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SAFE ANAESTHESIA

by

Lucille Bartholomeusz

1986

Third edition updated and revised

by

Jean Lees

2006

DrWael

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First edition 1986

Second edition 1996

Third edition 2006

Preface to the third edition

In revising this well-respected book I have tried to retain the original structure and, particularly, the ethos of the book in keeping with its origins as described in the preface to the 1st edition.
The whole text has been updated and revised in line with current anaesthetic teaching. I have added new chapters on hypothermia and acute pain relief and have amalgamated and re-arranged some other chapters for easier reference.
There have been many people (some unknown to me) involved in the revision and I thank them all. Andrew and Kellie Robinson who put the whole book on disc made an enormous contribution. Others have contributed sections, ideas and illustrations – Ruth Colman, Bill Casey, Ray Towey, Grant Haldane, Alex Bojarska and Roger Eltringham and John Main.
Lois Gibson encouraged me to tackle this enormous task and gave of her experience and invaluable help throughout the revision of the text.
Iain Wilson has been a great support and encouragement and, along with Isabeau Walker, has scrutinised the whole text, given advice and helpful suggestions throughout.

I have added a number of illustrations and diagrams and I gratefully acknowledge permission to reproduce these from:

- World Health Organisation.
  Anaesthesia at the District Hospital 2nd ed. Michael Dobson
- WA /WFSA
  Update journals edited by Walters & Wilson.

Thanks also to Lin and Paul for the cover design.
Finally thanks to my husband Norman who has collected and prepared many of the illustrations, assembled the index and encouraged me to continue when difficulties arose. Without his help and support this project would not have reached completion.
I hope this text will continue to assist all anaesthetists in developing countries who, with limited support and training opportunities, are working hard to improve conditions for their patients.

Jean Lees
MB,ChB. FRCA
August 2006

Preface to the first edition

How this book came to be written

I am a Christian. I believe that a life surrendered to Christ is used of Him to His glory and the benefit of others. In doing so the Christian is enriched and blessed a hundred fold.

While pursuing my postgraduate training in anaesthetics I was conscious of a very strong desire to do this. To let the knowledge and experience I had gained be available for His use.

One year after I finished my training as an anaesthetist an article appeared in 'Africa Now' (which was then the magazine of the Sudan Interior Mission). This described the need for an anesthesiologist to start a training program for nurse anaesthetists in order to equip them to carry out anaesthetic duties in Mission hospitals. The surgical workload in these hospitals was comparable to a small district hospital in Australia. The only language required for teaching was English. If my desire to serve was sincere, then surely this job must have been meant for me.

Arriving in Nigeria I found three very competent Nigerian nurses ready to commence the training program.

At the conclusion of the year, the course completed, the trainees dispersed to the various hospitals they came from, and I returned home pledged to go back if I was needed again.

Five years later it was suggested I organise another such training course. Awaiting me this time were four students; two males and two females. We had a very good year of work and fellowship.

Before my return home I had many requests that the notes used for the training program be published. This was suggested for the following reasons:

- Textbooks written for those anaesthetists with less than the conventional training were difficult to obtain.
- Library facilities were not available in the smaller hospitals. It is important for every anaesthetist to possess at least one book covering most aspects of anaesthesia. A need was felt for a text which dealt with basic anaesthetics and which would also be useful as a reference book when unusual surgical procedures were performed, unusual medical problems were met with and when complications set in during and after the anaesthetic.
- Techniques used successfully in Developing Countries, embodying safe modern principles of anaesthesia using drugs, equipment and facilities available, should be documented.

- The experience gained in setting up an anaesthetic service and organizing a training program for nurse anaesthetists should be documented for the use of others.

My first reaction to this was a very definite "no". I did not feel capable of fulfilling this request. I could think of many other anaesthetists who if they applied themselves to the task would do a far better job. It was too big a task and too time consuming anyway. A few months after I returned home a very dear friend from Sri Lanka, my country of birth, wrote to me with the suggestion that I write a book based on my experiences in Nigeria. This made me feel decidedly uneasy. Is this what I should be doing? I then prayed that if this was part of the work God wanted me to do I would be obedient in doing it. I pledged myself that if one more request was made I would consider writing a book.

A few months later, my very dear friend Lois Gibson, who was also one of my trainees, working in Niger, returned home unexpectedly. A phone call conveyed a special request that a Handbook of Anaesthesia be published.

The techniques described in this book are used successfully today in three hospitals in Nigeria, one hospital in Niger and one hospital in the Peoples Republic of Benin.

This book could not have been written without the help and encouragement of Lois Gibson and my many medical and Christian friends, both here and in Nigeria. A special word of thanks to my husband, John for his quiet encouragement and help, especially at those times when my enthusiasm was waning. To our dear friend, Harry Reynolds, without whose wisdom, expertise and hard work this book would not have been possible, my very sincere thanks. Acknowledgement is made to the following, for kind permission to reproduce the diagrams:

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- Churchill Livingstone
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- Oxford University Press
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My thanks go to my trainees and patients in Nigeria from whom I learned so much. This book goes out to those who labour to give of their best under very difficult circumstances.

Lucille Bartholomeusz MB BS, FFARACS December 1986

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CHAPTER 1

PRINCIPLES AND HISTORY OF ANAESTHESIA

Outline:

Principles of anaesthesia

Methods of surgical anaesthesia

History of anaesthesia

PRINCIPLES OF ANAESTHESIA

The first object of anaesthesia is the abolition of pain

Other features of a good anaesthetic:

- It must be completely reversible. Ideally, the patient must not be worse off for having an anaesthetic. It is not always possible to achieve this, as for instance in patients with chronic respiratory disease having general anaesthesia.
- It must be as safe as possible.
- It should provide good operating conditions. For example, good relaxation for abdominal surgery or lowered intracranial pressure for neurosurgery.
- It should be acceptable to the patient.

These features of an anaesthetic are ones that we must aim at achieving. However, it is not always possible to achieve them all.

Patient comfort is important but safety is essential and must come first. Nevertheless, the greatest possible regard must be paid to the patient's feelings. It is good to remember that the simplest methods will be acceptable to the patient if they are presented with gentleness and consideration.

Anaesthesia may be achieved with either:

- Regional anaesthesia alone e.g. spinal anaesthesia, arm block
- General anaesthesia
- A combination of regional and general
- Regional anaesthesia with sedation

METHODS OF SURGICAL ANAESTHESIA

Local or regional anaesthetic techniques

The conduction of painful impulses from the site of injury to the brain can be interrupted at one of many points, giving us:

- Surface anaesthesia
- Infiltration anaesthesia
- Nerve block/plexus block
- Epidural anaesthesia
- Spinal anaesthesia

General anaesthetic techniques

- Mask anaesthesia (including laryngeal mask)
  - Induction either intravenous or inhalational
  - Maintenance during surgery with the patient breathing spontaneously using an air/O_2/ inhalational agent +/– IV analgesia
- Endotracheal anaesthesia
  - Induction either intravenous or inhalational
  - Intubation using an IV muscle relaxant or an inhalational agent
  - Maintenance during surgery either with spontaneous respiration as for mask anaesthesia

    or

    intermittent positive pressure ventilation (IPPV) using a long acting muscle relaxant in addition to air/O_2/inhalational agent +/– IV analgesia
  - Reversal of the muscle relaxation at the end of surgery

Balanced anaesthesia

This is an important concept. It views general anaesthesia as comprising three parts:

- Anaesthesia (i.e. being asleep)
- Analgesia (i.e. pain relief)
- Muscle relaxation (which may or may not be necessary)

Awareness

The problem of awareness (where the patient is awake or partially conscious during surgery, when thought to be fully anaesthetised) is being acknowledged today as a preventable problem and the anaesthetist must ensure that the patient is not aware under general anaesthesia.

Responsibilities of the anaesthetist

Responsibility, for the anaesthetist, starts and finishes in the ward. Anaesthetised patients can be compared to people setting out on a journey – in this case a journey into unconsciousness and back – and it is the anaesthetist who steers the course.

Anaesthetists must therefore know as much as possible about their patients – how they are made and how they function. They must assess each patient carefully before the journey. They must know how to modify techniques to compensate for pre-existing problems and for when unexpected problems occur. The journey must be as safe as possible.

They must know their drugs, their equipment and their techniques – specific techniques for specific situations. They are honour-bound to watch their patients meticulously on the journey in order to detect any problems as soon as they arise and treat them appropriately. They must be experts in resuscitation.

A safe journey back to consciousness must be the anaesthetist's ultimate goal, though this cannot always be guaranteed. Ideally, the patient should be returned to the ward fully conscious, none the worse for the journey and if possible, even better.

HISTORY OF ANAESTHESIA

1540 Ether was prepared by Valerius Cordus, and called "sweet oil of vitriol". It was not used as an anaesthetic until the nineteenth century when William Morton, a Boston dentist, used it.

1772 Joseph Priestley in England prepared nitrous oxide but it was not used as an anaesthetic until about 1870.

1842 Crawford Long administered the first ether anaesthetic. The operation performed was the excision of a tumour from the neck of a young man. By 1846 Long had performed eight operations under ether anaesthesia but he failed to publish his work until 1849. He never got the recognition due to him as a pioneer in this field.

1846 William Morton in America was the first person to use ether to induce anaesthesia for a surgical operation.

1847 James Simpson, a Scottish obstetrician, used chloroform as an anaesthetic agent.

For the next 50 years ether and chloroform dominated the anaesthetic scene.

1848 Hannah Greener, aged 15, died while having her toenail removed under chloroform anaesthesia. This was the first reported death under chloroform.

1853 Queen Victoria received chloroform for the birth of Prince Leopold, thus giving the drug the royal stamp of approval.

1870 Nitrous oxide was introduced into clinical practice around this time.
It had been used since 1810 as 19th century "pot". At parties, observers noted that under its use pain sensation was abolished. In fact a dentist by the name of Horace Wells had tried to demonstrate the efficacy of nitrous oxide before a sceptical audience. The demonstration failed for no fault of the nitrous oxide. (The patient, as we know now, was most unsuitable for nitrous oxide anaesthesia). If only Horace Wells had known that nitrous oxide was destined to grow in popularity to become one of the most widely used anaesthetic agents today, he would have died a happy man.

The disadvantages of ether, the dangers of chloroform, and the lack of potency of nitrous oxide led to the discovery of other agents, some of which are still in use.

1884 Local Anaesthesia was the next big discovery. Cocaine was widely used at first, especially in E.N.T. surgery. Its stimulant action on the central nervous system and its effect on the heart and on respiration has taken it out of regular use.

1905 Procaine or novocaine was the first synthetic local anaesthetic drug to be used.

1920 Magill and Rowbotham used the first endotracheal tube to meet the needs of the plastic surgeons of the time.

1920's & 1930's ***These were important years for anaesthetic education and development***

- National Anaesthesia societies were established in Britain, USA and Australia.
- Chairs of Anaesthesia were established on both sides of the Atlantic. Sir Robert Macintosh was Professor of Anaesthesia in Oxford; Ralph Waters was his counterpart at the University of Wisconsin.

This resulted in rapid progress in pharmaceutical preparations and apparatus.

1934 Thiopentone was introduced by Lundy.

1930 – 1940
The circle absorber was developed. Blood transfusions were used more frequently than before.

1942 Curare or tubocurarine was employed by Harold Griffiths. This was followed by the use of artificial ventilation for patients paralysed with muscle relaxants.

1943 Macintosh introduced the curved blade laryngoscope.

1956 Michael Johnstone in UK first used halothane.

1966 Ketamine and enflurane were used clinically.

During the last 35 years, anaesthesia, whether local or general has been made safer and more pleasant for the patient as a result of:

- Safer drugs
- More precise anaesthetic and monitoring equipment
- Better training programmes for anaesthetists

CHAPTER 2

ANATOMY OF THE RESPIRATORY TRACT IN RELATION TO ANAESTHESIA

Outline:

Mouth

Palate

Nose

Pharynx

Larynx

Trachea

Bronchial tree

Lungs

Epithelial lining of respiratory tract

MOUTH

The vestibule of the mouth is formed by the gums and teeth inside and the lips and the cheeks outside. The parotid duct opens opposite the second upper molar tooth. The mouth cavity is bounded in front by the teeth and gums, above by the hard and soft palates and below by the tongue and the mucosa between the tongue and mandible. The floor of the mouth between the tongue and mandible bears the frenulum linguae, on either side of which are found the openings of the submandibular salivary glands.

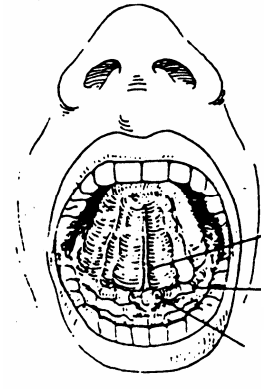


Fig. 2.1 Mouth

PALATE

The palate forms the roof of the mouth. The mucous membrane of the hard palate is closely attached to the periosteum of the bone. The soft palate is attached to the posterior edge of the hard palate. The uvula is in the free edge of the soft palate centrally, while the lateral ends of the soft palate are continuous with the side walls of the pharynx. The front of the soft palate faces the mouth cavity. The posterior surface is part of the nasopharynx.

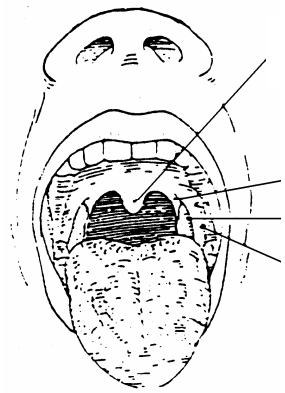


Fig 2.2 Palate

NOSE

The nose has five important functions.

- **Breathing**
  Breathing may be interfered with if there is obstruction in the nasal cavity, e.g. polyps or catarrhal inflammation of the mucous membrane. Deflection of the nasal septum can also interfere with breathing. These obstructions can also interfere with the passage of an endotracheal tube through the nose.
- **Defence and filtration**
  - Particles are filtered by the stiff hairs in the nose.
  - Organisms are filtered by the ciliated mucous membrane of the nose. The secretions of the mucous membrane are bactericidal.
- **Warming and humidifying inspired air**
  Temperature is kept constant by the great vascularity of the mucosa.
  Air is humidified by:
  - Transudation of fluid from the mucous membrane.
  - Active secretion from the glands and goblet cells. The daily volume of secretions is 1000 ml of which three quarters is used in saturating inspired air.

  An endotracheal or tracheostomy tube which bypassed the upper respiratory tract would result in cold dry gases reaching the trachea. If this continued for any length of time the cold dry gases would interfere with ciliary activity and cause tracheitis and other respiratory complications Hence patients who are intubated long term, or have a tracheostomy, must have inspired gases humidified.
- **Voice production or speech**
- **Olfactory function** or sense of smell

Nasal mucous membrane

- ***Stratified squamous epithelium.*** This lines the vestibule of the nose from where the stiff hairs proceed.
- ***Ciliated columnar epithelium*** *(see later).* This lines the nasal cavities and the sinuses and plays a part in warming and humidifying gases.
- ***Olfactory cells.*** These are found in the uppermost part of the septum and the upper lateral walls of the nose. The epithelium is concerned with the sense of smell.

PHARYNX

The pharynx extends from the posterior aspect of the nose at the base of the skull to the origin of the oesophagus at the level of the 6th cervical vertebra. The soft palate divides the pharynx into a nasopharyngeal and an oropharyngeal part. In the nasopharyngeal portion is the nasopharyngeal tonsil (adenoids).

The adenoids are important to the anaesthetist for several reasons.

- They may swell and cause upper respiratory tract obstruction.
- Nasotracheal intubation may be difficult if adenoids are present or large.
- Infection may result in a nasopharyngeal abscess. The passage of a nasal or endotracheal tube could rupture such an abscess. This is hazardous, especially in an unconscious patient.

Pharyngeal obstruction

This is a common problem under anaesthesia and in the recovery period. The commonest cause is the tongue falling back in the unconscious patient. The following measures should be taken to overcome the problem.

- Lift the lower jaw so that the lower incisors are in front of the upper jaw.
- Hyper-extend the head.
- Insert a Guedel airway.
- Insert a naso-pharyngeal airway.
- Place the patient in the lateral position if necessary.
- If the airway obstruction still persists intubation or insertion of a laryngeal mask (if available) may be necessary.

LARYNX

The larynx lies opposite the 4th, 5th and 6th cervical vertebrae. It is composed of a number of cartilages which are joined together by ligaments. The movement of the cartilages is controlled by muscles. The cartilages composing the larynx are the *thyroid*, *cricoid*, *paired arytenoid* and the *epiglottis*. The laryngeal inlet is bounded anteriorly by the epiglottis, laterally by the aryepiglottic folds and posteriorly by the mucous membrane between the arytenoid cartilages.

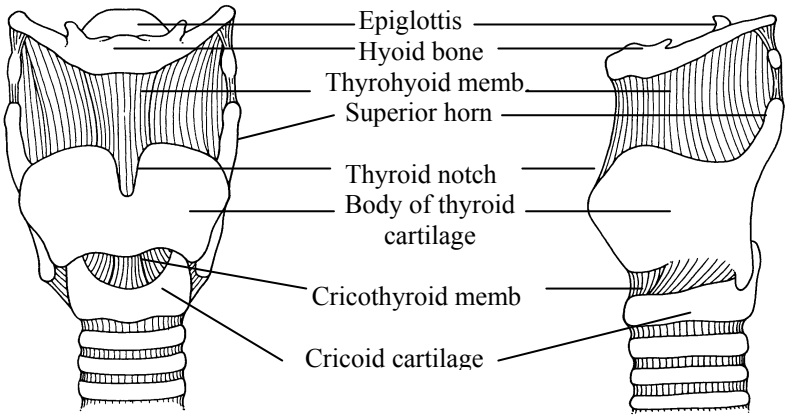


Fig 2.3 Trachea AP and Lateral

Vocal cords

These are two pearly white structures stretching from the angle of the thyroid cartilage in front to the vocal processes of the arytenoids behind. They are pale in colour because they do not have the usual submucous tissue with blood vessels. In adults the narrowest portion of the larynx is the opening between the vocal cords. In children below the age of 10 years the narrowest portion is at the level of the cricoid cartilage.

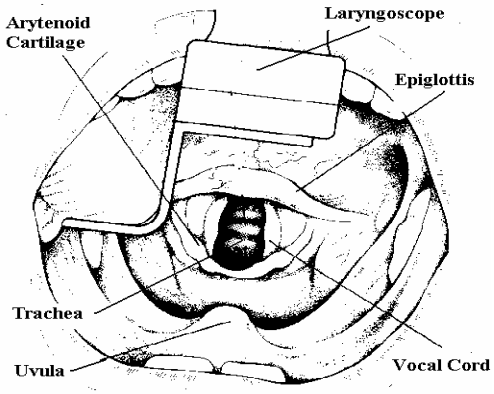


Fig 2.4
View of vocal cords at laryngoscopy

Vocal cord movements are controlled by 3 groups of muscles. The nerve supply of the larynx is from the superior laryngeal and the recurrent laryngeal nerves in the neck. These are both branches of the vagus nerve.

During inspiration the laryngeal inlet is opened. The cords are abducted.
During expiration the cords are adducted and the opening is closed.

Points to remember:

- To avoid injury to the cords extubate the patient during inspiration (cords abducted).
- Topical analgesia (local anaesthetic) applied to the larynx may block the sensory branches of the superior and recurrent laryngeal nerves. This may result in a gruff voice and a change in the shape of the vocal cords.
- The superior laryngeal or the recurrent laryngeal nerves may be injured during thyroid surgery. This injury may produce varying degrees of respiratory obstruction.

The vestibular folds. These are also called the false vocal cords. They lie above the true vocal cords.

TRACHEA

The trachea is a tube formed of 16-20 rings of cartilage. These are C-shaped rings, incomplete posteriorly. The trachea is 10-11cm long and extends from the 6th cervical to the 5th thoracic vertebra. It is lined with ciliated columnar epithelium. Red rubber endotracheal tubes are irritant to the tracheal mucosa and should not be left in place in patients who require prolonged intubation. Portex tubes and silver tracheostomy tubes are less irritant. As mentioned previously, humidification of gases reduces the incidence of tracheal irritation.

BRONCHI

The trachea divides into two bronchi, right and left. The right bronchus is wider, shorter and more vertical than the left.

This has implications for the anaesthetist.

- An endotracheal tube or a tracheostomy tube which is too long will pass from the trachea into the right main bronchus.
- Inhaled material e.g. vomitus has a slightly greater tendency to gravitate into the right side of the chest.
- It is more difficult to "block" the right main bronchus (using a bronchial blocker) because it is shorter than the left.

Divisions of the Main Bronchi

Right bronchus	Left main bronchus
Upper lobe bronchus divides into • Apical • Anterior • Posterior **Middle lobe bronchus divides into** • Medial • Lateral **Lower lobe bronchus divides into** • Apical • Medial • Lateral • Anterior • Posterior	**Upper lobe bronchus divides into** ***Superior Division*** • Apical • Anterior • Posterior ***Inferior Division*** • Lingula divides into – Inferior – Superior The **lower lobe bronchus** is the same as that on the right side except that there is no medial basal branch

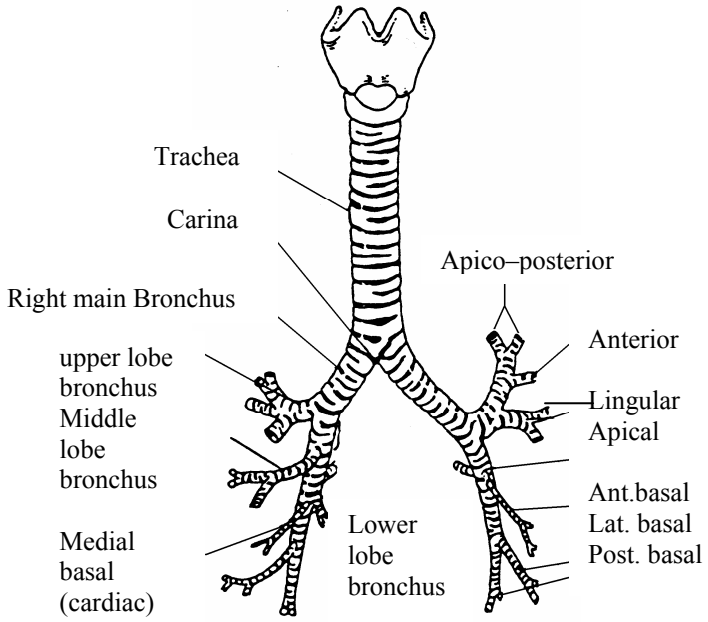


Fig 2.5 Trachea and main bronchi (viewed from the front)

Importance of the broncho-pulmonary segments to the anaesthetist.

We can see that each segment of lung has its own bronchus and its own blood supply from the pulmonary artery.

- A knowledge of bronchopulmonary segments is useful in locating the likely sites of lung abscesses.
- Lung resections are based on a knowledge of broncho-pulmonary segments.
- Postural drainage techniques depend on the bronchoscopic anatomy.
- Radio diagnostic techniques utilise this knowledge.

Lower respiratory tract

The main bronchi divide into branches, one to each lobe. These branches divide further into named branches, one to each bronchopulmonary segment. The bronchi are similar in structure to the trachea except that the cartilage is less complete.

Bronchioles: These are the finest bronchi. They have no cartilage. They contain vascular, fibrous and elastic tissue.

Alveoli: The alveoli are thin walled ducts and sacs and are the smallest and most peripheral parts of the airway. They are surrounded by a network of capillaries. Between the alveolar space and the capillary lumen is the alveolar/capillary membrane. The exchange of gases between the air in the alveoli and the blood in the capillaries takes place across this membrane.

LUNGS

The lungs are roughly conical in shape, and can be divided into apex, base, medial surface and lateral surface. The lungs lie free in the pleural cavity attached to the hilum. The right lung is larger, weighing 620 gm. The left lung weighs 570 gm. The apex of each lung reaches the root of the neck (2.5 cm above the midpoint of the clavicle). The base of each lung rests on the diaphragm. The right lung has three lobes divided by an oblique fissure and a transverse fissure. The left lung has two lobes.

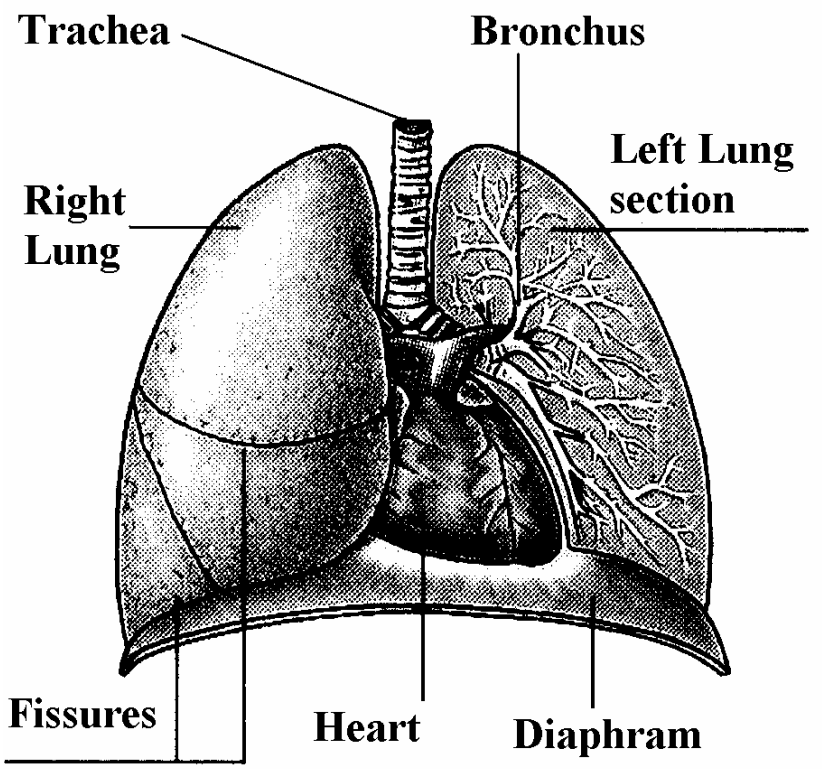


Fig. 2.6 Anatomy of the chest

EPITHELIAL LINING OF THE RESPIRATORY TRACT

Ciliated columnar epithelium lines the respiratory tract from the nose to the terminal bronchioles, becoming cuboidal then flat as it becomes the alveolar epithelium allowing gas exchange.

A cilium is a hair-like structure 0.7 mm long and 0.3 mm thick, with a mucous lining covering it. The tip of the hair is bent toward the direction of flow. At 30°C the cilium moves 10-15 times a minute.

Ciliary activity in the respiratory tract. The cilia have a very important function. They move continually, preventing accumulation of secretions. In the nose the cilia move towards the pharynx. In the upper respiratory tract the movement is upwards to the larynx.

Importance to the Anaesthetist

General anaesthesia can interfere with ciliary activity in several ways:

- The drugs themselves, both premedication (e.g. atropine) and the anaesthetic agents.
- Cold, dry gases administered via an anaesthetic machine.
- Anaesthetic techniques. Endotracheal intubation means the upper respiratory tract is by-passed, so the gases are neither warmed nor humidified.

Interference with ciliary activity causes accumulation of secretions, tracheitis, inflammation and pulmonary alveolar collapse.
Patients who undergo prolonged intubation or tracheostomy should have the inspired gases humidified.

CHAPTER 3

RESPIRATORY PHYSIOLOGY

Outline:

The mechanics of breathing

The respiratory centre

Arterial and venous blood gases

Definitions

Ventilation and perfusion

Dead space

Oxygen

Hypoxia

Carbon Dioxide

THE MECHANICS OF BREATHING

The lungs are contained in the thoracic cage, which is formed of ribs and muscles. The cage is triangular, its base formed by the diaphragm. The side walls are formed by the ribs and the intercostal muscles. The apex of the thoracic cage is in the neck.

Within the thoracic cage and surrounding the lungs are two layers of pleura. The outer layer (visceral) of the pleura is in contact with the chest wall; the inner layer (parietal) is in contact with the lungs. The space between the two layers is called the intrapleural space. The pressure in this space is negative (i.e. below atmospheric), while the pressure in the lungs is higher, close to atmospheric pressure. Since the pressure within the lungs is higher than the pressure outside, the lungs are kept expanded.

The pressure changes in the chest during respiration

The intra-pleural pressure is negative.		
During inspiration	-6 mmHg	(-0.8 kPa)
During expiration	-2.5 mmHg	(-0.33kPa)
The intra-pulmonary pressure:		
During inspiration	-1 mmHg	(-0.13kPa)
During expiration	+1 mmHg	(0.13kPa)

The chest cavity enlarges during inspiration because the muscles contract. Inspiration is therefore an active process. The enlargement of the chest is in three directions:

- Antero-posteriorly, i.e. from behind forwards. This is produced by the intercostal muscles drawing the ribs and the sternum forwards.
- Laterally, or from side to side. This is produced by the intercostal muscles drawing the lower ribs upwards and outwards.
- Vertically, or from above downwards. This is produced by the contraction of the diaphragm. The diaphragm accounts for 75% of the tidal volume. The distance the diaphragm moves varies from 1.5 cm to 7 cm during deep inspiration.

When the chest cavity enlarges, the pressure in the intrapleural space becomes more negative. This expands the lungs and the gases are drawn into the lungs from the atmosphere. For air to enter the lungs at inspiration the pressure gradient must be great enough to overcome the elastic resistance to expansion of the lung and chest wall (total compliance).

Expiration is passive, i.e. it is not produced by the active contraction of any muscles but by the relaxation of the muscles that were contracting during inspiration. These muscles relax or fall back into their resting position during expiration. The elastic recoil of the lungs brings them back also to the resting position. The chest cavity gets smaller, the lungs become smaller in size and the gases are pushed out of the lungs.

THE RESPIRATORY CENTRE

This term has been applied to a group of cells in the brain responsible for respiration. The cells are divided into two groups, dorsal and ventral. They are situated in the medulla oblongata. These cells discharge rhythmically, which means that they send out impulses or messages to the respiratory muscles at a special rate which is their own. These impulses are sent out automatically. However, this rate of discharge is influenced by many stimuli in such a way that the respiratory pattern meets the needs of the body.

Factors affecting the respiratory centre
The control of the respiratory centre has both direct and reflex components:

Direct control

- **Oxygen (O_2) lack**
  This can directly depress the respiratory centre which may seem surprising. A lack of oxygen *stimulates* the respiratory centre reflexly via the chemo- receptors (described below). However oxygen lack directly *depresses* the respiratory centre. The overall action of oxygen lack on the respiratory centre is stimulation. Respiration is increased and the oxygen lack is minimised.
- **Carbon dioxide (CO_2) excess or lack**
  An excess of CO_2 stimulates the respiratory centre directly. The respiratory centre is most sensitive to a rise in CO_2. The respiratory centre is stimulated not only directly but also through the chemoreceptors. A fall in CO_2 can depress the respiratory centre. This is sometimes seen following a general anaesthetic in which the patient has been over ventilated. The respiratory centre is depressed and breathing is slow to return.
- **Changes in the acid base balance of the blood**
  If the acid content of the blood is increased, then the respiratory centre is stimulated. (See notes on acid base balance, Chapter 59).

Reflex control

- **The chemoreceptors**
  These are a special group of cells in the neck. They are sensitive to changes in the chemical composition of the blood.
  The chemoreceptors are:
  - The carotid bodies found at the bifurcation of the common carotid artery.
  - The aortic bodies near the aortic arch.
  - Other chemoreceptors: special cells in the brain stem, pulmonary and coronary circulation.

  Afferents from the carotid body are carried to the medulla oblongata via the glossopharyngeal nerve (9th cranial nerve). Afferents from the aortic bodies travel via the vagus nerve (10th cranial nerve).
  The following conditions stimulate the chemoreceptors and cause impulses to travel up these nerves to the respiratory centre.
  - Oxygen lack.
  - Carbon dioxide excess.
  - An increase in the acidity of blood. This is seen in metabolic acidosis associated with diabetic coma.
- **The pressor receptors**
  These are special cells in the neck and the chest which are sensitive to changes in blood pressure. When these receptors are stimulated by a rise in blood pressure one effect is that respiration is depressed. (The main response, however, is on the cardiovascular system. See Chapter 4 Cardiovascular physiology).
- **Sensory stimuli**
  The respiratory centre is stimulated by pain, movement of joints etc.
- **Pulmonary stretch receptors**
  - The Hering Breuer reflex. During inspiration the alveoli in the lungs get distended. At a certain level of distension the alveoli send impulses to the respiratory centre. This causes inspiration to stop and expiration to start. This reflex is active at large tidal volumes, e.g.1 litre.
  - Irritant receptors in the lungs send impulses to the respiratory centre. These receptors are stimulated by irritant vapours.
  - J receptors are activated when the lung is deflated. Impulses are sent to the respiratory centre and inflation commences.

- **The higher centres**
  The higher centres have an effect on respiration. The pons contains an apneustic centre and a pneumotaxic centre both of which function with the medullary centre to produce respiration. The cerebral cortex and the hypothalamus influence the respiratory centre.

ARTERIAL AND VENOUS BLOOD GASES

The function of the lungs is to maintain a normal tension or pressure of gases in the arterial blood. This is achieved by ventilation.
In this process of ventilation, fresh oxygen is brought into the lung and excess carbon dioxide removed.

Exchange of oxygen

The venous blood from the tissues reaches the lungs. The tension or pressure of oxygen in the venous blood (40 mmHg /5.3 kPa) is less than the pressure of oxygen in the alveoli. Gases move from an area of high pressure to an area of low pressure, so the oxygen diffuses out from the alveoli to the venous blood oxygenating it. The blood thus becomes arterial.

Exchange of carbon dioxide

In addition to the exchange of oxygen, an exchange of CO_2 takes place.
The venous blood arriving in the lung is rich in CO_2. The pressure of CO_2 in the venous blood is 46 mmHg (6.1 kPa). The alveolar pressure is 40 mmHg. The CO_2 diffuses from the venous blood to the alveoli.

The arterial blood leaves the lung with an oxygen tension of 100 mmHg (13.3 kPa) and a CO_2 tension of 40 mmHg (5.3 kPa).

To summarise:

Arterial blood

Oxygen tension or pressure (Pa O_2) 100 mmHg. (13.3kPa)
Carbon dioxide tension (Pa CO_2) 40 mmHg. (5.3 kPa)

Venous blood

Oxygen tension or pressure 40 mmHg. (5.3 kPa)
Carbon dioxide tension 46 mmHg. (6.1 kPa)

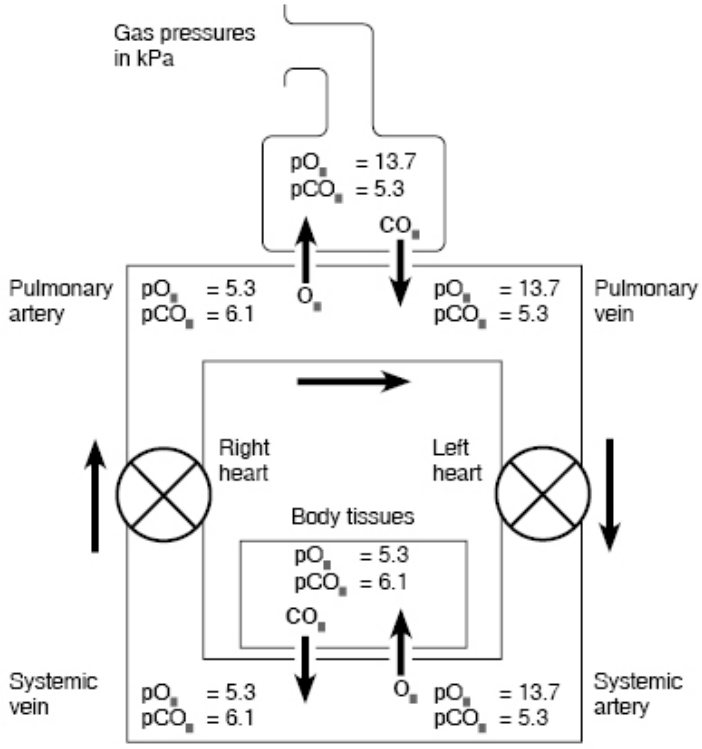


Fig 3.1 Tension of gases (kPa) throughout the body

DEFINITIONS OF LUNG VOLUMES

The tidal volume is the volume of gas passing into or out of the lungs per breath (10ml/kg or approximately 500 ml in the average adult).

Minute volume is the volume of gas passing in or out of the lungs per minute. It is equal to the tidal volume multiplied by the number of breaths per minute.

Minute volume = Tidal volume × Respiratory rate
= 500 ml × 10
= 5 litres (in the average adult)

Dead space is the part of the tidal volume that does not take part in ventilation (2ml/kg). It could be considered wasted ventilation in that it serves no useful purpose where gaseous exchange is concerned.

Alveolar ventilation is that part of the tidal volume that actually takes part in gaseous exchange.

Tidal volume = dead space + alveolar ventilation
500 ml = 150 ml + 350 ml (in 75kg adult).

Vital capacity is the greatest amount of air that can be expired after a person has breathed in as much as possible.

Functional residual capacity (FRC) is the volume of air in the lungs at the end of a normal expiration and is the sum of the ERV and the RV.
It is important to the anaesthetist because it contains a reservoir of oxygen (e.g. after pre-oxygenation and full nitrogen wash-out) which can maintain blood levels of oxygen during apnoea.
It is decreased by up to 25% in the supine position and also reduced in obesity, pregnancy and under anaesthesia.

Expiratory reserve volume (ERV) is the amount of air that can be forcefully expired at the end of a normal expiration.

Residual volume (RV) is the amount of air remaining in the lungs at the end of a maximum forced expiration.

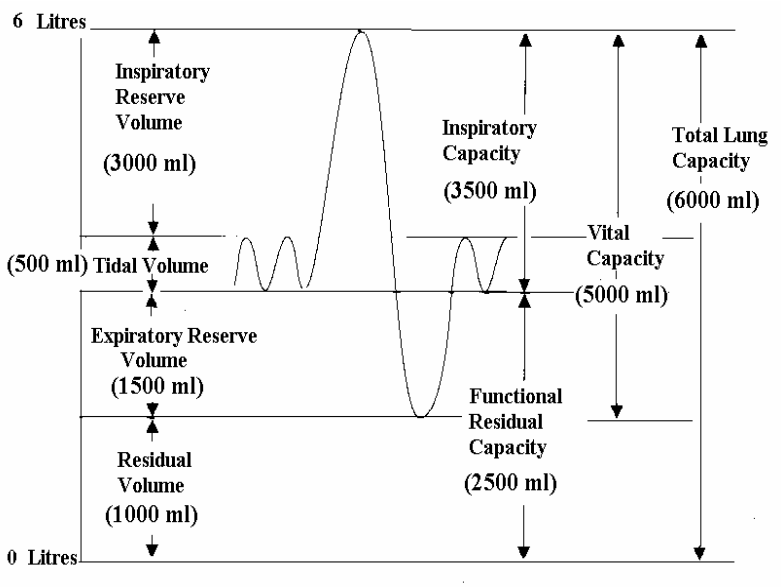


Fig 3.2 Lung volumes (av. male) measured with a spirometer

Diffusion is the passage of fluid or gas from an area of high pressure across a membrane. The gases in the alveoli pass into the blood in the capillaries by this process of diffusion. The gases have to pass through three layers:

- The alveolar lining or epithelium
- Interstitial fluid
- The capillary lining or endothelium

If any of these layers is thickened then diffusion is impeded.
The alveolar lining may be thickened by disease of the lung.
The interstitial fluid may be increased in heart disease.

Compliance is a measure of distensibility. Lung compliance is defined as the change in volume per unit change in pressure.

Airway resistance denotes the measure of obstruction to the flow of gas in the air passages. Flow of gas in a tube depends on a number of factors. Anything that reduces the size of the air passage will interfere with flow and therefore increase the airway resistance. Most of the resistance to air flow is in bronchi greater than 2 mm in diameter. Airway resistance is increased in asthma, emphysema, pulmonary congestion etc. In these conditions the work of breathing is increased.

VENTILATION AND PERFUSION

Ventilation (V)
Ventilation of the lung is not uniform. The lower part ventilates better than the upper. (The lower part of the lung, due to its weight and the effect of gravity tends to be less distended in the resting position than the upper part). Therefore during inspiration the lower part of the lung has a greater capacity to fill or distend.

Perfusion (Q)
The pulmonary blood flow equals the cardiac output. The pulmonary artery pressure is usually about 20% of the systolic blood pressure. The pulmonary circulation is affected by gravity. As with ventilation, perfusion is greater at the base than at the apex and so the lower zone has a better blood flow than the middle or upper zone. However, the overall result is that ventilation is relatively greater than blood flow at the apex and relatively less than blood flow at the base.

The V/Q ratio
This expresses the relationship between the regional blood flow and the regional ventilation in the lung. Ventilation/Perfusion ratios are not uniform throughout the lung but in the healthy person ventilation and perfusion are reasonably well matched with an overall ratio of 0.9. The V/Q ratio is higher at the apex and lower at the base.

In those areas of the lung where the V/Q ratio is high, the capillary blood gases returning from the lung approach those in alveolar air (high O_2 low CO_2). In those areas where the V/Q ratio is low the capillary blood gas tensions approach those of venous blood (low O_2 high CO_2). When pulmonary blood flow is high but ventilation is poor it is referred to as pulmonary shunting.

Regional ventilation perfusion ratios can be altered in pulmonary disease. In pulmonary embolism there is under perfusion. In certain lung disease there may be under-ventilation, e.g. pneumonia. These conditions affect gas exchange.

DEAD SPACE

The dead space has been defined as that part of the tidal volume that does not take part in gas exchange.
Three types of dead space will be considered:

Anatomical dead space
This is the volume of gas that fills the respiratory passages but which is not involved in gas exchange. It extends from the nostrils and mouth down to, but not including, the alveoli. In the average adult this is about 150 ml (2ml/kg body weight). The anatomical dead space is reduced by tracheostomy and endotracheal intubation.

Physiological dead space
This includes all the air (that for various reasons) does not take part in gas exchange. It includes the anatomical dead space **plus** air in any of the alveoli into which the blood does not flow and any air in over distended alveoli where gaseous exchange is impaired. Physiological dead space is increased in lung disease.

Anaesthetic dead space
Anaesthetic dead space is that part of the tidal volume that occupies the anaesthetic apparatus and is hence not available for gas exchange. The exact volume of anaesthetic dead space depends on the circuit used. Consider the "T piece" used for children. The anaesthetic dead space is the volume between the patient and the inflow of fresh gases into the "T piece". If the circle absorber is used, as for instance in the Heidbrink or Boyle's machine, then the anaesthetic dead space is the volume between the patient and the junction of the inspiratory and expiratory tubing.

If a face mask is used, the dead space is increased considerably because part of the patient's tidal volume occupies the space under the mask and is hence wasted. The use of an endotracheal tube or a tracheostomy tube reduces the dead space.

OXYGEN

Exchange between the lungs and the atmosphere

The amount of oxygen in inspired atmospheric air is 21%. The pressure or tension of this oxygen is 158 mmHg (21 kPa). The inspired air is drawn into the alveoli where it mixes with the air left from the previous respiration. Therefore, the oxygen is diluted, the CO_2 raised and water vapour is added. The pressure of O_2 in the alveolar air becomes 103 mmHg (13.7 kPa).

Exchange between the blood and the lungs

The venous blood arrives in the lung (from the tissues) with an O_2 pressure of 40 mmHg. Because of the difference in pressure, O_2 passes from the alveoli into the blood stream. This transfer occurs by the process of diffusion. The O_2 in solution determines the oxygen tension. It moves from the alveoli where the tension is high to the capillary blood where the tension is low. The capillary blood leaves the lungs after picking up O_2 and giving up CO_2. It is high in O_2 (both dissolved O_2 and O_2 in combination with haemoglobin).

The arterial blood leaves with an oxygen tension of 100 mmHg.

To summarise:

Inspired oxygen (atmospheric air)	Pressure Concentration	158 mmHg. (21 kPa) 21%
Alveolar air (diluted with gases already in the lung)	Pressure	103 mmHg. (13.7 kPa)
Arterial blood		100 mmHg. (13.3 kPa)
Venous blood		40 mmHg. (5.3 kPa)

Carriage of oxygen in the blood

Important:

- O_2 is carried in solution, which means it is dissolved in the water in plasma. This is only a very small quantity, 0.3 ml per 100ml of blood (when breathing air at sea level). It is important because this O_2 is responsible for the pressure or the tension of the O_2 in the blood.

- O_2 is also carried in combination with the haemoglobin in the red blood cells. 1 gm of haemoglobin can carry 1.34 ml of O_2. The normal adult with 14.8 g of Hb/100 ml of blood will therefore carry 19 ml of O_2 per 100 ml of blood. The Hb is almost fully saturated with this 19 ml O_2/100 ml blood.
  This O_2 is in the red blood cells. Each 100 ml of blood gives off 5 ml of O_2 as it passes through the tissues and returns to the lungs as venous blood carrying 14 ml of O_2 per 100 ml of blood. When the Hb combines with O_2 it is termed ***oxyhaemoglobin*** (oxyHb)**.** When it gives up the O_2 in the tissues it is termed ***reduced haemoglobin***.

The passage of oxygen from the blood to the tissues

In the tissues the capillary blood is exposed to a low O_2 tension. The oxygen diffuses out of the blood into the tissues. Initially the dissolved O_2 in the plasma diffuses out first. When this happens the O_2 in combination with the haemoglobin is no longer in equilibrium with the dissolved O_2. The O_2 passes from the red blood cells (haemoglobin) into the plasma and thence into the tissues. The O_2 is thus unloaded into the tissues.

There is a relationship between the O_2 tension in the plasma and the O_2 in combination with the haemoglobin. If the O_2 tension is low the O_2 content of the haemoglobin will be low. If the O_2 tension is high the O_2 content of the haemoglobin will be high. The graph below shows the relationship. It is referred to as the oxyhaemoglobin curve.

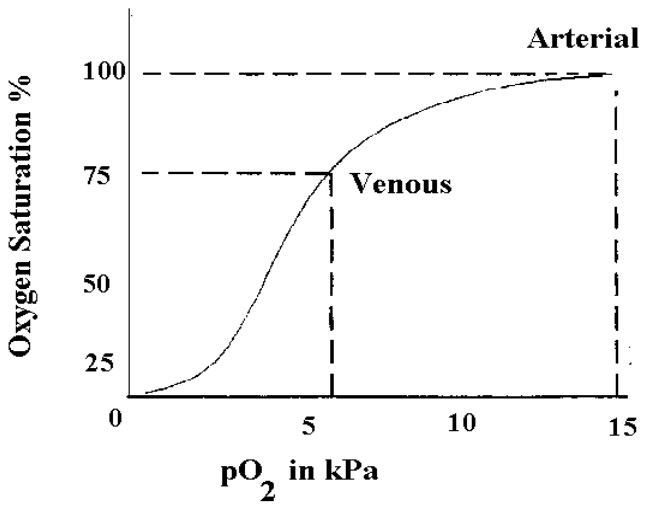


Fig 3.3 The oxyhaemoglobin dissociation curve showing the normal arterial and venous points

Po_2 mmHg	(kPa)	% sat of Hb	Dissolved O_2 (ml/100ml)
10	1.33	13.5	0.03
20	2.66	35	0.06
30	4	57	0.09
40	5.3	75	0.12
50	6.65	83.5	0.15
60	8	89	0.18
70	9.21	92.7	0.21
80	10.6	94.5	0.24
90	12	96.5	0.27
100	13.3	97.5	0.30

Fig 3.4 Relationship between Pressure and Saturation

The curve is flat between the pressures of 80 and 100 mmHg. This is the normal level of oxygen in the blood. If the oxyhaemoglobin is exposed to an O_2 tension below 40 mmHg there is a sudden fall in the O_2 content. This happens at tissue level. However at the upper end of the curve if the O_2 tension falls (less dissolved O_2) the haemoglobin content of O_2 is still maintained fairly constant.

The following situations shift the oxyhaemoglobin dissociation curve to the right and make it release oxygen more readily.

- An increase in the CO_2 tension in the blood.
- An increase in the H^+ ion concentration.
- An increase in temperature.
- An increase in 2,3 DPG (Diphosphoglycerate).

2,3 DPG is found in red blood cells. It is increased in chronic anaemia, hypoxia and thyrotoxicosis, and results in more O_2 being delivered to the tissues. It is decreased in polycythaemia, myxoedema and stored blood and inhibits the release of O_2 to the tissues.

HYPOXIA

Hypoxia means a lack of O_2 in the body (tissues). Hypoxaemia is a lack of O_2 in the blood. This may reveal itself as central cyanosis which is a blueness of the tissues of the lips, tongue and mucous membranes of the mouth. It is difficult to estimate the degree of hypoxia from the observation of cyanosis and by the time this is obvious oxygen levels will have fallen significantly. Oxygen saturation below 90% is a reasonable definition. Hypoxia must be diagnosed quickly and treated aggressively otherwise permanent neurological damage and cardiac arrest may quickly follow.

There are various types of hypoxia:

- Hypoxic hypoxia
- Anaemic hypoxia
- Stagnant hypoxia
- Histotoxic hypoxia
- Diffusion hypoxia

Hypoxic hypoxia

This occurs if the O_2 pressure in the blood is reduced for any reason. There is less O_2 carried in the blood, both in solution in the plasma and also in combination with the Hb. The quantity of Hb is normal.

Causes of hypoxic hypoxia

- ***Not enough O_2 in the inspired gases.*** This may occur under anaesthesia when the flow rate of gases used is incorrect, the cylinder is empty and escapes attention or the oesophagus is intubated and not immediately remedied (all inexcusable). It may also occur at a high altitude where inspired O_2 is at a reduced pressure.
- ***Reduced ventilation from any cause***. It may be associated with conditions at various locations.

Location	Condition
Respiratory centre	Drugs Trauma Infections Stroke
Spinal cord	Trauma
Peripheral nerves	Polyneuritis
Neuromuscular junction	Muscle relaxants Myasthenia Gravis
Muscles	Myopathies – especially those affecting intercostal muscles and diaphragm
Chest wall	Fractured ribs Flail chest
Pleural cavity	Pleural effusion Pneumothorax
Lung tissue	Collapse Pneumonia
Airway obstruction	Upper –oral –laryngeal Lower –bronchospasm

- ***Interference with diffusion*** between the alveoli and the blood as in lung disease.
- ***Circulatory disorders,*** e.g. in certain congenital heart disease where all the blood does not pass into the lungs.

Anaemic hypoxia

Here there is a reduction in the amount of circulating Hb. If the Hb is reduced then the oxygen carried in the blood is reduced.

Stagnant hypoxia

This occurs when the circulation is inadequate for any reason. The blood spends a longer time in the tissues and therefore loses more O_2 and the venous blood returning to the lungs also has less O_2. This is met with in 'shock' where the blood pressure is very low and the circulation sluggish. It also happens in heart failure.

Histotoxic hypoxia

This occurs when the tissues are poisoned with drugs or chemicals, e.g. in cyanide poisoning. It is also seen if the tissues are oedematous. In these situations the tissues cannot accept or use the O_2 brought to them by the blood.

Diffusion hypoxia.

This occurs at the end of an anaesthetic where N_2O/O_2 is used. The nitrous oxide passes out of the blood stream and into the alveoli faster than the nitrogen in air passes in and this may dilute the concentration of inspired O_2. If the patient is breathing atmospheric air then this falls to below 20%. This is overcome by giving the patient 100% O_2 at the end of the anaesthetic.

Effects of oxygen lack

The central nervous system: This is most sensitive to O_2 lack. The blood flow to the brain is first increased. This is followed by oedema or swelling of the brain and unconsciousness. Irreversible brain damage will occur within 4 minutes if severe hypoxia is not treated. The anaesthetist's main responsibility is to prevent this.

The respiratory system: There is an increase in respiration in conscious patients. This is a reflex stimulation of the respiratory centre via the chemoreceptors.

The cardiovascular system: The pulse rate and the blood pressure rise at first and then fall as the hypoxia increases.

Other tissues: The liver cells are damaged.

If a patient becomes hypoxic under anaesthesia, then:

- Increase the inspired oxygen concentration to 100%. Check oxygen analyser, if available, or oxygen cylinder levels, to exclude a failed oxygen supply.
- Check that ventilation is adequate:
  - Check end-tidal CO_2, if available. Note that oesophageal intubation must be excluded early as this needs to be corrected promptly.
  - Switch to hand ventilation to assess pulmonary compliance and auscultate both sides of the lung to exclude endobronchial intubation.
- Check blood pressure and pulse.
- Correct the cause:
  - Deficiency in inspired O_2.
  - Respiratory obstruction.
    i.e. laryngospasm (See Chapter 62 p 696 for management of laryngospasm)
    or inadequately cleared airway (try Guedel airway, nasal airway or laryngeal mask airway).
  - Inadequate ventilation (check capnograph). If difficulty in ventilation always consider a blocked endotracheal tube
    i.e. sputum plug or foreign body as well as the more common causes. Blocked endotracheal tube is excluded by the free passage of a suction catheter down the tube.
  - Bronchospasm.
  - Aspiration.
  - Pneumothorax.
  - Pulmonary Embolus.

CARBON DIOXIDE

Levels of carbon dioxide (CO_2)

Atmospheric air contains about 0.04% of CO_2. Expired air contains 4.5% of CO_2. The tension or pressure of CO_2 in arterial blood is 40 mmHg (5.3 kPa). The tension or pressure of CO_2 in venous blood is 46 mmHg (6.1 kPa).

Transport of CO_2 in the blood

The tissues produce CO_2 which is taken up by the blood. The blood arrives in the lungs with a CO_2 pressure of 46 mmHg. (This is the venous blood returning from the tissues). The pressure of CO_2 in the alveoli of the lungs is 40mmHg. There is a difference of 6 mmHg, sufficient to enable the CO_2 to diffuse from the venous blood into the alveoli of the lung, from where it is carried to the exterior with the expired gases.

The carbon dioxide is carried in the blood in three forms:

- In simple solution
- In combination with the Hb
- As bicarbonate

The greatest part of the CO_2 is carried as bicarbonate.

Effects of carbon dioxide excess

Central nervous system: There is an increase in the blood flow to the brain with a corresponding increase in cerebral volume and raised intracranial pressure.

The sequence of events is:

- Headache
- Nausea
- Vomiting
- Coma

Respiratory system: This is very sensitive to a rise in the CO_2 tension. The respiratory centre is stimulated both directly and reflexly via the chemoreceptors. If the pressure of CO_2 in the blood stream rises by 1.5 mmHg the tidal volume is doubled. Severe hypercapnia produces respiratory failure.

Bohr effect: the oxyhaemoglobin dissociation curve shifts to the right. This means more O_2 is given up at tissue level.

Cardiovascular system: Cardiac output is increased by direct effect of CO_2 and by the effect of the increase in circulating catecholamines.

- Heart rate increases and becomes irregular.
- Peripheral resistance increases.
- Blood pressure may rise at first, especially in the unanaesthetised person.

Causes of CO_2 excess (hypercapnia) in anaesthetic practice

- Respiratory obstruction causing inadequate ventilation.
- Inadequate ventilation, due to depression of the respiratory centre or the action of muscle relaxants.
- Faulty CO_2 absorption in circle absorbers.
- Accidental administration of CO_2.

Low CO_2

Excessive ventilation (hyperventilation) reduces the tension of CO_2 in the blood. The patient becomes hypocapnic (low CO_2).

Hyperventilation is best avoided during general anaesthesia. It can result in cerebral vasoconstriction and is therefore dangerous, especially in older people. It may also result in foetal asphyxia if used during a Caesarean section.

Hypocapnia may delay the onset of breathing at the end of an anaesthetic. Hypocapnia shifts the oxyhaemoglobin dissociation curve to the left and reduces the oxygen released to the tissues.

CHAPTER 4

CARDIOVASCULAR PHYSIOLOGY

Outline:

Functions of blood

Composition of blood

Anatomy of the heart

The cardiac cycle

The peripheral blood vessels

Arterial blood pressure

Regulation of blood pressure

FUNCTIONS OF BLOOD

- To take nutrients and oxygen to the cells.
- To remove carbon dioxide and other metabolites from cells.
- To transport water and electrolytes. It therefore plays a part in maintaining the blood volume, pH and electrolyte composition of the body.
- To help regulate temperature.
- To transport hormones and enzymes.
- To transport antibodies. It is therefore involved in the body's defence mechanism.

COMPOSITION OF BLOOD

The blood consists of plasma and cells. The cells are of 3 types: red blood cells, white blood cells and platelets.

Red blood cells (RBC)
These are very small cells, like a concave disc in shape, with a diameter of 7.2 microns. The red cell has no nucleus. In each one there are 200-300 million molecules of haemoglobin (Hb). Each molecule of haemoglobin consists of 1 molecule of protein (globin) and 4 molecules of pigment (haem).

Origins of RBC: At birth the RBC are formed, mainly in the marrow. Bone marrow can be yellow or red. The yellow marrow consists of fat cells, blood vessels and other supporting cells and fibres. The red marrow contains numerous blood cells both red and white and their precursors (earlier forms). With increasing age, the marrow becomes more fatty. In the adult, red marrow is present in the flat bones: sternum, vertebrae, ribs and skull. In the infant, red marrow is present in the flat bones and also in the long bones.

The red cells are produced in the red marrow from a nucleated cell called the erythroblast. The erythroblast eventually loses its nucleus and becomes the erythrocyte. Before the nucleus is completely lost, it disintegrates into fragments which are finally extruded. The name given to the cell with the nuclear fragments is a reticulocyte. The reticulocyte finally becomes the matured RBC.

Measurement of RBC count: The red cell count is 5 million/mm^3. The haematocrit (or PCV – packed cell volume) is the percentage of red blood cells in whole blood. Hence a haematocrit of 47% means that in 100ml of blood 47ml is cells and 53ml is plasma. Since the majority of cells in whole blood are red cells, the PCV can be used as an estimate of Hb in most patients. The haematocrit reading is approximately three times the Hb level. (See Chapter 32 Anaemia)
There are several types of haemoglobin. The most common are:

- Haemoglobin A (normal adult Hb)
- Haemoglobin F (foetal Hb)
- Haemoglobin S (a pathological Hb, found in sickle cell anaemia)

Destruction of the RBC: The life span of the RBC is 120 days. The RBCs, as they grow older, eventually rupture within the capillaries. Following this the reticuloendothelial cells carry out the process of phagocytosis (by which the ruptured cells are engulfed and removed by the phagocytes).

The haemoglobin breaks down into its component parts:

- Globin
- Iron (re-used)
- Bilirubin precursor

White blood cells (WBC)

The normal WBC count is 4000 - 11000/mm^3. *Leucocyte* is a general term for all white blood cells. White blood cells are of two kinds.

Granulocytes These have granules in their cytoplasm and make up about 70% of the normal WBC count. Most of them (50% - 70%) are neutrophils, which stain red-brown with Leishman dye. About 1% - 4% are eosinophils, which stain red and the remaining 0% - 1% are basophils, which stain blue.

Non-granulocytes make up about 30% of the normal WBC count. Non-granulocytes are either lymphocytes or monocytes.

If the WBC count exceeds 11000/mm^3 the condition is called leucocytosis. If the count falls below 3000/mm^3 it is called leucopenia.

Origins of white blood cells: After birth the granulocytes are produced mainly in the bone marrow, the non-granulocytes (lymphocytes and monocytes) in the lymphoid tissue.

Function of white blood cells:

- ***Neutrophils:*** Phagocytosis is the process whereby the bacterium or the foreign particle is engulfed into the cell and the enzymes in the cell then digest the bacterium. When there is a bacterial infection the neutrophil count increases. These white cells are transported by the blood to any part of the body.
- ***Lymphocytes:*** These are involved in antibody production, which helps to develop immunity.

Platelets

Platelets are small fragments of cells. They are formed in the bone marrow by fragmentation of large cells. Platelets are also known as thrombocytes and lack of them is called thrombocytopenia. The normal platelet count is 150,000 – 350,000 / mm^3.

Functions of platelets

- Haemostasis: minor injuries to a blood vessel result in the platelets clumping together to form a plug. This seals the damaged vessel and clotting follows.
- Storage: platelets store substances like histamine.

Plasma is the other component of blood.

Composition of plasma

- 90% water and crystalloids, e.g. mineral salts such as sodium, chlorine and potassium.
- 10% solutes: colloids, 1-100 millimicrons in diameter, e.g. proteins, such as albumin, globulin.

Another way of looking at the composition of plasma is as follows:

Nutrients**:** carbohydrates, amino acids
Metabolic wastes**:** urea, uric acid, creatinine, lactic acid
Respiratory gases**:** oxygen, carbon dioxide
Regulator mechanisms**:** hormones, enzymes, mineral salts
Protection substances**:** antibodies.

ANATOMY OF THE HEART

The heart has four chambers: 2 atria and 2 ventricles. The ventricles are larger than the atria and have thicker walls.

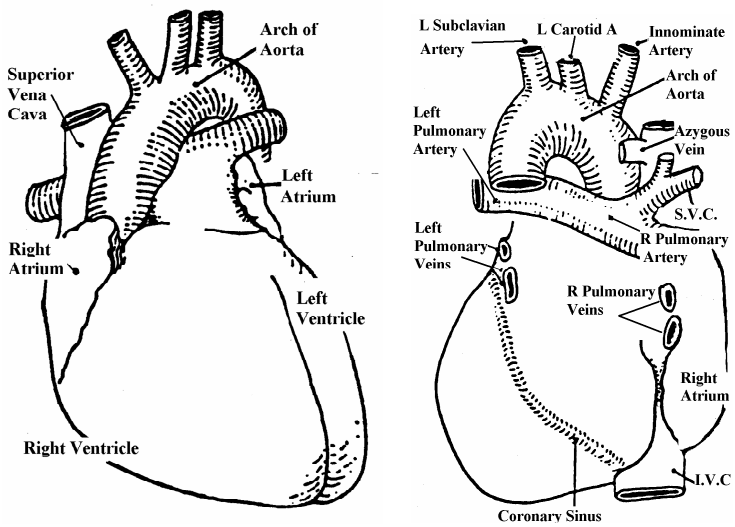


Fig 4.1 The heart: anterior view posterior view

Valves: The 2 atrio-ventricular or AV valves lie between the atria and the ventricles. The tricuspid valve lies between the right atrium and the right ventricle. The mitral valve lies between the left atrium and the left ventricle. There are 2 semilunar valves: one lies at the opening of the pulmonary artery from the right ventricle (pulmonary valve), the other at the opening of the aorta from the left ventricle (aortic valve).

Coverings of the heart: A thick fibrous layer forms a bag around the heart. This is called the pericardium.

THE CARDIAC CYCLE

This term refers to a complete heart beat, consisting of contraction (systole) and relaxation (diastole) of both atria and ventricles.

The heart makes a sound like "lubb-dupp, lubb-dupp". The first heart sound ("lubb") is caused by the closure of the AV valves as the ventricles begin to contract. The second heart sound ("dupp") is caused by vibration and closure of the aortic and pulmonary valves (semi-lunar valves).

THE PERIPHERAL BLOOD VESSELS

The blood that leaves the left ventricle enters ***the aorta and large arteries*** and then is distributed to:

The ***arterioles*** are pre-capillary resistance vessels, responsible for the peripheral resistance. The changes in their size affect the peripheral resistance and therefore the blood pressure. They also determine the blood supply to the different organs. The size of these vessels is controlled by nervous impulses (from the vasomotor centre) and also local factors acting on the blood vessel (peripheral resistance is explained on page 46).

The ***capillaries*** are tiny blood vessels lined by a single layer of endothelial cells. Solutes pass across the capillaries to and from the tissues. The arterioles become the capillaries and this change from arteriole to capillary is marked by a pre-capillary sphincter.

The capillaries themselves are not under nervous or hormonal control. The little sphincters however are controlled by nervous plus "local" factors and it is these sphincters that determine whether the capillary is available for exchange of substances, between blood and tissues. At any one time only 20-25% of the capillaries are available for exchange between the tissues and the vascular compartment.

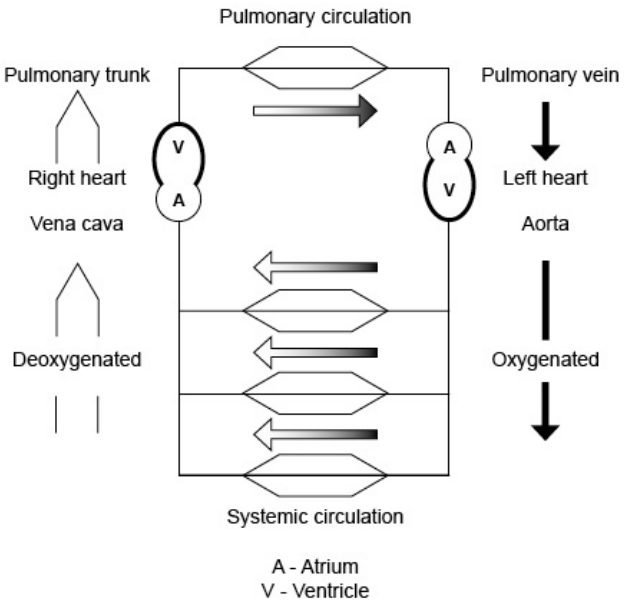


Fig 4.2 Normal blood flow

The ***venules*** comprise the "capacity" vessels. These vessels do not contribute to the peripheral resistance but they can dilate, so increasing the degree of peripheral pooling and decreasing the venous return or they can constrict increasing the venous return. They act as a reserve to compensate in shock. This dilatation and constriction is under the control of the sympathetic nervous system.

ARTERIAL BLOOD PRESSURE

This is pressure exerted on the arterial wall by the circulating blood.

The ***systolic pressure*** is the maximum pressure exerted on the arterial wall during systole. The ***diastolic pressure*** is the minimum pressure exerted on the arterial wall when the contraction of the ventricle has ceased, the ventricle is completely relaxed and the aortic valve closed.

Measurement of arterial blood pressure

- The sphygmomanometer can be used to estimate blood pressure.
  - By palpation of the radial artery. This enables the systolic blood pressure to be checked.
  - By auscultation. The pulsating brachial artery is auscultated and both the systolic pressure and diastolic blood pressure can be recorded.
- The oscillotomometer is an instrument that records the systolic and diastolic blood pressure by the oscillation of the needle. When the needle starts to swing it is the systolic blood pressure and when the needle ceases to swing it is the diastolic blood pressure.
- Automatic equipment with visual and auditory indicators is used to show both the systolic and diastolic blood pressure.
- Intra-arterial blood pressure estimation. A cannula is inserted into an artery, e.g. the radial artery. This is connected to a pressure transducer and a record of the blood pressure is produced in the form of a graph or printout.

REGULATION OF ARTERIAL BLOOD PRESSURE

Arterial blood pressure is affected by:

- The volume of blood in the circulation
- The cardiac output (which requires a normal blood volume)
  - Heart rate
  - Stroke volume
- The peripheral resistance
  - The size of the peripheral vessels
  - The viscosity of the blood

Each of these will be considered briefly:

Blood volume

Total blood volume is approximately 70ml/kg in adults, 80ml/kg in children and 90ml/kg in neonates. 70% of the total blood volume is contained within the venous system, 15% in the arterial system, 10% within the heart and 5% capillary. **Hypovolaemia** is a reduced circulating blood volume.
Hypovolaemia can result from the following:

- Loss of whole blood, as in acute haemorrhage
- Loss of plasma, as in burns
- Reduced intake, fasting (especially in the extremes of age)
- Excessive fluid loss, as for example in vomiting, diarrhoea, fistula, intestinal obstruction with accumulation of secretions in the gut
- Excessive sweating
- Excessive urinary loss

Cardiac output

The cardiac output is the volume of blood leaving the heart in one minute.
It depends on the heart rate and the stroke volume.
Both are now discussed in detail.

The heart rate is determined by the rate of spontaneous pacemaker activity at the sino-atrial (SA) node – a group of cells situated in the right atrium close to the SVC.

The heart rate and contractility can be increased or decreased by the activity of the autonomic nervous system through a number of nerve reflexes which detect and respond rapidly to changes in arterial blood pressure.

The **efferent** limb of these reflexes involves autonomic nerves regulated by centres in the medulla: The ***vasomotor centre*** and the ***cardio-inhibitory centre.***

- ***The Vasomotor centre*** is a collection of cells in the medulla, which have a sympathetic vasoconstrictor effect on the blood vessels and also an inotropic and chronotropic effect on the heart. A positive inotropic effect results in an increase in the force of contraction of the heart. A positive chronotropic effect results in an increase in the rate of the heart.

  The sympathetic nerves activated by the vasomotor centre have the following effects:
  - On heart – stimulates HR and contractility
  - On arterioles – release of nor-adrenaline leading to vasoconstriction
  - On veins – constriction leading to increase in venous return and cardiac output
  - On adrenal medulla – release of adrenaline and noradrenaline

- ***The cardioinhibitory centre.*** Immediately lateral to the vasomotor centre (in the medulla) is the dorsal nucleus of the vagus which has a cardio-inhibitory effect (i.e. on stimulation of these fibres there is a bradycardia and a decrease in the force of contraction via the vagus nerve).

The heart rate is hence controlled predominantly by a change in sympathetic or vagal tone.

The **afferent** limbs of the reflexes involve a number of cardiovascular receptors and reflexes – baroreceptors, chemoreceptors and low pressure volume receptors.

- ***Baroreceptors*** are specialised cells called "pressor receptors". They are sensitive to stretch which accompanies changes in pressure in the vessels or in the cardiac chamber where these receptors are situated. They are found in the carotid sinus and the aortic arch. The carotid sinus is the dilated part of the internal carotid artery at its commencement. An impulse passes upwards from these receptors. In the carotid sinus the nerve conveying the afferent impulses to the cardiac centres is the ninth or glosso-pharyngeal nerve. The vagus or the tenth cranial nerve carries these impulses from the aortic arch. As the pressure (and hence the stretch) in the carotid sinus and the aortic arch rises there is a decrease in the blood pressure. If the pressure in the aortic arch and carotid sinus falls there is a rise in the heart rate and an increase in the blood pressure, within the vessel or cardiac chamber where these receptors are situated. Atrial baroreceptors have been found which react in the same way to changes in pressure and help control blood volume.

- ***Chemoreceptors.*** These are of primary importance in the control of respiration. (See Chapter 3 Respiratory physiology). The chemoreceptors are situated in the carotid body at the point at which the common carotid artery divides into the external and internal carotid artery. They are also situated in the aortic arch. Their locations are close to but not identical with those of the baroreceptors. When the chemoreceptors are stimulated by a rise in arterial carbon dioxide or a fall in arterial oxygen, then, although the predominant effect is on the respiratory centre, there is also a reflex tachycardia and rise in blood pressure due to impulses conveyed via the 9th and 10th cranial nerves to the vasomotor centre.
- ***Low-pressure volume receptors.*** These are also stretch receptors found in the walls of the large veins, atria and the pulmonary trunk and are particularly sensitive to changes in the circulating blood volume. An increase in blood volume causes an increase in venous return and raises the cardiac output and arterial blood pressure. This stimulates the low-pressure volume receptors which can reduce blood pressure by two mechanisms.
  - Reduction of vasoconstrictor sympathetic activity thereby reducing peripheral resistance.
  - Inhibition of antidiuretic hormone (ADH).

  These effects are reversed following abnormal decreases in blood volume.

Other mechanisms involved in control of HR and BP

Hormones: A number of hormones are involved in the regulation of blood pressure. Some act rapidly and others have a delayed action.

