UNIVERSITY OF HONG KONG

Department of Anaesthesiology

LECTURE NOTES

PREFACE

These lecture notes are NOT intended to function as a textbook. Several useful texts already exist which are suitable for medical students, and are listed at the end of the notes.

The principle purposes of the following pages are:

1. To ensure that an accurate record of the lecture material is available to the student. Experience shows that notes taken in haste during a lecture are rarely accurate, and never complete.

2. To provide a concise document which will be useful for revision.

CONTENTS **PAGE**

INTRODUCTION TO ANAESTHESIA

Anaesthesia is a means to an end. It is neither diagnostic nor therapeutic except in certain very limited ways, but almost always serves to make a procedure (such as an operation) or an experience (like having a baby) painless. It is never trivial; even in the best circumstances, with highly trained individuals delivering the service, there is a hazard which can never be totally eliminated, although risk reduction has made great progress, and the mortality from anaesthesia is being steadily reduced.

The more you know about anaesthesia, the more able you are to anticipate what might go wrong, and the better prepared for trouble you are, the less likely it is to get out of control. The most important single principle of anaesthesia is that

IT IS BETTER TO STAY OUT OF TROUBLE THAN TO GET OUT OF TROUBLE

If risks are taken, the stakes are provided by the patient, not by the anaesthetist. There are no prizes for risks taken unnecessarily which you get away with. The penalty for failure is paid by the patient and usually he pays with his life. Everything must be done, therefore, which will minimise risk, and the most important method of risk reduction is adequacy of preparation. This means meticulous workup for the elective case, and vigorous resuscitation for the emergency.

Seriously ill patients who are adequately prepared and skilfully anaesthetised will never be better perfused or oxygenated than they are under anaesthesia, because at the same time they are connected to a life support system driven by a skilled person. Often the most dangerous time of the experience is when this support is withdrawn; so much so that it is frequently necessary to take the patient to the Intensive Care Unit where many of the techniques of anaesthesia in the operating suite are continued in a slightly modified manner until the patient is out of danger.

HISTORY OF ANAESTHESIA

By general agreement, the credit for administering the first anaesthetic is given to William T.G. Morton, a dentist of Boston, U.S.A. Although he was probably preceded by the physician Crawford Long of Georgia, it was Morton's successful public demonstration in 1846 to an audience of sceptical surgeons which won him the prize. The patient, Gilbert Abbott, is the central figure in a large painting which now hangs in the "Ether Dome" of the Massachusetts General Hospital, where the demonstration took place.

Not long after Morton's demonstration, Simpson, the Scottish obstetrician, introduced chloroform to relieve the pain of childbirth, which led to Queen Victoria requesting analgesia for the birth of Prince Leopold, her 8th child. She was attended for this purpose by John Snow, a remarkable and scientific anaesthetist. His untimely early death at the age of 45 deprived anaesthesia of an intellect which would have guided the specialty into better and more productive pathways than were followed in the second half of the 19th century, when the insatiable demand for anaesthetists led to its administration by the unqualified and the inexperienced, often with deplorable results.

It was not until 1929 that Ralph Waters was appointed to the world's first Chair of Anaesthesia at the University of Wisconsin; whilst the first British Professor, Robert (later Sir Robert) Macintosh, was appointed in 1937, a jubilee which was celebrated this year in Oxford.

Between these events, another significant step was taken by the Mayo Clinic, in appointing the first Director of Anaesthesia, John Lundy, who was largely responsible for the introduction of thiopentone (pentothal) into clinical practice. The significance of that event should not be overlooked; more patients have been given this drug than any other therapeutic substance administered by physicians. Every year it is given to 20 million Americans. 3.5 million Britons, and 1.5 million Australians. The figures for China are not available at this time.

In Hong Kong, the first qualified specialist anaesthetist, Dr. Z. Lett, arrived in 1954, and for some years was the only one. There are now two professors and nearly 100 qualified specialists, but there is room for many more.

PREPARATION OF THE PATIENT FOR

ANAESTHESIA AND SURGERY

An important event in the pre-operative work-up is the pre-anaesthetic consultation. The following summary of the reasons for the consultation, and how it should be done is adapted from the recommendations of the Faculty of Anaesthetists, Royal Australasian College of Surgeons:

The purpose of this consultation is to ensure that the patient is in the optimal state for anaesthesia and surgery. It requires additional skills and judgement which differ from those involved in the administration of the anaesthetic.

General Principles:

1. The pre-anaesthetic consultation should be performed by the anaesthetist who is to administer the anaesthetic.

2. This consultation should take place at the appropriate time prior to surgery and anaesthesia, so that any necessary therapeutic or investigatory measures can be performed.

3. Notwithstanding (2), it is acknowledged that early consultation is not always possible (e.g. emergency surgery), but assessment is still necessary prior to commencement of anaesthesia and surgery in these circumstances.

Recommendations:

The pre-anaesthetic consultation should include:-

1. Identification of the patient.

2. Confirmation of the nature of the procedure and where applicable, the side on which it is to be performed.

3. A medical history and clinical examination of the patient, including a review of any current medication and results of any special investigations.

4. Arrangement of any further therapeutic or investigatory measures.

5. Consultation with colleagues in other medical or surgical disciplines where appropriate. (Physicians, Intensivists, etc.).

6. An attempt to satisfy the psychological needs of the patient.

7. The ordering of appropriate pre-medication if considered necessary.

8. A written summary which becomes part of the medical record of the

Although the pre-anaesthetic consultation usually takes place in Hospital on the day before surgery, it may also be carried out on an outpatient basis, especially if the patient's operation is to be done in a day surgery unit.

The anaesthetist's assessment will include history-taking, physical examination and review of investigations, but with modifications directed towards the special considerations of anaesthesia, and the need to review as many patients as are for operation the following day within a reasonable time. Important aspects of history which must be covered are:

Previous history of anaesthesia, including complications, if any

Drug sensitivity
Relevant family history (of response to anaesthesia)
What medications, if any, the patient is taking

The principal systems which require review are as follows:

The heart: Is it healthy? Ask about exercise tolerance.
If not, what is wrong with it?
Has an ECG been done?
Has a chest X-ray been taken?
Can function be improved?
If so how?

The lungs: Are they healthy? Does the patient cough? Any sputum?
Does the patient smoke?
Has a chest X-ray been taken?
Has anyone looked at it?

The Kidneys: Are they functioning normally?
How do you know?
What tests have been done?

The blood: What is the haemoglobin?
Is the coagulation mechanism normal?

A particular concern for the anaesthetist is the patient's airway, and an assessment must be made of difficulties which may arise during intubation. Underdevelopment of the lower jaw, restricted opening of the mouth, and upper teeth which are considerably anterior to the lower teeth (Class II malocclusion) are all indicators of possible difficulty. Inability to visualise the posterior pharyngeal wall with the mouth open maximally and the tongue protruded is also believed to be associated with difficult intubation.

Loose teeth must be noted, and the patient warned of the possibility of dislodgment, whilst the veins of the arms should be checked for I.V. access which will be necessary.

If the patient is on medication,

Should it be continued?
Should it be discontinued or modified?
Will any of the drugs interact with anaesthesia?

Is any special preparation necessary for the procedure - e.g. beta blockers for thyrotoxicosis, parenteral vitamin K in the jaundiced patient, increased dosage for the steroid dependent.

Will blood be needed?
Has the patient been grouped?
Is cross-matching necessary?

Is Intensive care going to be necessary post-operatively,
e.g. in aortic surgery?
Is a bed available?

What kind of anaesthetic technique will be used?
Is all the necessary equipment ready and available?

Has the patient given consent?

This is not an exclusive list, but covers the main points which must be answered on every occasion.

At the conclusion of the consultation, the anaesthetist should make an assessment of the patient's risk status. This is normally recorded according to the American Society of Anaesthesiologists' classification, viz:

Class 1 - A normal healthy patient

Class 2 - A patient will mild to moderate systemic disease caused by the condition to be treated surgically or by any other disease

Class 3 - Severe systemic disease or disturbance from any cause

Class 4 - Severe systemic disorders that are already life threatening

Class 5 - The moribund patient with little chance of survival for more than 24 hours

Emergency is denoted by the letter E

PREMEDICATION:

The purpose of premedication is to relieve anxiety. Nevertheless it has been shown that the visit by a concerned, sympathetic anaesthesiologist is more effective than potent pharmacological agents in achieving this goal.

FASTING:

Prior to elective surgery, and in the interests of preventing complications from aspiration of gastric contents, complete fasting discipline is imposed for a minimum of 6 hours in adults; i.e. nothing to eat or drink. In children under 5 years this is modified to allow clear fluids up to four hours before anaesthesia, whilst infants may be breast-fed up to 3 hours previously.

SPECIAL CONDITIONS:

Diabetes, Thyrotoxicosis, anaemia and a number of other chronic conditions require special pre-operative management. In the case of diabetes hypoglycaemia is especially dangerous should it occur while the patient is unconscious, and the preparation of the diabetic patient is complicated by the need for fasting. Recent practice favours the combination of a dextrose and insulin infusion.

Thyrotoxicosis calls for particular care in producing a euthyroid patient. Apart from specific inhibitors of thyroxine production, beta blockade is also called for.

The chronically anaemic patient is a special case, and in some, but by no means all circumstances, may need pre-operative transfusion. It is potentially hazardous to attempt rapid improvement in haemoglobin since the anaemic patient often has an increased blood volume, and unless infusion is very cautiously carried out there is serious risk of overloading.

PRE-OPERATIVE PREPARATION IN EMERGENCIES

Time is short in the preparation of an emergency case for surgery and anaesthesia, but some conditions MUST be corrected before the patient is anaesthetised. Most important of these is BLOOD VOLUME, and to only slightly less extent EXTRACELLULAR FLUID VOLUME.

Electrolyte derangements and acid-base disturbance also require correction, and bronchospasm should be vigorously treated if present. In seriously ill patients for emergency surgery, the advice of the anaesthesiologist should always be sought so that the time available can be efficiently employed in making the patient as fit as possible.

GENERAL ANAESTHESIA AND THE CARE OF

THE UNCONSCIOUS PATIENT

In choosing the technique of anaesthesia to be employed, the first choice to be made is between general and local (or regional). General anaesthesia results in loss of consciousness or coma, a state which has hazards of its own, which will be discussed before proceeding to the details of how general anaesthesia can be produced.

THE HAZARDS OF UNCONSCIOUSNESS

When consciousness is abolished, either deliberately by the anaesthetist, or as a consequence of trauma or disease, serious physiological aberrations occur immediately which potentially threaten the patient's life and health. These are:

1. Muscle tone is lost. This involves the muscles of the upper airway, especially the tongue, which responds to the force of gravity. In the supine position, this can produce respiratory obstruction as the tongue falls back until it meets the posterior pharyngeal wall.

2. The laryngeal reflexes are deranged or abolished. Thus protection of the airways from contamination is either absent or inappropriate. In the latter case, the response is exaggerated so that laryngeal spasm may be so severe as to threaten asphyxia. In deeper states of unconsciousness, aspiration of liquids or solids may occur without impediment.

3. To a greater or lesser extent, depending on the depth of coma, vasomotor control is impaired, and hypotension follows. If this is accompanied by postural disadvantages, perfusion of the brain may fall to dangerously low levels.

4. The patient is unable to respond to nociceptive stimuli, and will therefore be prone to injuries of various kinds, e.g. to the eyes, peripheral nerves, and skin.

5. The bladder may overfill, and retention of urine be precipitated.

6. During the recovery phase the patient may be unwilling or unable to take sufficient fluids by mouth to maintain hydration.

All of these hazards may be reduced by special techniques, but even in expert hands cannot be totally eliminated. Some of them are not unique to anaesthesia (such as the lateral position during recovery), and have applications in the care of all unconscious patients.

Techniques of general anaesthesia therefore not only include the methods of putting patients to sleep, but also a range of other measures aimed at protecting them from the hazards of unconsciousness for the duration of anaesthesia and immediately afterwards.

The process of transforming a conscious patient into an unconscious or anaesthetised one is called INDUCTION. Some agents can achieve this result very rapidly - as little as one arm-brain circulation time. Others take much longer. The commonest method of induction in modern anaesthesia is by intravenous administration of a rapidly acting agent which crosses the blood-brain barrier promptly. By far the most frequently used drug of this kind is THIOPENTONE, a water soluble thio-barbiturate which has now probably been administered to more human being than any other drug in history.

This agent is made up freshly before use because its highly alkaline solution is unstable for more than a few days. It is administered in a 2.5% concentration, and is given intravenously as a SLOW bolus in doses ranging from 3-5mgm per kg. Older, frailer patients receive lower doses, and occasionally very resistant or apprehensive patients may need more.

Its action is rapid, since the time taken to cross the blood-brain barrier is negligible, and it is very predictable in its effects. As well as producing unconsciousness, it is a vasodilator and a myocardial depressant, actions which may therefore produce severe hypotension in predisposed patients - e.g. those suffering from unreplaced blood loss, or myocardial disease.

A single intravenous dose is rapidly REDISTRIBUTED throughout the ECF, and the blood level falls steeply so that recovery of consciousness is normally within 5 minutes of onset. Should additional doses be given, recovery is slower with each, because the drug is not metabolised rapidly, and cumulation occurs.

Furthermore, thiopentone, although a powerful hypnotic is not an analgesic, and the dose required to prevent movement from surgical stimuli is quite high. For all of these reasons, although it is an ideal induction agent, being quick and pleasant for the patient, it is unsuitable for the maintenance of anaesthesia, which must be achieved by other means.

Maintenance of general anaesthesia is therefore most often accomplished by adding INHALATIONAL agents after intravenous induction has been given. The inhalational agent may be A GAS or A VOLATILE LIQUID.

The only anaesthetic gas used nowadays is NITROUS OXIDE, and it is supplied to the patient via an anaesthetic machine which dispenses nitrous oxide, together with oxygen by means of flowmeters. The machine is supplied from sources which are either pipelines connected to a bulk supply, or reserve cylinders which are attached to the machine itself.

Nitrous oxide is a weak anaesthetic, and although when preceded by an intravenous induction it may enable a brief procedure to be done, it is insufficient for prolonged or stimulating surgical operations. The usual practice is therefore to supplement nitrous oxide by other agents, and these may be the volatile liquids referred to above, or intravenous narcotics, and sometimes both.

A range of volatile inhalational agents, all of them halogenated hydrocarbons, is now available. The commonest are:

HALOTHANE
ENFLURANE
ISOFLURANE

whilst of only historical value are

trichlorethylene
methoxyflurane

All of these agents necessitate the use of a VAPORISER, a device which converts the liquid to a vapour, and adds it to the nitrous oxide and oxygen mixture previously dispensed from the flow meters.

The final mixture of gases and vapours is administered to the patient by means of a BREATHING CIRCUIT, connected to the anaesthetic machine at one end, and to the patient at the other by a mask or an endotracheal tube. Breathing circuits are of different designs, some being arranged so that rebreathing of exhaled gases does not occur, whilst others permit rebreathing, and if so, may contain a chemical absorber which removes carbon dioxide from the gases before they are inhaled again.

The circulatory hazards of thiopentone have already been mentioned, but it should also be noted that it is a dangerous method of induction in patients with threatened airway obstruction, as in post-thyroidectomy bleeding, or upper airway compromise from malignant or infectious disease.

In the case of nitrous oxide, its dangers are solely related to the fact that it cannot support life unless oxygen is added to it in suitable concentrations. Thus if for any reason the oxygen supply fails, within a matter of minutes the patient breathing nitrous oxide alone will die from anoxia. Gas supplies and anaesthetic machines are fitted with a number of safety features to alert the anaesthetist to an oxygen supply failure, and also to prevent the accidental cross-connection of supply which might result in a patient being given nitrous oxide instead of oxygen.

The volatile agents are almost all potent agents which can kill through overdosage, respiratory failure usually preceding cardiac arrest, although sometimes the heart fails first. It is hardly ever necessary to use high concentrations of these agents, especially if they are used to supplement nitrous oxide and in combination with MUSCLE RELAXANTS.

These important drugs will be dealt with in another lecture, but at this time it can be noted that in combination with intravenous and inhalational anaesthetics, they can produce excellent operating conditions for the surgeon with much less cardiovascular impairment than would otherwise occur. They also have an important role to play in facilitating INTUBATION of the larynx.

The unconsciousness produced by anaesthesia results in the same problems as those which accompany coma from other causes, but because the anaesthetist is present, steps can be taken both before and after induction to minimise or even abolish these risks.

The anaesthetist's first concern in the management of general anaesthesia is the preservation of the airway. If the patient is expected to breathe spontaneously throughout the operation, and the case is an elective one, care of the airway may involve only postural manipulation of the head and jaw, or perhaps the insertion of a PHARYNGEAL AIRWAY. This device, usually a GUEDEL pattern, splints the tongue away from the posterior pharyngeal wall, and because it is hollow, provides a channel through which the patient can breathe.

In many circumstances, however, additional steps are necessary to maintain and protect the airway, and there is a need to intubate the larynx, inserting an ENDOTRACHEAL TUBE. It is possible to achieve this in a conscious patient with some difficulty, and it is not a pleasant experience for the patient. It can also be inserted after anaesthesia of some depth has been produced by inhalational agents. In by far the greatest number of cases, it is performed after the patient has been given an intravenous induction, followed by a muscle relaxant. This produces conditions which allow the early and non-traumatic insertion of a LARYNGOSCOPE by means of which the larynx is located, and the tube may be passed under direct vision.

The indications for endotracheal intubation are:

1. The presence of a full stomach, as in emergency cases.
2. Postural needs of the surgeon, e.g. the prone position.
3. Competition for the airway, e.g. in tonsillectomy
4. Need to control ventilation, i.e. when muscle relaxants are given, or the patient's own respiratory function is poor.
5. When the chest is opened.
6. To protect the lungs from aspiration when the surgery is intra-abdominal.
7. To reduce apparatus dead space, especially in children.
8. To overcome or prevent obstruction of the airway where this might be anticipated, e.g. in thyroidectomy.
9. When access to the patient is denied to the anaesthetist, e.g. in head and neck surgery.