- *Catecholamines* (adrenaline and noradrenaline) are released from the adrenal medulla as a result of sympathetic activity via the vasomotor centre. These hormones act within minutes causing vasoconstriction and heart rate and contractility.
- *Antidiuretic hormone* This hormone from the pituitary gland causes direct vasoconstriction but has a more important if less rapid effect by stimulating water reabsorption in the kidney and increasing blood volume.
- *Renin/angiotensin/aldosterone* This system is stimulated by a reduction in renal blood flow. Angiotensin acts as a vasoconstrictor, increasing peripheral resistance but also stimulates aldosterone secretion from the adrenal cortex. This promotes sodium and water reabsorption in the kidney increasing blood volume and blood pressure.

Emotions: Emotions, e.g. anxiety or excitement may increase the heart rate. Shock, another emotion, may decrease it. These impulses are transmitted to the vasomotor centre from the cerebral cortex and hypothalamus.

Respiration: In most adults, during quiet respiration, there is no change in the heart rate. However, in children during quiet breathing or in some adults during very deep breathing, the heart rate tends to vary. The heart rate in these cases, increases during inspiration and slows during expiration. This is termed "sinus arrhythmia". It is believed to be caused by impulses from the inspiratory (RC) centre, transmitted to the cardiac centre.

The stroke volume is the amount of blood leaving the heart with each contraction. It is affected by:

- ***Venous return:*** depending on
  - The tone in the arterioles (see peripheral resistance).
  - The capacity of the veins.
  - The thoracic pump. Pressure changes associated with respiration aid venous return. (There is an advantage in using laryngeal masks and spontaneous breathing).
  - The muscle pump. The contraction of skeletal muscles aids venous return.

  It is clear that under general anaesthesia all these are affected and the venous return falls. For this reason hypotension under anaesthesia is treated with intravenous fluids, in order to maintain venous return.

- ***Force of contraction:*** depending on
  - The balance between the sympathetic and parasympathetic return. The sympathetic nerves increase the force of contraction of the heart (inotropic effect).
  - The coronary flow. The force of contraction will be reduced if the coronary flow is inadequate and in the presence of other heart diseases.

(Starling's law states that the force of contraction of a muscle fibre is directly proportional to its resting length. One result of this is that in the normal heart cardiac output responds to venous return i.e. if there is an increase in venous return causing the ventricle to distend, the cardiac output will also increase until a steady state is again reached. It also means that output from both ventricles will be the same. This balance is disturbed in heart disease).

Peripheral resistance

This also influences the blood pressure. The peripheral resistance is the resistance to blood flow at the level of the peripheral arterioles and depends on:

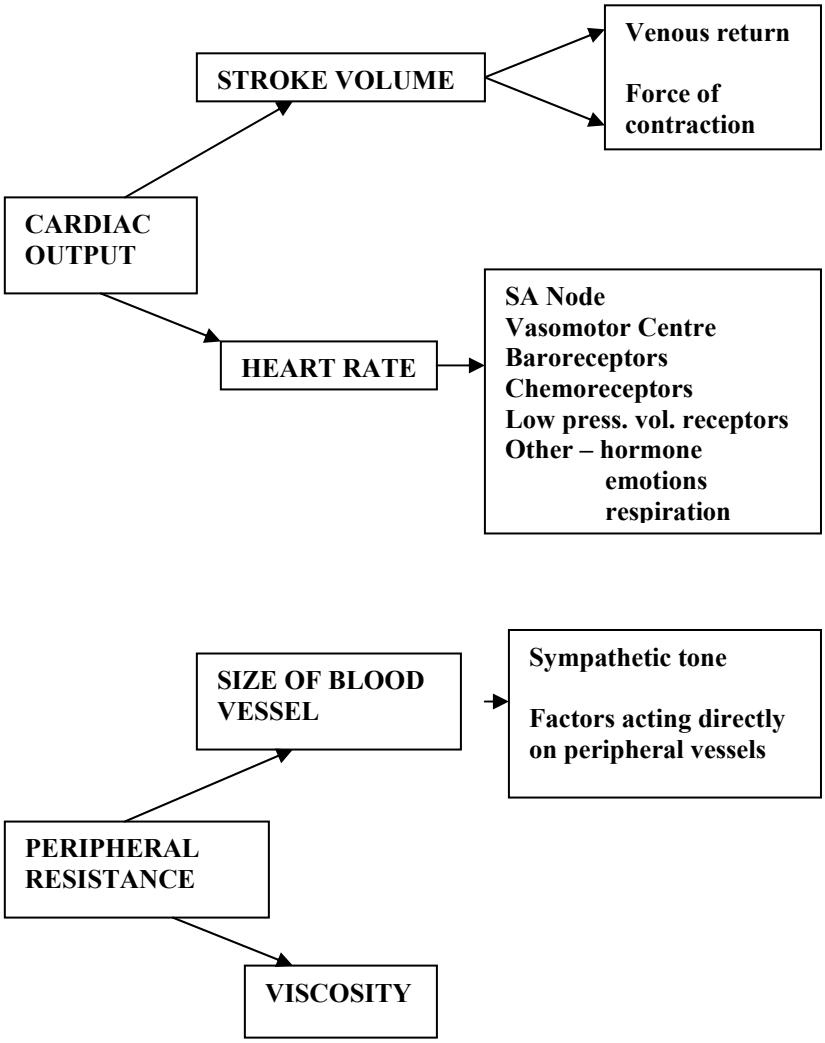
- The size or diameter of the arterioles which depends on sympathetic tone. The sympathetic outflow from the vasomotor centre (VMC) leaves via the thoracolumbar outflow to the peripheral vessels. When the venules constrict the blood is squeezed out of them and returned to the heart.
- The viscosity of the blood which imposes a resistance to flow. Viscosity is increased if the RBCs are increased. This raises peripheral resistance. Viscosity falls in anaemia and haemorrhage and this decreases resistance.

The following factors affect the VMC and hence the peripheral resistance.

- Impulses from higher centres. This will account for the peripheral vasoconstriction and rise in blood pressure associated with anger or fear.
- Baroreceptors. A fall in arterial blood pressure results in stimulation of the VMC. This results in peripheral vasoconstriction, tachycardia and in an increase in blood pressure.
- Chemoreceptors (see notes on heart rate).
- Direct effect on VMC.
- Carbon dioxide stimulates the VMC, and causes peripheral vasoconstriction. However, carbon dioxide acts on the vessel wall directly and causes vasodilation.

TO SUMMARISE

FACTORS CONTROLLING BLOOD PRESSURE



CHAPTER 5

THE SOMATIC AND AUTONOMIC NERVOUS SYSTEMS

Outline:

Definitions

The somatic nervous system: afferents, centres, efferents

Neuro-muscular junction (NMJ)

The autonomic nervous system: afferents, centres, efferents

Sympathetic nervous system

Parasympathetic nervous system

DEFINITIONS

Stimuli reach the central nervous system via two pathways:

The somatic nervous system

The autonomic nervous system

In each system there is

- An afferent impulse (an incoming message)
- A centre (in the CNS)
- An efferent impulse (an out-going message)

A ***neuron*** is the basic building block of the central nervous system. It consists of a cell body, dendrites which are short processes extending outwards from the cell body and an axon which is a long fibrous process which may be myelinated. Incoming signals affect the dendrites and cell body while outgoing signals are passed on via the axon.

THE SOMATIC NERVOUS SYSTEM

Afferents

Pain sensation is taken up by the fine nerve endings. Specific receptors are present for temperature, touch, pressure, vibration and proprioception and all these impulses are transmitted along larger afferent nerves formed by the union of smaller nerves.

Centres

These impulses reach the spinal cord and relay in the dorsal horn of the spinal cord. They next cross to the other side of the spinal cord and travel to the thalamus and then to the cerebral cortex.

The conduction of sensory impulses can be interrupted by drugs, by surgery or by disease at any of the following levels:

- Peripheral nerve endings
- Peripheral nerves
- Epidural space
- Subarachnoid space
- Spinal cord
- Cerebral cortex

Efferents

Efferents innervate the skeletal muscles. Efferents leave the pre–central gyrus in the brain, i.e. the cerebral cortex. They pass through the internal capsule and 80% cross the midline to form the lateral cortico–spinal tracts. The rest travel on the same side to form the anterior cortico-spinal tracts.
The pyramidal fibres converge on the anterior horn cells of the spinal cord. These cells supply the skeletal muscle via the motor nerve.

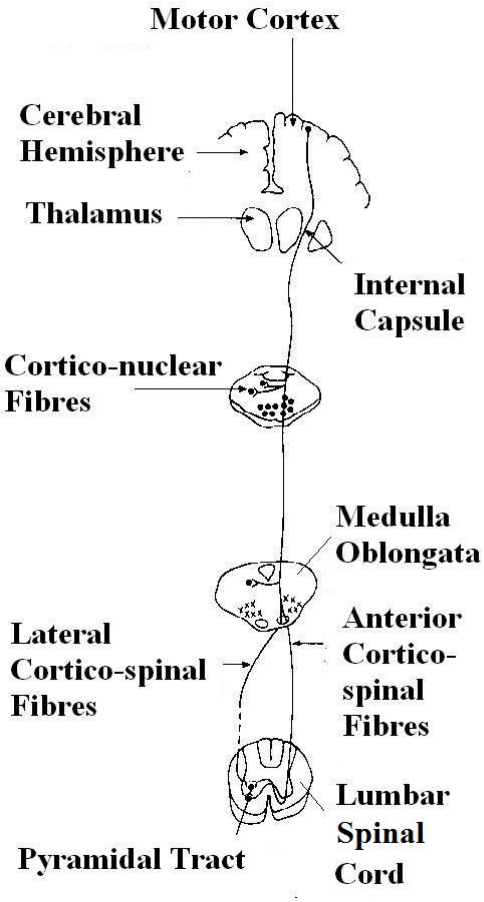


Fig 5.1 The Somatic efferent system

The efferent nerve innervates the muscles. The junction of the nerve and the muscle is termed the ***neuromuscular junction.***

THE NEUROMUSCULAR JUNCTION (NMJ) (See also Chapter 12)

At the neuromuscular junction the nerve loses its myelin sheath and expands into a broad foot-like structure.

The muscle into which the expanded end of the nerve fits is also modified. There is a depression in the muscle and a thickening of the muscle membrane which is thrown into folds. This area of the muscle is called the ***end plate***.

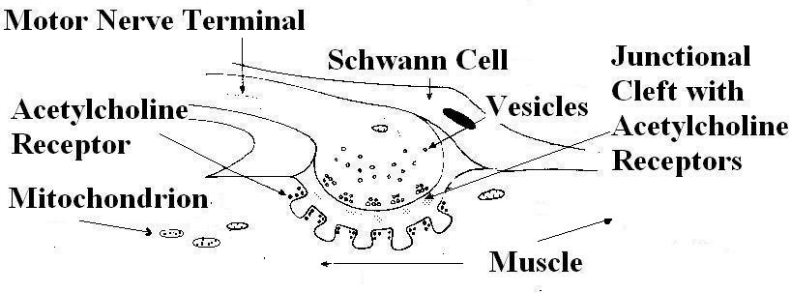


Fig 5.2 The neuromuscular junction

Sequence of events at the neuromuscular junction

- The nerve impulse travels down the motor nerve and reaches the nerve ending.
- Acetylcholine is liberated from the nerve terminals.
- Acetylcholine occupies special receptors on the end plate.
- This causes electrical and chemical changes in the muscle.
- These changes (called the end plate potential) result in muscle contraction.

Importance to the anaesthetist

By using various drugs we can either block or make possible the effects of acetylcholine at the neuromuscular junction. The muscle relaxant drugs commonly used in anaesthesia act at the neuro–muscular junction.

THE AUTONOMIC NERVOUS SYSTEM

The autonomic nervous system is divided into two separate parts sympathetic and parasympathetic, on the basis of their anatomical origins and physiological actions. Many organs have both sympathetic and parasympathetic innervation and the actions of the two divisions are usually antagonistic but can be complementary.
The autonomic nervous system is responsible for those functions in the body which are carried out automatically. (We do not think about them consciously). The autonomic nervous system is based (like the somatic nervous system) on the principle of the reflex arc. That is,

Stimulus > afferent impulse > centres > efferent impulse > effector organ.

In the autonomic nervous system, the receptors take various forms: osmo-receptors, baroreceptors, chemoreceptors and pain receptors (similar to the somatic nervous system).

Afferent pathways
These reach the central nervous system via the sympathetic and parasympathetic pathways.
Once these parasympathetic and sympathetic nerves enter the spinal cord they travel with the somatic sensory fibres to the thalamus.

The centres
These are integrated at various levels in the medulla oblongata, mid brain and hypothalamus.

The efferent pathways
These take the form of the parasympathetic and sympathetic nervous systems. Peripheral outflow is divided therefore into the sympathetic nervous system and the parasympathetic nervous system.
The parasympathetic nervous system and the sympathetic nervous system are made up of pre-ganglionic and post-ganglionic neurones.

THE SYMPATHETIC NERVOUS SYSTEM

The sympathetic nervous system fibres arise from the nerve cells in the lateral horn of grey matter of the thoracic and lumbar regions of the spinal cord. They pass out of the cord and synapse to form a chain of ganglia on either side of the vertebral column, from the base of the skull to the coccyx. From these ganglia, post-ganglionic fibres pass. These fibres form great plexuses in the front of the vertebral column, e.g. cardiac, coeliac, hypogastric plexuses.

Chemical transmitters in the sympathetic nervous system:

Pre-ganglionic endings:	acetylcholine
Post-ganglionic endings:	noradrenaline and acetylcholine

THE PARASYMPATHETIC NERVOUS SYSTEM

This is sometimes referred to as the cranio–sacral outflow.
In the cranial outflow fibres arise from the midbrain, pons, medulla and pass via the cranial nerves 3, 7, 9, 10 (especially the 10th or vagus nerve) to the organs in the head, thorax and upper abdomen.
In the sacral outflow fibres travel out with the 2nd, 3rd and 4th sacral nerves. They pass through the cauda equina and through the anterior sacral foramina.

The chemical transmitter in the parasympathetic nervous system:

Ganglionic and post-ganglionic sites:	acetylcholine.

Summary of functions of the different elements of the autonomic nervous system

- The autonomic nervous system is responsible for controlling those processes in the body not directly under conscious control.
- The sympathetic nervous system is closely associated with the action of adrenaline. This prepares the body to withstand threatening circumstances. It prepares us for " fight and flight".
- The parasympathetic nervous system slows down the body processes except digestion and absorption. It allows restoration to occur.
- All visceral organs are innervated by both the sympathetic and para-sympathetic nerves. These have opposing actions on the organs.

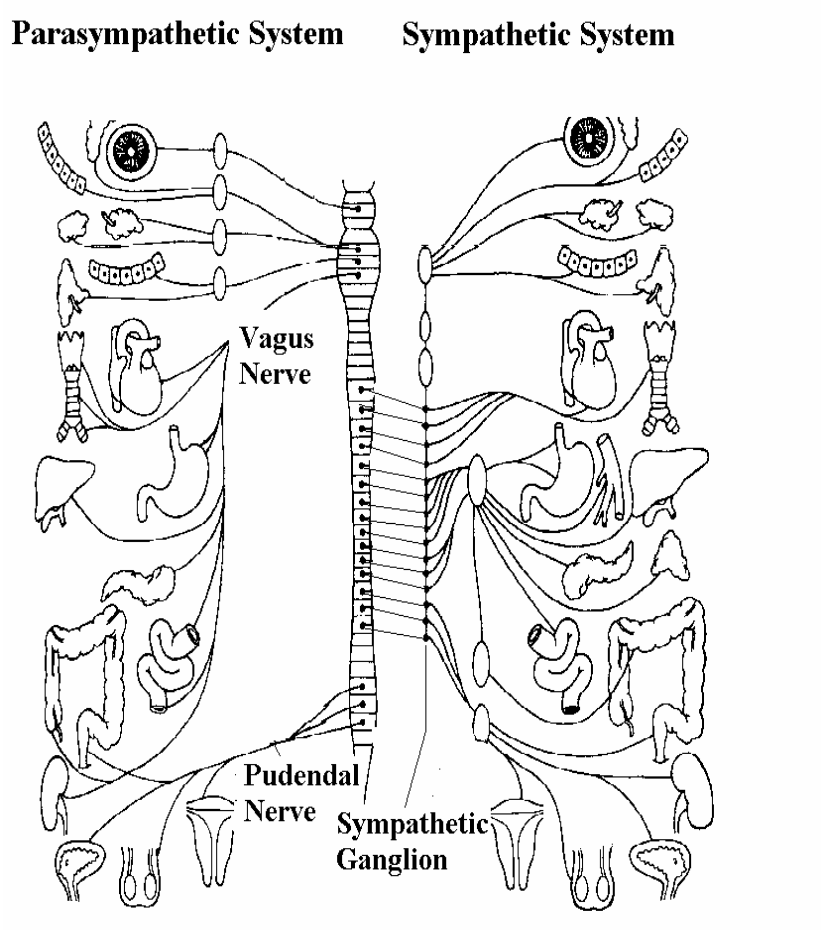


Fig 5.3 Distribution of the autonomic nervous system

Effects of autonomic stimulation:

Organ	Sympathetic stimulation	Parasympathetic stimulation
Eyes	Mydriasis (large pupil)	Myosis (small pupil) Accommodation for near vision
Exocrine glands		Salivary, pancreatic and lachrymal secretions are increased
Gastro–intestinal tract	Motility and tone are decreased	Motility and tone of GIT are increased Sphincters are relaxed Secretions are increased
Genito–urinary tract	Motility and tone are decreased	Motility and tone of ureters are increased
Respiratory system	Bronchi are dilated	Bronchi are constricted Bronchial glands are stimulated
Cardio–vascular system	Heart rate is increased Cardiac output is increased Rhythm: tachycardia Blood vessels: Coronary arteries are dilated Skin and viscera are constricted	Heart rate is slowed Cardiac output falls Rhythm: Bradycardia and atrio–ventricular block Coronary arteries are dilated

CHAPTER 6

PHARMACOLOGY OF ANAESTHETIC AGENTS

Outline:

Here is a list of the drugs most commonly used in anaesthetics.

Intravenous anaesthetics: thiopentone, methohexitone (Brietal), propofol (Diprivan), etomidate, ketamine

Inhalational anaesthetics: halothane (Fluothane), nitrous oxide, ether, trilene, enflurane (Ethrane), sevoflurane, isoflurane, desflurane **(See Chapter 11)**

Antisialagogues: (Drying agents): atropine, hyoscine, glycopyrronium

Analgesics: (Pain relievers):

- ***Non-narcotic analgesics***:
  Simple analgesic: aspirin, paracetamol
  Other non-steroidal anti-inflammatory drugs (NSAIDs) ibuprofen, ketorolac, diclofenac (Voltarol)
- ***Narcotic analgesics***: pethidine, morphine, papaveretum (Omnopon), pentazocine (Fortral), fentanyl, alfentanil

Sedative perioperative drugs: Benzodiazepines: diazepam (Valium), midazolam (Hypnovel)

Narcotic and sedative antagonists: naloxone (Narcan); flumazenil (Anexate)

Muscle relaxants: suxamethonium, tubocurarine, gallamine, pancuronium bromide, vecuronium bromide, atracurium besylate, rocuronium, mivacurium **(See Chapter 12)**

Anticholinesterases (reversal agents): neostigmine sulphate **(See Chapter 12)**

Dissociative anaesthesia: ketamine

Vasopressors: adrenaline, ephedrine, metaraminol, isoprenaline

PHARMACOLOGY OF ANAESTHETIC AGENTS

Drugs are the anaesthetist's most useful tools but when we administer a drug to a patient, especially by the intravenous route, we have an obligation to know its effects, both wanted and unwanted.

INTRAVENOUS ANAESTHETICS

THIOPENTONE

Thiopentone is a barbiturate. Barbiturates are a large group and they can be used as sedatives or hypnotics (e.g. amytal), as anticonvulsants (e.g. phenobarbitone) or as anaesthetics (e.g. thiopentone).
Thiopentone is a yellow powder, marketed in ampoules of 0.5g or 1g.
It is used in a concentration of 2.5%, prepared by dissolving 0.5g of powder in 20 ml of water, or 1g in 40 ml of water.
It is also obtainable in 2.5g bottles packaged with 100 ml water. The solution is discarded if cloudy. The solution if labelled and stored under sterile conditions in a refrigerator, can be kept for a few days.

Uses of thiopentone
As an induction agent
Treatment of convulsions

Dose
5mg/kg in the fit adult patient. No definite dose can be quoted. The appropriate dose must be determined, taking into account the conditions discussed below.

Mechanism of action
Like many anaesthetic agents thiopentone is highly fat soluble. The brain has a high blood flow and 90% of thiopentone in the cerebral capillaries crosses the blood brain barrier. Consequently the onset of action is rapid and the patient goes to sleep fairly quickly. The thiopentone is also taken up by the less well–perfused tissues, such as muscles, though it diffuses more slowly into these. Subsequently the blood concentration falls. This results in some of the thiopentone leaving the brain tissues to return to the blood and the patient shows signs of waking up again. This phenomenon is called redistribution and explains why a patient does not remain asleep for very long with thiopentone.
The drug is ultimately destroyed in the liver. Ten to fifteen percent of the injected drug is broken down per hour.

Systemic effects of thiopentone

Central nervous system: In the fit young adult the drug reaches the brain in thirty seconds. It causes sedation and has an anticonvulsant action. It decreases the oxygen consumption of the brain and also the cerebral blood flow. It has no analgesic action.

Respiratory system: Thiopentone is a potent respiratory depressant. Laryngospasm is more common with thiopentone compared to propofol.

Cardiovascular system: Thiopentone can cause a fall in blood pressure and serious hypotension by depression of the vasomotor centre in the brain and dilatation of the peripheral blood vessels, with resultant peripheral pooling. Myocardial depression can also occur. These side effects are more common in:

- Patients receiving a large dose of thiopentone
- Patients who have received a rapid injection of thiopentone
- Patients in shock or hypovolaemia
- Patients with hypertension or heart disease
- Patients with myxoedema

Foetus: Thiopentone crosses the placenta. The longer the time between induction and delivery the less effect there is on the foetus.

Muscle tone: At the point of maximal cerebral depression muscle tone is reduced. Sleep doses do not affect muscle tone.

Local side effects (i.e. at the site of injection)
If thiopentone is injected outside the vein certain complications can follow.

The most serious is

- **Intra-arterial injection:** gangrene of the limb is a real hazard. Using the medial aspect of the antecubital fossa increases the hazard. It is even easier to make this mistake if the patient struggles, is obese, has dark skin, or you are working in poor light.

  Symptoms and signs of intra-arterial injection
  - The patient will feel ***intense pain.***
  - The hand will be ***very pale or white.***
  - The hand will be ***cold*** (arterial spasm).
  - The hand will be ***oedematous***.
  - The ***pulse*** may be absent.

  When thiopentone enters an artery, there is spasm of the artery and also damage to the intima and crystal formation in the artery. This interferes with the flow of blood to the limb and if this ischaemia is not corrected, gangrene results.

Treatment Leave the needle in the artery. Cancel surgery if possible. Then:

- Flush with anticoagulant, e.g. heparin/saline (10 units/ml) 500 units.
- For relief of spasm inject procaine (10 ml of 1%), lignocaine (10mls of 1%) or papaverine (10-40 mg) into the artery.
- Sympathetic nerve blocks are recommended to dilate the collateral vessels and maintain the blood flow.
- Surgical exploration of the artery. Thrombectomy may be required.
- Full heparinisation may be required to prevent late arterial thrombosis.
- Occasionally amputation is needed.

Precautions

- Thiopentone must only be used in a concentration of 2.5% or less. This is prepared by diluting 0.5g of powder in 20 ml or 1g in 40 ml of water.
- During venepuncture the tourniquet must occlude only the vein and not the artery. The arterial pulsation will then be always palpable and reduce the chance of accidental entry into the artery.
- The medial side of the antecubital fossa is best avoided.
- It is wise to inject 2 ml of thiopentone and to inquire for any symptoms of pain. If the needle is not in the vein the patient will complain of an intense burning pain down the arm.

Other local side effects

- **Subcutaneous injection: tissue necrosis**
- **Injury to nerves**
- **Thrombophlebitis**

Contraindications to thiopentone

Absolute contraindications

- A known hypersensitivity to barbiturates
- Status asthmaticus
- Porphyria
- Airway obstruction
- Shock

Relative contraindications in which thiopentone must be used with care

- Severe heart disease
- Severe liver and renal disease
- Addison's disease, myxoedema, asthma

METHOHEXITONE (Brietal)

This is a methylated oxybarbiturate. It comes as a white powder and is used in a 1% solution as an alternative to thiopentone. It should not be used in epileptics.

Uses of Methohexitone

As an induction agent
Drug of choice for ECT (if available)

Dose : 1-1.5 mg/kg (1% solution)

Mode of action: similar to thiopentone

Systemic effects

Recovery is three times as quick after thiopentone
Less cardiovascular depression
Less irritant to the tissues as only 1% solution is used

Disadvantages

Abnormal muscle movements, tremors, coughing, hiccups.
Should not be used with epileptics as it can precipitate convulsions.
Causes pain on injection.

PROPOFOL (Diprivan)
This is a white aqueous and isotonic emulsion. The vehicle contains glycerol soyabean oil and egg phosphatide. It is produced in a 1% solution (10mg/ml) for induction of anaesthesia, in a 1% or 2% solution in a 50ml vial and a 50ml prefilled syringe.

Uses and Mode of action
Induction of anaesthesia.
Total intravenous anaesthesia by infusion (TIVA).
Target controlled infusion (TCI).
Sedation in ICU.

It is a short acting intravenous anaesthetic agent with a half-life of 1.8 – 8.3min (rapidly distributed). It is conjugated in the liver and the inactive metabolites are excreted in the kidneys.

Dose
Induction in the fit adult patient 1.5 - 2.5mg/kg titrated at the rate of 4ml/10sec. Elderly patients may only require 1mg/kg.
For children over 8 years the dose is 2.5mg/kg.
Children 3-8 years require a higher dose of 2.5 – 5mg/kg.

Maintenance
For short procedures repeated bolus doses of 25–50mg according to response may be given.
Propofol infusion: (Total intravenous anaesthesia or TIVA)
This requires a prefilled syringe and electronic syringe pump for safe administration. For more details of this technique consult a more advanced textbook of anaesthesia.

Systemic effects

Central Nervous system
- Produces rapid loss of consciousness and rapid recovery.
- Low incidence of nausea and vomiting.
- Reduces cerebral blood flow, ICP and intra-ocular pressure.
- Epileptiform movements occur very occasionally but sometimes some hours after recovery, so it is best avoided in epileptics but can be used for ECT.

Cardiovascular system
- Hypotension is greater than that after thiopentone. This is seen especially in the old, hypertensive and hypovolaemic patients.
- No increase in heart rate and may cause bradycardia.
- Hypertensive response to laryngoscopy is less than with thiopentone.

Respiratory system: Causes respiratory depression, often with a transient period of apnoea after induction. Obtunds airway reflexes. Because of these effects it is the agent of choice for insertion of a laryngeal mask

In pregnancy: Rapid placental transfer but is an acceptable induction agent for caesarean section with no greater effect on the foetus than thiopentone.

Adverse reactions

- Local pain: 28% if the veins on the dorsum of hand are used, 5% when the antecubital fossa is used. The incidence of pain is reduced by injecting 20 mg (2ml of 1%) lignocaine before or mixed with the propofol. (In animals subcutaneous and intra-arterial injections did not induce serious effects).
- Anaphylactic–type reactions (bronchospasm, erythema and hypotension) have been reported but are very rare. Propofol is best avoided in patients with a true egg allergy.
- Bradycardia: responsive to atropine.

ETOMIDATE

Etomidate was introduced as an I.V. induction agent in 1973. It is presented as a clear liquid 0.2% solution in propylene glycol

Dose : 0.3mg/kg

Systemic effects

It has a high safety margin and has very little effect on the cardiovascular system with only mild decreases in peripheral vascular resistance and blood pressure. It also causes minimal respiratory depression and does not cause histamine release. This makes etomidate a useful drug for induction of anaesthesia in hypovolaemic and frail patients.

Disadvantages

Pain on injection.
Excitatory movements during induction.
High incidence of nausea and vomiting.
Steroid suppression is now recognised to occur even after a single dose.

ANTISIALAGOGUES (DRYING AGENTS)

Atropine, hyoscine and glycopyrrolate are antimuscarinic drugs which act on the autonomic nervous system and competitively block the effect of acetylcholine at the post-ganglionic parasympathetic nerves.
Since these drugs are non-selective they have an effect on all types of cholinergic receptors to some degree.
The areas of action are:

Eye: dilatation of the pupil. In glaucoma there is an increase in the intraocular pressure.

Exocrine glands: decrease in salivary and bronchial secretions.

Cardiovascular system: an increase in the heart rate (a small dose of atropine if given IM can cause initial slowing of the heart rate before the tachycardia).

Gastro-intestinal tract and genito-urinary tract: decreased motility.

ATROPINE

This is a naturally occurring alkaloid and can be given orally but more usually IM or IV.

Dose 10 micrograms/kg. Average adult dose is 600 micrograms IM.
It causes an increase in heart rate and moderate smooth muscle relaxation. It has a drying effect on secretions and causes dilatation of the pupils.

HYOSCINE (Scopolamine)

This is also a naturally occurring alkaloid and can be given orally, IM or sub-cutaneously.

Dose: 8 micrograms/kg. Usual dose 200-600 micrograms.
It has a marked sedative, anti-emetic and amnesic effect but only a mild action on the heart. It is a more effective drying agent than atropine. It also causes marked dilatation of the pupil.

GLYCOPYRROLATE

This is a synthetic compound which does not easily cross the blood–brain barrier. It can be given IM or IV.

Dose: 200-400 micrograms.
It is the most effective agent for drying secretions and has a moderate effect on heart rate and smooth muscle.

ANALGESICS (PAIN RELIEVERS)

NON-NARCOTIC ANALGESICS

- **Simple analgesics**

ASPIRIN

This is widely available and is taken orally in tablet or soluble tablet form and as suppositories rectally.

Uses and mode of action

Mild to moderate pain.
Antiplatelet action by inhibiting thrombin formation in the arterial circulation.
It is rapidly metabolised in the gut to the active compound salicylic acid.

Dose: 300-900 mg 4-6 hourly. Max. 4gm daily.

Disadvantages

It should not be used in children under 12 yrs.
It has gastrointestinal side effects – nausea, vomiting and gastro-intestinal bleeding due to antiplatelet effect.
Not advisable for patients on warfarin.

PARACETAMOL

This is widely available and is taken orally in tablet, soluble tablet and suspension form or as suppositories rectally.

Uses and mode of action

Mild to moderate pain.
Treatment of pyrexia.
Well absorbed orally. Metabolised in the liver.

Dose: Adult: 500 mg – 1 gm 4-6 hourly. Max 4gm daily.
Child: 20mgs/kg orally. Max 90mg/kg/day

Side effects

Rarely causes side effects.
Causes hepatotoxicity in overdosage.

- **Non-steroidal anti-inflammatory drugs (NSAIDs)**

Uses and mode of action
These drugs are suitable for relief of mild to moderate post–operative pain especially pain related to bone surgery.

- They have analgesic, anti-inflammatory and antipyretic properties.
- They can be given orally, rectally and parenterally.
- They have long half lives and so can be given twice daily.
- They reduce opioid requirement and do not cause sedation or respiratory depression.

Side effects

- Increase in bleeding time.
- Can cause gastric erosions.
- Cause a reduction in renal blood flow.
- Can cause bronchospasm in asthmatic patients (10% incidence).

Contraindications

- Patients with history of peptic ulcer disease.
- Patients with renal disease.
- Pregnant patients.
- Dehydrated patients.

The lowest effective dose should always be used.
Three commonly used examples are:

IBUPROFEN
Less anti-inflammatory effect than others but fewer side effects.

Dose: 1.2 - 2.4 gms daily in divided doses.
Presented as tablets and syrup.

DICLOFENAC (Voltarol)
Can be given orally, rectally or IM or by IV infusion.

Dose: 75-150 mg O
12.5-100 mg suppositories
25mg/ml for injection.

KETOROLAC
Can be given orally or by IM or IV injection (10mg/ml).

Dose: 10mg O 4-6 hourly
By injection 10-30mg

NARCOTIC ANALGESICS
The narcotic analgesics can be divided into 3 large groups.

Naturally occurring alkaloids
- morphine
- papaveretum (omnopon)
- codeine

Semi synthetic alkaloids
- diamorphine
- dihydrocodeine

Synthetic agents
- benzmorphone derivatives
  - pentazocine (Fortral, Ortalwin)
- piperidine derivatives
  - pethidine (Demerol)
  - fentanyl
- diphenylheptan derivatives
  - methadone (Physeptone)

Opioid Receptors
There are at least 3 types of opioid receptors, known by the Greek letters μ(mu), ∑ (sigma) and K (kappa). Some of them are found in the dorsal horn of the spinal cord. The discovery of opioid receptors in the spinal cord meant that the opioid drugs (e.g. morphine) could be introduced into the spinal and epidural spaces, for the relief of chronic and acute pain.

The **advantages** of opioid drugs over local anaesthetic drugs in the spinal and epidural space are:
- No block of sympathetic nerves, therefore no hypotension or bradycardia.
- No motor block, so no weakness or paralysis.
- No loss of other sensation apart from pain.
- Accidental IV injection can be treated with naloxone.
- Analgesia is effective and prolonged and achieved with smaller doses than used systemically.

BUT

- Respiratory depression has been reported as long as 16 hours after injection. (This is reversible with naloxone but requires very careful observation).
- Other complications such as pruritus, nausea and vomiting and urinary retention are common. Intrathecal opioids, although used in some major centres, are not recommended in smaller hospitals.

Commonly used narcotic drugs

MORPHINE

Uses
Relief of moderate to severe pain.
Metabolised by the liver and excreted in the urine.
Available for oral administration and in concentrations from 10mg-30mg/ml for IM and IV injection and infusion.

Dose: Adult: 10-15mg 1M or subcutaneous (SC) 3 hourly PRN
2.5–10mg IV.
Child: 0.1–0.2mg/kg IV/SC 1-2mg boluses as required.

Duration: 3 hours.

Systemic effects

Central nervous system
- Depressant action; relieves anxiety
- Analgesic
- Respiratory depressant
- Can cause euphoria, addiction

Respiratory system
- Respiratory rate is decreased
- Tidal volume may increase
- Bronchoconstriction may occur (histamine release)

Gastrointestinal tract
- Sphincters of gut may be constricted
- Nausea and vomiting may occur
- It can produce biliary spasm

Eyes: Pupil constriction. This is one reason why morphine must not be used for head injury.

Side effects
Nausea and vomiting
Respiratory depression

CODEINE
This has one sixth the analgesic potency of morphine.
It is available as tablets, syrup and IM injection.

Uses and mode of action
It is used for mild to moderate pain and as a cough suppressant.
It is metabolised in the liver. About 10% is converted to morphine and this is probably responsible for its analgesic properties.

Dose: 30-60mgs orally 4 hourly, 30-60 mgs IM 4hrly.

PETHIDINE
Available for oral (50mg tabs), IM and IV (50mg/ml) administration.

Uses and metabolism
Moderate to severe pain. Also used for analgesia in obstetrics.
The duration of action is shorter than with morphine lasting about 2 hours.
90 percent undergoes metabolism by the liver to an active substance with a long half-life (20–40 hours), called Norpethidine, which can cause hallucinations and convulsions. Norpethidine is excreted by the kidneys, so care must be taken in patients with renal failure if repeated high doses are being used.

Dose:	Adult:	50-150mg O 4 hourly
		50-100 mg IM/ SC 2 hourly PRN
		25-50 mg IV 4 hourly
	Child:	0.5-2.0mg/kg O
		0.5-2.0mg/kg IM 2 hourly PRN

Systemic effects
Similar to morphine.
Pethidine has an atropine-like effect causing dilatation of the pupils and drying of secretions.

Contra-indications
Should not be used for patients who are taking monoamine oxidase inhibitors.

PENTAZOCINE (Fortral)
This has actions similar to morphine and pethidine but has opioid agonist and antagonist properties. It is less effective than morphine or pethidine and may cause hallucinations.
It is available as 25 and 50mg tablets and 30mg/ml for IM and IV injection.

Dose: Adult: 30 to 60mg (depending on the patient).
Child: 0.5 mg/kg IM.

60 mg of pentazocine gives analgesia equal to 10mg of morphine.

PAPAVERETUM (Omnopon)
This is a mixture of purified opium alkaloids.
Fifty percent of the mixture is morphine. The other alkaloids are codeine, papaverine, narcotine etc.
It is available for IM and IV injection 15.4mg/ml.

Uses
It is used for the relief of moderate to severe pain and for premedication, often combined with hyoscine.

- To relieve anxiety
- To reduce the dose of anaesthetic required
- To prevent tachycardia when used with trilene

Dose: Adult: 7.7-15.4mg IM
Child: 6-12 years 4 -7.7mgs IM

Side effects
As for Morphine

Take special care with

- Infants less than 6 months
- Aged and debilitated patients
- Patients on monoamine oxidase inhibitors (MAO inhibitors)
- Patients with metabolic diseases (e.g. liver and renal diseases), respiratory disease (e.g. asthma, COAD), endocrine diseases (e.g.Addisons, hypothyroidism).
- Very ill patients

10mg morphine =100mg pethidine =15mg papaveretum =60mg pentazocine

FENTANYL CITRATE
Fentanyl is a narcotic analgesic like morphine, pethidine and pentazocine.

Uses and mode of action
For analgesia as part of an anaesthetic.
Used transdermally for chronic cancer pain.
It is highly lipid soluble giving it rapid onset of action.

Dose: 1 microgram/kg IV

Duration of action: 30mins–1hr in normal doses.

Systemic effects
Fentanyl produces analgesia, sedation, euphoria, respiratory centre depression, miosis and suppression of the cough reflex. It has minimal effect on the cardiovascular system.
The incidence of nausea and vomiting is minimal. Incidence is 4% after fentanyl as opposed to 20% after pethidine.

Side effects
Can cause delayed respiratory depression with higher doses.
Can cause bradycardia which is responsive to atropine.

Not suitable for use as an anaesthetic drug with:
- Very young patients less than 1 year of age.
- Comatose patients.
- Those with head injuries.

Fentanyl can be habit forming.

ALFENTANIL
Is a rapid onset, short duration opioid drug similar to fentanyl, good for outpatient surgery. Potency is a quarter that of fentanyl but onset of action is four times more rapid. Duration of action is one third of fentanyl.

Dose: 5-10 micrograms/kg.

SEDATIVE PERI-OPERATIVE DRUGS: Benzodiazepines

Benzodiazepines are useful in anaesthesia principally for premedication and as sedation for short procedures. They are also used in ICU as sedation in patients receiving assisted ventilation.

DIAZEPAM (Valium)
Diazepam can be taken orally, IM, IV or rectally.

Dose: 5-10 mg orally as premed. 0.1mg/kg by injection.
Diazepam is relatively insoluble in water and can be painful on injection with a high incidence of venous thrombosis. It can be mixed with dextrose 5% or saline 0.9%. Diazepam also comes in the form of an emulsion (Diazemuls) which reduces the risk of thrombophlebitis.

Uses and mode of action
Premedication
Sedation with amnesia
As an anti-convulsant
Useful in combination with ketamine to reduce unpleasant after effects
Its muscle relaxant properties are useful in the treatment of Tetanus

It has a long duration of action with active metabolites which can produce a second period of drowsiness.
It is metabolised in the liver.

Systemic effects
It produces a sedative and tranquillising effect with some amnesia and also has good anti-convulsant properties.
It can help relieve muscle spasm and spasticity.

Side effects
- Can cause respiratory depression
- Hypotension
- Ataxia and confusion
- Pain on injection

MIDAZOLAM (Hypnovel)
This is a water soluble benzodiazepine. It is available for IM and IV injection as 1mg/ml or 5mg/ml. Can be given orally to children.

Uses
Conscious sedation for short procedures
Long-term sedation for ventilated patients in ICU
Can be used as part of a general anaesthetic both for induction and maintenance
Anticonvulsant

Midazolam is shorter acting than diazepam.

Dose: 30-100micrograms/kg IV should be given slowly (approx.2mg/min)
Reduce dose and give more slowly in the elderly
Premedication in children: 0.4mg/kg orally given with paracetamol syrup

Systemic effects
Sedative and tranquillising effect
Provides anterograde amnesia
Has minimal cardiovascular and respiratory effects in small doses

Side effects
Respiratory depression
Respiratory arrest especially with higher doses

Precautions
- Always watch for signs of under-ventilation.
- Don't use it in children under the age of 8 years except for premedication.
- Should not be used for nursing mothers.
- Take special care with elderly and high risk patients and reduce the dose by 25% at least. See note on midazolam and conscious sedation at the end of Chapter 25 (Anaesthesia for dental surgery).
- Midazolam can be mixed with sterile water, 5% dextrose in water, normal saline and Hartmann's solution.

COMPARISON BETWEEN	
MIDAZOLAM (Hypnovel) &	**DIAZEPAM** (Valium)
Water soluble	Insoluble in water
Not irritant to veins Twice as potent as diazepam	Irritant
Amnesic and hypnotic actions better than diazepam	
Half life 1-4 hours	Half life 21-35 hours
Metabolises in liver to hydroxy midazolam which is weaker than the parent compound	Metabolised in liver to desmethyl diazepam and also oxazepam. Drowsiness can persist because of metabolites.
Dose: 30-100microgm/kg IV Slowly titrate to response. Give 1-2mg IV slowly. Wait 2 min before next increment	**Dose:** 0.1 - 0.2mg/kg orally or 0.1mg/kg IV

ANTAGONISTS FOR CENTRAL AND RESPIRATORY DEPRESSION

NALOXONE

Central depression caused by **narcotics** can be reversed by **naloxone** by competing with them at the site of action and can counteract the respiratory depression produced by narcotic analgesics.

Dose: 100-200 micrograms, then if necessary give increments of 100 micrograms every 2 minutes

Neonate : 10 micrograms/kg

Duration: 20 minutes

As naloxone has a short duration of action the dose may need to be repeated.

Naloxone also antagonises the analgesic effect of the narcotic.

FLUMAZENIL (Anexate)
Respiratory depression due to sedation with **benzodiazepines** e.g. diazepam and midazolam following anaesthetic, intensive care or diagnostic procedures can be reversed by **flumazenil.**

Dose: 200 micrograms over 15 secs then 100 micrograms at 60 second intervals as required up to maximum total dose of 1mg.
It is short acting. A single IV dose lasts between 15 and 140 minutes (average 58 minutes). Patients who have taken long acting benzodiazepines such as diazepam may need a further dose of flumazenil for the antagonising action to continue, though this will not be a problem if they have taken midazolam which is short acting.

Mode of action
Benzodiazepines in increasing doses produce the following effects:

- Relief of anxiety (anxiolysis)
- Relief of convulsions (anticonvulsant effect)
- Mild sedation
- Amnesia
- Deep sedation
- Muscle relaxation
- Anaesthesia

Note that each effect is stronger than the one before it.

Flumazenil antagonises these effects in reverse order. Therefore, if a patient has lost consciousness because of an excessive dose of diazepam, flumazenil will reverse this. It can also be used in the management of a patient who has taken an overdose of diazepam or any other benzodiazepine. It can also be used as a diagnostic tool if the nature of the drug taken is not known.
Flumazenil has no action of its own on the central nervous system.

DISSOCIATIVE ANAESTHESIA

KETAMINE
Ketamine is a phencyclidine derivative. Following injection, patients become mentally dissociated or removed from the surroundings. This is referred to as dissociative anaesthesia. Ketamine (Ketalar) is rapidly absorbed after intramuscular and intravenous injection. It is metabolised and excreted in the urine.

Indications

As an induction agent (in place of thiopentone):

- In shocked patients **or** poor risk patients
- In asthmatics
- In obstetric patients for caesarean sections:
  - It causes less hypotension or foetal depression
  - It may increase uterine tone

As a sole anaesthetic for minor operations, dilatation and curettage, fractures, diagnostic procedures, burns dressing, debridement (bolus plus infusion or infusion only).

As an infusion with a relaxant anaesthetic (see Chapter 14 Low cost techniques).

Dose

IV ketamine 1-2mg/kg.

Onset of action less than 15 seconds; unconsciousness in 30 seconds.

Duration 5-10 minutes.

IM ketamine 5 - 7mg/kg, onset may take 2-8 minutes.

Duration 10-20 minutes. Subsequent dose is half of the initial dose.

Unless there is a strong contraindication an atropine premedication should be used to reduce the effects of the increase in salivation.

Systemic effects

Central nervous system: the patient is sedated, amnesic and analgesic.

It is difficult to detect the point at which sleep commences as patients appear to gaze into space and may not close their eyes for several minutes. The eyelash and corneal reflexes remain. There is dissociation from the surroundings.

Cerebral blood flow and intracranial pressure is increased. Awakening from anaesthesia may take several hours and may be associated with vivid dreams and hallucinations, especially in adult patients.

Cardiovascular system: cardiac output and the systolic and diastolic blood pressures are increased.

Respiratory system: respiration may be depressed if large doses of ketamine are used. The larynx and pharynx usually remain competent but this cannot be guaranteed.

Other actions of ketamine

Rise in intra-ocular pressure. Eye movements may be noticed.

Uterus not relaxed.

At 1 mg/kg given slowly over 1 minute it does not depress the foetus, though it has been known to contract the pregnant uterus and cause foetal distress.

Advantages

- Minimal cardiovascular depression, which is good in the shocked patient.
- Minimal depression of pharyngeal and laryngeal reflexes, so the airway is maintained without intubation, provided the patient has had the right amount of ketamine. The airway is not, however protected from aspiration. If the patient is at risk of aspiration a rapid sequence induction should be undertaken.
- Very good analgesic.
- Easy to administer.

Disadvantages

- Emergence reactions: vivid dreams and hallucinations occur, especially in adults. Decrease the incidence of emergence reactions by:
  - Not using ketamine in nervous patients or those with psychiatric problems
  - Using diazepam with the ketamine
  - Preventing stimulation in the recovery period
- Hypertension.
- It is difficult to know when a sufficient dose is given. Eyes may be open and there may be spontaneous movement.
- No relaxation of muscles.
- Salivation increases.
- Respiratory depression.

Precautions with ketamine

- Select patients carefully.
- Don't use ketamine in hypertensive patients, patients with head injuries or nervous patients.
- See that the patient fasts for 6 hours before surgery.
- Weigh the patient before surgery.
- Make sure there is oxygen and a means of ventilation and suction available.
- Check blood pressure before starting the anaesthetic.
- Leave the needle in the vein.
- Inject ketamine slowly (if IV).
- Don't leave the patient unattended.
- Don't test eyelash reflex as for general anaesthesia. It is not abolished with ketamine.
- If the patient shows purposeless tonic or clonic movements of the limb it does not mean the anaesthesia is too light.

VASOPRESSORS

These are mostly sympathomimetic drugs. The sympathetic nervous system has many actions which have been discussed in Chapter 5. It causes vasoconstriction of the skin and splanchnic vessels (this is referred to as stimulation of the "alpha receptor"). It also stimulates the heart, that is it increases the force of contraction and the rate of the heart (this is referred to as "beta" stimulation).

Some vasopressors act by stimulating the "alpha" receptors causing peripheral vasoconstriction thus increasing the venous return and raising the blood pressure. Others act on the heart, that is, on the "beta" receptors and raise the blood pressure.

It is important to be familiar with the action and dosage of two or three vasopressors:

ADRENALINE (Epinephrine)

Available for IV, IM and subcutaneous injection in concentrations
of 1:1,000 (1mg/ml)
and 1: 10,000 (1mg/10ml)

Uses and mode of action

Resuscitation of seriously ill patients
Treatment of anaphylactic shock
Treatment of hypotension intra-operatively
As an additive to local anaesthetics to prolong duration

Acts on alpha 1, beta 1 and beta 2 receptors

Dose: Bolus doses of 100 micrograms – 1mg IV (preferably through a central venous catheter) depending on the patient's condition. Some patients may require a continuous infusion (2–20 micrograms/min) with careful monitoring.
During resuscitation from cardiac arrest the injection can be given directly into the heart or down the lumen of an endotracheal tube if there is no venous access.

Systemic effects

Cardiovascular system: Increase in heart rate and force of cardiac contraction. Increase in systolic blood pressure.

Respiratory system: Relaxes bronchial smooth muscle causing bronchodilatation.

Side effects
Ventricular arrhythmias
Hypertension
May cause arrhythmias during anaesthesia with halothane

EPHEDRINE
Available for IM and IV injection in concentrations of 30mg/ml and 50mg/ml

Uses and mode of action
Treatment of hypotension due to vasodilatation e.g. after spinal or epidural anaesthesia.
Ephedrine acts on alpha 1, beta 1 and beta 2 receptors and mainly causes a rise in heart rate and blood pressure.

Dose: 3-10mg IV as a bolus –may require to be repeated.
Duration of action 5-15 mins.

Side effects
Tachycardia
Hypertension
Arrhythmias during halothane anaesthesia

METARAMINOL (Aramine)
Available IV, IM or S/C injection or for IV infusion in concentration of 10mg/ml

Uses and mode of action
Acts on alpha 1 receptors
Increases blood pressure and cardiac output
Treatment of hypotension following spinal anaesthesia

Dose: 1 mg bolus IV
1-20 mg/hr by IV infusion

ISOPRENALINE
Available for IV and IM injection

Uses and mode of action
Acts on beta 1 and beta 2 receptors.
Causes an increase in force of contraction and rate of heart.
Used for treatment of complete heart block or severe bradycardia.

Dose: 5-20 microgram bolus
0.5-10 micrograms/min by infusion

Side effects
May cause tachycardia and ventricular arrhythmias.
May worsen coronary perfusion in ischaemic heart disease.

CHAPTER 7

PRE-OPERATIVE CARE

Outline:

Pre–operative assessment
- History
- Examination
- Investigations
- ASA Classification

Pre–operative preparation
- Principles
- Emergency surgery – special problems
- Fasting Guidelines

Premedication
- Purposes
- Drugs used for premedication
- Routes of administration
- Choice of drugs

PRE-OPERATIVE ASSESSMENT

The role of the anaesthetist begins not in the operating theatre but in the ward. The pre-operative assessment is designed to present the patient for surgery in the best possible condition.

HISTORY

Anaesthetic

Any problems encountered during past anaesthetics must be fully investigated. Records of previous occasions yield a wealth of information on response to various drugs, intubation difficulties, allergic responses and post–operative problems.

Family anaesthetic history is also important because certain abnormal and possibly dangerous responses to drugs (e.g. malignant hyperpyrexia, suxamethonium apnoea) tend to run in families.

Further, several diseases which can give rise to "anaesthetic problems" for instance sickle cell disease, dystrophia myotonica and blood dyscrasias, have a familial incidence.

Medical

- Respiratory problems: cough, sputum, smoking, asthma, breathlessness and exercise tolerance. History of previous chest disease (TB, bronchitis etc)
- Cardiovascular disease:
  - Difficulty in breathing, palpitation, chest pain, ankle oedema
  - Previous heart attacks
  - Exercise tolerance
  - Hypertension
- Other illnesses, e.g. diabetes, renal disease, hiatus hernia, epilepsy
- Alcohol and drug intake
- Allergies
- Smoking habits

Surgical

Past surgical procedures as well as that for which the patient is being assessed are important.

Some operations, such as those on the heart, lungs, kidneys and CNS may tend to interfere with vital functions under anaesthesia.

Drug history

The following drugs, previously or currently being taken may influence present anaesthesia.

Steroids

Prolonged steroid therapy (> 10mg prednisolone/day) results in atrophy of the adrenal glands so that they cannot secrete extra hormones in time of stress. Collapse, with a fall of blood pressure, may ensue. Many different regimes have been described to provide perioperative steroid cover. The principles are as follows:

- Hydrocortisone sodium succinate (rapidly acting) is the drug most frequently used.
- Steroid cover is provided if the patient has had steroids in the three months before surgery unless the surgery is very minor and imposes very little stress on the patient.
- Hydrocortisone is administered at induction.
  Dose: 25mg IV (adult)
- Any unexplained fall in blood pressure either during or after surgery is treated with steroids. However hypotension from more common causes i.e. blood loss or hypoxia must be excluded.
- The steroid cover is maintained until the stress of the operative and post-operative period is over and then gradually reduced.

For a more detailed description of this regime see Chapter 40

Antihypertensive drugs

These drugs produce their effect by a reduction in peripheral vascular tone. This tends to interfere with circulatory homeostasis under anaesthesia. Most patients are left on these tablets until the day of operation. The anaesthetist must bear in mind that these patients cannot compensate for such stresses as blood loss, changes in posture, intermittent positive pressure ventilation (IPPV), etc. in the same way as normal patients can. Further, they may react badly to drugs such as thiopentone which can cause a fall in blood pressure.

Monoamine oxidase inhibitors (MAOI)

The actions of these drugs are imperfectly understood. They interact with narcotic analgesics, e.g. pethidine and morphine and result in various bizarre reactions - severe hypo or hypertension, coma, convulsions, Cheyne Stokes respiration and death. They also react abnormally with pressor drugs and potentiate the side effects of barbiturates. The effects of MAOI last from 1 to 2 weeks depending on the drugs. Suspension of MAOI will be necessary for major surgery requiring post-operative analgesia. This must be done 10 to 14 days pre-operatively.

Beta-blockers
Commonly used for the treatment of hypertension, cardiac arrhythmias, ischaemic heart disease etc. In most cases the patients may remain on these drugs but remember that they cannot compensate efficiently, in the face of cardiovascular stress.

Diuretics
Prolonged diuretic therapy interferes with electrolyte balance.
This must be checked pre-operatively (especially potassium).

Insulin
Patients on antidiabetic treatment must be carefully assessed by the anaesthetist for pre and post-operative care (see Chapter 39).

Antibiotics
Large parenteral doses of antibiotics – neomycin, streptomycin and others – have been known to potentiate the action of non-depolarising relaxants.

Phenothiazines
These cause peripheral vasodilatation and result in a fall in blood pressure under anaesthesia. They also potentiate the action of narcotics and barbiturates and these drugs must be used in smaller doses.

EXAMINATION

General examination

Note the following:

General appearance of the patient in bed including age and approximate weight. For instance, is the patient dyspnoeic? nervous? sweating ? Or suffering from anaemia, cyanosis, jaundice, oedema, dehydration (any evidence of early dehydration must be detected. Note skin turgor, tongue, urine output, pulse, superficial veins etc). Note temperature.

Pathological problems: difficulty in opening the jaws, difficulty in extending the neck, tumours or inflammation of the neck, burns, contractures of the neck.

Finally, assess the ***psychological state*** of the patient. This will influence the choice between regional or general anaesthetic and also the premedication required.

Airway examination

Note any ***anatomical features*** that would hinder the maintenance of a clear airway or interfere with endotracheal intubation, e.g. bull neck, receding lower jaw, high arched palate or protruding teeth.

A simple and easy test known by the name of *Mallampati,* who first described it, involves sitting in front of the patient and asking them to open their mouth fully and stick their tongue out. If the faucial pillars, soft palate, posterior pharyngeal wall and uvula are visible laryngoscopy should not be difficult. The likely degree of difficulty increases as less of the anatomy is visible and if only the hard palate is visible a difficult laryngoscopy is probable.

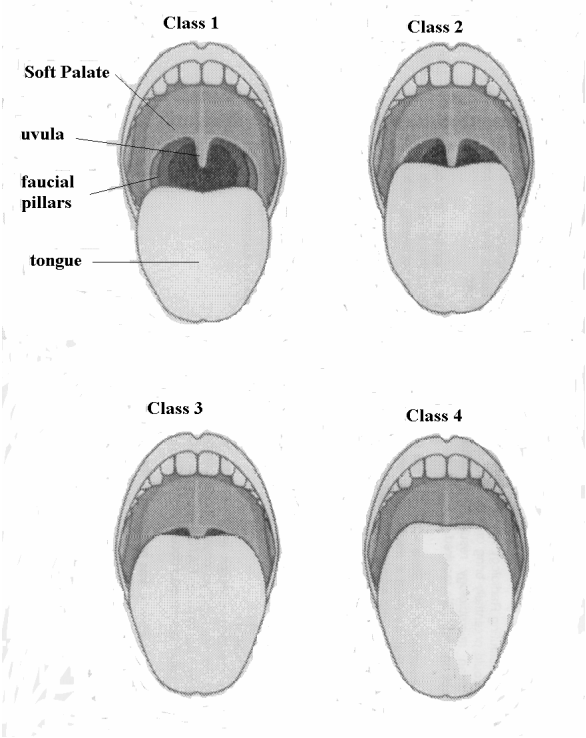


Fig 7.1 Mallampati classification and view obtained

Class 1 Faucial pillars, soft palate and uvula
Class 2 Faucial pillars and soft palate
Uvula is masked by base of the tongue
Class 3 Only soft palate visible
Class 4 Soft palate not seen

This test is useful but is not a substitute for a good history and examination

System Examination

RESPIRATORY SYSTEM

Inspection

- Note rate and type of breathing — is it noisy, laboured, obstructed.
- Shape of chest and movement of the chest.
- Deformities of the spine.
- Sputum colour, quantity (if any) of nasal discharge.
- Cyanosis central or peripheral.
- Clubbing of fingers.

Palpation

- Confirm chest movement.
- Note the position of the trachea.
- Check for the presence of enlarged supraclavicular lymph nodes.

Percussion

Compare the percussion notes at equivalent positions on both sides of the chest.

Auscultation

- Breath sounds.
- Accompaniments: crepitations, rhonchi.
- Vocal resonance.

CARDIOVASCULAR SYSTEM

The pulse Note the rate, rhythm, volume and character of the pulse wave and vessel wall. Check the peripheral pulses, including arterial pulsations in the neck.

Jugular venous pressure

Blood pressure

Colour of mucous membrane: cyanosed or anaemic

Oedema (especially dependent)

Examination of the heart

Inspection and palpation To detect and confirm the position and quality of the apex beat. Displacement of the apex beat may suggest cardiac enlargement.
To confirm also the presence of any thrills over the precordium.

Auscultation
To listen to the heart sounds and identify murmurs.

OTHER SYSTEMS

Depending on the patient it may be necessary to examine the other systems e.g. the central nervous system.

INVESTIGATIONS

These depend on the facilities available. In the smaller hospital urinalysis and Hb may be all that is available.
In a larger hospital some or all of these tests may be considered routine, depending on the patient and the surgery.

- FBC or at least haemoglobin
- Urinalysis
- Chest x-ray where indicated
- ECG in patients with a history of cardiac disease
- BUN/creatinine and serum electrolytes

Specific investigations

This depends on the underlying medical condition of the patient.

If there is ***respiratory disease*:**

- Lung function studies: vital capacity and forced expiratory volume (FEV_1), arterial blood gases
- Sputum culture and sensitivity
- More specialised radiological examination
- Bronchoscopy, etc.

If there is ***liver disease:***

- Liver function tests
- Prothrombin index

If there is ***diabetes*:**

- Four hourly blood (glucometer) or urinalysis (in the ward) for sugar and acetone
- Fasting blood sugar
- Glucose tolerance tests

If the patient is ***anaemic*** or the proposed surgery would necessitate ***blood transfusion*** then the patient's blood must be grouped and cross–matched.

ASA CLASSIFICATION - Assessment of physical status

The American Society of Anaesthesiologists (ASA) has classified patients as follows

Class	1	A normal healthy patient
	2	A patient with mild systemic disease
	3	A patient with severe systemic disease limiting activity but not incapacitating.
	4	A patient with incapacitating disease – a constant threat to life
	5	A moribund patient

If it is an emergency the category number above should be followed by the letter E (denoting emergency).

PRE-OPERATIVE PREPARATION

GENERAL PRINCIPLES (ELECTIVE AND EMERGENCY):

Any medical condition that can be corrected or improved must first be treated so that the patient is in the fittest possible physical state before surgery. The following are some medical problems that may require treatment.

Anaemia

Depending on the time available for treatment and the Hb deficit, iron deficiency anaemias may be treated with:

oral iron
parenteral iron
blood transfusion

Anaemia must always be investigated before treatment. Anaemia decreases the oxygen carrying power of the blood. (In anaemia grave hypoxia is not accompanied by cyanosis). Ideally major surgery should not be performed if the patient's Hb is less than 10g% but this is not always possible and a minimum of 8g% can be accepted. The urgency and cause of the anaemia e.g. menorrhagia, may have to be taken into account.

Sickle cell disease is a common cause of anaemia in patients from African and Caribbean countries and if there is a family history or any past symptoms suggestive of a sickling crisis a sickledex test should be performed if possible.

For more detail about sickle cell disease see Chapter 33.

Cardiovascular disease

- ***Myocardial infarction:*** A minimum of three months and preferably six months, must be allowed before elective surgery.
- ***Cardiac failure*** must be treated before elective surgery.
- ***Arrhythmias:*** Arrhythmia is not a contraindication for surgery but an attempt must be made to correct the arrhythmia to the best possible degree, e.g. in atrial fibrillation the ventricular rate must be reasonably slowed with digoxin or beta-blocker before anaesthesia. The arrhythmia must not be severe enough to interfere with the patient's cardiac output.
- ***Hypertension:*** This must be treated pre-operatively. Uncontrolled hypertension can result in left ventricular failure, arrhythmias and cerebrovascular disturbances under anaesthesia. Ideally the blood pressure should be stabilised to a diastolic pressure of 90–100mmHg. It is probably safe to go ahead with elective surgery in a patient with a diastolic pressure of 110 mmHg or less provided there are no complications of hypertension.

Respiratory disease

- ***Acute respiratory disease*** is a contraindication to GA.
- ***Chronic respiratory disease*** e.g. COAD (Chronic Obstructive Airways Disease) must first be investigated and then treated with the usual measures of physiotherapy, no smoking, bronchodilators and antibiotics if necessary.
- ***Asthma*** must be treated with the appropriate bronchodilators until the chest is clear for auscultation, before elective surgery is contemplated.

Metabolic diseases

- ***Diabetes mellitus*** must be first investigated and assessed and then controlled before elective surgery is performed. The anaesthetist must very carefully work out a regime for the control of the diabetic state during the operative period.
- ***Liver disease*** especially in relation to the prothrombin index. After a severe case of infective hepatitis, operation is best postponed for a minimum of six months.
- ***Thyroid disorders:*** Both hyperthyroidism and hypothyroidism must be corrected before elective surgery. In addition to the problems of arrhythmias and heart failure in the toxic patient the danger of "thyroid storm" occurring in the post-operative period necessitates complete control of the toxic state. (See Chapter 26)

Fluid Imbalance

Whenever possible the volume of circulating fluid should be corrected before anaesthesia. Fluid loss may result from a variety of causes, including blood loss, burns, vomiting, diarrhoea, loss through fistulae, loss into the gut (ileus), deficient intake, excessive loss through the skin (especially in the extremes of age) and excessive urinary loss.
Briefly, the following symptoms and signs suggest dehydration:

- Thirst
- Dry mouth
- Diminished skin turgor
- Rapid pulse
- Decreased urine output
- In the later stages a fall in blood pressure
- Central venous pressure (CVP) if measured will be low
- A high BUN and a raised specific gravity of urine confirm the diagnosis

The appropriate fluid must be administered with a close watch on these parameters.

Electrolyte imbalance

Sodium and potassium imbalance especially must be corrected pre-operatively. A low potassium level can result in hypotension, arrhythmias and cardiac arrest. It can also result in skeletal and smooth muscle weakness and interfere with the action of relaxant drugs. A high potassium level is also associated with cardiac arrhythmias.

Fluid and electrolyte imbalance will be more common in patients for emergency surgery.

Smoking

This increases intra and post-operative morbidity due to associated bronchial exudation and bronchospasm. It should ideally be given up three days pre-operatively. However, cessation for even 24 hours pre-operatively reduces the morbidity.

Dental treatment

Should be carried out if necessary before any major surgery, for instance cardiac surgery.

SPECIAL PROBLEMS RELATED TO EMERGENCY SURGERY

In addition to the problems already listed, patients presenting for emergency surgery pose the problems of:

- The unfasted patient (full stomach).
- Hypovolaemia due to blood or fluid loss.

The unfasted patient (full stomach)

The dangers of vomiting or regurgitation under anaesthesia are discussed elsewhere. (See Chapter 46)

In considering the pre-operative measures we can take to prevent this complication, the following deserve mention:

- Postpone surgery for at least six hours. However, the gastric emptying time is usually prolonged in emergencies.
- The stomach may be emptied using a naso-gastric tube or orogastric tube of the largest possible bore.
- Sodium citrate (a non-particulate antacid) can be used pre-operatively to counteract the acidity of the gastric contents. It takes approx. 10 minutes to work and its effects last approx. 20 minutes.
- H_2 blockers i.e. ranitidine 300mg given at least 1 hour prior to surgery may also decrease the acidity of gastric contents. Metoclopramide 10mg given at the same time may benefit. Omeprazole 40mg given 2–6 hours prior to surgery also helps reduce gastric acid.
- Regional techniques may be the safest.
- Special rapid sequence induction techniques should be used in general anaesthesia unless an awake intubation is indicated.

Hypovolaemia

As already described fluid imbalance should be corrected as far as possible and cross matching of blood, if likely to be required, should be underway before anaesthesia commences.

FASTING GUIDELINES

The most recent well-researched fasting guidelines are as follows: -

For elective surgery

No solids after midnight or for 6 hours before surgery but clear fluids can be allowed up to 2 hours before surgery. (clear fluids are: water, clear fruit juice, black tea / coffee).

For babies and small children breast milk may be allowed up to 4 hours before surgery and water until 2 hours before surgery.

For emergency surgery

The period of fasting will depend on the urgency of the procedure.

All emergency patients should be treated as potentially at risk of aspiration and anaesthetised using a rapid sequence induction.

Gastric emptying is delayed in the following situations: patients in labour, with head injuries or severe trauma and patients receiving drugs eg opiates.
Very ill patients have a delayed gastric emptying time.

Before the patient leaves the ward a senior nurse should:

- Check the identity of the patient
- Check the site and side of the operation
- Ensure the consent form is signed in accordance with the rules of the hospital
- It is good to weigh every patient At least every child under the age of 12 must be weighed to allow calculation of drug dosages
- Grossly underweight and overweight adults must also be weighed
- Ensure that fasting rules have been observed
- Remove lipstick, nail varnish, etc.
- Remove dentures, artificial limbs and artificial eyes
- Empty bladder
- Dress the patient in a linen gown with an identification label
- Give premedication if any

PREMEDICATION

The term **premedication** is used to describe the administration of drugs before operation, with the general aims of lessening anxiety and fear and contributing to the ease and safety of the anaesthetic. The term was first used in 1920.

PURPOSES OF PREMEDICATION

- To alleviate anxiety and fear
- To reduce the volume and acidity of gastric contents
- To reduce secretions especially salivary and bronchial
- To prevent undesirable reflexes, e.g. bradycardia
- To provide anti–asthma and anti–allergy therapy if relevant
- To provide pre and post operative analgesia
- To reduce post-operative nausea and vomiting
- To facilitate induction and reduce the dose of anaesthetic required

DRUGS USED FOR PREMEDICATION

Narcotic, sedative and tranquillising drugs

Narcotic analgesics, e.g. papaveretum, pethidine, morphine. These drugs are excellent agents for premedication.

Sedative drugs, e.g. barbiturates.

Tranquillisers, e.g. diazepam, midazolam, phenothiazines, butyrophenones.

Sedative drugs and tranquillisers are useful when narcotics are contraindicated. They are useful too when patients are not in pain pre-operatively and when they are to have a regional anaesthetic.
The tranquilliser group of drugs is especially useful for patients who are emotionally disturbed before the anaesthetic.

Anticholinergic drugs

Atropine, hyoscine (Scopolamine) and glycopyrrolate are commonly used. They are especially useful when ether is used.

Atropine has a more pronounced effect on the heart (tachycardia).

Hyoscine is a more effective drying agent. It also has a central sedative effect and is a good antiemetic but may cause confusion and restlessness in the elderly patient, so is then used in smaller doses.

Glycopyrrolate has the best drying action and the least central effect.

Dosage of atropine = 300–600 micrograms IV/IM
Dosage of hyoscine = 200–600 micrograms IV/IM/SC
Dosage of glycopyrrolate = 200–400 micrograms IV

Antacid drugs

These have already been discussed in connection with the management of an unfasted patient. See above.

Anti-emetics

Phenothiazines, butyrrophenones, antihistamines, metoclopramide, hyoscine and $5HT_3$ antagonists e.g. ondansetron, can be used to help reduce incidence of post-operative nausea and vomiting.

ROUTES OF ADMINISTRATION

Oral administration

This has become much more popular in recent years. It has the advantage of avoiding an injection and is often favoured by paediatric anaesthetists. It is suitable where only a sedative, tranquilliser or analgesic tablet is required. Its disadvantage is that absorption is slow and not dependable and the tablets or syrup have to be given 1-2 hours pre-operatively. Tablets may be taken with a sip of water.

Intramuscular injection

Premedicant drugs such as narcotic analgesics and anticholinergic drugs are usually given intramuscularly. IM injection is also used when the patient cannot take anything by mouth e.g. intestinal obstruction or vomiting.

Intravenous injection

Occasionally premedication is given intravenously in the operating theatre before the induction of anaesthesia. It is given in the following situations:

- In patients requiring emergency surgery when time cannot be allowed for the 1M injection to take effect.
- In patients presenting for elective or routine surgery who for some reason have not been given premedication.
- In patients who have an intravenous infusion running and where it is decided to avoid an intramuscular injection.
- In the shocked patient where intramuscular absorption is slow. Often no premed is given here.
- In patients with a bleeding tendency where intramuscular injections may be associated with bruising.

Rectal administration

This route is rarely used. Patient acceptance is low.

No premedication

There is a place for no premedication in anaesthesia. Very ill and frail patients fall into this category.

CHOICE OF DRUGS

The choice and dose of drugs will depend on a variety of conditions.

- Age
- Sex
- Weight of patient
- Nature of surgery (long? painful?)
- Regional or general anaesthetic (relaxant with IPPV or spontaneous breathing)
- Degree of apprehension
- Anticipated post operative pain

Important points regarding choice of drugs

- Narcotics such as papaveretum, pethidine or morphine should be used (unless otherwise contraindicated) if the patient is in pain, e.g. from a fractured femur.
- Narcotics cause respiratory depression and should not be used in head injured patients unless facilities for pre and post-op long-term mechanical ventilation are available.
- A sedative premedication should be avoided in patients with raised intracranial pressure.
- Narcotics are avoided in Caesarean sections and operative obstetrics, e.g. forceps delivery. They can be given when the baby is born.
- Narcotic premedication is not used in children under the age of twelve months.
- Smaller doses of narcotics are used in
  - Poor risk patients from whatever cause
  - The elderly
  - The very young
  - Patients who should not be too drowsy at the end of the operation, e.g. patients who have had surgery on the upper airway, those with full stomachs, diabetic patients in whom prolonged drowsiness may mask a hyper or hypoglycaemic state.
- Papaveretum is given in a dose of 0.3 mg/kg.
  Pethidine is given in a dose of 1 - 1.5 mg/kg.
  Papaveretum + hyoscine is an excellent combination, given 1-1.5 hours pre-operatively.
  Pethidine and atropine are prescribed 30-45 minutes pre-operatively.

- In asthmatics a good premedication is a combination of pethidine, promethazine and atropine. A salbutamol nebule 30 minutes prior to surgery is often beneficial.
- In regional anaesthesia tranquillisers such as midazolam or diazepam may be used.
  A narcotic such as pethidine or papaveretum may be used to provide analgesia so that the patient will be comfortable when the local anaesthetic begins to wear off.
- Chlorpromazine (Largactil) or other benzodiazepines are good drugs to use in premedicating patients receiving ketamine anaesthesia. The hypertension and hallucinations that follow ketamine are minimised.

In conclusion, a variety of drugs are available for premedication. The drugs selected must be tailored to the patient's needs.

CHAPTER 8

THE AIRWAY AND ITS MAINTENANCE

Outline:

The airway: definition

Measures available to maintain a clear airway

The obstructed airway: diagnosis and treatment

Anaesthesia for patients with an obstructed airway

THE AIRWAY

The airway is the passage that conveys the inspiratory gases from the atmosphere to the alveoli in the lungs. It therefore includes the nasal cavities, pharynx, larynx, trachea, bronchi, bronchioles and alveoli. The airway is a major lifeline. An open, clear airway is essential for life.

MEASURES AVAILABLE TO MAINTAIN A CLEAR AIRWAY

Position

By positioning the patient correctly it is possible to minimise the risk of the tongue falling back and obstructing the pharynx.

- The left lateral position is the best for the unconscious patient.
- If the patient is placed supine the head must be extended so that the nostrils point upwards, the chin is pulled forward and the angles of the mandibles lifted upwards.

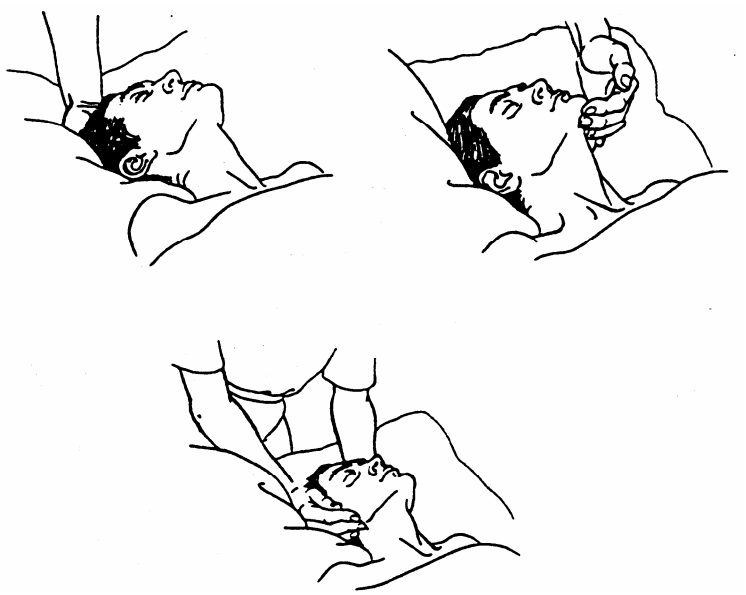
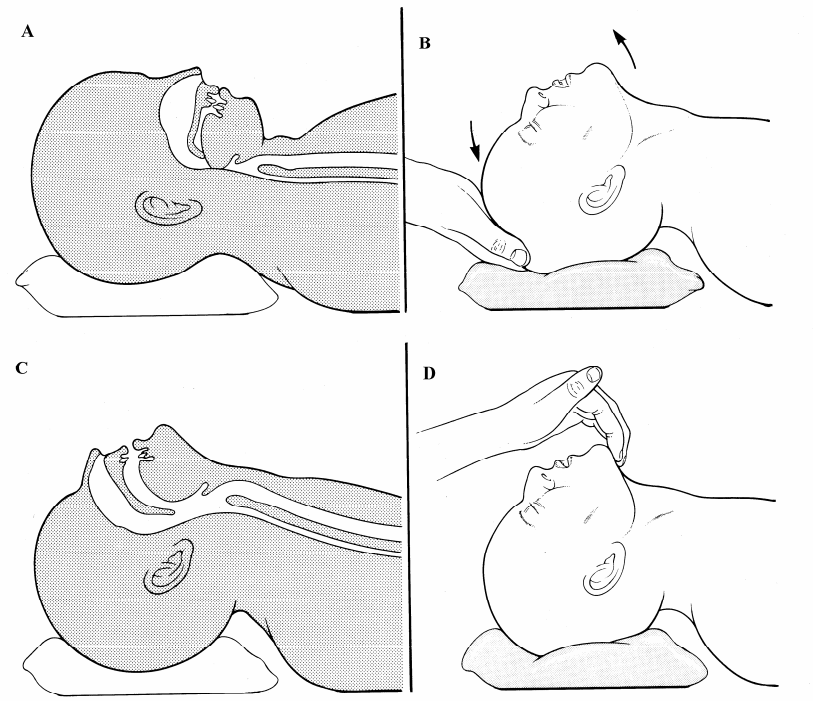


Fig. 8.1 Head extension with chin raised and the angles of the mandible lifted forwards



A - Mechanism of airway obstruction when prone. B, C - Exten tion of the Head.
D - Maintaining a clear airway by supporting the jaw

Fig. 8.2

Suction

Aspiration of the secretions:

- Oropharyngeal/nasopharyngeal suction
- Orotracheal/nasotracheal suction

Pharyngeal airway

This is a rigid or semi–rigid tube made of rubber or plastic which fits the upper airway. The pharyngeal airway is for short-term use and must be replaced by endotracheal intubation if the patient's ability to maintain a patent airway is in doubt.

- **Oropharyngeal airway**
  The airway comes in various sizes. It conforms to the curvature of the palate and extends from the lips to the pharynx. The head must be extended. A flange fits outside the lips. The airway displaces the tongue anteriorly and allows the patient to breathe through it and around it. Injury to teeth can result.
- **Nasopharyngeal airway**
  Made of soft rubber or latex. Extends from the nostril, through the pharynx to just below the base of the tongue.

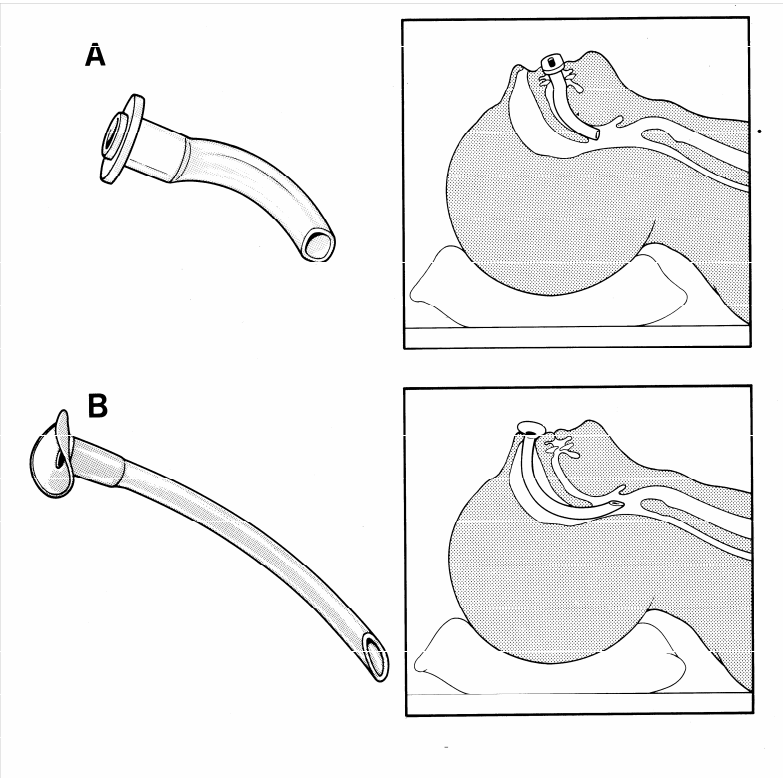


Fig. 8.3 A Oropharyngeal airway B Nasopharyngeal airway

Face mask and self-inflating bag

If the above measures do not succeed in securing a clear airway or if the patient's respiratory effort is inadequate, a more invasive method will be required. Respiration can be assisted temporarily by the use of a facemask with a self-inflating bag or anaesthetic bellows until equipment for LMA insertion or endotracheal intubation is assembled.

Laryngeal Mask Airway

Endotracheal tube (ETT)

Tracheostomy

These topics will be extensively dealt with in Chapter 9.

THE OBSTRUCTED AIRWAY

Signs and symptoms depend on whether the patient is breathing spontaneously or being ventilated mechanically.
The signs and symptoms fall into two categories:

Mechanical signs

Spontaneous respiration

- Noisy breathing. ***Remember that noisy breathing always indicates obstruction but an obstructed airway is not always noisy.*** The airway may be almost completely obstructed and yet respiration may be quiet.
- Diminished chest movement.
- Retraction of the chest wall and the infra and supra–clavicular spaces.
- Excessive abdominal movement.
- Paradoxical respiration: the natural heave of the chest and abdomen are replaced by the indrawing of the chest and the pushing outwards of the abdominal wall.
- Nostrils may be dilated.
- Breath sounds may be absent on auscultation with a stethoscope.

Controlled ventilation

- Increased pressure is required to ventilate the patient.
- Chest movements are diminished.
- Breath sounds are soft or absent.

Signs of oxygen lack

- Cyanosis. This may be absent if the Hb is less than 5g %.
- Respiratory rate increases at first.
- Pulse. Initially there is a rise.
- Blood pressure. At first this rises and then falls.

The use of oximeters and capnographs record changes in blood O_2 and CO_2.

The different causes of airway obstruction and their management are outlined in the following table

Causes of airway obstruction	Management of obstruction
Apparatus	
Kinking: compression or obstruction in the hose	The patient must be removed from the ventilator and ventilated by hand If a face mask held by a harness is in use, the harness must be removed and the mask held by the anaesthetist.
Valves improperly functioning	Check the hoses and valves for defects.
Pharynx	
The tongue falling back on the posterior pharyngeal wall	Extend the head so that the chin points upwards. Lift the angles of the jaw upwards. If the anaesthetic is sufficiently deep an oropharyngeal airway is inserted. If the airway is obstructed and the jaws are clenched, a mouth gag is used to force open the jaws, the tongue is pulled forward and the oropharyngeal airway is inserted. Occasionally, pharyngeal obstruction can be overcome only by endotracheal intubation or use of a laryngeal mask.
Swelling of the base of the tongue or the floor of the mouth (inflammatory, traumatic, neoplastic).	Endotracheal intubation or tracheostomy may be necessary.
Foreign material, such as Food Dentures Pack Secretions	Suction or manual removal of the foreign matter.

Causes of airway obstruction	Management of obstruction

Larynx	
	The treatment of laryngeal spasm
Laryngeal spasm which may be ***Direct:*** due to stimulation of the larynx by secretions, vomitus, anaesthetic vapours, laryngoscope blades or airways. **or** ***Reflex:*** under light anaesthesia, surgical stimulation can produce laryngeal spasm.	Stop surgical stimulation and deepen anaesthesia. Institute CPAP (continuous positive airway pressure) with an FiO_2 of 100%. Maximise efforts to open the airway with jaw thrust, head tilt, oral or nasal airway. If vomitus or secretions are present, use suction. A small dose i.e. 10-20mg Scoline (in the adult) will correct laryngospasm. If this fails then the diagnosis is not laryngospasm. Consider inadequate airway (try Guedel airway), incorrectly placed laryngeal mask (try a bigger size), anaphylaxis etc. If these fail, 100% O_2 is administered to the patient by mask. The anaesthetic gases and vapours must be turned off. If there is still no improvement, then give 100mg suxamethonium (in an adult patient) and ventilate and intubate the patient if necessary. Surgical airway if this fails. Laryngeal spasm can recur when the patient is extubated. For the best results, start treatment at the onset of laryngeal spasm. The hypoxia associated with laryngeal spasm, if persistent, can cause a cardiac arrest.

Causes of airway obstruction	Management of obstruction

Larynx (cont)	
Laryngeal stenosis Laryngeal tumour Vocal cord injury due to involvement of the recurrent laryngeal nerve.	Endotracheal intubation or tracheostomy may be necessary.
Foreign material in the larynx: Food Vomitus Dentures Secretions	Suction and possibly bronchoscopy
Laryngeal oedema This may occur in children within 6 hours of extubation. The usual signs of respiratory obstruction are present in addition to stridor and a croupy cough.	Give oxygen via face mask. Adrenaline nebules; (1mg of Adrenaline+ 1ml Saline) Give continuously until improvement up to 5mgs Give Dexamethasone 4 mg. If the child does not improve and is still hypoxic you will need to consider intubation or tracheostomy.

Trachea	
Tracheal obstruction could be due to: stenosis tumours	Tracheostomy may be necessary.
Foreign material such as dentures vomitus or food blood	Suction Bronchoscopy
Pressure from outside: Inflammatory and neoplastic lesions. Thyroid gland enlargement.	Endotracheal intubation or tracheostomy may be needed.
Haematoma formation after thyroidectomy.	Evacuation of haematoma.

Causes of airway obstruction	Management of obstruction

Bronchi and bronchioles	
Bronchospasm	Treatment
This may occur in Asthmatics Heavy smokers Acute respiratory infections Following vomiting and aspiration	If the patient is to be anaesthetised, check for predisposing factors, e.g. thiopentone.
Under anaesthesia in susceptible patients, e.g. certain drugs such as thiopentone and prostigmine produce bronchospasm. The presence of an endotracheal tube can bring on an attack Surgical stimulation under light anaesthesia.	Deepen anaesthesia using a drug that produces bronchodilatation e.g. ether or halothane. Aminophylline 250 mg slow IV Hydrocortisone 100 mg IV If patient is not anaesthetised, adrenaline 0.5 ml 1:1000 solution SC, aminophylline, hydrocortisone, salbutamol.

Other causes	
As part of an anaphylactic reaction.	Salbutamol via nebuliser or aerosol or slow IV, 250 micrograms (the adult dose).
Obstruction of the endotracheal tube.	See notes on endotracheal intubation Chapter 9

ANAESTHESIA FOR THE PATIENT WITH AN OBSTRUCTED AIRWAY

Patients for surgery may present with an obstructed airway, e.g. patients for thyroidectomy, or with tumours or abscesses in the pharynx or neck. (See Chapter 26).

Principles

If there is pre-operative obstruction, the airway must first be secured before anaesthesia is commenced.

Either intubate the patient under local anaesthesia

or perform tracheostomy first, then anaesthetise.

If there is no pre-operative obstruction but obstruction is likely when the patient is anaesthetised, **do not** use a relaxant for intubation. A relaxant must be used only when the anaesthetist is certain that the patient can be ventilated. With possible airway obstruction this cannot be guaranteed. The patient must be intubated awake or under a deep inhalational anaesthetic.

Have the following available:

- Laryngeal mask (if available)
- Failed intubation drill 'set-up'
- Facilities for a tracheostomy
- Minitrach set (Portex) is useful, (if available)

See Chapter 21 (Obstetric anaesthesia) for full description + failed intubation algorithm.

CHAPTER 9

THE LARYNGEAL MASK AIRWAY, ENDOTRACHEAL INTUBATION and TRACHEOSTOMY

Outline:

Laryngeal Mask Airway

Equipment for intubation

Indications for intubation

Techniques of intubation

Packing the pharynx

Suction through an endotracheal tube

Extubation

Dangers of endotracheal anaesthesia

What to do when intubation fails

Diameters and lengths of endotracheal tubes

Tracheostomy

LARYNGEAL MASK AIRWAY

The laryngeal mask (LMA) was introduced in 1985 and is intermediate in design and functions between the oropharyngeal airway and a tracheal tube.

Construction

The LMA is made from silicone. It withstands repeated autoclaving. The manufacturers recommend the mask should be discarded after 40 uses. Many have been autoclaved more than 40 times.

The LMA consists of two sections

- An oval mask surrounded by a cuff which inflates to 1.5cm width. The cuff is inflated through a pilot tube which has a soft pilot balloon. The cuff provides an oval seal around the tube.
- A tube delivering gases to the mask.

There are various sizes of laryngeal mask:	***cuff inflation***
Size 1 for use in infants up to 5 kg	4ml
Size 1 ½ for use in infants of 5-10 kg	7ml
Size 2 for use in infants of 10- 20 kg	10ml
Size 2 ½ for use in children of 20-30 kg	14ml
Size 3 for use in children and small adults of 30-50kg	20ml
Size 4 for small or normal-sized adults.	30ml
Size 5 for normal or large sized adults	40ml

The mask is sterilised by autoclaving at 134° C. Deflate the cuff before autoclaving.

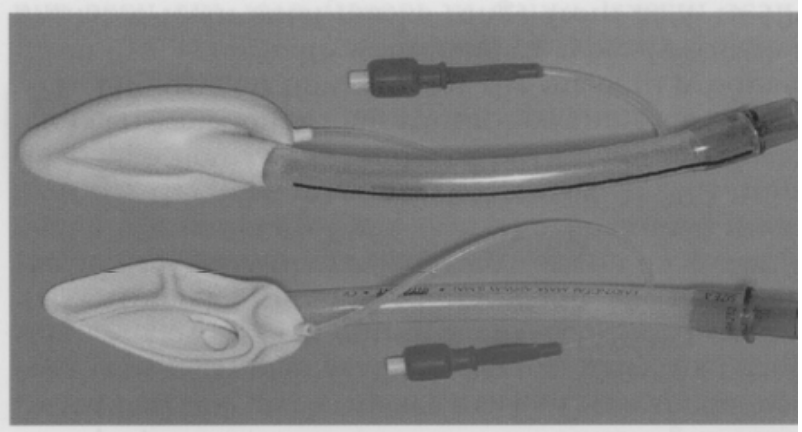


Fig. 9.1 The Laryngeal Mask Airway

Upper – with cuff inflated **Lower – with cuff deflated**

Features of the Laryngeal Mask

- Insertion is rapid and atraumatic.
- Laryngoscopy is unnecessary.
- Insertion with flexed neck is possible.
- The operator can work from the front or side of the patient.
- Permits a lighter plane of GA, because it is less stimulating in situ. (It requires a deep plane of anaesthesia for initial insertion).
- Accidental intubation of the oesophagus or bronchus is not possible.
- It frees the anaesthetist's hands. Mandibular support is not necessary.
- It enables the use of capnography with spontaneous respiration.
- It is useful in difficult intubations.
- It enables early postoperative recovery of cough reflex.

Reinforced Laryngeal Mask

The reinforced laryngeal mask has spiral strengthening throughout the length of the tube designed to prevent kinking. It is flexible and can be used where the anaesthetist has no ready intraoperative access to the airway or where the head or larynx has to be manipulated during surgery e.g. in ophthalmological or some types of dental surgery.
Re-inforced tubes are "floppy" and may have to be inserted using an introducer.

Method of insertion

- Test the cuff by inflating with air, then deflate it.
- Lubricate the back of the cuff with lignocaine jelly before insertion.
- Insert when the patient is deeply anaesthetised (therefore induce with thiopentone, follow this with an inhalational agent, e.g. halothane or enflurane, before attempting to insert the LMA). It is also very suitable for use with a propofol induction (if it is available) together with midazolam and fentanyl.
- Maintain the normal intubation position, i.e." sniffing position".
- Assistant pulls the lower jaw down.
- Grasp the tube near the mask end.
- Left hand extends head, right hand inserts the mask.
- The aperture of the laryngeal mask faces forward and the mask tip should be pressed back against the hard palate while introducing the mask. This must be done under direct vision. Once the mask reaches the posterior pharyngeal wall, grasp the top end of the mask, maintaining a downward pressure on it. Next straighten the tube and push the mask down with one rapid movement. The position of the black line must face the upper lip.

- Inflate the cuff with air. For recommended volumes see page 108. As the cuff inflates release grasp on the mask and allow the mask to find its own position.

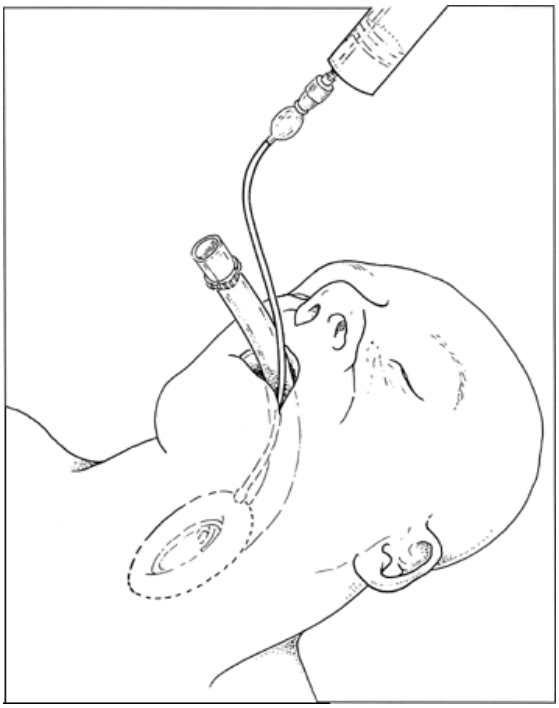


Fig 9.2 Laryngeal mask in place

- Then connect the mask to the gas supply. An oropharyngeal airway at the corner of the mouth or a bite block between the teeth at one side can be used to prevent occlusion of the tube.
  Check that the airway is clear. It can be helpful to listen with a stethoscope over the trachea before and after insertion. Clear breath sounds confirm a good placement. Airway obstruction is a hazard that must always be borne in mind and it can develop during anaesthesia even when initial placement of the tube appears correct.

After surgery leave the laryngeal mask in place until the patient responds to verbal commands. The cuff does not require to be deflated before removal as the inflated cuff helps to remove secretions which may have collected above it. Patients, as they waken, can be encouraged to remove their own laryngeal masks.

EQUIPMENT FOR ENDOTRACHEAL INTUBATION

Before the days of muscle relaxants blind nasal intubation was very popular. Today endotracheal tubes are passed through the nose or mouth into the trachea so that the tip lies below the larynx but well above (3cm) the carina.

Endotracheal tubes can be passed into the trachea:

- Without anaesthetic, e.g. in very ill patients.
- With topical or local analgesia, especially if a difficult intubation is expected.
- Under inhalational anaesthesia (with spontaneous respiration).
- Under general anaesthetic with muscle relaxation.

The anaesthetic is then continued via the endotracheal tube.

Tubes

Early endotracheal tubes (the Magill tube and the Oxford tube) were made of red rubber. The Magill tube came in an oral and nasal version with or without an inflatable cuff. The Oxford tube was a short right-angled preformed oral tube. Red rubber is irritant at the point of contact and these tubes would only be used now if there were no modern PVC tubes available. There are several different styles and makes of endotracheal tubes but the most common groups are:

The standard transparent PVC curved tubes based on the original Magill red rubber tube. These tubes come in oral and nasal versions with or without a cuff. Both versions are very similar except that in a nasal tube the pilot balloon for the cuff comes off more proximally and care should be taken if the tube has to be shortened. These tubes have a sideways facing bevelled tip to facilitate passage through the cords. The size of the tube is marked on the wall together with the outside diameter and a scale measuring the distance from the tip in centimetres.

The inflatable cuff sits immediately below the cords. Its purpose is to seal the trachea in order to:

- Prevent or minimise gas leakage during IPPV.
- Prevent foreign material entering the tracheo-bronchial tree, e.g. gastric contents, blood, or secretions from the upper respiratory tract.

The cuff is inflated by injecting air from a syringe into a small tube running down the side of the endotracheal tube. A small pilot balloon at the upper end of this tube indicates the degree of inflation of the cuff. Always test the cuff by inflating it before use. It must be inflated until no further leakage of gases around the endotracheal tube, with ventilation (IPPV), is heard.

Dangers of over inflation

- The cuff may rupture.
- Herniation of the cuff and occlusion of the tube at the lower end by a distended cuff.
- Excessive pressure on tracheal mucosa, which may lead to ulceration, fibrosis and stricture. This is especially dangerous in children, as they have narrow air passages.

Remember that the cuff is not a foolproof device to prevent aspiration of foreign matter.
If excessive post-nasal bleeding is expected, pharyngeal packing should also be employed. (See below)

Preformed tubes (cuffed or plain) e.g. RAE tubes, are suitable for procedures requiring access to the airway, face and head. The preformed tube is less likely to kink and its shape allows the tube to be directed away from the site of operation. This avoids the use of bulky connectors close to the head. They have the disadvantage that they are of fixed length and cannot be adjusted to suit the variation in patient size. It is also difficult to pass a suction catheter through this type of tube.

Armoured or reinforced tubes have a spiral reinforcement within the wall to prevent kinking and are used where the head or larynx may have to be manipulated during surgery e.g. facio-maxillary and thyroid surgery. They are also commonly used in procedures where the anaesthetist has no access to the airway during surgery. Armoured tubes cannot be cut and as they are very flexible they have to be inserted using an introducer.

There are some ***other specialised tubes*** for use during ENT surgery e.g. laryngoscopy tubes, tubes safe for using in laser treatment and for thoracic surgery (endobronchial double lumen tubes) to allow single lung ventilation. These will be discussed in the relevant chapters.

Connectors

These are inserted into the upper end of the endotracheal tube and then into a catheter mount which connects the tube to the anaesthetic machine. Modern plastic tubes come complete with plastic connectors.

Magill oral and nasal metal connectors are available.
They are curved and offer less resistance to gas flow. The chief disadvantage is that they have no provision for suction.

Portex endotracheal adaptors and taper tip connectors:
There are two types of adaptors, one right angled with a swivel, the other straight. They fit all taper tip connectors from 2.5 to 11mm. They are made of plastic material and can be autoclaved.

Cobb's elbow and taper tips
The taper tips have one end which fits into tubes from 2-12mm. The other is tapered and is standard for all sizes. It fits into the Cobb's elbow. This is right-angled and fits into the catheter mount. An attached rubber stopper can be removed and a suction tube inserted into the endotracheal tube and beyond.

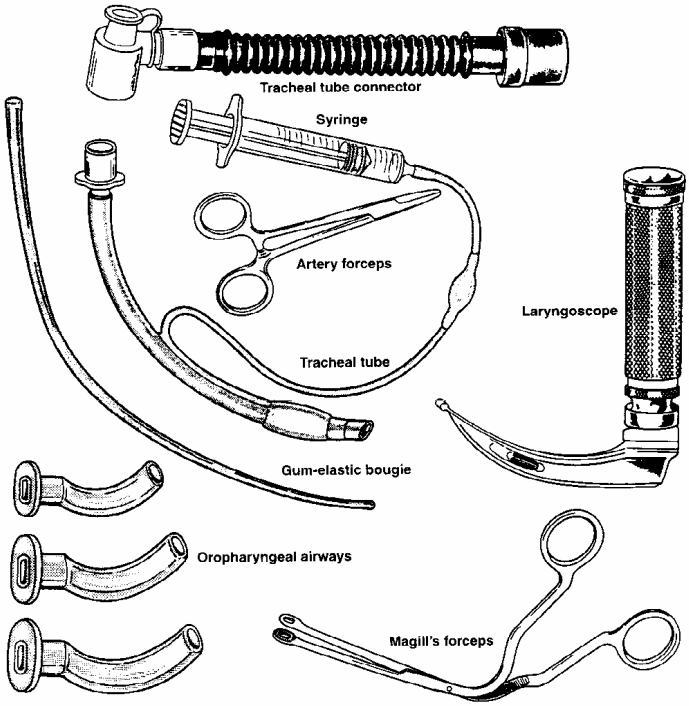


Fig 9.3 Some of the equipment needed for endotracheal intubation

Catheter mounts

These connect the endotracheal tube and the anaesthetic circuit. They may be made of plain (usually black) rubber or plastic and are usually corrugated, which is useful when the patient's head has to be moved during the procedure or the patient has to be placed in an unusual position. Kinking is minimised and traction on the tube is less likely.
Plastic catheter mounts are now available with angle piece and suction port all in one.

Laryngoscopes

There are many designs but basically only two main types: curved blade and straight blade. On some laryngoscopes curved and straight blades are interchangeable.

Curved blade laryngoscope (e.g. MacIntosh)
This is designed not to touch the posterior aspect of the epiglottis which is supplied by the vagus nerve. The blade is passed over the tongue and inserted in front of the epiglottis (in the vallecula). It lifts the base of the tongue forwards and with it the epiglottis so that the cords can be seen.

Advantage of the curved blade:

- It can be used in a lighter plane of anaesthesia with less danger of precipitating laryngeal spasm or cardiac arrhythmias, although these complications may still occur.
- The curved blade does not hide the cords from view when you are intubating. It should be inserted from the right side of the mouth, moving the tongue to the left.

Straight blade laryngoscope
This may be right-angled or acute-angled. It is useful for children and for nasal intubation. The blade is designed to lift the epiglottis up, exposing the cords to view.

McCoy laryngoscope

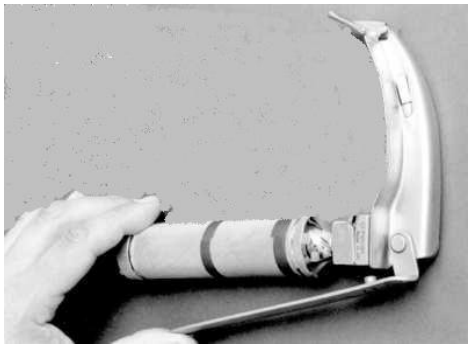


Fig 9.4
McCoy laryngoscope

This is a specialised laryngoscope designed for difficult intubation. It is similar in shape to the MacIntosh laryngoscope but has a hinged tip controlled by a lever on the handle. The tip is placed in the vallecula in the normal way and when the hinged tip is elevated it pulls the soft tissue and epiglottis forward without putting extra pressure on the pharyngeal tissues.

Fibreoptic laryngoscopes (where available) provide excellent conditions for intubation.

INDICATIONS FOR ENDOTRACHEAL INTUBATION

Non-Surgical

- To maintain a clear airway
- To protect the airway from blood, secretions etc
- To control respiration, IPPV
- For suction of tracheobronchial secretions
- To reduce dead space
- Unconscious patients needing long term intubation and ventilation (use an oral endotracheal tube as there is less risk of sinusitis which can occur with nasal tubes)

Surgical

- If a clear airway cannot be otherwise maintained
- Unfasted patient (full stomach)
- Surgery of head, neck and airway
- Thoracic surgery: where controlled respiration is essential
- Upper abdominal surgery
- Surgery performed in abnormal positions, e.g. lateral or prone, where a clear airway cannot be guaranteed
- Procedures which take a long time
- Very ill patients

Contraindications (relative)

- Acute laryngitis
- Open pulmonary TB
- Known hypersensitivity to red rubber or latex tubes

Indications for nasal intubation

- To give the surgeon access to the patient's mouth or pharynx.
- When anatomical or pathological problems make laryngoscopy very difficult or impossible, e.g. inability to open the mouth, flex the neck, extend the head. A nasal tube is better tolerated for long-term intubation.

Contraindications to nasal intubation

- In patients with head injuries especially to the base of the skull
- Blocked nasal passages e.g. polyps, large adenoids
- History of severe nose-bleeds or blood clotting abnormalities

TECHNIQUES OF INTUBATION

Standard Techniques

Oral	direct vision
Nasal	direct vision

Special Techniques

Blind nasal	routine method
Awake intubation	
Difficult intubations	

The endotracheal tube can be inserted through the mouth (oral) or through the nose (nasal). Nasal intubation can be done under direct vision (using a laryngoscope) or blind. We will consider these techniques in more detail.

Standard techniques

- **Oral, with direct vision**

Anaesthesia for intubation may be

- awake (no anaesthesia)
- local
- general
  - spontaneous respiration
  - relaxant

Before intubation, do the following with care:

- Observe the patient noting the shape of the jaws, condition of teeth and any other anatomical and pathological abnormalities.
- Check that all equipment necessary for intubation is available: tubes, connections, adaptors, anaesthetic machine and circuit, suction apparatus, laryngoscope with functioning light, a 10ml syringe for inflating the cuff and artery forceps for clamping when the cuff is inflated, local anaesthetic spray and lubricant and an ETT introducer.
- Oxygenate the patient.
- Anaesthetise the patient with adequate quantities of both hypnotic and muscle relaxant to allow adequate time for intubation.

Technique using the curved blade laryngoscope.

- Put the patient in the correct position: neck flexed and head extended at atlanto-occipital joint.
- The right hand separates lips and opens mouth.

– The left hand holds the laryngoscope; the blade is introduced from the right corner of the mouth along the dorsum of the tongue, pushing the tongue to the left. Locate the uvula and the base of the epiglottis. Slide the tip of the laryngoscope blade in front of the epiglottis, between the epiglottis and the base of the tongue. Then lift the laryngoscope up towards the roof and forwards towards the patient's feet.

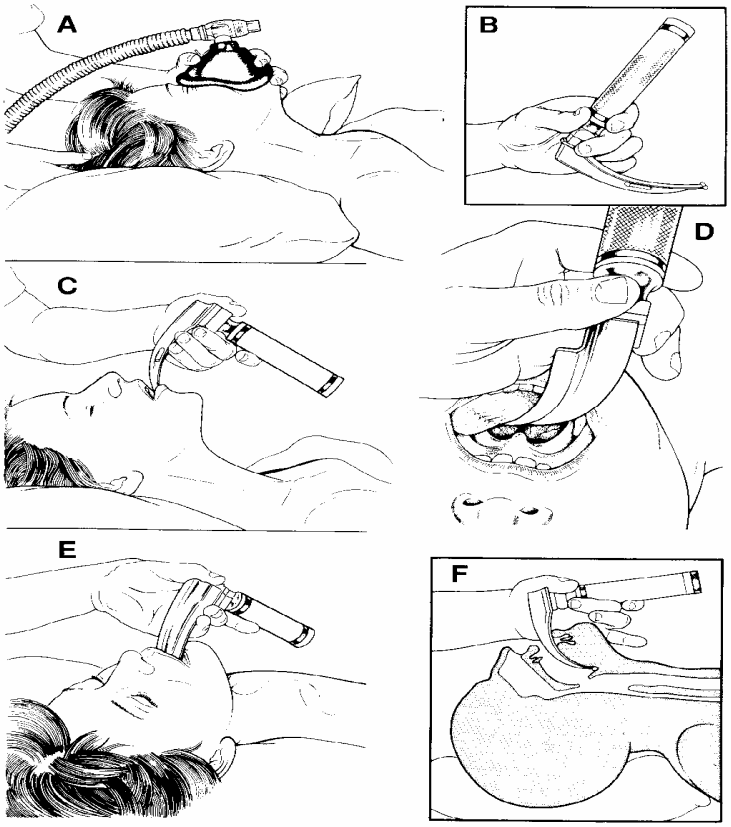


Fig 9.5 Technique of endotracheal intubation

– External pressure on the thyroid cartilage may bring the larynx into view. Look for the cords and when they can be seen clearly, pass the tube (which is held in the right hand) between the cords. The concavity of the tube should face anteriorly, (i.e. face upwards) with the patient lying supine.

Never use force to introduce the tube. **If the cords are in spasm do not attempt intubation**. Instead, withdraw the laryngoscope and if the patient is breathing spontaneously, deepen the anaesthesia. If a muscle relaxant has been used, postpone intubation until it has taken full effect. If conditions are still unsatisfactory give more relaxant. The patient must be ventilated until fully paralysed. If intubation has taken more time than usual the patient may wake up and require more induction agent. The patient must be well oxygenated before you attempt intubation. This is extremely important. The anaesthetist must get used to noting the colour of the tongue and lips during this procedure and checking a pulse oximeter reading if available.

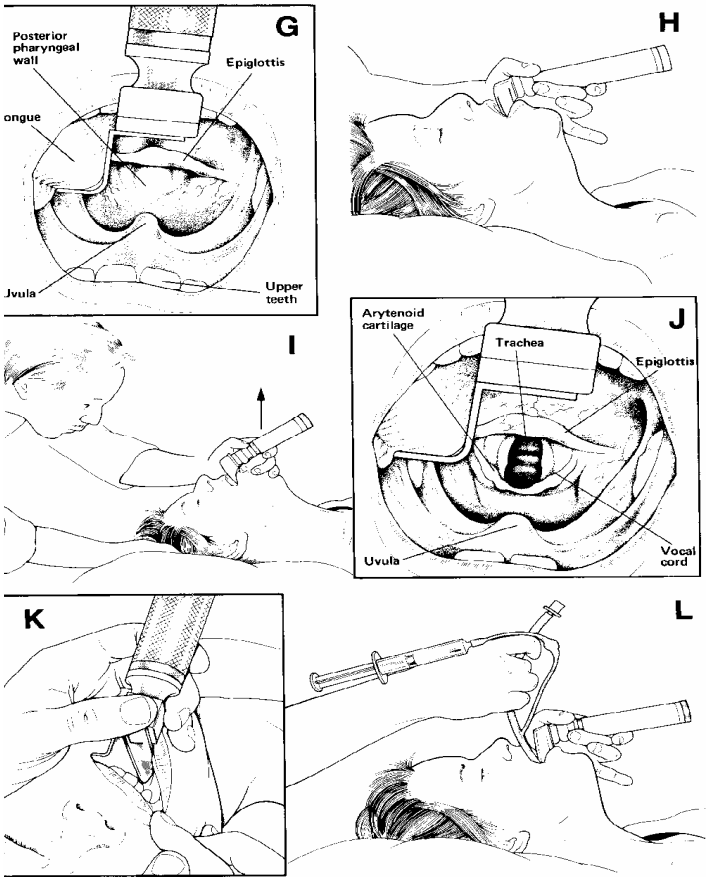


Fig 9.6 Technique of endotracheal intubation

- Once the tube is in the trachea, connect it to the anaesthetic circuit and ventilate the patient, checking by auscultation that it has not entered the oesophagus or the right main bronchus. Place the stethoscope over the lower lobe of the lungs in the mid axillary line. If the tube is in the oesophagus the sounds heard will not be breath sounds but gurgling noises. If the tube is too long, or pushed down too far, it may enter the right main bronchus and the breath sounds will be louder on the right side. They may even be absent on the left. If so, pull the tube back gently until the sounds are equal on both sides.
- Try to estimate the correct length of tube required before intubation. The average adult female will require a tube of 21cm and the average male 23cm. (See the table on page 129). Cut the tube accordingly and insert a connector into it before passing the tube. (It is difficult to get the connector in once the patient has been intubated.) The anaesthetist must be absolutely sure the tube is placed in the trachea and not the oesophagus. To observe the tube pass between the cords ensures this. The movements of the chest with respiration and with auscultation are helpful.
- **If in doubt** re-insert the laryngoscope and check the position of the tube. Ventilation with a mask and re-intubation may be necessary.

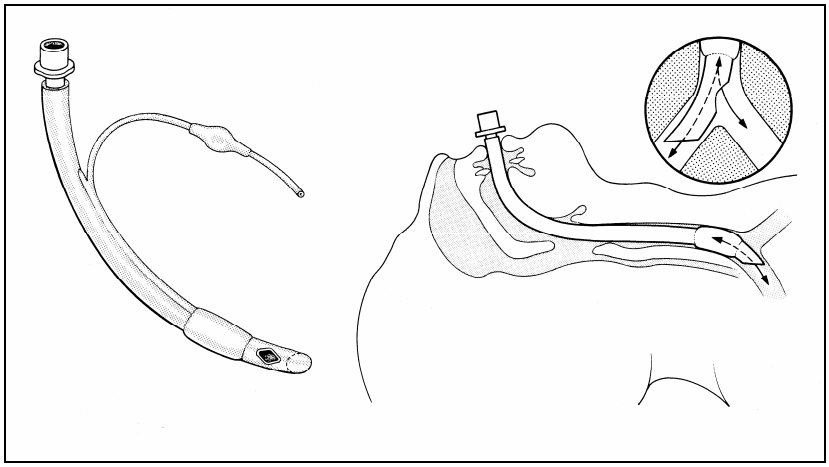


Fig 9.7 Accidental intubation of a bronchus

- **Nasal, with direct vision**

  Select the largest size tube which will pass through the nostril (average 7mm for women, 8 for men). Lubricate it and pass it gently through the nose into the pharynx. Then, with a laryngoscope, get a clear look at the cords. Guide the tube through the larynx with Magill's intubating forceps. The tip of the tube may touch the anterior laryngeal wall but lifting the head and flexing the neck will usually allow the tube to slide into the trachea. Alternatively, a straight blade can be used. Posteriorly directed pressure of the blade against the tube will have the same result.

Special Techniques

- **Blind nasal intubation: routine method**

(Always make sure facilities for a tracheostomy are readily available before attempting this technique).

  - Spontaneous respiration is employed. Induce anaesthesia with oxygen-nitrous oxide-halothane, (or enflurane or isoflurane) or ether. Make sure the patient is anaesthetised deeply enough to tolerate intubation.
  - Position the head as for oral intubation, i.e. the neck is flexed and the head is extended.
  - Lubricate a nasal tube and pass it into the pharynx.
  - Place your ear at the upper end of the tube and advance the tube. As it approaches the larynx the breath sounds become louder and as it enters the trachea the patient coughs. If the tube enters the oesophagus, breath sounds disappear and gurgling noises may be heard.
  - When the tube is in the trachea connect the tube to the anaesthetic circuit.

In well-equipped hospitals **a fibre-optic laryngoscope** is used for difficult intubations.

- **Awake intubation under local anaesthesia**

  Patients who have some form of upper respiratory obstruction and in whom a clear airway cannot be guaranteed are sometimes best intubated under local analgesia and then put to sleep and paralysed. It is very dangerous to paralyse such patients without first making quite sure that they can be ventilated.

Method:

- Explain carefully to the patient what you intend to do and that functions such as swallowing, speaking and coughing will temporarily be altered.
- Give the patient a local anaesthetic lozenge to suck (e.g.amethocaine).
- Dry the mouth as much as possible to prevent dilution of the local anaesthetic.
- Spray the pharynx and back of tongue with 4% lignocaine.
- Give a cricothyroid injection of local anaesthetic. (2ml of plain 4%.) Inject through the cricothyroid membrane during expiration.

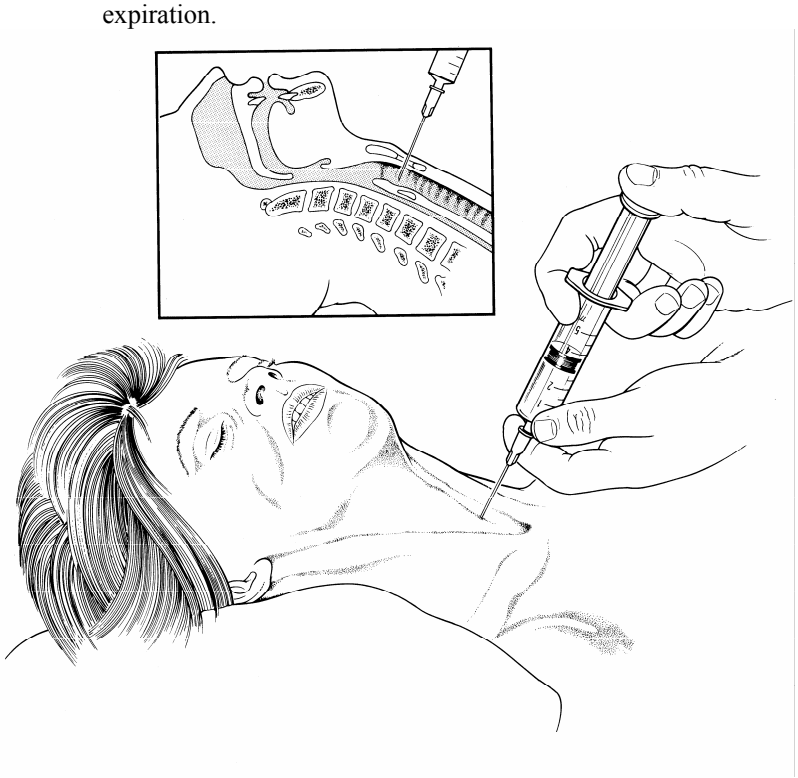


Fig 9.8 Cricothyroid injection of local anaesthetic

- Insert the laryngoscope, bring the cords under direct vision and spray.
- Pass the tube, smeared with lignocaine ointment, into the larynx.

Superior laryngeal nerve block

Blocking the superior laryngeal nerve can improve airway anaesthesia and some more experienced anaesthetists may do this. Locate the greater cornua of the hyoid bone and place index finger over the tip. Use a 5ml syringe containing 1% lignocaine with a 23g needle attached. Insert the needle into the notch of the thyroid cartilage and advance to a point under the cornua of the hyoid bone. Aspirate and then inject 2.5 ml of solution. Repeat this procedure on the opposite side.

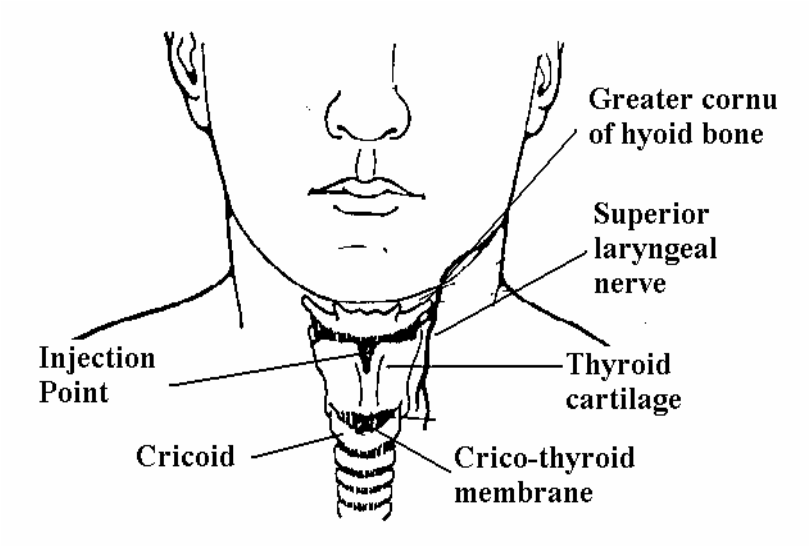


Fig 9.9 Superior Laryngeal nerve block

As discussed before, in well-equipped hospitals the use of a fibre-optic laryngoscope makes this technique easier for both the anaesthetist and the patient.

Examples of other types of patient who may have to be intubated awake are:

- Very young neonates in the first hours of life
- Very ill or moribund patients (children or adults)
- Those with high risk of aspiration, e.g. when the stomach contains food, blood or fluid which cannot be emptied satisfactorily
- Those with laryngeal obstruction

Summary of steps for awake intubation under local anaesthesia

	Dose in mgs
Spray the mouth, pharynx and epiglottis using a laryngoscope with 10% lignocaine spray (one puff usually gives 10 mg. Read the instructions).	100 mg (approx)
Nebulise 4% topical lignocaine 2 ml	80 mg
Superior laryngeal nerve block (optional) 1% plain 2 ml bilateral to the hyoid bone.	40 mg
Crico–thyroid puncture 4% topical lignocaine 2 ml.	80 mg

Remember that the total dose of plain lignocaine should not exceed 3mg/kg, so adjust the technique and volumes accordingly.

DIFFICULT INTUBATIONS

Difficult intubations may occur unexpectedly but careful preoperative examination will usually help detect them. Therefore, watch out for anatomical and pathological problems which will make intubation difficult.

Anatomical problems

- Short neck and bull neck
- Receding chin
- Prominent "Adam's apple" (thyroid cartilage)
- Protruding upper incisor teeth
- High arched palate

Pathological problems

- Tumours of mouth and pharynx
- Inability to flex neck and extend head, perhaps as a result of cervical spine fracture or arthritis
- Inability to open mouth, as in arthritis of the temporomandibular joints and trismus
- Burn contractures in the front of the neck
- Injuries to airways

WHAT TO DO WHEN INTUBATION FAILS
Every anaesthetist should have a plan of action when faced with failure to intubate a patient who is anaesthetised and paralysed.

This is discussed in detail in Obstetric anaesthesia in Chapter 21

The view obtained at **laryngoscopy** has been **graded** as follows

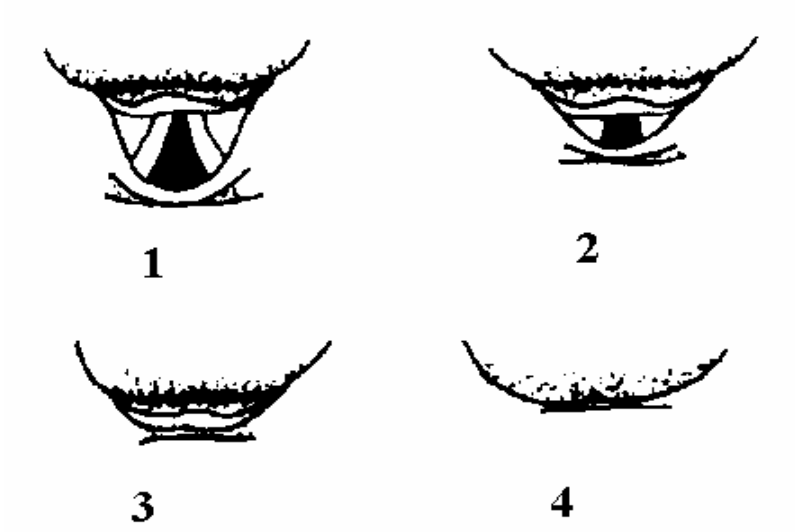


Fig 9.10 Classification of Laryngoscopy views

Grade	**1**	Most of the glottis is visible.
	2	Only the posterior extremity of the glottis is visible. Pressure on the neck improves exposure.
	3	No part of the glottis is visible but the epiglottis can be seen. This can cause severe difficulty.
	4	Not even the epiglottis can be seen. This is very rare, and may require complex methods of intubation.

Grades 3 and **4** indicate likely difficult intubations.

PACKING THE PHARYNX

Indications: Wherever bleeding from the upper airway is likely, e.g. following ENT operations. Dental extraction or restorations may present further problems associated with teeth or filling material entering the trachea or the larynx after extubation.

Material: A gauze bandage is best. Sometimes a dressing roll cut to a suitable length is used. *Do not use paper or similar material*. The pack should be soaked in saline or water and wrung out.

Dangers:

- The pack can be forgotten and left in
- Sore throat
- Infection

The pack should have tapes that remain outside the mouth and the surgeon and anaesthetist should both be vigilant when a pack is used.

SUCTION THROUGH AN ENDOTRACHEAL TUBE

This is done with a soft, small diameter rubber or plastic tube. It is not done routinely except in known chest disease with increased secretions and in thoracic surgery. It is used also when it is suspected that secretions are the cause of difficult lung inflation. If it is possible that the patient has aspirated gastric contents before intubation, suction may be necessary several times during the procedure.

EXTUBATION

This should be done only when the patient is breathing spontaneously and adequately and only during inspiration. It should be preceded and followed by suction. It is safer to extubate patients awake with them opening their eyes to command. After extubation the patient is made to breathe oxygen through the anaesthetic mask.

Ideally the patient should be extubated lying in the lateral position while still being monitored.

DANGERS OF ENDOTRACHEAL ANAESTHESIA

Failure to recognise oesophageal intubation is the most serious complication of endotracheal intubation.

Other complications are:

Mechanical

- Blockage of the tube by mucous, clot etc.
- Disconnection from anaesthetic circuit
- Kinking of the tube
- The patient may bite the tube
- Herniation of cuff
- The bevel may be forced against the tracheal wall
- Intubation of right main bronchus
- Kinking of the catheter mount

Trauma Rough or difficult manipulation of the laryngoscope or tube may damage lips, teeth, nose, throat and pharynx. Epistaxis may be troublesome, or teeth may lodge in the bronchus.

Inhalation of foreign matter from the stomach before the airway can be protected with a tube.

Infection may be spread from the oral cavity to the trachea and bronchial tree. It may be introduced by the equipment.

Cardiovascular disturbances

Arrhythmias caused by stimulation of the vagus nerve which supplies the lower surface of the epiglottis. Arrhythmias are less likely if anaesthesia is not too light and if both hypoxia and hypercarbia are avoided.

Arterial blood pressure often rises with intubation and is an indication that anaesthesia is not deep enough. It takes some time to settle afterwards.

Laryngeal spasm (see also under Airway obstruction Chapter 8)
This usually occurs in light anaesthesia if intubation is attempted, or some other stimulant factor is present, such as suction or mucous. It can be overcome by:

- Deepening the plane of anaesthesia

  or

- Using O_2 (100%) and a muscle relaxant (suxamethonium). It is less likely to occur if the pharynx is cleared gently and extubation is done smoothly.

Laryngeal oedema

This occurs more often in children, in whom it causes greater respiratory distress than in adults, because the child's air passages are still narrow. It can commence within an hour or two of extubation. Laryngeal oedema can be reduced by:

- Careful selection of the tube (done according to the formula in Chapter 20 Paediatric anaesthesia). A tube one size smaller must also be available for immediate use. The tube must not be forced into the larynx.
- Don't use a cuffed tube in a child.
- Be extremely gentle when intubating a child.
- Always have a "leak" around the tube in a child.
- Use only a sterile tube.
- Some procedures, e.g. cerebral tomography have to be done under general anaesthesia in children and involve frequent movement of the head with consequent mechanical stimulation of the cords. This frequently results in oedema, which must be expected. It is best not to use a red rubber tube.

Other laryngeal complications

- Granuloma of the larynx. This is slow to develop and follows an ulcer on the vocal cords or on the vocal process of the arytenoid cartilage. If the ulcer does not heal a granuloma develops and requires surgical removal.
- Bruising of the larynx may cause dysphonia and dysphagia. It usually clears up in a few days.
- Stretch injury of the vocal cord. This may be due to injury to the recurrent laryngeal nerve during acute extension of the head while intubating, or the injury may occur while extubating but the underlying cause is not known with certainty.

COMPARISON OF ENDOTRACHEAL TUBE AND LARYNGEAL MASK

Endotracheal tube	Laryngeal mask
Trauma esp. to teeth more likely	Less likely
Cardio vascular stimulation more likely	Less likely
Endo bronchial or oesophageal intubation possible	Not possible
Bronchospasm and bucking may be a problem	Bucking is a problem with light GA
Laryngoscopy and intubation may be difficult in certain anatomical and pathological situations.	Laryngeal mask insertion is easier under these conditions

ADVANTAGES	ADVANTAGES
More suitable when higher inflation pressures are called for, e.g. COAD	Does not require such deep anaesthesia
Head and neck surgery	No muscle relaxant required
Surgery in abnormal postures	Less incidence of sore throat
In cases of prolonged IPPV	**However**, it gives less protection against regurgitation and aspiration.
Palatal surgery, where the Boyle gag is used	Airway obstruction can still occur.
Full stomach- protected airway.	Failure of insertion < 5%.
Hiatus Hernia- protected airway.	

GUIDE TO ENDOTRACHEAL TUBE SIZE AND LENGTH

Age	ET size int. diam (mm)	ET length (cm) Oral	Nasal
0 – 3 months	3.0	10	
3 – 6 months	3.5	12	15
6 – 12 months	4.0	12	15
2 – 3 years	4.5	13	16
4 – 5 years	5.0	14	17
6 – 7 years	5.5	15	18
8 – 9 years	6.0	16	19
10 – 11 years	6.5	17	20
12 – 13 years	7.0	18	21
14 – 15 years	7.5	21	24
Adult female	8.0	21	24
Adult male	9.0	23	26

Points to note:

Below 8–10 years uncuffed tubes should be used.
It is always advisable to have prepared a tube one size smaller than calculated.
This table is a guide only. In many countries where children may be malnourished and smaller than normal for age / weight it may be necessary to choose a smaller tube.

TRACHEOSTOMY

Indications are similar to those for endotracheal intubation except that a tracheostomy is performed only if prolonged intubation is anticipated. A tracheostomy may also be performed when insertion of an endotracheal tube is not possible.

Indications

- Upper airway obstruction
- Tracheal toilet
- To prevent contamination of the upper airway
- To reduce dead space
- Prolonged intermittent positive pressure ventilation (IPPV)

Technique
Except in the case of sudden complete upper airway obstruction tracheostomy is not an emergency procedure. The patient must first be intubated and the operation must be performed in the theatre. The opening into the trachea is made between the 2nd and 3rd tracheal rings. A window is cut and a flap fashioned to aid the changing of the tubes.
Refer to surgical textbooks for more details.

Tracheostomy tubes

Silver tubes: These consist of

- An outer tube which sits in the trachea.
- An inner tube which can be removed for cleaning.
- An obturator or introducer which is placed in the outer tube when it is inserted into the trachea. The silver tubes are rigid, uncuffed and expensive but if no intensive care facilities are available, they are the best. An inner tube that can be regularly removed for cleaning reduces the chances of the tracheostomy getting obstructed.

Portex (PVC)
These are the most commonly used and are available with low pressure cuffs.

Rubber tubes
These are irritant to the trachea and should only be used if there is no alternative.

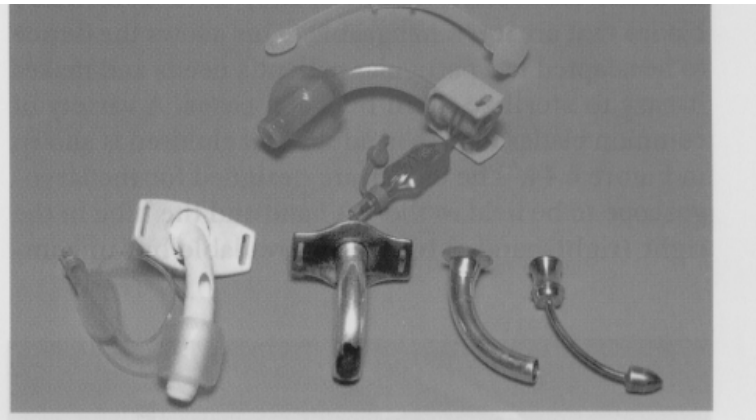


Fig 9.11 Tracheostomy tubes

Complications of tracheostomy

Early complications

- Haemorrhage
- Pneumothorax and mediastinal emphysema
- Airway obstruction
  - Occlusion of the cannula by secretions
  - Herniation of the cuff
  - Kinking
  - Displacement of the tube
  - Misplacement of the tube
  - Obstruction of the lower end of the tracheostomy tube against the wall of the trachea
  - Endobronchial intubation

Late complications

- Tracheal stenosis.
- Tracheomalacia (loss of cartilaginous support of tracheal wall).
- Tracheal erosions, anteriorly into the innominate artery, posteriorly into the oesophagus.

Advantages of endotracheal intubation over tracheostomy

- It is performed quickly and easily.
- It leaves no scar.
- It avoids some of the serious complications of tracheostomy such as wound infection, misplacement of the tube in the pretracheal tissues, ulceration into the innominate artery, pneumothorax etc.

Disadvantages of endotracheal intubation over tracheostomy

- Fixation of the tube is more difficult.
- Suction is more difficult.
- Laryngeal damage which may follow is more difficult to treat.

Except in cases with acute obstructive lesions of the upper airway, endotracheal intubation is normally used as the initial method of treatment even when prolonged artificial ventilation is anticipated. The incidence of severe laryngeal complications increases after 48-72 hours. Since by this time the pattern of the disease process has become clear the intubated patient is reassessed with a view to tracheostomy after the second or third day. If it is likely that intubation could be terminated in another 2-3 days, then the tube is left in place. If not, tracheostomy may be considered. However, with the introduction of the soft, low-pressure cuff, it is possible to leave an endotracheal tube in place for as long as 10 days if there is a possibility of avoiding tracheostomy.

Nursing care of a patient with a tracheostomy tube

Care of the tube

The cuff

- The cuff is deflated for 5 minutes every 1-2 hours but this must not be done till at least 30 minutes has elapsed after oral or tube feeding.
- Deflation of the cuff must be preceded and followed by nasopharyngeal and oropharyngeal suction.
- A sterile catheter must be used for suction of the tracheostomy tube.
- The cuff should be reinflated slowly. Air is admitted into the cuff sufficient to maintain a seal.
- The cuff is clamped (distal to the pilot balloon) with a pair of artery forceps which are rubber lined.

The tube

- The tracheostomy tube is changed twice a week. In the first 48 hours the tube must be changed by a medical officer, experienced in the procedure.

Procedure for changing tracheostomy tube

Requirements

- Sterile gloves and sterile suction catheters
- A source of oxygen and a means of ventilating the patient (this could be the anaesthetic machine or a self-inflating bag)
- Suction machine
- An endotracheal tube
- A laryngoscope
- A sterile replacement tracheostomy tube
- Good lighting

Method

- Check the new tracheostomy tube for leaks.
- Position the patient flat.
- Perform oxygenation through the tracheostomy tube for 4 minutes.
- Suction the pharynx and tracheobronchial tree.
- Remove the tracheostomy tube and insert the new one. If there is difficulty in the passage of a new tracheostomy tube:

  either Insert an endotracheal tube so that the cuff is below the tracheal stoma.

  or Cover the stoma (opening) and then ventilate the patient with a bag and mask.

Suction

Suction is performed whenever necessary to keep the tracheostomy airway free of secretions. It must be carried out as a sterile procedure, so the hands should be washed and sterile gloves used each time.

The suction catheter used in the oropharynx must **not** be used in the tracheostomy tube. It should have an external diameter less than half the internal diameter of the tracheostomy tube.

Suction should be applied on withdrawing the catheter, avoiding excessive negative pressure to the airway. The patient should be oxygenated before and after suction. The quantity and the nature of the secretions should be noted.

Care of the stoma

Dress the tracheostomy wound every 8 hours and do it as a sterile procedure. Antibiotic ointment is applied to the wound. The tracheostomy tube should be secured firmly with cotton tape and any traction on the tracheostomy tube should be avoided.

Humidification

It is essential to humidify the inspired gases in a patient with a tracheostomy, because the upper respiratory tract, which is the normal means of humidification, is bypassed. If the gases are not humidified the secretions will dry, harden and obstruct the tube and the respiratory passage.

Methods available

- Cold bubble humidification, in which the oxygen is bubbled through cold water. This is not very effective.
- Heated humidifiers. Water is heated to about 37°C and the temperature controlled by a thermostat. The gases are carried to the patient through a large bore tube and a T-piece. This is a safe and effective method. The dangers associated with the system are overheating through faulty control of the temperature. There is also a danger of the water tank running dry.
- Instillation of sterile saline (0.5 ml) through the tracheostomy tube at intervals.
- Condenser humidifiers (the artificial nose). This consists of pleated material (aluminium / paper / foam) in a plastic cylinder. The total area of gas exchange is about 4,000 square cm (cm^2). The dead space is 5 ml. It is small, lightweight and effective. The water vapours in the expired air (80%) condense on the mesh and humidify inspired air. It is especially useful in ambulant patients with tracheostomies who require added oxygen.

- Nebuliser. The water is broken up into very small particles and very effective humidification is produced.

Tents and chambers are not effective means of humidification.

A patient with a tracheostomy, if unconscious and on a ventilator will need detailed nursing care and monitoring. (This is discussed in Chapter 10 Artificial ventilation).

CHAPTER 10

ARTIFICIAL VENTILATION

Outline:

Definition

Indications for artificial respiration

Methods: First aid: mouth-to-mouth
Using simple apparatus: self-inflating bag with non-rebreathing valve and mask e.g. Ambu/Laerdal
Artificial respiration using mechanical ventilators

Management of patients receiving artificial respiration (mechanical ventilation)

Additional modes of ventilatory support

Harmful effects of intermittent positive pressure ventilation (IPPV)

DEFINITION

Artificial respiration or ventilation is the process we use when the normal method of spontaneous respiration fails.

INDICATIONS FOR ARTIFICIAL RESPIRATION

(See Chapters 57, 58 and 62 for more information)

- Respiratory arrest
- Established respiratory failure
- Incipient respiratory failure
- Pulmonary oedema

METHODS

- **First aid methods**

  Mouth-to-mouth resuscitation

  Approach the patient's head from the side and ensure that the airway is clear. This may require suction or manual removal of any foreign matter. Next hold the chin up and the jaws forward, place your mouth on the patient's mouth and pinch the patient's nose with your disengaged hand. Take a deep breath and then inflate the patient by expiring forcibly into the subject 10-15 times per minute. Allow two seconds inflation and three seconds for deflation. It is very important to watch the patient's chest and note the rise and fall of the chest with each inflation and deflation.

  Advantages

  - Adequacy of ventilation can be observed.
  - Respiratory obstruction can be sensed.
  - Secretions and vomitus can be observed.
  - Return of spontaneous respiration can be detected.
  - Both hands of the rescuer are free.
  - No equipment is needed.

  Disadvantages

  - It is unpleasant for the rescuer.
  - Inflation, if the airway is obstructed, can lead to distension of the stomach and regurgitation.
  - It cannot be maintained for more than 15 minutes because of exhaustion on the part of the rescuer. This is associated with a fall in carbon dioxide.
  - The patient receives only 16 percent oxygen. This is not enough.

Of the first aid methods, however, mouth-to-mouth respiration is the most effective.

- **Methods using simple apparatus**

  Self-inflating bags with one or more non-rebreathing valves eg Ambu or Laerdal bags.
  These can be used to ventilate the patient with atmospheric air or oxygen.
  Two principles must be observed when we use this equipment:
  - The airway must be clear. The chest must rise with inflation.
  - The bag must fill. An airtight fit of the mask on the face will ensure this.

  The self-inflating bag fills by its elastic recoil during expiration. When the bag is squeezed the valve is shut and the patient's lungs are inflated. This method is superior to the mouth-to-mouth method, as ventilation is more efficient and more oxygen can be provided.

  Self-inflating bags can also be used with a LMA or an endotracheal tube if these are available in the emergency situation.

- **Artificial respiration using a mechanical ventilator**
  Two types of ventilators are available: negative pressure and positive pressure.

  Negative pressure ventilators

  Examples are the tank ventilator or the iron lung.
  The patient is placed in a rigid tank with the head protruding outside the tank and an airtight fit around the neck. During inspiration a negative pressure is created outside the chest wall within the tank. Air is sucked into the chest. During expiration the tank pressure becomes atmospheric and air flows out of the patient's lungs.

  Positive pressure ventilators

  Here positive pressure is applied to the patient's upper respiratory tract. The gases are forced into the patient's lungs. An endotracheal tube with inflated cuff is required for efficient use of these machines. There are two types of positive pressure ventilators: those which pre-set the pressure and those which pre-set the volume.
  - ***Pressure preset ventilator*** e.g. Bird, Blease
    Inspiration ceases (i.e. the entry of gases into the lungs stops) when a set pressure is reached. The disadvantage is that if a patient has very stiff lungs or is asthmatic then the pressure builds up very quickly before a sufficient tidal volume has been delivered to the patient.
  - ***Volume preset ventilators*** e.g. Manley
    These ventilators are set to deliver a chosen volume. Hence inspiration ceases and expiration starts when a certain volume of gas has been delivered.

Ventilators can be powered electrically or by compressed gas or both and are cycled by pressure, time or volume. Modern ventilators have many different features and the anaesthetist must always be familiar with the functioning of the ventilator in use whether in theatre or ICU.

MANAGEMENT OF PATIENTS RECEIVING MECHANICAL VENTILATION

In addition to nursing and monitoring the patient the ventilator also has to be managed.

Managing the ventilator

The ventilator should be set so that it produces an alveolar ventilation sufficient to maintain blood, oxygen and carbon dioxide levels within normal limits. The following controls must be selected:

Time control rate For adults the rate should be 12-16 times per minute.

Inspiratory time control at 1-1.5 seconds. I/E ratio (inspiratory time/expiratory time ratio) should be 1:2.
This means that expiration should be longer than inspiration.

Tidal volume There are many ways to assess the tidal volume.

- The movement of the chest.
- Using the spirometer.
- Estimation of the arterial blood gases (about half an hour after the patient has been placed on the ventilator).

Airway pressure. An inflation pressure of 10-25 cm of water is usually adequate.

Humidification. Patients ventilated for more than 12 hours should receive humidified gases.

Oxygen enrichment. This should be determined in the light of pulse oximetry and arterial blood gas readings.

Nursing the patient

- Routine care of the endotracheal or tracheostomy tube.
- Humidification of gases.
- The nutrition of the patient, especially fluid and calorie requirements. An average patient needs *2800 calories* per day and *3000 ml of water*. These can be administered via naso-gastric feeds and IV fluids.
- Management of pain. A combination of narcotic analgesic and sedative drugs is recommended.
- Position
  - Turn the patient from side to side to prevent pressure sores.
  - Perform percussion of the chest to prevent accumulation of secretions.
  - Perform passive joint movements.
- Eyes: lubricate with saline and gently close every 15 minutes.
- Mouth: oral hygiene with an antiseptic solution.
- Bladder: catheter should be checked and volumes recorded at regular intervals.
- Rectum: the regular use of suppositories or enemas.
- Communication: pencil and paper should be provided.
- Morale.

Monitoring the patient

- Ventilation. Monitor the tidal volume using Wright's spirometer.
- Blood pressure
- Pulse
- Central venous pressure. Use a central venous catheter inserted into the great veins draining into the heart. It is indicated in certain types of patients, e.g. those with renal dysfunction.
- Fluid balance charts showing intake and output.
- Temperature
- Blood gases
- Chest X-Ray
- Hb percent
- Serum electrolytes

ADDITIONAL MODES OF VENTILATORY SUPPORT

Positive end expiratory pressure (PEEP)

A positive pressure of 5-10 cm water is applied at the end of expiration. This holds the airway open. It is used in situations where the arterial oxygen is low despite high inspired oxygen, e.g. fat embolism.
This may embarrass the heart and cardiac output especially if there is underlying cardiac dysfunction or if the patient is hypovolaemic. If PEEP is used to improve oxygenation and hypotension develops, treatment consists of IV fluids plus vasopressors (if needed).

Intermittent mandatory ventilation (IMV)

This set-up enables the patient to breathe spontaneously through another circuit but the ventilator 'boosts' the patient's inspiratory effort by providing a selected tidal volume at a selected respiratory rate. This is used when 'weaning' a patient off a ventilator.

HARMFUL EFFECTS OF INTERMITTENT POSITIVE PRESSURE VENTILATION

Fall in cardiac output and blood pressure.

This occurs especially in sick, old and hypovolaemic patients. The negative pressure in the chest during spontaneous respiration is replaced with positive pressure. The negative pressure maintains the venous return into the heart. This is called the thoracic pump. During IPPV the 'thoracic pumping' stops, the venous return and the cardiac output fall. In the normal patient, this is compensated for by peripheral vasoconstriction. In the hypovolaemic, shocked patient, compensatory vasoconstriction may not occur.

Increase in the pressure in the pulmonary capillaries.

This may cause right heart strain, as the right ventricle now has to work harder to pump blood through the pulmonary capillaries.

CHAPTER 11

INHALATIONAL ANAESTHETIC AGENTS

Outline:

Uptake and excretion of inhalational anaesthetics

Stages of anaesthesia (Guedel's)

Balanced anaesthesia

The inhalational anaesthetic drugs
- Nitrous Oxide
- Ether
- Halothane
- Trichloroethylene
- Enflurane
- Isoflurane
- Sevoflurane

A summary of properties

UPTAKE AND EXCRETION OF THE INHALATIONAL ANAESTHETICS

A volatile anaesthetic agent is a liquid which when it evaporates into a gas has anaesthetic properties. A good volatile anaesthetic agent easily evaporates into a gas at normal room temperature. There are two phases in the uptake of anaesthetic gases and vapours: the pulmonary phase and the circulatory phase.

The pulmonary phase

The gas has to be inspired and reach the alveoli in the lungs. A significant concentration has to be built up in the lungs and then diffusion occurs from the lungs into the blood stream. The pulmonary capillaries are close beside the alveoli. The air in the alveoli is separated from the blood in the capillaries by two thin layers of cells and a film of interstitial fluid. This interface is a large area of 55 square metres.

As the concentration of the inhaled anaesthetic builds up in the alveoli the anaesthetic diffuses or moves across into the blood stream. It is then carried in the pulmonary vein to the left side of the heart and from there to the arterial circulation via the aorta and other arteries.

The circulatory phase

The drug enters the blood stream and is carried to the brain and the other organs where its effect is experienced. When the agent dissolves in the brain it produces the state of anaesthesia.

Many theories have been considered to explain how anaesthesia is produced but it is still somewhat of a mystery. The agent may reduce the ability of the nerves to conduct impulses to each other and activity is reduced, or stopped altogether if you give an overdose. Fortunately, the higher centres controlling consciousness are the first affected and the vital centres such as the respiratory and vasomotor centres are more resistant to this effect.