During general and local anaesthesia it is essential to have reliable venous access, which usually means the insertion of a cannula of appropriate size into an accessible vein in the arm or hand, rarely the leg, and occasionally the neck.

COMA

In addition to the acute dangers referred to on p. * , additional problems arise during prolonged coma. Although principally respiratory and metabolic in origin, they can secondarily affect the circulation, the kidney and in turn, the brain. It is often these complications which determine the outcome of the illness, and their prevention and/or management is vital to the survival of the patient suffering from prolonged unconsciousness from whatever cause.

Even if the airway is protected from obstruction and contamination, when the normal mechanisms of coughing are abolished, secretions accumulate at a rate which depends on the prior health of the respiratory tract. Eventually, however, even in a previously normal respiratory tree, retained secretions will cause blockage of small airways, become infected, and a deadly combination of collapse and pneumonia will reduce arterial oxygen saturation.

Impaired tissue delivery of oxygen will have serious effects on an already compromised brain, and may even affect cardiac output. Every effort must therefore be made to postpone these pulmonary complications, and if they do occur, to deal with them vigorously.

Prevention is in every way preferable. To achieve drainage of secretions, posture and physiotherapy are the mainstays of conservative prophylaxis. Turning the patient at regular intervals, and not less often than second-hourly, encourages drainage of the uppermost lung, and most of the secretions will enter the trachea, and even drain into the pharynx, where they can easily be retrieved by suction.

Physiotherapists use techniques of percussion and vibration ("percs and vibes") designed to loosen secretions situated more peripherally. By a combination of these methods, useful drainage can be achieved, and in coma lasting only two or three days may be all that is needed to keep the lungs healthy until recovery has occurred.

In more prolonged states, and especially where it has been necessary to intubate, invasion of the bronchial tree by suction catheters will be necessary. Indeed, intubation may be required to enable efficient removal of secretions which threaten to drown the patient. Once an endotracheal tube is in place, problems of HUMIDIFICATION and INFECTION now arise.

Humidification of the lower respiratory tract is normally very efficient, the nose and upper airways ensuring that gas is at 100% saturation with water vapour at 37 degrees C by the time it enters the glottis. However, bypassing the upper airways by ET tube results in gas being delivered to the bronchi which is at whatever ambient conditions prevail. If the patient is being ventilated by piped or bottled gases, these are completely dry unless steps are taken to provide humidity artificially. Secretions may now become extremely tenacious and difficult to remove, even by suction. Occasionally, even bronchoscopic removal will be necessary.

Apparatus exists which can provide humidification, but its technical details are beyond the scope of this lecture.

Respiratory infection is a constant threat to the unconscious patient, and can only be prevented by meticulous attention to the principles of asepsis in dealing with the airway, as well as such other measures as mouth hygiene and the prompt removal of accumulated secretions. Suction and manipulation of ET tubes and their connections must always be done with gloved hands and sterile disposable equipment, and exceptional care must be taken when moving from one patient to another. The contents of humidifiers must at all times be sterile, and breathing circuits, when used, changed at frequent intervals, as well as being carefully sterilised before use.

Apart from the pulmonary complications of prolonged coma, there are also metabolic consequences which require careful management. Voluntary fluid intake is impossible, and a valuable regulatory mechanism is therefore lost. Enteral feeding may be possible, but is risky if the patient's airway is not protected by an ET tube or tracheostomy, whilst if it is, the presence of an indwelling naso-gastric tube introduces problems of erosion and fistula formation, due to compression between the two appliances of the intervening soft tissues of the oesophagus and trachea.

For the most part, therefore, comatose patients' fluid, electrolyte and even nutritional needs are supplied intravenously.

During the first 24 hours, whilst an assessment is being made of the likely duration of coma, fluid and electrolyte requirements only need be given. Considerable controversy surrounds the rules which should be observed at this time. In general, fluid restriction is practised, with a ceiling of 1ml/kg/hour. Some work suggests that dextrose may encourage raised intracranial pressure (ICP), and injury to the brain often leads to inappropriate ADH secretion with water retention and hyponatraemia, a situation which will be greatly worsened by the administration of large volumes of salt-free solutions.

Should raised ICP be suspected, another problem arises in how to treat it. The common practice of giving an osmotic diuretic such as mannitol, is effective temporarily, but there is always a rebound in a few hours, and repeated infusions have less and less effect. There is also the possibility of haemoconcentration.

Not only, therefore, is the fluid management of coma no easy matter, but it is difficult to lay down routines. Each patient must be very closely followed for electrolyte status, urinary output, arterial blood pressure and possibly central venous pressure. In the case of comatose closed head injuries, monitoring of intracranial pressure is also regarded more and more as an essential feature of management.

Nutritional requirements need not be considered until it is evident after, say, 48 hours, that the patient will remain unable to be enterally fed for some time. At that stage it is important to initiate a regime of total parenteral nutrition which will conserve protein and body weight, but once again the potentially unfavourable effect of dextrose on ICP must be kept in mind.

Irrespective of whether enteral feeding is being given or not, stress ulcer prophylaxis is essential, particularly if head injury is the cause of coma. H2 blockers should therefore be commenced early.

Temperature must be controlled, and hyperthermia is especially to be prevented. Mild hypothermia, to 35 degrees may be beneficial, but the deeply comatose patient may rapidly cool to very low temperatures, with resulting arrhythmias and reduced cardiac output.

Remember that the skin and underlying nerves are vulnerable to pressure injury. Frequent changes of posture are necessary, and sources of heat must be guarded. The bladder must be drained in as sterile a manner as is possible, if the patient does not void. The eye may be abraded or ulcerated if the cornea is allowed to dry out. Due to the generalised lack of tone, wrist and foot drop can develop with surprising rapidity, and may take months to recover.

CLOSED HEAD INJURY

The special problems of closed head injury merit separate consideration. No attempt will be made here to deal with open head injuries, or those which require drainage of intracranial collections, so the ensuing discussion applies only to the non-operative management of head injury.

Even though no skull fracture or large intracranial vessel injury has occurred, damage to the brain may be such as to produce unconsciousness. Nevetheless, the prognosis for these injuries, i.e. those in which no laceration of the brain substance, and no massive contusion has taken place, should be good, provided that care is well organised and directed.

The aims of management are:

1. Early detection of neurological deterioration
2. Prevention of secondary cerebral insults
3. Early treatment of concurrent medical and surgical problems

Hence immediate attention must be paid to the airway, breathing and circulation on admission, and if in doubt, the patient should be intubated. Institution of a reliable method of observation must commence immediately. The Glasgow Coma Scale is now an internationally recognised system, and is reproduced below:

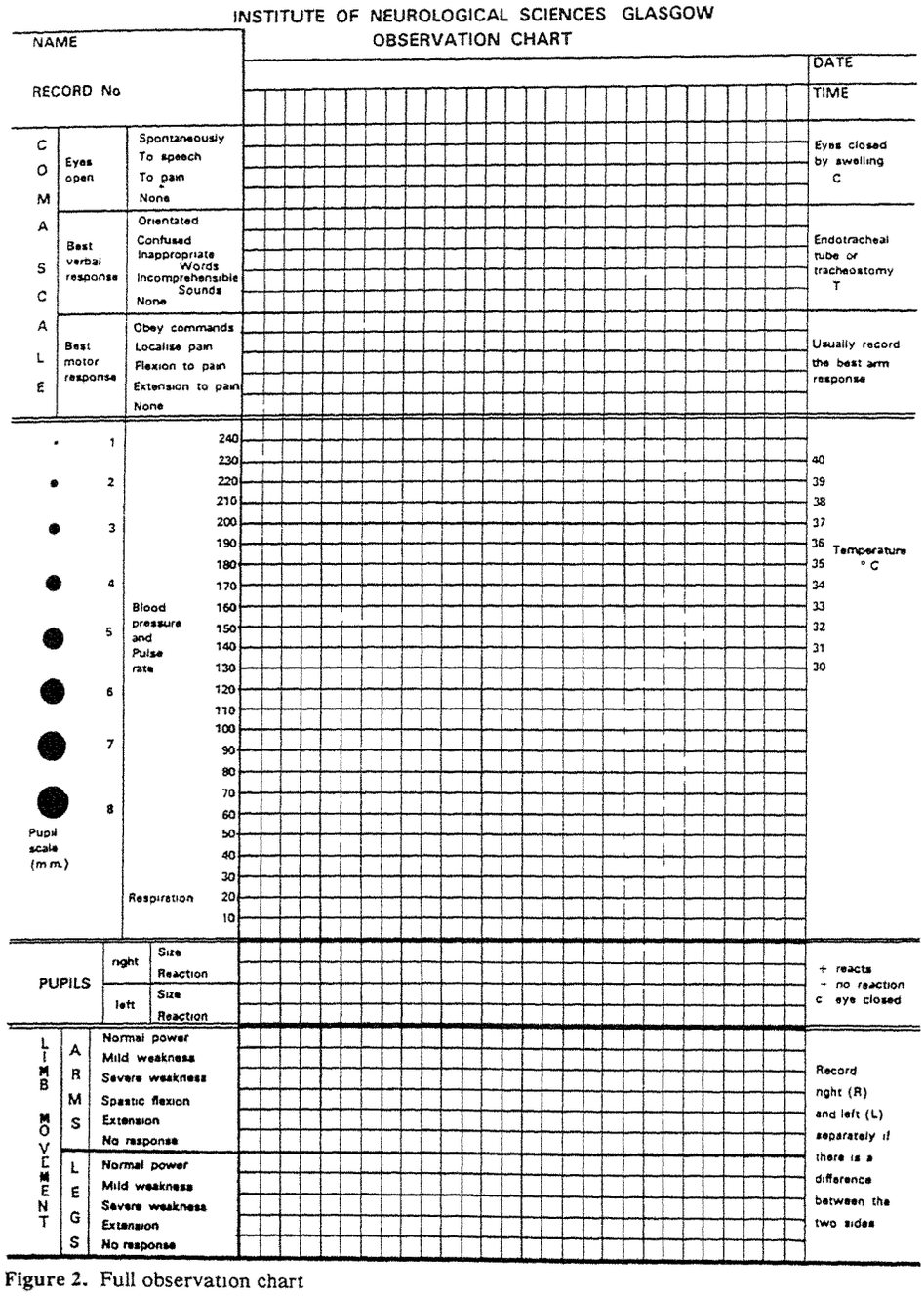


Figure 2. Full observation chart

Scoring the Glasgow "Coma" Scale

Eye Opening		
spontaneous	E	4
to speech		3
to pain		2
nil		1
Best motor response		
obeys	M	6
localizes		5
withdraws		4
abnormal flexion		3
extensor response		2
nil		1
Verbal response		
orientated	V	5
confused conversation		4
inappropriate words		3
incomprehensible sounds		1
nil		1

Coma score (E + M + V) = 3 to 15

* 90% of patients with a score of 8 or less define coma

b. Motor Response Pattern - hemiplegia
- bilateral or unilateral
- focal signs

c. Pupil Reaction and Size - focal damage to globe or cranial nerves
- brainstem function
- depth of coma
- drugs - either systemic or local

d. Eye Movements - with head movement (oculocephalic)
- with caloric stimultion (oculovestibular)
- spontaneous

* a high coma score > 8 is almost always associated with intact eye movements.

If observation along these lines reveals a deterioration in the patient's condition, the cause must be sought and action taken. As in all unconscious patients, it is essential to prevent hypoxia and hypocarbia, but in the case of closed head injury, the need is so vital as to require intubation and ventilation in any patient whose Glasgow score is less than 8.

Apart from hypoxia and hypocarbia, other factors which are less well understood may contribute to a rise in intracranial pressure (ICP). Such a rise is always deleterious, and many methods have been advocated in dealing with it, or preventing it, but none is as important as the preservation of blood gas homeostasis.

Measures which are advocated for the prevention/treatment of raised ICP are as follows. The effectiveness of each is shown on a +/- scale.

Posture (head up, neck not twisted)	+++
Controlled ventilation	++++
Ventricular drainage	+++
Mannitol	++++ initially +/- later
Steroids	+/-
Frusemide	+/-
Barbiturates	+++ for paroxysmal rises - in long term IC hypertension

A serious additional insult is seizure activity. Even if the muscular component is controlled and the airway safeguarded during fits, there is still a substantial rise in cerebral oxyegn consumption. Control of seizure activity is therefore always necessary. Acutely, thiopentone is the most effective drug (2 mgm/kg), followed by diazepam in a titrated dose beginning with 2 mgm IV. Phenytoin is only suitable for long-term prophylaxis, and is useless in the management of active convulsions. Once this drug has been given, it must be controlled by serum estimations, and kept within the range of 40-80 mmol/L.

Always keep in mind the possibility of hyponatraemia and pyrexia as contributing causes to seizures.

The existence of concurrent medical and (particularly) surgical problems demand attention if the head injury itself is to be managed successfully. For example, compound fractures of limbs must at the very least be immobilised, since the stimulation which otherwise occurs when the patient is moved will lead to rises in ICP. If abdominal or thoracic injuries are present they must be dealt with in the normal manner. In particular, visceral lesions which involve bleeding or perforation require urgent treatment, the priority for which takes second place only to drainage of an intracranial collection.

BRAIN DEATH

Sometimes the damage to the brain is such that survival is impossible, and the stage is reached when, although other systems are still functioning nearly normally, there is no neurological activity detectable in the brain. If this is so, it is important to determine whether the brain is indeed "dead" or whether any other factor might be responsible.

The two most important causes to eliminate are drugs and hypothermia. If both can be excluded, the former usually by the withholding of medication for 24 hours, then confirmatory tests of brain death can be carried out, viz:

Clinical tests of brainstem function:

Pupillary light reflexes
Corneal reflexes
Oculocephalic (doll's eye reflex)
Oculovestibular reflex (cold caloric reflex)
Pharyngeal and laryngeal reflexes
Apnoea in the presence of a pCO_2 of 60 mm (8 kpa)
Absence of cardiac response to atropine

The straight-line EEG is no more reliable than the above clinical tests, and may indeed be less so. Medico-legally, however, it is probably necessary to perform the EEG. It is not necessary to perform angiography, but if the latter is done, and demonstrates no cerebral perfusion (due to an ICP which exceeds mean arterial pressure) this is conclusive proof of brain death.

In most communities now, this condition is recognised as being the equivalent of clinical death, and the victim's organs may, with the consent of the family, be harvested for transplantation. It is important that, not only should these opportunities for life not be denied to other patients, but also that scarce ICU resources should not be devoted to the futile maintenance of cardiopulmonary function in patients with no chance of cerebral recovery.

Hospitals have, or should have, well documented procedures for the proper and ethical certification of brain death, as well as sympathetic programmes for the support of bereaved relatives in these difficult circumstances.

CARDIOPULMONARY RESUSCITATION (C.P.R.)

BACKGROUND:

1. Modern CPR was commenced in 1960 with the rediscovery of mouth to mouth ventilation, external chest compression and application of external defibrillation.

2. Ventricular fibrillation is the cause of cardiac arrest in the majority of victims and carries a better prognosis. However the most important factor in producing a favourable outcome is minimising the interval between collapse and the institution of basic CPR.

Recent Advances in the Physiology of External Cardiac Massage (ECM)

1. Coronary blood flow and survival are related to mean aortic pressure.

2. Coronary blood flow is nearly zero during chest compression.

3. Output during ECM is not due to direct compression.

Technical factors which are important in successful ECM are:

1. Complete release of compression to allow adequate diastolic coronary perfusion.

2. Use of adrenaline to support mean aortic pressure.

3. Compression must be applied over the lower HALF (NOT third) of the sternum.

4. Adequate ventilation must be performed, and simultaneous chest compression and ventilation is not recommended.

Other points on technique:

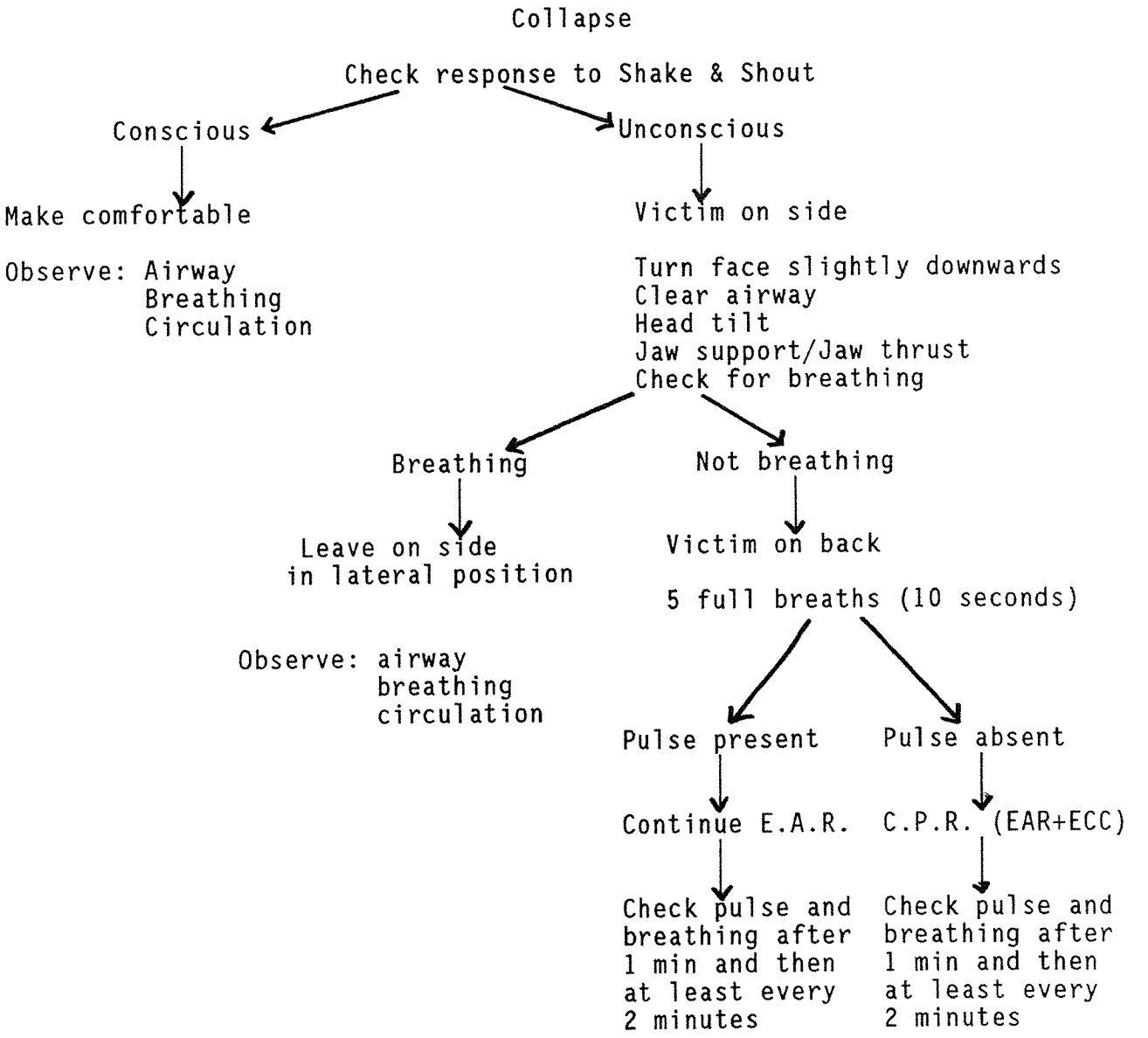
The patient must be on a firm, horizontal surface. Elevating the lower limbs may be useful by augmenting the central blood volume. A praecordial thump or blow is not effective and may even convert a weak sinus rhythm to a ventricular arrhythmia.