The main physical properties of inhalational agents are: -

- Blood-gas partition coefficient/solubility
- MAC / potency
- Volatility

The blood gas partition coefficient. This is defined as the ratio of the amount of anaesthetic in the blood to the amount in the gaseous phase when the phases are in equilibrium (i.e. the pressures are the same). It is really a measure of the solubility of the anaesthetic in blood. The higher the blood gas solubility, the slower the induction and the slower the recovery from anaesthesia.

Ether has a high blood gas solubility and is therefore a slow induction agent. Nitrous oxide has a low blood gas partition coefficient, so both induction and recovery are fast. Halothane has a coefficient in between nitrous oxide and ether.

MAC or Potency. MAC (minimum alveolar concentration) is defined as the concentration of an anaesthetic agent which at equilibrium will prevent a reflex response to a skin incision in 50% of patients.
Thus the potency of different agents can be compared, by showing how much is needed to produce the effect required. This is expressed as a percentage vapour strength.
An agent with a low MAC is a potent agent because only a small amount is required to produce anaesthesia. A high MAC means the agent is weak because a lot of agent is required to produce anaesthesia. Ether has a high MAC and is a weak agent, while trichloroethylene has a very low MAC, is potent and produces its effects at a fraction of the concentration of that needed for ether. Halothane has the ideal MAC, somewhere in between.

Volatility. An agent with a low ***boiling point*** will evaporate readily and enable a higher concentration to be administered than with an agent with a high boiling point.
Ether is highly volatile and can be given in high concentration. Trichloroethylene is difficult to vaporise and it is harder to give an adequate concentration. Halothane lies between these agents and has good physical properties.
Another index of volatility is the ***Saturated Vapour Pressure*** or SVP. It indicates the maximum proportion of atmospheric pressure which can be occupied by the vapour of an agent. Ether has an SVP of 425 mmHg and theoretically will allow a maximum concentration of 56% (425/760 x 100). SVP is dependent only on the temperature and not on atmospheric pressure.

The uptake of inhalational anaesthetics in the pulmonary phase is affected by:

- ***The inhaled concentration.*** This determines the concentration in the alveoli and hence the rate at which the gases diffuse into the blood stream.
- ***Alveolar ventilation.*** The greater the alveolar ventilation the more rapidly the concentration in the alveoli approaches the concentration of the inspired gas.
- ***The blood gas partition coefficient*** – see above
- ***MAC or potency of the agent*** – see above
- ***Pulmonary blood flow***. This equals the cardiac output except in certain cases of congenital heart disease.
- ***The alveolar membrane.***
- ***Diseases of the lung*** which alter the relationship between the blood flow and the actual ventilation tend to interfere with the uptake of anaesthetic gases.

The uptake of inhalational anaesthetics in the circulatory phase is affected by:

- ***The cardiac output.*** Normally about 70% of the cardiac output goes to the brain, heart, liver and kidneys, although these organs comprise only about 7% of the total body weight. In certain conditions this distribution of blood flow can be altered. For example, in muscular activity, fever, fear, apprehension, or thyrotoxicosis, there is an increased blood flow to the peripheral organs. This may delay the onset of general anaesthesia. On the other hand, in the shocked patient (as in a patient with decreased circulating blood volume following haemorrhage, dehydration, burns etc.) there is peripheral vasoconstriction and a relative decrease in the blood flow to the skin, gut and periphery. This means that the brain receives a higher concentration of the drug (because of the redistribution of the blood flow). Induction is therefore more rapid and smaller doses of drugs produce the same effect.

Factors increasing the speed of induction

- The inhaled concentration.
- Hyperventilation with both spontaneous and controlled ventilation.
- A poor peripheral blood flow, e.g. in patients who are shocked.

Factors slowing down the speed of induction

- Decrease in the inhaled concentration.
- Respiratory obstruction.
- Hypoventilation from any cause.
- Increase in the circulation to non-vital organs.

During induction a higher concentration of anaesthetic is needed, because at this time the vital organs like the brain, with a greater blood supply, take up larger quantities of anaesthetic agents. The other tissues, with a smaller blood supply, take up the anaesthetic more slowly. When these tissues do start taking up anaesthetic, it is drained away from the brain to the tissues via the blood stream. As the tissues are saturated the rate of diffusion from the brain decreases and only a small amount of anaesthetic is required to maintain anaesthesia. Inhalational anaesthetics are excreted mainly by the lungs, so the reverse of what happens during induction takes place during recovery.

STAGES OF ANAESTHESIA (GUEDEL'S)

Guedel first described the stages of anaesthesia using open ether. The stages of anaesthesia that he described are really a series of physical signs indicating the depth of anaesthesia. Much of what he describes is no longer seen because of modification of the stages by drugs (induction agents, muscle relaxants and narcotic analgesic agents) but may help in understanding what is happening to the patient as anaesthesia is deepened.

Following IV induction the patient goes from consciousness to surgical anaesthesia (plane 2-3) in 2 minutes.

Stage 1 Analgesia From the beginning of induction to the loss of consciousness. At the end of this stage the eyelash reflex is lost.

Stage 2 Excitement From the loss of consciousness to the onset of automatic respiration. During this stage struggling and vomiting may occur.

Stage 3 Surgical anaesthesia This commences at the onset of automatic respiration. It ends at the onset of respiratory paralysis. It is divided into four planes:

- ***Plane I*** From the onset of automatic respiration to the cessation of eyeball movements.
- ***Plane 2*** From the cessation of eyeball movements to the beginning of intercostal paralysis.
- ***Plane 3*** From the start to finish of intercostal paralysis.
- ***Plane 4*** From complete intercostal paralysis to diaphragmatic paralysis, respiratory failure, cardiac failure and death.

The most useful signs are therefore:

- The eyelash reflex
- The movement of the eyeball
- Dilatation of the pupil
- Changes in respiration

There is now a tendency to divide the stages of Surgical Anaesthesia into three planes:

- Light anaesthesia – until the eyeballs are fixed
- Medium anaesthesia – increasing intercostal paralysis
- Deep anaesthesia – diaphragmatic respiration

BALANCED ANAESTHESIA (Triad of anaesthesia)

Gray has described three 'components' to an anaesthetic:

- Narcosis or sleep
- Relaxation
- Pain relief and reflex suppression

The present concept is not to use a single drug, e.g. ether, to provide all of these components. Instead different agents are used, each with its special action to provide the various components of an anaesthetic.

Thiopentone, for instance, may be used to induce sleep, *nitrous oxide* and *opioids* to provide pain relief and *muscle relaxants* to provide relaxation for surgery.

THE INHALATIONAL ANAESTHETIC DRUGS

NITROUS OXIDE

Physical and chemical properties

Nitrous oxide (N_2O) is a colourless gas, with a faint smell. It will support combustion but is not explosive. It is provided in a cylinder under pressure. It is manufactured by heating ammonium nitrate to 240°C.
Nitrous oxide can contain nitric oxide and nitrogen dioxide as impurities. About 80 percent of the cylinder is liquid. As it evaporates it uses up heat, which explains the condensation of moisture on the outside of the cylinder. A reducing valve reduces the pressure from 750 pounds/square inch to 50 or 60 p.s.i. depending on the machine.

The impurities of nitrous oxide can cause the following effects:

- Cyanosis, a few minutes after induction. The nitric oxide converts the Hb to methaemoglobin, which is unable to carry oxygen.
- Respiratory difficulty, caused by non-specific pulmonary oedema and broncho pneumonia.
- Circulatory failure

Nitrous oxide has a low blood gas partition coefficient and so anaesthesia is induced relatively rapidly. However it is a weak anaesthetic and a high concentration is required to produce loss of consciousness.

Systemic effects of nitrous oxide

- ***Cardiovascular system***
  - Causes direct cardio-vascular depression. This is not significant in healthy patients but in patients with poor myocardial contractility nitrous oxide can cause a reduction in cardiac output and BP.
- ***Central nervous system***
  - Causes depression of the CNS but it is a poor anaesthetic and cannot provide surgical anaesthesia alone.
  - It has mild analgesic properties.

Advantages

- Rapid onset and recovery
- Provides analgesic supplement for other agents
- Has minimal cardiovascular and respiratory effects

Disadvantages and unwanted side effects

- Nitrous oxide is regarded as a relatively non-toxic anaesthetic although prolonged administration (6–24 hours) can result in depression of bone marrow DNA synthesis and this period may be much shorter in very ill patients.
- Nitrous oxide diffuses very easily into air filled cavities and should be avoided or used with caution in patients with pneumothorax or bowel obstruction as it may cause these air collections to enlarge.
- ***Diffusion hypoxia***. There is a 35 fold difference in the solubility of nitrogen and nitrous oxide. If the mask is removed (or the patient extubated) and if this patient is allowed to breathe room air, the alveoli will soon contain nitrogen and oxygen from the atmosphere.
  Capillaries in contact with the alveoli will contain a high concentration of nitrous oxide and oxygen since gases move from an area of high concentration to one of low concentration. Nitrous oxide moves out into the alveoli while nitrogen moves into the blood stream. The rate of diffusion of the nitrous oxide is 35 times faster than that of the nitrogen gas.
  Therefore for every 35 molecules of nitrous oxide that move into the alveolus from the blood stream, only 1 molecule of nitrogen will move out of the alveolus into the blood stream. The alveolar oxygen will thus be diluted by the nitrous oxide. The patient can get hypoxic simply because of a difference in the rates of diffusion of nitrogen and nitrous oxide.

After every general anaesthetic (mask or endotracheal) always try to ensure that the patient has 10 to 12 deep breaths of high oxygen mixture using the anaesthetic circuit. This could even be 100% oxygen.

ETHER

Physical and chemical properties

Ether is a colourless highly volatile liquid with a boiling point of 35°C and is flammable in air and explosive in oxygen. It has a high blood gas partition coefficient and a low MAC value. Ether is mainly excreted by the lungs with only about 15% being metabolised.

Ether is manufactured by heating concentrated sulphuric acid with ethyl alcohol. Ether may contain peroxides and aldehydes as impurities, because of its decomposition by heat, light and air. Decomposition is retarded by copper (Cu), iron (Fe) and mercury (Hg).

Ether should be stored in dark bottles in a cool dry place
The presence of impurities reduces the potency of ether. Peroxides are the first impurity to develop. To test for peroxides add 10mls of ether to 1ml of 10 percent potassium iodide. Store this for 30 minutes in a dark room. A yellow coloration indicates the presence of peroxides. To test for aldehydes use Nessler's reagent.

Systemic effects of ether

- ***Cardiovascular system***
  - ***Heart rate***. There is an increase in the heart rate, which is due to the liberation of catecholamines by the ether.
  - ***The peripheral blood vessels*** are first constricted and later dilated.
  - ***Myocardium*** may be depressed but because the sympathetic nervous system is being stimulated, the cardiac output is well maintained.
  - ***Blood pressure*** is initially maintained because of the stimulant effect on the sympathetic nervous system.

  In deep surgical anaesthesia the heart rate begins to slow and the blood pressure falls. The direct effects of ether on the cardiovascular system begin to predominate over the effects of the sympathetic nervous system stimulation.

  IMPORTANT: Patients who are on anti-hypertensive treatment with ganglion blocking drugs or beta blockers and patients who have had spinal anaesthesia before an ether anaesthetic, may be prone to greater falls in blood pressure under ether anaesthesia. Cardiac arrhythmias are rare with ether. Adrenaline can be used with ether.

- ***Respiratory system***
  The respiratory centre in the brain is the initial stimulator of the respiratory system. Ether works on the tracheo-bronchial tree giving further stimulation to respiration. There may also be stimulation of the irritant receptors in the lung. At deeper levels of anaesthesia, respiratory depression occurs.

- ***Central nervous system***
  Ether induced analgesia is followed by anaesthesia. Cerebral blood flow and intracranial pressure rises. Convulsions have been reported but the cause is not clear. Many of the convulsions occurred in febrile children who had atropine in their premedication and a deep ether anaesthetic.

- ***Gastro-intestinal tract***

  The incidence of nausea and vomiting depends on the concentration of ether used. If we are using 4-6% ether, e.g. spontaneous ventilation, the incidence is 50 percent. If we use 2-3% e.g. ether with muscle relaxants, the incidence is no higher than with any conventional relaxant-nitrous oxide-oxygen anaesthetic.

- ***Genito-urinary tract***

  Urinary flow is diminished because of the renal vasoconstriction. The uterus is relaxed. Ether can cross the placental barrier and depress the foetus so high concentrations of ether are best avoided in Caesarean sections.

- ***Skeletal muscle***

  A myoneural block occurs of the non-depolarising type.

- ***Blood sugar***

  Ether stimulates gluconeogenesis and may cause hyperglycaemia. Patients with impaired glycogen stores may form ketones leading to a metabolic acidosis.

Dosage and techniques

Ether has a high therapeutic ratio and is safer than more modern agents for administration from an uncalibrated vaporiser or by less experienced anaesthetists.

It is usually administered via an EMO inhaler using a draw over technique but can also be given using a plenum system with a Boyle's bottle. It can be used in a closed circuit and may still occasionally be administered using a Schimmelbusch mask.

The easiest method is to give ether to the patient after they have been intubated following atropine, thiopentone and suxamethonium. IPPV is commenced initially with 10-15% ether and then according to the patient's requirements, the ether is cut back after 2-8 minutes to 4-6%. Poor risk, septic or shocked patients can be kept just asleep with 2% ether. It is best to discontinue the ether well before the end of the operation to avoid a prolonged recovery. If the patient is given a long acting muscle relaxant and the ventilation controlled, then the ether may be reduced to around 2-3%.

Advantages of ether

- It is relatively non-toxic, especially if used as described with IV induction, intubation and muscle relaxants with low concentrations of ether. (see Chapter 14)
- Respiratory depression usually precedes cardiovascular depression.
- There are no associated cardiac arrhythmias and cardiac output is well maintained.

- It can be used as the sole anaesthetic agent and is the agent of choice when general anaesthesia is needed but no oxygen is available.
- It gives good abdominal muscle relaxation.
- It is cheap.

Disadvantages

- It is explosive in oxygen or nitrous oxide.
- Diathermy should not be used.
- It gives slow induction and slow recovery.
- It stimulates salivation and is best used with atropine premedication.
- It can cause a high incidence of nausea and vomiting if used in a high concentration (which is not recommended).
- It should be avoided if possible in diabetic patients and in patients with liver disease.

Prevention of convulsions (see CNS effects)

- Febrile children need little or no atropine before surgery.
- Avoid deep ether anaesthesia if possible.
- Keep the patient cool.
- Start IV fluids.
- A relaxant anaesthetic is preferable to a deep ether anaesthetic.

Treatment of convulsions

- Stop the ether anaesthetic.
- Oxygenate the patient.
- Control the convulsion with:
  - Diazepam IV
  - Thiopentone (50-100mg IV)
  - If intubation and artificial ventilation is necessary the patient can be given suxamethonium 1mg/kg IV.
- Reduce temperature by the use of fans and cold sponge.

HALOTHANE

Physical and chemical properties

Halothane is a colourless liquid with a relatively pleasant smell. It is decomposed by light and is stored in amber coloured bottles. Thymol 0.01% is added as a stabilising agent and can be responsible for causing sticking of the lever on the OMV. Halothane can be used safely with soda lime.

Halothane has a relatively low blood gas solubility so induction is rapid though slower than enflurane, isoflurane and sevoflurane. Halothane is mainly excreted by the lungs with only about 20% metabolised in the liver and excreted in the urine.

Systemic effects of halothane

- ***Central nervous system***
  Halothane is a CNS depressant six times as potent as ether. Unlike ether it is not a good analgesic. It increases cerebral blood flow and raises intracranial pressure.
- ***Respiratory system***
  There is a decrease in the tidal volume. Its broncho-dilator action is good in asthmatics.
  Always use at least 30 percent oxygen with halothane
  The respiratory centre is usually depressed before myocardial depression occurs. *If the patient is breathing spontaneously and then stops, act as follows:*
  - Turn off the halothane.
  - Ventilate the patient with 100% oxygen.
  - Check pulse and blood pressure.
- ***Cardiovascular system***
  Halothane is a potent myocardial depressant and these effects are augmented in the presence of beta-blockers. Blood pressure and heart rate may also fall by a similar mechanism to that after thiopentone so the effect will be augmented when both are given together.

  Arrhythmias: There is increased myocardial irritability with halothane. This may result in ventricular extra–systoles, ventricular tachycardias or even ventricular fibrillation.

Factors increasing the risk of arrhythmias with halothane:

- Carbon dioxide retention
- The injection of adrenaline (it is safer not to use adrenaline under halothane anaesthesia but if necessary then the dose should not exceed 10mls of 1:100,000)
- The injection of atropine
- Very light anaesthesia resulting in sensory stimulation.

- ***Gastrointestinal tract***
  - Nausea and vomiting (rare).
- ***Liver***
  - Liver damage and jaundice have been reported but this is rare.
  - A type of hepatitis (clinically indistinguishable from infective hepatitis) has been described. See Halothane hepatitis in Chapter 46.
- ***Genito-urinary tract***
  - ***Kidneys.*** No harmful effects have been reported.
  - ***Uterus.*** Uterine relaxation is profound in the pregnant uterus. It is best not to use halothane for caesarean sections, dilatation and curettage (for miscarriages). It is useful in cases of external version and uterine rings where uterine relaxation is especially sought after. Halothane also depresses the foetus.
- ***Other actions***
  - Postoperative shivering: Treat this by
    - Reassuring the patient
    - Giving oxygen
    - Keeping the patient warm
    - Pethidine 25–50 mgs IV may help
  - Potentiation of muscle relaxants. The non-depolarising agents e.g. pancuronium or vecuronium may be potentiated.

Dosage and techniques

Inhalational induction with halothane

The mask is placed closely over the patient's face. (If a draw–over technique is being used the mask has to be placed firmly on the patient's face). The vapour strength is increased gradually from 0.5% to 4% (if the patient is a fit adult) **or** 0.5% to 2% (in ill or frail adults or children). Once the eyelash reflex has disappeared, the mask is placed on the patient's face and the concentration of halothane dropped to 2% and adjusted as required.

Uses

Halothane is used

- As an induction agent
  - In children. This is often more acceptable than an injection and halothane is the agent of choice for inhalational induction in paediatric anaesthesia.
  - In adults with poor veins or when IV agents are contraindicated.
  - In adults where airway obstruction is a possibility.
- For maintenance
  - With spontaneous respiration using nitrous oxide and oxygen.
  - With controlled respiration using nitrous oxide and oxygen and relaxant.

Methods of vaporisation of halothane

- Open methods are very wasteful and there is no control of the inspired concentration.
- Semi–closed methods:
  - The ***Oxford miniature vaporiser (OMV).*** This is the ideal method for delivering halothane with draw-over apparatus. It has low resistance, has some thermo-compensation using a mixture of water and antifreeze inserted at manufacture and delivers an accurate concentration on a scale of 0-4.
  - The ***Fluotec vaporiser.*** This is the most accurate method of administering halothane. The vaporiser is temperature compensated. As the temperature falls with vaporisation, then more carrier gas passes through the Fluotec.
  - ***Boyle's bottle.*** If this is to be used safely, then an extended scale would be required for the lever. Using a Boyle's bottle for administration of halothane is not recommended.
  - The ***Goldman vaporiser*** is a simple bottle with no temperature compensation. The Goldman vaporiser cannot yield more than a maximum of 3% halothane.

  (For more details about these vaporisers see Chapter 15)

Advantages of halothane

- Gives a rapid smooth induction.
- Is relatively non-irritant to the respiratory tract.
- Is a bronchodilator.
- Is a vasodilator – this may aid venepuncture in patients with difficult veins.
- Gives relatively rapid recovery.

Disadvantages

- Halothane is very potent and the delivered concentration should be known at all times.
- Poor analgesic properties.
- Causes cardiac arrhythmias especially when adrenaline is used.
- Causes hypotension.
- Uterine relaxation resulting in haemorrhage in obstetric patients.
- Shivering in the post-operative period.
- There is the possibility of liver damage especially after repeated administration.

Safety measures with halothane

- Induction with halothane. ***Always*** reduce the concentration of halothane when the eyelash reflex has disappeared and introduce the drug used for maintenance, either a relaxant or nitrous oxide.
- Halothane and spontaneous respiration. The pulse and blood pressure must be monitored very carefully. The respiratory rate and the oxygen saturation or colour of the blood at the site of operation are other useful parameters to monitor. If these indicate that the patient has received too much halothane then reduce the concentration immediately.
- Halothane with controlled ventilation. Intermittent ventilation with 0.5% halothane will usually suffice if N_2O or IV opioids are used. It is safer not to ventilate a patient with more than 1% halothane.

TRILENE (Trichloroethylene)

Trichloroethylene is no longer manufactured for anaesthetic purposes although there may be some stores still available.

Physical and chemical properties

It has a high blood/gas solubility and a MAC of 0.17%.
It is non-flammable and non-irritant to the respiratory tract.
Trichloroethylene is stabilised in solution by the addition of 0.01% thymol and should be protected from light.

Uses and dosage

Trichloroethylene is mainly used as an analgesic supplement to halothane or used on its own for minor procedures such as fracture manipulation.
Dose 0.5-1% initially, reducing to 0.2-0.5%.

Advantages

- It is a non-irritant safe agent.
- It provides good analgesia during and after surgery.
- It maintains cardiac output and is inexpensive.

Disadvantages

- Trichloroethylene takes effect slowly.
- It has weak anaesthetic properties.
- It may result in a rapid respiratory rate in spontaneously breathing patients.
- Arrhythmias may occur and adrenaline administration is contraindicated.
- If high doses are used a prolonged recovery will occur - particularly in elderly patients.

Contraindications

Trichloroethylene must not be used in a circle system with soda lime as the toxic compounds phosgene and carbon monoxide are produced.

ENFLURANE

Physical and chemical properties

Enflurane is a clear, colourless, volatile anaesthetic with a pleasant smell. It is non-flammable in clinical concentrations, can be used with soda lime and does not require preservatives.
It has a low blood/gas solubility resulting in rapid induction of and recovery from anaesthesia and a MAC of 1.67.

Systemic effects

- ***Central nervous system***
  - Depression with increasing depth of anaesthesia
  - Jerky movements may appear
- ***Cardiovascular system***
  - As anaesthesia deepens there is a fall in blood pressure
  - Arrhythmias may occur as with halothane but less commonly
- ***Respiratory system***
  - No increase in bronchial secretions
  - Enflurane is a good bronchodilator
- ***Skeletal muscle***
  - Potentiates non-depolarising relaxants

Uses and techniques

Induction concentration 3% to 4%
Maintenance concentration 1% to 2%

Enflurane should not be used in patients with seizure disorders, e.g. epilepsy, hydrocephalus or patients with cerebral irritation from any cause.
Enflurane can be administered from a conventional anaesthetic machine using an appropriate Tec series vaporiser or for draw-over anaesthesia using the OMV with an interchangeable Enflurane scale.

Advantages of enflurane

- Rapid induction and recovery
- Low risk of hepatic dysfunction
- Potentiation of muscle relaxation
- Low incidence of arrhythmias

Disadvantages

- Relatively high cost
- Shows evidence of seizure activity on electro-encephalogram

ISOFLURANE

Physical and chemical properties

This is a newer inhalational anaesthetic now widely used in Europe. It is a halogenated methyl ethyl ether. It is a colourless volatile liquid, with a saturated vapour pressure similar to halothane. Some have suggested using it in a halothane vaporiser but this is not recommended.
Blood gas solubility is lower (1.4) than that of halothane or enflurane. It therefore provides a more rapid onset of and recovery from, anaesthesia. Further, fat solubility is also lower, which has the same effect.
Minimum alveolar concentration (MAC) is 1.15 in oxygen.
It has an irritant smell.
Isoflurane is almost totally excreted by the lungs.

Systemic effects of isoflurane

- ***Central nervous system*** Like other inhalational agents, it causes
  - CNS depression and anaesthesia but it has no convulsant effect like enflurane.
  - Cerebral blood flow and therefore intracranial pressure increases, though this is less than with halothane and ethrane.
- ***Cardiovascular system*** (CVS)
  - Little effect on myocardial contractility.
  - A fall in blood pressure due to a decrease in peripheral vascular resistance, though this depends on the dose.
  - Heart rhythm is stable. Isoflurane does not increase the sensitivity of the myocardium to adrenaline.
- ***Respiratory system***
  - Decrease in tidal volume.
  - Increase in respiratory rate.
- ***Muscle tone***
  - Relaxes muscles.
  - Potentiates non-depolarising muscle relaxants.
- ***Liver and kidneys***
  - Negligible changes in liver function.
  - Negligible changes in renal function. An association with malignant hyperpyrexia is under investigation.

Advantages of isoflurane

- Rapid onset and recovery from anaesthesia
- No convulsant effect on central nervous system even with hypocapnia
- Low risk of cardiac arrhythmias and no increased sensitivity to adrenaline
- Low toxicity to liver and kidneys
- Non explosive and non-flammable

Disadvantages

- Expensive

Administration

As with enflurane, isoflurane can be administered using a Tec vaporiser on a conventional anaesthetic machine or in a draw-over system using an OMV with the appropriate scale.

SEVOFLURANE

Physical and chemical properties

This is a newer inhalational agent. It is non-flammable with a pleasant smell.
It has a very low blood/gas solubility and provides the most rapid induction and recovery of all currently available inhalational agents.
It has a MAC of 1.7%.
5% is metabolised in the liver.

Systemic effects

- ***Central nervous system***
  - No significant effect on ICP or CBF at clinical levels
- ***Cardiovascular system***
  - Does not sensitise the myocardium to catecholamines
- ***Respiratory system***
  - Decrease in minute volume
  - Increase in respiratory rate
  - No airway irritation

Uses

It is becoming the agent of choice for inhalational induction especially in children but also in adults.

Advantages of sevoflurane

- Rapid induction
- Rapid recovery
- Minimum cardiovascular effects

Disadvantages

- High cost
- If being used as sole inhalational agent in a draw-over system it would require two OMVs in series to give an adequate concentration.

(See Chapter 13 for this set–up)

SIGNIFICANT FEATURES OF THE INHALATIONAL ANAESTHETICS

NITROUS OXIDE (N_2O)

ADVANTAGES	DISADVANTAGES
Pleasant Non-explosive Analgesic (mild) No effect on metabolism Can be used with adrenaline	Weak relaxant Weak anaesthetic: cannot produce anaesthesia alone It should be supplemented with volatiles to prevent awareness Necessitates a reduction in oxygen concentration since at least 50% conc. of N_2O is required for analgesia Causes increased distension of air-filled cavities e.g. the middle ear Post-operative nausea and vomiting

ETHER

ADVANTAGES	DISADVANTAGES
Minimal respiratory and cardiovascular depression Good relaxation Bronchodilator Clear signs of depth of anaesthesia Cheap Good analgesic Can be used with adrenaline No dangerous arrhythmias	Irritant Explosive Nausea and vomiting Secretions Can cause myocardial and respiratory depression if deep anaesthesia is induced Dangerous with • Unfasted patients • Diabetes (uncontrolled) Slow induction, slow recovery

TRILENE

ADVANTAGES	DISADVANTAGES
Pleasant Non irritant Analgesia Non-emetic Non-explosive	Weak and poor relaxant Weak anaesthetic. Causes tachypnoea Causes cardiac irregularities Myocardial depressant Cannot be used with soda lime Should not be used with adrenaline

HALOTHANE (FLUOTHANE)

ADVANTAGES	DISADVANTAGES
Pleasant No secretions Non irritant Rapid action Rapid recovery Minimal nausea or vomiting Non-explosive Bronchodilator	Myocardial depressant Arrhythmias may occur Respiratory depressant Poor relaxant Narrow margin of safety – dangerous in concentrations above 2% Uterine relaxation – should not be used in caesarean sections or miscarriages Post-operative shivering may occur Can cause hepatitis in hypersensitive patients Should not be used with adrenaline Poor analgesic Malignant hyperpyrexia risk factor More expensive than ether

ENFLURANE (ETHRANE)

ADVANTAGES	DISADVANTAGES
Pleasant Rapid induction and recovery Non-irritant Bronchodilator Minimal nausea and vomiting Non-flammable	Myocardial depression May produce hypotension and arrhythmias with progressive depth of anaesthesia Shivering May unmask a latent epileptic focus in the brain Expensive Malignant hyperprexia risk factor

BRIEF COMPARISON of HALOTHANE, ISOFLURANE and SEVOFLURANE

AGENT	HALOTHANE	ISOFLURANE	SEVOFLURANE
CVS	Myocardial depression Arrhythmias common esp. bradycardia Sensitises myocardium to adrenaline	Less depression of myocardium Stable cardiac rhythm Does not sensitise myocardium to adrenaline	Similar to isoflurane Does not sensitise myocardium to adrenaline
CNS	Depression	Depression No convulsant effect	Depression Autoregulation preserved
RS	Depression Bronchodilator	Depression Bronchodilator	Depression Bronchodilator
Skeletal Muscle	Relaxation Potentiates non-depolarisers	Relaxation Potentiates non-depolarisers	Relaxation Potentiates non-depolarisers
Disadvantages	Arrhythmias Sensitises myocardium Hepatitis Associated with malignant hyperpyrexia	Pungent odour Can be irritant Expensive Associated with malignant hyperpyrexia	Unstable with soda lime Very expensive Associated with malignant hyperpyrexia
Hepatic metabolism	20%	0.2%	5%
Maintenance concentrations	1%	1.5 - 2.5%	0.5 – 3%

Volatile agents and their physical properties.				
AGENT	**SVP in mmHg**	**MAC**	**Blood/Gas coefficient**	**Comments**
Ether	425	1.92%	12	High volatility High solubility
Trichloroethylene	60	0.17%	9	Low volatility High solubility
Halothane	243	0.75%	2.3	Volatile Low solubility
Sevoflurane	160	1.8%	0.6	Volatile Very low solubility
Isoflurane	250	1.15%	1.4	Volatile Low solubility
Enflurane	175	1.68%	1.9	Volatile Low solubility

CHAPTER 12

MUSCLE RELAXANTS

Outline:

The neuromuscular junction

The depolarising relaxants

The non-depolarising relaxants

Dual block

Mixed block

Practical points for using relaxants

Reversal of non-depolarisers (neostigmine)

Uses of relaxants

Incomplete reversal of the relaxant

Prolonged apnoea at the end of surgery

THE NEUROMUSCULAR JUNCTION (NMJ)

Consider briefly the anatomy and the physiology of the neuro–muscular junction:

Anatomy:
The junction between the motor nerve and the muscle is the neuro-muscular junction. At this point two important changes occur in the nerve and two changes in the muscle.

At the nerve terminal

- The nerve loses its myelin sheath.
- It breaks up into a foot-like structure.

The muscle

- There is a depression in the muscle at this site.
- The muscle membrane is thrown into several folds.

The nerve impulse has to travel across the gap between the nerve and the muscle at the NMJ.
The specialised area of muscle is referred to as the *motor end plate.*
On the motor end plate are special areas called *receptors*, stimulated by a*cetylcholine.*

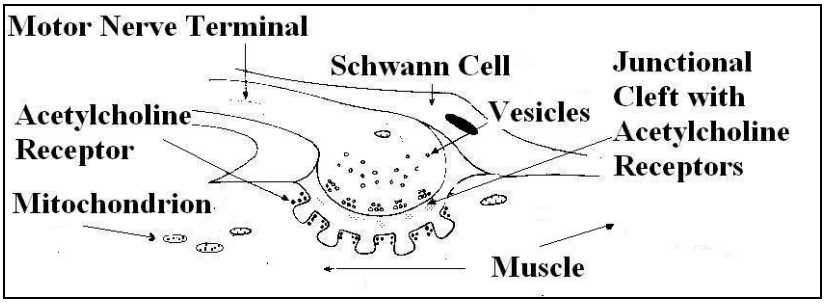


Fig 12.1 Anatomy of the Neuro–muscular junction

Physiology
The following sequence of events takes place at the NMJ with the arrival of the nerve impulse:

- The nerve impulse liberates acetylcholine from the nerve ending.
- Acetylcholine occupies receptors at the motor end plate.
- This is followed by changes (both ionic and electrical) at the motor end plate.
- Muscular contraction results.
- The released acetylcholine is hydrolysed fairly quickly by cholinesterases found in the region of the motor end plate.

Neuromuscular block:
Any interference with the passage of an impulse between the nerve and muscle produces a neuromuscular block. There are four types of block:

- Depolarising block
- Non-depolarising block
- Dual block
- Mixed block

THE DEPOLARISING RELAXANTS

These are very much like acetylcholine in structure. They occupy the receptors, depolarise the muscle and cause contraction initially ***but then*** they continue to occupy the receptors and the muscle becomes electrically unresponsive to further stimulation by acetylcholine. This action persists until the depolarising relaxant is hydrolysed by cholinesterase. This takes a few minutes.

SUXAMETHONIUM CHLORIDE (Scoline, Anectine, Succinylcholine) is the commonly used depolarising relaxant.

Uses
The main use of suxamethonium is to give relaxation when it is essential to insert an ETT rapidly. It is also used by infusion or intermittent IV injection during general anaesthesia to produce relaxation for short procedures.
After a standard intubating dose of suxamethonium, muscle relaxation lasts 3-5 minutes but since it is always combined with other anaesthetic agents the apparent duration of muscle relaxation is dependent on the combined effect of all drugs given.

Suxamethonium solutions cannot be mixed with thiopentone.

Dose
Suxamethonium chloride 1-1.5 mg/kg IV or 4mg/kg IM.
Maximal neuromuscular block occurs in 1 min. (IM administration is reserved for rare occasions where inhalational induction is undertaken in children without IV access and obstruction develops. It is best to establish IV access prior to induction if possible.)

Duration
3-5 minutes depending on other drugs administered. If it lasts more than 15 minutes, this should be regarded as an abnormal response.

Systemic effects

- Rapid onset of action – 1 min.
- Short acting.
- Associated with muscular fasciculation (initial stimulation) especially in the face, neck and limb muscles before paralysis.
- The action of the depolarisers wears off when the drug is hydrolysed, so no specific reversal of the drug is required.
- Action is increased or potentiated by anticholinesterase drugs.

Side effects of suxamethonium chloride

- Muscle pains – usually experienced from a few hours to 1-2 days post-operatively, particularly in ambulant patients.
- Prolonged apnoea may occur if the patient has either a low pseudo-cholinesterase level or an abnormal one.
- Cardiac arrhythmia, especially bradycardia in children.
- It raises intraocular pressure and therefore its use is a relative contraindication in patients with glaucoma or in patients having intraocular operations or with penetrating eye injuries.
  However, securing a safe airway takes precedence over a small rise in intraocular pressure.
- Suxamethonium chloride can trigger a hyperthermic reaction called malignant hyperpyrexia.

Contraindications to suxamethonium

- There can be a release of potassium from the tissues, especially after major burns. Suxamethonium can safely be used in the first 48 hours after a major burn. However, it is unsafe to use suxamethonium after 48 hours for at least 6 weeks and possibly for as long as 2 years. There is an increased incidence of cardiac arrest if it is used in these patients.
- Following chronic spinal cord lesions with upper motor neurone denervation the motor endplate extends to cover the entire muscle cell membrane. When suxamethonium is administered, depolarisation occurs over the whole endplate area causing massive K^+ efflux and risk of cardiac arrest. Suxamethonium should be avoided in these patients from 3 days up to 9 months post injury.

Destruction of suxamethonium compounds

Suxamethonium compounds are broken down by serum or pseudo-cholinesterases, just as acetylcholine is broken down by acetyl cholinesterases.

Acetyl cholinesterase is specific to acetylcholine. It is found in the nerve terminals, synapses, brain and red blood cells. Pseudocholinesterase is non-specific. It has little effect on acetylcholine but acts on other compounds. The levels of pseudocholinesterase may be low in certain medical conditions and these patients are very sensitive to the drug.

Low pseudocholinesterase levels occur

- In liver disease
- In severe anaemia
- In malnutrition
- In the second half of pregnancy
- After therapeutic irradiation
- After contamination with organic phosphorous insecticides

Abnormal pseudocholinesterase

Certain patients may have an abnormal pseudocholinesterase. This abnormality runs in families. It is very important not to disregard an "anaesthetic complication" that has arisen in a family especially when the information is reliable. The presence of the abnormal cholinesterase can be confirmed by certain specialised laboratory tests (the Dibucaine No. and the Fluoride No.)

In all these instances of low or abnormal pseudocholinesterase the patient requires only very small doses of suxamethonium chloride. If a normal dose is given the effect of the suxamethonium chloride does not wear off for several hours and the patient remains paralysed.

THE NON-DEPOLARISING RELAXANTS

Uses and mode of action

These combine with the receptor and prevent acetylcholine getting to the receptor at the motor end plate.

- They are long-acting relaxants, their effects lasting 20-45 minutes as opposed to 3-5 minutes for the depolarising relaxants like suxamethonium chloride.
- Their onset of action is 2.5-3.0 mins.
- They do not produce muscular fasciculation.
- The action can be reversed by increasing the concentration of acetylcholine. This is done by using an anti-cholinesterase. The receptor is now presented with an increased concentration of acetylcholine which overcomes the effect of the non-depolarising relaxant at the NMJ.
- Their action is potentiated by ether, halothane and other inhalational agents.
- As a general rule doses in the upper range are given to achieve intubation and moderate doses are given as initial doses for abdominal relaxation. Approximately one third of the initial dose is given as required as a maintenance or subsequent dose in fit patients.

Examples of non-depolarising agents

TUBOCURARINE CHLORIDE

This was first obtained from the bark, leaves and stems of the plant *Chondrodendron tomentosum*. It has been used on arrow tips in South America for centuries. Its action at the NMJ has been described previously. (See also Chapter 5)

Dose	20 - 30 mg for intubation for an adult 0.3-0.5mg/kg (15-20 mg) supplementary dose
Duration	20 - 30 minutes. It is absorbed if given IV, IM, SC It crosses the placental barrier in small doses only.

Side Effects

- Hypotension (ganglion blocking action)
- Histamine release may result in bronchospasm and further hypotension

GALLAMINE TRIETHIODIDE (Flaxedil)

Uses
Gallamine has general effects similar to tubocurarine but its duration is shorter, 20-25 minutes.
A precurarising dose of gallamine is sometimes used for its ability to prevent muscle pains caused by suxamethonium.

Dose 80 - 100 mg (for IPPV) (1mg/kg)

Systemic effects
It has a sympathomimetic effect and induces tachycardia and hypertension. Histamine release is rare.

Contraindications
- Renal disease because it is excreted by the kidney
- Pre-existing tachycardia
- Not the drug of choice for obstetrical procedures such as caesarean section as it crosses the placental barrier

Both tubocurarine and gallamine are less commonly used since the introduction of pancuronium, vecuronium and atracurium.

PANCURONIUM BROMIDE (Pavulon)

Pancuronium belongs to the amino steroid group of non-depolarising muscle relaxants.
It does not have the hypotensive action of tubocurarine nor cause such a marked tachycardia as gallamine.

Uses
As a muscle relaxant for surgery. It has a relatively long duration of action and is sometimes used for long-term ventilation in ITU e.g. respiratory problems / tetanus.

Dose: Adults 4 - 6 mg IV initially (0.05-0.1mg/kg), 2mg thereafter.

Duration
Its action lasts for about 45 minutes but is dependent on dose and other agents used.

Metabolism
It is mainly excreted unchanged in the urine but some is also metabolised in the liver.

Side effects
It has a vagolytic and sympathomimetic effect and can cause tachycardia and a rise in blood pressure.

VECURONIUM BROMIDE (Norcuron)

This is also an amino steroid compound and is an intermediate acting non-depolariser. It is marketed as a powder (4 mg/ampoule generally) for mixing with water. The powder can be stored at room temperature.

Dose	initial dose	0.08-0.1 mg/kg
	supplementary dose	0.03 mg/kg
	(infusion 0.2 mg/kg/hr)	
Duration	20 minutes	

Metabolism
It is broken down mainly in the liver. A small proportion of the drug (20%) is excreted unchanged in the urine.

Systemic effects

- ***Cardiovascular system:*** There are minimum changes in pulse and blood pressure. No histamine release.
- ***Placental barrier:*** Does not cross the placenta in clinically important amounts.
- ***Reverses well***: It is reversed in the usual manner with atropine and neostigmine 20 minutes after the last dose.

ATRACURIUM BESYLATE (Tracrium)

Atracurium is a non-depolarising blocking agent derived from curare which undergoes spontaneous breakdown into inactive fragments at body temperature and pH.

Dose	0.3mg-0.6mg/kg supplementary dose ⅓ of initial dose
Duration	Onset of action is 2½ mins Duration 20-40 minutes

Systemic effects

- ***Cardiovascular system:*** Minimal bradycardia may be present with halothane.
- ***Histamine release:*** It does cause histamine release but to a much less significant degree than tubocurarine
- ***Placental barrier***: There is some transfer but it is insufficient to produce a block.
- Agent of choice in ***renal or hepatic disease*** and in ***elderly patients***.
- ***Reverses well*** (after 20 minutes) with atropine and neostigmine.

Both vecuronium and atracurium give good intubating conditions but are not recommended as an alternative to suxamethonium when rapid intubation is required. Both drugs show a tendency towards spontaneous recovery of neuromuscular function but it is recommended that they be reversed in the usual way.

ROCURONIUM BROMIDE (Esmeron)

Rocuronium like vecuronium is an intermediate acting non-depolariser and has the most rapid onset of action of this group of drugs. It gives good intubating conditions in less than 2 minutes and is sometimes used as an alternative to suxamethonium e.g. in ophthalmic surgery.

Dose	0.6–1mg/kg for intubation Supplementary dose 0.15mg/kg
Duration	20–40 minutes
Actions	As for vecuronium

DUAL BLOCK (OR A BI-PHASIC BLOCK)

This occasionally follows a repeated dose of suxamethonium. A single dose causes a pure depolarising block. This is termed a *phase I block*.
A repeated dose causes the same drug to act differently and occasionally produce a non-depolarising block.

MIXED BLOCK

This may follow the simultaneous injection of depolariser and non-depolariser. The receptors may manifest both types of blocks at the same time.
It is good to be aware of dual and mixed blocks. However, they are rare.

The important point is to ventilate these patients until they start breathing spontaneously again.

PRACTICAL POINTS FOR USING RELAXANTS

- Never use a relaxant unless all the equipment for ventilation and intubation is available and checked.
- Never use a relaxant unless you are sure you can ventilate the patient.
- All the inhalational agents commonly used potentiate the non-depolarisers, therefore the dose of relaxant (non depolarising) can be reduced when used with ether, halothane, etc.
- If the patient is hypotensive, pancuronium bromide is a good agent to use.
- Don't use gallamine in obstetric patients.
- Don't use gallamine in renal disease.
- Many of the non-depolariser relaxants can release histamine.
- All the non-depolarising relaxants must be reversed with neostigmine.
- A minimum of 20 minutes must elapse between the last dose of relaxant and reversal of the relaxant.
- Always note on the chart the time the relaxant drugs are given.
- The effects of tubocurarine, gallamine, pancuronium and alcuronium chloride are additive, so if one non-depolariser relaxant is replaced by another, make sure the previous drug has started to wear off.
- Depolarisers, e.g. suxamethonium, must not be used after non-depolarisers. Larger doses of depolarisers than is normally necessary may be required and reversal may not be easy. A mixed block may result.
- When non-depolarisers are given after depolarisers, make sure the depolariser has commenced to wear off before you administer the non-depolariser.
- Certain groups of patients are very sensitive to non-depolarisers, e.g. neonates and myasthenics.

REVERSAL OF NON-DEPOLARISERS

Anticholinesterases

NEOSTIGMINE

This prevents the normal hydrolysis of acetylcholine by the cholinesterases. Therefore there is a build-up of acetylcholine. This antagonises and therefore reverses the effect of the non-depolariser.
If acetylcholine is built up in very large quantities it could result in a depolarising block. Hence neostigmine methyl sulphate (also called Prostigmine), if given in too large a dose, could itself result in a neuro-muscular block of the depolarising kind.

Dose 2.5mg along with atropine 1.2mg or glycopyrrolate 500 micrograms. Maximum dose 5mg

Neostigmine can also result in

- Bradycardia
- Bronchoconstriction
- Increase in secretions

These are counteracted by combining the neostigmine with atropine which antagonises the unwanted effects of acetylcholine.
Neostigmine 2.5 mg is used with atropine 1.2 mg or glycopyrrolate 500 micrograms for reversal (adult dose).
There has to be some return of neuromuscular function before patients can be reversed with neostigmine and atropine. This is possible 20 minutes (roughly) after a standard induction dose of vecuronium, atracurium and rocuronium and 20-30 minutes for pancuronium.
The degree of muscle relaxation can be monitored with a nerve stimulator if available. (For more detailed information on this see Chapter 50). There needs to be at least one twitch in a train of four stimulus before the patient can be reversed with neostigmine and atropine.

Measures taken to prevent cardiac arrhythmias with neostigmine

- Use atropine or glycopyrrolate (referred to above)
- Avoid hypoxia
- Avoid hypercarbia
- Inject the dose **slowly**
- Give small increments of neostigmine and don't exceed 5 mg.

USES OF RELAXANTS

- Intubation
- Abdominal relaxation
- IPPV: Chest surgery
  Neuro–surgery
  Long operations
  Very ill patients
- Electro-convulsive therapy and cardioversion
- Treatment of laryngeal spasm (Suxamethonium)
- For use with patients on ventilators

It is important that a patient on a ventilator be managed with sedation only. If this fails and reliable staff are available to observe the patient while on the ventilator, then and only then should a relaxant be used.

INCOMPLETE REVERSAL OF THE RELAXANT

The anaesthetist must never leave patients unattended until they are able to breathe deeply and regularly and muscle power has **completely** returned.

Signs of incomplete reversal

- Respiratory movements of chest are shallow and spasmodic
- Stridor
- Jerky 'wriggling' movements of arms and legs
- Tracheal tug
- Inability to lift the head off the pillow
- Inability to keep eyes open
- Inability to cough

A nerve stimulator if available may be used, so that evidence of a persistent non-depolarising block may be detected. (See Chapter 50 or more advanced textbooks of anaesthetics).

Treatment of incomplete reversal

- Leave the ET tube in place.
- Ventilate the patient. Keep the patient well oxygenated. Wash out any anaesthetic vapours that may have been previously administered but keep the patient asleep to avoid him becoming distressed by the presence of the tube and the muscle weakness (see page 178).
- Carefully monitor the pulse and blood pressure.
- Correct acidosis, electrolyte imbalance, hypothermia and hypovolaemia.
- A maximum dose of neostigmine 5.0 mg may be given.

PROLONGED APNOEA AT THE END OF SURGERY

Causes

- **Central depression of respiratory centre**
  Respiration is slow, there is no tracheal tug and no pause after inspiration.
  - Premedication
  - Thiopentone
  - Halothane, ether or other inhalational anaesthetic
  - Analgesic agents e.g. pethidine, fentanyl
- **Relaxant (Peripheral NM Block)**
  The rate is normal but breathing is jerky and there is a pause after inspiration and expiration. A tracheal tug is present.

  Following non-depolarisers possible causes are:
  - Early reversal within 20 minutes of the last dose
  - Overdose
  - Electrolyte imbalance
  - Acidosis
  - Antibiotics e.g. neomycin, gentamicin
  - Myasthenia gravis
  - Hypothermia

  Following depolarisers possible causes are:
  - Low serum cholinesterase
  - Abnormal serum cholinesterase
  - Absent serum cholinesterase
  - Electrolyte imbalance
  - Dual block
- **Hypocarbia**
  Over ventilation during the operation causes hypocarbia. The normal stimulus to the respiratory centre is now absent. (The $PaCO_2$ level in arterial blood is about 40 mmHg.).
- **Hypercapnia**
  The high Pa CO_2 depresses the respiratory centre.
- **Metabolic Acidosis**
- **Very serious illness or frailty**
- **Reflex laryngeal apnoea** (breath holding)

MANAGEMENT OF A PATIENT WITH POST OPERATIVE APNOEA

- ***Ventilate the patient*** with 35 percent oxygen and a low concentration of volatile agent. This keeps the patient asleep. Alternatively, sedate the patient with intravenous diazepam or small doses of ketamine. If patients are paralysed and ventilated with pure oxygen there is a chance that they will wake up and this can be very frightening to them.

  Do not over or under ventilate the patient.

- ***Support the patient's circulation*** (i.e. make sure the pulse and blood pressure are within normal limits).

- Use a ***nerve stimulator*** if available, to find out whether the depression is central or peripheral and to find out whether the block is a non-depolarising or a depolarising type.

  Correct the above-mentioned abnormalities.

  Non-depolarising block will require atropine and neostigmine if not already given or may require a small additional dose.

  Depolarising block can be treated with fresh blood or plasma if available.

IT IS VERY IMPORTANT TO MAINTAIN THE PATIENT'S VENTILATION AND OXYGENATION UNTIL THE PROBLEM HAS BEEN RESOLVED
THIS MAY TAKE SEVERAL HOURS AND SUPPORT MUST BE CONTINUED UNTIL THE PATIENT CAN MAINTAIN ADEQUATE VENTILATION WITHOUT EFFORT

CHAPTER 13

LOW COST EQUIPMENT

Outline:

The EMO vaporiser

The Oxford inflating bellows

The Ruben valve, Ambu E valve, paedivalve

The Oxford miniature vaporiser

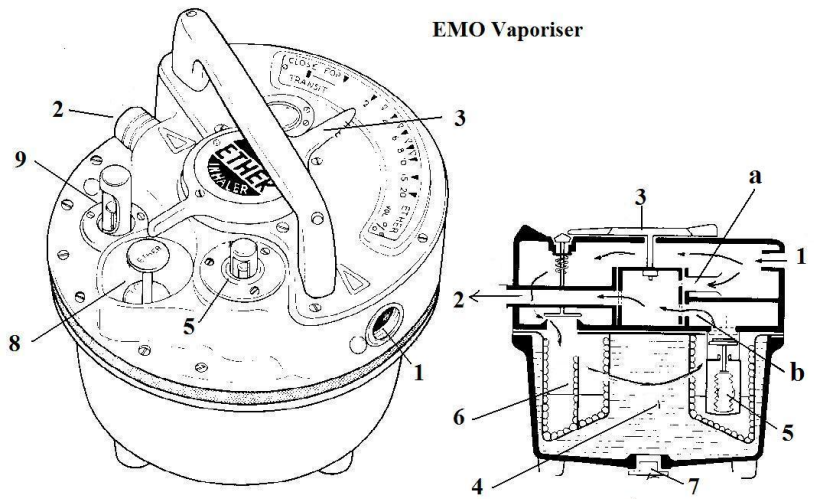
Oxygen concentrator

Ventilators:
- Manley multivent
- Glostavent

THE EPSTEIN MACKINTOSH OXFORD (EMO) VAPORISER

GENERAL DESCRIPTION

The EMO vaporiser is a calibrated, draw-over vaporiser which will accurately deliver a pre-set concentration of ether in room air. Additional oxygen can be added if required. The easiest way to describe the EMO vaporiser is to consider it in three sections.



1) Inlet port 2) Outlet port 3) Concentration Control 4) Water Jacket 5) Thermocompensator Valve 6) Vaporising Chamber 7) Filling Port for Water 8) Filling Port for Anaesthetic 9) Anaesthetic Level Indicator

Fig 13.1 The EMO Vaporiser and cross section

The lower section consists of an ether vaporising chamber surrounded both inside and outside by a water chamber which acts as a buffer against changes in temperature of the surrounding air. A tap at the bottom of the water reservoir is used for filling or draining the chamber. At the entrance to the ether chamber there is a special closing device. This closes the chamber when the indicator is in the *transit* position. Also incorporated in the closing device is ***an air inlet relief valve*** which enables air to enter the inhaler if the main inlet becomes blocked.

The outlet of the ether vaporising chamber is controlled by a thermo-compensating valve. This regulates the volume of ether vapour leaving the vaporising chamber and ensures that the concentration of ether reaching the patient is constant, independent of the temperature of the liquid ether.

To summarise, the lower part of the vaporiser consists of the following:

- An ether vaporising chamber.
- A water jacket both inside and outside the ether vaporiser.
- At the entrance to the ether vaporiser an ***inlet closing device.***
- At the exit from the ether vaporising chamber a thermo–compensating valve.

The middle section of the EMO consists of a central chamber just above the water compartment already described. This has two entry ports.

- One entry port (a) conducts the gases which have bypassed the ether vaporising chamber.
- The second entry port (b) conducts gases which have passed through the ether vaporiser and picked up ether vapour.

These gases then mix and leave the central chamber through the main outlet of the EMO.

In the transit position the entry port (b) is completely closed and (a) is completely open. As the concentration of ether is increased, then (b) progressively opens and (a) progressively closes. Thus more and more of the gas passing through the EMO machine is made to pass through the ether vaporising chamber so with each movement of the indicator more ether vapour is delivered to the patient.

The upper section of the EMO consists of a scale and a concentration pointer. Concentration shown by the indicator is kept constant because of the action of the thermocompensator and to a lesser extent the action of the water jacket.

The concentration is kept constant for a range of respiratory frequencies and tidal volumes. This is achieved by the design of the apparatus: the volume of the chambers and the flow directed by the bell mouthed and other orifices. The chambers are large and have a minimal resistance.

The ether level indicator at the left front consists of a float riding on the liquid ether inside the vaporising chamber and indicates the degree of filling of the ether chambers. The ether filler and the temperature compensator are also in the upper section of the vaporiser.

FEATURES OF THE EMO

- The concentration delivered by the EMO is constant over the 15-30°C temperature range despite the ***temperature*** of the liquid ether. This is achieved by an efficient thermocompensator. The active element is a sealed capsule containing ether. As the temperature of the ether falls, more air passes through the ether chamber.
  It also adjusts to changes in barometric pressure.

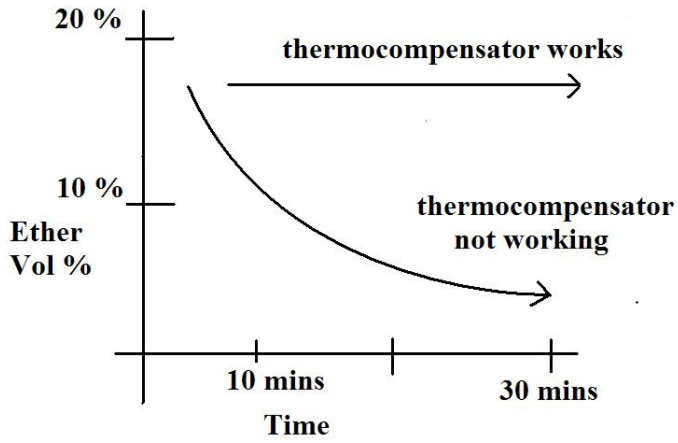
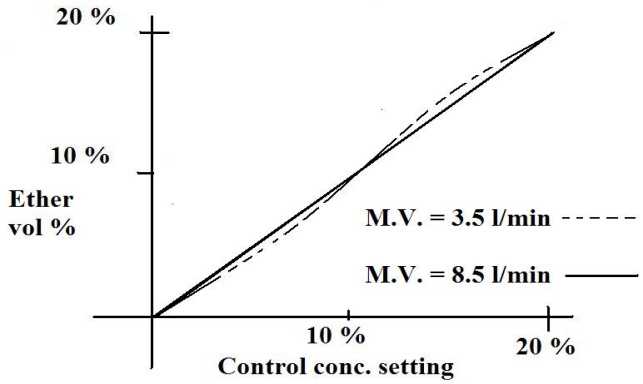


Fig 13.2 Shows the effect of immobilising the thermocompensator

- The concentration of ether is constant at different ***minute volumes*** in draw-over mode as shown below in **Fig 13.3**



- Introduction of oxygen to the EMO circuit via a ***T-Piece***

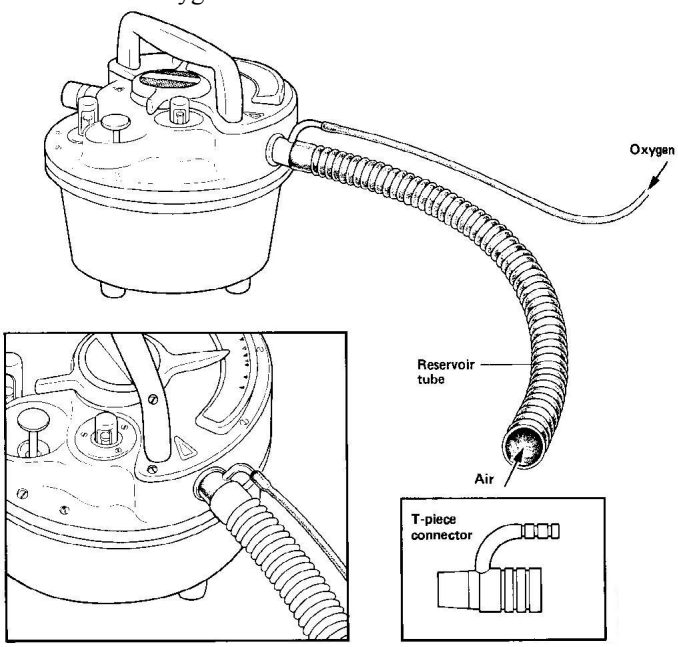


Fig 13.4 Introduction of Oxygen to the EMO with a T–piece

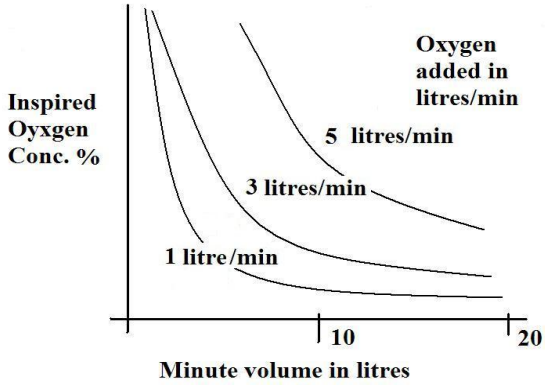


Fig 13.5 The concentration of inspired oxygen

The concentration will depend on:

- The oxygen added to the system.
- The patient's minute volume.
- The volume of the reservoir tubing. One metre (450ml) is the ideal length, delivering 30-40% FiO_2 with 1L/min O_2 flow.

INSTRUCTIONS FOR USING THE EMO

Check the water compartment

Mark I	every three months (serial numbers 1 – 2859)
Marks II - V	every year

When filling the water compartment:

- Move the control lever to the transit position.
- Turn the EMO upside down and unscrew the ***water filler plug***.
- Pour in 1200 ml of water at room temperature.

Fill with Ether

- Turn the control to zero (rather than to the transit position), so that the air can escape.
- To fill the vaporiser on a Mark I model, lift out the filler and rotate it. For Mark II-V models press the filler down.
- 150 ml will soak the wicks. A further 300 ml are needed to fill the chamber. Do not overfill.

Check the temperature compensator indicator

This consists of a rod, a black band and a red band surmounted by a cap.

- If the black band shows it means that the EMO is at the correct operating temperature.
- If the red band shows it means that the apparatus is too hot. This may happen if room temperature is above 32° C. It may be cooled by filling the water compartment with water below room temperature **or** by forcing the ether to evaporate.
- If the black band is not visible then the apparatus is too cold. Put it in a warm room for some time **or** refill the water jacket with water at 25°C.

OTHER EQUIPMENT USED WITH THE EMO

THE OXFORD INFLATING BELLOWS (OIB)

This consists of two main components:

- Spring loaded bellows.
- Two one-way flap valves.

A tap at the base of the bellows enables oxygen to be added. (This is used only during resuscitation).
A magnet is used to immobilise the distal flap valve when a non-rebreathing valve, e.g. a Ruben or Ambu E valve is used.

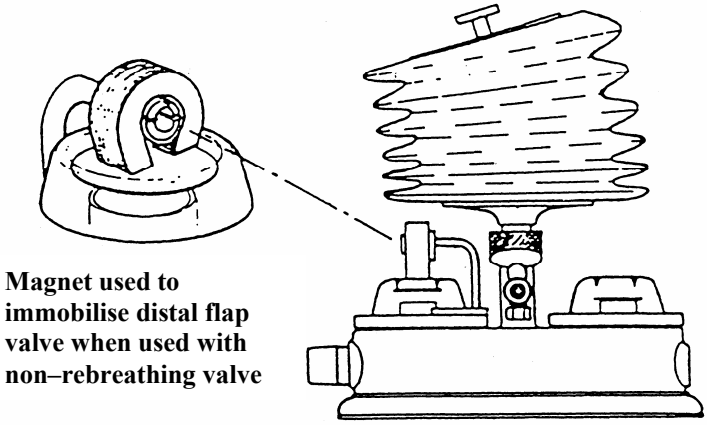


Fig 13.6 Oxford inflating bellows with magnet in position

THE RUBEN VALVE

This is a non-rebreathing valve, which means that the patient inspires only from the inhaler and expires into the atmosphere. This is brought about by the to-and-fro movement of the bobbin.
These valves can stick. Always have a spare valve available. The valves are sterilised by chemical means: 5% chlorhexidine (Hibitane) for 5 minutes.
It is important to ensure the valve is dry before use.

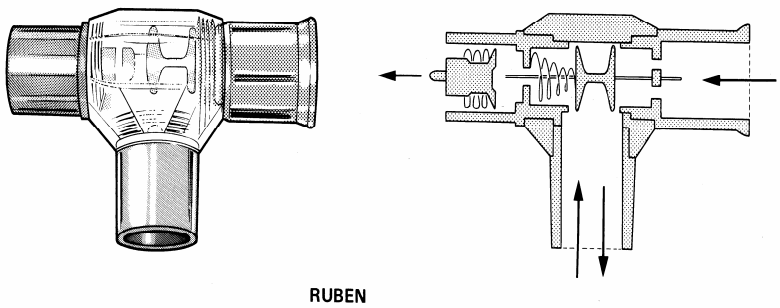


Fig 13.7 Ruben Valve

THE AMBU ANAESTHETIC VALVE

The Ambu E valve with 2 flaps is a non-rebreathing valve. This means that the expired carbon dioxide is not rebreathed. It can be used for both spontaneous and controlled ventilation. The Ambu valve is like the Ruben valve except that the bobbin is replaced by 2 yellow silicone rubber flaps. The valve can be dismantled easily for cleaning and sterilisation.
The Ambu E valve comes in ***2 models***: the ***anaesthetic model*** (2 flaps) for both spontaneous and controlled ventilation and the ***resuscitation model*** used for IPPV. This has only 1 flap, is **not** a non-rebreathing valve and is **not suitable** for anaesthetic use.

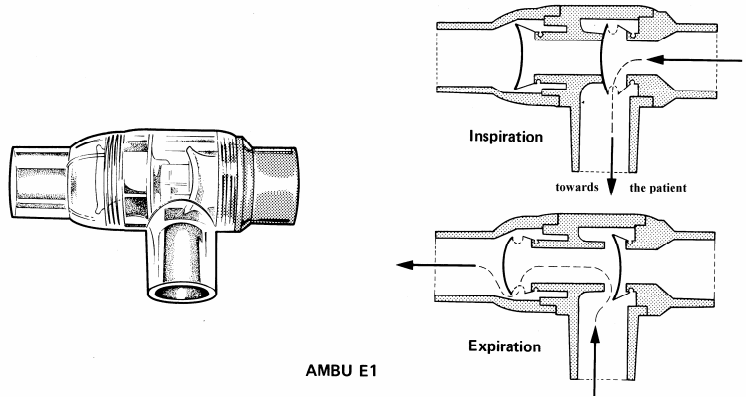


Fig 13.8 Ambu Anaesthetic Valve

Paedivalve

This is the paediatric version of the Ambu E valve which can be used for children under 15 kg. It can be used for spontaneous and controlled ventilation.

SETTING UP THE APPARATUS:

Apparatus here means the EMO, the OMV, the OIB and a non–rebreathing valve.
It is possible to combine the vaporisers, bellows, valves and other components in a variety of ways.

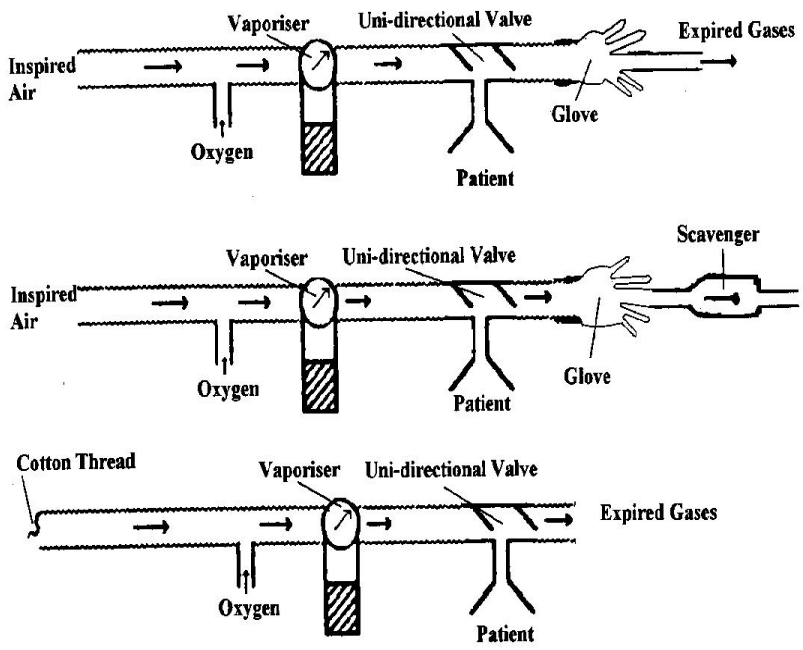
Principles

- If more than one vaporiser is to be incorporated in the circuit then the more volatile agents must be placed further from the patient. If this is not done, the less volatile agent will condense as the vapour passes through the second vaporiser. The EMO must therefore always be placed further from the patient than the OMV.

- Position the bellows (or the inflating bag) between the vaporiser and the patient.
- Introduce the oxygen at the vaporiser inlet via a T piece and 1 metre reservoir tube (i.e. further from the patient).
- When you are using a non-rebreathing (inflating) valve such as the Ruben valve, always use the horseshoe magnet with the OIB. It must be used to immobilise the valve closer to the patient.
- Before using the set-up, always check that the flow of air is in the right direction i.e. towards the patient.

The difficulty of monitoring respiration in spontaneously breathing patients can be overcome by a simple and inexpensive method. Attach a rubber glove to the expiratory limb of the uni-directional (non-rebreathing) valve and cut a small hole in one finger to allow the expired gases to escape. This can then be attached to the scavenging system if desired. Respiratory excursion can be monitored by observing the movements of a piece of cotton attached to the end of the reservoir tube, observing movements of the uni-directional valve and listening to the chest with a stethoscope.

Fig 13.9 Methods of monitoring spontaneous respiration



Standard set up using the EMO, O1B and non–rebreathing valve.

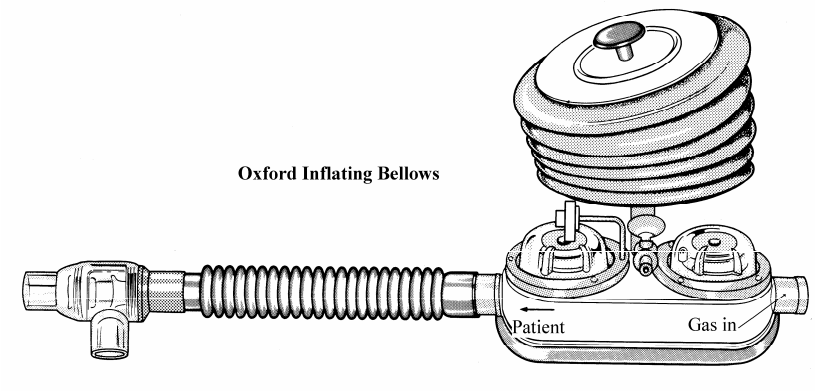


Fig 13.10 OIB and non–rebreathing valve

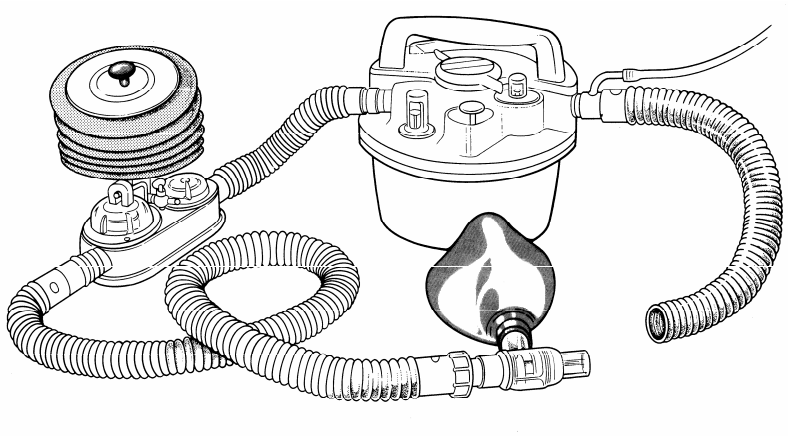


Fig 13.11 EMO, OIB and non–rebreathing valve

THE OXFORD MINIATURE VAPORISER:

The OMV was designed by Macintosh and Epstein. It delivers fairly accurate concentrations of halothane, trilene, enflurane, isoflurane and sevoflurane within defined limits. It was originally designed to assist induction of anaesthesia with the EMO. The OMV is then plugged into the outlet of the EMO. Small variations occur in the delivered concentration depending on the temperature and the duration of use. There is no increase in concentration with positive pressure. The scale ranges from 0 to 4. A special filling device is incorporated. The filler must be pressed down fully (or unscrewed) to open the filling port and the liquid flows into the vaporising chamber. The liquid level is visible in the glass window. The vaporiser should be turned off before refilling to avoid air being drawn in which would cause an increase in the concentration. The unused anaesthetic can be poured back into the bottle. Only 3 to 4 ml are retained in the wick.

When combined with a non-rebreathing valve and a means for inflating the lungs, the OMV (50ml capacity model) can also be used as a draw-over vaporiser on its own. This set up is suitable for relaxant or spontaneous breathing techniques. Adequate anaesthesia must then be given by the IV route. The carrying gas is room air supplemented with oxygen.

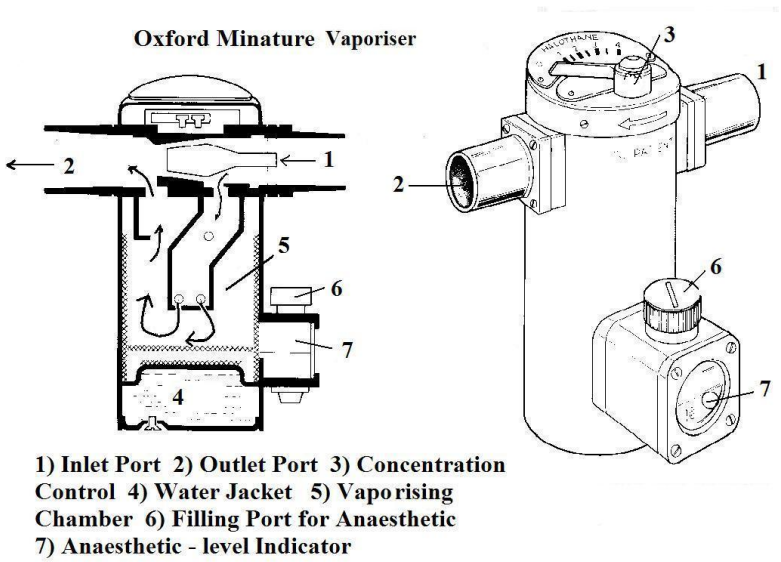


Fig 13.12 Oxford Miniature Vaporiser in section and front view

OMV vaporisers can be coupled together to use 2 volatile agents simultaneously, e.g.. halothane upstream (for its anaesthetic properties) then trilene (for its analgesic properties).

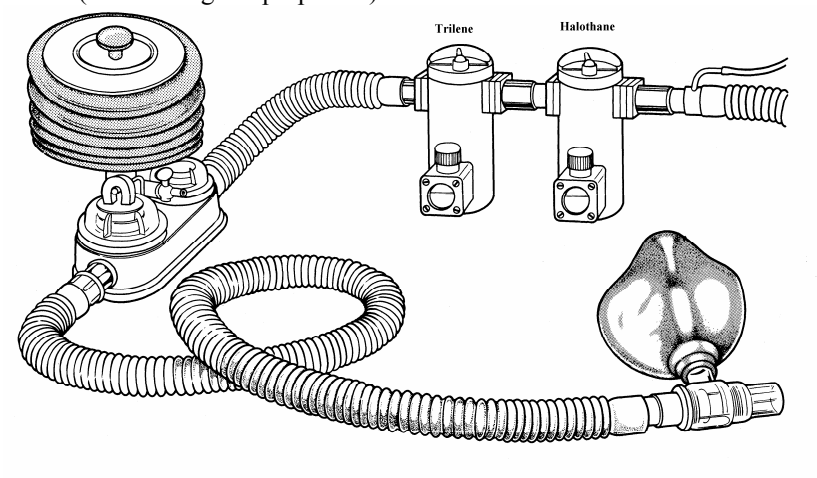


Fig 13.13 OMVs in series

The OMV is suitable for draw-over paediatric use (under 15 kg), provided three important steps are taken. Use paediatric bellows or self–inflating bag instead of the adult size on the OIB, use the **paedivalve** instead of the adult non-rebreathing valve and use tubing of a smaller diameter. Alternatively, oxygen at 4–6L/min can convert the OMV vaporiser into a plenum device for use with the Ayre's T– piece.

Fig 13.14 Paediatric set–up using paediatric self–inflating bag

THE OXYGEN CONCENTRATOR

This machine concentrates oxygen from atmospheric air. Atmospheric air consists of approximately 80% nitrogen and 20% oxygen. An oxygen concentrator separates these two components. Zeolite granules are used to selectively adsorb nitrogen from compressed air.

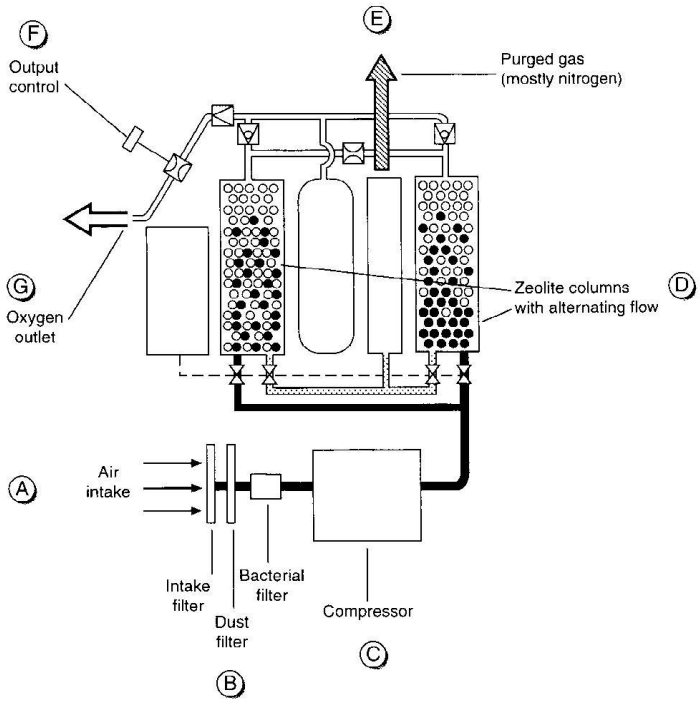


Fig 13.15 Simplified diagram of an oxygen concentrator

Atmospheric air is drawn into the oxygen concentrator. It is filtered and raised to a pressure of 20psi (1.5kg/cm^2) by an air compressor. Two canisters filled with zeolite granules are used alternately to adsorb nitrogen. The compressed air passes through the first canister, the nitrogen is adsorbed and the oxygen is made available to the patient. After about 20-30 seconds the compressed air is diverted into the second canister where the same thing happens.

While the second canister is being used, the first canister rests. The pressure is reduced to zero, the zeolite is regenerated and the nitrogen that was adsorbed is released into the atmosphere. By using the two canisters alternately a continuous supply of oxygen is maintained.

Fig 13.16 An Oxygen Concentrator

Oxygen concentrators have an output of up to 5 L/min with an oxygen concentration of 95%.

The World Health Organisation lists tested models which meet the recommended Performance Standard and details can be obtained from WHO, 1211 Geneva 27, Switzerland. They can be powered by the mains and if this fails by a small generator. Warning lights indicate a fall in oxygen concentration below 85%.

The oxygen concentrator provides an adequate supply of oxygen to be used with the EMO or OMV and also the Ambu bag in the case of the ketamine relaxant technique. It is also useful in resuscitation with IPPV and in supplementing inspired oxygen by mask or nasal catheter. Routine maintenance consists of changing the filter at regular intervals as specified by the manufacturers. The output of the oxygen concentrator must be regularly analysed using an oxygen analyser.

VENTILATORS

Manley Multivent

This is a versatile ventilator designed mainly to meet the needs of developing countries and is one of the less expensive makes. In practical terms, it is a mechanised OIB with a control panel. There are 2 types available, standard and ether compatible.

It has an on/off switch and 3 other controls, one each for tidal volume, respiratory rate and inspiratory: expiratory ratio.

Standard settings for a normal adult are indicated by colour coding and there is a low-pressure alarm system. The ventilator is powered by an oxygen cylinder or compressed air at 20psi (1.5kg/cm^2) from a modified oxygen concentrator (a De Vilbiss model is currently produced). If the oxygen cylinder runs out, or there is a power failure it can be used as a hand ventilator.

If the drive gas is supplied by an oxygen cylinder, the gas can be recycled into the breathing circuit to give approximately 34% inspiratory oxygen concentration.

A rechargeable battery operates the electronic circuits and if it is kept fully charged it will function for 150 hours. The weight on the arm compressing the bellows ensures complete emptying with each compression. Tidal volumes can be set to accommodate child as well as adult patients.

GLOSTAVENT

The Glostavent is the name describing the ***Manley Multivent*** mounted on a trolley, which has on the lower shelf a modified oxygen concentrator, incorporating an inbuilt oxygen analyser. This is capable of producing compressed air at 20psi (1.5kg/cm^2), to power the ventilator as well as producing oxygen to entrain into a draw-over system.

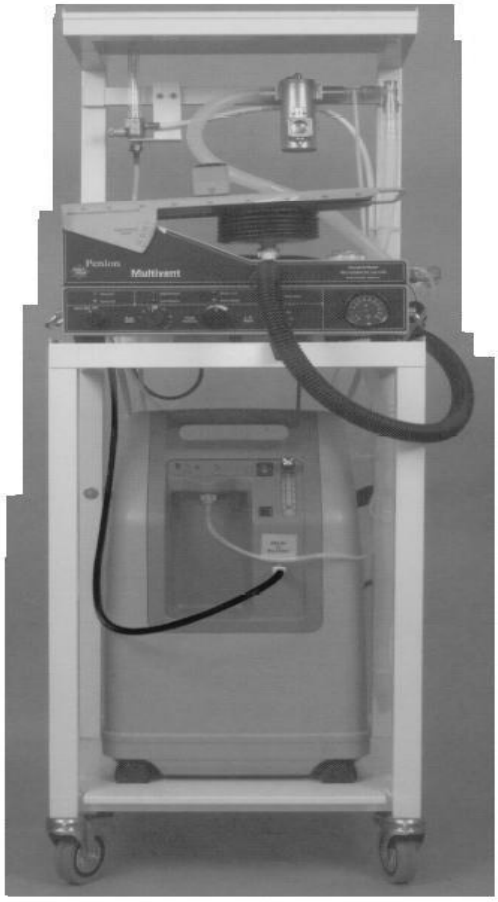


Fig 13.17 The Glostavent

(Manley Multivent with O_2 concentrator mounted on trolley)

CHAPTER 14

LOW COST TECHNIQUES

Outline:

Basic anaesthetic technique using the EMO and controlled ventilation

Basic anaesthetic technique using the OMV and controlled ventilation

Ketamine-relaxant anaesthesia and controlled ventilation

Ketamine as a sole anaesthetic

A BASIC ANAESTHETIC TECHNIQUE USING THE EMO AND CONTROLLED VENTILATION

Outline of technique

Induction with thiopentone 2-5mg/kg (or ketamine 1-2mg/kg).
Intubation using suxamethonium (Scoline) 1mg/kg.

- Ventilate with air and oxygen (4 L/min delivers FiO_2 of 60-80%) and ether 10%.
- Intubate and secure the tube.
- Continue ventilation with air and oxygen (4L/min) and ether 10%.

Maintenance When the suxamethonium wears off:

- Give 4-6mg pancuronium IV (50-100 micrograms/kg) or another non- depolarising drug such as vecuronium or atracurium.
- Reduce the concentration of ether from 10% to 3%.
- Turn off the entrained oxygen and continue ventilation with air.

(If the suxamethonium has a prolonged action, turn down the ether from 10% to 3% in 4-5 minutes).

Reversal with atropine/glycopyrrolate and neostigmine.

Pre-operative management

- Assess the patient's ***general condition*** very carefully, especially with regard to the cardiovascular and the respiratory system.
- Before elective surgery, allow a minimum 6 hours fasting for solids and 2 hours for clear liquids.
- ***Weigh*** all children under the age of 10 years and adults who are grossly under or over weight, pre-operatively. If facilities are available every patient should be weighed before anaesthesia.
- ***Premedication*** A variety of premedicant drugs may be used. See Chapter 7 for more detail.
  Opiates should be avoided in the following patients:
  - Infants under the age of 6 months.
  - Patients with head injuries.
  - Patients requiring caesarean sections.
  - Very ill patients.
  - Ophthalmic patients.
  - Many anaesthetists do not give opioids to anyone as a premed.

Atropine should be reduced by half in very hot climates. It should also be reduced in febrile patients and those with acute blood loss. It is best omitted in cases in which the pulse rate is above 120/min. Agents to reduce the acidity of gastric contents (if available) should always be considered.

The following equipment should be available: laryngoscope, endotracheal tubes with connectors, LMAs, syringe, artery forceps, catheter mount, mask, airway, stethoscope, strapping and suction.

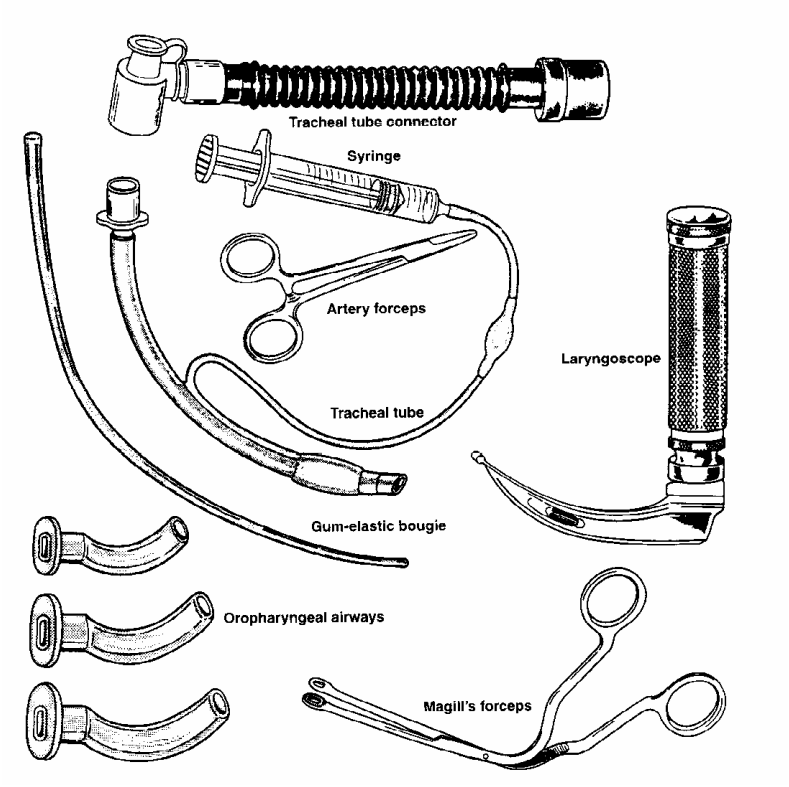


Fig 14.1 The intubating tray

The standard EMO set-up with the Oxford inflating bellows and Ruben or Ambu E valves as described earlier, is used.
A source of oxygen and the drugs used for resuscitation must be available in the operating theatre.

Intra-operative management

- Draw up the drugs and label the syringes.
- Check the blood pressure.
- Establish venous access.

Induction: Firstly, preoxygenate the patient by giving oxygen via the face mask to breathe for at least 3 minutes (4L/min). In the fit patient give ***thiopentone*** 5mg/kg. In the poor risk patient give thiopentone in a smaller dose, e.g. 3-4mg/kg; or ***ketamine*** 1mg/kg IV.

Thiopentone must always be used in a 2.5% solution for reasons described under the pharmacology of thiopentone. (See Chapter 6 page 59)
A 2.5% solution of thiopentone is prepared by mixing 0.5g of powder with 20mls of water or 1.0g of powder in 40mls of water. The dose of thiopentone is reduced to 2-4mg/kg in the following situations:

- Advanced age
- Anaemia
- Recent blood loss or hypovolaemia from any cause
- Cardiac disease
- Hypertension
- Severely obese patients
- Very ill patients from any other cause

The dose may be increased to 6 mg/kg (if necessary) in:

- Alcoholics
- Drug abusers
- Very apprehensive patients

Ketamine 1mg/kg IV is used in the poor risk patient and may also be used at 1mg/kg IV as the induction agent for caesarean sections. In higher doses it may cause harmful prolonged contractions and foetal anoxia.
Ketamine is not used in eclampsia and hypertensive patients.

Intubation: This involves the use of a relaxant drug and an endotracheal tube. The relaxant drug used is suxamethonium chloride in a dose of 1 mg/kg IV. Infants and very young children will require a dose that is higher on a weight basis than adults. Infants are technically more difficult to intubate and they have a more dynamic circulation which makes the relaxant wear off more quickly.

The endotracheal tube : Cuffed tubes are used routinely for adults. The average sized male would need size 8.5-9, the average sized female size 7.5-8. (For children see the guide to tracheal tube size in Paediatric anaesthesia Chapter 20)
The size of an endotracheal tube is its internal diameter in mm.

Once the relaxant drug has been given, prepare to ventilate the patient with a mask.

The relaxant paralyses the patient for 3-5 minutes. While the relaxant is taking effect use the time as follows:

1st minute:

- Ventilate the patient using the Oxford inflating bellows.
- Use an anaesthetic mixture of ether 10%, air and oxygen.
- Oxygen is entrained via a T piece and reservoir tube at 4L/minute.
- When the jaw is relaxed insert a Guedel airway of the appropriate size.

At first the ventilation is mainly to assist the patient's respiratory efforts. As the patient becomes fully paralysed the anaesthetist controls the patient's respiration completely.

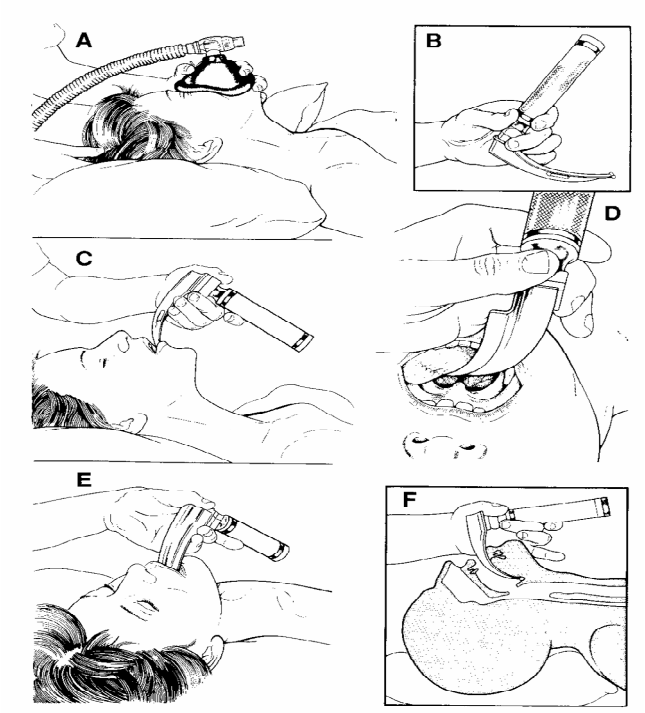


Fig 14.2 Ventilating the patient and then inserting the laryngoscope

2nd minute:

- Intubate the patient using a tube of the appropriate type and size.

3rd minute:

- Connect the endotracheal tube to the Ruben valve or Ambu E valve and the EMO/OIB set-up.

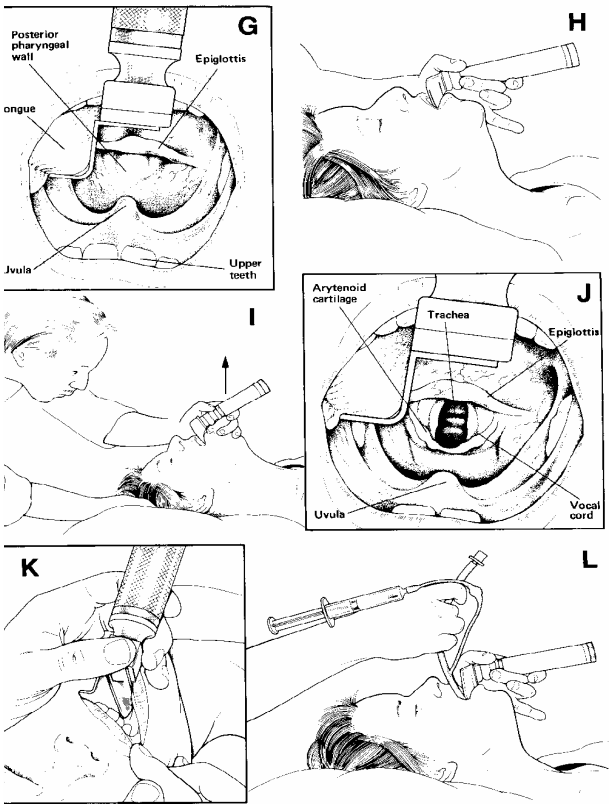


Fig 14. 3 Inserting the endotracheal tube

Check the position of the tube by auscultation. Breath sounds must be heard equally on both sides of the chest. Inflate the cuff of the endotracheal tube until the leak of air around the tube disappears. Then clamp the cuff using an artery forceps. Insert an airway as a bite block.

The anaesthetist must be satisfied with the movement of the chest with each respiration and the colour of the mucous membrane of the tongue. Then strap or tie the tube in place. The patient must be ventilated with the ether 10% /air /oxygen mixture while this procedure is carried out. Check the blood pressure at this stage. Cardiac arrhythmias can occur with high concentrations of ether 10-15%, so keep a finger on the pulse unless ECG monitoring is available.

Maintenance of anaesthesia: This requires an anaesthetic mixture to keep the patient asleep and a relaxant to keep the patient paralysed and ventilated. The anaesthetic mixture used for maintenance is ether 2-3% and air. The suxamethonium used for intubation will show signs of wearing off in 3 –5 minutes. The patient may begin swallowing, moving limbs or attempting to breathe. When this occurs:

- ***A non-depolarising muscle relaxant should be given***
  Doses:
  - Pancuronium 0.05-0.1mg/kg, **or**
  - Vecuronium 0.08-0.1mg/kg **or**
  - Atracurium 0.3-0.6mg/kg

  Ether relaxes skeletal muscles and adds to the effects of the non-depolarising muscle relaxants.
- ***Reduce the concentration of ether*** from 10% to 2-3%. Occasionally 3% ether is not sufficient to keep the patient asleep and ventilation with 6% ether may be required for a few minutes.
- ***Consider discontinuing the oxygen*** that was fed into the EMO circuit via the T-piece attachment. However, if the patient is hypotensive, with systolic blood pressure below 85mmHg, **or** hypoxic, the oxygen must be continued as before.
  If the patient is a "poor risk" for any reason at all, oxygen should be entrained at 2L/min during the entire anaesthetic. Ideally diathermy should not be used with this technique as ether plus oxygen is explosive.
  Finally oxygen should always be entrained in areas of high altitude.
- ***Ventilate*** the patient at a rate of 12-15 inflations/minute.
  Aim at a tidal volume 50% greater than the patient's resting tidal volume. Watch the patient's chest continuously during ventilation. The chest must rise during inspiration.
  Expiration must be longer than inspiration, with a ratio of 1:1.5 or 1:2 so that adequate time is given for the lungs to empty. The patient must be monitored carefully while under anaesthesia.

- ***Monitor the following vital signs***: pulse, blood pressure, colour of the blood, colour of the mucous membrane of the lips, respiration, blood loss, urine output. Pulse oximetry and capnography, if available, are invaluable.
- ***Calculate requirements*** and give intravenous fluids as required.
- ***Turn down the ether concentration*** after the peritoneum is closed. Depending on the speed of the surgeon, the ether concentration can be turned off completely if you think the operation will be completed ten minutes after the peritoneum is closed. Maintain ventilation until the operation is over. If by this stage the patient is attempting to breathe spontaneously then the patient's respirations must be assisted.

Reversal: This is carried out after the last stitch is in place, when a standard dose of atropine 1.2mg / neostigmine 2.5mg or glycopyrrolate 0.5mg / neostigmine 2.5mg is given.

Rules for extubation

- The patient must be breathing deeply and regularly before extubation is attempted.
- The patient should preferably be awake at the end of the procedure.
- Oxygen (4L/min) should be given to the patient prior to extubation.
- Suck out the pharynx then turn the patient into the lateral position.
- Suck out the pharynx again before and after removing the tube.
- Administer oxygen by mask after extubation and make sure the patient is breathing adequately. Watch the movement of the chest.
- Observe the patient until fully awake. At this stage the patient can be sent back to the ward.

Advantages of the low ether/relaxant technique

The technique is safe

- The patient is intubated and this reduces the risk of airway obstruction and laryngeal spasm. The trachea is protected against contamination in the case of a full stomach. ***However, it cannot be emphasised too much that intubation minimises but does not completely eliminate these risks.***
- The patient is ventilated, so an adequate tidal volume is maintained during surgery. IPPV is certainly safer in prolonged surgery and surgery performed in abnormal postures. A relaxant technique with a high concentration of oxygen is the safest anaesthetic in the seriously ill patient.

- Postoperative complications are reduced to a minimum. Only a very low concentration of ether is used –2-3%. At this concentration there is minimal central depression. The patient is awake at the end of the surgical procedure. Hence the incidence of post operative complications such as airway obstruction, vomiting and aspiration and chest complications e.g. atelectasis and pulmonary embolism is reduced significantly. Postoperative vomiting using the low concentration of ether is no more frequent than it is with a nitrous oxide / halothane / relaxant anaesthetic. The patient is able to get out of bed and also tolerate a normal diet (if appropriate) more quickly.
- The hazard of compressed gas cylinders running out unnoticed is less serious with the EMO technique.

Good operating conditions are provided by this relaxant anaesthesia for most surgical procedures.

Cost

- The capital outlay for the equipment used is much less with the EMO technique than with techniques using the conventional anaesthetic machine.
- The drugs used are much cheaper. Ether costs much less than nitrous oxide and halothane.
- The dose of relaxant required is much less with the EMO technique than with a conventional nitrous oxide/oxygen/relaxant technique. Ether potentiates the non-depolarising relaxants and reduces the dose required. The volume of ether used is very low. Oxygen is often a very precious commodity in developing countries. Using oxygen only before intubation and again before and after extubation conserves the oxygen supply but if there is adequate oxygen it should be used for all patients and will help to reduce post-operative complications.

Disadvantages of the technique

- Ether is a flammable agent. Cautery must be attempted with caution and not with the use of oxygen. If you use this technique, you need to pay careful attention to the state of electrical equipment used in the operating room. If possible, electrical equipment should be at least 1 metre from the floor.
- The help of an assistant is imperative if the patient is to be ventilated and monitored at the same time.
- The use of a mechanical ventilator, a spirometer to ensure adequate tidal volumes and a scavenging device would greatly improve the technique.

A BASIC TECHNIQUE USING THE OXFORD MINIATURE VAPORISER AND CONTROLLED VENTILATION

Outline of technique

Induction with thiopentone 2-5mg/kg (or ketamine 1-2mg/kg).
Intubation using suxamethonium (Scoline) 1mg/kg.

- Ventilate with air and oxygen (4L/min delivers FiO_2 of 60-80%) or 100% oxygen.
- Intubate and secure the tube.
- Continue ventilation with air and oxygen (4L/min) and halothane.

Maintenance When the suxamethonium wears off

- Give a non-depolarising drug such as pancuronium, vecuronium or atracurium
- Continue ventilation with oxygen, air and halothane

Reversal with atropine/ glycopyrrolate and neostigmine.

Pre-operative management is the same as for using the EMO (see above)

Preparation of the OMV with Oxford Inflating Bellows (OIB) and a non-rebreathing valve

- Fill with halothane up to the line indicated on the glass window.
- Check that the pointer is sliding freely.
- Disable the proximal valve nearest the patient on the OIB to allow proper function of the non-rebreathing valve.
- Check the apparatus for any leaks in the system.
- Check the non-rebreathing valve shutters are moving freely.

As in the use of the EMO a source of oxygen and the drugs used for resuscitation must be available in the operating theatre.

Intra-operative management.

Induction and intubation is the same as for using the EMO (see above) but during the intubation:

1st minute:

- Ventilate the patient using the Oxford inflating bellows with oxygen 95% or 100%.
- When the jaw is relaxed insert a Guedel airway of the appropriate size.

At first the ventilation is mainly to assist the patient's respiratory efforts. As the patient becomes fully paralysed the anaesthetist controls the patient's respiration completely.

2nd minute:

- Intubate the patient using a tube of the appropriate type and size.

3rd minute:

- Connect the endotracheal tube to the Ambu E or Ruben valve OMV/OIB set-up.

Check the position of the tube by auscultation. Breath sounds must be heard equally on both sides of the chest. Inflate the cuff of the endotracheal tube until the leak of air around the tube disappears. Then clamp the cuff using an artery forceps. Insert an airway as a bite block.

Check the movement of the chest with each respiration and the colour of the mucous membrane of the tongue. Then strap or tie the tube in place. Ventilate the patient with halothane at mark 1 on the dial and an oxygen / air mixture while this procedure is carried out.

Check the blood pressure at this stage. If the BP is high halothane at mark 2 may be given for a short time but continue to measure the BP every 2 minutes to detect any sudden fall in BP. Hypotension is likely and cardiac arrhythmias can occur with high concentrations of halothane.

Maintenance of anaesthesia: This requires an anaesthetic mixture to keep the patient asleep and a relaxant to keep the patient paralysed and ventilated. The anaesthetic mixture used for maintenance is halothane mark 1 with air / oxygen and most anaesthetists would add a dose of IV opioid. The suxamethonium used for intubation will show signs of wearing off after 3-5 minutes. The patient may begin swallowing, moving limbs or attempting to breathe. When this occurs:

- ***A non-depolarising muscle relaxant should be given***
  Doses:
  - Pancuronium 0.05mg/kg, **or**
  - Vecuronium 0.05mg/kg **or**
  - Atracurium 0.2mg/kg
- If the patient becomes ***hypotensive*** turn off the halothane until the hypotension is corrected. (If the patient is likely to awaken a ketamine infusion of 500mg/500mls of dextrose 5% or normal saline at a rate of 0.5 drops/kg to keep the patient asleep and provide analgesia could be commenced).
- ***Ventilate*** the patient at a rate of 12-15 inflations /minute.

Aim at a ***tidal volume*** 50% greater than the patient's resting tidal volume. Watch the patient's chest continuously during ventilation. The chest must rise during inspiration.

- ***Expiration must be longer than inspiration,*** with a ratio of 1:1.5 or 1:2 so that adequate time is given for the lungs to empty.
- The patient must be monitored carefully while under anaesthesia. ***Monitor the following vital signs***: pulse, blood pressure, colour of the blood, colour of the mucous membrane of the lips, respiration, blood loss, urine output. Pulse oximetry and capnography, if available, are invaluable.
- ***Calculate requirements*** and give intravenous fluids as required.

When using halothane turn it off depending on the speed of the surgeon and length of the incision when the skin wound is sutured. When using a ketamine infusion turn off the ketamine when the surgeon commences suturing the skin. Maintain ventilation until the operation is over. If by this stage the patient is attempting to breathe spontaneously then the patient's respiration must be assisted.

Reversal: This is carried out after the last stitch is in place, when a standard dose of atropine 1.2mg / neostigmine 2.5mg or glycopyrrolate 0.5mg / neostigmine 2.5mg is given.

Rules for extubation as for using the EMO (see above)

Advantages of the halothane/relaxant technique using the OMV

- Similar to ether although halothane is a more expensive agent and does not potentiate the effect of muscle relaxants to such a great extent as ether.
- Halothane is non-flammable so the use of diathermy is safe.

Disadvantages of the technique

- Halothane may cause cardiac arrhythmias.
- Halothane may cause hypotension.

THE KETAMINE-RELAXANT ANAESTHESIA AND CONTROLLED VENTILATION

This is another technique providing anaesthesia for major surgery.

The equipment required is simple

- A means of ventilating the patient, e.g. a self-inflating bag or the Oxford inflating bellows. The Oxford inflating bellows has an oxygen nipple on the side.
- An oxygen source.
- Suction.
- Equipment for intubation.
- Equipment for monitoring the patient.

The drugs required

- Ketamine infusion prepared by using 500mg ketamine in 500mls 5% dextrose or 0.9% saline. Use a normal adult giving set with 15 drops = 1ml (i.e. 1mg = 1ml).
- Suxamethonium 1mg/kg.
- Any non-depolarising relaxant.
- Neostigmine and atropine for reversal of the relaxant.
- Diazepam.
- Drugs used for resuscitation.

Pre-operative management

Perform a routine assessment of the patient and then give premedication of diazepam or opioid and atropine.

Intra-operative management

Check the blood pressure and insert an intravenous cannula.

Induction: This is achieved with ketamine 2mg/kg IV and diazepam 5mg IV in the fit adult.

Intubation: Suxamethonium 1mg/kg, then ventilation using a self-inflating bag or the Oxford inflating bellows. Ventilate the patient for 1 minute with air and oxygen and intubate as described for the EMO technique. Check the position of the tube and secure it.

Maintenance: The ketamine infusion containing 500mg of ketamine in 500mls of 5% dextrose is commenced. The infusion rate of 40-60 drops / minute (0.5-1 drop/min/kg). This is equivalent to 2-4mg/kg/hour. Give a dose of a non-depolarising relaxant when the suxamethonium wears off. Ventilation is carried out with air, and oxygen is added if necessary. Vary the rate of the ketamine infusion to keep the patient asleep and free from pain.
Depth of anaesthesia is hard to assess with this technique. Observe closely for changes in pulse rate and BP.
Stop the drip 10 minutes before the end of the operation.

Reversal of relaxant: Atropine 1.2mg. or glycopyrrolate 0.5mg plus neostigmine 2.5mg.

Take the same precautions as described for the EMO technique regarding reversal and extubation.

Two disadvantages that may be encountered with this technique are:

- Hypertension.
- Hallucinations.

THE USE OF KETAMINE AS A SOLE ANAESTHETIC

- Bolus — IV or IM
- As an infusion

The pharmacology of ketamine has been discussed earlier. Ketamine is useful as the sole anaesthetic in the following situations, provided certain precautions are taken.

Indications for ketamine

- Cystoscopies
- Dilatation and curettage
- Closed reduction of fractures (note that ketamine provides no muscle relaxation)
- Removal of foreign bodies from the ears, nose and subcutaneous tissues
- Suture of lacerations
- Incision of abscesses
- Other minor procedures, e.g. anal, or urethral dilations, etc.

Precautions to be taken

- Select patients carefully.
- A premedication of atropine is sometimes given (see below).
- Use IV diazepam to reduce the emergence delirium.
- Allow patient to awaken spontaneously after surgery in quiet surroundings.

Patient Selection

- Patients must be medically fit for an anaesthetic. The usual criteria in selecting patients for general anaesthetic should be applied. Acute respiratory infection, cardiac failure, electrolyte imbalance, etc. would be a relative contraindication to ketamine anaesthesia.
- Ketamine must not be used in patients with hypertension, raised intracranial pressure (e.g. following a head injury) or raised intraocular pressure.
- Patient should be fasted according to the guidelines for any general anaesthetic. (See Chapter 7)
- Every patient receiving a ketamine anaesthetic should be weighed.

Premedication

A drying agent such as hyoscine, atropine or glycopyrrolate can be used.

Bolus technique

- Record the patient's blood pressure before giving the ketamine. Leave the cuff in place for blood pressure recordings during surgery.
- Access a vein, using a scalp vein needle or IV cannula.
- Give diazepam in a dose of 0.1mg/kg IV.
- ***Dose:*** Give ketamine 2 mg/kg IV slowly over 1 minute (in the fit patient), or 5-7 mg/kg IM.
  The ***onset of action*** is 30 seconds IV or 2-8 minutes IM.
  Duration is 5-10 minutes IV or 10-20 minutes IM.
  If further supplements are required half the initial dose of ketamine may be used. The same dose of diazepam may be repeated.

Ketamine infusion technique

Indications: Most of the procedures listed above and also longer procedures not requiring muscular relaxation, e.g. herniotomy in an older child.

Pre–operative Management

Assessment and pre-medication are the same as for other ketamine anaesthetics.
Prepare ketamine solution 1mg/ml (i.e. 10mls of 50 mg/ml ketamine in 500mls of normal saline or 5% dextrose).
Start IV infusion of 0.9% saline, 5% dextrose or dextrose/saline mixture.
Use a cannula in a larger vein.
When commencing the anaesthetic, change to the ketamine infusion.

Intra-operative Management

Induction and maintenance

- Induction with bolus: IV ketamine 1-2 mg/kg and diazepam (Valium) 0.1 mg/kg or start drip at 2 drops/kg/min until surgical anaesthesia is reached. At this point the patient does not respond to pain but still has the eyelash and pharyngeal reflexes.
- **Slow** the infusion to 1 drop/kg/min (= 4mg/kg/hour).
- If the patient seems to react to the pain of the operation, increase the drip rate to 2 drops/kg/min until operating conditions are satisfactory.
- Monitoring is routine as for other ketamine anaesthetics, e.g. pulse, BP, respiration, colour and oxygen saturation if available.
- Stop drip about 10 minutes before the end of surgery.

Post-operatively

The patient must be nursed in a quiet environment and should be observed but must not be stimulated after surgery.

Treatment of emergence reactions

If emergence reactions or hallucinations occur, the following drugs will be useful:

- Diazepam.
- Chlorpromazine.
- Pethidine (in combination with the other drugs) if the patient is in pain.

Reassurance of the patient and relatives is important.

Other intra-operative disadvantages of ketamine

- Hypertension
- Occasional respiratory depression

CHAPTER 15

CONVENTIONAL ANAESTHETIC EQUIPMENT

Outline:

The anaesthetic machine
- Gas source
- Flow meter
- Vaporisers
- Delivery System or circuit
  - The Magill circuit
  - The Bain circuit
  - The Circle System

Checking the anaesthetic machine

Other features of the anaesthetic machine
- alarm devices
- oxygen flush valves
- scavenging devices

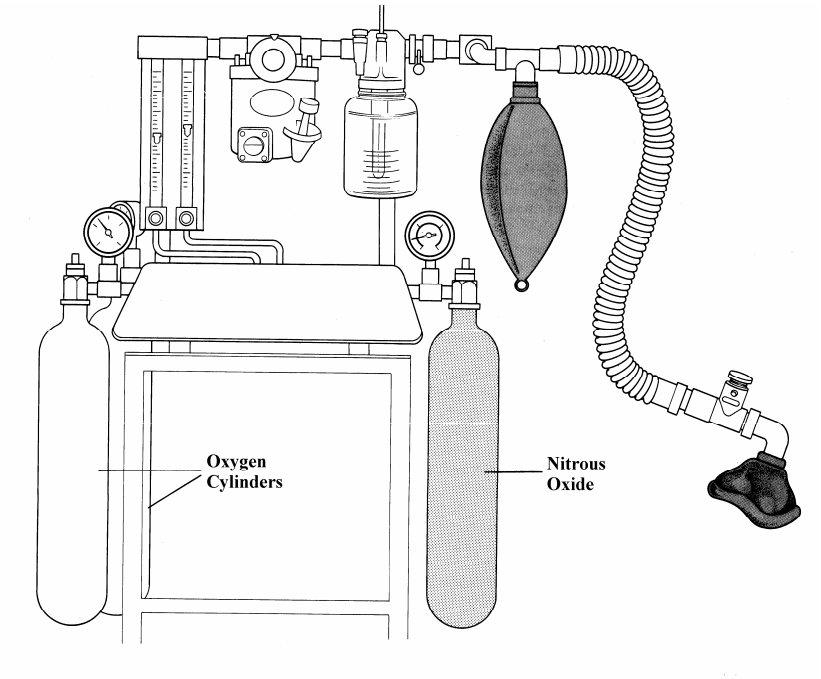


Fig 15.1 A continuous flow (Boyle's) anaesthetic machine

The introduction of gaseous anaesthetics and the need to provide more accurate control of anaesthesia over long periods of time led to the development of the anaesthetic machine.

At first glance the anaesthetic machine appears to be a formidable and complicated bit of machinery but if it is considered in its four components it becomes much simpler to understand.

The four basic components or modules are the following:

- The gas source – either piped gas or supplied in cylinders
- The flowmeter
- The vaporisers
- The gas delivery systems
  - The T-piece
  - The Magill circuit (semi closed)
  - The Bain circuit
  - The circle absorber

THE GAS SOURCE

Piped gas

Piped gases are stored in a "bank", remote from the operating room. The gases are piped into the operating room and connected to the anaesthetic machine via hoses with special connections to ensure that the nitrous oxide cannot be connected to the oxygen inlet and vice versa.

Cylinders are fitted directly on to the anaesthetic machine by means of yokes.

The pressure within the cylinders: The cylinders contain gases under a very high pressure. Oxygen is compressed at a pressure of about 147 bar (2000 psi). Nitrous oxide is compressed at a pressure of about 44 bar (600 psi).

Construction of the cylinders: The cylinders have to be constructed according to definite specifications. They are made entirely out of steel that meets certain chemical and physical requirements **or** out of a chrome molybdenum mixture that is 20% lighter than steel. Each cylinder is designed to contain a gas under a specified pressure, so the cylinder must possess a minimum strength. Each cylinder has within it a valve which will seal the contents and also provide an exit for the gases and a means of filling the cylinders.

Cylinder sizes: The size of the cylinder is designated by a letter stamped on the outside of the cylinder. Depending on the country of manufacture these letters will vary.

Labelling and marking: All the cylinders used in the operating room have certain markings on them, stamped on the top end of the cylinder.
They include:

- The specification number, then the service pressure in psi.
- A letter designating cylinder size.
- The date the cylinder was tested (done at least every 5 years).

Storing the cylinders

- Store cylinders in a definitely assigned location, specific for cylinders. No other items should be stored at that site.
- Storage rooms should be dry, fireproof and well ventilated. Cylinders should be protected from the following hazards:
  - Excessive rises in temperature
  - Highly flammable substances, e.g. gasoline, kerosene
  - Sparks or flames
  - Radiators
  - Corrosive agents
- Store the used and unused cylinders separately.

Colour code for cylinders: There is an international standard for colour coding gas cylinders but not all countries follow it. The anaesthetist must know the appropriate colour coding used in the country of origin of the cylinders being supplied.

Rules for handling cylinders

- Do not allow oil, grease or other flammable substances to come into contact with cylinders, valves, regulators, gauges, hoses etc. Do not use oil or grease for lubrication.
- Do not handle cylinders or apparatus with oily hands or gloves.
- Keep connections to cylinders, regulators etc, always securely fixed to prevent gas leaks. Always make sure the hose is in good condition.
- Do not allow sparks or flame from any source to come into contact with cylinders or other anaesthetic equipment.
- Gauges and regulators are designated for use with specific gases. Do not use them for any other gases.
- Keep the cylinder valve fully open while the cylinder is in use.
- Before you connect a cylinder to a machine, make sure paper wrappings are completely removed and the cylinder label is completely visible.
- Never attempt to repair, alter or repaint a cylinder.
- Keep cylinder valves closed whenever the gas is not being used.
- Have defective or damaged equipment repaired by the manufacturer.
- Do not allow compressed gases to be handled by anyone who is not properly instructed and experienced.
- Handle cylinders and associated equipment gently. Avoid dropping cylinders or even knocking them.

Pin index system: An ever-present hazard in anaesthesia is the danger of attaching a cylinder to the yoke meant for a different gas. This is eliminated by the pin index system. The system consists of two pins projecting from the yoke in the anaesthetic machine, designed to fit into matching holes in the body of the cylinder valve. For any one gas there is only one combination of pins and holes. Unless the correct cylinder valve is attached to the correct yoke these pins and holes will not match and the cylinder will not fit. Therefore it is not possible with this system to fit a nitrous oxide cylinder to an oxygen yoke or vice versa.

Reducing valves: These reduce the pressure of the gases in the cylinders. As mentioned earlier, the pressure in the oxygen cylinder is about 147 bar (2000 psi). This pressure must be reduced to about 4 bar (60 psi) if the gas delivered to the patient is going to be controllable and safe.

Gauges: Oxygen is entirely in its gaseous form in the cylinder, whereas nitrous oxide is primarily in the liquid state. The gauge in the oxygen cylinder therefore registers the contents in the cylinder fairly accurately. The gauge in the nitrous oxide cylinder, however, will tend to record the vapour pressure of the gas above the liquid in the cylinder. As long as there is any liquid in the cylinder of nitrous oxide the gauge will register full and will only fall when there is only gas left in the cylinder.

THE FLOWMETER

The gases pass from the reducing valve, via pressure tubing, to the flowmeter calibrated for each gas. The flowmeters record the volume of gas flowing to the patient per minute. There are various designs for the flowmeters. We will describe those used in the Boyle's machine.

The flowmeter in the Boyle's machine is referred to as a "Rotameter". It consists of a vertical glass tube tapered at the lower end. Rotating a knob at the base of the machine permits the entry of gases into the flowmeter. In the glass tube is an indicator or a bobbin. The height of the float in the tube indicates the flow of gases through the flowmeter. The flow should be read at the top of the bobbin.

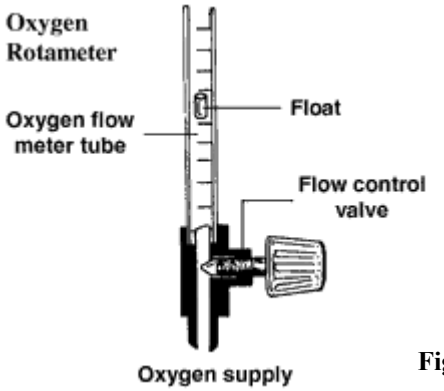


Fig 15.2 Oxygen rotameter

The indicator/bobbin has a rim on the top with grooves cut obliquely on it. The stream of gases entering the flowmeter impinges on these grooves and causes the float to rotate. It is **essential** that this float should rotate all the time. Sometimes grease and dust may cause the float to stick. The flowmeter cannot then be relied on to give an accurate reading.
Since the glass tube tapers with the narrow end at the bottom the markings on the flowmeter are not equally spaced. The markings at the lower end are further apart and the markings at the upper are closer together.

Two other makes of anaesthetic machine are **Ohmeda** and **Drager**.

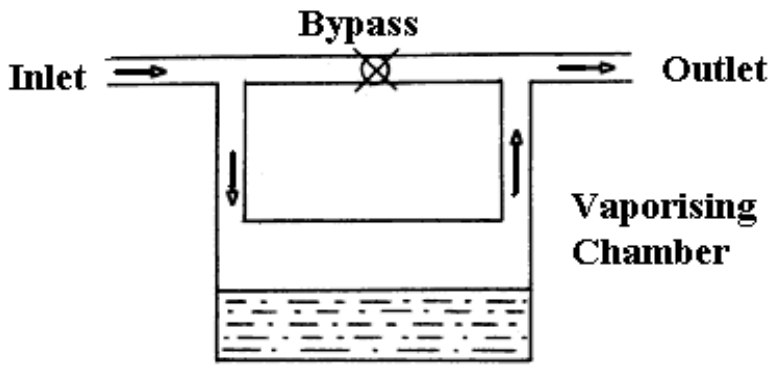


Fig 15.3 Vaporiser design

From the flowmeters the gases pass in the direction of the vaporisers. The vaporiser enables volatile agents to be introduced into the gaseous mixture. These volatile agents are liquids at room temperature and do not need to be stored under pressure. The function of the vaporiser is to vaporise this liquid. The ideal vaporiser should yield an unchanging (constant) concentration despite variations in gas flow, temperature and the amount of liquid anaesthetic contained in the vaporiser.

Boyle's bottle

The *simplest type* of vaporiser for an anaesthetic machine is the **Boyle's bottle**. The volume of gas diverted into the Boyle's bottle can be controlled by adjusting a lever. When this volume is at its maximum the vaporisation of the liquid can be further increased by depressing a "cowl" which brings the gases entering the Boyle's bottle closer to the liquid anaesthetic. The output of the vaporiser is not constant. As the liquid vaporises and its temperature falls then the evaporation of the liquid falls too and the concentration delivered to the patient drops with time. Hence this vaporiser is **not temperature compensated.**

This vaporiser is also **not calibrated**, so we cannot know how much is given to the patient with each position of the lever.

The Boyle's bottle can be used for ether and trilene. It can also be used for halothane but this is not recommended. Halothane is a very potent anaesthetic and should be administered very carefully in a specially designed vaporiser so that the concentration being administered to the patient can be known at all times.

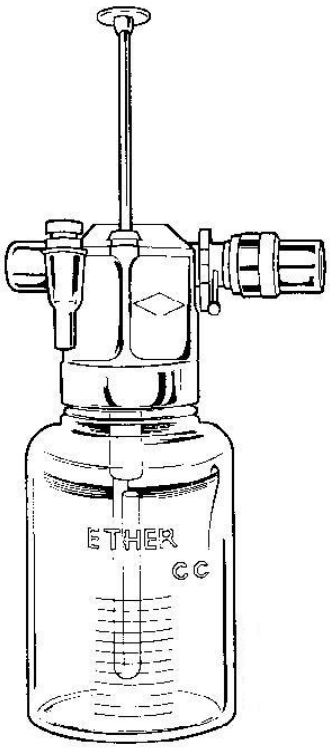


Fig 15.4
A Boyle's bottle suitable for vaporising Ether

Fluotec vaporiser - Mark II

The Fluotec vaporiser provides most of the features desirable in a good vaporiser.

- It is temperature compensated.
- It provides a constant concentration at various gas flows (4 to 15L/min).
- Its efficiency does not depend on how much liquid halothane is in the vaporiser.
- The control dial allows concentrations to be altered from 0 to 4% in 0.5% gradations.

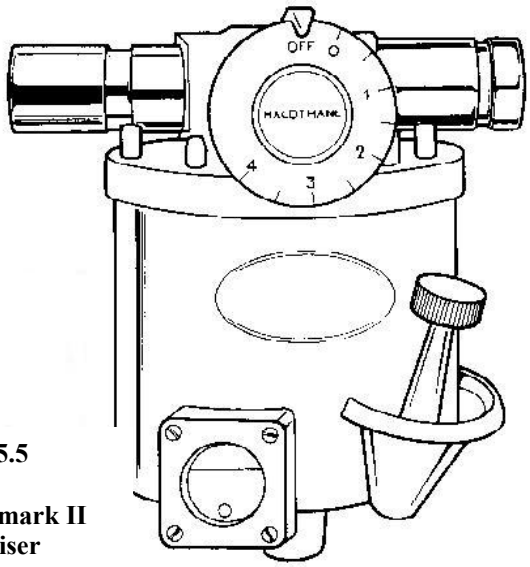


Fig 15.5

A Fluotec mark II Vaporiser

The Fluotec can be fitted on the back bar of the Boyle's machine. Rotation of a spindle increases the flow of gases diverted through the vaporising chamber. The dial must be pulled forward before it can be turned on.
It is important to remember that with controlled ventilation and low gas flows the concentration of halothane delivered may be greater than that shown on the dial. Use of more than 1% halothane with IPPV is best avoided if possible.

Fluotec Vaporiser - Mark III, IV, V

This delivers concentrations ranging from 0 to 5%. The concentration dial locks when it is turned to the OFF position. It must be unlocked before it can be turned to the ON position. A special filling device ensures that only halothane is used in the Fluotec Mark III vaporiser.
There are many variations on the 'TEC' concept made by different manufacturers. They are all temperature and flow compensated.

The Goldman Vaporiser

This is a simple glass bowl which will hold up to 20 ml of liquid.
The bowl is attached to a head. The gases are partly diverted into the vaporising chamber and the rest of the fresh gas flow travels through the bypass, depending on the position of the lever. There are three positions between the OFF and ON positions in the Mark I and Mark II models of the Goldman Vaporiser.

The Mark III model has two settings between the OFF and ON positions. The Goldman vaporiser is simple, easy to clean and can be used with multiple agents, e.g. halothane, trilene. The Goldman vaporiser is not ideal but is relatively safe, as it cannot yield a vapour strength greater than 3% halothane. Further, the small vaporising chamber prevents a dangerously high concentration of vapour being delivered to the circuit when the vaporiser is first turned on.

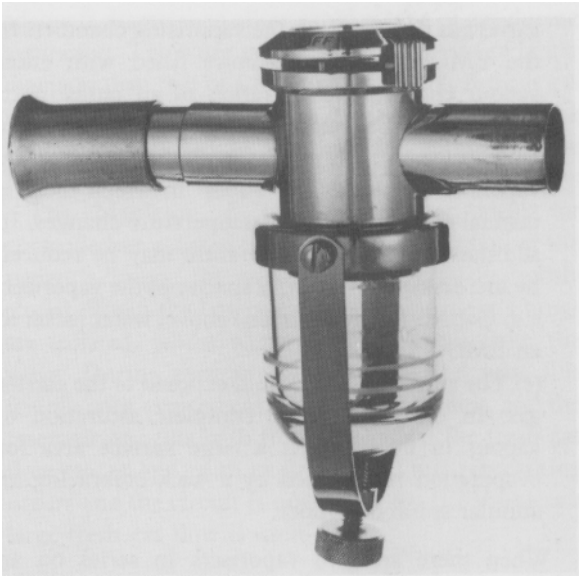


Fig 15.6 The Goldman vaporiser

THE DELIVERY SYSTEM

The commonly used delivery systems are:

- The Ayres T-piece
- The Magill's circuit
- The Bain circuit
- The circle system

The Ayres T–piece is discussed under Paediatric Anaesthesia in Chapter 20.

The Classification of Breathing Systems

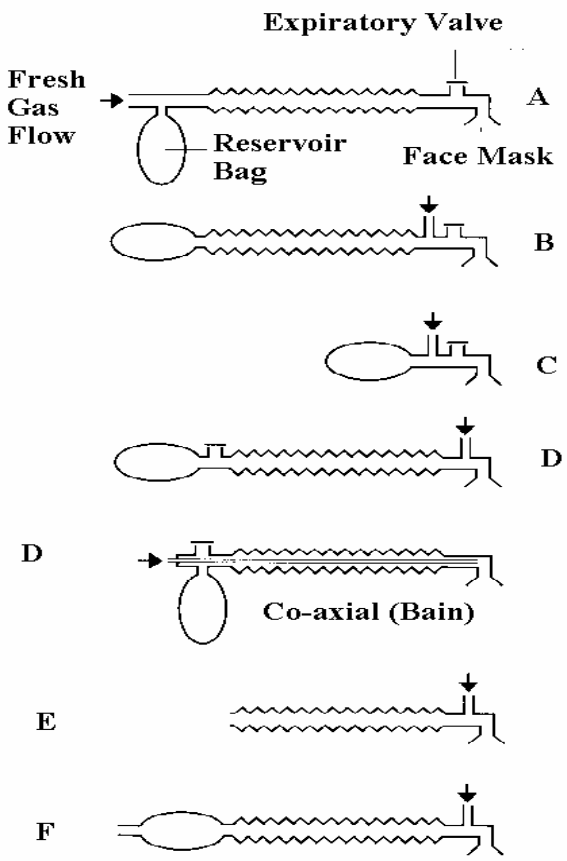


Fig 15.7 Mapleson's classification of breathing systems

THE MAGILL CIRCUIT

This is the Mapleson A circuit. It is suitable for adults and children over the age of 3 years where spontaneous respiration is to be used. It consists of a bag and an expiratory valve arranged as in the diagram. For spontaneous respiration the fresh gas flow should be equal to the patient's minute volume. The circuit is not suitable for IPPV because of the danger of rebreathing but if the expiratory valve is replaced with a non-rebreathing valve then the Magill circuit is suitable for IPPV. The fresh gas flow must be equal to the minute volume. Non–rebreathing valves can carry their own hazards, e.g. sticking of the valve.

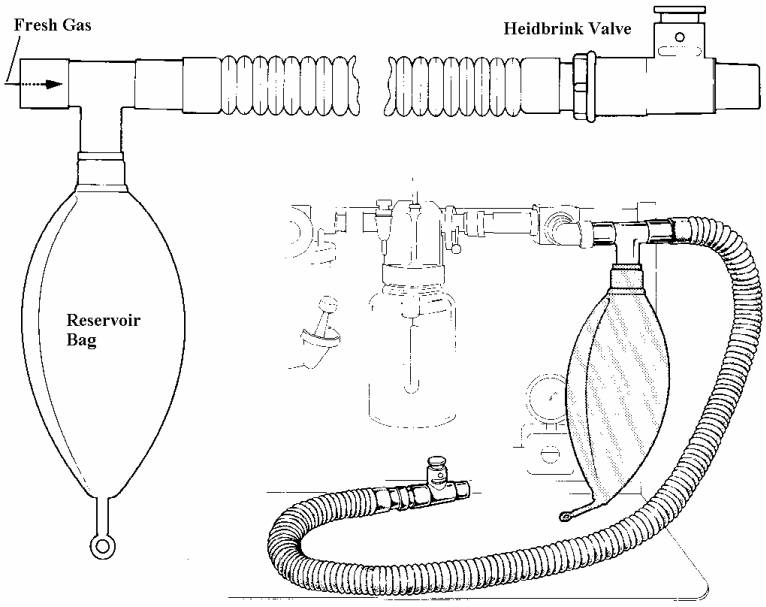


Fig 15.8 A Magill circuit

THE BAIN CIRCUIT

The Bain (co-axial) circuit is a modification of the Mapleson D system, in which fresh gas enters at the patient end but without any bulky inlet port. An expiratory valve and reservoir bag is placed at the other end. The Bain circuit is 1.8 metres in length and is composed of an outer tube made of light weight conductive corrugated plastic tubing 22 mm in diameter and an inner tube 7 mm in diameter.

The Bain circuit is connected to the anaesthetic machine using a special valve which allows fresh gas to be delivered to the patient through the small bore inner tube while exhaled gases pass through the outer tube to a reservoir bag and expiratory valve. A scavenging device is incorporated in the expiratory valve.

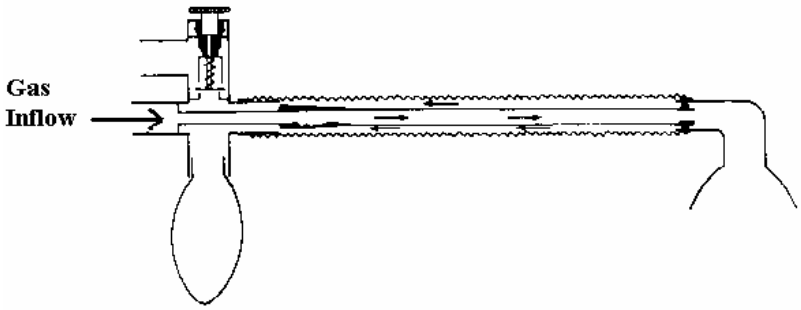


Fig 15.9 The Bain circuit

Fresh gas flow and the Bain circuit

The fresh gas flow in the adult patient requiring IPPV is 70 ml/kg. For patients less than 50 kg use a minimum flow of 3.5 L.
For spontaneous respiration, 100 ml/kg body weight is required. The circuit must be checked before use in case the inner tube has become dislodged. (See also Chapter 20 Paediatrics)

Advantages of the Bain circuit

- It is a single tube from the machine to the patient.
- There are no valves at the patient end so is less bulky and can be hidden under the drapes, e.g. in head and neck surgery.
- It is light in weight.
- It can be used for all ages.
- It can be used for spontaneous or controlled ventilation.
- It is easily sterilised.
- A scavenging valve is available for it.

THE CIRCLE SYSTEM

Principle: The circle system works on the principle that if carbon dioxide is absorbed from the anaesthetic circuit then the same oxygen and anaesthetic mixture can be used over and over again, with the addition of only minimal amounts of oxygen and anaesthetic agent.

By means of one-way (uni-directional) valves the oxygen and anaesthetic mixture passes into the patient and then the expired gases pass through a canister containing soda lime. The carbon dioxide is absorbed and the oxygen and anaesthetic gases and vapours pass again to the patient. Somewhere between the bag and the patient these gases are joined by the fresh gas flow from the machine. The circle system was first designed as a closed circuit. The valve (pop-off valve close to the mask) was closed completely and a very minimal flow of oxygen between 250-400 ml was used. However this took for granted that there was absolutely **no** leak in the circuit. In practice the circle system is used as a "semi closed" system, i.e. there is a small leak at the pop-off valve and higher gas flows are used. A total flow rate of 4 L/min nitrous oxide and oxygen is used at the beginning of the anaesthetic, in order to flush out the system and achieve a steady state. After three minutes, a total flow rate of 3L/min is used, comprising 1 L/min of oxygen and 2 L/min of nitrous oxide (N_2O).

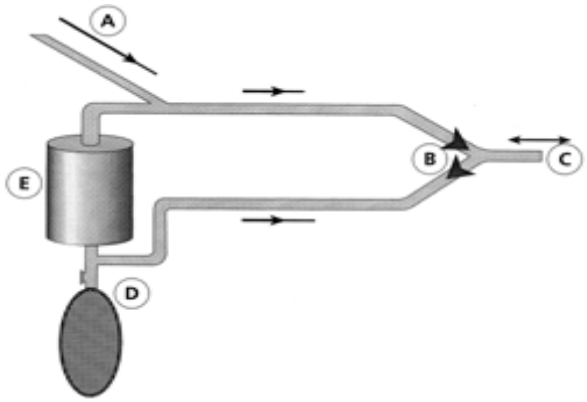


Fig 15.10 The scheme of a circle system

The Circle Absorber

In the canister the carbon dioxide (CO_2) produced by the patient is absorbed by either ***soda lime***, a mixture of 4% sodium hydroxide and 94% calcium hydroxide with a small amount of silica added to make the mixture hard or ***baralyme,*** a mixture of 80% calcium hydroxide and 20% barium hydroxide. Baralyme is sufficiently hard to prevent crumbling and the formation of alkaline dust, which sometimes may occur with soda lime.

Important points about soda lime

The chemicals in soda lime combine with carbon dioxide to form carbonates and water. Heat is released as a result of this reaction. The soda lime canister therefore feels warm.

The granules of soda lime are of a special size so that the maximum absorption is achieved with minimum resistance. An "indicator" is incorporated into the soda lime and this changes colour when the soda lime is exhausted. The canisters used may have single chambers or dual chambers.

The soda lime canister is filled to the brim. A single chamber canister of soda lime can be used for two hours continuously. After a canister of soda lime has been rested for several hours it can be used again for an hour and this can be repeated a few times. The soda lime must then be discarded. However, the exhaustion time of the canister depends on the size, the packing of the canister, the quality of the soda lime used, the fresh gas flow and the rate of carbon dioxide production. *The colour change of the indicator and the temperature of the canister should not be a substitute for careful clinical observation of the patient for signs of carbon dioxide retention such as a rise in heart rate and blood pressure, a deepening of respiration if the patient is breathing spontaneously, sweating and arrhythmias.*

Advantages and disadvantages of the circle system

Advantages

- The cost of anaesthesia is reduced because of the lower fresh gas flows required.
- Operating theatre pollution is reduced if a scavenging system is employed.
- Inhaled gases are warmed and humidified.
- The length of the corrugated tubing can be increased (e.g. in neurosurgery and surgery of the head and neck), giving maximum surgical exposure without interference by anaesthetic equipment.
- Bacterial filters, humidifiers and spirometers can be used in conjunction with the circle absorber.

Disadvantages

- The circle absorber is composed of many parts that can be disarranged or may malfunction.
- Resistance is greater than with other systems. It is therefore not suitable for use in very young children.
- The dead space extends from the Y-piece or the junction of the inspiratory and expiratory tubes. This too makes it unsuitable for use in very young children.
- Cross infection may be a problem as some of the components are difficult to clean.
- The system is bulky.
- Trilene cannot be used with soda lime as it produces a toxic substance.

CHECKING THE BOYLE'S MACHINE

- Check that adequate supplies of gas and oxygen are available before commencing an anaesthetic with the Boyle's machine.
- Check that ***both the cylinders*** of oxygen and nitrous oxide are not empty.
  Ensure that you have a reserve cylinder of oxygen available for replacement.
- Turn on the ***flow meter*** by turning the knob at its base. Make sure the bobbin rotates and does not stick.
- Check that the liquid anaesthetic to be used is in the ***vaporiser.***
- Make sure the soda lime is fresh (i.e. that the indicator has not changed colour). Make sure that the canister is filled to the brim. With the usual gas flows used, about 500g of soda lime in a small canister has to be changed at least every two hours. If a large canister with two compartments is used then the canister must be "reversed" when the indicator changes colour. It will last five times longer than the small canister.
- Check the machine and circuit for gas leaks. The expiratory valve is completely closed. Cover the opening at the patient's end of the Y-piece completely with the palm of your hand. The oxygen and nitrous oxide flow meters are adjusted to give a flow rate of about 4 L/min. The reservoir bag will fill and will not deflate when the bag is squeezed if there are no leaks in the system.

OTHER FEATURES OF ANAESTHETIC MACHINES

- **Alarm devices** are designed to initiate a signal when the oxygen pressure is low. Various designs are available. It is important that every anaesthetic machine be fitted with an oxygen alarm device.
  The alarm should be triggered by a low oxygen pressure and **not** by nitrous oxide flow. Alarm devices triggered by nitrous oxide flow will not function if the nitrous oxide and oxygen fail simultaneously **or** if there is prior failure of the nitrous oxide supply.

- **Oxygen flush valves** are available on most machines. It is important to be familiar with the position of the oxygen flush valve on the machine you are using. The valve directs a very high flow of oxygen (at least 30L/min) to the outlet of the machine. The valve is useful if it is necessary to fill the reservoir bag quickly with 100% oxygen.

- **Scavenging devices** have now become standard in operating theatres. They are discussed under the heading "Hazards in the Operating Theatre" (See Chapter 61).

CHAPTER 16

CONVENTIONAL ANAESTHESIC TECHNIQUES

Outline:

Making the choice of technique

General anaesthetic checklist

Spontaneous respiration

Controlled ventilation

Special techniques
- techniques used for patients who are likely to regurgitate or vomit under anaesthesia
- inhalational induction
- pre-oxygenation

CHOICE OF TECHNIQUE

The anaesthetic technique used will depend on several considerations. There is firstly the choice between a regional and a general anaesthetic. This will depend on:

- **The patient** – time of last meal, general medical state, general mental state.
- **The surgery** – the site of operation, the anticipated duration of surgery, the nature of surgery, (bleeding, intestinal obstruction etc), position in which the surgery is to be performed, the use of diathermy, the use of adrenaline by the surgeon.
- **The anaesthetic** – availability of anaesthetic gases, experience of the anaesthetist (especially in relation to regional techniques), cost.

Four important checks must be made. To help the memory we can call them the 4 Ms.

- The ***m***edical state of the patient.
- The time of the last ***m***eal.
- ***Medication***. This means that pre-operative medication, if ordered, must be given.
- The ***m***achine to be used must be checked, as described in the previous chapter.

If a general anaesthetic is decided on, then the most suitable anaesthetic technique using the gas machine must be chosen - either **spontaneous respiration** or **controlled ventilation.**

Whichever one *is* chosen, all of the items in the following list will be needed. Use it as a checklist.

GENERAL ANAESTHETIC CHECKLIST

Equipment which should always be available on the machine or trolley

- Laryngoscope
- Endotracheal tube (the cuff and connector must be checked)
- Syringe for inflating the cuff
- Artery forceps
- Catheter mount
- Mask
- Airway
- Suction apparatus (pharyngeal sucker)
- Stethoscope
- Strapping
- IV needle/cannula
- Blood pressure cuff
- ETT introducer
- Laryngeal mask, if available
- Oximetry, BP monitor, ECG and capnography if available

Anaesthetic drugs

- Thiopentone and water for injection or propofol
- Ketamine
- Suxamethonium chloride
- Pancuronium, vecuronium or any other long acting relaxant
- An inhalational agent e.g. halothane or ether
- Atropine sulphate
- Neostigmine
- Opioid for analgesia

Drugs for resuscitation

- Sodium bicarbonate
- Metaraminol (Aramine)
- Ephedrine
- Aminophylline
- Hydrocortisone
- Frusemide
- Naloxone
- Adrenaline
- Lignocaine
- Calcium gluconate or chloride

SPONTANEOUS RESPIRATION

Preliminary measures

- Check the four Ms. (See above)
- Make sure all the items of equipment are available and ready.
- Draw up the drugs to be used and label the syringes. It is essential to check at the beginning of every operating list, that all emergency drugs required are available.
- Check the patient's blood pressure and leave the cuff in place.
- Insert a cannula or start an intravenous infusion.

Induction

In the fit adult patient 2.5% thiopentone is used. This is prepared by mixing 0.5 g of thiopentone in 20 ml of water (1.0 g in 40 ml or 2.5 g in 100 ml). Each ml of thiopentone contains 25 mg. Dose of thiopentone is 4-5 mg /kg for a patient of average size. 200 mg (i.e. 8 ml) of this solution can be injected first. Test the eyelash reflex. If it is absent then it may be assumed that sufficient thiopentone has been given (though the patient often may require a further 3-4 ml). If the eyelash reflex is still present after 200 mg, then give further increments of 50 mg at a time and test the reflex.

Once the eyelash reflex has disappeared, no further doses are generally required. Test the eyelash reflex by brushing the eyelashes with a finger. The reflex is said to be absent when the patient does not blink when stimulated in this way. The loss of the eyelash reflex is taken to indicate the loss of consciousness. Loss of consciousness is often followed by a deep breath and a period of respiratory depression. Once normal breathing has resumed, **but not before**, another 2-3 ml of thiopentone may be given if necessary.

In the elderly or very ill or "poor risk" patient, induction may be carried out using IV ketamine 1mg/kg **or** etomidate **or** thiopentone in very small doses. Thiopentone must be used cautiously, bearing in mind the patient's prolonged circulation time. Inject 100mg (4ml) of thiopentone slowly. After a pause, test the eyelash reflex and if present inject another 25 mg of thiopentone. Repeat this pattern until the eyelash reflex is no longer discernible. Do not use more than 150mg of thiopentone in a premedicated elderly patient or a very ill patient.

To summarise the technique of induction for elective surgery:

Fit patient: Thiopentone; sleep dose of 200mg, increments of 50mg. Loss of eyelash reflex; period of respiratory depression. A further 50-100mg of thiopentone if required.

Poor risk patient: Ketamine IV 1mg/kg or etomidate 150 micrograms/kg or thiopentone. Sleep dose of 100 mg then PAUSE, then 25mg increment, then PAUSE. Repeat increment once more (total dose to 150mg maximum). No further thiopentone is required.

Maintenance

A clear airway is maintained by extending the head and lifting up the jaw. Place the mask on the patient's face. Use an oxygen flow rate of 3L/min and a nitrous oxide flow rate of 5L/min. Increase the concentration of halothane gradually starting with 0.5% halothane, to a maximum of 2% halothane. After 3 - 4 minutes check the tone in the jaw muscles. Once the jaw is relaxed, insert a Guedel airway of the appropriate size. If the patient does not cough or react to the airway lower the concentration of halothane approximately 1%. If the patient resists the airway (e.g. by coughing or holding the breath) it is best to remove it and re-insert it after the anaesthetic has been deepened. The following parameters must be monitored at least every 5 minutes and more frequently if necessary: blood pressure, pulse, colour and respiration. If oximetry is available monitor the SaO_2 (saturation of arterial oxygen) as well.

Uses of the technique

This technique can be used for limb surgery, hydrocele, "superficial" surgery of the chest or abdominal wall, curettage, cystoscopies and can be used for all herniotomies if a GA is required. It is not advocated for very prolonged procedures.

Dangers of the technique

- The airway may become obstructed, especially by the tongue falling back on the posterior pharyngeal wall.
- Spasm of the vocal cords may occur under the following conditions:
  - Premature surgical stimulation while the patient is still only lightly anaesthetised.
  - Direct stimulation of the vocal cords by secretions or vomitus.
  - Stimulation of the laryngeal area, by the insertion of an airway in a patient who is only lightly anaesthetised.
- Hypoventilation may occur if the technique is used for prolonged periods.
- There may be an overdose of halothane.

Technique using the LMA

An alternative technique would be to insert an LMA at the point where the patient's jaw muscles are relaxed and the Guedel airway is tolerated. Continue maintenance as before with the patient breathing through the LMA.

Advantages

- Provides a more secure airway with less risk of obstruction.
- The anaesthist's hands are free from holding the facemask and jaw throughout the procedure.

CONTROLLED VENTILATION

Preliminary measures

- Check the 4 Ms.
- Make sure all the items of equipment are available and in working order on the trolley.
- Draw up the drugs and label the syringes.
- Check the patient's blood pressure and leave the cuff in place.
- Insert an indwelling needle or cannula or start an intravenous infusion.

Induction

As described previously for the spontaneous respiration technique.

Intubation

Once the eyelash reflex is lost, give the patient suxamethonium chloride in a dose of 1mg /kg. Suxamethonium chloride (Scoline) paralyses the patient for approximately 3 to 5 minutes. After the relaxant is injected, the patient is managed as follows:

First minute

- Ventilate the patient with 100% oxygen.
- Insert a Guedel airway if needed when the jaw is relaxed.
- A flow rate of 6-8 L/min is used. The inflation rate is approximately 20/minute. The chest must rise and fall with each inflation.
- After an injection of suxamethonium fine muscle twitching (fasciculation) of the face and body muscles will usually be seen. When the fasciculation of the facial muscles stops, proceed to laryngoscopy and intubate.

Second minute

- Intubate

Third minute

- Auscultate the chest in the apex of both axillae to ensure the tube is correctly placed (breath sounds must be equal on both sides).
- Inflate the cuff of the tube with a syringe until the leak of air disappears (less than 5 ml of air should be enough).
- Insert a Guedel airway as a bite-block.
- Strap tube in place.
- Check the blood pressure.

If intubation has not been achieved after the second minute, ventilate the patient with oxygen for another minute before attempting intubation again. If the patient is breathing by this time (i.e. the last dose of relaxant has worn off) half of the original dose of suxamethonium may be given before a second attempt at intubation is made. Atropine 0.3mg IV is sometimes needed to block the bradycardia caused by the second dose of suxamethonium. It is important not to let the patient become hypoxic while intubation is being attempted. If still unsuccessful, consider waking the patient up and getting a more experienced person to help.