Adult ECM at 60/minute should allow 0.5 second for compression and other 0.5 second for ventricular filling.

Do not use intracardiac injections.

Adrenaline, atropine and lignocaine can be given via the ET tube, but IV administration is better. Always use veins in the upper half of the body.

CPR FLOW CHART



E.A.R. = Expired air resuscitation
E.C.C. = External cardiac compression
C.P.R. = Cardiopulmonary resuscitation = E.A.R. + E.C.C.

BASIC C.P.R.

First Steps of CPR

1. Establish unresponsiveness

2. Call for assistance

3. Place patient in the lateral position

   advantages:

   a. gravity assists the tongue to fall away from the pharynx
   b. vomitus, saliva, mucus will gravitate to the lower cheek or from the victim's mouth
   c. the victim is stable in this position
   d. the victim doesn't suffer further injury to the lower brachial plexus or arm

4. Clear AIRWAY

   a. tilt head backwards with face turned slightly downwards
   b. clean mouth of foreign material using fingers if necessary
   c. extend the neck
   d. support jaw at the point of the chin or use jaw thrust with forward pressure at the angle of the lower jaw

5. Check for BREATHING

   a. if breathing leave patient in lateral position and attend to the airway
   b. if not breathing, turn patient on his back and commence E.A.R. immediately - give 5 <u>full</u> breaths within 10 seconds

6. Check for CIRCULATION

   a. feel for the carotid pulse
   b. if the victim has a pulse but is still not breathing, continue E.A.R.
   c. if the victim has no pulse commence C.P.R.

EXPIRED AIR RESUSCITATION

Methods:

1. Mouth to Nose - resuscitating babies where the rescuer's mouth covers the victim's mouth and nose.

2. Mouth to Mouth

   i. tilt - place palm of one hand on top of the head, support the chin with the other hand and firmly but gently tilt the head backwards.

   ii. blow - take a deep breath, open your mouth as widely as possible and place it over the victim's slightly open mouth, sealing the nose with the cheek. Blow to inflate the victim's lungs.

   iii. observe the victim's chest - if it does not rise, there is

      a. airway obstruction
      b. an inadequate seal
      c. inadequate air being blown into the lungs

   iv. listen and feel for exhalation - following inflation, lift your mouth from the victim's, turn your head towards the chest and with your ear about 2cm from the victim's mouth listen and feel air being exhaled.

   v. give 5 inflations in 10 seconds.

   vi. check the carotid pulse - if a pulse is felt, continue EAR at 15/minute until the victim breathes effectively.

   - check the carotid pulse every 2 minutes.

   If the pulse is absent the victim has had a cardiac arrest, he is:

   UNCONSCIOUS
   NOT BREATHING
   PULSELESS

   - commence C.P.R.

EXTERNAL CARDIAC COMPRESSION

Position for the hands:

The rescuer should identify

a. the lower 1/2 of the sternum (compression site for adults)

and

b. the mid point of the sternum (compression site for infants and children)

Method of Compression:

a. Place the heel of one hand on the compression point with the fingers relaxed, parallel to the ribs and slightly raised so that pressure is not exerted directly on the ribs.

b. The other hand is placed securely on top of the first.

c. All pressure is exerted through the heel of the under hand, and the body weight should be the compressing force, so that the shoulders should be vertically over the sternum and the compressing arm kept straight.

Position of Rescuer:

The rescuer should kneel opposite and at right angles to the victim's chest.

Rate of ECM:

60 compressions per minute in an adult

and

100 compressions per minute in a child or baby.

C.P.R.

One operator

For Adults:

2 ventilations and 15 compressions in 15 seconds (i.e. 4 cycles/minute) .

For children:

2 ventilations and 15 compressions in 10 seconds (i.e. 6 cycles/minute).

Two operators

For Adults:

1 ventilation and 5 compressions in 5 seconds (i.e. 12 cycles/minute).

For Children:

1 ventilation and 5 compressions in 3 seconds (i.e. 20 cycles/minute).

* Where there are two operators, there should be no pause in compressions and the inflation should be interposed between the last compression of one cycle and the first of the next.

* Check for the return of carotid pulse every 2 minutes.

ADVANCED LIFT SUPPORT (A.L.S.)

The A.L.S. team requires some essential equipment

1. Oxygen source and method of delivery to the patient.
2. Self inflating bag/non-rebreathing valve/mask/universal connector (15/22mm).
3. Oropharyngeal airways, endotracheal tubes, laryngoscopes.
4. Suction apparatus.
5. ECG monitor.
6. Defibrillator.
7. I.V. apparatus and drugs.

Plan of Management

1. Secure the airway and ventilate with 100% oxygen.
2. Blind defibrillation if there is any delay in obtaining ECG monitoring.
3. Continuing CPR if pulse is absent or unsatisfactory.
4. Management of arrhythmias.
5. Post-resuscitation care.

DEFIBRILLATION

a. If the ECG diagnosis is VF, immediately defibrillate using 5 Joules/kgm (400 Joules for the average adult).

   i. If successful reversion to a supraventricullar rhythm then administer an antiarrhythmic such as lignocaine I.V.

   ii. If unsuccessful repeat shock (400J) should immediately be given.

b. resistant ventricular fibrillation (VF)

   1. check airway and ventilation
   2. maintain external chest compression
   3. establish I.V. line
   4. give adrenaline 10mls of 1:10,000 solution I.V.
   5. repeat defibrillation (400J)
   6. repeat adrenaline if V.F. persists
   7. if arrest time > 5 minutes give 1ml/kg of $NaHCO_3$ 8.4% I.V.
   8. repeat defibrillation (400J)

DRUGS IN CARDIAC ARREST

1. Adrenaline

   1. Presently the drug of choice because of α + ß receptor stimulating properties, particularly its peripheral vasoconstrictor action.

   2. Elevates perfusion pressure and hence the contractile state of the heart and stimulates spontaneous contraction.

   3. Adrenaline should be repeated at 5 minute intervals becuase of its short half life.

2. Lignocaine

   a. Intravenous lignocaine is the antiarrhythmic drug of first choice for

      1. treatment of VT
      2. prevention of VT and VF after reversion of VF by defibrillation to a supraventricular rhythm

   b. Loading dose is 1mg/kg IV over 3 minutes (or 2mg/kg via ETT) followed by infusion of 2gm lignocaine/500ml 5% dextrose run at

      4mg/min (60ml/hour) for first hour
      3mg/min (45ml/hour) for second hour
      2mg/min (30ml/hour) for third hour in adults

   c. Lignocaine raises the defibrillation threshold and its use in resistant VF is contraindicated.

3. Sodium Bicarbonate

   a. The most important time for bicarbonate administration is immediately after restoration of an adequate perfusion pressure when lactate is "washed out" of tissue beds which were previously inadequately perfused.

   b. In the hospital setting bicarbonate administration should always be guided by frequent analysis of blood gases.

   c. Hazards of bicarbonate therapy are

      i. hyperosmolality
      ii. disequilibrium coma
      iii. congestive cardiac failure
      iv. rebound alkalosis
      v. increased lactate production
      vi. shift Hb/O_2 saturation dissociation curve to the left

d. Effects of bicarbonate on the cardiovascular system

$NaHCO_3$ --> increases extracellular fluid osmolality\
increases extracellular $[Na^+]$ | decreases
decreases ionized $[Ca^{++}]$ | myocardial
decreases serum $[K^+]$ / contractility

--> increases extracellular fluid volume } increase left ventricular stroke work
increases preload } increase cardiac output and blood pressure
decreases peripheral resistance (afterload) } increases cardiac outp

4. Calcium

a. Calcium is no longer recommended in the management of cardiac arrest except when hyperkalaemia is known to be present.

b. The reason for this change is because Ca^{++} ions are known to be mediators of cell death after hypoxic injury to the brain.

WHEN THE HEART FAILS TO START

a. check for recording malfunction

b. secure airway and ventilation

c. continue CPR

d. insert I.V. line

e. give adrenaline 10ml 1:10,000 I.V. over 30 seconds

f. if estimated arrest time > 5 minutes give 1ml/kg 8.4% sodium bicarbonate I.V.

g. repeat I.V. adrenaline 1ml of 1:1000

h. if patient remains in asystole continue CPR for five minutes then repeat I.V. adrenaline 1ml 1:1000

How Long should resuscitation be continued?

1. Once begun it should continue until all attempts to achieve a stable cardiac rhythm have failed.

2. In general it is reasonable to curtail resuscitation attempts when the adult patient has been in asystole for more than about 20 minutes and is not hypothermic.

3. The pre-arrest condition of the patient should influence the arrest team's decision.

POST-RESUSCITATION CARE

1. Transfer and monitoring in a critical care unit

2. Immediate blood gas and serum electrolyte estimations

3. Continued antiarrhythmic therapy

4. Circumstances leading to the collapse should be clearly defined and if possible, prevented from recurring.

THE NEUROMUSCULAR JUNCTION

Skeletal (voluntary) muscles are innervated by myelinated motor nerve fibres which arise in the anterior horn cells of the spinal cord, or in the brain stem. Where these fibres innervate the muscle, they do so via a complex structure which is the site of action of many potent pharmacological agents.

Ultramicroscopically, the neuromuscular junction has been revealed in great detail, and the following is a simplified description of the histology of the region.

As the axon approaches the surface of the muscle fibre, it expands and divides into a number of motor endplates, each endplate contacting an individual muscle fibre. Where the membrane of neural origin comes into contact with the muscle fibre, a specialised area of the muscle membrane also develops, and the combination of the two forms the physiological unit via which nerve impulse effects a contraction of the muscle fibre.

Within the neural component of the structure, vesicles containing acetylcholine await the stimulus of a nerve impulse for their rupture and discharge into the cleft between the two components of the endplate. Precursors of acetylcholine exist within the cytoplasm of the neural component.

The muscle-derived component contains receptors which respond to acetylcholine, but they are not wholly specific, and can be occupied by other substances, in particular by the class of drugs known as muscle relaxants.

Within the cleft itself, cholinesterase begins to hydrolyse acetylcholine as soon as it is discharged, ensuring that the effect is transient. The components are reabsorbed into the neural terminal where they are recycled. Drugs with anticholinesterase activity therefore allow the accumulation of acetylcholine in the cleft, prolonging and intensifying its action.

Once acetylcholine has occupied its receptor on the muscle component of the neuromuscular junction, a complex series of electrochemical events takes place culminating in the contraction of the muscle fibre. Channels in the membrane open, admitting sodium and calcium ions, whilst potassium ions escape into the extracellular space. A substantial shift in electrical charge depolarises the muscular component of the end-plate, and it is this electrical event which is then followed by muscle contraction. Return to the previous resting potential of -90 mv is necessary before another contraction can be initiated.

The foregoing basic physiology is essential to an understanding of the action of muscle relaxants, of which there are two main types in clinical practice.

The non-depolarisers, also sometimes called competitive blockers, occupy the acetylcholine receptors without activating them. When 70% of the receptors are so occupied, the onset of clinical paralysis ensues. Effective muscle relaxation for the performance of abdominal surgery requires 90% occupancy.

A non-depolarising block has the following characteristics:

1. there is decreased reaction to a single impulse

2. the response to a tetanic stimulus is not sustained, i.e. it fades
3. At levels of occupancy of 70% and over, response to repeated stimuli at 1 Hz is characteristic. The "train-of-four" stimuli can be used as a rough guide to percentage occupancy.

4. no fasciculation occurs prior to the onset of paralysis

5. a single stimulus arriving after a period of tetany produces an enhanced response ("post-tetanic facilitation")

6. it is antagonised by anticholinesterases.

There are many drugs in clinical practice which produce these effects:

Generic name	Commercial name	Onset	Duration
d-tubocurarine (curare)	tubarine	slow	long
alcuronium	alloferin	medium-slow	medium
pancuronium	pavulon	medium-slow	long
vecuronium	norcuron	medium	short
atracurium	tracrium	medium	short

The other class of muscle relaxants, the depolarisers, is nowadays represented by a single drug, suxamethonium (succinylcholine). When this molecule occupies the receptor it triggers a depolarisation similar to that which follows acetyl- choline occupancy, but the membrane does not recover as rapidly because the destruction of succinylcholine depends on pseudo-cholinesterase, and takes longer. Nevertheless, the onset of paralysis is very rapid, and duration is ultra-short, making this agent ideal for certain purposes in which speed of onset and brevity of duration are important considerations.

The characteristics of depolarising block are:

1. Decreased response to a single impulse

2. Decreased amplitude, but sustained response to tetanic stimulation, i.e. there is no fade

3. the train of four is not diagnostic of receptor occupancy

4. muscle fasciculations occur prior to paralysis

5. there is no post-tetanic facilitation

6. anticholinesterases prolong the block

The pharmacokinetics of muscle relaxants vary. Protein-binding, the predominance of the liver (pancuronium) and kidney (alcuronium) in excretion, other sites and mechanisms of metabolism and/or breakdown (succinylcholine, atracurium), all contribute to their special characteristics and usefulness in different clinical situations.

Other important considerations are their effects on the cardio-vascular system, which range from virtually nil in the case of vecuronium to quite significant ganglion-blockade and therefore hypotension in the case of d-tubocurarine. Pancuronium can produce both hypertension and tachycardia.

Other drugs may potentiate the effects of muscle relaxants, notably the volatile anaesthetics and some antibiotics. There is also a relationship between electrolyte levels and neuromuscular blockade, especially in the case of potassium and magnesium. Acidosis potentiates non-depolarising block.

Reversal of nondepolarising block can be achieved with anticholinesterases provided that receptor occupancy less than 90%, although it is better to await a lower level of occupancy before administering these drugs. By far the commonest, and the longest in duration is neostigmine. Because anticholinesterases produce powerful parasympathomimetic effects, such as bronchospasm, gut contraction and profuse upper airway secretions, it is customary to combine them with atropine.

Anticholinesterases must NOT be used in association with depolarising block.

Clinical tests of the effectiveness of reversal are those of grip strength and the ability to raise the head. Neither is particularly reliable as a guide to the patient's ability to ventilate, still less with respect to the effectiveness of airway protective reflexes. Hence it is vital to observe patients who have been given muscle relaxants for a period in a well-staffed environment (the recovery room). Even after effective reversal as measured by vital capacity and inspiratory force, residual paralysis can persist in especially sensitive muscles such as those of accommodation.

MECHANICAL VENTILATION

Modern mechanical ventilators all operate on the intermittent positive pressure principle, and must therefore be connected to the patient's airways either by an endotracheal or a tracheostomy tube. The connection between machine and tube is vital, since if a disconnect occurs, the patient will no longer be ventilated, even if the machine is otherwise working perfectly. Hence one of the important components of the ventilator/patient complex is an alarm system which will warn of such an accident having taken place.

All ventilators need a POWER SOURCE. This is either electricity or compressed gas (air or oxygen). Many ventilators which are powered by compressed gas also utilise a separate electrical supply to operate the displays, alarms, sensors and controls which determine how much and in what manner the patient is being ventilated. Conversely, ventilators which are electrically powered still need a supply of oxygen at least, so as to be able to enrich the inspired gases; many require a compressed air supply as well. Unless the power source(s) and/or gas supplies are connected and switched on, either nothing will happen, or an alarm system on the machine will tell you that something needs to be done.

Similarly, all ventilators must <u>cycle</u>, i.e. they must inflate the patient and then stop inflating to allow expiration to occur. Inflation is active, in the sense that this is when the machine is actually performing the work which the patient's respiratory muscles normally do. Expiration, on the other hand, is passive, and the machine simply allows the patient's respiratory tract to decompress, so that gas can escape. More sophisticated ventilators are capable of maintaining slight residual positive pressure at the end of expiration (PEEP - see below).

Cyling methods vary, and this is one of the ways in which ventilators may be classified. Cycling may be:

By TIME
By PRESSURE
By VOLUME

Unmodified time cycling simply means that the changeover from inflation to expiration occurs after a preset time, usually 1 - 2 seconds, irrespective of the amount of gas which has entered the patient's lungs, or the pressure which has been generated. Obviously this could be dangerous if the pressure is too high, so time-cycled ventilators are almost always fitted with pressure-limiting devices which will not allow pressure inside the breathing circuit to rise above a certain level.