A maximum of three attempts at intubation is allowable. It is dangerous to continue trying to intubate. This leads to hypoxia and airway oedema, making possible ventilation more difficult. The priority at this point is maintaining adequate ventilation. Wake the patient up and review the options for anaesthesia. (See Chapter 21 for difficult and failed intubation)

Maintenance of anaesthesia

Begin with a gas mixture of nitrous oxide 5 L/min and oxygen 3 L/min. These flow rates are reduced after the first 3 to 4 minutes depending on the type of circuit being used. (See Chapter 15 for flow rates required with each circuit). When the patient shows signs of returning muscle power e.g. swallowing or breathing, in the fit adult 6 mg of pancuronium (Pavulon) may be given (See Chapter 12 Muscle relaxants). It is important to note the time the muscle relaxant is given on the anaesthetic chart. Further increments may be needed later. The anaesthetic will require to be supplemented and this will depend on the availability of drugs. Halothane 0.5%, enflurane 1% or isoflurane 0.5% may be added. If N_2O is not available then higher concentrations of inhalational agents may be required. For painful operations, boluses of IV opioid are given as well (i.e. morphine 1mg, pethidine 10mg, or fentanyl 20 micrograms for adults) so that the patient will be comfortable when they wake up. (See Chapter 55 Acute pain relief.)

Reversal

This is attempted at the end of the operation. Wait a minimum of 20 minutes after the last dose of relaxant before attempting reversal. Atropine 1.2 mg or glycopyrrolate 0.5mg and neostigmine 2.5 mg are used in the adult.

Rules for extubation

- The patient must breathe deeply and regularly before extubation is attempted. Neuromuscular function should have returned to normal.
- Administer 100% oxygen for at least 3 minutes before extubation to allow the nitrous oxide to be eliminated and to provide a safe reservoir for the patient.
- Suction the pharynx before and after deflating the cuff and also after extubation. Active reflexes will protect the airway after extubation.
- Do not extubate patients who are hypotensive or cyanosed until the underlying problem has been understood and corrected.
- It is very important that an active cough reflex be present after extubation in certain types of patients:
  - Patients who have had surgery of the upper respiratory tract and in whom post-operative bleeding may occur.
  - Patients with a full stomach, e.g. intestinal obstruction or a recent meal. In these patients, it is necessary to leave the tube in place until the patient is fully awake, i.e. responds to verbal stimuli.
- Give oxygen using the anaesthetic mask.
- If there is any risk of aspiration the patient should be extubated awake in the left lateral position. A high proportion of aspiration syndromes occur on extubation.

SPECIAL TECHNIQUES

RAPID SEQUENCE INDUCTION (RSI)

Technique used for a patient who is likely to regurgitate or vomit under anaesthesia

Preliminary measures:

Check the 4 Ms -

- The **m**edical state of the patient and the availability of blood
- The time of the last **m**eal
- The pre-**m**edication
- The **m**achine

Check the equipment required as described previously (page 229).

Draw up the drugs and label the syringes.

Take special precautions for patients with a "full stomach".

- Empty the stomach using a naso-gastric tube.
- Check the suction equipment, making sure it is turned on and the suction head is left tucked under the mattress of the operating table. It must be easily accessible and ready for use when needed.
- Check the cuff of the endotracheal tube. The inflating syringe is left attached to the cuff.
- Explain the principle of **cricoid pressure** to the assistant (and to the patient). Cricoid pressure is applied over the cricoid cartilage to compress the oesophagus that lies behind it. This prevents gastric contents rising up the oesophagus and contaminating the respiratory tract. The cricoid cartilage lies immediately below the thyroid cartilage (Adam's apple) which is easily palpable. The cricoid pressure is applied as the patient is being induced (i.e. before the loss of protective airway reflexes). The cricoid pressure is maintained until the patient is intubated and the cuff of the endotracheal tube is inflated and tested for the presence of a leak.

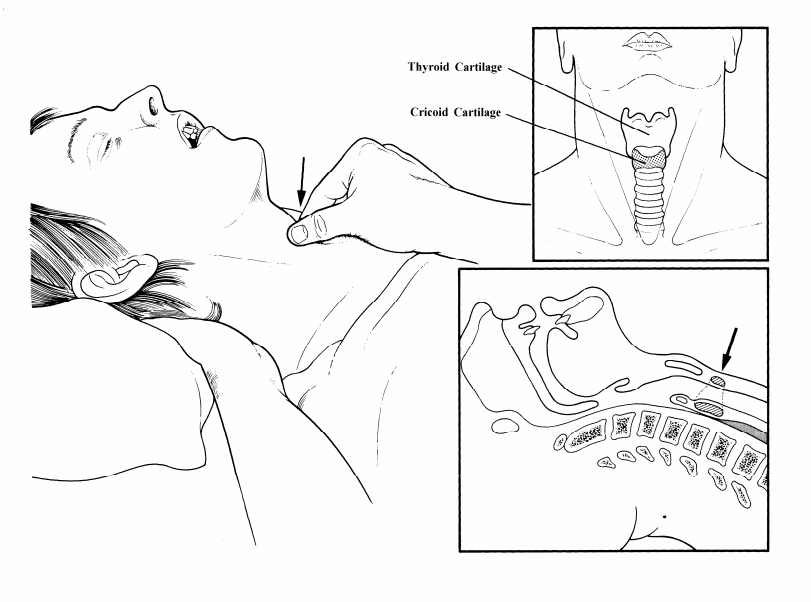


Fig 16.1 The technique of cricoid pressure

ANAESTHETIC TECHNIQUE – RAPID SEQUENCE INDUCTION

- Check the blood pressure.
- Insert an indwelling needle or cannula into the vein.
- Start an intravenous infusion if necessary.
- Place the patient on the table in the horizontal position.
- Give 100% oxygen for 4 minutes.
- Give a pre-selected dose of thiopentone (or ketamine), followed by a selected dose of suxamethonium. (This is the one instance where the dose of the induction agent is not titrated by testing the eyelash reflex).
- The assistant applies cricoid pressure and maintains this until the cuff of the endotracheal tube is inflated. (The patient is not ventilated with a mask before intubation).
- Insert the laryngoscope when the jaw is relaxed and intubate the patient.
- Inflate the cuff of the endotracheal tube. Hand ventilate the patient while the position of the tube is being checked by auscultation of the chest and upper abdomen. This is to ensure that the oesophagus or the right main bronchus has not been intubated. It is best to listen out in the axillae to ensure bilateral air entry. Strap the tube in place.

  Note: it is possible to hear transmitted sounds in the axilla from an oesophageal intubation. Capnography is extremely useful. If there are any doubts remove the tube and re-intubate.

- Connect the patient to the anaesthetic machine. Insert a Guedel airway as a bite block.
- Maintain the anaesthetic by air- nitrous oxide-oxygen-volatile-relaxant.
  - Give I.V opioid as required.
  - Monitor carefully for signs of inadequate anaesthesia.
  - Calculate and give IV fluids as necessary.
- Reversal
  - Atropine / glycopyrrolate and neostigmine are used in the dosage described above.
  - The rules for extubation are followed but extubate these patients when they are ***fully awake***.

INHALATIONAL INDUCTION

This is an alternative to intravenous induction in the following situations:

- Very young children where an experienced anaesthetist prefers this mode of induction.
- Patients with poor veins.
- Patients who request inhalational anaesthesia for fear of an injection.
- Patients who may have intubation problems and where the use of a relaxant may be dangerous, e.g. patients with known or possible airway obstruction after induction of anaesthesia. **Do not paralyse any patient who may be difficult to ventilate**. Partial airway obstruction can become complete airway obstruction on induction with loss of airway tone. An awake technique must be used.

Enflurane, halothane and ether are suitable agents for inhalational induction of anaesthesia. This technique will often be used with only oxygen or air but if N_2O is available it can be used in addition to the halothane or ether and may aid a smooth induction. The use of halothane as an induction agent is described under the pharmacology of halothane. If ether is being used as an induction agent it is important to remember that the concentration of ether must be increased very gradually. Start the induction with the lowest concentration of 1% and make the patient take 8 breaths before you increase the concentration by a further 1%. Induction is therefore a slow process.

It is important that the patient be deep enough before intubation is attempted, or laryngeal spasm will result. The patient must be able to tolerate a Guedel airway and breathe 2-4% halothane or 10-15% ether after the insertion of the airway for at least another minute before intubation is attempted. If when the airway is introduced, or later the laryngoscope, the patient swallows, holds breath, reacts in any way, withdraw the airway or laryngoscope immediately and deepen the anaesthesia.

An inhalational induction or intubation can never be a hurried process; be as comfortable as possible. For example sit near the head end of the table while inducing anaesthesia.

PRE-OXYGENATION

Many anaesthetists believe that all patients should be pre-oxygenated but oxygen should always be administered before the induction of anaesthesia in the following situations:

- Very ill or moribund patients.
- Patients with a full stomach (e.g.unfasted or with intestinal obstruction) in which a rapid sequence induction with cricoid pressure is used.
- Patients with possible intubation problems, where a slightly longer time interval may be required for intubation.
- Patients who are not desperately ill but who have some medical problem which cannot guarantee a normal degree of oxygenation by conventional anaesthetic technique, e.g. severe anaemia, chronic obstructive airway disease, obesity.
- Patients in whom even a small fall in the oxygen tension would have very adverse effects. For instance, those with
  - Sickle cell anaemia
  - Ischaemic heart disease
  - Liver disease
- Obese patients.

If a patient breathes 100% oxygen by mask, the nitrogen is eliminated from the lungs in 2 minutes, from the blood stream in 5 minutes and from the brain in 20 minutes.

The average healthy adult has some oxygen reserves in the body which will be utilised during a period of apnoea. The FRC (see Chapter 3) is the most important source of this and under normal circumstances i.e. breathing room air, allows approximately 90 seconds before hypoxia develops.
Four minutes of pre-oxygenation with 100% oxygen increases the FRC stores of oxygen significantly delaying the onset of hypoxia after apnoea by 3-4 minutes.

If pre-oxygenation is considered necessary, administer 100% oxygen by mask for 4-5 minutes.

Note: it is important to ensure a good seal of the mask to prevent dilution of oxygen with air.

CHAPTER 17

PHARMACOLOGY OF LOCAL ANAESTHETIC DRUGS

Outline:

History of local anaesthesia

Pharmacology of local anaesthetic drugs

Toxicity and treatment of toxic reactions

Individual drugs

Autoclaving drugs

Vasoconstrictors

HISTORY OF LOCAL ANAESTHESIA

Cocaine was the first local anaesthetic drug in use. It occurs naturally in South America. Cocaine, however, fell in popularity because of its toxicity and the danger of addiction. Procaine (Novocaine) was the next agent used fairly widely. This was followed by cinchocaine and lignocaine.

PHARMACOLOGY OF LOCAL ANAESTHETIC DRUGS

Several local anaesthetic drugs are available. It is important to become thoroughly familiar with the commonly used ones. ***Lignocaine*** (Xylocaine, Lidocaine) is now one of the most popular local anaesthetic agents. ***Bupivacaine*** (Marcaine) is used when more prolonged anaesthesia is required. ***Levobupivacaine*** (Chirocaine) and ***ropivacaine*** (Naropin) are recently introduced drugs with similar properties to bupivacaine but less toxic. ***Amethocaine*** is also useful and available in many countries.
Local anaesthetics are used mostly in the form of hydrochlorides. The alkaline tissues tend to release the bases which then combine with the nerve tissues. In infected tissues the acid of the pus makes these drugs less active. Local anaesthetics retard or stop the propagation of impulses along the nerves.

TOXICITY OF LOCAL ANAESTHETIC SOLUTIONS

Causes of toxic reactions

- The use of the drug in a dose greater than the safe maximum for that drug.
- The accidental injection of the correct dose into a blood vessel. This will result in a very high blood concentration.
- The use of the correct dose in a patient who is especially sensitive to the drug.

Factors affecting the toxicity of local anaesthetic agents

- Quantity of solution used
- Concentration of solution used
- Presence or absence of adrenaline
- Vascularity of the site of injection, e.g. absorption of the drug from the nose, pharynx and respiratory tract is very rapid
- Hypersensitivity to the drug (this is rare)
- Age, physical status and weight of the patient
- The presence of liver or renal disease

Symptoms, signs and emergency treatment of toxic reactions
(See also Chapter 25)

Central nervous system (CNS)

- Stimulation – tinnitus, circumoral numbness, nystagmus, restlessness, tremor, convulsions *followed by*
- Depression – depressed conscious level, coma

  Treatment
  - Give oxygen
  - Give sufficient diazepam or midazolam to control the convulsion. Thiopentone may also be used in a small dose (e.g. 50 mg).
  - If associated apnoea, give artificial ventilation with 100% oxygen following intubation with a dose of suxamethonium chloride.

Cardiovascular system (CVS)

- Pulse – bradycardia, arrhythmias, cardiac arrest
- Blood Pressure –hypotension, cardiovascular collapse

  Treatment:
  - Give IV fluids.
  - Give vasopressors and atropine, if necessary.
  - Give oxygen by mask.
  - Watch for and be ready to treat cardiac arrest.

Respiratory system

- Respiratory depression, apnoea.

  Treatment
  - Clear airway
  - Give oxygen by mask
  - Assist or control ventilation

Other side effects

Allergic phenomena (rare)
These may take the form of bronchospasm, urticaria, hypotension, etc.

Reaction to vasoconstrictor drugs
Pallor, tachycardia, palpitation, hypertension.

INDIVIDUAL LOCAL ANAESTHETIC DRUGS

COCAINE produces surface anaesthesia and intense vasoconstriction.
Its use is now restricted to ENT surgery. It is rapidly absorbed through mucous membranes. It should never be injected.
Cocaine should not be used on a patient having a general anaesthetic, unless ECG monitoring is available.

Systemic effects

CNS Stimulation.
Euphoria (a sense of well being) which may lead to addiction.

CVS Sudden cardiac standstill has occurred; cardiovascular collapse.

Muscle Curare-like effect.

Safe maximum dose *(adults):* 1.5 mg/kg by direct application to nasal mucosa.

LIGNOCAINE (Lidocaine, Xylocaine)
Lignocaine has a faster onset and longer duration than procaine. Lignocaine has low toxicity and is also used as an anti-arrhythmic.

Onset of action: 2-5 mins

Duration of action*:* Plain lignocaine: 45 minutes to 1 hour
Lignocaine with adrenaline: 1 to 1.5 hours

Concentrations of lignocaine commonly used:

- Topical: larynx, pharynx 2 – 4% or 10%.
- Infiltration: 0.25 – 0.5% with adrenaline 1/200,000.
- Nerve block: 1 – 2% with adrenaline 1/200,000.
- Spinals: 5% lignocaine for hyperbaric spinals.

Safe maximum dose:

Plain lignocaine:	200 mg (i.e. 3 mg/kg)
Lignocaine with adrenaline:	500 mg (7 mg/kg)

It is important to calculate the volume of local anaesthetic that can safely be used.

- 1% solution has 10 mg/ml
- 2% solution has 20 mg/ml
- 5% solution has 50 mg/ml

BUPIVACAINE (Marcaine) is a newer local anaesthetic drug. It is four times as potent as Lignocaine, so a 0.5% solution of bupivacaine is equivalent to 2% lignocaine.

Onset of action: 20 mins

Duration of effect: 5 to 6 hours or longer

Formulations: 0.25% plain
0.5% plain
0.25% with adrenaline 1/200,000
0.5% with adrenaline 1/200,000
0.5% heavy (with glucose)

Safe maximum dose: 2 mg/kg for plain bupivicaine
2.5 mg/kg for bupivacaine with adrenaline

LEVOBUPIVACAINE (Chirocaine) is an isomer of bupivacaine introduced recently. It has similar properties to bupivacaine but is less cardiotoxic.

Onset of action:	20 mins
Duration of effect	as for bupivacaine
Formulations	0.2% 0.5% 0.75% (not for use in obstetrics)
Safe maximum dose	2mg/kg

AMETHOCAINE HYDROCHLORIDE (Pontocaine, Tetracaine) belongs to the cocaine and procaine group of drugs. It is used to produce surface infiltration and caudal and spinal anaesthesia. Amethocaine is potent and relatively toxic. It may deteriorate on autoclaving.

Onset of action: 5-10 minutes

***Duration*:** 2 hours

The ***maximum dose*** that could be used for spinals is 15- 20 mg. It is mixed with equal quantities of CSF or 10% Dextrose.

LOCAL ANAESTHETICS USED FOR SPINAL in NON-PREGNANT PATIENTS

(Doses in the sitting position)

Drug	**Concentration (%)**	**Duration (hours)**	**Dose Range for fit adult**
Lignocaine	5	1.5	1 – 2 ml
Amethocaine	1	1.5 – 2	1 – 2 ml
Bupivacaine	0.5	5 – 8	2 – 3 ml

Fixation time

For spinals, using lignocaine: 5 to 20 minutes.
Amethocaine takes longer: a minimum of 10 minutes.

For Caesarean section under spinal anaesthetic see Chapter 19

EMLA is a local anaesthetic cream, a 50:50 mixture of lignocaine and prilocaine (25 mg of each per ml). It produces surface anaesthesia. A thick layer of cream is applied to the skin and covered with a plastic dressing.

***Onset of Action*:** > 1 hour

Indications

- IV cannulation in children and nervous adults.
- Any superficial but painful skin procedure, e.g. skin grafts.

Problems

- EMLA produces pallor, oedema and vaso-constriction at site of application.
- Absorption of drugs can be excessive, especially in neonates.

AUTOCLAVING LOCAL ANAESTHETIC SOLUTIONS

The commonly used solutions are very stable and it is possible to autoclave lignocaine, procaine and bupivacaine without any deterioration in potency. Repeated autoclaving of solutions used for spinal anaesthesia, however, can lead to some deterioration (especially with amethocaine) and therefore should be avoided. If the spinal anaesthetic solution changes colour it should be discarded.

VASOCONSTRICTORS

Local anaesthetic solutions are available with or without added vasoconstrictor drugs (adrenaline).

The local anaesthetic drugs (all except cocaine) dilate blood vessels. This results in an increased rate of absorption and a decreased duration of action on the nerves.

The vasoconstrictor drugs (e.g. adrenaline) produce vasoconstriction at the site of action of the local anaesthetic solutions. This results in an increased duration of action and a decrease in the absorption of the drug into the circulation. Hence, the side effects of the local anaesthetic drugs are minimised. A bigger total dose of local anaesthetic can be used.

The concentration of adrenaline used is 1/200,000 (i.e. 1mg of adrenaline in 200 ml of solution). The total dose of adrenaline used must not exceed 1mg (i.e. 1ml of a 1:1000 solution) for the adult patient.

Caution

- The use of adrenaline in spinal anaesthetic solutions is not recommended although it is sometimes added to lignocaine where there is not a longer acting drug available. There is a risk of causing permanent neurological damage.
- Adrenaline–containing local anaesthetics should not be used on fingers, toes, ears or penis.
- The use of adrenaline in surface anaesthetic solutions applied to mucous membranes does not prolong the duration of the block.

CHAPTER 18

SELECTED LOCAL ANAESTHETIC TECHNIQUES

Outline:

Advantages of local anaesthesia over general anaesthesia

Sites of action of local anaesthetic drugs

Preparation of patient for local anaesthesia

Selected local anaesthetic techniques:
- Axillary block
- IV Lignocaine block
- Field block for herniorrhaphy
- Penile block
- Caudal block

Spinal anaesthesia (See Chapter 19)
Dental blocks (See Chapter 25)

ADVANTAGES OF LOCAL ANAESTHETIC TECHNIQUES

Any operation that can be safely and successfully performed under local anaesthesia should be managed with a regional technique.

- The technique is simple, needing a minimum of drugs and equipment.
- There is less interference with physiological functions. It is especially useful in patients with cardiac disease, respiratory disease, hepatic and renal disease, endocrine problems, e.g. diabetes and thyroid disease, muscle disease, e.g. myasthenia gravis.
- There are fewer post-operative complications such as nausea, vomiting and respiratory problems.
- It is safer in the patient who has not fasted, as the cough reflex remains intact.
- Less post-operative care is necessary.
- It is cheap.
- There is no pollution of the atmosphere.
- There is less bleeding during surgery. This may be achieved by vasoconstriction (when adrenaline has been added to the solution) or by positioning a patient after a spinal or epidural block.
- Prolonged post-operative analgesia may be achieved by using a long-acting local anaesthetic such as bupivacaine.
- In minor surgery, the duration of stay in hospital is reduced.

SITES OF ACTION

Local anaesthetics are used to interrupt the conduction of painful impulses from the site of injury to the brain. This may be done at one of several points:

- Surface anaesthesia
- Infiltration anaesthesia
- Nerve and plexus block
- Epidural blocks
- Spinal (or subarachnoid) block

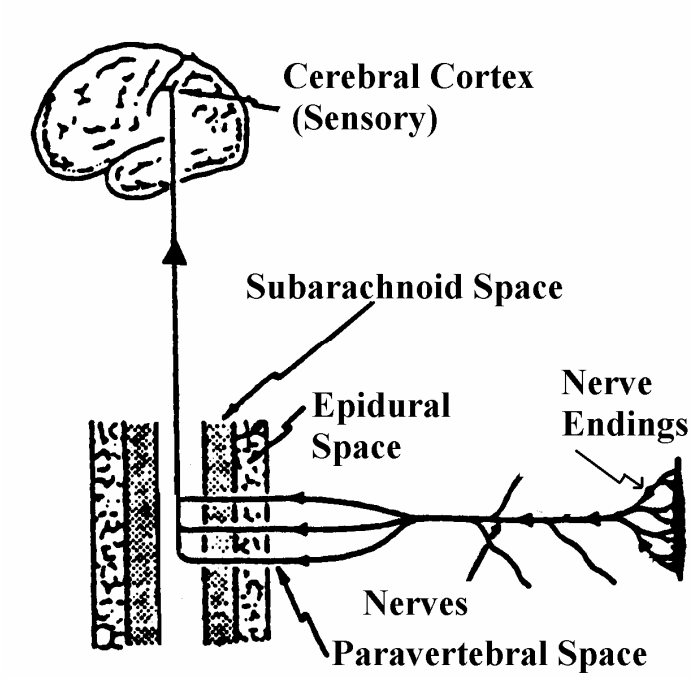


Fig 18.1 Sites at which conduction of pain impulses can be blocked

Surface anaesthesia

The nerve impulses can be blocked by applying the local anaesthetic on the surface of the mucous membrane in places such as the mouth, nose, pharynx, eye and urethra. This means that endoscopies, for example cystoscopies and bronchoscopies, can be performed under local anaesthesia.

Local infiltration

Subcutaneous injection of local anaesthetic can produce anaesthesia by blocking the nerve terminals. This is useful for suture of wounds and other minor procedures.

Nerve and plexus blocks

For a nerve block a small volume of local anaesthetic is injected near a nerve. The passage of impulses along that nerve is blocked. The nerve must be identified in relation to an anatomical structure such as a bone or artery.
A plexus block works on the same principle as a nerve block except that the plexus or collection of nerves is blocked and a larger area anaesthetised.

Epidural block

The local anaesthetic is deposited outside the dura mater. The site of action of the local analgesic solution is probably the nerve roots.

Spinal block

The local anaesthetic solution is injected into the subarachnoid space. The nerve roots in the subarachnoid space contain sensory fibres (posterior nerve root) and motor fibres (the anterior nerve root). They also contain autonomic fibres. The smallest diameter fibres are the most sensitive. The autonomic fibres are blocked to the maximum extent. The sensory fibres are next and the largest motor fibres are most resistant.

PREPARATION OF A PATIENT FOR LOCAL ANAESTHESIA

- The patient having a regional technique should be assessed preoperatively. Explain the technique to the patient and allow questions
- Fast adult patients for 6 hours; children for 4 hours. All patients may have clear fluids up to 2 hours before anaesthesia. (See Fasting guidelines chapter 7 p.93)
- Premedication is advisable especially in nervous patients.
- Access to a vein is essential. This may take the form of a needle or cannula placed in the vein or an intravenous infusion.
- The blood pressure must be monitored and the cuff left in place before the block.

- All equipment required to treat a toxic reaction to the local anaesthetic must be available.
- ECG and pulse oximetry monitoring equipment (if available) is used for all regional blocks.
- The total dose of local anaesthetic and adrenaline used must be calculated prior to the injection. Ensure this is within the safe maximum dose.
- The technique must be carried out under aseptic conditions.
- The patient who has had a regional technique must not be abandoned by the anaesthetist. The latter must be ever watchful for toxic effects of the drugs and complications of the technique.

SELECTED LOCAL ANAESTHETIC TECHNIQUES

- Brachial plexus block: axillary approach
- IV Lignocaine block (Bier's block)
- Field block for herniorrhaphy
- Penile block
- Digital block
- Caudal block

Local anaesthesia for Caesarean section is described in Chapter 21

BRACHIAL PLEXUS BLOCK

The upper limb is supplied by a collection of nerves referred to as the brachial plexus. The plexus is formed by the anterior primary rami of the 5th to the 8th cervical nerves and the 1st thoracic nerve. These nerves converge on the upper surface of the 1st rib, in close relation to the subclavian artery. The plexus of nerves then passes between the anterior and middle scalenus muscles into the neck. The nerves, together with the axillary artery and vein, are contained in a sheath of connective tissue. They traverse the axilla in close proximity to each other.

The brachial plexus can be blocked at three different points each giving a different level of anaesthesia.

The highest point is at the upper border of the 1st rib. This is called a ***supraclavicular block*** of the brachial plexus. It carries the risk of pneumothorax, stellate ganglion block, phrenic nerve block, haematoma formation and intravascular injection.

The plexus can also be blocked as it descends into the neck between the 2 scalenus muscles; this is referred to as an ***interscalene*** block of the brachial plexus.

Thirdly the plexus can be blocked in the axilla; this is termed an ***axillary block*** of the brachial plexus. The axillary block provides complete analgesia below the elbow joint.

Technique for axillary block of the brachial plexus

- Premedicate the patient appropriately, e.g. an analgesic or sedative such as an opioid or diazepam may be used.
- Start pulse oximetry and ECG monitoring if available.
- Insert a needle into the vein.
- Check the blood pressure and leave the cuff in place.
- Position the patient as follows:
  - Supine
  - Upper arm abducted at 90 degrees
  - The forearm flexed and externally rotated.
- Shave the axilla if necessary.
- Hands should be washed and gloves worn for the procedure.
- Draw up the local anaesthetic solution as follows:
  30-40ml 1% lignocaine with 1/200,000 Adrenaline,
  or 50ml 0.25% bupivacaine with 1/200,000 Adrenaline
  These doses could be used in a fit adult patient weighing 70kg.
- Attach the syringe containing the local anaesthetic solution to a 2in 23G needle via an extension tube.

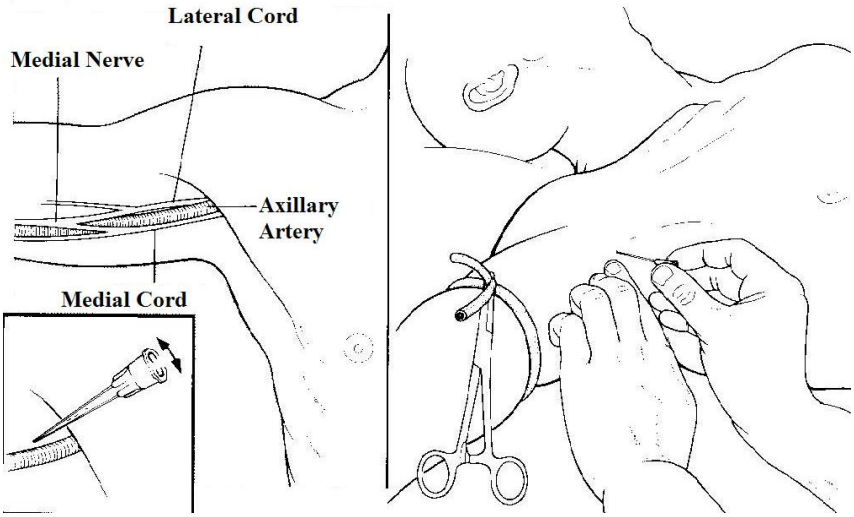


Fig 18.2 Anatomy and technique of axillary block

- An assistant applies a tourniquet (e.g. Penrose drain) distal to the site of injection to prevent the downward spread of the local anaesthetic solution. Contraindications to the use of the tourniquet are sickle cell anaemia and peripheral vascular disease. The block can still be undertaken but without the tourniquet.

- Palpate the axillary artery:
  - At the highest point of the axilla below the lower border of the pectoralis major muscle.
  - With the index finger the axillary artery is "fixed" against the humerus.
- Placement of the needle:

  One needle technique

  Insert a 23G needle very close to the axillary artery:
  - The pulsation of the axillary artery is transmitted to the needle.
  - A definite “click” may be heard as the needle enters the sheath.
  - Paraesthesia (electric shock or pain) radiating down the arm to the fingers confirms a needle placement close to the nerve.

  Two needle technique (This method is used by some anaesthetists but suitable needles may be difficult to obtain).

  Position two 23 G scalp vein needles as follows:
  - Immediately superior to the axillary artery.
  - Immediately inferior to the axillary artery.
  - After aspiration to ensure the needles are not in a blood vessel, inject half the volume of local anaesthesia into each needle while the other is closed off.
- If blood is obtained on aspiration, the needle must be withdrawn until the aspiration of blood ceases and then the local anaesthetic is injected.
- The last 3ml of local anaesthetic are injected as the needle is withdrawn (this blocks the intercostobrachial nerve).
- Leave the tourniquet on for 5-10 minutes.
- Two nerves may escape getting blocked:
  - ***The musculo-cutaneous nerve*** which supplies the lateral side of the forearm. It is important to inject as high as possible in the axilla in order to block this nerve. If it has not been blocked, inject 5ml of local anaesthetic solution at a point 2.5cm distal to the elbow crease and lateral to the biceps tendon.
  - ***The intercostobrachial nerve*** which supplies the medial half of the upper arm. This is blocked by injecting the last few ml of local anaesthetic as the needle is withdrawn.

Complications
- Intravascular injection.
- Infection.
- Haematoma formation.
- Injury to nerves.

THE IV LIGNOCAINE BLOCK (BIER'S BLOCK)

This is a very old technique which can be used for the arm or the leg. It is best used for operations below the elbow, especially hand injuries and infections.

Precautions

- This technique should not be used unless sickle cell anaemia is ruled out (the tourniquet can precipitate a sickling crisis).
- **Use plain lignocaine** (xylocaine, lidocaine). Adrenaline in the lignocaine can cause gangrene of the extremities. Prilocaine is a good alternative if available.
- Do not use bupivacaine for this block.
- **Have equipment ready to resuscitate the patient in case of toxic effects of the local anaesthetic.**
- Follow carefully the rules given for deflating the cuff.

Technique

- Premedicate the patient appropriately.
- Insert an indwelling cannula on the side opposite that to be blocked.
- Monitor the blood pressure on the side opposite that to be blocked. Use pulse oximetry and ECG if available.
- Place a blood pressure cuff as high as possible on the arm to be operated on and secure it with a piece of strapping at least 5cm wide.
- Insert an indwelling needle (scalp vein needle or cannula 21G) and secure it with a piece of strapping.
- Use an Esmarch bandage to exsanguinate the limb.

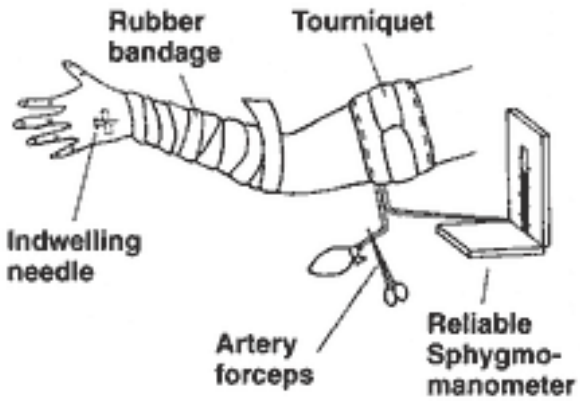


Fig 18.3 Bier's block

- Inflate the cuff to 100 mm/Hg above the systolic blood pressure. Clamp the cuff to prevent leakage.
- Inject 30-40ml of 0.5% lignocaine without adrenaline. This provides 30-40 minutes of operating time. (30 - 40 ml of prilocaine 0.5% could also be used, if available).
- Rules for deflating the cuff:
  - It is advisable to wait 20 minutes after the injection of the lignocaine before the cuff is deflated. Then the cuff can be deflated in one step, over 2-3 minutes.
  - If the cuff has to be deflated before the 20 minutes are up, the deflation must be done in steps, i.e. deflate quickly and then inflate again. This must be done several times before the cuff is finally deflated.
  - Always watch the patient very carefully for the next 15 minutes after deflation of the cuff for signs of toxicity of the local anaesthetic solution.
  - It is important not to leave the tourniquet on for more than 1 hour.

Dangers of the technique

- Absorption of the local anaesthetic into the circulation causing possible toxic reactions.
- The area under the blood pressure cuff may become painful, so it may be necessary to apply a second cuff distally (i.e. further down the arm) and deflate the first cuff after the second cuff is inflated. A double cuff, specially designed for this block, if available, is ideal.

Indications

Operations below the elbow.

Contraindications

- Psychologically unsuitable patients.
- Patients with peripheral vascular disease or neurological disease.
- Hypersensitivity to the local anaesthetic agents.
- Sickle cell disease.
- Children under 7 years.

INGUINAL FIELD BLOCK

Anatomy

Very briefly, the ***inguinal canal*** is 4cm long and extends from the external ring (which lies above and lateral to the pubic crest) to the internal ring. The posterior wall is formed by the fascia transversalis in its length and by the conjoint tendon in its inner two thirds. The floor of the inguinal canal is formed by the inguinal ligament. The roof is formed by the fibres of the conjoint tendon, curling over. A direct hernia is one that leaves the abdominal cavity through a deficiency in the wall. An indirect hernia is one that traverses the inguinal canal.

The ***nerve supply*** of the inguinal region comes from the last two thoracic and the first two lumbar nerves through the ilio-hypogastric nerve, the ilio-inguinal nerve and the genito-femoral nerve. The ilio-hypogastric and ilio-inguinal nerves arise from the 1st lumbar nerve and are blocked at a point one fingerbreadth medial to the anterior superior iliac spine. The genito-femoral nerve comes from the first and second lumbar nerves and divides into a femoral and genital branch. The genital branch enters the inguinal canal through the internal ring.

Technique of field block

- Weigh the patient and calculate the maximum safe dose and volume required for the patient's weight and general condition.

Maximum volumes and doses which should not be exceeded

	Small adult 50-60 kg	**Medium adult 60-70 kg**	**Large adult 70-100 kg**
0.5% lignocaine plain	30ml (150mg)	36ml (180mg)	42 ml (210mg)
0.5% lignocaine + adrenaline	70ml (350mg)	84ml (420 mg)	98ml (490mg)
1% lignocaine + adrenaline	35ml (350mg)	42ml (420mg)	49ml (490mg)
0.25% bupivacaine	40ml (100mg)	48ml (120mg)	56ml (140mg)

- Premedicate the patient appropriately e.g. opioid or diazepam.
- Insert an IV cannula or set up an intravenous infusion.
- Check the blood pressure and leave the cuff in place.

Volumes for varying weights using lignocaine 0.5% + adrenaline

Weight kg	1st weal	2nd weal	3rd weal	skin	surgeon
70-100	25-30ml	15-20ml	15-20ml	10-15ml	10ml diluted
60-70	20-25ml	15ml	15-20ml	10-15ml	10ml diluted
50-60	20ml	10-15ml	10-15ml	10-12ml	10ml diluted

For example for a sick or frail patient weighing 50 –60 kg the following regime would be suitable*. (See table above).*

- Draw up 70ml 0.5% lignocaine with adrenaline. This is done by mixing 17ml of 2% with 1/200,000 adrenaline plus 53ml saline.

  or

  35ml 1% lignocaine with 1/200,000 adrenaline plus 35ml saline.
- Prepare and drape the inguinal and scrotal region as for a surgical procedure.
- Wash your hands and put gloves on.
- Weals (small subcutaneous injections) are made at the following sites with local anaesthetic solution, using a 25G needle (sharp).
  - ***First weal*** 2.5cm medial to the anterior superior iliac spine
  - ***Second weal*** over the spine of the pubis
  - ***Third weal*** 1.25cm above the midpoint of the inguinal ligament

Through the first weal introduce a needle (23G slightly blunted) vertically backwards to pierce the aponeurosis of the external oblique with a click. (this means the external oblique aponeurosis has been pierced). Aspirate and deposit approximately 15 ml of solution. Through the same weal and still under the external oblique aponeurosis direct the needle medially (pointing to the midline) and inject 5ml of solution.

Through the second weal an intradermal and subcutaneous injection of approximately 12ml of local anaesthetic solution is made towards the umbilicus. A sharp 23G needle is used.

Through the third weal, insert a slightly blunted 23G needle perpendicular to the skin until you feel it “give”. Use up to 15 ml of local anaesthetic but before you inject, confirm the placement of the needle further by placing your little finger on the scrotal wall of the source side and introducing it through the external ring as far as the internal ring, so that the needle tip is right beside the little finger tip. The line of incision is infiltrated subcutaneously with 12 - 15 ml of local anaesthetic.

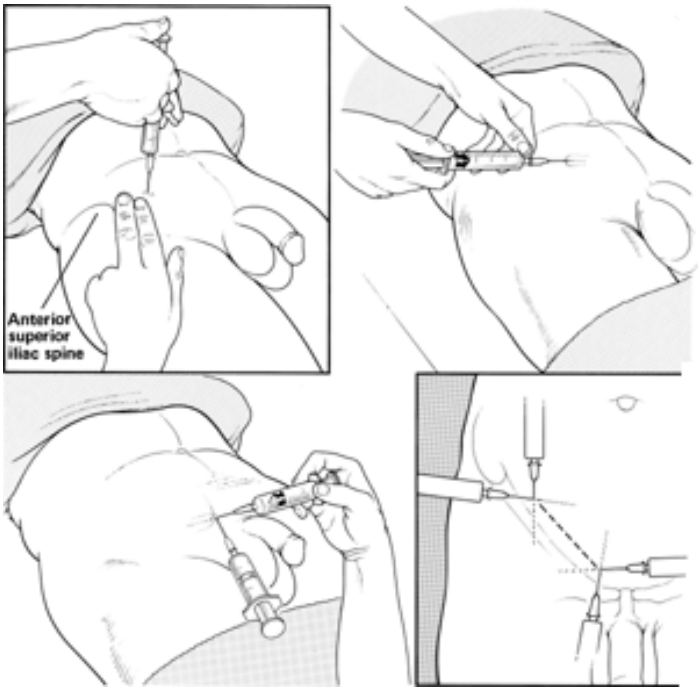


Fig 18.4 Inguinal field block

Keep the remaining 10ml of local anaesthetic sterile. It is diluted with an equal quantity of water to give 20ml of 0.25% Lignocaine. This is given to the surgeon in case the neck of the hernial sac has to be removed.

Indications for field block for herniorrhaphy

- In very sick elderly patients for whom the risks of a general or spinal anaesthetic are too great.
- Day care surgery.

PENILE BLOCK

This may be used as an alternative to a caudal block, for penile surgery such as circumcision. It can also be performed after the patient is anaesthetised in the supine position.

Anatomy

Sensation of most of the shaft and glans of the penis is transmitted by the dorsal penile nerves. These nerves with the accompanying arteries emerge under the pubic symphysis close to the midline and traverse the dorsum of the penis.

Method

Bupivacaine 0.5% plain is used in the following doses, administered using a 25G needle. **Adrenaline must not be used.**

0 - 12 months	1 ml	0.5%	plain bupivacaine
1 - 5 years	3 ml	0.5%	,,
6 - 12 years	4 ml	0.5%	,,
13 - 20 years	5-7 ml	0.5%	,,
> 40 years	7 ml	0.5%	,,

Cleanse the skin with hibitane in spirit. Use the 2nd and 3rd fingers of the left hand to palpate the lower border of the pubic symphysis.

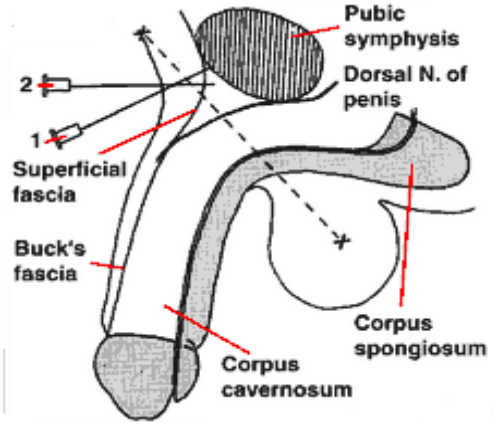


Fig 18.5 Penile block

Using the right hand insert the 25G needle attached to the syringe at right angles to the skin between the 2nd and 3rd fingers of the left hand, until bony contact is made. Then redirect the needle to pass just inferior to the lower border of the arch of the pubic symphysis but not deep to it. The blood vesssels run in the midline, therefore direct the needle either side of the midline, about 2mm below the inferior border. Aspirate the syringe and inject half of the local anaesthetic on either side.

Complications

- Direct IV injection and toxicity of the local anaesthetic.
- Haematoma due to puncture of the blood vessel.

DIGITAL NERVE BLOCK

There are two dorsal and two palmar or plantar branches of the digital nerve for each finger and toe. These can be blocked by injecting a ring of local anaesthetic at the base of the digit. A tourniquet can be applied after injection to localize the anaesthetic and reduce bleeding but should not be in place for more than 15 minutes.

Maximum volume 4ml (e.g. 1% lignocaine) **and no adrenaline should be used.**

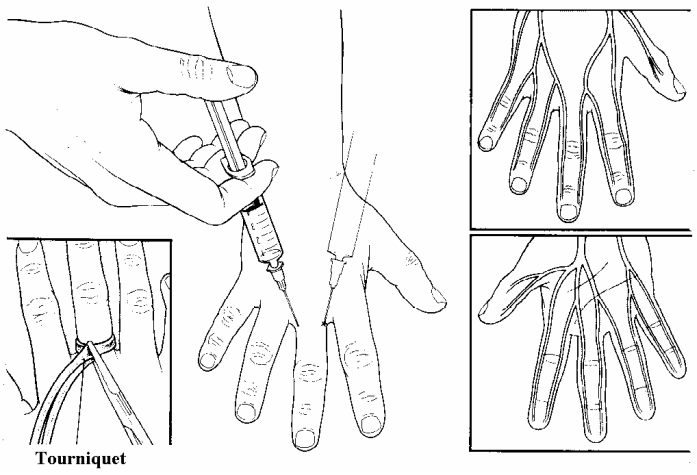


Fig 18.6 Digital nerve block

CAUDAL (SACRAL) EPIDURAL ANAESTHESIA

The local anaesthetic solution is injected into the sacral epidural space through the membrane covering the sacral hiatus.

Anatomy of the sacrum, sacral canal and sacral hiatus

The sacrum is a large triangular bone formed of 5 sacral vertebrae.
It articulates above with the body of the 5th lumbar vertebra and below with the coccyx. The front (pelvic surface) of the sacrum is concave from above downwards and from side to side. Anteriorly are four openings, the anterior sacral foraminae which transmit the first 4 sacral nerves. Posteriorly the sacrum is convex and down the middle runs the sacral crest with 3-4 rudimentary spinous processes. The laminae of the 4th and 5th sacral vertebrae have not fused posteriorly in the midline and this gives rise to the sacral hiatus. The tubercles of the inferior articular processes of the 4th sacral vertebra are prolonged downwards to form the sacral cornua. The rudimentary spine of S4 and the 2 cornua form the boundary of the sacral hiatus. Four pairs of openings on the posterior surface of the sacrum transmit the posterior branch of the sacral nerve. The lateral surface of the sacrum articulates with the ilium.

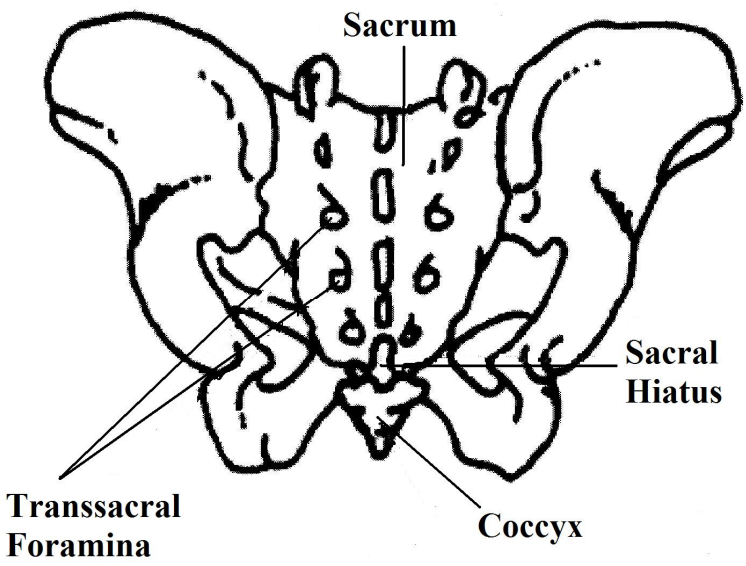


Fig 18.7 The anatomy of the sacrum

The sacral canal is a cavity within the sacrum. Superiorly it communicates with the lumbar vertebral canal. The lower extremity is the sacral hiatus covered by the sacro-coccygeal membrane.

Contents of the sacral canal

- The dural sac which ends at the lower border of S2 at the level of a line joining the posterior superior iliac spines.
- The pia mater which continues down as the filum terminale.
- Sacral and coccygeal nerves surrounded by a thick dural sheet.
- Venous plexus.
- Areolar and fatty tissue.

The sacral hiatus is a triangular opening caused by failure of the 4th and 5th sacral laminae to fuse. It is bound by the 4th sacral spine and the two cornua on either side. It is covered over by the sacrococcygeal membrane and it is pierced by the 5th sacral and coccygeal nerves.

Landmarks of the hiatus (adults)

- 4 to 5 cm above the tip of the coccyx.
- The upper end of the intergluteal cleft.

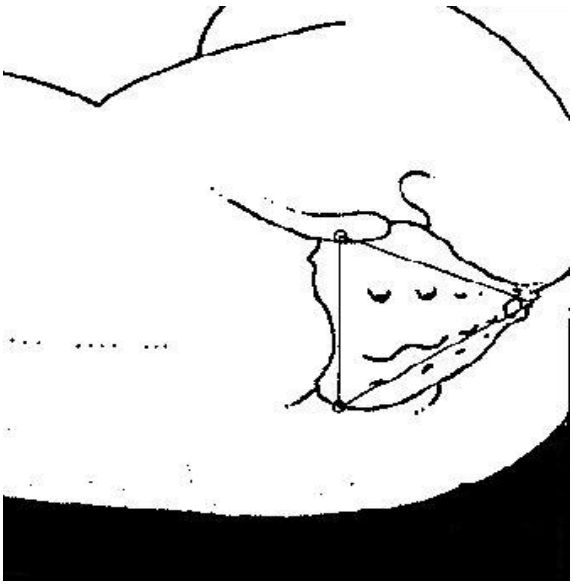


Fig 18.8 Landmarks of the hiatus

Technique

- Place the patient in the left lateral position with the knees drawn up to the chest.
- Identify and mark the sacral hiatus by:
  - Identifying the tip of the coccyx and palpating the hiatus 4 to 5 cm above it.
  - Palpating the hiatus at the top end of the intergluteal cleft.

  Another way of locating the sacral hiatus is to palpate the two posterior-superior iliac spines and form an equilateral triangle, the tip of which is at the point of the sacral hiatus.
- Swab the skin and drape.
- Raise a small weal over the hiatus.
- Insert a 21G needle 35mm through the weal at an angle of 20° from a line drawn at right angles to the skin surface.
- Once through the membrane, depress the needle until it is nearly horizontal towards the intergluteal cleft. Insert it into the sacral canal, keeping it in the midline.

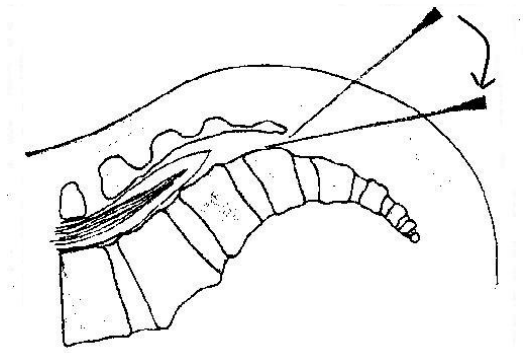


Fig 18.9 Needle placement for caudal injection

- The depth of insertion is very important. It must not penetrate the dural sac which ends at the lower border of S2. This is along a line joining the posterior superior iliac spines. The mean distance between the apex of the hiatus and the dural sac is 45mm. Remember the needle used must not be longer than 35mm but **note** that you can use an IV cannula size 20 for the purpose.
- Aspirate for blood and CSF.
- Inject a small test dose. Wait for 5 minutes and inject the rest.
- Signs of successful entry:
  - No swelling on the dorsum of the sacrum after injection.
  - No resistance to the injection.
  - The syringe and needle stay unsupported in the sacral canal.

Drugs and dosage

Adults 25-30 ml of 5% bupivacaine

or

20 to 30 ml of 1.5% lignocaine

Adrenaline is not recommended as it may cause spinal ischaemia.

Children 0.5ml/kg of 0.25% bupivacaine for lumbosacral block. If the volume is greater than 20ml it is recommended that the bupivacaine be diluted 3 parts local to 1 part saline (0.19% bupivacaine).

Advantages

- No post–operative headache (dura is not punctured).
- Less cardiovascular depression.
- Good anaesthesia and post–operative analgesia.

Disadvantages

- Length of time taken to develop analgesia.
- Less accurate control of analgesia.
- Technical difficulty: 10 percent fail because of anatomical abnormality.
- Risk of subarachnoid injection due to dural puncture.
- Drug toxicity possible because of absorption of large volume of local anaesthetic or inadvertent injection into the blood vessel.
- Hypotension.
- When a caudal is used on a mother in labour there is a danger of injuring the baby's head with the needle.

Indications

- Perineal operations not requiring anaesthesia of the anterior abdominal wall. Especially good for outpatients. Suitable for anal, gynaecological, urological and obstetric procedures.
- Post–operative analgesia after haemorrhoidectomy or circumcision.

Contraindications

- Infection over sacrum
- Bleeding problems
- Unco-operative adults
- Children unless under GA
- Caesarean section
- Anatomical abnormalities
- Gross obesity

CHAPTER 19

SPINAL ANAESTHESIA

Outline:

Anatomy

The spinal pack

Technique

The spread of the analgesic solution

Drugs and doses

Care of the patient under spinal anaesthesia

Complications:
- Immediate
- Later

Indications for spinal anaesthesia

Contraindications

Advantages

Disadvantages of spinal anaesthesia

Extradural block

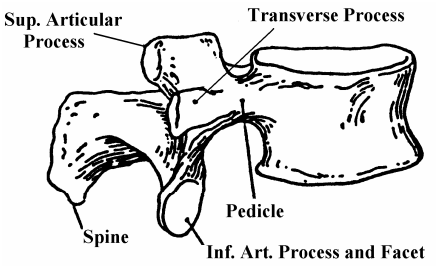
ANATOMY

The vertebral column consists of:

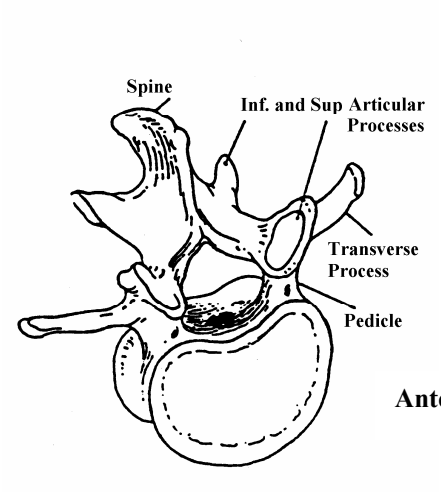
7 cervical
12 thoracic
5 lumbar
5 sacral
4 or 5 coccygeal vertebrae

A typical lumbar vertebra consists of

- A body which bears and transmits weight.
- An arch composed of a pedicle and a lamina. The pedicle is notched both inferiorly and superiorly to form part of the intervertebral foramen.
- 2 upper and 2 lower articular processes, arising from the junction of the pedicles and the laminae.
- A spinous process.
- Transverse processes.



Lateral



Antero–superior

Fig 19.1
A lumbar vertebra

Intervertebral disc

This is a fibro-cartilaginous disc that exists between two vertebrae.
In the centre is the nucleus pulposus. Surrounding this is the annulus fibrosus. Above and below this is a thin plate of cartilage.
If the spinal needle penetrates the nucleus pulposus it could herniate and cause chronic backache.

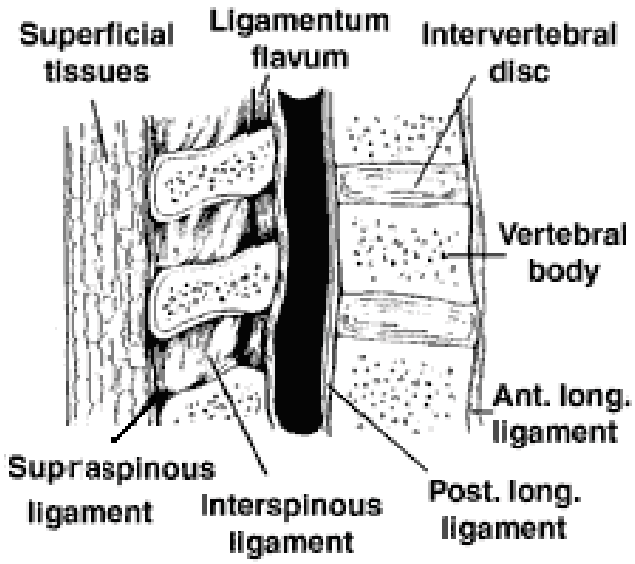


Fig 19.2 Cross–section of the vertebral column at lumbar level

The vertebral canal

This is bound in front by the vertebral bodies and the intervertebral disc. Behind, by the spines, are the supraspinous and interspinous ligaments on the sides by the pedicles, the laminae and the ligamentum flavum. There are two bony deficiencies or "openings" in the vertebral column which will enable a needle to be introduced into the vertebral canal.

- At the site of the intervertebral foramina. This is difficult. The nerves leave through this foramen.
- Posteriorly between the laminae. Half the posterior wall of the vertebral column is made up of bone (the laminae) and half by ligaments.

Vertebral ligaments

- Supraspinous ligaments extend from the tip of one spine to another.
- Interspinous ligaments extend between two adjacent spines.
- Ligamenta flava extend from one lamina to another lamina. They are composed of yellow elastic fibres. Half of the posterior wall of the vertebral canal is composed of bone (i.e. laminae) and half of ligaments (ligamenta flava).
- The posterior longitudinal ligament connects the posterior aspects of the bodies of the vertebrae.
- The anterior longitudinal ligament connects the front aspects of the bodies of the vertebrae.

The curvature of the vertebral column

There are four curves in the vertebral column: 2 curves are convex anteriorly, in the cervical and lumbar regions and 2 curves are concave anteriorly, in the thoracic and sacral regions. These curves determine the spread of local analgesic solution.

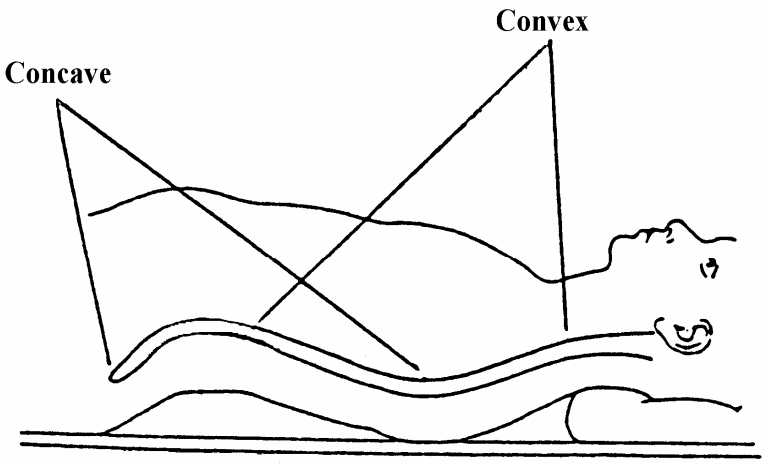


Fig 19.3 The curves of the vertebral canal

The spinal cord

The spinal cord is the continuation of the brain and occupies the vertebral canal. It extends from the upper border of the first cervical vertebra (the atlas) to the upper border of the second lumbar vertebra.

In the foetus, the spinal cord occupies the whole of the vertebral canal. However, the vertebral canal grows faster than the spinal cord and at birth the cord terminates at L3.

In the adult, the spinal cord occupies the upper two-thirds of the vertebral canal. It is 45cm long and ends in the conus medullaris, from the apex of which the filum terminale descends to the coccyx. Spinal puncture above L2 may result in cord damage.

Coverings of the spinal cord

These are three in number:

- ***The dura mater*** is strong and fibrous and ending at the lower border of S2, it is separated from the bony wall of the vertebral column by the extradural space. The dural fibres are longitudinal and this must be remembered when introducing the spinal needle. Ensure the bevel faces upwards (in the lateral position of lumbar puncture), so that the fibres are not divided but separated.
- ***The arachnoid mater*** is a very thin and transparent layer closely adhering to the dura mater.
- ***The pia mater*** is a vascular membrane closely adhering to the spinal cord. It invests the cord and sends septa into it.

The cerebrospinal fluid

The cerebrospinal fluid occupies the space between the pia mater and the arachnoid mater. This space is called the subarachnoid space. The local anaesthetic is deposited into it during spinal anaesthesia.

Spinal segments

The cord is divided into segments by the spinal nerves that leave it. Each segment gives off an anterior root and a posterior root. Each anterior root fuses with the corresponding posterior root at the intervertebral foramen.

Spinal nerves

There are 31 pairs of spinal nerves each arising by the fusion of the anterior and posterior nerve root. The anterior root is motor (i.e. it supplies the muscles). The posterior root is sensory.

The nerve roots are blocked by the analgesic solution during spinal anaesthesia. The analgesic solutions block the finest fibres first. The sensory nerves, being of smaller diameter than the motor, are blocked first.

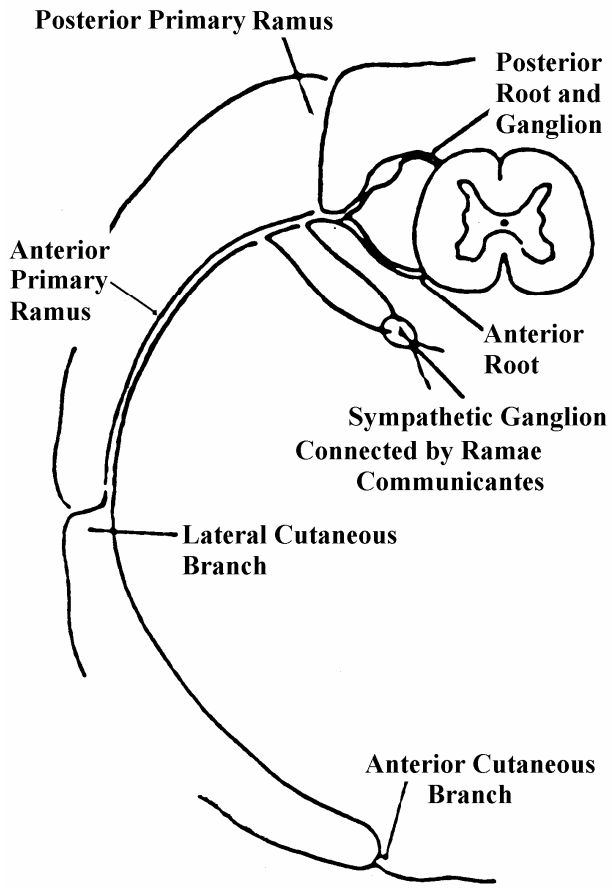


Fig 19.4 A typical spinal nerve

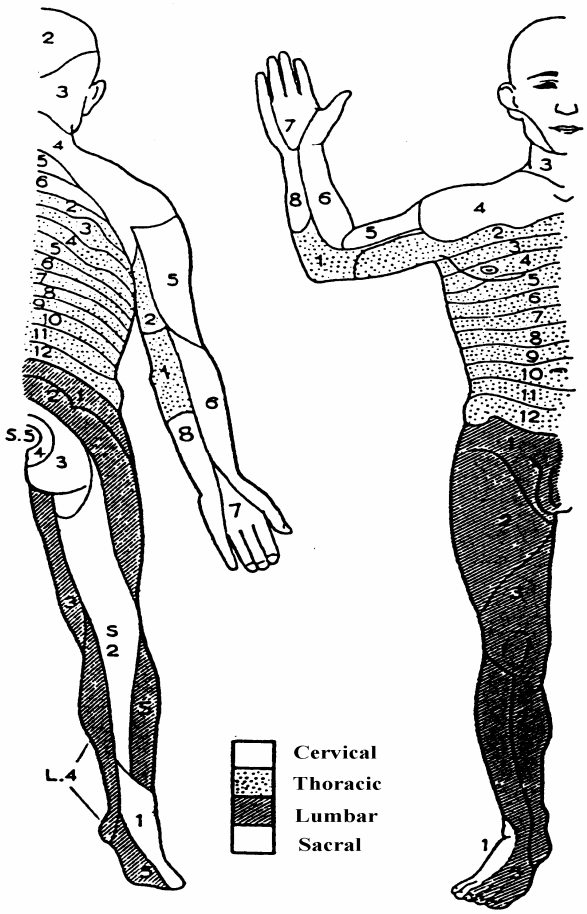


Fig 19.5 The segmental cutaneous nerve supply of the body

Segmental Levels

The following areas are supplied by the segmental areas of the spinal cord mentioned below:

Perineum	S1-4
Inguinal Area	L1
Umbilicus	T10
Xiphisternum	T8
Nipple	T4-5
Clavicle	C3-4

Structures penetrated by the lumbar puncture needle

Midline approach:

- Skin
- Subcutaneous tissue and fat
- Muscles
- Supraspinous ligament
- Interspinous ligament
- Ligamentum flavum
- Extradural space
- Dura mater and arachnoid mater
- Cerebrospinal fluid

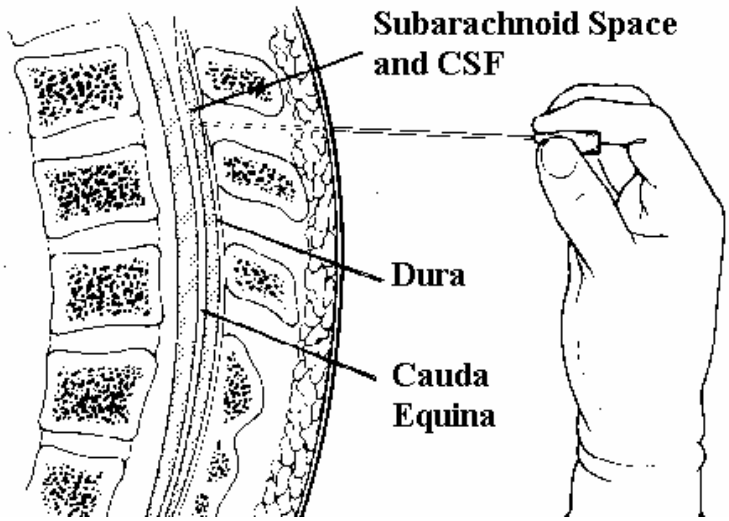


Fig 19.6 Path of needle during lumbar puncture

Lateral approach:

- Skin
- Subcutaneous tissue
- Muscles
- Ligamentum flavum
- Extra dural space
- Dura mater and arachnoid mater
- Cerebrospinal fluid

Average depth penetrated by the spinal needle is 4 to 5 cm. This is greater in the obese patient.

The spinal cord ends at the upper border of L2. Therefore make sure the lumbar puncture is done below this level, or else the cord may be damaged. In the elderly patient the supraspinous ligament may be calcified and the lateral approach may be easier.

THE SPINAL PACK

This is a collection of items to be kept ready for spinal anaesthetics.

The following list is an example of a basic spinal set but the contents may vary from hospital to hospital.

All sterile

- 1 fenestrated drape or 4 towels.
- 2 bundles of gauze swabs.
- 2 gallipots and 1 kidney dish.
- 1 spinal needle with stilette gauge 22 and/or 25G/27G, plus introducer (19 gauge needle).
- 1 23G hypodermic needle (skin analgesia).
- 1 18G hypodermic needle (for drawing up LA).
- 1 ampoule file.
- 1 swab holder.
- 1 ampoule local anaesthetic.
- 2 syringes: 5ml and 2ml. These must be glass if in a pack to be autoclaved.

Several such bundles should be kept in theatre, with the date of autoclaving marked on the seal and every unused pack re-autoclaved every week at a pressure of 20psi for half an hour. Most local anaesthetics can be autoclaved but should be discarded if the solution is discoloured.

Amethocaine (Pontocaine, Tetracaine) must not be re-autoclaved.

Chemical sterilisation is very dangerous. Never use needles or syringes soaked in spirit or any other antiseptic solution. It is ineffective and can cause neurological damage. Autoclave (or boil if altitude is less than 1000 metres).

TECHNIQUE OF SPINAL ANAESTHESIA

- Wash the patient's back (shave it if necessary).
- Check the blood pressure and leave the cuff on. Apply ECG and pulse oximetry monitoring, if available.
- Put in an indwelling needle or start an intravenous infusion if necessary.
- Make sure the following are available in the theatre before proceeding with the spinal anaesthesia:
  - Vasopressors (ephedrine or metaraminol)
  - Atropine
  - Oxygen
  - A gas machine or a means of ventilating the patient
  - Suction
- Select either the lateral or sitting position for the patient.

Lateral Position

Place the patient on his side, right or left. The buttocks and the shoulders should be parallel to the edge of the table. The nurse stands in front of the patient and places one hand behind the patient's neck and the other hand behind the patient's knees. The back is arched to open up the intervertebral and the interlaminar spaces.

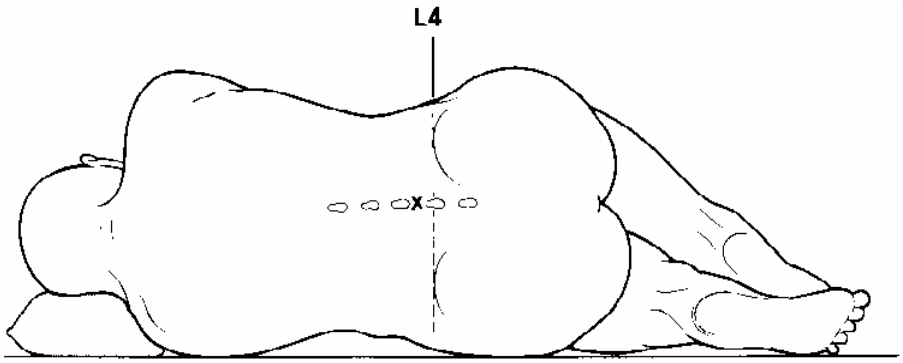
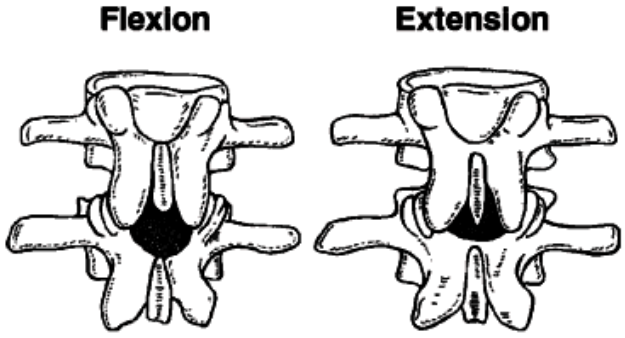


Fig 19.7 Patient in lateral position for lumbar puncture

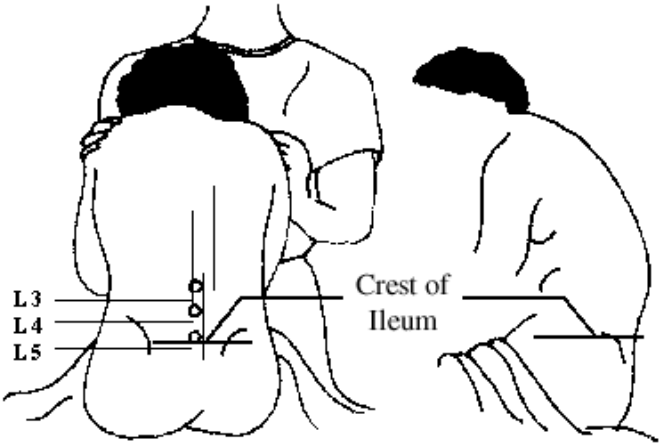


Effect of flexion & extension on the intervertebral space

Fig 19.8

Sitting position

The patient sits on the table. He is instructed to arch his back like a cat or to "push out" his back. The feet are placed on a stool, arms folded across the chest and the head flexed. A nurse supports the patient.



Ideal sitting position for spinal anaesthesia.

Fig 19.9

With the patient in an appropriate position

- Scrub as for a surgical operation. Wear a sterile gown and gloves.
- Swab the patient. Use a fresh swab each time and discard it after use. Swab the lumbar spine from above downwards three times. Betadine solution or any other antiseptic that is used to prepare the site of surgery in theatre can be used.
- Open the pack.
- Draw up the drugs. In the 2ml syringe, draw up the drug to be injected into the spinal space. In the 5ml syringe, draw up the drug for local infiltration of the skin. If using bupivacaine a 5ml syringe is needed and great care must be taken to identify the contents of each syringe.
- Drape the patient, preferably with a single "window" drape or with 4 drapes, 2 vertical and 2 horizontal, leaving a square in the lumbar region uncovered.
- Sit on the stool so that the site of the lumbar puncture is at eye level.
- Choose the site of injection:
  - Feel for the highest point of the iliac crest. This passes through the body of the 4th lumbar vertebra or the interspace between 4th and 5th lumbar vertebrae.
  - Choose the widest space between 2 spines with the above point as a guide. It does not matter exactly at which level entry is made, provided it is at or below the space between L2 and L3.

Technique for Midline approach (This is the most common approach)

The patient is positioned as described. The interspace between L3 and L4 or L4 and L5 is identified. Make a skin weal, then insert the spinal needle in the interspace at right angles to the skin.
Once through the ligament there is a distinct loss of resistance. The needle is advanced slightly forwards, to pierce the dura mater and the stilette should be removed to check for flow of cerebrospinal fluid through the needle. If the flow is poor, replace the stilette, rotate the needle gently, push it forwards or withdraw it slightly.

Always replace the stilette before advancing the needle, or skin or other tissue may be introduced into the CSF when the local anaesthetic solution is injected. Remember that unless the flow of cerebro spinal fluid is free, the block will not be satisfactory.

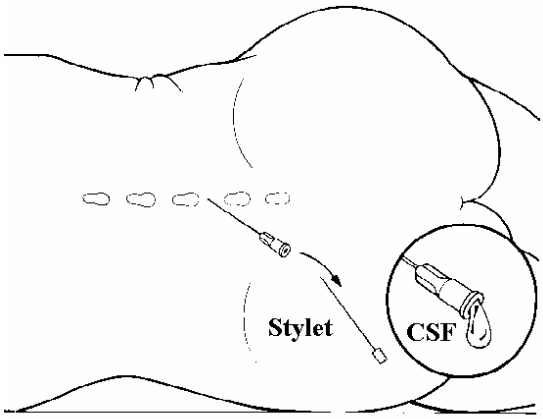


Fig 19.10 Removal of stilette and flow of CSF

The same approach, i.e. lateral or midline, can be used in the sitting position. The beginner is advised to commence spinal anaesthesia using a 22G needle. Once experience is gained a 23G needle and later a 25G needle can be used. The use of a 23G or 25G needle would necessitate the use of a 19G hypodermic needle as an introducer. Ensure the introducer is not inserted too deep as it may pierce the dura.
Using a 25G needle may require aspiration of CSF into the syringe.