Pressure cycled ventilators switch from inflation to deflation as soon as a preset presure in the airways has been reached, irrespective of the time taken or the volume delivered. These machines therefore are very inefficient in the presence of stiff lungs or obstructed airways, since the cycling pressure will be reached very early in these conditions, and before any significant quantity of gas has entered the alveoli. On the other hand, they are unlikely to produce pressure injury to the lungs, and since they will not cycle unless their preset pressure has been reached, they give visual and usually audible evidence of disconnection.

The disadvantages of time and pressure cycling have led to the development of the VOLUME VENTILATOR, i.e. one which is set to deliver a preset volume to the patient's lungs. In these machines the airway pressure which results is a function of

. delivered volume
. rate of inflation (i.e. inspiratory flow rate)
. lung compliance (NORMAL = 0.05 Litres/cm H_2O)
. airway resistance (NORMAL = 6 cm H_2O/litre/sec)

If an attempt is made to force the volume of gas into the lungs more rapidly than they can comfortably expand to receive it, then the peak pressure reached will be quite high. Hence volume ventilators always have some kind of time-setting which enables the operator to determine how much time will be allowed for inflation, and how much for deflation.

Sometimes these settings are individual, but many machines achieve the aim by means of a rate switch, knob or lever which sets the number of cycles per minute and another control which alters the ratio between inflation and deflation, usually called the I:E ratio. However other volume ventilators may make use of a flow control, which adjusts the speed with which gas is delivered to the patient. In this kind of system, a fast flow delivers the same volume at a higher pressure in a shorter time, and a slower flow will take longer to get the gas in, but the pressure will not be so high. Note, however, that if the machine is set on too slow an inflation rate, there won't be much time for deflation before the next inflation comes along!

Obviously, if there are settings for volume to be delivered, time to be taken to deliver it, time allowed for inspiration and time allowed for expiration, it is possible to set a ventilator an impossible task, and some kind of fault signal will be generated to alert the operator in these circumstances.

Most ventilators used in Intensive Care (but usually not those in anaesthesia) have mechanisms which allow the patient to "trigger" the ventilator, that is, if the patient takes a breath, the ventilator will switch to an inflating cycle, even if one was not due to occur at that precise time had the machine been left to do its own thing. This "assist" mode may be deliberately used in the "weaning" of patients from mechanical ventilation, and can also be useful in acclimatising a conscious patient to artificial ventilation without the problem of "fighting the ventilator" arising.

"Smart" ventilators can also be fitted with intermittent mandatory ventilation (IMV) modes, which allow the patient to breathe spontaneously, but intervene with a positive pressure inflation at intervals, in order to maintain ventilation at a certain (mandatory) minute volume. Again, the purpose of this arrangement is to give the patient a chance to breathe on his own, but not to allow hypoventilation to occur.

Reference has already been made to the use of PEEP. It has been found that intermittent positive pressure ventilation (IPPV) results in "shunting" within the lungs, resulting from the closure of small airways, and failure of some alveoli to ventilate. Blood flowing to these areas is not oxygenated, and paO2 falls. Correction is possible by increasing the FiO2 (the percentage of oxygen in the inspired gases), but only up to a point, and the longer that IPPV goes on, the higher the FiO2 has to be. Eventually, especially in patients who have poor lung function to begin with, or pulmonary damage due to septic or traumatic causes, even 100% oxygen cannot produce a satisfactory paO2. This is, of course, a critical situation.

It has been found that by not allowing the pressure in the lungs at the end of expiration to fall to zero (i.e. atmospheric pressure), but by maintaining a positive end-expiratory pressure (PEEP) of 5 - 15 cm H_2O, this problem can be overcome, and FiO2 can be reduced, whilst paO2 can be maintained at a satisfactory level. In intensive care, most patients on prolonged ventilator care are on some degree of PEEP to help keep the FiO2 of their inspired gases down to below 50%.

BREATHING CIRCUITS

The gases which leave the ventilator do so via a breathing circuit which is connected to the patient's airway via an endotracheal or tracheostomy tube. Breathing circuits are made (usually) of corrugated wide-bore plastic tubing, and incorporate a number of components, as follows:

Breathing Hoses:

These are designed to resist kinking (hence the corrugations) and are of a diameter similar to that of the adult trachea, viz 2-2.5 cm. Their length is such as to enable the ventilator to be conveniently positioned, and is usually approximately 1 metre.

Connectors:

These are rigid components which join hoses to each other (Y-piece) join a hose to a ventilator, or connect an ET tube to the breathing circuit. They have standard tapers by international convention, viz 22mm male and female and 15mm male or female. A 22mm male connector often has a 15mm female co-axial taper.

Valves:

Whilst many ventilators incorporate valves in their main mechanism, some make use of valves which form part of the breathing circuit. The commonest of these is the pressurised expiratory valve which is closed as the inspiratory cycle begins and opens as soon as the ventilator switches to the expiratory mode.

Moisture traps:

Due to the necessity for humidification (see below) there is sufficient condensation ("rain-out") is some breathing circuits to require inclusion of a receptacle for excess water.

Filters:

In many ICU's the need to control infection has led to the use of bacterial filters in both arms (inspiratory and expiratory) of the breathing circuit. Note that these components are often undirectional, so that incorrect installation is potentially fatal.

Thermometers:

Most breathing circuit manufacturers provide a port near the patient end at which a thermometer may be mounted in order to monitor the temperature of inspired gases.

Spirometers:

Some breathing circuits vent the expired gases from the patient into a device which measures their volume - i.e. a spirometer. This may in turn be fitted with an alarm which will signal failure to reach a preset volume.

HUMIDIFICATION

The need for humidification complicates all long-term IPPV, since the gases as delivered by the machine having come from cylinders or a liquid oxygen reservoir are by definition absolutely dry, and the patient's own humidification system has been by-passed by the endotracheal or tracheostomy tube. Failure to humidify has no short-term consequences - i.e. for up to an hour, but as time goes on, secretions will begin to dry, cilia will be knocked out, and the vulnerability of the respiratory tract to infections will increase.

Hence humidification must be provided, but how? Most ventilators do not have their own built-in humidifier, and rely on a separate device. All the latter are heated, since not enough water vapour can be obtained from water at room temperature. The warmed gases leaving the humidifier will cool during the journey along a corrugated hose to the patient, and some moisture will condense out. How to ensure that what reaches the patient is still gas saturated to 100% humidity at 37 degrees C is a challenge both to the manufacturer and to the operator, as is the disposal of any condensed moisture without hazard to the patient.

VENTILATOR ALARMS

Alarms are nowadays fitted to all new ventilators, at the very least to warn of disconnection, but usually also to alert the operator to the development of pressures outside a range which has been set by the operator. Hence a warning of leakage, or of decreasing compliance, partial airway obstruction, and a range of other less likely possibilities, is available. It is important that these alarms be visual as well as audible, since it can be extremely difficult to determine which of four ventilators in a row is emitting an audible signal. Alarms also usually indicate failure of the gas supply, reduction of F_IO_2, and even power failure - such as might result from accidentally switching off the power outlet, which can be signalled by means of a battery-powered alarm.

The ultimate in ventilators are those which continually monitor and display such parameters as end-expired carbon dioxide, the exact composition of expired gases, oxygen consumption by the patient, and so on. Non-activation of these luxuries is always possible, and the machine will still work. Hence it pays to concentrate on the basics of setting the volume, rate and I:E ratio controls, then the high and low pressure alarms, and making sure that all the connections fit. Fancy monitoring can be activated later if needed.

The ideal ventilator would be one which most closely meets the following criteria:

1. Simple operation with minimal direct controls

2. Visual readout of circuit pressure
   volume expired
   frequency
   inspiratory time
   inspiratory waveform

3. Direct control of inspired oxygen concentration (F_IO_2)

4. Small, portable, dual power source

5. Reliable, suitable for ICU, Theatre and Transport

6. Suitable for all patients with multiple ventilatory mode selection

7. Circuit easily sterilised
   minimal dead space
   low internal compliance
   minimal resistance and minimal connections

8. Provision for humidification

9. Audible and visual safety alarms

10. Quiet, cheap, good service back-up with easy access for repairs

Once you know that you are in a position in which you may have responsibility for a ventilated patient, you must at the earliest opportunity read the operator's manual for the machine which you will have to operate. If at all possible, obtain a "test lung" (most machines come with one and experiment with the machine in this manner until you feel that you understand it. Always make sure that the manual back-up resuscitator is available and in working order, in case anything goes wrong. If in doubt, ask. Remember that if a patient "fights" the ventilator, it may be because the ventilator is trying to kill him; in this contest, you should be on the side of the patient, not the machine! Until you have made quite sure that the machine is working properly, take the patient off it, check the airway, and use the manual resuscitator on 100% oxygen.

ADVERSE EFFECTS OF I.P.P.V.

1. Cardiovascular system:

   (a) decrease in venous return ---> decreased cardiac output

   (b) pulmonary and systemic vascular pressures altered

   (c) increased impedance to right ventricular ejection, may cause increased intraluminal pressure, increased ventricular wall tension, myocardial ischaemia and ventricular dysfunction.

   (d) sympathetic nervous system stimulation with intubation

2. Respiratory System:

   (a) increased intra pulmonary shunt - V/Q, altered pulmonary vascular resistance

   (b) increased dead space with high inflating pressures

3. Renal System:

   (a) inappropriate ADH secretion:

   fluid retention, particulary assocated with a fall in cardiac output, decreased renal blood flow and excessive I.V. fluid administration

   (b) increase aldosterone secretion - Na^+ retention

   (c) decreased cortical blood flow - decreased urine flow and increased diuretic usage

COMPLICATIONS OF I.P.P.V.

1. Infection of upper airways and tracheostomy sites due to invasion especially in the presence of moisture. Pseudomonas and staphylococci are especially troublesome.

2. Barotrauma, due to high inflation pressures which may be necessary to overcome poor compliance.

3. Nasal, laryngeal and tracheal trauma, ulceration and stenosis as a consequence of prolonged indwelling tubes, especially if size is poorly chosen or cuffs over-inflated.

4. Rise in I.C.P. during suction of upper airways, manipulation of tubes or inadvertent hypoventilation.

5. Alkalosis and arrhythmia due to inadvertent hyperventilation.

6. DISCONNECTION, EXTUBATION AND OTHER MACHINE OR HUMAN FAILURE.

REGIONAL ANAESTHESIA AND ANALGESIA

Regional anaesthesia is the reversible abolition of sensation in a part of the body, without loss of consciousness. Regional analgesia is the reversible reduction or abolition of pain in a part of the body, without loss of consciousness. To obtain the above conditions, drugs are used which when placed in the proximity of nerve fibres, interrupt impulses passing along those fibres by blocking the sodium channels of the axonal membrane.

PHYSICO-CHEMICAL STRUCTURE

Clinically useful compounds that demonstrate local anaesthetic (L.A.) activity usually possess the chemical arrangement:

Aromatic Portion - Intermediate chain - Amine Portion

There are two distinct chemical groups.

(a) AMINO-ESTERS - ester linkage between the aromatic portion and the intermediate chain.
(e.g. procaine, 2-chloroprocaine, amethocaine, cocaine and benzocaine)

(b) AMINO-AMIDES - amide link between the aromatic end and the intermediate chain.
(e.g. lignocaine, mepivacaine, prilocaine, bupivacaine and etidocaine)

The differences between these two groups of compounds are:

a. their metabolism:

- esters are hydrolysed in the plasma by pseudocholinesterase
- amides undergo enzymatic degradation in the liver

b. their allergic potential:

- ester hydrolysis forms para-aminobenzoic acid which will induce allergic phenomena in a small number of patients
- the amides do not form para-aminobenzoic acid and allergic reactions to these agents are extremely rare.

Except for cocaine, all local anaesthetic agents are vasodilators.

Following injection of a local anaesthetic agent, some of the drug is taken up by the nerve and some is absorbed by the vascular system. The degree of vascular absorption is related to blood flow through the area into which the drug is deposited. Duration of action is affected by the site in the body into which the drug is deposited (greater vascularity produces a briefer block) and by the addition of vasoconstrictors (such as adrenaline), which by delaying absorption, prolong the duration of action.

ANAESTHETIC CHARACTERISTICS OF L.A.^{S}.

The anaesthetic profile of a chemical compound is dependent on:

a. lipid solubility
b. protein binding
c. pK_a

a. Lipid Solubility:

* 1. primary determinant of intrinsic anaesthetic potency

2. lipid solubity is proportional to the drug's partition coefficient and therefore procaine with a partition coefficient less than one is the least lipid soluble of the L.A.^{S} and is therefore, the least potent.

3. partition coefficients of bupivacaine, tetracaine and etidocaine vary from 30-140, indicating a high lipid solubility and an intrinsic anaesthetic potency approximately 15 times greater than procaine.

4. the relationship between lipid solubility and anaesthetic potency is consistent with the lipoprotein composition of the nerve membrane.

b. Protein Binding-

* 1. primary determinant of duration of action.

2. the less protein binding, the shorter the duration of action. Procaine is poorly bound to protein and therefore short acting (less than one hour) whereas bupivacaine and tetracaine are highly protein bound and therefore long acting (> 2 hours).

3. the relationship between protein-binding of local anaesthetic agents and their duration of action is again consistent with the basic structure of the nerve membrane.

c. pK_a:

1. pK_a of a chemical compound is the pH at which its ionized and nonionized forms are in equilibrium.

2. the unchanged base form of a local anaesthetic is primarily responsible for diffusion across the nerve sheath.

3. onset of anaesthesia is directly related to the rate of epineural diffusion which depends upon the amount of free base present at a pH of 7.4.

4. Lignocaine has a pKa of 7.74, is 65% ionized and 35% nonionized at pH 7.4 and therefore, as the pKa is relatively close to the pH, the onset time is rapid.

TOXICITY OF LOCAL ANAESTHETIC AGENTS

All excitable membranes will be affected by local anaesthetic agents if they achieve a sufficient tissue concentration.

Toxicity may be due to:

a. inadvertant intravascular administration of the correct dose of LA

b. administration of an excessive does of LA

c. direct tissue toxicity due to the agent or its associated preservatives

d. rarely, an anaphylactoid or anaphylactic reaction

The signs and symptoms of toxicity will depend upon the dosage of the drug administered and the rate at which the agent enters the intravascular compartment.

Effects on the Central Nervous System (C.N.S.):

a. Initially - circumoral numbness
dizziness
difficulty in focusing and tinnitus

b. Excitation Phase - shivering, muscle twitching and tremors
generalised tonic-clonic convulsions

c. Depression Phase - unconsciousness
respiratory arrest

The excitatory phase is due to selective blockade of inhibitory pathways, however with large doses of LA^S there is inhibition of both excitatory and inhibitory pathways producing generalised CNS depression.

The relative CNS toxicity of bupivacaine, etidocaine and lignocaine is approximately 4:2:1, which is similar to the relative intrinsic anaesthetic potency of these agents for the production of epidural anaesthesia in man.

Effects on the Cardiovascular System (C.V.S.):

The doses and blood levels of LA^S which cause significant CVS depression are approximately 2 to 7 times higher than those which will produce convulsions.

a. Initially - hypertension and tachycardia during CNS excitation.

b. Intermediate Phase - prolongation of conduction time
negative inotropic action
decrease cardiac output and moderate hypotension

c. Terminal Phase - peripheral vasodilation
profound hypotension
sinus bradycardia
conduction defects and ventricular arrhythmias
circulatory collapse

All LA^S exert a dose dependent negative inotropic action which correlates with their local anaesthetic activity. The more potent, highly lipid soluble, highly protein bound LA^S are more cardiotoxic. (e.g. inadvertent I.V. bupivacaine 0.75%) Bupivacaine markedly inhibits sodium conductance in cardiac muscle, and whereas lignocaine's action is short, the bupivacaine action persists for a prolonged period (so called "fast-in, slow out" drug). Ultimately, the combined peripheral vasodilation, decreased myocardial contractility and depressant effects on rate and conductivity lead to cardiac arrest.

Local Tissue Toxicity

The potential for LA agents to cause localised nerve damage is very low; however the local neural toxicity reported with 2-chloroprocaine solutions is believed to be related to the antioxidant, sodium bisulphite, and the low pH of the anaesthetic solution.

Local anaesthetics are relatively safe if administered properly. However comparison of the dose required to produce a clinically useful blockade and that which will produce toxic symptoms reveals a poor therapeutic ratio.

PREVENTION OF TOXICITY

Rules:

1. Never use a more concentrated solution than is necessary. For infiltration anaesthesia in peripheral lesions, 0.5% lignocaine is sufficient.

2. For vascular areas, stronger concentrations and large volumes, always use a vasoconstrictor.

3. Always aspirate before injecting. Do not inject if blood is aspirated.

4. Always have another person check the identity and concentration of the drug intended for use.

5. Always calculate the maximum dose (on a per kg basis) and verify that the dose to be administered is within the safe range.