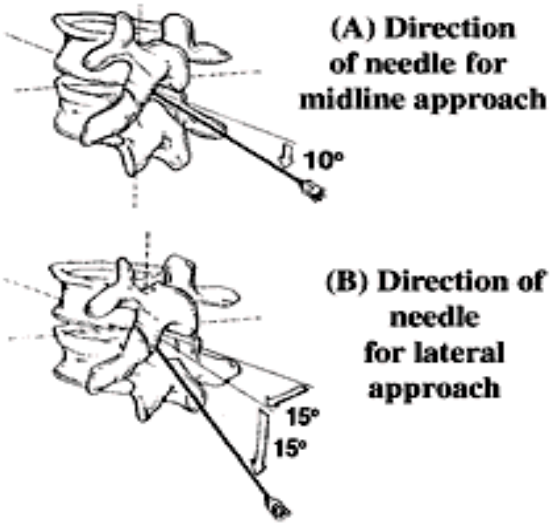


Fig 19.11 Midline and lateral approach

Technique for the lateral approach

Note the space between L3 and L4 or L4 and L5 spines in the midline.
Raise a skin weal at the selected interspace using a 23 gauge needle and 1 ml of lignocaine 0.5%.
Hold the lumbar puncture needle by the hub. With the patient in the lateral position the needle is inserted 1cm above the midline between two spines. It is directed inwards at an angle of 25 degrees to the midline. Once through the ligament there is a distinct loss of resistance. The needle is advanced slightly forwards, to pierce the dura mater and the stilette should be removed to check for flow of cerebrospinal fluid through the needle. As for the midline approach if the flow is poor, replace the stilette, rotate the needle gently, push it forwards or withdraw it slightly. **Always replace the stilette before advancing the needle, or skin or other tissue may be introduced into the CSF when the local anaesthetic solution is injected**. Remember that unless the flow of cerebrospinal fluid is free, the block will not be satisfactory.

Once the space has been identified and a free flow of CSF obtained

- Inject the drug, place a sterile swab over the site of injection and hold it in place with a piece of strapping. After the injection some of the drug is fixed to the nervous tissue and some is absorbed into the venous plexuses. Position the patient in such a way as to get the desired block.
- Remember a minimum of 10 minutes fixation time is required after injection.
- A blood stained CSF should clear after 5 or 6 drops. If it clears, proceed, if not, repeat at another interspace.

THE SPREAD of the ANALGESIC SOLUTION

The vertebral column if viewed from the side has a concavity in the thoracic region maximal at the 6th thoracic vertebra and a convexity in the lumbar region maximal at the third lumbar spine. At the usual site of injection most of the anaesthetic "rolls" down the lumbar curve. This is of little use for abdominal surgery where the thoracic segments must be anaesthetized, so when the patient is placed on the back, a slight head down tilt is employed to help the distribution to the thoracic segments. In women there is a natural tilt, because the female pelvis is broad and the shoulders are narrow.

The spread of local anaesthetic in the subarachnoid space is affected by

- Posture.
- The specific gravity of the solution in relation to CSF, i.e. whether the solution is lighter or heavier than CSF. This is really determined by what is added to the local anaesthetic solution. If the solution is heavier than CSF it tends to sink.
- The volume of the local anaesthetic solution injected.
- Speed of injection. With a fine needle it is difficult to inject fast enough to affect the spread of the drug.
- Barbotage: the aspiration of CSF into the syringe and then injection into the spinal space.
- Changes in pressure and volume of CSF during pregnancy reduce the volume of local anaesthetic required.
- Age. Smaller doses of local anaesthetic are needed with advanced age.
- The interspace chosen for injection. The higher the interspace the higher the spread of LA.

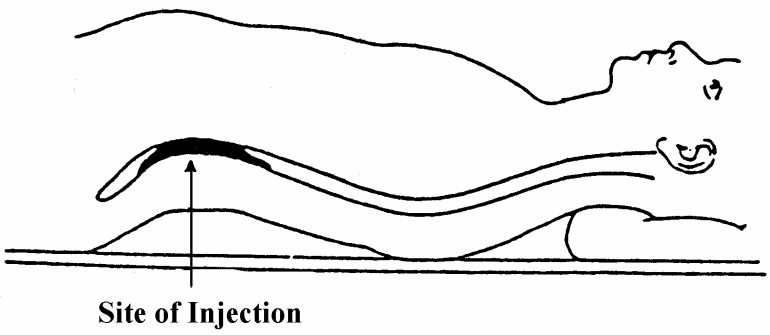


Fig 19.12 The effect of the curvature of the vertebral column on the spread of local anaesthetic

DRUGS AND DOSAGES FOR FIT ADULTS

Bupivacaine 0.5% plain (Marcaine)

2.5-3 ml may be used in the sitting or lateral position for lower limb and lower abdominal surgery.

Bupivacaine 0.5% hyperbaric

Heavy or hyperbaric bupivacaine is a 0.5% solution mixed with 80mg/ml dextrose. (SG 1.026)
This is the recommended choice for spinal anaesthesia and is the most predictable in onset and distribution of anaesthesia.

Dosage:	Perineal surgery (saddle block)	1-1.5ml
	Prostatectomy, lower limb surgery	2-2.5ml
	Hysterectomy, appendicectomy	3-3.5ml

Lignocaine 5% heavy

Perineal surgery (saddle block)	1-1.5ml
Prostatectomy, lower limb surgery	2ml

Amethocaine (pontocaine, tetracaine)

This may be used as crystals dissolved in CSF. It could also be used as a 1% solution dissolved in 10% glucose.
10-15 mg is normal for lower abdominal surgery, depending on the patient and the preparation used.
The maximum intrathecal dose is 20mg.

Never use a spinal anaesthetic for upper abdominal surgery.

For older patients (above 55 years of age)

- Start an intravenous infusion before the spinal.
- Give a minimum dose.

For Caesarean section (see also Obstetric anaesthesia Chapter 21**)**

Lignocaine 5%	1.2-1.5ml
Bupivacaine 0.5% heavy	2-2.5ml
Amethocaine	10mg (1ml)

Adrenaline in local anaesthetic solutions.

The advantages and disadvantages of the use of adrenaline, with the local anaesthetic, for spinals are considered with Obstetric anaesthesia in Chapter 21. The practice is not recommended.

CARE OF THE PATIENT UNDER SPINAL ANAESHESIA

In the operating theatre

- **Check the blood pressure** every minute for the first 5 minutes and every 5 minutes thereafter. Record it on the anaesthetic chart. The treatment of hypotension is discussed later.
- **Monitor the pulse rate** every minute for the first 5 minutes and every 5 minutes thereafter. If the pulse rate slows and if this is associated with a fall in blood pressure atropine may be required.
- **Respiration:** the depth and rate of breathing should be observed. The management of respiratory depression is discussed later.
- **Colour or oximetry:** Pallor or cyanosis require urgent attention.
- **Observe the general condition** of the patient carefully. Sweating, nervousness, nausea, dry retching, should be noted and appropriately treated.

In the recovery area and the ward

If otherwise stable a patient can be returned to the ward when there are signs of the spinal anaesthesia beginning to wear off.

The following vital signs should be observed for four hours after the return of the patient to the ward **or** as long as the spinal is still effective (i.e. as long as the patient is paralysed).

- **Record blood pressure** every 10-15 minutes for the first hour and half-hourly thereafter.
- **Record pulse** every 10-15 minutes for the first hour and half-hourly thereafter.
- **Record SaO_2** or **observe colour** every 10-15 minutes.
- **Observe respiration** rate and depth every10-15minutes.
- **Level of consciousness**
- **Position**: Patients are usually nursed lying flat for the first 6 –12 hours but may ambulate if a fine needle has been used.

COMPLICATIONS

The complications of spinal anaesthesia can be immediate or late.

Immediate complications

Cardiovascular system

There may be a fall in blood pressure. This is usually seen in the first twenty minutes and could be explained by several mechanisms.

- Sympathetic blockade resulting in vasodilation of the peripheral blood vessels. This causes a fall in venous return and a drop in blood pressure.
- The sympathetic nerves to the heart may be blocked. These nerves increase the rate of contraction of the heart. A block of the cardiac sympathetic nerves results in bradycardia and a fall in the cardiac output.

  Treatment of hypotension. In the event of a fall in blood pressure, exclude other causes e.g. blood loss, packs in the abdomen.
  If hypotension occurs:
  - Tilt the table head down. This increases venous return. This must only be done if the spinal is 'fixed', otherwise the legs could be elevated, manually or by raising the lower half of the table.
  - If blood pressure falls below 90mm Hg, increase IV fluids (Hartmann's solution). Infuse 500ml rapidly.
  - If blood pressure falls below 80mm Hg give ephedrine 5mg IV and repeat if necessary **or** aramine (Metaraminol) 0.25 - 0.5 mg IV in increments.
  - Give oxygen by mask.
  - Give 0.3mg of atropine IV if the hypotension is associated with a bradycardia. **Do not give** aramine (Metaraminol) if the pulse rate is less than 60 as it causes a reflex bradycardia. This dose of 0.3mg atropine could be repeated if necessary until a dose of 0.6mg has been given.
  - Always exclude and treat aorto-caval compression in obstetric patients.

Respiratory system.

The breathing may become slow and shallow, perhaps as a result of the intercostal nerves being blocked by the spinal anaesthesia. The breathing may actually stop owing to paralysis of the respiratory centre after a total spinal (see below). If respiratory depression occurs, the patient must be given oxygen by mask first. If the respiratory excursions are inadequate, assist respiration using a mask. If still inadequate, intubate the patient and control respiration.

Nausea and vomiting

Nausea and vomiting could be due to a variety of causes. Always attempt to find out the cause. It may be associated with any of the following:

- Hypotension. **Always** check the patient's blood pressure, if there is nausea or vomiting.
- Hypoxia.
- Traction on the nerve endings by the surgeon.
- Morphine or pethidine given as pre-medication.
- Nervousness, fear, etc.

Treatment

- Treat the cause. The main cause of nausea is hypotension. (See above for treatment.)
- Re-assurance.
- Encourage deep breathing.
- Phenergan IV slowly (if the patient's blood pressure is within normal limits) or IM.
- Metoclopramide (Maxalon) 10 mg IV or IM.
- Ondansetron 4 mg. IV.

Broken needle

A broken needle is a serious complication. Never apply force when inserting a needle into the subarachnoid space. If a needle does break leave the proximal part of the needle in place. Insert another needle along the tract. The patient must be X-rayed and the needle must be removed as soon as possible.

Total spinal

This may occur accidentally. If a "total spinal" occurs, it means that either an excessive dose of local anaesthetic has been given or, because of faulty positioning, the injected drug has affected the spinal nerves at a higher than expected level. It may follow the accidental injection into the subarachnoid space of drugs intended for epidural block. Occasionally the cause is not obvious.

The results of a total spinal are:

- The blood pressure falls. If this is severe a cardiac arrest may result.
- Respirations become shallow and may even cease.
- The patient becomes cold and blue.

Treatment is supportive until the spinal wears off:

- Ventilation with facemask or LMA.
- Tracheal intubation and IPPV (with sedation).
- Treat hypotension with fluid +/– vasopressor.

Systemic reaction to the injected local anaesthetic

It is rare for the local anaesthetic injected into the subarachnoid space to be absorbed in such quantities into the blood stream as to produce toxic effects. It may be associated with the following:

- A high dose of local anaesthetic.
- Accidental injection of a local anaesthetic into a blood vessel.
- Rapid absorption, perhaps because of an increased vascularity of the meninges.
- Decrease in the rate of breakdown of the local anaesthetic, e.g. in liver disease.

Later complications

Headache

There are many explanations given. The most widely accepted is that the leakage of CSF through the hole in the dura mater lowers the pressure in the subarachnoid space. This results in traction on the blood vessels etc.

Features of a spinal headache

- The spinal headache is different from any the patient has experienced before.
- It is worse on sitting up. It is relieved by lying down.
- External stimuli, such as light and noise, make the headache worse.
- The headache is mainly at the back of the head (occipital) and is associated with pain down the neck.
- It is relieved by increasing abdominal pressure.

The incidence of spinal headaches is related to the size of the needle.

Needle size	Incidence of headaches
20G	10–20%
22G	8–15%
25G	2–5%
26G	<1%

Prevention

- Use a fine needle.
- Make sure the fibres of the dura mater are divided and not cut by adjusting the bevel of the needle.
- Avoid multiple punctures.
- Nurse the patient flat for 6 hours post-operatively.
- Avoid coughing and straining post-operatively.

Treatment

- Reassurance
- Frequent long drinks
- Sedation and analgesia
- IV infusion if not contraindicated
- Epidural "blood patch" (see more advanced text books)

Backache

Treatment

- Small pillow under the lumbar region
- Sedation

Prevention: Avoid traumatic punctures.

Retention of urine

Treatment

- Nursing measures and stand patient out of bed if possible.
- If retention persists, catheterise.

Sepsis or infection

This may take various forms. All of them are serious.

- Extra dural abscess
- Meningitis (inflammation of the coverings of the brain and spinal cord)
- Encephalitis (inflammation of brain and spinal cord)
- Radiculitis (inflammation of nerve roots)

Meningism

This is an irritation of the meninges without actual infection. It presents like a meningitis and is diagnosed on a normal CSF tap. It is due to introduction of irritants into the subarachnoid space during the tap.

Paralysis of the cranial nerves

This is a rare occurrence.

The 6th cranial nerve (which is the longest) is the most vulnerable. The paralysis of this nerve presents usually as double vision, between the 5th and 11th post-operative day. It may be associated with headaches. It occurs in 1 in 300 patients. The cause of cranial nerve palsies is most likely due to the loss of CSF and traction on the unsupported brain.

Treatment: Dark glasses with a patch on the outer third of the glass over the affected eye may help. Fifty percent of patients recover within 1 month. Surgery must be performed to correct the double vision if recovery does not occur in 2 years.

INDICATIONS FOR SPINAL ANAESTHESIA

- Routine anaesthetic for surgery below the umbilicus in the fit patient.
- In a patient with a full stomach a spinal anaesthetic is safer than a general from the point of view of aspiration of stomach contents. If possible all patients should still be fasted according to the guidelines whatever type of anaesthetic is planned.
- Patients who have a contraindication or relative contraindication to a general anaesthetic e.g. acute respiratory tract infection, known difficult intubation / airway, severe liver or renal disease.
- In situations in which oxygen is available but must be conserved. **Remember that oxygen must be available for a spinal anaesthetic to be given.**
- In situations where cost is an important factor.

CONTRAINDICATIONS TO A SPINAL ANAESTHETIC

- Local infection over the site of injection.
- Raised intracranial pressure.
- Young children.
- Unco-operative patients and patients with chronic headaches.
- Very ill patients, e.g. those with reduced blood volumes as from:
  - Bleeding from any site.
  - Dehydration.
  - Shock.
  - Uncorrected hypotension from any cause.
  - Heart disease, e.g. unstable angina or recent myocardial infarction, severe aortic stenosis.
- Deformed backs (relative contraindication as may be more difficult but may be worth trying).
- Disorders of blood clotting.
- Multiple sclerosis.
- Enlarged prostate in which the surgery performed is **not** on the prostate. The spinal anaesthetic is sometimes associated with retention of urine. This would be more likely in patients with an enlarged prostate (relative contraindication).

ADVANTAGES OF SPINAL (AND EPIDURAL) ANALGESIA

- Simple technique. Epidural analgesia is a slightly more difficult technique than spinal and requires special needles.
- Cheap. The cost of drugs and equipment for a spinal is much less than for general anaesthesia.
- Safe. The patient is not exposed to a multiplicity of drugs, e.g. IV drugs, inhalational agents and relaxants. There is minimal danger of aspiration and airway obstruction.
- Minimal interference with physiological functions as the patient is conscious and breathing spontaneously.
- Good operating conditions, for instance, for intra -abdominal surgery below umbilicus:
  - Relaxed abdomen.
  - Good analgesia.
  - Reduced bleeding.
- No explosion risks.
- Fewer post-operative chest complications.

DISADVANTAGES

- Patient acceptability. Not all patients accept being awake.
- The procedure is not without complications. These have been discussed in detail.

EPIDURAL BLOCK

This is not discussed in detail. Please see other anaesthetic textbooks.
The local analgesic solution is deposited outside the dura mater. The local anaesthetic acts on the nerve roots.

Advantages of an EDB over a spinal

- No postoperative headache. However if the dura is punctured the incidence of headache is far higher, as a thicker needle is used.
- Less danger of infection in the CSF as the dura is not punctured.

Disadvantages compared with a spinal

- Technically the EDB is a little more difficult. The "end point", indicating the needle is in the epidural space, depends on the detection of the negative pressure. The "end point" in the spinal is CSF.
- Time taken to insert an EDB is a little longer than for a spinal.
- The onset of analgesia after an EDB is slower.
- A large volume of local anaesthetic is used for an EDB. This makes it more expensive and it increases the danger of toxic reactions.
- Accidental injection into the subarachnoid space of a large volume of local anaesthetic can be very dangerous.

It is recommended that provided there are no contraindications, for surgery below the level of the umbilicus, a spinal anaesthetic can be used as the standard method of anaesthesia.

CHAPTER 20

PAEDIATRIC ANAESTHESIA

Outline:

The differences between adults and children
Anatomical, physiological, pharmacological, psychological, pathological

Anaesthetic equipment used in paediatric anaesthesia
Breathing systems: Ayre's 'T' Piece, Jackson Rees modification, Bain circuit, Magill circuit, the Paedivalve and OMV/OIB

Other equipment
Laryngoscopes, endotracheal tubes and connectors, masks, airways

Anaesthetic management

Peri–operative fluids

Post–operative complications

Paediatric Basic Life Support
Advanced Life Support

THE DIFFERENCES BETWEEN THE ADULT AND CHILD

These can be classified as follows:

- Anatomical
- Physiological
- Pharmacological
- Psychological
- Pathological

Anatomical differences

- Children are ***small*** and they require special equipment of the appropriate size (the neonate is one twentieth the size of the adult). Drugs and fluids are given according to the weight of the child. All children should be weighed prior to surgery but the following table may be used as a rough guide to weight in healthy children.

Birth:	3 – 3.5kg
3 – 12 months:	weight (kg) = [age (months) + 9] / 2
1 – 6 years:	weight (kg) = [age (years) + 4] x 2

- The ***surface area is large*** in relation to weight. This means the child loses heat more easily and loses more fluid from the skin.
- The ***veins*** may be difficult to cannulate.
- The ***respiratory tract*** shows many differences and the child has limited respiratory reserve. Breathing in infants is relatively inefficient and takes a lot of the child's energy. Anything which makes the work of breathing harder will rapidly cause respiratory failure. Neonates and infants should be intubated and ventilated for all except brief surgical procedures.
  - The infant's head and tongue are large and the airway is easily obstructed. The older child may have large tonsils and adenoids. Under anaesthesia a clear airway is maintained by lifting the chin, using jaw thrust and avoiding compression of the floor of the mouth. An oropharyngeal airway of the correct size may be useful.
  - The larynx in the child is placed higher at (C3/C4) than in the adult (C5/C6). It is also more anterior. The epiglottis is large, floppy and is more easily seen using a straight blade laryngoscope, which lifts the epiglottis out of the way. This factor, together with limited respiratory reserve may make intubation difficult in infants.

- The narrowest part of the adult larynx is between the vocal cords. In the child it is lower down at the level of the cricoid cartilage. The implication of this is that the tube may pass quite easily between the cords but fit tightly at the level of the cricoid, resulting in laryngeal oedema post-operatively. Avoid using a tracheal tube which is too large.
- The trachea in children is relatively short and it is easy to put the tracheal tube down too far. The position of the tube should always be checked by listening with a stethoscope.
- The airways are small and easily blocked by secretions. A clear nasal airway is especially important for small infants as they breathe predominantly through their noses while feeding.
- In infants the ribs are horizontal and elastic, the lungs are very compliant and distal airway collapse is common. The diaphragm is an important muscle of respiration – abdominal distension will splint the diaphragm and make respiration inefficient. A nasogastric tube should be passed to relieve abdominal distension.
- The apparatus dead space is relatively large compared with the small tidal volume of the child (7–10 ml/kg). Dead space and the resistance of the breathing circuit should be minimised to reduce the work of breathing.

- The ***central nervous system***. In the infant the spinal cord ends at the level of L3 (L1 in children > 1 year and adults). Lumbar punctures should be performed at the level of L5 / S1 in infants.

Physiological differences

- Energy metabolism. The ***basal metabolic rate is higher*** in the child than the adult. The oxygen consumption in the neonate is 7ml/kg compared with 4 ml/kg in the adult. Infants rely on a rapid respiratory rate to provide adequate minute ventilation (resting respiratory rate is twice that of adults). Interruption of breathing for any reason results in rapid desaturation.
- The ***respiratory system.*** The respiratory centre in newborns is immature and they are prone to stopping breathing (apnoeas) for the first few weeks of life, especially if they become hypoxic. Young infants are extremely sensitive to respiratory depressants (e.g.opioids).

- The ***cardiovascular system***
  - The heart in infants is immature and sensitive to the depressant effects of anaesthetic agents.
  - The cardiac output is high. The cardiac stroke volume is relatively fixed and cardiac output is maintained by a relatively high resting heart rate (at least 120 beats/min in infants). Bradycardia results in a rapid fall in cardiac output.
  - Vagal tone is well developed in infants and they are prone to reflex bradycardias (intubation, hypoxia, drugs). Atropine is useful as a premedication or should be readily available.
  - The blood pressure in newborns is 60-90mmHg and increases with increasing sympathetic tone to reach adult levels by 10 years of age. In measuring BP in a child, it is important to use a cuff of the correct width for the arm or leg. It should be the largest size which will fit the limb.
  - The blood volume, which is calculated on the basis of the body weight, may appear very small. For example: If a child weighing 25 kg has a tonsillectomy, then a loss of 200 ml would constitute 10% of total blood volume (25 kg x 80 ml = 2000 ml). If an adult lost 200ml of blood it would not be significant but a child losing that amount would need a transfusion.
  - Allowable blood loss may be small and will depend on the child's starting haemoglobin and the circulating blood volume. Blood transfusion should be considered when there is a 10–15% loss in blood volume. Circulating blood volume is calculated using the formula:

	Circulating blood volume
Newborn	90ml/kg
Infant	85ml/kg
Child	80ml/kg
Adult	70 ml/kg

- ***Fluids and electrolytes.*** Isotonic solutions (Ringer's or 0.9% saline) should be used during surgery for all children. Neonates have limited glycogen stores and are prone to hypoglycaemia. Added dextrose (5% dextrose in Ringer's or 0.9% saline) should be considered for neonates and other children requiring a dextrose infusion prior to surgery to maintain blood glucose. Children are prone to iatrogenic hyponatraemia, and postoperative fluids should be given as 0.45% saline or 0.9% saline with dextrose.

  Renal function in infants is immature. They produce large volumes of dilute urine and can become dehydrated if fasted for prolonged periods of time. Conversely fluid requirements may be small in absolute terms and fluid overload should be avoided.

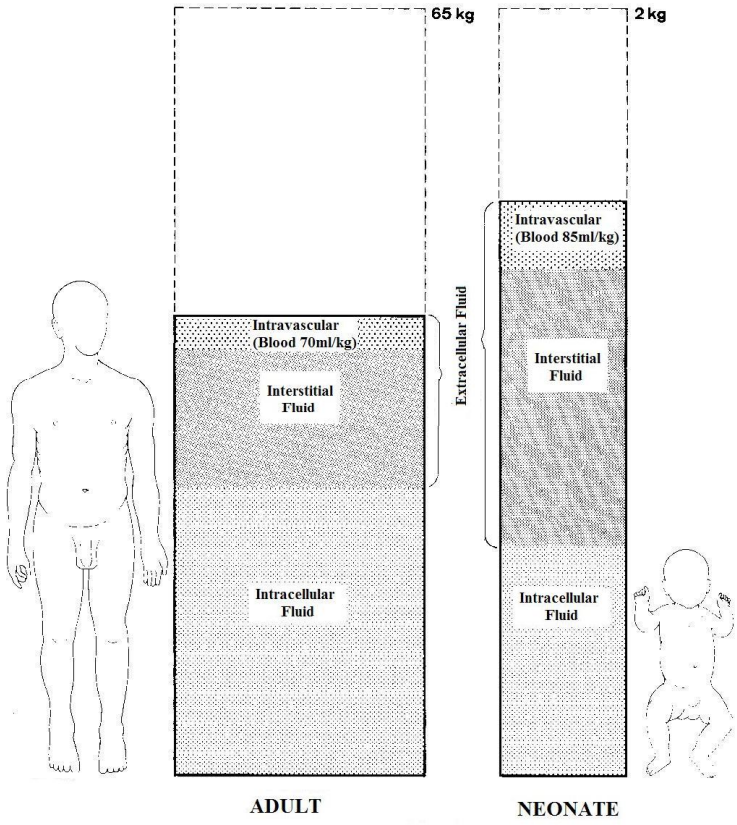


Fig 20.1 A comparison of fluid compartments of the body between adult and neonate

- ***Temperature control***. Infants have immature thermoregulatory mechanisms and are prone to heat loss, especially under anaesthesia, due to vasodilatation and loss of shivering. The body temperature tends to follow that of the environment and active measures should be taken to minimise heat loss, at the same time avoiding hyperthermia. The dangers of hypothermia include clotting abnormalities, delayed drug metabolism (opioids, muscle relaxants), impaired wound healing and infection. (Discussed in detail in the Complications of Blood Transfusion Chapter 49 and Hypothermia Chapter 51).

Doses of commonly used drugs (in healthy children)

DRUG	DOSAGE	ROUTE	NOTES
Premedicants and Sedatives			
Atropine	10micrograms/kg 20micrograms/kg	IV IM	
Paracetamol	20mgs/kg	Orally	
Trimeprazine	2mgs/kg	Orally	
Midazolam	0.5mg/kg	Orally	Give 20–30 mins pre–operatively.
Diazepam	0.2mg/kg	IV	Not recommended if alternative available.
Induction agents			
Thiopentone	5mg/kg	IV	
Propofol	3-5mg/kg	IV	
Etomidate	0.3mg/kg	IV	
Ketamine	1-2mg 5-7mg	IV IM	
Relaxants			
Suxamethonium	2mg/kg (neonate) 1.5mg/kg (child)	IV IV	
Pancuronium	0.1mg/kg	IV	1/3 of initial dose can be given as an increment.
Atracurium	0.5mg/kg	IV	
Vecuronium	0.1mg/kg	IV	
Rocuronium	0.6mg/kg	IV	
Reversal agents			
Atropine	20 micrograms/kg	IV	
Glycopyrrolate	10 micrograms/kg	IV	
Neostigmine	50 micrograms/kg	IV	
Analgesics			
Paracetamol	20mgs/kg	Orally	Max. 90mgs/kg/day
Ibuprofen	5-10mgs/kg	Orally	
Diclofenac	1mg/kg	Rectally	
Fentanyl	1-2 micrograms/kg	IV	
Pethidine	1mg/kg	IV or IM	
Morphine	100 micrograms/kg	IV, IM or SC	
Pentazocine	500 micrograms/kg 1mg/kg	IV IM or SC	In children over one year.

Pharmacological differences

Both the actions and side effects of many drugs differ in children from adults. Doses are calculated on a weight basis.

Note that in the case of muscle relaxants the neonate has unusual requirements.

***Suxamethonium*:** neonates and young infants are resistant to suxamethonium, rarely show fasciculation and are more prone to bradycardia.

Non-depolarising agents: neonates are very sensitive to non-depolarising drugs.

In all children less than 5 years of age the risk of laryngeal spasm and bradycardia can be reduced by giving atropine 10 -20 micrograms/kg IM or IV either pre-operatively or at induction.

For older children a friendly and simple explanation along with the use of topical creams, if available, will often avoid the need for preoperative sedation.

Psychological differences

- There is difficulty in communication, especially in the first five years.
- Deprivation develops rapidly, especially after separation from the parents.
- There is a great fear of injections and a greater fear of the unknown in young children.

Pathological differences

- Children presenting for surgery are often very ill.
- There may be other associated congenital abnormalities, especially in the neonates.

ANAESTHETIC EQUIPMENT

BREATHING SYSTEMS

Paediatric anaesthetic equipment should:

- Have minimal resistance to breathing
- Have minimal dead space
- Be light (not bulky)
- Be simple to use
- Provide humidification of inspired gases.

The following systems are described

Ayres T–piece
Jackson Rees modification
Draw-over system with Paedivalve

The Bain circuit, the Magill circuit and the circle absorber are described in Chapter 15.

Ayre's T–Piece

This is a simple metal T Tube (Mapleson E circuit). Disposable plastic versions are available.

- It connects to the patient via an endotracheal tube connector.
- It connects to an extension tube which acts as a reservoir and then opens into the atmosphere.
- It connects to a gas source, e.g. an anaesthetic machine. Nitrous oxide, oxygen and inhalational agents such as halothane may be fed into the T–piece.
- Adjustment of the fresh gas flow can prevent rebreathing of expired air. Accumulation of CO_2 is prevented.
- Intermittent occlusion of the open end allows controlled ventilation.

Jackson Rees modification of Ayre's T–Piece

Jackson Rees added an open-ended 500 ml bag to the expiratory limb. This makes controlled ventilation possible on application of continuous positive airway pressure (CPAP). The movement of the bag indicates the patient's respiratory activities during spontaneous respiration. During spontaneous ventilation the fresh gas flow should be 2-3 times the minute volume. During controlled ventilation the flow rate can be 200 ml/kg with a minimum flow of 3L/min. (Some degree of rebreathing may occur at these flow rates but it is compensated for by hyperventilation).

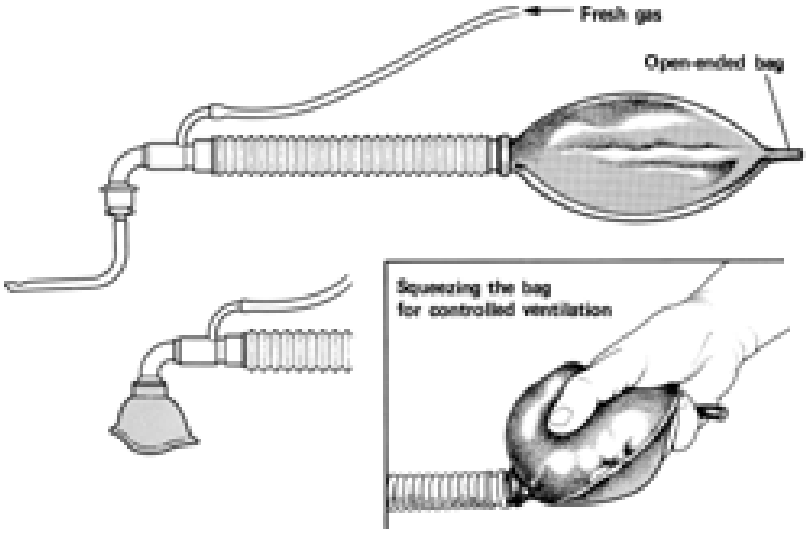


Fig 20.2 The Ayre's T–piece with open-ended bag

Advantages of a T– piece

- The dead space is minimal, equal to the volume of the apparatus between the point of entry of the fresh gases and the patient.
- Resistance to flow is minimal because there are no valves.
- Rebreathing is virtually eliminated if the prescribed fresh gas flows are used.
- It is light.
- It is relatively cheap.
- It is simple.

Disadvantages of the T Piece

- It needs a high flow rate. This makes it expensive to use, so it is a wasteful and expensive system to use where anaesthetic gases and agents are in short supply or for children > 25kg.
- The gases inhaled are dry unless they are artificially humidified.
- Theatre pollution becomes an even greater problem with the T piece. A scavenging system is necessary.

The circle absorber

This is described in detail under anaesthetic machines in Chapter 15. The adult circle can be used in children over 25 kg in weight or over 5 years of age. This would be reasonable for controlled ventilation. The decision to use controlled ventilation would be made on surgical indications. It can be used for spontaneous respiration in older children above 35 kg in weight.

Draw-over system with Paedivalve

This arrangement is used for children under 15kg and is a low cost draw–over technique.
The paedivalve is a non-rebreathing valve (like a miniature Ambu E valve) and is used in conjunction with paediatric bellows replacing the standard adult bellows on the OIB base or with a paediatric self-inflating resuscitation bag. This delivery system can then be used with the OMV, using halothane and entraining oxygen from an oxygen concentrator.

Fig 20.3 Draw–over system with paediatric self–inflating bag and paedivalve

In summary

	Spontaneous Respiration		Controlled Respiration
T–Piece (Mapleson E)	Flow rate 2.5 to 3xminute volume		200 ml/kg body weight (min FGF of 3L)
In practice lower flow rates are used.			
	Rough guide	Weight	Flow rate
		Less than 15 kg.	4L/min
		15 – 20 kg.	6L/min
Bain Circuit (Mapleson D)	Flow rate 100 to 150 ml/kg body weight		Flow rate 70 ml/kg body weight
Magill's Circuit (Mapleson A)	Flow rate 2-3 x minute volume 70ml/kg body weight		Very high flow rate needed Not practical to use

OTHER EQUIPMENT

Laryngoscopes

A wide range of laryngoscopes is available for paediatric use.
The straight blade laryngoscope is useful in neonates and infants (up to the age of 1 year). The curved blade may be used for children above the age of 1 year but the choice of the blade depends on the anaesthetist's preference. The straight blade laryngoscope is introduced under the epiglottis. It is especially suitable in neonates who have a floppy U shaped epiglottis but be careful of vagal reflexes. The curved blade is inserted in front of the epiglottis, between the epiglottis and the base of the tongue.

Endotracheal tubes and connectors

Polyvinyl chloride (PVC) tubes are available for paediatric use as for adults. The tubes are non-irritant and though disposable, may be re–used after cleaning.
Uncuffed tracheal tubes are usually used in children to prevent problems with sub–glottic oedema or stenosis.
A guide to uncuffed tracheal tube size is:

Weight or age	Tracheal tube size (internal diameter, mm)
>2kg	2.5
Newborn	3.0–3.5
3 months – 1 year	4.0
Over 2 years	Age/4 + 4

Appropriately sized cuffed tubes may be considered in children of all ages but especially those at risk of aspiration or with stiff, poorly compliant lungs. The size of cuffed tube is one size smaller than an uncuffed tube (age/4 + 3). Care should be taken to avoid over–inflation of the cuff.
In all cases, a tracheal tube one size above and below that calculated should be available.

RAE tubes These are PVC tubes with a bend in them suitable for both nasal and oral use.

Latex or reinforced tubes are incompressible, almost unkinkable but floppy and accidental extubation is possible.
Latex deteriorates rapidly after sterilisation.

Cole (or Foregger or Rusch tubes). These have an expanded oral part which prevents insertion too far down. The smallest size can be used if the other tubes are too large. The shoulder on the Cole tube is said to prevent insertion too far down the trachea but it may act as a laryngeal dilator and cause serious laryngeal oedema. This type of tube is easy to insert but should not be left in place for more than a few hours because of this hazard.

The tubes most recommended are PVC and RAE tubes.

Masks

The Rendell-Baker mask was designed to fit the contours of the face and reduce dead space. In the smallest of these masks the dead space is 4 ml. They are available in sizes 0, 1, 2, 3 and 4.
Cushioned clear plastic facemasks (when available) are the current standard as they are easy to use, non–threatening to the child and regurgitation is easily detected.

Airways

The Guedel oropharyngeal airways are most commonly used. They are made of PVC or firm rubber. A metal insert prevents obstruction of the airway by the patient biting on it. The Guedel airway is available in sizes 000 to 4. The airway is sized from the distance from the incisors to the angle of the jaw. It is important to use the correct sized airway.

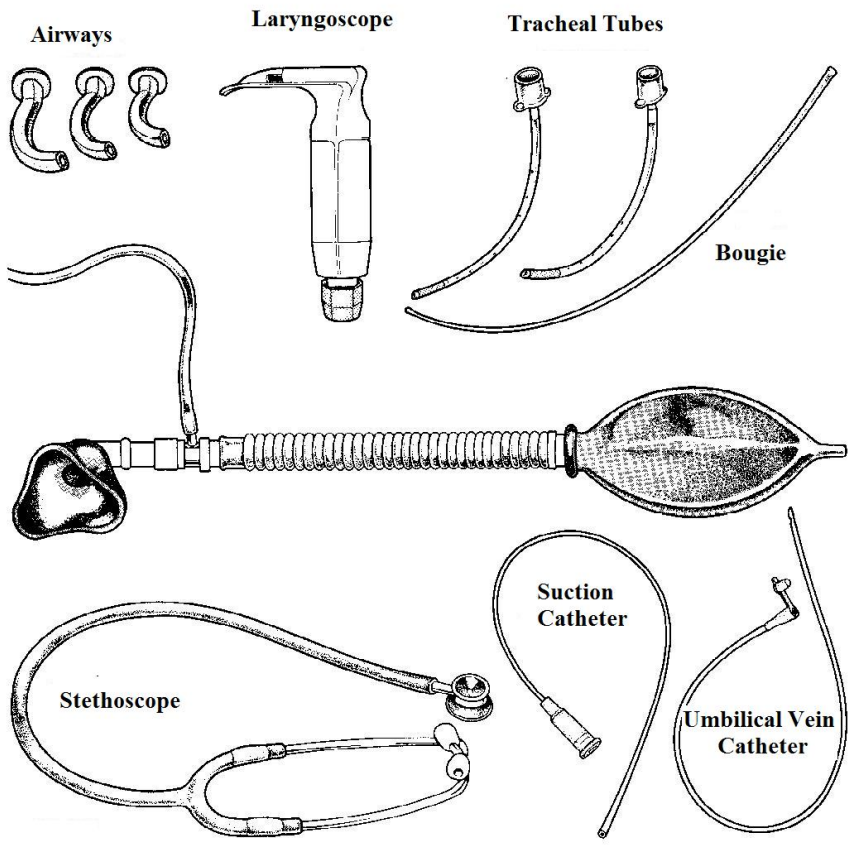


Fig 20.4 Some paediatric equipment for neonatal use

ANAESTHETIC MANAGEMENT

Regional techniques as the sole anaesthetic are better avoided in those less than 15 years of age.

Pre-operative preparation of patient

- Check the medical state of the infant, look for congenital abnormalities, any other underlying medical disorders, the degree of dehydration, electrolyte imbalance etc.
- Check the time of the last feed. Prolonged starvation should be avoided. Babies may be breast-fed 4 hours before surgery but can be given clear fluids up to 2 hours pre-op. Infants and older children may be given food or milk up to 6 hours pre–operatively and clear fluids up to 2 hours before surgery. The fluids can be solutions containing glucose, as children are prone to hypoglycaemia. In emergency surgery it is important to note that the gastric emptying time can be delayed for the same reasons as it is in adults, e.g. peritoneal irritation, fear, head injuries etc. The patient is best considered to have a full stomach and the usual precautions of pre oxygenation, rapid sequence induction and cricoid pressure applied.
- Weigh the patient.
- Psychological preparation for surgery:
  - Seek to establish a rapport with the child, trying to allay any fears (e.g. the fear of venepuncture, anaesthesia, surgery).
  - Discuss any fears expressed by the parents.
  - Discuss the method of induction with the child if the child is old enough to understand. If there is no contraindication to either an inhalational or intravenous induction and there is no special benefit to be derived from using one technique over the other, then the child's wishes should be taken into account.
  - EMLA cream (if available) may be applied to the skin on the back of the hand to reduce the pain and discomfort of venepuncture.
- Premedication:
  This is often omitted now but may be used:
  - To dry secretions
  - To block undesirable reflexes due to surgery
  - To sedate the child before surgery
  - To reduce the dose of anaesthetic drugs required

  Drugs used for premedication:
  Atropine 10 micrograms/kg IM as an antisialogogue
  Midazolam 0.5mg/kg PO, trimeprazine 2-4mg/kg PO or
  chloral hydrate 50mg/kg PO for sedation

Opioids are rarely used for premedication and painful IM injections should be avoided in paediatric anaesthesia wherever possible.
Opioids should be used with care in infants under the age of 6 months due to the difficulty of calculation of the correct dose, administration and the risks of respiratory depression.

Pre–operative preparation of equipment

- Check the machine, equipment and drugs as described in Chapters 14, 15 and 16 under Techniques of anaesthesia. This must be done before the child is brought into theatre.
- Work out the following details and write them out on a sheet of paper before commencing the anaesthetic.
  - The dose of each drug to be used, based on the child's weight.
  - The size of the endotracheal tube to be used, based on the child's age. Formula: $\frac{\text{Age in Years}}{4} + 4$
    (Always have a tube one size smaller and larger also available).
  - The maintenance fluid that the child will need while in the operating theatre (based on the child's state of hydration and weight).
  - The child's blood volume. This will depend on the child's age and weight. Knowing the blood volume will indicate the significance of any blood loss in theatre and the need to transfuse the patient.
- Draw up the drugs and label the syringes before commencing the anaesthetic. This is essential.

Intra-operative management

- ***Monitoring***
  Always monitor the following:
  Cardiovascular System:
  - Pulse.
  - Heart rate. Use a precordial or oesophageal stethoscope.
  - Blood pressure. Make sure the cuff is the right size.
  - ECG if available.
  - CVP if necessary.
  - Blood Loss. Weigh the sponges. Blood on the drapes and gowns and also on the floor must be taken into account. If 10–15 % of the blood volume is lost consider replacement with blood.

  Respiratory system: Observe the colour of the mucous membrane and the blood continuously. The respiratory rate and the tidal volume must be noted if possible. Use pulse oximetry and capnography if they are available.

Temperature: The usual sites of temperature monitoring are rectal, oesophageal or axillary. It has been mentioned previously that neonates and young children have poor control over body temperature, so every effort should be made to conserve heat. Here are some of these measures:

- The temperature of the theatre environment should be between 75 and 80 degrees F (21-24°C).
- Neonates and very young infants should be brought to the operating room in a humidicrib.
- A method of warming should be used for all patients weighing less than 5kg, or patients over 5kg who need prolonged surgery or large transfusions of cold blood.
- Minimise skin exposure by wrapping the patient in velband (cotton wool).
- Inspired gases should be humidified if possible.
- Warm all blood and plasma solutions before they are infused. Large volumes of crystalloids if administered to the patient need to be warmed. (For Methods of warming see Chapter 51).

- ***Induction:***
  - Particular care should be taken in the moribund patient and also in the patient who is at very great risk of regurgitating (e.g. very severe abdominal distension).
  - Inhalational induction is frequently used with children. Air or nitrous oxide/oxygen and halothane or ether/air/oxygen can be used. If the patient is very ill the oxygen concentration can be increased to 50–100%. An intravenous line is established as soon as possible if not established pre- induction. This will require a skilled assistant to maintain the airway. .
  - Intravenous induction. This is perhaps the kindest and quickest method in children who have accessible veins or an infusion running. The drugs available for intravenous induction have been discussed under pharmacology of anaesthetic agents. Thiopentone can be used in the fit patients. Ketamine IV may be used in the poor risk patients and atropine should be used to reduce secretions.
  - Intramuscular induction may be carried out using ketamine IM in patients who have no accessible veins and in whom an inhalational induction is not possible, for instance anyone with facial burns.
  - Rectal induction. Thiopentone and methohexitone have been used in the past for this purpose. The technique is not recommended.

- **Intubation:**
  Intubation can be performed as follows:
  (Awake intubation is no longer recommended)
  - ***Intubation under deep general anaesthesia*** (inhalational)
    Anaesthetise the patient with air/nitrous oxide/oxygen and halothane or ether/air and oxygen. Once the jaw is relaxed, insert a Guedel airway and give the patient a few more breaths of anaesthetic. Intubation is then performed. If there is any evidence that the patient is light when the laryngoscope is inserted, then the patient should be anaesthetised further and intubation deferred until the patient is in a deeper plane of anaesthesia. Swallowing, breath-holding, coughing etc. are signs that the patient is not ready for intubation and if it is attempted then laryngeal spasm may result.

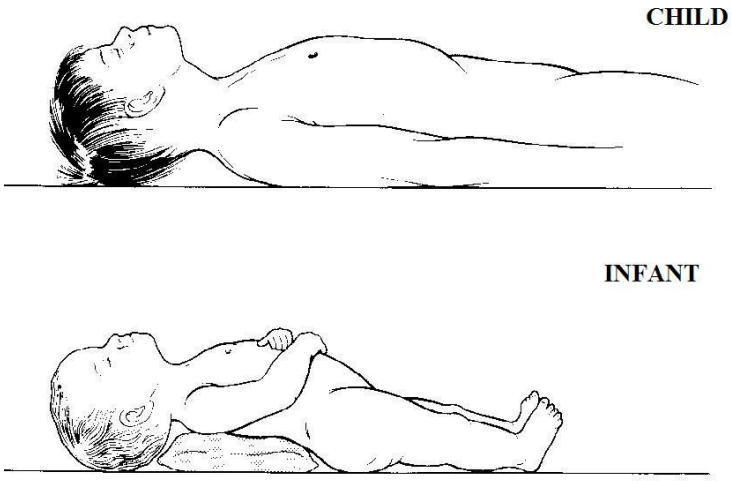


Fig 20.5 Intubation position for children and infants

This method (inhalational anaesthesia) for intubation may be appropriate in the following conditions:

- Very young infants.
- Children with airway problems. These patients may be difficult to ventilate with a mask and this makes the use of a relaxant hazardous.
- Lack of intubating experience on the part of the anaesthetist.

The problems of intubating a patient under deep inhalational anaesthesia are:
The tongue may fall back and obstruct the airway during inhalational induction.
Laryngeal spasm may occur if the intubation is performed too early.
Bradycardia may result from the use of halothane or during intubation. Circulatory collapse or cardiac arrest may occur if the patient is too deep.

– ***Intubation following the use of a relaxant.*** A relaxant makes intubation easier and is the preferred method in all children. Relaxants should be used before intubation only if the anaesthetist is able to ventilate the patient with a mask.
Suxamethonium can be used for intubation at a dose of 2mg/kg IV in the neonatal period and 1.5mg/kg IV thereafter.
Suxamethonium is useful in the following cases:
  - A patient with a full stomach.
  - Patients likely to have intubation problems and who need good intubating conditions (provided they can be ventilated by mask).
  - Very ill patients who need quick intubation and for whom oxygenation is mandatory. A modified rapid sequence induction may be used (ventilation with 100% oxygen with the application of cricoid pressure) as it is difficult to pre–oxygenate an anxious, upset child.
  - Patients who may be difficult to ventilate by mask but who need rapid intubation e.g. treatment of laryngeal spasm.

The choice of the non-depolarising relaxant will depend on the relaxant available.

- ***Maintenance:***
The anaesthetic is maintained with air or nitrous oxide/oxygen, halothane, relaxant (e.g. pancuronium) or ether/air/oxygen and pancuronium.
Analgesia is titrated IV as required.
A peri–operative combination of simple analgesics (paracetamol, NSAIDs), local anaesthesia +/– opioid or ketamine analgesia should be used whenever possible.
Position the patient with care.

PERI-OPERATIVE FLUIDS

Fluids may be classified as replacement fluids (including fluid for resuscitation) or maintenance fluids. Children are vulnerable to cerebral oedema from hyponatraemia (low plasma sodium) and so hypotonic fluids, (especially 4% dextrose/0.18% saline), should be avoided in the peri–operative period.

Pre–operative fluids

Prolonged starvation should be avoided and free clear fluids can be given orally up to 2 hours before elective surgery.
Fluid resuscitation may be required if the child is unwell. Diagnosis of hypovolaemia or dehydration is made on clinical grounds. Important clinical signs include delayed capillary refill time greater than 2 seconds (a child who is well hydrated and warm should have an instantaneous capillary refill time), cool peripheries, altered mental status, either lethargic or agitated and rising heart rate. Hypotension, cold white peripheries and a child who is unresponsive are late and extremely ominous signs. In case of difficult venous access, intraosseous access should be considered (20G butterfly needle inserted directly e.g. proximal tibia).
The immediate treatment is 100% oxygen by facemask and a rapid fluid bolus of 0.9% saline 20 ml/kg. The child should be re–assessed and the fluid bolus repeated if necessary. A common problem in management of severe hypovolaemic shock is failure to administer enough fluid. Blood or colloid should be considered if there is no improvement after 40ml/kg of fluid.

Intra–operative fluids

Replacement fluid with isotonic solution (Ringers or 0.9% saline), colloid or blood should be administered as required. Fluid may be required to counter the effects of anaesthesia or replace intra–operative fluid losses. Intra–operative fluid losses depend on the type of surgery and should be guided by clinical monitoring (heart rate, capillary refill, blood pressure). Most children do not require dextrose containing solutions during surgery.

Minor surgical procedures 10ml/kg bolus (only).
Major surgical procedures up to 20ml/kg/hour with additional bolus 20ml/kg as required.

If 10–15% of the blood volume is lost then blood transfusion should be considered. As a rough guide, 4ml/kg of packed cells (or 8ml/kg whole blood) will raise the haemoglobin by 1g/dL. Once the decision is made to transfuse the child, use as much of a single donor unit as possible to limit exposure to other donors.

Post–operative fluids

Intravenous fluids should only be given if the child is unable to tolerate oral fluids.
Replacement with isotonic fluids may be required to replace ongoing losses such as blood loss, vomiting, nasogastric fluid loss or wound drain losses.
Maintenance fluids should be given to provide the requirements for water, sodium and potassium (potassium not required on the first post–operative day, 1–2 mmol/kg/day thereafter). These may be given as 0.45% or 0.9% saline with 5% dextrose, calculated at the rate of either 10ml/kg/hour for the first hour and 5 ml/kg/hour thereafter **or** 4ml/kg/hour for the first 10kg + 2ml/kg/hour for the next 10kg + 1ml/kg/hour for any additional kgs**.**
An easily remembered formula based on the weight of the child is as follows:

Weight	Fluid required ml/day	ml/hr
0–10kg	100ml/kg	4ml/kg
10–20kg	1000ml+50ml/kg for each kg more than 10kg	40ml+2ml/kg for each kg more than 10kg
20–30kg	1500ml+20ml/kg for each kg more than 20kg	60ml+1ml/kg for each kg more than 20kg

Example:

8kg child
8kg x 4ml/kg = **32ml/hr maintenance**

12kg child (10kg + 2kg)
10kg x 4ml/kg = 40ml/hr
+ 2kg x 2ml/kg = 4ml/ hr
Total = 40=4 = **44ml/hr maintenance**

25kg child (10kg + 10kg +5kg)
10kgx4ml/kg = 40ml/hr
+10kg x 2ml/kg = 20ml/hr
+ 5kg x 1ml/kg = 5ml/hr
Total = 40+20+5 = **65ml/hr maintenance**

Fluid balance should be measured carefully to avoid fluid overload

Reversal

At the close of the operation the relaxant is reversed.
Atropine 20 micrograms/kg or glycopyrrolate 8 micrograms/kg is given intravenously, followed by neostigmine 50 micrograms/kg. It is easier to draw up the atropine and neostigmine in separate syringes as the doses are much smaller than adult doses. The same precautions must be taken before extubation as have been described for adults. Before extubation the patient must breathe deeply and regularly and must be given at least 10 breaths of 100% oxygen. The pharynx must be suctioned before and after extubation. Administer oxygen by mask after extubation and ensure that the patient is still breathing adequately.

The patient should be nursed on his side during the recovery period.
Never leave patients unattended (even for a minute!) until they are awake and fully conscious.

Other anaesthetic techniques available

- Spontaneous respiration using air or nitrous oxide/oxygen/volatile. This is similar to the technique described for adults (Techniques of anaesthesia Chapter 16). A mask and an airway of appropriate size or a LMA can be used.
- Ketamine, given either intravenously or intramuscularly in combination with diazepam. It is believed but not confirmed that the incidence of hallucinations after ketamine is less frequent in children.

These techniques are used in fit infants who are due to have brief, minor, superficial surgery e.g. removal of foreign bodies from the ear, cystoscopies, reduction of fractures, removal of lesions from the limbs.

Ketamine infusions are used widely in older children when muscle relaxation is not necessary. (See Chapter 14)

Post–operative care

The patient is observed in the recovery ward until fully awake, with careful monitoring of analgesia, fluids and oxygen therapy. Post-operative complications as outlined in the relevant chapters must be watched for and treated vigorously.

Pain relief: A combination of analgesic techniques should be used whenever possible – simple analgesics such as paracetamol and NSAIDs, local anaesthetics, either infiltrated or regional blocks such as a caudal block and/or ketamine or opioid analgesia. (See Chapter 55 Acute pain relief for more detail).

EARLY POSTOPERATIVE COMPLICATIONS

Laryngeal oedema

This may occur in young children following extubation and is caused by the endotracheal tube in the larynx. Laryngeal oedema is associated with:

- Large endotracheal tubes.
- Endotracheal tubes made of irritant material, e.g. red rubber or irritant chemicals used in sterilising tubes.
- Any infection in the upper respiratory tract or a history of croup.
- Trauma to the upper airway, e.g. difficult, clumsy or repeated laryngoscopies or bronchoscopies. These will predispose to oedema of the mucosa of the larynx.

The small diameter of the child's respiratory tract makes it especially susceptible to obstruction even with a small degree of oedema of the mucous membrane.

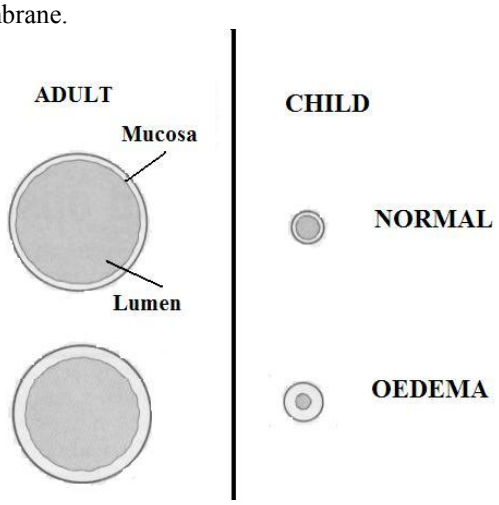


Fig 20.6 Cross–section of the airway showing the effect of mucosal oedema

Stridor, which is a harsh noise during inspiration, is one of the cardinal signs of laryngeal oedema but if the anaesthetist is conscious of this complication arising in young children and watches out for it after extubation, the condition can be treated in the early stages. The early signs of respiratory obstruction have been detailed elsewhere (The airway and its management Chapter 8). Late and worrying signs are restlessness, sweating and cyanosis. The pulse and respiratory rate rise initially and then fall indicating impending respiratory arrest. Treatment needs to start at the earliest indication of the problem.

Treatment:

- Humidified oxygen.
- Careful observation. If there is no relief, then try:
- Adrenaline nebules 0.5mg/kg (1:1,000 solution containing 1mg/ml) up to a maximum of 5mg. Can be repeated after 2–3hours.
- Steroids: dexamethasone 0.25mg/kg IV to a maximum of 8mg.
  hydrocortisone 1–2mg/kg IV.
- If the condition is still not relieved, it may be necessary to consider re-intubation or even tracheostomy.

Laryngospasm

Another frequent acute complication of anaesthesia in children is laryngospasm. This can be caused by:

- Secretions or vomit
- Inhalational anaesthetic agents
- Attempts at intubation
- Light anaesthesia
- Surgical stimulus
- Extubation

Treatment:

- 100% oxygen/a tight fitting face mask/ CPAP
- Jaw thrust
- Airway suction
- Deepen anaesthetic if possible
- Stop surgical stimulus

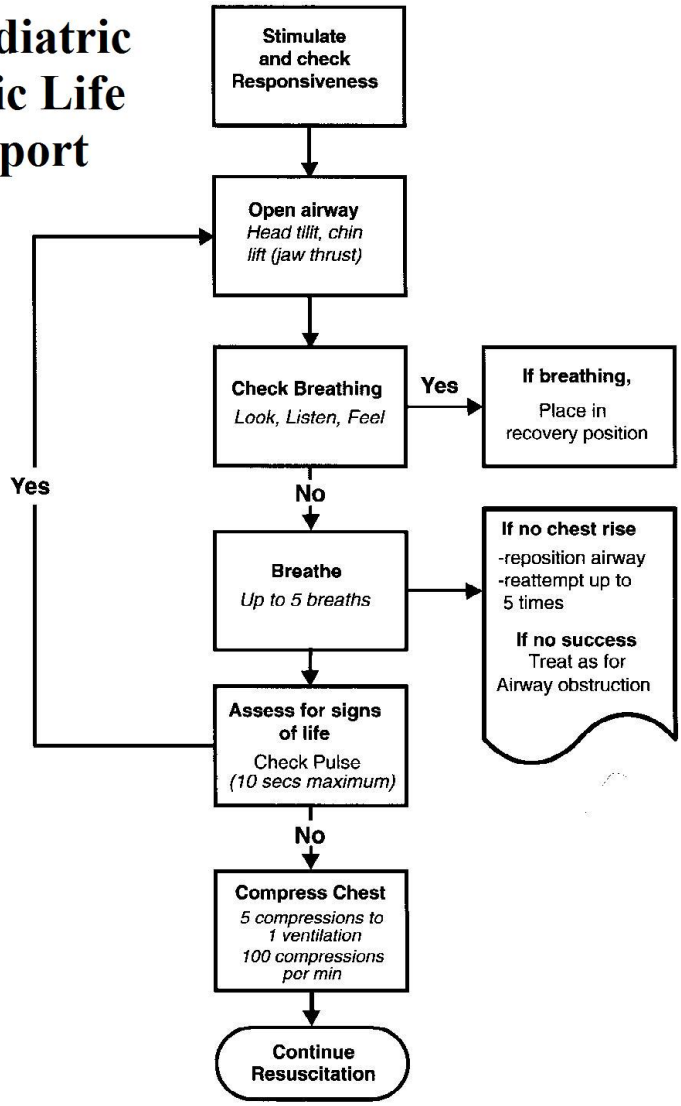
If this fails

- Give suxamethonium 0.25- 0.5mg/kg
- Ventilate with face mask if possible
- Consider re-intubation

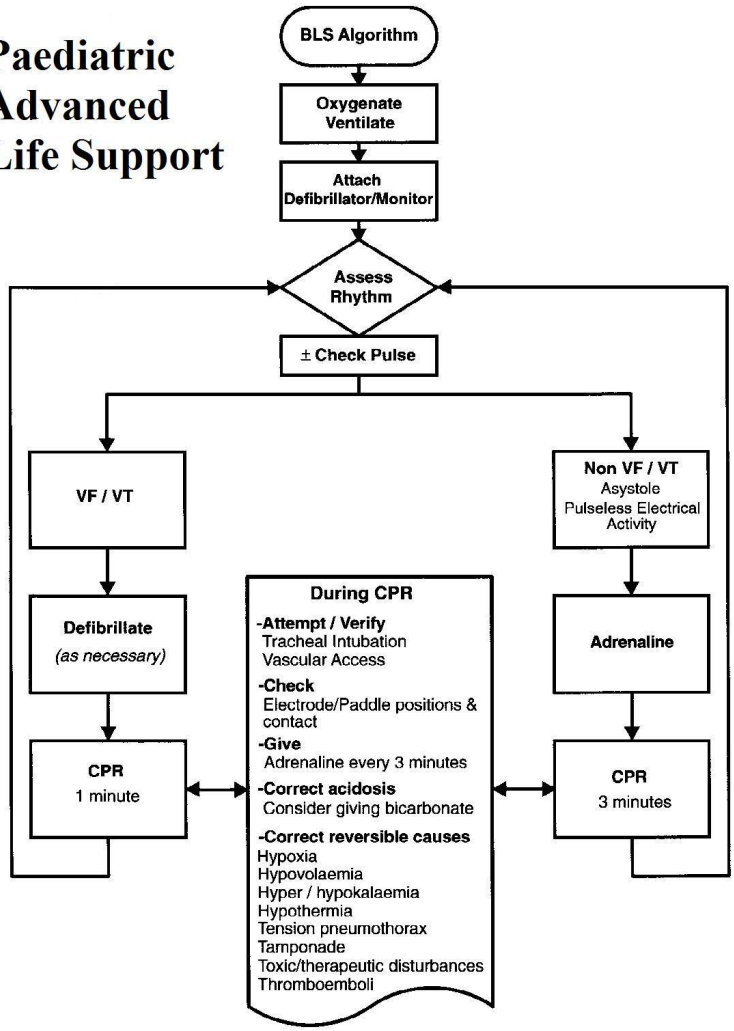
A summary of the important points in anaesthetising neonates and very young infants.

- The need for intubation
- The need for ventilation
- The prevention of heat loss
- Maintenance of careful fluid and electrolyte balance
- The use of appropriate equipment
- Veins are more difficult to find.

Paediatric Basic Life Support



Paediatric Advanced Life Support



CHAPTER 21

OBSTETRIC ANAESTHESIA

Outline:

- Maternal changes during pregnancy
- Anaesthesia for obstetric surgery
  - Spinal anaesthesia
  - General anaesthesia
  - Failed intubation drill
  - Local infiltration
- Pain relief during labour
- Toxaemia of pregnancy
- Neonatal resuscitation

MATERNAL CHANGES DURING PREGNANCY

Cardiovascular changes

- **Inferior vena caval compression.** The pregnant uterus can obstruct the inferior vena cava and this reduces the venous return to the heart. This is overcome by intense peripheral vasoconstriction and an increased flow through the vertebral venous plexuses. If these compensatory mechanisms do not work the patient presents with the supine hypotensive syndrome.
  - Supine hypotension must be watched for and treated as described later. No patient with the supine hypotensive syndrome should be anaesthetized, either with a general or regional anaesthetic until the situation is corrected.
    (See page 319 for treatment of supine hypotensive syndrome).
  - The engorged vertebral plexus means a smaller amount of local anaesthetic is required in the pregnant patient for both subarachnoid and epidural block (2/3rd the dose used in the non-pregnant females) and the onset of action is more rapid and the duration shorter in the pregnant patient.

- **Aortic compression** can lead to a reduction in the placental circulation. No pregnant patient after the 28th week should lie supine (i.e. on her back) on a hard surface. The lateral position must be encouraged.

- **The enlarged uterus** pushes the diaphragm upwards and this may result in a change in the position of the heart. This causes a change in the apex beat, heart sounds and ECG. The heart rate increases during pregnancy, reaching a maximum between the 28th to 32nd week.

- **Cardiac output increases** because stroke volume and heart rate increase maximally between the 28th and 32nd week. Blood volume increases, with maximum increase again between 28 and 32 weeks. Within a few weeks post–partum values have returned to normal. The plasma volume increase causes the physiological anaemia of pregnancy.
  The increase in cardiac output and blood volume increases the workload on the heart. The average patient compensates for the increase in cardiac output by a fall in peripheral resistance and in a fall in blood pressure.

- **The CSF pressure increases** during uterine contractions. The local anaesthetic should not be injected into the subarachnoid space during a uterine contraction.

Respiratory changes

- **Capillary engorgement of respiratory tract.** The mucosa of the nasopharynx, larynx, trachea and bronchi are congested.
  The upper respiratory tract must be treated very gently in the pregnant patient, especially during intubation and suction.
- **Minute volume increased by 50% at term.** This is due to an increase in tidal volume and respiratory rate. Both induction and recovery from an inhalational anaesthetic are faster. The FRC is reduced and the patient can get hypoxic more quickly from respiratory obstruction or breath-holding. Oxygen consumption is increased.

Gastrointestinal changes

- A **raised intragastric pressure** at term
- A greater incidence of **hiatus hernia**
- A **delayed gastric emptying** time. There is a greater risk of regurgitation and aspiration of gastric contents. Treatments include:
  - The use of oral antacids e.g. 30ml of sodium citrate (0.3M), within 20 minutes of induction. Magnesium trisilicate should be avoided, as it is a particulate antacid and can cause pneumonitis if inhaled.
  - Histamine 2 Receptor Antagonists (cimetidine or ranitidine, if available, can be given orally several hours before induction). Omeprazole is also a useful drug.
  - a rapid sequence induction with cricoid pressure is essential.

Renal changes

There is an increased GFR with an increase in urine output and a decrease in the BUN. There is an increase in urinary urea and in glucose excretion.

Hormone changes

The thyroid and pituitary glands show increased activity.

Fluids and electrolytes

Sodium and water retention occur during pregnancy. The IV fluid of choice is Hartmann's solution, or 0.9% saline. 5% dextrose is to be avoided particularly in patients with signs of pre-eclampsia and as the infusion fluid for the use of Syntocinon.

Psychological changes

Fear and anxiety are common. This calls for more time to be spent on the preoperative visit. Reassurance is very necessary.

ANAESTHESIA FOR OBSTETRIC SURGERY

- ***Spinal anaesthesia*** is recommended as the standard procedure for caesarean section.
- ***General anaesthesia*** is used when spinal anaesthesia is contraindicated.
- ***Epidural anaesthesia*** The technique is mentioned briefly under Regional anaesthesia in Chapter 19.
- ***Local infiltration*** This can be used in desperate situations, e.g. the absence of a trained anaesthetist, or a moribund patient.

SPINAL ANAESTHESIA FOR CAESAREAN SECTION

This method is considered dangerous by some but it is used as the standard anaesthetic technique in hospitals with limited facilities for the following reasons:

Advantages

If it is done cautiously and if the anaesthetist is aware of (and watching out for) complications, it has the following advantages:

- It gives effective pain relief very quickly with almost 100% success.
- There is no maternal depression of CNS. The cough reflex is present.
- There is no direct foetal depression.
- Only a very small dose of local anaesthetic is needed, so systemic toxicity is low.
- The anaesthetist is free to resuscitate the baby if necessary.
- There is no direct effect on uterine contractility unlike high doses of volatile agent.
- There is good relaxation of abdominal muscles.

Disadvantages

- Hypoxia. The enlarged uterus interferes with the movement of the diaphragm. Respiratory difficulties can arise with a high block but rarely cause serious problems. If the spinal block interferes with intercostal movement then there is a definite danger of hypoxia.
- Hypotension. During pregnancy the circulation of the CSF is altered because of high intra-abdominal pressure. The injected local anaesthetic travels higher up, causing a block of sympathetic nerves. Because of this smaller doses of LA are used in pregnancy and most hypotension is caused by aorto–caval compression and sympathetic block.
- Post spinal headache - the incidence of this is low especially if a 25/27G needle is used.
- Danger of total spinal.

Precautions to take while using a spinal anaesthetic for caesarean section

- Pre-operative preparation as described for general anaesthetic.
- Take steps to prevent supine hypotensive syndrome. The patient must be lying on her side or with a left tilt at this stage. If her blood pressure is lower than her normal blood pressure then you must not start any kind of anaesthesia until the blood pressure has been corrected. (See page 331).
- Insert a 16G needle and give at least 1 litre of Hartmann's solution before inserting the spinal.
- Use an appropriate dose of local anaesthetic:
  - 2.0ml of heavy 0.5% bupivacaine from a non-multidose vial
    or 2.5ml of plain 0.5% bupivacaine.
  - 1.2 – 1.5 ml 5% lignocaine or1ml (10mg) amethocaine may be used.

  These doses are sufficient if the spinal is performed in the lateral position using a 22G - 23G needle.
  (It is recommended that the patient lie on her right side while the spinal injection is being made then turned to the left tilt position with a wedge or pillow under the right hip).
  The spinal injection can also be made with the patient in the sitting position if preferred.
- Don't inject the local anaesthetic while a uterine contraction is in progress.
- Check the blood pressure every 60 seconds for the next 15 minutes.
- Give the mother oxygen by mask, to breathe.
- Place a wedge under her right hip when she is turned to the supine position as described above.
- If the block is patchy or starting to wear off toward the end of the operation, ketamine 25-50mg IV can be given every 10-15 minutes or by infusion to supplement anaesthesia.

Treatment of post-spinal hypotension:

- Increase fluids
- Give oxygen by mask
- Manual displacement of uterus
- Place in lateral position
- Give ephedrine 5 – 10 mg IV in repeated doses

THE USE OF ADRENALINE WITH LOCAL ANAESTHETIC SOLUTIONS (FOR SPINALS)

The addition of adrenaline is not recommended routinely but is sometimes added to lignocaine if the operation is expected to take longer than I hour.

Concentration required – 0.1mg (0.1ml of 1:1000)

Advantages

- Decreased absorption of local anaesthetic solution because of vasoconstriction
- The intensity and duration of the block are increased

Disadvantages

- Local anaesthetic solutions containing adrenaline are more acid (i.e. locally irritant) unless adrenaline is freshly added
- It can cause vasoconstriction of the spinal artery leading to permanent neurological damage
- The addition of adrenaline introduces a risk of contamination/ infection and must be performed using a sterile technique.

TECHNIQUES OF GENERAL ANAESTHESIA

Potential anaesthetic problems

- ***Inhalation of gastric contents*** This is a special hazard in the pregnant patient for the following reasons: there is a high gastrin level and a delayed gastric emptying time during pregnancy; the increased intra-abdominal pressure predisposes to vomiting; the oesophago-gastric sphincter is less efficient during pregnancy; the lithotomy position (in which a lot of obstetric procedures are performed) predisposes to regurgitation. Many obstetric procedures are emergencies and the patient may present for an anaesthetic after a recent meal.
- ***Depression of the foetus*** by drugs given to anaesthetise the mother.
- ***Supine hypotensive syndrome*** or obstruction of the inferior vena cava. This is not uncommon. Always watch out for it! It is caused by the pregnant uterus pressing on the inferior vena cava and reducing the venous return on which the blood pressure depends. The blood pressure therefore falls. Occasionally blood pressure remains normal but tachycardia and sweating occur.

The signs and symptoms of this syndrome should always be corrected before an anaesthetic is given.
Several anaesthetic deaths have resulted from this. The hypotension must first be corrected by the following methods:

- administer intravenous fluids rapidly.
- place a pillow under the right hip and tilt 15 degrees to the left.
- try to push the uterus away from the IVC manually (if above fails).
- place the patient in the left lateral position if above fails (very rare) while fluid is being given.

- ***Aortic compression*** interfering with blood supply to the foetus.
- ***Shock*** In the obstetric patient this may have several causes, e.g. haemorrhage; septic shock; traumatic shock; acute inversion of the uterus; amniotic fluid embolism (here the membranes rupture and amniotic fluid enters the circulation causing a "shock like" state). The most common cause of shock is blood loss, the signs of which often present late as these patients have the ability to compensate for a significant degree of blood loss. Therefore vigilance is important, including good IV access (16G) and treatment of the cause before an irreversible situation arises.
- ***Impairment of uterine contractility*** due to anaesthetic agents.

Pre-operative preparation

- Check the patient's medical state, e.g. present and past medical problems; anaesthetic history; degree of dehydration; extent of blood loss (if any). Check blood pressure with the patient in the lateral position and again 5 minutes after the left lateral tilt position is assumed. Tell the patient to lie on her side until she comes to theatre. Find out the time of the last dose of analgesia, the reason for the caesarean section and the presence or absence of foetal distress.
- Premedication consists of H_2 antagonist, i.e. ranitidine 300mg orally (if available) and metoclopramide 10mg IM, IV or O, 1 hour prior to surgery.
- Give an antacid, 20-30ml sodium citrate (0.3M) 20 minutes before surgery.