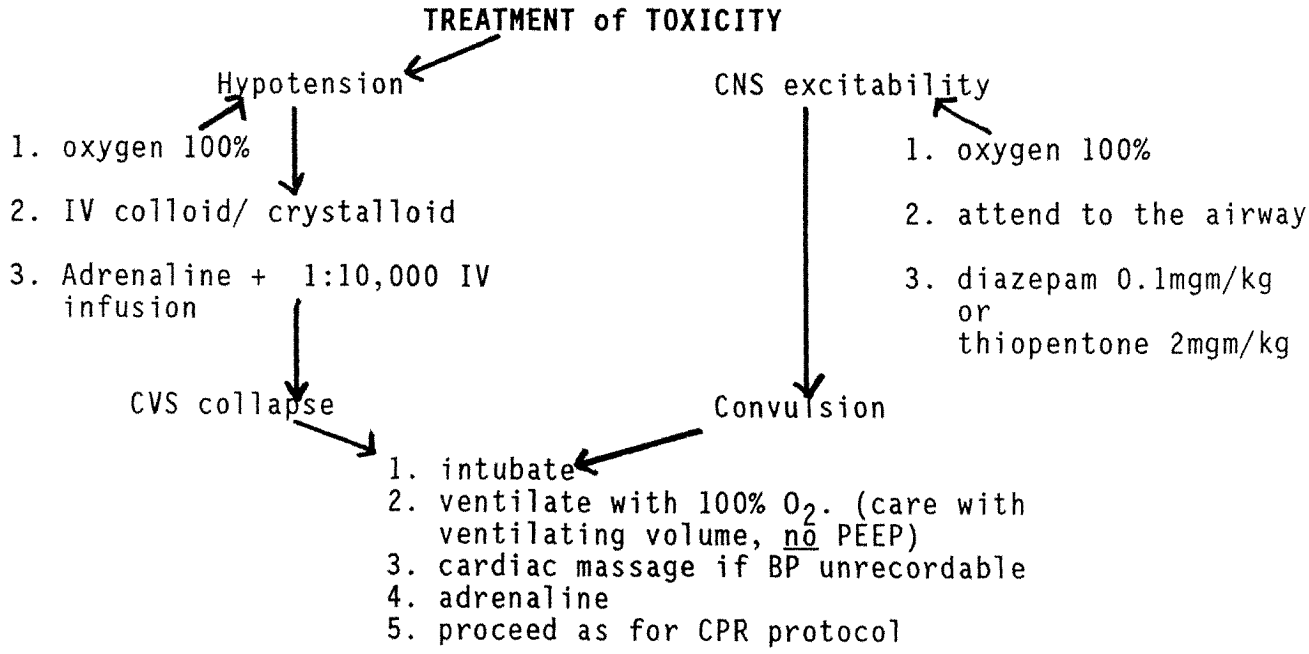
6. I.V. line must be in place for all regional blocks except for limited topical and infiltration anaesthetics.

7. A sucker and a means of ventilating the patient with oxygen must be close at hand.

Vasoconstrictor:

The vasoconstrictor of choice is adrenaline. It should never be used in concentrations greater than 1:200,000, and the total dose should not exceed 0.5mgm at a single session.

Never use a Vasoconstrictor in an end-artery situation. It is neither necessary, nor desirable to use adrenaline in the subarachnoid space.



FACTORS WHICH INFLUENCE THE CHOICE OF REGIONAL ANAESTHESIA:

Controversy has long existed as to the optimal and appropriate use of regional as opposed to general anaesthesia for a variety of surgical operations.

1. Patient suitability.
2. Skilled anaesthesiologist.
3. Other personnel - surgeons, nursing staff.
4. Operative site - the periphery is easier.
5. Special risks associated with unconsciousness, e.g. in obstetrics.

But in the long run the choice must be made principally on the basis of what is safer for the patient, whilst at the same time providing effective anaesthesia.

TECHNIQUES:

Local anaesthetic agents may be:

1. Applied to a surface; this is TOPICAL anaesthesia.
2. Injected into the vicinity of a lesion; this is INFILTRATION.
3. Injected around the trunk of a major nerve; this is a NERVE BLOCK.
4. Injected around a plexus of nerves; this is a PLEXUS BLOCK.
5. Injected into the extradural space; this is an EPIDURAL BLOCK.
6. Injected into the subarachnoid space; this is a SPINAL BLOCK.
7. Injected into the exsanguinated veins of a limb distal to an arterial tourniquet; this is a BIER'S BLOCK.
8. Refrigeration.

TOPICAL ANAESTHESIA

Cocaine, a very powerful surface anaesthetic, is used by ENT surgeons in the nose because of its additional vasoconstrictor activity. It is, however, a very difficult drug to use and to avoid toxicity. Many junior medical officers will use amethocaine 0.5% on the cornea to allow the painless removal of embedded foreign bodies, or the examination of a painful eye. Remember that the continuing anaesthesia renders the eye vulnerable to damage, and that it must be covered until sensation returns.

When mucous surfaces, as in the nose or throat are anaesthetised topically, absorption may be very rapid, especially in the presence of inflammation. Very careful attention must therefore be paid to dosage, particularly using Xylocaine aerosol in children, as it contains lignocaine 10% delivered as a metered dose of 10mg per puff.

INFILTRATION

Infiltration with a local anaesthetic solution via a syringe and hypodermic needle is the commonest anaesthetic procedure in the world, and one of the few techniques which non-anaesthetists can carry out safely and effectively provided they observe a few simple rules.

Because of the limitations of dosage, only a relatively small area of the body can be anaesthetised in this way at one time. Peripheral lesion excisions, cannulations, dental procedures and blood donations are painlessly performed every day using this technique.

NERVE BLOCK

By placing a relatively small quantity of local anaesthetic agent (Femoral Nerve block -Adult-10ml of 0.5% bupivacaine) - in close proximity to a nerve trunk, block of all sensation in the distribution of that nerve can be achieved. The advantages of this technique are:

- the small amount of drug used and the reduced likelihood of toxicity.

- the nerve block is remote from the area on which the operation is to be performed. This is particularly useful if the operation site is infected.

Nerve block techniques vary with respect to the precision of anatomical knowledge they require for success. Some, such as maxillary nerve block are technically very challenging; others, such as digital nerve block, can be easily performed.

Higher concentrations of local anaesthetic agent are required for a successful nerve block compared to infiltration. Vasoconstrictors are useful, but must never be used in the "end-artery" situations of the digits or penis.

PLEXUS BLOCK

Even larger body areas can be anaesthetised by blocking the plexus of nerves which supply them. This is particularly the case with the upper limb, where the accessibility and anatomical constancy of the brachial plexus in the root of the neck feasible. Interscalene, supra clavicular and axillary approaches have been described; the most popular is the axillary approach which is virtually free of anatomical complications.

Relatively large volumes of local anaesthetic solution (Axillary Plexus Block-Adult-- 30-40mls lignocaine 1% with adrenaline 1:200,000) are necessary for successful plexus blocks, and careful attention must be paid to dosage. Once again, vasoconstrictors are a big help in controlling absorption and prolonging block.

IMPORTANT

In both plexus and major nerve blocks, time must be allowed for penetration of the nerve trunks by the local agent, and up to 20 minutes must elapse before the larger nerves will be completely blocked. Surgery must therefore not be prematurely commenced.

EPIDURAL ANAESTHESIA/ANALGESIA

Amongst the most useful of all regional techniques is that of epidural anaesthesia and analgesia. The former is suitable for a wide range of surgery below the umbilicus, whilst the latter can be used for the abolition of pain in labour or for post-operative pain relief.

Before epidural block is attempted, an intravenous cannula must be placed, and a fluid load, of 500ml of crystalloid solution is administered, before the onset of sympathetic blockade. If extensive block is intended, a systemic vasoconstrictor such as ephedrine I.V. may be required.

The procedure is technically difficult until practice has been acquired. Placement of the needle in the epidural space is via the interspinous route from the back, and several tests for entry of the needle tip to the extradural space have been described. The most common in use is the loss of resistance test, in which an air or fluid-filled syringe is attached to the needle as it is advanced through the ligaments, whilst firm pressure is maintained on the plunger. As the space is entered, resistance falls and a quantity of air or solution (usually saline) is injected.

Local anaesthetic solution may now be injected, or a catheter may be introduced to allow more than one dose to be given. This is especially useful in obstetrics, since longer periods of analgesia may be required than a single dose of bupivacaine is capable of producing.

Successful epidural blockade of a number of segments requires volumes and concentrations verging on the toxic threshold; many anaesthetists therefore believe that addition of vasoconstrictor to the solution is essential.

The bilateral block which results includes the sympathetic outflow. The denervation eventually produces sufficient vasodilation for blood pressure to fall. In fit patients, even extensive sympathetic block is of little consequence, although if the hypotension is sudden, nausea may result. In old, sick and compromised patients, sudden, extensive sympathetic blockade may be lethal. Very careful consideration must therefore be given to the choice of agent and extent of epidural anaesthesia in poor risk patients.

The difference between anaesthesia and analgesia is largely a matter of the strength of solution employed. For caesarean section (anaesthesia) 0.5% Bupivacaine is necessary, whereas for pain relief during labour, the concentration is reduced to 0.25%.

Very careful attention must be paid to charting the total dose of drug given if repeated injections are necessary, as well as BP, pulse and respiratory rate at frequent intervals. There is a limit to the amount of agent which the patient can metabolise, and the manufacturers' literature should be consulted for the amount which is safe to administer within a 24 hour period.

SPINAL BLOCK

Essentially similar results to that of epidural block can be obtained from introducing a much smaller volume (e.g. 2-3mls of isobaric bupivacaine 0.5%) of local anaesthetic solution into the subarachnoid space, and bringing it into contact with the spinal cord. All sensation below that level will be lost, as well as vasomotor tone, as in epidural block. The concentrations of solution required are high, but the total dose of drug is less.

Spinal block is technically simpler, and more likely to succeed in less experienced hands. Its principal drawbacks are that it cannot be prolonged via a catheter and the dural integrity is breached.

All of the problems of sympathetic blockade are the same as in epidural anaesthesia, and similar precautions apply.

Very fine needles (25G) are used to minimise post spinal puncture leakage of CSF and consequent low pressure headache.

VENOUS PERFUSION (BIER'S) BLOCK

If a limb is exsanguinated and an arterial tourniquet applied, dilute local anaesthetic solution (e.g. 30ml Prilocaine 1.0%) introduced into the veins below the tourniquet produces anaesthesia for as long as the tourniquet is left on. Removal of the tourniquet terminates anaesthesia within a few minutes.

This simple technique is especially applicable to the upper limb, and can be carried out as an outpatient procedure. Risks are present from local anaesthetic toxicity, especially if any defect develops in the tourniquet within 15 minutes of the drug having been administered. Bupivacaine is especially dangerous and should never be used for this block.

DRUGS

A wide variety of local anaesthetic agents is available, and not all are available in every country. A limited selection is given below:

Agent	Duration	Onset	Toxicity	Applications
Lignocaine	Medium	Rapid	Medium	Topical Infiltration Nerve block Plexus Block Epidural Spinal
Bupivacaine	Long	Slow	High	Epidural spinal
Cinchocaine	Long	Rapid	High	Spinal only
Prilocane	Short	Rapid	Medium	Bier's Block
Cocaine	Medium	Rapid	Very high	Topical only

DOSAGE OF LA^S

It is inadvisable to exceed:

1. **Lignocaine** plain **3mg/kg**
   Lignocaine with adrenaline **5mg/kg**

2. **Prilocaine** with adrenaline **7mg/kg**

3. **Bupivacaine** plain or with adrenaline **1.5mg/kg**

4. **Mepivacaine** plain **3mg/kg**
   Mepivacaine with adrenaline **5mg/kg**

PAINFUL CONDITIONS-- Relief by Regional Block

Some examples:-

1. Fractured shaft of Femur - Femoral Nerve Block

2. Herpes Zoster - Thoracic Epidural
   Cervical Sympathetic Block

3. Reflex Sympathetic Dystropy - Cervicothorocic sympathetic block
   Lumbar sympathetic block

4. Carcinoma of the Pancreas - Coeliac Plexus Block

5. Skin graft donor site - Topical bupivacaine 0.25%
   with adrenaline 1:200,000

6. Ischaemic Peripheral Vascular Disease - Lumbar sympathetic blockade

OXYGEN THERAPY

Oxygen is a drug: it has

a. physiological actions

b. pharmacological actions

c. a dose-response relationship

d. adverse effects

Supplemental oxygen should be administered when tissue oxygenation is inadequate or threatened.

PHYSIOLOGY

Oxygen is utilized in aerobic metabolic pathways to produce biological energy from food fuels. Anaerobic metabolism produces fewer high-energy molecules for an equivalent amount of substrate. Conversely greater glucose consumption is required to produce an equivalent amount of energy, because the cellular uptake of glucose is enzyme limited. A decrease in biological energy results from anaerobic metabolism and those tissues that have a high utilization of energy and limited cellular stores (cerebral, adrenal, hepatic and renal) will be damaged by hypoxia. The accumulation of biological acids as a consequence of anaerobic metabolism have further deleterious effects on cellular performance.

The "oxygen cascade" (oxygen-tension gradient from the inspired gases to the mitochondria) acts as a pressure head of oxygen which determines the oxygen supply to the mitochondria. The major oxidative enzymatic processes occur in the mitochondria and the physiological partial pressure of oxygen in the mitochondria is around 1-5 kPa. Anaerobic metabolism occurs when the oxygen tension falls below the Pasteur point which is about 0.27 kPa.

Oxygen is delivered by ventilation and pulmonary gas exchange to arterial blood and then is carried to the tissues via the vascular system. Oxygen passes from capillaries by diffusion to the mitochondria. This diffusion requires a pressure head of at least 2.7 kPa and an arterial oxygen tension of less than 4.0 kPa will jeopardize mitochondrial function, particularly if regional blood flow is also compromised.

ARTERIAL OXYGEN CONTENT

The normal value of 18 to 20 volumes %) is a measure of the number of ml of oxygen contained in 100ml of blood. Since oxygen is both dissolved in plasma and bound to haemoglobin, the calculation of oxygen content has two components.

$$C_aO_2 = 1.37 \times Hb \times S_aO_2 + 0.003 \times P_aO_2$$

$$\simeq \quad 19.7 \quad + \quad 0.3$$

Hb = haemoglobin in gram per 100ml blood
S_aO_2 = % haemoglobin saturation

1.37 = number of mls of oxygen bound to one gram of fully saturated haemoglobin

P_aO_2 = arterial oxygen partial pressure in kPa

0.003 = solubility of O_2 in plasma, Vol% / kPa

From the above equation it can be seen that the haemoglobin term is much larger than the dissolved term and therefore C_aO_2 should not be very sensitive to P_aO_2. This would be true except for the dependence of S_aO_2 on P_aO_2 expressed in the oxyhaemoglobin dissociation curve.

The points to remember are:

1. 90% saturation with P_aO_2 60mmHg (shoulder of curve)
2. 75% saturation with P_aO_2 40mmHg (venous point)
3. 50% saturation with P_aO_2 26mmHg (P_{50})

The curve is displaced to the right by four factors:

1. increasing P_aCO_2 (Bohr effect)
2. increasing hydrogen ion concentration (decreasing pH)
3. increasing temperature
4. increasing 2, 3 DPG concentration

The position of the curve is defined by the 50% saturation point known as the P_{50}.

"Oxygen Flux" is the amount of oxygen delivered to the tissues per minute. Oxygen flux equation describes the global oxygen availability and illustrates the reservoir above the average resting oxygen consumption of 115-165ml/min M^2. As the amount of oxygen in the arterial blood is known (C_aO_2) and the rate at which this oxygen is delivered to the tissues is determined by the cardiac output, and the body surface area is used to index the cardiac output for patients of different size, then the oxygen delivery index can be calculated.

Oxygen Flux = cardiac index x C_aO_2 x 10 = 550 - 650mlO_2/min M^2 ($ml/min/M^2$)

Sufficient oxygen must be available to provide for the capillary cell diffusion gradient and tissue differences in oxygen extraction may be important. The normal range given above is for a patient at rest and is not necessarily adequate to meet the oxygen delivery demands of the tissues during periods of stress.

Oxygen Consumption (VO_2) is a relatively stable parameter for a patient at rest and is calculated from the oxygen balance:

oxygen in - oxygen out = VO_2

$CI \times C_aO_2 - CI \times CvO_2 = VO_2$

therefore, $VO_2 = CI\ (C_aO_2 - CvO_2)$

where CvO_2 = mixed venous blood oxygen content (sampled from the pulmonary artery)

The tissues usually consume an average of 5ml of O_2 per 100ml of circulating blood. The mixed venous O_2 is a global measure of the adequacy of oxygen supply relative to oxygen demand. If the oxygen delivery decreases, more O_2 will be extracted from the arterial blood, therefore lowering the CvO_2. An increase in oxygen consumption will produce the same result if there isn't a compensating increase in oxygen delivery.

Mixed venous oxygen (physiological values):

Oxygen content CvO_2 = 12 to 15 vol%
Hb Saturation SvO_2 = 72 to 78%
Partial Pressure of Oxygen PvO_2 = 5.5 to 6.5 kPa

HYPOXIA

Hypoxia is defined as inadequate tissue oxygenation. The types of hypoxia are:

I Ischaemic Hypoxia — ↓ Blood flow, normal CaO_2

II Hypoxaemic Hypoxia — ↓ CaO_2

1. Hypoxic Hypoxia — P_aO_2, ↓ S_aO_2, normal Hb
2. Anaemic Hypoxia — Hb, normal P_aO_2, normal S_aO_2
3. Toxic Hypoxia — ↓ S_aO_2, normal P_aO_2 (↑ met Hb COHb)

Hypoxic Hypoxia is caused by hypoventilation
impaired diffusion
shunts (Qs/Qt)
ventilation/perfusion mismatch V/Q

When a pulmonary shunt is present, poorly oxygenated venous blood mixes with well oxygenated end pulmonary capillary blood to produce a lower P_aO_2. (i.e. venous admixture). The larger the shunt the greater the effect of PvO_2 on P_aO_2. Consequently, in the presence of a shunt, any further decrease in PvO_2 will also lower P_aO_2.

Clinically venous admixture is due to

1. anatomical shunts (bronchial and thebesian veins)
2. pulmonary atelectasis, oedema and infection
3. pulmonary arterio venous shunts
4. congenital right-to-left heart disease
5. ventilation/perfusion mismatch

EQUIPMENT TO ADMINISTER OXYGEN

Oxygen may be delivered to spontaneously breathing patients by:

1. facial mask
2. nasal cannulae
3. anaesthetic circuits (via a mask or ETT)
4. resuscitation devices (laerdal, Air-Viva and Ambu)
5. headboxes
6. cots
7. tents
8. incubators

Favourable Specifications

a. patient comfort

b. low resistance to breathing

c. no accumulation of carbon dioxide

d. accurate control of F_IO_2

A patient's alveoli can only receive 100% oxygen if

1. $F_IO_2 = 1.0$
2. Oxygen delivery flow > patients peak inspiratory flow rate (PIFR) (Adult, at rest = 25-35L/min)
3. Circuit has a reservoir bag + is "closed" (i.e. airtight seal)

Therefore patients CANNOT inspire 100% oxygen with a conventional bedside mask because the maximum flow of a conventional flowmeter is only 14 L/min.

There are two categories of oxygen delivery devices:

I Fixed Performance Systems : the F_IO_2 will remain constant and independent of patient factors

II Variable Performance System : the F_IO_2 varies with the patient's ventilation

FIXED PERFORMANCE SYSTEMS

The inspired oxygen concentration is determined by the oxygen flow rate and is independent of the patient's ventilation. To accomplish this, the system must deliver the prescribed gas mixture at a flow greater than the patient's Peak inspiratory flow rate (PIFR).

e.g. anaesthetic machines
continuous positive airway pressure (CPAP) masks
venturi-type masks

a. Venturi Type Masks:

The oxygen flow to a venturi-type mask entrains room air by the venturi principle, resulting in a high total flow of gas which provides an accurate fractional inspired oxygen concentration.

e.g. Ventimask (Vickers U.K.) preset 24%, 28%, 35%, 40% and 60%; Inspiraton; Hudson; M^{c}Gaw and Medishield.

- these use a mask with a short interchangeable delivery hose with a varying aperture : flow rate ratio which provides various F_IO_2.

Oxygen flow rate is usually set at 6-8L/min and will produce gas flows of approximately 40-60L/min, depending on the oxygen concentration which is prescribed.

High flows maintain a constant F_IO_2 without rebreathing and a tight fitting face mask is unnecessary. In severe dyspnoea with high PIFR^S, the oxygen flow rate should be increased to 14L/min to maintain the intended F_IO_2.

b. CPAP Mask

A continuous positive pressure delivered throughout the respiratory cycle can improve oxygenation by increasing functional residual capacity, reducing shunt and thereby reducing venous admixture in patients who have a dimished FRC and are hypoxic. The supply of air/O_2 mixture must be greater than the patients minute ventilation. A CPAP circuit and reservoir bag will allow the F_IO_2 to be set by the fresh gas mixture.

VARIABLE PERFORMANCE SYSTEMS

The F_IO_2 depends on the oxygen flow rate, patient ventilation and device factors.

1. Nasal Cannulae

Oxygen flow rate of 0.5-4L/min delivers a F_IO_2 0.22-0.4, however an accurate prediction of the F_IO_2 is not possible. The F_IO_2 will vary from breath to breath depending on the rate and depth of breathing, nose or mouth breathing and the length of the expiratory pause. High oxygen flow rates are very uncomfortable and dry the mucosa. In infants, a high F_IO_2 can be achieved by a catheter tip placed at the uvula and taped to the side of the face. The nasopharynx acts as a reservoir and is loaded with oxygen for the next breath during the expiratory pause.

2. Face Masks

Hudson, MC, Medishield: simple, semi-rigid, plastic disposable. Oxygen is fed to the mask and the patient expires via lateral perforations and between the mask and the face. On inspiration the patient entrains a variable amount of air and so the F_IO_2 varies, but is always less than 100%. Provided the oxygen flow rate is greater than 4L/min rebreathing is not a problem and flow rates between 4-14L/min provide F_IO_2 0.35-0.6.

Clinical Significance of Some Oxygen Tension and Saturation Values

Arterial partial pressure of oxygen (kPa)	(mmHg)	Saturation	Clinical significance
20.0	(150)	99%	Inspired air at sea-level
12.9	(97)	97%	Young normal adult
10.6	(80)	95%	Young normal adult asleep; old normal adult awake; inspired air at 5800m
9.3	(70)	93%	Lower limit of normal
8.0	(60)	90%	Respiratory failure
6.7	(50)	85%	Moderate-to-severe respiratory failure requiring hospital admission
5.3	(40)	75%	Normal venous blood values; severe respiratory failure; acclimatized adult at rest at 5800m
4.0	(30)	60%	Usually unconscious if not acclimatized
3.5	(26)	50%	P_{50} (50% saturation) value
2.7	(20)	36%	Acclimatized adult exercising at 5800m; hypoxic death

In severely dyspnoeic patients, the actual oxygen concentration that is inspired may be considerably less than intended. Dual flow-meters can give a maximum flow of 28L/min in an attempt to match the patients PIFR.

In children, by virtue of their small size, PIFR approximates more closely with a flowmeter flow rate and F_IO_2 0.8 can be achieved with a flow rate of 8L/min. Masks with unidirectional expiratory valves and a reservoir bag can deliver F_IO_2 0.9. These masks have to be used with great care as the rebreathing of carbon dioxide and asphyxia are potentially lethal complications if the rather primitive valves malfunction.

3. Face tents, tracheostomy mask and T-pieces.

Artificial humidification should be supplied if the normal humidifying mechanisms are bypassed, laryngeal oedema or thick tracheobronchial secretions are present.

The humidified oxygen mixture is delivered from the apex of the face tent and gases are expired through the open upper part. A T-piece is a non-rebreathing, large bore circuit that is attached directly onto the ETT. The oxygen mixture is delivered by way of one limb of the T-piece and the expired gas leaves by way of the other limb, which also acts as a reservoir. Rebreathing is prevented by having sufficient flow rate (approximately 3 x minute volume) to wash expired gases out of the expiratory limb.

A tracheostomy mask is a small, plastic mask placed over the tracheostomy stoma. The patient will inspire less humidified oxygen than that delivered, because dilution from room air occurs.

4. Oxygen Headbox

The F_IO_2 depends on the oxygen flow rate, size of the headbox, size of the leak around the neck, the position of the head and the frequency of lid-opening. An F_IO_2 of 1.0 can be achieved but falls rapidly on box opening, and the recovery half-life is long. Rebreathing of carbon dioxide can occur at low flow rates and with a seal around the neck.

5. Incubators

These are a means to deliver oxygen and provide a neutral thermal environment. It is possible to deliver F_IO_2 0.85 however the need for high oxygen flow rates. patient access and long recovery after each opening are major problems.

6. Oxygen tents and cots

Larger children can be nursed in these devices however oxygen concentrations above F_IO_2 0.4 are difficult to achieve. Recovery after opening the tent is very slow and access and observation of a sick child is severely restricted.

CLINICAL ASPECTS OF OXYGEN ADMINISTRATION

1. Oxygen should be administered when oxygen flux is decreased and the P_aO_2 falls below 8.0kPa. Haemoglobin saturation falls steeply with oxygen tensions below this because of the shape of the oxy-haemoglobin dissociation curve. Profound hypoxaemia is present and death is imminent when the P_aO_2 is less than 4.0kPa.

2. Patients who are seriously ill from non-respiratory causes require oxygen therapy:

    1. shock
    2. sepsis
    3. cardiac failure
    4. AMI
    5. burns
    6. trauma
    7. unconscious patient
    8. neuromuscular disease
    9. immediately post-anaesthesia

3. Physiological humidification of gases requires a heated humidifier. Cold water bubble-humidifiers are inefficient, potential sources of infection. Humidification is necessary not for venturi masks where large volumes of room air are entrained.

4. Patients with severe chronic obstructive airways disease (COAD) rely on a hypoxic stimulus to the medullary respiratory centre, whose normal hypercarbic drive is depressed by chronic carbon dioxide elevation. The uncontrolled administration of oxygen may lead to respiratory depression and death. These patients require accurately-metered low concentrations of oxygen which are titrated against P_aO_2 and close patient observation.

    Rationing of oxygen to every hypoxic patient who has a raised P_aCO_2 is illogical, as the central depression may be associated with another cause. Profound hypoxaemia is commonly associated with acute asthma which may be aggravated by ß-adrenergic bronchodilators and methylxanthine substances. Adequate oxygen, with simple masks (not low concentration venturi masks) should be administered in acute asthma many of whom have low $PaCO_2$ anyway.

    COAD patients should receive controlled oxygen therapy commencing with F_IO_2 0.24 venturi mask and blood gas analysis after 30 minutes. If the P_aCO_2 remains below 10kPa and the rise in P_aCO_2 is < than 1.3kPa, the F_IO_2 should be increased to 0,28 and later to 0.35 if required. It is unnecessary and hazardous to aim for large improvements in P_aO_2, remembering that a small increase in P_aO_2 will provide a large increase in tissue oxygen availability because of the shape of the oxy-haemoglobin dissociation curve. A patient with COAD is NOT sensitive to oxygen if the P_aCO_2 does not rise with increases in F_IO_2.

5. Inspired oxygen displaces air in alveoli, and being more soluble than nitrogen, will lead to increased pulmonary microatelectasis. Acute cessation of supplementary oxygen may cause substantial hypoxaemia and is hazardous.

6. Facemasks should be replaced by nasal cannulae with restless patients, during meals and physiotherapy sessions to maintain a continuous oxygen supply

COMPLICATIONS OF OXYGEN THERAPY

1. Carbon dioxide narcosis in carbon dioxide - sensitive COAD patient.

2. Pulmonary oxygen toxicity - depends upon the $P_A O_2$.

3. Retinopathy of prematurity - depends upon the P_aO_2.

4. Cerebral oxygen toxicity - hyperbaric oxygen therapy.

No pulmonary toxicity has been reported when the F_IO_2 has been < 0.5. Breathing 100% oxygen for periods less than 24 hours is usually not associated with toxicity, but symptoms can be expected after 36 hours of 100% oxygen therapy.

Retrolental fibroplasia in some infants is unavoidable but it is recommended that the P_aO_2 be maintained between 6.6-10.7kPa.

High inspired oxygen concentrations, even 100%, should never be withheld if profound hypoxaemia is present.

OXYGEN MEASUREMENT AND MONITORING

Oxygen therapy must be titrated against frequent arterial blood-gas estimations or some other form of oxygen monitoring, such as pulse oximetry or transcutaneous P_aO_2 measurement. The oxygen flow rate, F_IO_2, device used, ventilation mode should be quoted with each P_aO_2 that is measured. A solitary P_aO_2 represents oxygenation at one specific point in time and is meaningless without the concomitant information.

Oxygen measuring devices can be divided into two groups, those which measure partial pressure and those which measure haemoglobin saturation.

PERIOPERATIVE FLUID MANAGEMENT

The primary determinant of fluid and electrolyte therapy is the current clinical state of the patient. During anaesthesia and surgery the patient's well being is threatened by shifts in intravascular volume and cardiovascular performance caused by haemorrhage, unwise fluid management, pharmacologic or mechanical effects on the heart and vessels, or aggravation of pre-existing cardiovascular disease.

The state of hydration is assessed by skin turgor, urine output, pulse rate, central venous pressure, arterial blood pressure, haematocrit, serum osmolality, serum sodium and urine osmolality. None of these are accurate predictors by themselves and even collectively do not provide accurate information about fluid needs.

Degree of Dehyration

	Mild	Moderate	Severe
Body weight	5% loss	10% loss	15% loss
Skin turgor	↓	↓↓	↓↓↓
Mucous membrane	dry	dry	very dry
Skin colour	pale	grey	mottled
Urine	± oliguria	oliguria	azotaemia
Blood pressure	normal	± normal	decreased
Pulse	± ↑	↑	↑↑

The most useful clinical signs of moderate dehydration are skin turgor and a low urine output. Dry mucous membranes depend on whether the patient is mouth breathing and acidotic, rather than being a specific sign of dehydration.

Overhydration is manifested as oedema. The factors which cause oedema are excess fluid administration, high levels, of ADH, "leaky" capillary syndrome, heart failure, renal failure and hypoalbuminaemia. In the intensive care setting there are often a number of these causes contributing at one time.

Replacement intravenous therapy should be considered as a dose-response relationship; the dose is the amount and type of fluid administered and the response is the Physiological interaction between the body fluids, the intravascular compartment and the kidneys.

The response is assessed by clinical signs, urine output and biochemical estimations. Alterations to the dose may be necessary during replacement therapy.

Signs of Hypovolaemia are:

1. Narrow pulse pressure
2. Decreased systolic pressure
3. Increasing heart rate
4. Paling mucous membrane (conjunctive, tongue, lips)
5. Flattening of neck veins
6. Retarded capillary refill after blanching
7. Cycling of systolic B.P. with positive phase of IPPV
8. Decreased peripheral venous pressure flash back in I.V. infusion on lowering the infusion bag.

The significance of central venous pressure readings is blurred in the presence of myocardial disease, and in any event a single observation is of much less valve than a series of measurements taken at intervals.

HAEMORRHAGE AND BLOOD TRANSUFSION

Assessment:

Bleeding, as a cause of acute illness should be considered in a number of groups of patients:

1. Trauma victims
2. Recent surgery or invasive procedures
3. Altered coagulation states
4. Acute abdomen
5. Gynaecological problems
6. History of upper gastrointestinal symptoms

Postural hypotension (i.e. significant fall in systolic BP > 10 mmHg when patient moves from supine to erect position) is only certain to be present after more than 15% of blood volume is lost. There are several causes of postural hypotension other than hypovolaemia, viz:

1. Drugs - narcotics, vasodilators
2. Autonomic neuropathies (diabetes)
3. Spinal cord injuries
4. Increasing age

Symptoms and signs of acute blood loss

Blood loss (% of blood volume)	Arterial blood pressure (systolic mmHg)	Symptoms and signs
10 - 15	Normal	Postural hypotension Mild tachycardia
15 - 30	Slight fall	Tachycardia Thirst Weakness
30 - 40	60 - 80	Pallor Oliguria Confusion Restlessness
> 40	40 - 60	Anuria Air hunger Coma Death

The urgency of volume replacement and the rate of resuscitation are both functions of the amount and rate of loss. The initial assessment will suggest how much replacement is needed. If after this replacement is given the patient is still not stable, then it should be assumed that there is ongoing, unrecognised bleeding.

Typical amounts of blood loss associated with differing types of fractures in an average adult

Type of fracture	Associated blood loss
Forearm	400 - 800 ml
Humerus	500 - 1,000 ml
Tibia and fibula	750 - 1,200 ml
Femur	1,000 - 1,500 ml
Pelvis	1,500 - 2,500 ml or more
Lumbar or thoracic spine	500 - 1,000 ml

MANAGEMENT:

In the initial phase of resuscitation in the emergency department cross-matched blood is usually not available. It is appropriate to use a colloid initially and then to use a mixture of packed red cells and clotting factors later as they become available. Since the patient usually has no oral intake in this situation it is important to consider maintenance fluid requirements as well.

Guidelines:

1. Attend to the priorities of intravascular volume and tissue perfusion. Don't wait for blood to be available - start volume replacement with colloid.
2. Determine the required rate of repair (e.g. slow onset gives some compensation)
3. Titrate to an appropriate patient end-point
4. Correct 1/2 the abnormality and then re-evaluate
5. Repair deficits
6. Cover continuing losses
7. Provide maintenance

Advantages of Polygeline ("Haemaccel")

1. Long shelf life (5 years)
2. Packaged in an unbreakable, collapsible plastic flask which does not require an airway
3. Similar to plasma and 50% remains in the intravascular space
4. Isotonic
5. T1/2 4-5 hours, renal excretion
6. Does not interfere with cross-matching of blood
7. No limit to the volume that can be infused
8. Very few side-effects

Massive Blood Loss

Where 50% of the circulating blood volume is lost in a short period the optimal fluid for replacement is fresh whole blood. This is often not available but group-specific uncross-matched blood is very safe serologically and if available can be given until fresh cross matched blood is procurable.

It is useful to review the priorities in management of massive bleeding:

1. Cardiopulmonary resuscitation (CPR)
2. First-aid control of haemorrhage
3. Restitution of circulating volume
4. Restitution of oxygen carrying capacity
5. Medical and surgical haemostasis
6. Management of specific injuries

Potential hazards of massive and rapid blood transfusion:

A. Impaired oxygen transport
   - Microaggregates *
   - Fluid overload
   - Defective red cell function *
   - Impaired haemoglobin function *
   - ARDS (adult respiratory distress syndrome) *

B. Haemostatic failure
   - Dilution
   - Depletion of clotting factors *
   - Decreased production

C. Electrolyte and metabolic disturbances
   - Hyperkalaemia *
   - Sodium overload *
   - Acid-base disturbances *
   - Citrate toxicity *
   - Hypothermia *

D. Vasoactive reactions
   - Kinin activation *
   - Damaged platelets and granulocytes *

E. Serological incompatibility

F. Plasticiser toxicity

G. Impaired reticuloendothelial function *

* Complications arising from changes which occur during storage.

CHANGES OCCURRING IN STORED WHOLE BLOOD

Blood is a complex fluid which rapidly deteriorates outside its normal intravascular milieu. Compliated procedures are undertaken to prolong the life of its various components. These manoeuvres, together with the deterioration that occurs in blood in vitro, cause problems when the blood is returned to the cardiovascular system of the patient being transfused.

	Fresh	A.C.D.	$7/_7$	$14/_7$	$21/_7$	$28/_7$
rbc survival	<5% lysed	10%	10%	20%		30% lysed
rbc activity	10% of normal, flexible-----> rigid spherocytes					70% survive after 24hrs
wbc^s	100%-->0 in 24 hrs		0	0	0	0
platelets	3.5×10^5 0 in 24 hrs		0	0	0	0
plasma free Hb						200mgm%
VII					↓70%	
VIII	↓50% in 24 hours		50%			
V	↓50% in 10/7					
Fibrinogen	↓					
Temp	37^0C	$4\text{-}37^0C$	4^0C	4^0C	4^0C	4^0C
pH	7.40	7-10	6.8	6.7	6.6	6.5
pCO_2	46	46	60	150	200	300
pO_2	40-100	40		40	20	5-15
HCO_3	24	15		7-10	2-5	2-5
Na^+	140	150-170	160	150		140
K^+	4-5	4-5	12	20	25	30
Lactic A(mgm%)	20	20		120	140	150
Glucose (mgm%)	80-100	700		500	400	400
Ammonium (ug%)	50	50		500		700
Ca^{++}(mmol/L)	4-5	0.5	0.5	0.5	0.25	
Citrate		500		500		

P_{50}	25.2			19±1.5		
23DPG Activity	100%	ACD	20%	15%	10%	
		CPD	60%	50%		5-10%
23DPG Level (ACD Blood)	0.76mmol/Mole Hb			0.24±0.13		
Microaggregates	few	$15x10^6$	$35x10^6$	$35x10^6$	$70x10^6$	

1 Litre of cold blood removes 25KCal of heat energy from the patient.

Post transfusion: Level of 23DPG returns to normal in 4/7

With increasing expertise and decreasing volumes of whole blood, more use is made of blood components. The acutely haemorrhaging patient who continues to bleed or has established shock is likely to require many of the components of blood.

Available blood components:

Stored whole blood
Fresh whole blood (< 5 days old)
Ultra fresh whole blood (uncooled)
Red cell concentrates
Plasma protein solution
Concentrated 25% albumin
Fresh frozen plasma
Supernatant plasma (Factor 8 depleted plasma)
Cryoprecipitate (Factors 8, VWF, Fibronectin, Fibrinogen)
Platelet concentrates
Fibrinogen concentrates

The patient requiring massive blood transfusion is of course critically ill. If the patient survives, complications invariably occur in which blood transfusion therapy may be contributory - for example:

Respiratory failure
Renal failure
Multisystem failure
Hyperbilirubinaemia
Sepsis
Pyrexia
Haemostatic failure
Venous thrombosis

PRACTICAL POINTS:

For fluid administration in the seriously ill patient.

1. One or more large bore (14G) cannulae in situ plus the facility for measuring CVP.

2. Formulae are imprecise and are only a guide because they may not be based on accurate data and various assumptions are made which may not necessarily apply.

3. Different fluids have different fates

   5% Dextrose --> TBW - 1/3 in ECF and 5% in plasma
   0.9% Saline --> ECF - only 25% remains in plasma
   Colloid --> primarily to the plasma space

4. K^+, Ca^{++} and $Lactate^-$ (Hartmann's solution) are not always appropriate
   Whole blood resuscitation should aim for a haematocrit (HCT) 30%
   Dextrans should NOT be given more than 20ml/kg/day

5. An abnormal laboratory result should always be viewed with caution and checked, especially if it is unexpected or inconsistent.

6. Concomitant problems must be treated particularly

   hypoxia
   acidosis
   sepsis
   major electrolyte disturbances of K^+, Ca^{++}, Mg^{++}.

7. Therapy other than fluid administration may also be helpful during resuscitation, such as steroids, inotropes, antibiotics and anti-arrhythmics.

Modification of fluid intake

Decrease	Adjustment
Humidified inspired air	x 0.75
basal state (e.g. paralysed)	x 0.7
High ADH (IPPV, asthma, brain injury)	x 0.7
Hypothermia	- 12% per °C
High room humidity	x 0.7
Renal failure	x 0.3 (+urine output)
Increase	
Full activity + oral feeds	x 1.5
Fever	+ 12% per °C
Room temperature > 31°C	+ 30% per °C
Hyperventilation	x 1.2
Neonate - preterm (1-1.5kg)	x 1.2
- radiant heater	x 1.5
- phototherapy	x 1.5
Burns - first	+ 4% per 1% area burnt
- subsequently	+ 2% per 1% area burnt

NARCOTIC ANALGESICS

The treatment of chronic pain has improved considerably over the past 10 years. Too little time, staff resources and funding have been devoted to acute pain, which in practical terms is still a largely unresolved problem. Many forms of acute pain are equally in need of attention. e.g.

post traumatic pain
acute "medical" pain (e.g. AMI, acute pancreatitis)
obstetric pain
paediatric pain

HARMFUL EFFECTS OF UNRELIEVED ACUTE PAIN

1. Adverse Psychological Effects

Unrelieved acute pain progressively results in increasing anxiety and interferes with sleep. This rapidly demoralises patients and depletes their psychological reserves.

Children tend to become withdrawn and appear not to be in any discomfort. However, in reality they are in a state of catatonia and severe depression caused by unrelieved pain.

2. Adverse Physiological Effects

a. Atelectasis, hypoxia and hypercarbia.

b. Inability to cough, sputum retention, lobar collapse and pneumonia.

c. Increased sympathetic activity, increased myocardial work , increased myocardial oxygen consumption can produce myocardial ischaemia.

d. Compromised regional blood flow to the brain, kidney & uterus.

e. Gastric stasis.

f. Activation of neuro humoural stress response - α -stimulated vasoconstriction, decreased urine flow, decreased insulin, increased cortisol.

g. Increased platelet aggregation, decreased venous blood flow and skeletal muscle spasm producing splinting and giving rise to deep vein thrombosis and pulmonary embolism.

PHYSIOLOGY OF PAIN

It is important to emphasise individual variability in the pain response, which may be substantially due to psychological factors, and also possibly due to individual differences in levels of endogenous opioids and in receptor responses. Therefore each patient requires an individual pain treatment regimen.

A. CENTRAL MECHANISMS:

Melzack and Wall demonstrated that pain can be modulated in the spinal cord and brain to produce powerful influences on pain perception. This "descending modulation" may help to explain the influence of psychological factors and is certainly important with the centrally acting analgesics. Modulation also occurs as a result of ascending input due to collaterals at several levels.

Interest in acute pain management has focussed on spinal modulation at the level of the substantia gelatinosa (SG), in the dorsal horn of the grey matter. Recent work has indicated the following possibilities for post-operative pain control.

1. At least two different populations of opioid receptors.
2. A noradrenergic system (e.g. relief of postoperative pain by the agonist epidural clonidine).
3. A serotonergic system.
4. Possibly a GABA-ergic system.
5. The eleven amino acid peptide substance P may be depleted or blocked, to interfere with primary afferent transmission.

Since the discovery of opiate receptors, it has been possible to identify endogenous opiate substances (e.g. endorphins, enkephalins) and to increase the understanding of the analgesic actions of endogenously administered opioids. A high density of opiate receptors are found in the limbic system, medial thalamic nuclei, periaqueductal and periventricular grey matter, midbrain reticular formation and the substantia gelatinosa of the spinal cord. The density of opiate receptors increases 20 times between birth and adulthood. Differences in the pharmacological effects of opiates have been ascribed to interactions with different receptor sites e.g. mu (responsible for analgesia and sedation) and sigma (responsible for tachycardia, hypertonia and tachypnoea). Compounds that preferentially interact with the kappa receptor are of potential interest because they would be capable of inducing analgesia and sedation without respiratory depression.

The relative efficiency and safety of these options remains to be evaluated in comparison to older techniques.

B. PERIPHERAL MECHANISMS

There are various factors which decrease or increase the sensitivity of peripheral nociceptors. One example of modifying pain at this level is the inhibition of prostaglandin synthetase by aspirin-like drugs. Local anaesthetics can be applied directly at the receptor level using subcutaneously implanted catheters for wound pain, or at the level of afferent fibres.

C. REFLEX ACTIVITY

Since afferent fibres ascend or descend at least three segments before synapsing in the substantia gelatinosa, reflex activity may extend well beyond the level of noxious stimuli. Such reflexes may include viscero-somatic, viscerosympathetic and viscero-visceral reflexes.

Visceral sympathetic afferents converge on the same dorsal horn neurone as somatic nociceptive afferents. Visceral noxious stimuli are then conveyed, together with somatic noxious stimuli via the spinothalamic pathway to the brain. Note the following:

1. "Referred" pain is felt in the cutaneous area corresponding to the dorsal horn neurones upon which visceral afferents converge. It is accompanied by hyperalgesia in this skin area.

2. Reflex somatic motor activity results in muscle spasm which may stimulate parietal peritoneum and initiate somatic noxious input to the dorsal horn.

3. Reflex sympathetic efferent activity may result in spasm of sphincters over a wide area.

4. Reflex sympathetic efferent activity may result in visceral ischemia, and further noxious stimulation. Also, visceral nociceptors may be sensitized by norepinephrine release and microcirculatory changes.

5. Increased sympathetic activity may influence cutaneous nociceptors and this may be at least partly responsible for "referred pain".

NARCOTIC PHARMACOKINETICS

The absorption, distribution, and elimination of opioid compounds determines their onset, intensity, and duration of action. Pharmacokinetic data are most useful clinically when there is a close relationship between the plasma drug concentration and its effect on the CNS. Analgesics that rapidly equilibrate between the plasma and neuronal tissue will enable effects in the CNS to parallel changes in the plasma levels.

CNS opioid penetration is dependent upon

1. free, non-ionized fraction (non protein-bound)

2. oil / water partition coefficient at pH7.4 (lipophilicity)

Fentanyl therefore enters the CNS approximately 150 times faster than morphine.

Pharmacokinetic data provide the anaesthetist with an understanding of how the body handles a given drug.

The t1/2 = distribution phase (rapidity of drug uptake)

t1/2 ß = elimination phase (persistence of the drug in the body)

Vd_C = central volume of distribution

Vdd_{SS} = steady state volume of distribution

CL = clearance (high hepatic clearance of opioids is dependent on liver blood flow)

$$t1/2 = \frac{0.69\ Vd}{CL}$$

opioid Pharmacokientic and Related Data:

A Summary of Mean Pharmacokinetic Properties of Various Opioids Following Intravenous Administration

	I.V. Potency Ratio$_b$	Ionized pH7.4 (%)	Plasma Protein Binding pH7.4 (%)	Topical Fast Half life (min) (T1/2 α)	Slow Half life (hr) (t1/2β)
Fentanyl	292	91	83	2.3	2.5c
Alfentanil	73	11	91	3	1.3-3.3
Sufentanil	4,521	80	92	1	2.5
Pethidine	0.53	95	65	4.2-11.4	3-7
Morphine	1	76#	35	25	1.4-4
Methadone	1	99	85	10	25-45
Buprenorphine	33	91#	96	3	2-4.5d

Opioid Pharmacokinetic and Related Data: (Continued)

	CL (L/min)	Vss (L) (Vd_{ss})	V (L) (Vd_c)	MEC (ng/ml) (Cp)
Fentanyl	0.8-1.2	375	40-80(v)	1-3
Alfentanil	0.92	36	9-15(v)	100-300
Sufentanil	0.73	98	7-10(v)	
Pethidine	0.5-1.8	250	40-80(v)	300-650
Morphine	0.9-1.5	200	15-30(v)	12-24
Methadone	0.1-0.2	420	50-100(v)	30-70
Buprenorphine	1.1-1.5	180	12-18(a)	d

Vss = (total volume of distribution at steady-state equilibrium;
CL = mean total body clearance;
V = initial dilution volume;
(a) arterial samples;
(v) venous samples;
MEC = minimum effective analgesic blood concentration;
# = ionization of amine moiety;
b rat tail withdrawal model
c Fentanyl undergoes extensive redistribution in the body. This results in a short duration of action after small or infrequent doses. However, after large or frequent doses, the slow half-life may be > 13 hours.
d Buprenorphine blood concentration and slow half-life do not determine analgesic response. This is determined by rate of dissociation from receptors.

The durations of analgesia produced by various opioids are related to redistribution (fentanyl) and elimination (methadone) processes. For the short acting alfentanil, both processes are important. Although fentanyl is generally considered to be short-acting as a result of its rapid redistribution, if given in high-dose, repeated or continuous administration, fentanyl can become long acting because of its long elimination half-life (t1/2ß), if enough of the drug is given.

The therapeutic plasma concentration (Cp) or MEC (minimum effective analgesic blood concentration) has a wide range depending on the surgical stimulus and anaesthetic conditions. The required Cp depends upon an individuals:

1. CNS sensitivity
2. type of operation
3. presence of adjunctive drugs (sedatives, muscle relaxants, N_2O)

Pharmacokinetic knowledge has now provided the rational basis for <u>increased efficiency</u> and <u>safety</u> of opioids delivered as

1. controlled rate of I.V. infusion
2. on demand patient activated infusion
3. orally
4. regular, prescribed intramuscular injection (NOT p.r.n.)
5. transdermally.

Methadone can provide up to 24 hours of pain relief after a single I.V. dose, due to its long elimination half like which reflects the drugs large volume of distribution and low clearance rate.

By using a continuous infusion technique, a drug can be titrated to the patients needs and thereby:

1. minimise the amount of drug administered
2. improve pain relief
3. decrease recovery time.

The loading dose (L.D.) and initial maintenance infusion rate (MIR) are calculated as follows:

$$Ld\ (\mu g/kg) = Cp(\mu g/ml) \times Vd(ml/kg)$$

$$MIR(\mu g/kg/min) = Cp(\mu g/ml) \times Cl(ml/kg/min)$$

Because achievement of a steady state drug concentration is slow with a constant infusion, a loading dose is required to boost plasma levels of the drug initially. This can be administered as a bolus or short priming infusion. If a smaller LD is given then a higher initial MIR will be required because the amount of drug infused must equal that which is effectively removed from the brain by both redistribution and elimination.

In time, redistribution becomes less important and the infusion rate is dependent on the drugs clearance from plasma. Thus the MIR decreased during the infusion. Clearance is often lower than predicted in steady state infusions, particularly in the critically ill, morbidly obese and with cardiopulmonary bypass.

PHARMACODYNAMICS

The pharmacologic effects of opioid compounds on the various organ systems include:

CNS - Opioid compounds produce a similar spectrum of action on the CNS, with dose-dependent depression resulting in analgesia, sedation, and ultimately, loss of consciousness.

Cardiovascular - Hemodynamic stability is usually associated with the opiates. However, bradycardia (due to stimulation of the medullary vagal nucleus) and hypotension (due to arterial and venous dilation) can occur during high - dose narcotic techniques.

Respiratory - A dose-dependent decrease in the responsiveness of the respiratory center to carbon dioxide is seen with all opioid. agonist compounds.

Gastrointestinal - Narcotic analgesics directly stimulate the emetic chemoreceptor trigger zone and increase smooth muscle tone (e.g. pylorus, sphincter of Oddi, ileocaecal valves).

Renal - Opiates increase bladder detrusor and urethral sphincter tone, which can result in urinary retention.

Neuromuscular - Truncal muscle rigidity may occur when opiates are rapidly administered in high doses and may be further enhanced by N_2O.

Metabolic - High-dose narcotic anesthesia (e.g. fentanyl) is alleged to be more effective than inhalation anaesthesia in blunting the release of "stress" hormones (e.g. catecholamines, glucocorticoids, aldosterone) in response to surgical stimulation .

All the opioids are readily absorbed from the GIT, nasal mucosa, lungs and of course parenterally. The oral dose of morphine is considerably less effective than the equivalent I.V. dose because the drug undergoes "first pass" hepatic metabolism. Less of the drug reaches the systemic circulation, and that which does is mostly as the pharmacologically inactive glucuronide. It must also be remembered that the majority of strong analgesics are excreted by the kidneys to a clinically significant extent as the unchanged drug.

Alfentanil is a rapid, short-acting analgesic useful in outpatient anaesthesia and to block the stress response of intubation. Sufentanil is a potent, rapid, but longer acting analgesic. The agonist-antagonist compounds have a "ceiling" effect on respiratory depression and a lower incidence of side effects, however their analgesic potency is limited.

Agonist <----------> Antagonist

(strong narcotic Analgesic) (narcotic antagonist)

Fentanyl	Morphine	Pethidine	Pentazocine	Naloxone
sufentanil	Methadone		Butorphanol	
Alfentanil			Nalbuphine	

Spectum of Activity of the Opioid Drugs

Classification of Analgesics According to time to Peak Effect:

Ultrashort	Short	Intermediate
Alfentanil	Fentanyl	Morphine
	Sufentanil	
	Pethidine	
	Methadone	

Classification According to Duration of Action:

Short	Intermediate	Long
Alfentanil	Fentanyl	Morphine
	Sufentanil	Methadone
	Pethidine	Buprenorphine
	Butorphanol	
	Nalbuphine	

In spite of all the physiological, pharmacokinetic and dynamic data if the drug is not administered, the patients pain remains unalleviated.

Administrative and Logistical Problems in Opioid Use

Problem	Cause
Inadequate doctor's orders:	
1. Dose too small, interval too long	Inadequate pharmacokinetic knowledge and failure to observe analgesic effect
2. Range of doses ordered	Unwillingness or inability to assess opioid needs of individual patients
3. Opioid order prn	
4. Alternative nonopioid analgesics ordered	
Inadequate interpretation or implementation of orders by nurses:	
1. Use of lower range of opioid dose, encouraging long intervals between doses. Interpretation of prn as "as little and infrequently as possible"	Fear of addiction and respiratory depression
2. Delay in administering opioid when patients report pain.	Delays due to: Deciding if nurse thinks patient has pain Convincing senior nurse Staff attitude to that patient Availability of senior nurse Finding key to drug cupboard Demands of other patients
Difficulties in patient communicating analgesic needs:	
1. Problems due to language, intelligence, or age (e.g. very young and old).	
2. Ethnic, cultural, and psychological background leading to over, or under reporting of pain.	

Most of the non-compliance of medical practitioners in administering narcotics is due to ignorance:

Misconceptions in the Use of Opioid Drugs in Acute pain

---

1. Doses should be as small and infrequent as possible to avoid development of addiction.

2. Doses larger than the standard do not increase pain relief and cause heavy sedation and respiratory depression.

3. Nursing and/or medical staff know when and how much pain relief each patient needs.

4. Patients requesting more pain relief than the standard are psychologically abnormal or are becoming addicted.

5. If nonopioid drugs are also ordered, these should be tried in preference to opioids.

6. What is needed is a powerful nonaddictive pain reliever.

THE ABOVE ATTITUDES ARE **WRONG** AND **INAPPROPRIATE** TO THE PATIENTS **REQUIREMENTS!!**

CLINICAL USES OF NARCOTICS

A. PREOPERATIVE ADMINISTRATION

The major problem with the routine use of analgesics preoperatively is the high incidence of side effects (nausea, retching, dizziness, flushing, chest tightness, dysphoria). Sedative-anxiolytic drugs can potentiate opioids and therefore in combination can allow a reduced dose to be administered. The side effects which are dose may be reduced, but excessive sedation can occur. An opiate (e.g. alfentanil 0.3mg/kg I.V.) can be administered 90 seconds prior to induction to blunt the haemodynamic responsed to intubation and as continuous alfentanil infusion will decrease subsequent anaesthetic requirements.

B. INTRAOPERATIVE ANALGESICS

Advantages Claimed of Narcotic Anaesthesia

1. Minimal cardiac depression.
2. No sensitization of the heart to catecholamines.
3. Preservation of blood flow autoregulation (CNS, heart, kidney).
4. No interference with autonomic or cardiovascular drug, actions.
5. Tolerance of an endotracheal tube and airway manipulation.
6. Facilitation of mechanical ventilation.
7. Postoperative analgesia.
8. Arousable patient.
9. Antagonist available.
10. No primary hepatic or renal toxicity.
11. Not a trigger of malignant hyperpyrexia.
12. Not teratogenic.
    - but newborn depression after maternal dose
13. No environmental pollution.
    - but potential for abuse and dependence.

To exert their "sole anaesthetic" properties, analgesics must be administered in doses 10-30 times more than those used for conventional analgesia. Of the available analgesics, fentanyl has been the most successful for the induction and maintenance of anaesthesia. When a high dose fentanyl technique is used, plasma concentrations in excess of 18ng/ml are required to maintain haemodynamic stability during cardiovascular surgery. A loading dose equal to 50-70 micg/kg followed by a MIR of 0.3-0.6 micg/kg/min would be expected to achieve a steady state fentanyl level of > 20 ng/ml.

One of the most serious problems associated with opiate "anaesthesia" is the high incidence of inadequate anaesthesia (awareness, movement, sympathetic autonomic hyperactivity). Supplementary drugs are added to provide greater depth of anaesthesia without increasing the incidence of intraoperative hypotension or producing prolonged post-operative depression.

Supplementation of Narcotic Anaesthesia

To prevent or control:	Supplemental drugs
Awareness intraoperatively	hypnotic, nitrous oxide, or volatile anaesthetic
Recall of perioperative events	nitrous oxide, benzodiazepine, or Hyoscine
Hypertension	vasodilator or volatile anaesthetic
Tachycardia	ß- sympatholytic or volatile anesthetic
Muscular rigidity	muscle relaxant or volatile anaesthetic

Opioid infusions are now frequently used in "balanced" anaesthesia with N_2O and muscle relaxants. In fact, pharmacokinetic/dynamic studies with fentanyl and alfentanil have defined therapeutic plasma concentrations for a variety of surgical procedures.

C. POSTOPERATIVE ANALGESIC THERAPY

1. Intermittent intramuscular injection:

This often results in inadequate control of postoperative pain because of widely fluctuating blood levels, especially if administered p.r.n.. The minimum effective blood concentration has been shown to be exceeded for only 35% of a 4 hour period, when the patient required analgesia .

2. Opioid Infusions:

The use of an infusion of a medium to high clearance opioid with a short terminal half-life has become a routine in some surgical wards and intensive care units. The infusion is adjusted until the MEC for analgesia has been exceeded and this blood level is maintained. In this way continuous analgesia is given with minimum side effects. Repeated injections, discomfort and fluctuating blood levels are avoided.

The risk of overdosage must be carefully monitored by

1. analgesic response

2. respiratory rate

Therefore the infusion equipment must be simple, safe, reliable and the opioid infusion should follow a prescribed protocol. Trained personnel should recognise and treat appropriately both inadequate and excessive blood narcotic levels.

3. Patient Controlled Analgesia (P.C.A.)

PCA adresses two important issues

a. variability in patient requirements
b. pain at rest and "incident" pain (e.g. physiotherapy) can be treated.

PCA allows patients to increase the dose rate when the pain increases and reduce it when they have less pain. Hourly usage rates are similar to those for "maintenance infusions".

4. Single Dose Analgesia

Methadone has a long elimination half-life. Stable "analgesia" blood concentration lasting 20-30 hours after a 20 mg bolus dose are possible. There is however considerable patient variability and initial doses must be carefully titrated.

Supplementary doses must be given by

1. trained personnel

2. only if pain is reported spontaneously

3. only if the unstimulated respiratory rate is greater than 12/min.

A further feature of methadone is its high bioavailability when given orally.

5. Intrathecal Opioids

Morphine 0.25-0.5 mg gives good analgesia after an onset time of 15-45 minutes, with a duration of about 12 hours. Delayed respiratory depression occurs in 0.36% of patients. The slow onset (3-6 hours) relates to the slow cephalad spread of the drug to the brain stem. This is predisposed to by high dose, sensitivity to opioids, administration of other opioids, serious illness and advanced age. The incidence of respiratory depression with the more lipid soluble drugs (fentanyl) appears much lower. Other problems such as urinary retention, itching, nausea and vomiting do however occur.

6. Epidural Opioids

Low dose epidural opioid infusion as a mixture with dilute bupivacaine is very effective and has a low incidence of side effects. Epidural morphine seems most appropriate for acute pain of wide spread origin (post thoracotory, upper abdominal surgery) and a lumbar catheter is just as effective as a thoracic one because of morphine's CSF migration. Lipid soluble opioids such as fentanyl or pethidine may be more appropriate with pain from a more limited segmental area.

Management of Severe Postoperative Pain

First 24 hours:

? Epidural local anesthetic by infusion, in critical care area (± epidural opioid)

From 24 hours onwards:

Lower abdominal and lower limb surgery: Lumbar epidural catheter and fentanyl or pethidine infusion or top-ups by trained staff in medium care area. Naloxone available.

Upper abdominal-thoracic surgery (extensive pain stimulus). Epidural morphine tops-ups by trained staff in critical care area. (Lumbar catheter as effective as thoracic). Naloxone available.

For marked increase in pain stimulus (e.g. vigorous physiotherapy to chest, hip or knee) Epidural local anesthetic, 0.25% bupivacaine (avoids problems of additive respiratory depression of epidural and parenteral opioids)

Side Effects
Spinal Opioids vs Local Anesthetics

	Opioids	Local Anaesthetics
C.V.S.	No change in HR, CO or BP unless toxic dose	HR, CO and BP may be decreased with high dose
	Vasoconstrictor response intact	Postural Hypotension
RESP	Early decrease (1-2h) - Drug in Plasma Late decrease (6-24h) - Drug in CSF	Usually unimpaired

7. Transdermal Fentanyl

   Effective blood concentrations can be achieved and sustained, with an efficacy similar to IV infusion, however there is a 6-8 hour "lag time" in achieving analgesia and a slow washout time from the skin after removal of the patches.

8. Sublingual, Oral "lollipop", Subcutaneous Infusion

9. Local Anaesthetic Neural Blockade

   Local anaesthetic drugs provide the best quality analgesia as pain can be abolished rather than diminished. However there is always some degree of motor and sympathetic block accompanying the sensory block.

   a. Intercostal Nerve Blocks
   b. Paravertebral Block
   c. Continuous Epidural Blockade
   d. Epidural local anaesthetic/opioid mixtures
   e. Femoral N. Block
   f. Continuous Brachial Plexus Block

10. Transcutaneous Electrical Nerve Stimulation (TENS)

    There is a 50% reduction in opioid requirements, though TENS is not effective by itself.

11. Cryoanalgesia - especially for post thoracotomy and inguinal herniorraphy.

SUMMARY:

There are now many drugs, techniques and a considerable amount of pharmacokinetic data available for pain control. To utilise these techniques properly there must be:

1. commitment to provide staff and equipment to supervise and deliver acute pain relief.

2. training of medical and nursing staff in the basic principles of pain control.

References:

Cousins MJ, Philips GD, (eds): Acute Pain Management, Churchill Livingstone, Edinburgh, 1986.

Cousins MJ, Bridenbaugh PO, (eds): Neural Blockade in Clinical Anaesthesia and Pain Management 2nd ed, Lippincott, Philadelphia, 1987.

Prys-Roberts C., Hug C.C. Jr eds. Pharmacokinetics of Anaesthesia, Blackwell Scientific, London 1984.

COMPLICATIONS OF ANAESTHESIA

Like all therapeutic or diagnostic intervention, anaesthesia has its complications. Some are virtually unavoidable, although their frequency can be reduced. The common complications, fortunately, are minor, but they are nevertheless important, since in many instances they are what the patient remembers most, and influence his or her subsequent attitude to surgery.

MINOR COMPLICATIONS

By far the commonest minor complication of anaesthesia is VOMITING. Although there are a few patients who seem to vomit after any anaesthetic, the incidence is normally affected markedly by the following:

1. Premedication

   Unpremedicated patients vomit only one third as often as those who receive a premedicant of any kind.

2. Duration

   Vomiting is commoner after long procedures.

3. Nature of surgery

   Eye and ear operations are notorious for the frequency with which patients vomit post-operatively

4. Anaesthetic Agents

   Some agents are much more inclined to produce vomiting, and the abandonment of cyclopropane and ether has to some extent been due to the high incidence of vomiting after their use. Every new agent introduced into anaesthesia is claimed by its manufacturer to be less likely to produce vomiting.

Apart from the avoidance of certain agents, and perhaps the omission of premedication, other factors are not under the anaesthetist's control. The question which then arises is how to deal with the patient who claims that they "always" vomit after anaesthesia. Antiemetics are an obvious answer, but their timing is controversial, especially since some are soporifics as well, and can prolong recovery times. Others (prochlorperazine, thiethylperazine) have the ability to produce dystonic reactions in a percentage of patients, and are particularly contraindicated in day surgery for this reason.

The drug which is least likely to cause problems seems to be metaclopramide, but it also has a lower effectiveness. It has to be admitted that the problem of vomiting after (general) anaesthesia is as yet unsolved.

MINOR TRAUMA is the next commonest complication. It ranges from the trivial, such as a haematoma associated with venepuncture, to the potentially significant, such as a corneal abrasion. The following list is probably not complete.

Bruising or laceration to lips and/or tongue. This can arise as a result of the anaesthetist's manipulations, or may occur during emergence, due to the patient biting.

Tooth trauma, especially when the upper anterior teeth are loose or prominent, or the patient is difficult to intubate.

Sore throat, especially when packs have been used. It was also a very common complication of intubation, but has become less so now that plastic tubes are replacing red rubber.

Haematoma, as mentioned above, can be avoided by the prompt application of pressure dressings after the removal of needles or cannulae. In special circumstances, e.g. when large cannulae have been used, or an attempted cannulation has been unsuccessful, a crepe bandage should be applied for the duration of the case.

Infusion phlebitis which is a chemical or physical irritation to the vein producing an inflammatory reaction, can be a very troublesome minor complication, leading to prolonged hospital stay. It is caused, for the most part, by inappropriate solutions (e.g. hypertonic dextrose), antibiotics, prolonged infusion time, and the selection of small veins for cannulation.

Eye trauma is uncommon, largely due to the almost universal practice of protecting the cornea by taping the eyes shut. If the cornea is abraded, immediate application of an oily-based antibiotic ointment, such as chloramphenicol, is an effective treatment.

Epistaxis may complicate transnasal intubation, but always settles down.

A very small incidence of granuloma of the vocal cords may be due to intubation during anaesthesia.

Minor neurological complications, such as ulnar paraesthesia, due to prolonged pressure on the nerve against a hard object during the case. Perhaps commoner are the temporary paraesthesias or neuralgias which follow local blocks, and which may be due to transfixion of a nerve by an exploring needle with resulting intraneural haematoma.

MAJOR COMPLICATIONS

The obvious major complication of anaesthesia is cardiac arrest, which may arise due to a number of causes, more often associated with the patient's prior medical condition, or the surgery being performed. A few such events are of wholly anaesthetic origin, but although uncommon, they should be mentioned first because of their importance.

Hypoxia

However produced, must ultimately lead to cardiac arrest, and death of the patient. Rarely is it due to oxygen deprivation by failure of the gas supply. More often it results from an airway complication, such as accidental extubation or oesophageal intubation. Where mechanical ventilators are in use, disconnection is a potent cause of hypoxia. More subtle causes are one-lung ventilation, due to a tube being displaced into the bronchus after insertion, cuff herniation with increasing obstruction, or pneumothorax arising as a complication of surgery or central venous line insertion.

Drug overdose

Was once a common cause, but with the introduction of calibrated vaporisers which are always placed outside the breathing circuit, volatile agent overdosage is now rare. Intravenous agents, especially thiopentone, are also nowadays used in lower doses, and rarely cause trouble. In the case of local anaesthesia. arithmetical errors in calculating dosage, or accidental injection into a vessel can lead to serious toxic reactions which are dealt with elsewhere.

Drug Incompatibility

When volatile agents and adrenalin are administered sequentially, the myocardium may first be sensitised, then stimulated into an arrhythmia leading to arrest.

Anaphylaxis

Once rare, anaphylactic or anaphylactoid responses to anaesthetic drugs are becoming commoner, and may be very life-threatening. The onset of the condition is marked by sudden hypotension, increased resistance to ventilation, cutaneous flushing or cyanosis and tachycardia. Adrenalin is the specific treatment, and is life-saving. It must be given generously, in doses up to 1 mgm every 3 minutes, administered intravenously, in a dilution of 1 in 10,000. Fluid infusion and maximum oxygenation are helpful in overcoming hypotension on the one hand and hypoxia on the other. No other measures have been shown to be useful.

Aspiration of gastric contents

A justifiably feared complication of general anaesthesia is regurgitation or vomiting and aspiration of this material into the lungs. Apart from the imminent danger of asphyxiation by drowning in liquid vomitus, or the much rarer obstruction of the airway by a large chunk of undigested food, there is the chemical pneumonitis which results from the effect of highly acid fluid on the tissues of the lungs and bronchi.

This acid-aspiration syndrome is also named after the New York obstetrician, Mendelson, who first described and reproduced it in rabbits. It may develop within minutes after inhalation, or may not be manifest for several hours, and is diagnosed by the triad of hypoxia, wheezing and moist rhonchi accompanied by dyspnoea, whilst the chest X-ray often shows a pattern of widespread, discrete nodules of consolidation spreading out fan-wise from both hila, often described as a "snowstorm".

Treatment is urgent, and consists of oxygen by appropriate means to elevate the PaO_2, and may require intubation and mechanical ventilation. Intravenous fluids, with possible inotropic support in severe cases, will also be required. Steroids are useless, and antibiotics of prophylactic value only against a complicating pneumonia.

The condition was once common in obstetrics, but the universal practice of cricoid pressure and intubation, and the even more effective substitution of regional for general anaesthesia in obstetrics has produced a marked decline. It should be noted that aspiration can and does occur in the post-anaesthetic period, especially if the patient is nursed supine whilst still semiconscious and/or under the influence of muscle relaxants.

Major Trauma

Major nerve injury can occur to the brachial plexus or the radial nerve due to poor posturing or pressure from hard objects such as screens or retractor supports. Stirrups can also compress the common peroneal nerve against the head of the fibula.

Very occasionally, an unconscious patient can actually fall from a table or trolley due to clumsiness in handling or instability of the patient just before consciousness is lost.

Contrary to some texts, major neurological complications are exceptionally uncommon after spinal anaesthesia. Paraplegia has been reported, but hardly at all in recent times, whilst the incidence of permanent neurological defect after epidural anaesthesia/analgesia is also extremely rare, being many times less than mortality following general anaesthesia, which has already reached the respectably low figure of 1 in 10,000. Mortality due to surgical causes on the other hand is approximately 1%, although of course there is wide variation from operation to operation.

Burns

Can occur with the combination of diathermy, flammable skin preparations and anaesthetic gases.

Pneumothorax

May result from supraclavicular brachial plexus block or injury to the pleura and lung during the insertion of a central venous line by the internal jugular or subclavian approach.

Malignant Hyperpyrexia:

This rare condition is a genetic abnormality of muscle which is unmasked by the administration of certain anaesthetic agents, notably the volatile halogenated hydrocarbons. The muscle relaxant succinylcholine is also a relatively weak trigger. The sequence of events which takes place begins with an increased production of CO_2, followed by a tachycardia and decreased arterial oxygen saturation. Temperature rise and muscle rigidity are late signs, and later still acidosis and hyperkalaemia appear. The condition had an extremely high mortality until the availability of a specific antidote, dantrolene, and even now can be fatal unless it is recognised early and energetically treated. Its incidence is low in Chinese, but in certain areas of the United States, notably Wisconsin, is very common.

Succinylcholine Apnoea

A commoner, but much less serious complication is prolonged apnoea following succinylcholine administration, which is due to congenital absence of pseudocholinesterase. Paralysis which normally does not exceed 5 minutes after this relaxant is prolonged for up to 3 hours. If effective artificial ventilation is provided, the patient comes to no harm. The incidence of the homozygous genetic situation is variously calculated at 1-3000 to 1 in 5000 of the population.

AWARENESS

The complication of general anaesthesia which is most seriously feared by patients is that they will be awake when they should be asleep. This possibility, once remote, has been made much more possible by the extensive use of muscle relaxants, and has been estimated by Utting to be as high as 1%-2% of all patients undergoing general anaesthesia with muscle relaxants. Of course many of these patients will be aware for only the briefest of intervals, perhaps during intubation. A few, however, are paralysed but awake during a particularly painful part of the operation, and are understandably terrified by this experience.

Prevention obviously consists of the administration of adequate anaesthesia. In this connection, the common practice of administering more muscle relaxant if a patient moves is to be deplored. If a patient claims that he or she has felt or heard something during anaesthesia, the statement must be sympathetically heard, and a reasonable explanation given. What the patient wants to know is whether this will always happen, and if reassured that the forewarned anaesthetist can take steps to prevent awareness occurring again, this will relieve their anxiety.

THE RECOVERY ROOM

One of the most significant advances in anaesthesia has been the incorporation into all modern operating suites of a Recovery Room, in which patients are detained during the transition from anaesthesia to full consciousness, return of muscle power and circulatory stability.

The effectiveness of this environment in detecting and successfully managing problems which can arise during the early post-anaesthetic period depends on the presence of a well-trained nursing staff and the availability nearby of anaesthesiologists with the necessary resuscitative skills. To a lesser degree, the presence of specialised equipment in the appropriate numbers is helpful - the defibrillator, suction, oxygen and the means of inflating the lungs and so on.

A period in the Recovery Room should follow both regional and general anaesthesia, especially if the latter has involved significant autonomic blockade.

The foregoing incomplete description of complications which are possible after even the shortest anaesthetic reinforces the concept that there is no such thing as a "trivial" anaesthetic, no matter how brief or straightforward the surgical procedure.