- Theatre procedure:
  - Transfer the patient to theatre in the lateral position.
  - Check when the patient had her last meal. A routine caesarean section should not be done within 6 hours of a meal. Even so, the risk of vomiting is higher in the pregnant patient.
  - Check the cuff of the endotracheal tube for leaks. Leave the syringe attached.
  - Check the suction, turn it on and put it under the mattress of the operating theatre.
  - Explain the technique of cricoid pressure to the assistant and to the patient and why it is necessary.
  - Check the anaesthetic machine and equipment.
  - Draw up the drugs.

Intra-operative management

- Anaesthetise the patient in the supine position with a wedge or pillow under her right hip. The table is horizontal.
- Note the blood pressure. If it is low follow the measures described to treat the supine hypotensive syndrome.
- Start an IV infusion of Hartmann's solution (500 -1000ml of fluid). Use a 16G cannula if possible.
- Pre-oxygenate the patient for four minutes (by the clock).
- Give a pre-selected sleep dose of ketamine or thiopentone intravenously (ketamine 1 – 2 mg/kg IV or thiopentone 3 –4 mg/kg IV). The standard method of titrating the induction agent by checking the eyelash reflex is not used in this instance. Avoid the use of ketamine in pre-eclampsia and eclampsia.
- Follow the induction agent (thiopentone or ketamine) with a dose of short acting relaxant, e.g. suxamethonium 1.5mg/kg.
- Apply cricoid pressure after consciousness is lost.
- Support the jaw until it is relaxed enough. (Note: The patient should not be ventilated after the relaxant. The four minutes pre-oxygenation ensures that there is sufficient oxygen in the lungs to tide over the period of apnoea).
- When the jaw is relaxed, intubate the patient and inflate the cuff. Check the position of the endotracheal tube by auscultation.

Maintain the anaesthetic with air or nitrous oxide, oxygen (50%) and volatile (e.g. halothane 0.5%) or intermittent doses of ketamine 0.5mg/kg **or** ether 2 - 3% air/oxygen. When the effect of the suxamethonium wears off give a dose of long-acting relaxant, e.g. vecuronium 500 micrograms/kg, pancuronium 500 micrograms/kg). Intermittent doses of suxamethonium may also be used. Control the patient's ventilation, avoiding hyperventilation. Ether/air/oxygen, suxamethonium/intubation followed by spontaneous respiration can also be used if no non–depolarising relaxants are available.

Once the baby is born the following is carried out:

- Give the mother 25- 50mg pethidine IV or other analgesic.
- Watch for awareness under anaesthesia.
- Use syntocin: 5 units IV and 30 units/Litre in Hartmann's solution (or normal saline) over 4 hours.
- Have the assistant ventilate the mother (or use a mechanical ventilator).
- Inspect the baby and resuscitate if necessary.

Reversal is carried out in the usual way with atropine and neostigmine. Halothane and high concentrations of ether may cause uterine relaxation and post partum bleeding. The low concentration (2-3%) of ether does not interfere with uterine contractility.

Post-operative care

Give routine post–operative care. Watch for post partum bleeding. Encourage early ambulation.

Advantages of a general anaesthetic

There is less cardiovascular disturbance than with spinal or epidural anaesthesia. If it is properly performed harmful effects on the mother or foetus are few.

Disadvantages of a general anaesthetic

- There is a danger of aspiration.
- The incidence of difficult/failed intubation is higher in obstetric patients.
- The anaesthetist is not as free to resuscitate the baby.

General anaesthesia is required when a spinal anaesthetic (recommended as the standard anaesthetic in situations for which this manual is intended) is contraindicated e.g.

- Shocked patient
- Bleeding patient
- Transverse lie where uterine relaxation may be required by the surgeon
- Where other contraindications to a spinal exist (see Chapter 19).

DIFFICULT AND FAILED INTUBATION

Difficulties with intubation can occur unexpectedly with any general anaesthetic but are more frequent and have a higher risk of complications in pregnant patients. A plan of action is essential in the event of failure to intubate an anaesthetised (and paralysed) patient.

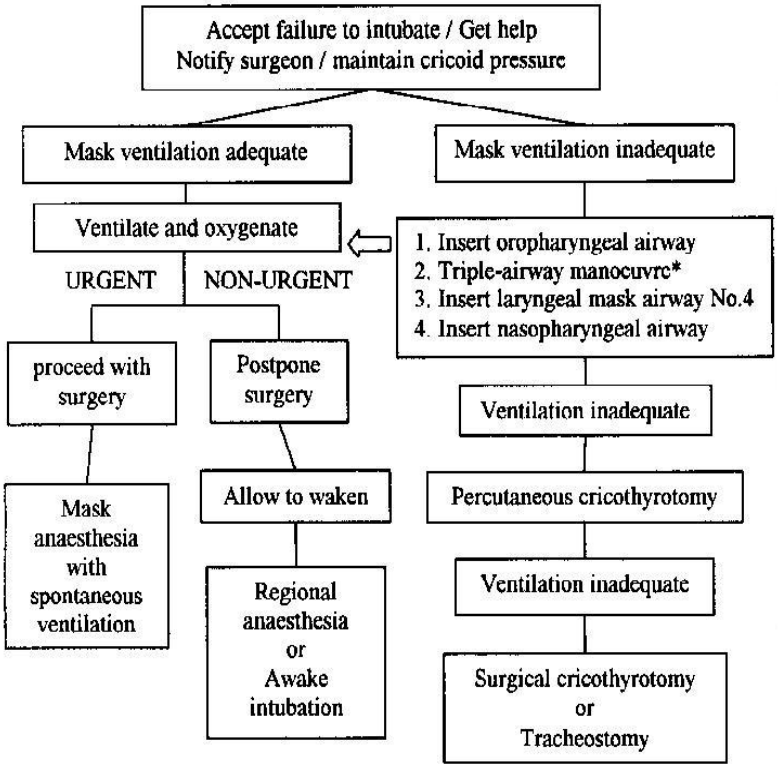
FLOW CHART FOR MANAGEMENT OF DIFFICULT INTUBATION

Attempted Intubation **DIFFICULT**	
MAINTAIN CRICOID PRESSURE + **OXYGENATION** **(with oral or nasal airway, 2 hands on mask, LMA)** Draw up more suxamethonium, atropine, ketamine or thiopentone An assistant will be necessary for this	
DIFFICULT LARYNGOSCOPY	**DIFFICULT TO PASS TUBE THROUGH CORDS**
Check "sniffing" position ↓ Check cricoid pressure is applied correctly ↓ Try larger blade or McCoy blade ↓ Use gum elastic bougie	Try smaller ETT tube ↓ Curve tube with introducer to hockey stick shape ↓ Use gum elastic bougie

If above measures are unsuccessful

- An early decision must be made to abandon repeated attempts at intubation. Maternal hypoxia must be prevented.
- Maintain cricoid pressure.
- The surgeon and sister must unscrub and be available to help the anaesthetist.
- Ventilate the patient with 100% oxygen.

FLOW CHART FOR MANAGEMENT OF FAILED INTUBATION



* 1. Flex neck
2. Extend head (at atlanto-axial joint)
3. Lift angles of mandible forward
(Consider two hands with assistant squeezing the bag)

HOW TO PROCEED FOLLOWING FAILED INTUBATION:

Elective caesarean section
Allow the patient to wake up and use a regional technique (e.g. spinal, epidural or local infiltration).

Emergency caesarean section
Consider waking the mother up and using a spinal. This is mandatory if the airway is not being maintained as both the mother and baby will die if hypoxia continues.
To repeat, the most important point is to maintain the airway so that the mother continues to receive oxygen. If you cannot do this, wake the mother up.

Otherwise
- Give inhalational anaesthesia using spontaneous respiration (mask anaesthetic) and maintain cricoid pressure throughout the operation. Insert a laryngeal mask airway if the airway is difficult to maintain with a mask and Guedel airway; maintain cricoid pressure. Nitrous oxide 50% in oxygen with halothane 1-1.5% may be used.
- In the absence of nitrous oxide use ether 4-8% in oxygen.
- Ketamine either in intermittent IV doses or as an infusion 500mg in 500ml of 5% dextrose may be used.

Emergency ventilation
If an apnoeic patient cannot be intubated and cannot be ventilated by face mask plus Guedel airway you may try the following methods of ventilation:
- Use two hands on the face mask, ensure the head position is correct (jaw thrust, head tilt) and ask an assistant to squeeze the anaesthetic bag or bellows if draw–over is being used.
- Try inserting a LMA
- Use failed intubation set up or minitrach.
- Needle cricothyrotomy and trans-tracheal ventilation. This surgical airway (or Mini-Trach Commercial kit or emergency tracheostomy) may have to be used if a patient can be neither intubated nor ventilated.

Technique for cricothyrotomy.

- Position the patient with her neck extended and identify both thyroid and cricoid cartilages.
- Mark a cross (X) in the space between the two cartilages, in the midline. Swab the skin.
- Choose a large bore needle and cannula (size 12 or 14 G) and pass it through the cricothyroid membrane into the trachea. The tip of the needle should point downwards towards the carina.

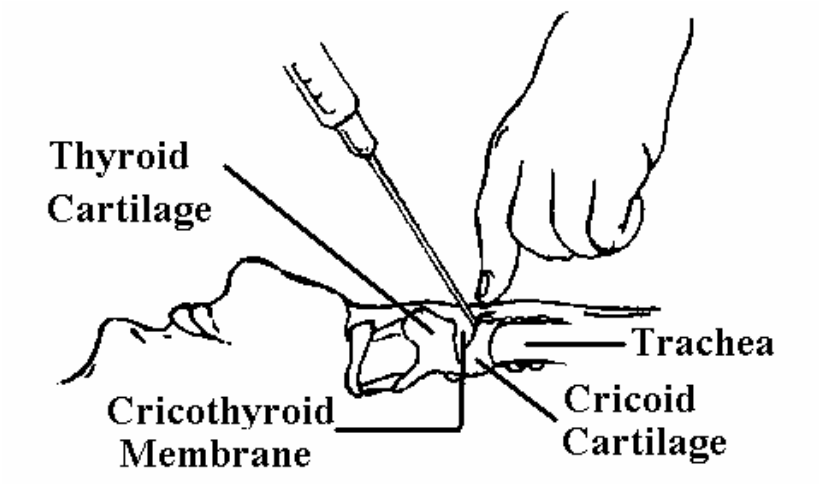


Fig 21.1 Insertion of cricothyrotomy needle / cannula

- Confirm the position of the cannula by aspirating air.
- Remove the needle and tape the cannula in place.
- The cannula can be connected to a 2 ml syringe and a 7.5mm ETT connector, thence to the anaesthetic machine or resuscitation bag.

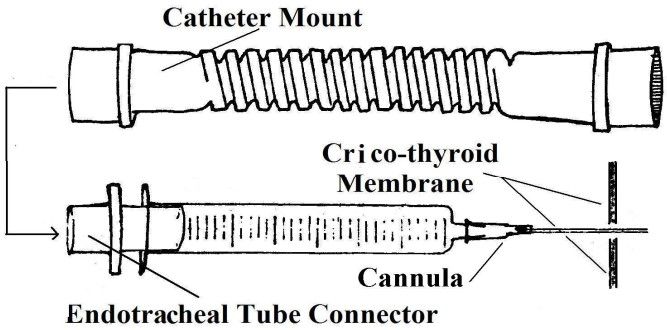


Fig 21.2 Equipment for emergency ventilation

- During expiration the gas escapes also through the larynx.

Hopefully this is a short-term ventilation method to provide oxygenation until spontaneous ventilation resumes and the patient wakes up.

EPIDURAL ANAESTHEIA

See Chapter 19. Epidural anaesthesia is not dealt with in detail in this book. See a specialist textbook for further information.

LOCAL INFILTRATION FOR CAESAREAN SECTION

Up to 100ml of 0.5% lignocaine with adrenaline 5 micrograms /ml (1:200,000) can be used to raise two weals on either side of the midline from the symphysis pubis to a point 5cm above the umbilicus. The layers of the abdominal wall should be infiltrated with the solution using a long needle. The abdominal wall is very thin in patients at term and great care should be taken not to pierce the peritoneum or the uterus.
A small dose of ketamine may be given to the mother to provide analgesia without the risk of foetal depression. Once the baby has been delivered additional analgesia or sedation can be given to the mother.

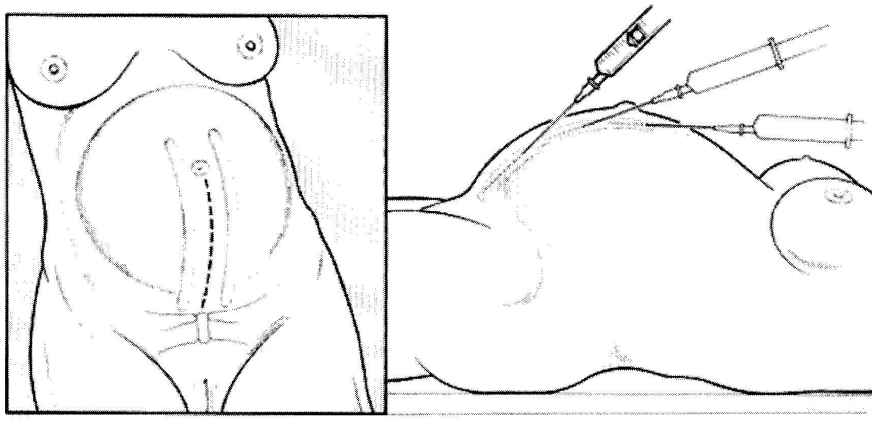


Fig 21.3 Field block for caesarean section

Advantages of local infiltration
No ill effects on mother or baby

Disadvantages of local infiltration
- Unsuitable for nervous patient
- Needs the surgeon's cooperation.

There is no absolutely safe anaesthetic for caesarean section. The spinal anaesthetic with the precautions taken above is recommended but is contraindicated in the patient who is bleeding (internally or externally) or shocked. The other contraindications to spinal anaesthesia (mentioned in Chapter 19) are applicable in obstetrics also.

The use of ephedrine

Ephedrine does not interfere with uterine blood flow and is recommended as the vasopressor of choice for the treatment of hypotension during spinal anaesthesia. Dose: 5 – 10 mg in repeated doses up to 30mg IV.
For resistant hypotension, assuming that caval compression and hypovolemia have been corrected a small dose of metaraminol (0.5 -1 mg) may be used. If this is not available a low dose of dilute adrenaline may be required.

The use of oxytocics

Oxytocin or syntocinon is the first line agent to help contract the uterus after delivery of the placenta. It can be administered as a bolus dose of 5 units slowly IV or 30 units/litre in normal saline or Hartmann's over 4 hours. This is usually effective. However, occasionally ergometrine (0.2mg IM or IV) may be required. Ergometrine is a second line agent. It can elevate blood pressure and therefore should be used cautiously in severe pre-eclampsia. It produces nausea, vomiting and vasoconstriction which is hazardous in patients with pre-eclampsia.
The third line agents are prostaglandins, if available. Prostaglandin f2 alpha (250micrograms) injected intramyometrium is effective. Side effects include nausea, vomiting, diarrhoea and bronchoconstriction.

PAIN RELIEF DURING LABOUR

There are several methods:

- Non-pharmacological methods
- Analgesics and sedatives
- Inhalational methods
- Regional anaesthesia

Each of these methods will be considered briefly:

Non-pharmacological methods
These avoid the use of drugs. "Natural childbirth" teaches the patient to relax. The exercises are taught during pregnancy and this relaxation is used at the time of the birth to help with the delivery.

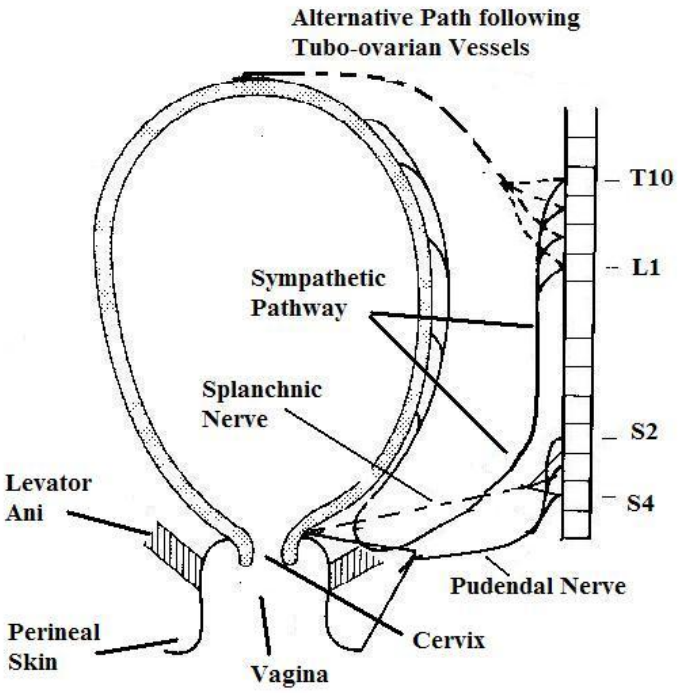


Fig 21.4 Uterine innervation

Analgesics

Pethidine or morphine. Pethidine relieves pain, having an action midway between morphine and codeine. The pharmacology of these drugs has been described in Chapter 6.

Pethidine is the most widely used analgesic drug in labour. It is effective when given early. It may also reduce cervical spasm and hence is very useful in the case of a rigid slowly dilating os.

Dose: Initial dose is 100mg when labour is well established. This dose may be modified, i.e. reduced to 75mg or 50mg in smaller patients.

Morphine has the following effects

On the mother:

- Elevates the pain threshold
- Produces a relative indifference to pain
- Acts as a hypnotic

On the foetus:

- Before the onset of labour there are no harmful effects
- During labour it severely depresses the foetal respirations

On the progress of labour:

In small doses morphine increases the interval between pains. In larger doses, it may slow down labour. Morphine should not be given later than 3 hours before delivery.

Side effects of opioid analgesics:

- A fall in blood pressure if these drugs are given IV
- Nausea and vomiting
- Vertigo and tingling in the extremities
- Opioids depress foetal respirations and must not be given in the three hours before delivery
- They can depress maternal respiration

Recent research suggests there may be some benefit in giving very low dose ***ketamine*** (0.5mg/kg IM) where epidurals are not available for pain relief during labour.

Inhalational methods

As labour advances the degree of discomfort experienced usually exceeds the relief provided by the narcotics and most big hospitals use either inhalation methods with anaesthetic vapours, or regional techniques.

Nitrous oxide: 50% nitrous oxide is needed to produce analgesia. It must always be mixed with oxygen and should be self administered by the patient. It has no adverse affect on the foetus.

Regional techniques

- Spinal anaesthetic can be used for either an instrumental or a forceps delivery.
- Epidural anaesthesia is used to provide pain relief in labour in many larger hospitals.
- Caudal anaesthesia (epidural or sacral block) can be used for an instrumental or forceps delivery.
- Pudendal nerve block and local infiltration can be used for suturing an episiotomy.

A more advanced textbook should be consulted for these techniques.

TOXAEMIA OF PREGNANCY
(PREGNANCY–INDUCED HYPERTENSION or PRE–ECLAMPTIC TOXAEMIA)

Definition: A multisystem disease occurring after the 20th week of pregnancy.

Pre-eclampsia complicates 7-10% of all pregnancies. It is characterised by:

- Hypertension > 140/90 (mild-diastolic > 90, severe-above 160/100)
- Oedema
- Proteinuria > 0.3 gm/l in 24 hours

Eclampsia is further complicated by tonic/clonic convulsions which may lead to coma.

Pathophysiology

The underlying cause is not known but it is thought it may be due to substances released from the placenta affecting endothelial cells. This endothelial cell damage disrupts capillary integrity throughout the body. Pre-eclampsia is a multisystem disease. The following pathological changes have been described:

- Vasoconstriction leading to hypertension and tissue hypoxia.
- Retention of sodium and water above that found in normal pregnancy.
- Localised intravascular coagulation especially in the placenta and kidneys. Pre-eclampsia is categorised as mild, moderate or severe.

Factors differentiating mild from severe pre-eclampsia*

	Mild	Severe
Systolic arterial pressure	<140mmHg	≥160mmHg
Diastolic arterial pressure	<90mmHg	≥110mmHg
Urinary protein	>0.3g/24hr dipstick + or 2+	≥5g/24hr dipstick 3+ or 4+
Urine output	>500ml/24hr	≤500ml/24hr
Epigastric pain	no	yes
Right upper quadrant abdominal pain	no	yes
Pulmonary oedema	no	yes
Cyanosis	no	yes
Headache	no	yes
Visual disturbances	no	yes
Platelet count	>100,000/mm^3	<100,000/ mm^3
HELLP (see below)	no	yes

*Not all features may be present in the same patient.

Pre-eclampsia is more common in:

- First pregnancies
- Diabetics
- Patients with polyhydramnios
- Multiple pregnancies.

Maternal changes

Cardiovascular

- Vasoconstriction causing hypertension and hypoperfusion.
- Reduced blood volume (relative to normal).
- Oedema secondary to leaky capillaries and salt retention. The oedema usually presents peripherally but pulmonary oedema may also occur.

Renal

- Decreased renal blood flow.
- Decreased urine output.
- Proteinurea.

Haematological

There is increased fibrinogen, fibrin and platelet turnover.

- Platelet count may be reduced. If less than 100,000 x 10^9/L check coagulation profile, i.e. INR or prothrombin time. If less than 75,000 x 10^9/L it is best to avoid a spinal anaesthetic as there may be an increased risk of spinal haematoma.
- Platelet function may be impaired.
- HELLP Syndrome (Haemolysis, elevated liver enzymes, low platelets).

Neurological

There is hyper-excitability and hyper-reflexia. Visual symptoms and headache suggest severe pre-eclampsia and the possibility of an impending convulsion (eclampsia).

Placenta

Decreased blood flow and possible infarcts leading to intra-uterine growth retardation and increased incidence of foetal distress.

Note: Pre-eclampsia is an unpredictable condition. Eclampsia can develop without severe hypertension.

The changes of pre-eclampsia continue for up to 48 hours after delivery and may peak in the first 24 hours after delivery. The highest risk for severe complications such as pulmonary oedema, eclampsia and thrombocytopaenia occur in the first 24 hours post partum.

TREATMENT OF TOXAEMIA

The aim of treatment in pre-eclampsia is to control blood pressure, prevent eclampsia and plan delivery of the foetus at the appropriate time. Good communication between the obstetric, anaesthetic and paediatric teams is important. After assessment of the patient, including history, examination and appropriate blood tests where available (full blood picture, urea, electrolytes, creatinine, clotting profile if platelet count is less than 100,000 x 10^9). The principles of treatment are:

Management of hypertension

The aim here is to protect the mother from the complications of extreme hypertension. To control an acute hypertensive episode where the systolic blood pressure is greater than 170 or the diastolic is greater than 110 (or both), drugs such as nifedipine, labetalol, atenolol, methyldopa or hydralazine are used depending on what is available. Labetolol and hydralazine can be given IV in hypertensive crises. The management of severe hypertension should be in a controlled setting, in hospital with appropriate close BP monitoring.

Seizure prophylaxis

Magnesium sulphate is the drug of choice for seizure prophylaxis in the following settings:

- After an eclamptic seizure, to prevent further seizures.
- In severe pre-eclampsia with signs of cerebral irritability, i.e. headaches, visual disturbance, etc.

Dosage:

- Magnesium sulphate is usually given IV (4g load over 15 minutes), followed by a continuous infusion of 1g/hr. Therapeutic levels of magnesium are 4-6mEq/L. Levels can be monitored but toxicity is extremely unlikely with this regime and clinical monitoring would be sufficient (see below). Magnesium also has an anti-hypertensive effect and prolongs the effect of muscle relaxants.
- Magnesium can be given intramuscularly (4 grams deep IM into each buttock as a loading dose – total 8 grams). This can be monitored clinically using assessment of the patella deep tendon reflexes. The point at which the deep tendon reflex is abolished is equivalent to a blood level of 10mEq/L which is above the therapeutic range but lower than levels causing more serious side effects (i.e. arrhythmias and severe muscle weakness). If the tendon reflexes are abolished the next IM dose (4 grams deep IM into 1 buttock) is given when the reflexes return. This will usually be about 4 hours after the loading dose. Therefore, magnesium can be given intramuscularly with regular monitoring of tendon reflexes to monitor toxicity. Magnesium toxicity is treated with IV calcium.

The management of convulsions

- Protection of the airway: This may be achieved by placing the patient in the left lateral position with high flow oxygen. If the seizure is prolonged and unresponsive to IV treatment (see below), endotracheal intubation using a rapid sequence induction may be necessary.
- The treatment of convulsions: Give IV diazepam (2.5mg /30 secs up to 20mg). Thiopentone in small doses can also be used.
- The prevention of further convulsions. Give magnesium sulphate (as described under *Seizure prophylaxis*, earlier).

Fluid management

Patients with pre-eclampsia have a relative hypovolemia and require careful fluid management. The fluid management is guided by urine output to maintain a urine output of 1ml/kg/hr. Careful IV hydration with Hartmann's solution (or 0.9% saline) is used to maintain an adequate urine output. These patients are at higher risk of developing pulmonary oedema, therefore their fluid management must be frequently reassessed.

Foetus

Delivery of the foetus must be timed carefully. The main reason for delivery is either foetal distress or intrauterine growth retardation. There are some maternal relative and absolute indications for delivery also. See specialised textbooks

Anaesthetic problems

- Uncontrolled hypertension.
- Imminent convulsions.
- Hypovolaemia (shift of fluid).
- Electrolyte imbalance: sodium may be low because of diet and diuretics. The potassium level may be low because of the use of diuretics.
- The magnesium level in the blood may be high if oliguria is present. Magnesium is excreted through the kidneys, so if an oliguric patient has been treated with magnesium sulphate, look for signs of magnesium toxicity. Prolonged neuromuscular block is common, potentiating non-depolarising drugs. Note however that magnesium improves renal blood flow and hence urine output.
- The foetus is premature, placental function is impaired and foetal hypoxia is likely.
- The foetus is often depressed because of decreased placental blood flow or as a result of the drugs used in treating of the eclampsia.

- Inadequate pain relief may cause the blood pressure to increase further and result in convulsions.
- Pulmonary oedema causing hypoxia.
- Detoxification and excretion of drugs are interfered with because of impaired liver and renal function in severe cases.

ANAESTHETIC TECHNIQUE IN PRE-ECLAMPSIA

Regional techniques are superior to general anaesthesia in pre-eclamptic patients ***without cerebral symptoms*** for the following reasons:

- Avoids difficult/failed intubation. Patients with pre-eclampsia have increased oedema of the airway.
- They provide maximum analgesia, eliminating the risk of pain, apprehension and so on, which can raise the blood pressure and precipitate a convulsion.
- They have no direct effect on the patient's heart, lungs, kidneys or liver (if the spinal is given carefully). However, coagulation tests may be necessary before a spinal.

Pre-operative care

- Give IV fluids until the patient is considered to be in fluid balance, as evidenced by the vital signs of pulse, blood pressure and urine output. (However, in the eclamptic patient these vital signs may be affected by the underlying pathology and may not be as useful). A large bore cannula must be inserted.
- Monitor vital signs until the patient is brought to the operating room. Intra-arterial monitoring is very useful in severe pre-eclampsia.
- Check the following:
  - The drugs given pre-operatively, especially the central depressants like pethidine
  - The time these drugs were given
  - The dose
  - The availability of naloxone for the mother and baby.
- Premedicate with ranitidine, metoclopramide and sodium citrate if available. Avoid prophylactic ephedrine.
- Make sure all the equipment necessary for a general anaesthetic is available, especially suction, oxygen, airways, endotracheal tubes, laryngoscopes.

- Follow the routine for a spinal anaesthetic for an obstetric patient, taking the usual precautions. Following spinal anaesthesia there may be a large drop in the patient's blood pressure which must be treated with small doses of ephedrine (3-6mg) and 250-500 ml boluses of Hartmann's solution.
- Be ready to resuscitate the baby (see the following notes on neo-natal resuscitation).

Contraindications to spinal anaesthesia in pre-eclamptic patients

General anaesthesia is the anaesthetic of choice in all patients with ***diminished level of consciousness*** e.g. those who have had eclamptic convulsions or are showing signs of increased cerebral irritability.
General anaesthesia may also be necessary in pre-eclamptic patients because of ***coagulation problems, maternal haemorrhage, or severe foetal distress.***

General anaesthesia technique in pre-eclamptic and eclamptic patients

- Assess airway as there may be damage or swelling as a result of convulsions.
- Prepare all equipment for difficult intubation.
- Preoxygenate as for any emergency case.
- Induce anaesthesia with thiopentone (4-5mg/kg) or propofol 2–3mg/kg followed by suxamethonium (1-1.5mg/kg) as per rapid sequence induction with cricoid pressure.
  Ketamine is contra-indicated because it causes hypertension.
- To prevent the hypertensive response during laryngoscopy and intubation in severe pre-eclampsia give a generous dose of induction agent and use another agent, such as esmolol (0.5mg/kg over 15-30 secs) magnesium (loading dose as above), lignocaine (1.5mg/kg), fentanyl (1.5 micrograms/kg) or alfentanil (10 micrograms/kg). The neonate may need naloxone if opioids are used.
  Esmolol can also be used to prevent a hypertensive response at extubation.
- Maintain anaesthesia with small doses of a non-depolarising relaxant or suxamethonium infusion and a low concentration of volatile agent. An opioid can be given as soon as the baby is delivered.

NEONATAL RESUSCITATION (resuscitation of baby)

After delivery

- Place the baby at a 45 degree head down tilt.
- Suck out secretions if indicated.
- Quickly assess the condition according to the following observations using the Apgar score at birth, 1min and 5mins.

Assessment of neonate

	Apgar score	**2**	**1**	**0**
1.	**Respiration**	regular	gasping	absent
2.	**Heart rate**	greater than 100/min	less than 100/min	absent
3.	**Colour**	normal (pink)	body pink extremities blue	blue
4.	**Tone**	strong flexor	average	limp
5.	**Reflex irritability**	cry +	some response	absent

At birth the foetal circulation ceases and the cardiovascular and respiratory systems of the infant are called into play. Approximately 80% of all infants undergo these changes without any problems. The others need some help.

For normal respiration the neonate needs:

- Ample respiratory stimuli
- An ample supply of oxygen
- An "undepressed" respiratory centre (no foetal distress or hypoxia ante partum and no drugs that would depress the respiratory centre)
- Normal neuromuscular mechanism
- Normal thoracic cage
- Normal lung structure: normal alveoli, normal capillaries, normal alveolar capillary membrane
- Clear airway
- Normal CVS

Factors stimulating respiration at birth

- Stimuli arising from extra-uterine existence e.g. the external temperature, movement, drying with a towel etc.
- The increasing $PaCO_2$ following birth asphyxia
- The mild acidosis following birth asphyxia

In infants who remain apnoeic at birth the mild acidosis that is present (caused by the birth process) is greatly exaggerated. Unless the baby is resuscitated promptly, there is further depression of the respiratory centre and myocardium, loss of muscle tone and permanent damage to the CNS. Bradycardia or cardiac arrest in neonates is almost always the result of hypoxia the correction of which is the mainstay of neonatal resuscitation. Thus the treatment of apnoea and airway obstruction must be prompt and effective.

Be prepared for the resuscitation of any baby born by caesarean section. In the presence of foetal hypoxia this is even more necessary.

SOME CAUSES OF FOETAL HYPOXIA

Maternal

- Toxaemia (pre-eclampsia)
- Chronic medical conditions
  - Hypertension
  - Diabetes
  - Renal disease
  - Cardiac disease
  - Pulmonary disease
- Drugs: sedative, analgesic, anaesthetic

Foetal

- Prematurity
- Abnormal foetal heart rate
- Meconium stained liquor
- Intrauterine growth retardation
- Hydrops foetalis
- Multiple births

Labour and Delivery Problems

- Prolonged labour
- Unusual presentations eg. breech or transverse
- Bleeding - anaemia
- Prolapsed cord
- Placental infarction

Always find out

- **The reason for a caesarean section being done.**
- **Whether foetal distress is present.**

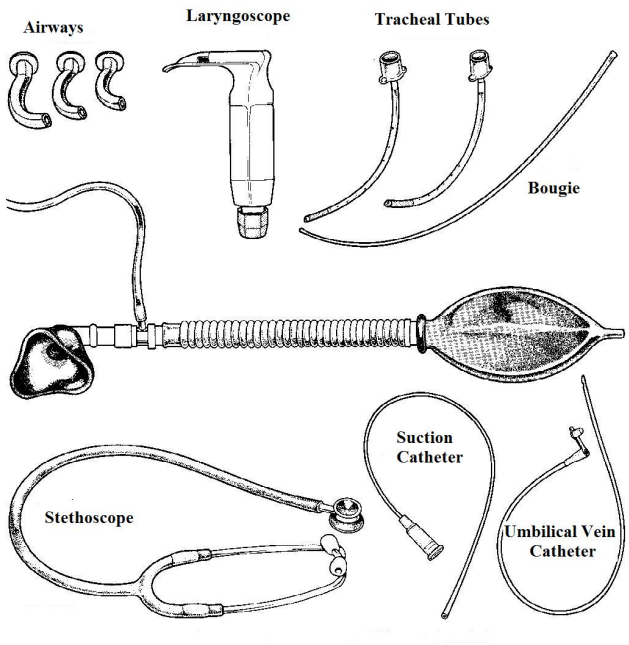


Fig 21.5 Some equipment for neonatal resuscitation

EQUIPMENT for RESUSCITATION

- A means for keeping the baby warm: a radiant heater, blankets
- An oxygen source, with flowmeter, or an oxygen concentrator
- A T-piece with infant bag or paedivalve and self inflating paediatric bag or bellows (250ml- pressure relief valve set at 30-35cm H_2O)
- A face mask - Rendell– Baker sizes 00, 0
- Airway - size 00
- Laryngoscope and infant blade- straight blade (size 0-1). A second laryngoscope should also be available.
- Endotracheal tubes size 2.5, 3.0, 3.5
- Infant stethoscope
- Suction bulb
- Suction machine with catheters
- Drugs
- Needles and syringes
- A stop watch

Principles of resuscitation

- Oxygen is essential for the function of the respiratory centre.
- Warming and drying and gently slapping the baby's feet is permissible.
- Gentle oropharyngeal suction may be necessary and if meconium aspiration has occurred tracheal suction should precede IPPV.
- IPPV is initiated promptly as mentioned with bag and mask +/- pharyngeal airway or LMA, or if this fails, prompt endotracheal intubation.

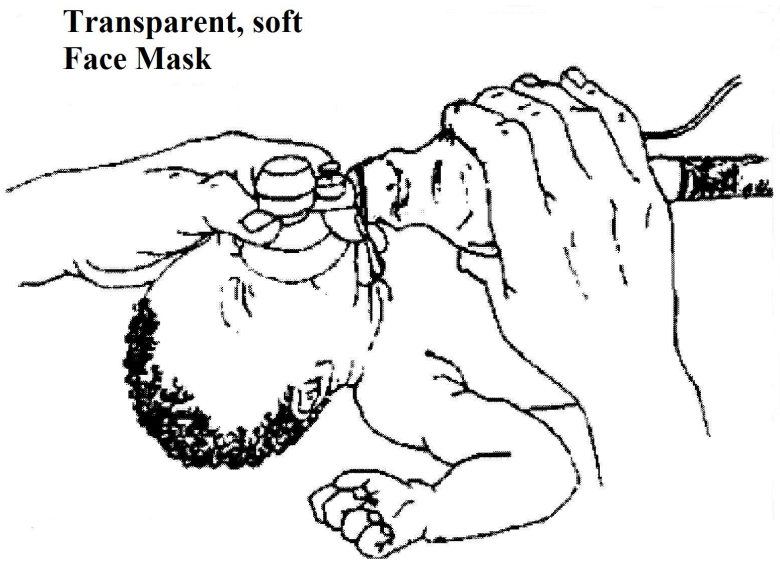


Fig 21.6 Bag and mask ventilation

- Neonatal heart rate is a good guide of adequate oxygenation. As a guide a heart rate of less than 100 is treated with oxygen and IPPV while a heart rate of less than 60 is treated with cardiac massage (CPR), oxygen + IPPV and appropriate drugs.
- For cardiac massage:
  - rate = 100.
  - two fingers on mid-sternum compressing 1.5-2.5cm. at a rate of 3 compressions : 2 ventilations.
- Prompt control of ventilation or assisted ventilation with oxygen is the most critical measure. Once breathing has been established and the infant's condition is satisfactory oxygen administration can be reduced in keeping with oxygen saturation measurements.

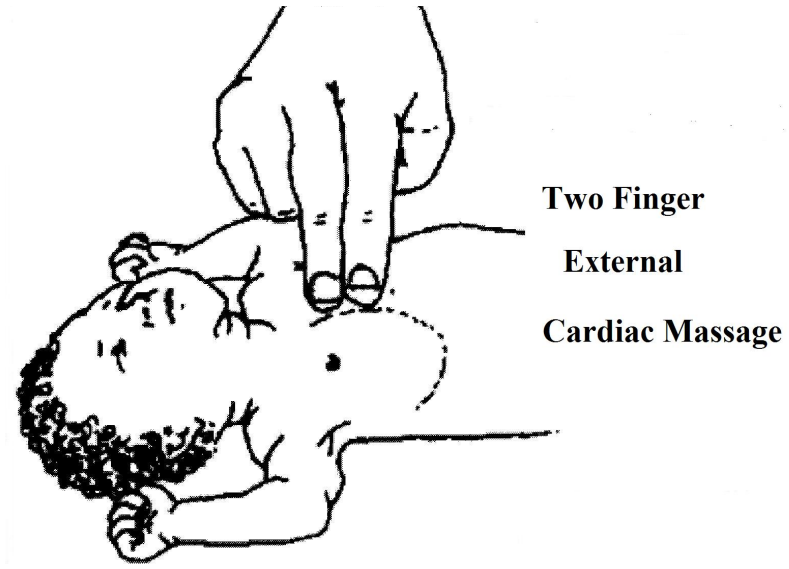
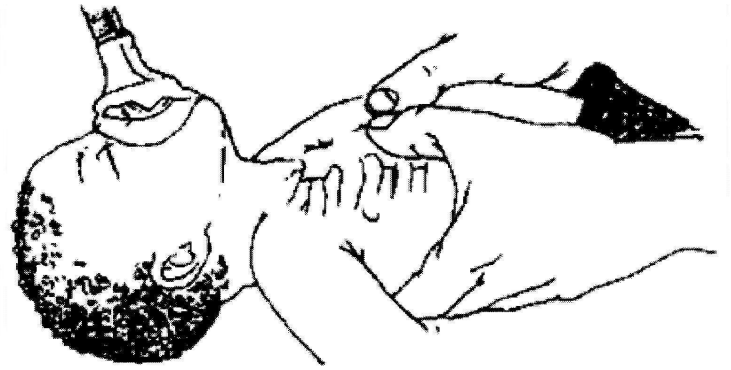


Fig 21.7 Cardiac massage in neonates

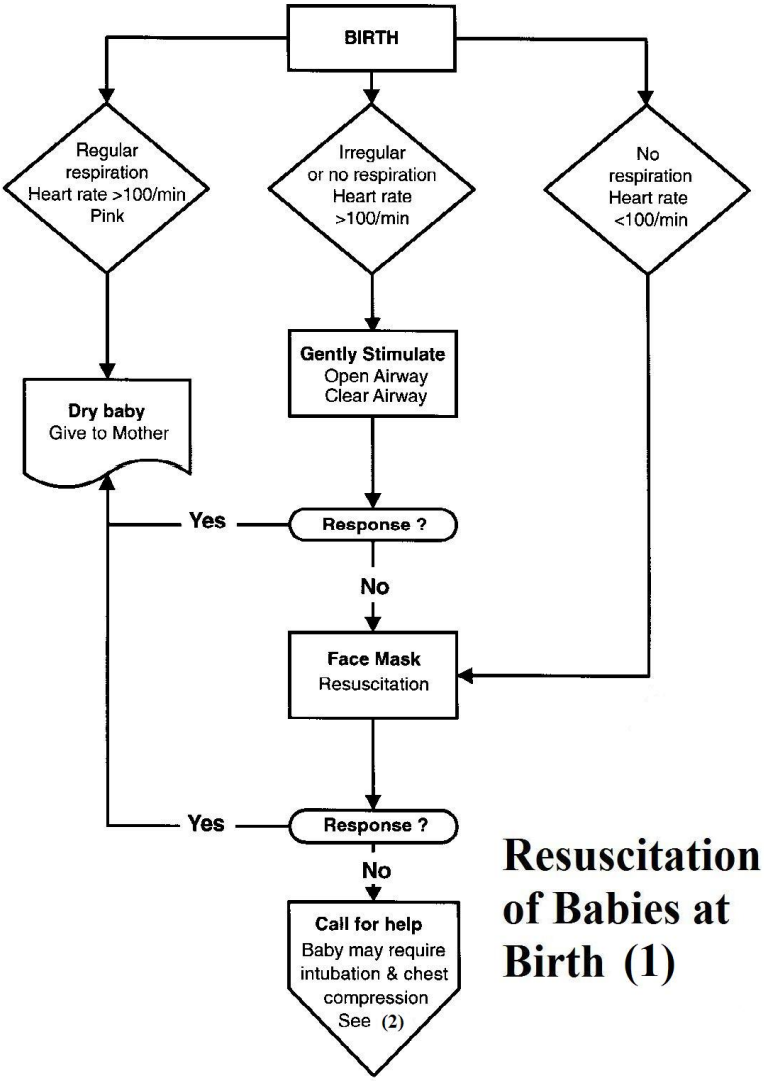


Oxygenation by Bag and Mask

and External Cardiac Massage

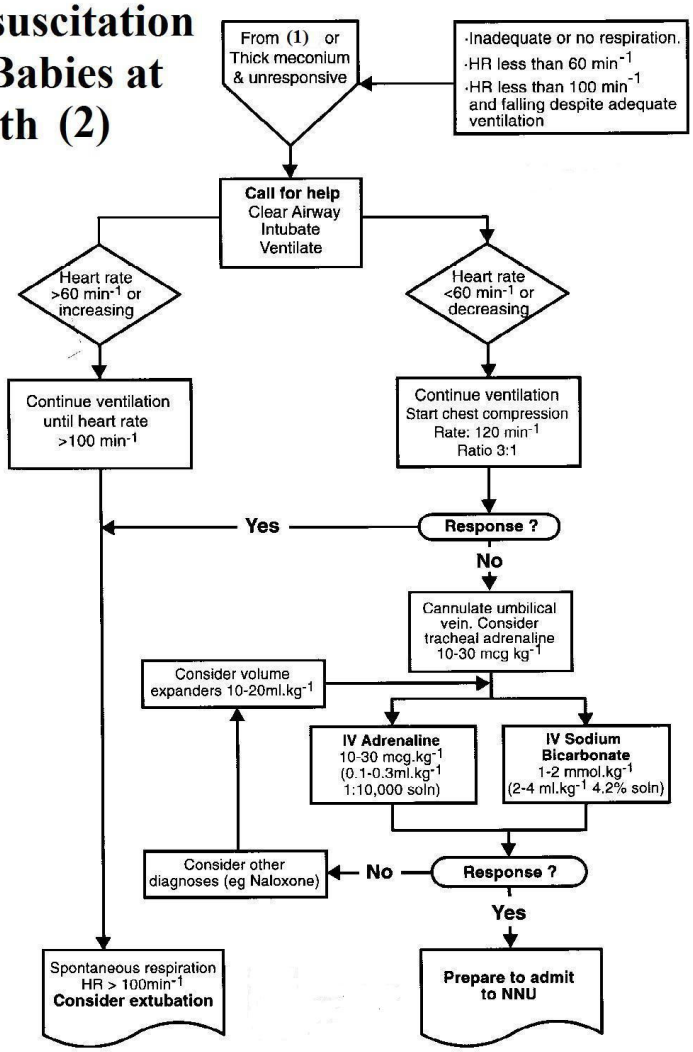
Fig 21.8

- Always consider hypoglycaemia as a cause of a "flat baby."
- Fluid bolus of 10 ml/kg of 0.9% saline or colloid if hypovolaemia.
- Naloxone if mother has received narcotics < 3 hours pre-delivery.



Resuscitation of Babies at Birth (1)

Resuscitation of Babies at Birth (2)



CHAPTER 22

NEUROSURGICAL ANAESTHESIA

Outline:

Types of neurosurgical cases likely to be encountered

Intracranial pressure
- Factors affecting ICP
- Signs and symptoms of raised ICP
- Dangers of raised ICP

Potential anaesthetic problems

Techniques of anaesthesia
- Local anaesthesia
- General anaesthesia

Special measures available to lower intracranial pressure

Problems facing the anaesthetist in neurosurgical anaesthesia and the principles of anaesthetic management of these cases will be discussed only briefly. For more detailed information consult a specialist text.

TYPES OF CASES

Neurosurgery can be elective or 'emergency'. Elective neurosurgery will include the removal of brain tumours and surgery for aneurysms. Such cases will be operated on in the larger hospitals and anaesthesia for procedures with them will be found in more advanced textbooks. Emergency surgery, however, will present itself. Head injuries are becoming more and more common as road traffic accidents increase in number.

Head injuries can include:

- Scalp lacerations
- Lacerations and contusions of the brain tissue
- Intracranial haemorrhage: extradural, subdural, subarachnoid
- Depressed fracture of the skull
- Cerebral oedema.

The danger of most of these head injuries (apart from the injury to the brain tissues and the blood loss, etc.) is that they can be associated with a rise in intracranial pressure. A rise in ICP results in ischaemia to the vital centres and later to 'coning'. It is with this danger in mind that patients with head injuries should be observed in the ward. The subject of intracranial pressure deserves consideration.

INTRACRANIAL PRESSURE

The intracranial contents consist of:

Brain tissue	24%
Water	60%
Blood	3-5%
CSF	11-13%

The intracranial pressure is the pressure exerted by the intracranial contents. It is usually 60 - 150 mm of H_2O (5-13 mmHg) in the horizontal position.
The cranium is a rigid structure and cannot expand to accommodate any increase in its contents. Any increase in the brain tissue, blood flow or CSF will cause a rise in ICP.

The intracranial pressure can rise for several reasons:

- Physiological causes: coughing, sneezing, straining, etc.
- Pressure from outside the brain e.g. the cranial bones and the outer coverings of the brain:
  - Bony tumours
  - Craniostenosis
  - Bleeding into the coverings of the brain.
- Pressure from within the brain i.e. any increase in the components of the intracranial space:

  Brain tissue – tumours
  CSF – hydrocephalus
  Blood – venous or arterial engorgement from any cause
  Water – cerebral oedema from any cause.
- Anaesthetic causes: straining, coughing, hypoxia, hypercarbia, over hydration and faulty positioning may raise ICP.
- Posture. The ICP is higher in the horizontal position and temporarily lower when standing erect.

Signs and symptoms of a raised intracranial pressure:

Signs:

- Pupils enlarged due to stretching and paralysis of the 3rd cranial nerve
- Papilloedema
- Blood pressure - systolic often rises significantly and the diastolic may fall
- Bradycardia
- Respiration may become irregular

Symptoms:

- Headache
- Vomiting
- Visual disturbances
- Other neurological changes, e.g. altered consciousness

Dangers of a rising intracranial pressure:

Before surgery:

- Ischaemia to the vital centres. This results in changes in the level of consciousness and changes in blood pressure and respiration.
- Danger of coning. By "coning" is meant the herniation of the contents of the cranial cavity into the vertebral canal. The vital centres in the medulla oblongata are interfered with and sudden death may occur.

During surgery:

The danger of coning is not present once the cranial cavity is opened but a raised intracranial pressure results in the following:

- The brain tends to swell. This makes the "closing up" procedure more difficult.
- The same danger of ischaemia to the vital centres is present.

POTENTIAL ANAESTHETIC PROBLEMS

Maintenance of a clear airway

The airway is especially important in neurosurgical anaesthesia:

- If the airway is even minimally obstructed the result will be hypoxia and hypercarbia and this leads to a rise in intracranial pressure.
- Coughing and straining on the tube has the same effect - raising intracranial pressure.
- Inaccessibility of the ETT and the connectors during a long operation makes it very important to secure the tube carefully.

The following rules must be remembered in relation to the airway:

- Intubation is essential in even the shortest case.
- A reinforced armoured tube is best. Size 9 for adult males and size 8 for adult females.
- Spray the cords with a local anaesthetic solution to prevent the patient from straining on the tube.
- Lubricate the tube well with lignocaine ointment.
- A cuffed tube is essential and a pharyngeal pack may be indicated in certain procedures, e.g. frontal craniotomies and fractures of the base of the skull.

Blood loss

- Make sure blood volume is restored before anaesthetising the patient.
- Make sure blood is cross-matched and ready before the operation is started.
  There is only one reason for starting an operation when blood is not available and that is when intracranial pressure is rising and causing rapid depression of the vital centres.
- A large intravenous cannula must be inserted before anaesthetising the patient.

The poor general condition of the patient

It is always important to look for other complications:

- Injuries to chest, jaw and face, abdomen and limbs.
- The chest may be contaminated by inhaled material.
- There may be a pneumothorax or haemothorax.

Careful monitoring

This is especially important in neurosurgical cases. Changes in the vital signs may reflect changes in the intracranial pressure. They may also reflect injury to vital centres as a result of surgery. If an oesophageal stethoscope, ECG monitor and capnography /oximetry are available they should be used.

Air embolism

The danger of air embolism is present, especially in the steep reverse Trendelenburg (head up) position and the sitting position. This is covered in the Complications of Anaesthesia. (See Chapter 46)

Positioning.

Reverse Trendelenburg, lateral and sitting positions are those commonly employed and this poses further problems for the anaesthetist. These have been described elsewhere. (See Chapter 30)

Diathermy

If diathermy is required, ether should not be used.

ANAESTHETIC TECHNIQUES

Intracranial Pressure must not be raised by the anaesthetic technique.

LOCAL ANAESTHESIA

If it is at all possible to operate on a head injury under local anaesthesia, do so. Local anaesthesia has the advantages of not increasing the intracranial pressure and also keeping the airway clear. Most of the intracranial structures are insensitive to pain.

Disadvantages: It is unsuitable for children and very uncooperative adults.

Indications for local anaesthesia:

- If experienced anaesthetists are not available.
- Minor surgery if the patient has an increased ICP.
- A rapid rise in ICP, e.g. following a middle meningeal artery haemorrhage.
- If a general anaesthetic is contraindicated because of the patient's medical condition. The following structures are sensitive and require local infiltration:
  - Skin
  - Scalp
  - Dura mater

GENERAL ANAESTHESIA

Pre-operative assessment. Note the following:

- Level of consciousness.
- Airway obstruction.
- Any signs of an increased intra cranial pressure.
- Any additional injuries.
- General condition of the patient.

Premedication is usually avoided. Opioids (e.g. pethidine or papaveretum) raise ICP by causing depression of respiration and carbon dioxide retention. Opioids also interfere with vital signs, e.g. constriction of the pupil.

Intra-operative management for emergency neurosurgical procedures.

Induction and intubation: This must be smooth. If the patient is in deep coma, or semiconscious, consideration of raised intracranial pressure management must be made. This means that induction and airway management must avoid hypotension (reducing cerebral perfusion) or hypertension, if possible. Head injuries are associated with a prolonged gastric emptying time. A rapid sequence induction is appropriate for all head injured patients, as described in Techniques of Anaesthesia (Chapter 16).

- Use a lubricated armoured tube.
- Preoxygenate the patient for 4 minutes.
- Give a selected dose of thiopentone (a smaller dose if the patient is in depressed coma or is unconscious, i.e. 1-2mg/kg) and suxamethonium.
- Apply cricoid pressure and intubate the patient.
- Do not use ketamine.
- A pharyngeal pack is advisable.

For elective neurosurgical procedures a rapid sequence induction is not necessary. Propofol and etomidate which reduce ICP can be used for induction. Spray the cords before intubating.

Maintenance:

- Air or $N_2 0/0_2$/Volatile-(low concentration) and non–depolarising muscle relaxant.
  Hyperventilation, ideally before the volatile agent is introduced, is necessary if there is raised intracranial pressure. However, the effects of hyperventilation only last for 8 - 12 hours and therefore this is only a temporary measure. The aim is for a PCO_2 of 30 – 35mmHg. Excessive hyperventilation can cause cerebral vasoconstriction and cerebral hypoxia.
- Maintain careful monitoring as described above
- Fluids: Hartmann's solution or 0.9% saline.
- Head up tilt may be useful.

Reversal:

- Atropine or glycopyrrolate and neostigmine
- Do not extubate the patient unless the respirations are regular and deep. If the patient tolerates the ETT leave it in place as it will protect the airway from contamination until the patient is awake.

Post-operative care

Monitor and record the following:

- Level of consciousness (See **Glasgow Coma Scale** below)
  - Fully conscious: awake and orientated. (score 15)
  - Semiconscious: unable to answer questions but responds to painful stimuli.
  - Unconscious: Does not respond at all to stimuli. (score 3)
- Pupils
- Pulse
- Blood pressure
- Respirations
- Ability to move limbs
- Fluid balance.

Sedation

Avoid narcotics (opioids). If the patient is restless always look for another cause, e.g. hypoxia, hypotension etc. Give narcotics only under the doctor's instructions.

IV Fluids

Give normal maintenance fluids i.e. 3L per day for adults (normal saline or Hartmann’s solution). 5% dextrose should be avoided if possible.

Eyes open:	*Score*
Spontaneously	4
To speech	3
To pain	2
Never	1
Best motor response:	
Obeys commands	6
Localises pain	5
Withdrawal from pain	4
Abnormal flexion	3
Extends to pain	2
No response	1
Best verbal response:	
Orientated	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
Silent	1

Fig 22.1 The Glasgow Coma Scale
(For assessing improving or deteriorating level of consciousness)

SPECIAL MEASURES TO REDUCE INTRACRANIAL PRESSURE.

- Hyperventilation using adequate muscle relaxation. Increase inspired oxygen concentration to 50%.
- Steroids: Dexamethasone - 8-12 mg IV may be used to reduce intracranial pressure (especially for cerebral tumours).
- Hypertonic agents.
  Mannitol 20% solution (20gms /100ml) in a dose of 0.25-1.0 g/kg. Average dose 0.5g/kg. Effect lasts for up to 2 hours.
- Head up tilt.
- Where the facilities for measurement are available maintain an adequate cerebral perfusion pressure of greater than or equal to 70mmHg. (Cerebral perfusion pressure = mean arterial blood pressure minus intracranial pressure or CVP, whichever is greater).
- Induced hypothermia.
- Diuretic e.g. frusemide in dose of 1mg/kg.
- Direct surgical tapping of the ventricle to allow CSF drainage. This should be of a small volume (< 5ml/min).

CHAPTER 23

ANAESTHESIA FOR OPHTHALMIC SURGERY

Outline:

Points of importance to the anaesthetist

Anaesthetic management
- Local anaesthesia
- General anaesthesia

Special problems associated with common eye operations

POINTS OF IMPORTANCE TO THE ANAESTHETIST

Intraocular pressure must not be allowed to rise, particularly in eye injuries. The eye should be soft, i.e. the intraocular pressure should be low when the anterior chamber is opened. The normal intraocular pressure is 15-21 mmHg.
General anaesthesia should not cause a rise in intraocular pressure.

Intraocular pressure is raised by:

- Pressure from an anaesthetic face mask
- Coughing, straining on the endotracheal tube
- Hypoxia
- Hypercarbia
- Suxamethonium
- Atropine (in patients with closed angle glaucoma) although it rarely causes a problem
- Hypertension
- Post-operative restlessness and vomiting.

Intraocular pressure is lowered by:

- Hypocarbia.
- Hypotension
- Diamox but this causes metabolic acidosis and hypokalaemia and a risk of arrhythmias
- Mannitol
- IV anaesthetics and analgesics
- Inhalational anaesthetics
- Non-depolarising relaxants

The Oculocardiac Reflex
Reflex bradycardia is caused by pressure or traction on the eyeball, especially in relation to the medial and lateral rectus muscles. Adequate monitoring to detect the arrhythmia and then prompt treatment with atropine is important. Cardiac arrests have been reported.

Control of the airway.
Intubate the patient with a tube or LMA with a minimum tendency to kink. It must have secure connectors. A pharyngeal pack may be necessary in certain types of operations, e.g. dacryocystorhinostomy.

Careful monitoring is essential.

Protection of other eye
Always protect the other eye, with ointment and/or tape and make sure that cleaning fluids do not pool in the eye.

Eye surgery and drugs

- Atropine occasionally raises the intraocular pressure in patients with closed angle glaucoma. This condition may occur during general anaesthesia if the patient is not being treated for the glaucoma. Post-operative red eye with poor vision and pain is acute glaucoma until proven otherwise.
- Suxamethonium raises the intraocular pressure by 7-8 mmHg or more. The onset of this rise is 30 seconds after the drug is injected. The duration usually coincides with the period of apnoea. The rise is minimised by:
  - Using a small dose of a non-depolarising muscle relaxant (⅓ of normal intubating dose) 3 minutes before suxamethonium.
  - Using 500mg Diamox IV before the induction of anaesthesia.

  Note: if needed, the use of suxamethonium to establish an airway is more important than any consideration of raised intraocular pressure.
- Non-depolarisers, intravenous induction agents such as thiopentone and inhalational agents all decrease the intraocular pressure.
- Ketamine raises intraocular pressure by a small amount.

Medical problems associated with eye surgery

- Dystrophia myotonica may be associated with congenital cataracts.
- Diabetes mellitus may be associated with congenital cataracts.
- Malignant hyperpyrexia has been linked with muscle disorders, e.g. squints, dystrophia myotonica.
- Patients who may have exophthalmos secondary to thyrotoxicosis may present for tarsorrhaphy.
- Many patients are in a poor general state of health, especially those presenting for cataract surgery.

ANAESTHETIC MANAGEMENT
If surgery can be safely and efficiently done under local anaesthesia, then local anaesthesia should be used.

LOCAL ANAESTHESIA

Contraindications to local anaesthesia:
- A coagulation defect
- The patient is unsuitable e.g. unable to co-operate, demented, deaf, having language problems, extremely nervous
- The patient refuses
- An uncontrollable cough
- The patient cannot lie flat because of cardiac or respiratory disease
- An unstable medical condition
- An allergy to local anaesthetic
- Perforating eye injuries.

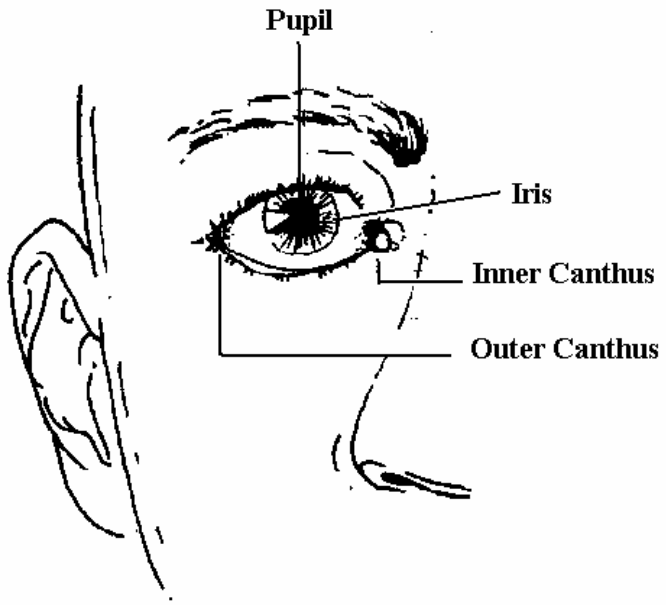


Fig 23.1 Anterior view of the eye and surrounding tissues

SUBCONJUNCTIVAL LOCAL ANAESTHESIA

Method

- Prepare peri-orbital skin with betadine solution 5% or equivalent.
- Instil two drops of ophthalmic local anaesthetic.
- Instil 5 drops of local anaesthetic eye drops (benoxinate, amethocaine, lignocaine 4% ophthalmic drops or proparacaine, NOT the injection solution) into the conjunctival sac while gently pulling the lid away from the eye to form a little reservoir for the drops to pool. Lignocaine 4% drops will sting, so use a drop or two of another ophthalmic anaesthetic before the lignocaine drops
- Wait five minutes, then inject the local anaesthetic solution:
  - 1 ml of lignocaine 1% with 1:100,000 adrenaline for routine cataract or glaucoma surgery. Hyaluronidase is **not** recommended, because it shortens the half life of the local anaesthetic.
  - Either hold the eyelids open, or use a speculum. Instruct the patient to look into the position of gaze opposite to that of the chosen site of injection (usually to the opposite shoulder or feet). With the bevel of the 30 mm (long) 25 gauge needle facing the conjunctiva, enter the conjunctiva for about 2 mm. This will allow the membrane to be lifted away from the sclera so that the end of the needle can be seen at all times in the subconjunctival space as the drug is slowly injected. For glaucoma surgery the needle entry site has to be made at least 10 mm away from the site of the intended fistula.
  - For penetrating keratoplasty (corneal grafting) and intracapsular cataract extractions inject an additional 0.5 - 1 ml into the opposite quadrant.
  - For strabismus and retinal detachment surgery, give additional anaesthetic by local infiltration around the muscles as necessary.

Some patients can tolerate an hour and a half of eye surgery, others only an hour.
Mild sedation and systemic analgesia will help.

Eyelid anaesthesia usually helps in all intraocular surgery and is used for repairs and removal of lesions.

- 0.5 ml Lignocaine 1% with 1:100,000 adrenaline is injected in the sub-muscular plane of the outer third of the upper lid to block the cutaneous branch of the lacrimal nerve.
- Block the supra-orbital nerve with another 0.5 ml over the supra-orbital foramen. Then, take great care to keep the needle under the skin in order to advance it posteriorly and medially above the medial canthus for about 5 mm and inject 0.5 ml subcutaneously. This is to anaesthetise the supra-trochlear nerve supplying the medial part of the upper lid.
- Gentle aspiration is necessary here because of the danger of injecting into the anterior facial vein.
- Inject 0.5 ml over the infra-orbital foramen to anaesthetise the lower eyelid.

For minor lid surgery, local anaesthetic drop instillation makes lid infiltration virtually painless if it is performed after 5 minutes through the conjunctival surface of the everted lower lid. The upper lid is not as easy to evert.

Eye movements may be a nuisance particularly during certain delicate stages of intraocular manoeuvres. Most patients fortunately are able to maintain a steady gaze on demand. About 10% of patients are unable to keep their eyes still during surgery for more than a few minutes, while about 1 in 50 cannot prevent constant eye movements at all. Eye movements are prevented in this situation by very light sedation. Avoid heavy sedation if possible because of the real chance of sudden wakening, violent eye and head movements and confusion.

Important advantages of subconjunctival local anaesthesia

- The eyes are very soft without any pre-operative massage.
- There is no risk of retrobulbar haemorrhage.
- Because the needle end is visible at all times, perforation of the eye is most unlikely.

Other advantages of subconjunctival local anaesthetic or drops

- The complication rate is very low.
- Anaesthesia is easy.
- Very soft eye for cataract and glaucoma surgery, probably because there is no retrobulbar mass effect from the anaesthetic and slow ooze from capillary bleeding.
- It is cheap.

Complications of subconjunctival local anaesthesia

- Failure of anaesthesia, usually due to a low potency drug, is easily avoided with another injection.
- Perforation of the globe is very rare. Prevent it by keeping the end of the needle in view and by injecting no more than 3 to 5 mm posterior to the limbus.
- Excessive eye movements (1 in 50 patients approximately) are prevented or reduced by light sedation, or ketamine. Such movements are often caused by poor anaesthesia of the eyelids, e.g. as a result of low potency anaesthetic or out of date drugs.
- If a painful superior rectus bridal suture is to be used, supplement local anaesthetic in the region of the superior rectus muscle. The surgeon may use 6/0 corneal fixation suture (spatulate needle) to avoid this in glaucoma surgery.

Drops for eye surgery

Lignocaine 4% ophthalmic drops, used after one or two standard local anaesthetic drops alone, will also provide enough analgesia to perform cataract and glaucoma surgery. Facial nerve block and probably lid infiltration should be used as well.

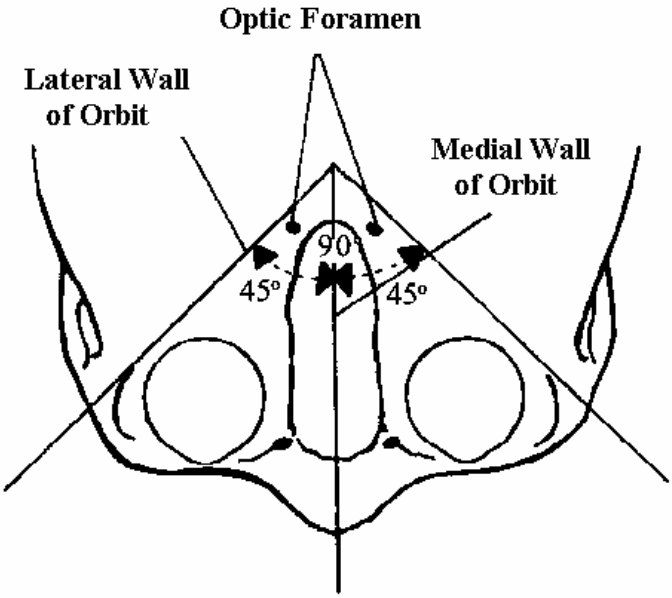


Fig 23.2 The orbit viewed in section from above

PERIBULBAR BLOCK

Pre-operative management

- Assess the patient as for GA. The medical state must be optimal before elective surgery.
- Explain the procedure to the patient.
- Check blood pressure and leave the cuff on the patient's arm. Check pulse, ECG, oxygen saturation (SaO_2) available.
- Give IV sedation only if absolutely necessary, for instance if there is danger of losing the patient's co-operation, if there is airway obstruction or respiratory depression.

Intra-operative management

- Drugs and equipment:
  - Surface anaesthetic for the eye, e.g. Benoxinate
  - 2% Lignocaine with Adrenaline 1/200,000 (Adrenaline use is controversial, so check with eye surgeon before use).
  - Hyaluronidase if available: 6 - 10 Turbidity Reducing Units.
  - 25G needle for weal. 23G x 35mm needle for block.
- Technique of peribulbar block:
  - Establish IV access and monitoring
  - Light sedation can be used if required
  - Instil topical local anaesthetic drops to conjunctiva
  - The patient should lie supine and look straight ahead
  - Palpate the inferior orbital rim to find groove at junction of medial two thirds and lateral one third
  - Just lateral to and 1mm above this point insert a 25G/25mm long needle, with syringe attached, slowly along the orbit floor until the hub of the needle reaches the plane of the iris

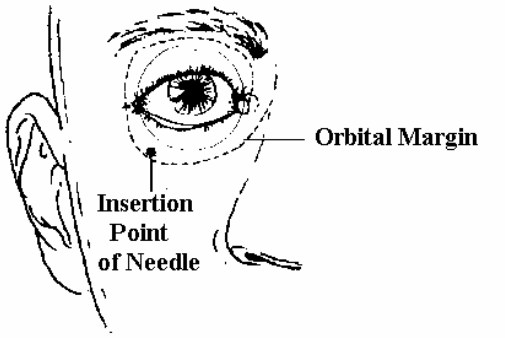


Fig 23.3 View from the front

- Aspirate then inject 6-8 ml of local anaesthetic. Check for tension in globe and stop injecting if this occurs

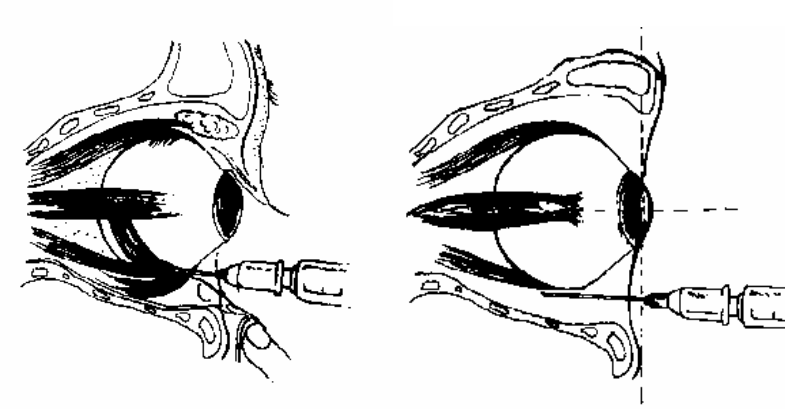


Fig 23.4 View from the side **Fig 23.5 View from above**

- Withdraw needle and inject small volume of local anaesthetic in orbicularis of lower lid
- Sometimes a second injection is required. Using a similar needle, insert it on the nasal side medial to the caruncle and parallel to the medial orbital wall. A further 4–5ml of solution is injected

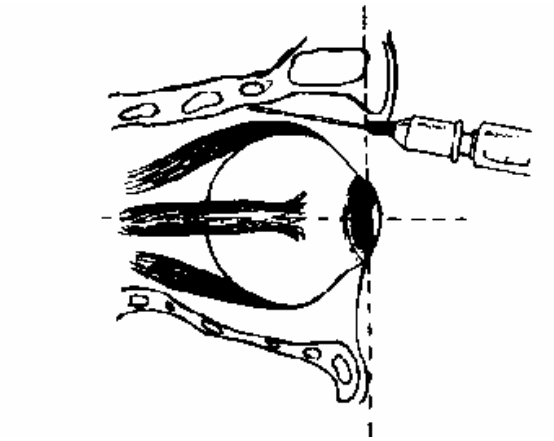


Fig 23.6 View from above

- Apply digital compression or a compression device to spread local anaesthetic and normalise intraocular pressure.

Advantages of peribulbar block

- Large volumes of local anaesthetics are placed outside the muscle cone. They spread to the apex and all compartments of the eye.
- Provides good analgesia while reducing the risk of dangerous complications.
- Well accepted by the patient with very little discomfort during injection.
- Facial nerve block is not necessary.

Disadvantages of peribulbar block

- Onset slower than the retrobulbar block.
- Large volumes of local anaesthetic used can cause toxicity.
- Globe perforation is possible.
- Intraocular pressure may be raised by the volume of solution.

RETROBULBAR BLOCK

The technique described involves introducing a needle percutaneously from an infero-temporal point aiming at the apex of the orbit. A small volume of local anaesthetic is deposited in the extraocular muscle cone. To prevent the contraction of the orbicularis oculi muscle, a facial nerve block is performed. Preparation of the patient is as for peribulbar block.

- Technique of retrobulbar block:
  - Patient in supine position
  - Gaze supero-nasal
  - Instil 2 drops of local anaesthetic solution.
  - Swab eyelids and surrounding area with antiseptic.
  - Palpate the inferior orbital rim with the index finger.
  - Raise a skin weal at the junction of the lateral and inferior orbital margin.
  - Introduce the 35mm long needle through the weal and direct it along the orbital floor until the tip of the needle has passed the equator of the eye (approx. 15mm). Redirect the needle upwards and inwards, in the direction of the apex of the orbit. Penetrate the fascia of the muscle cone and aspirate and inject 2.5 ml. Apply intermittent digital compression of the eye for 2-4 min.
  - Examine the eye for akinesia (i.e. check the movement of the eyeball). There should be little or no movement of the anaesthetised eyeball.
  - If there is significant movement of the eye, make a supplementary injection alongside the rectus muscle that is active.
- Monitoring:
  Level of consciousness, pulse, blood pressure, respiration, SaO_2.

Complications of retrobulbar block

Local complications:

- Retrobulbar haemorrhage
- Globe perforation
- Central retinal artery occlusion
  - Secondary to retrobulbar haemorrhage
  - Puncture of dura around optic nerve
- Optic nerve damage
- Periorbital ecchymoses

Systemic complications:

- Subarachnoid injection of local anaesthetic.
  - Cranial nerve block
  - Respiratory arrest
  - Cardiac arrest
- Intravascular injection: grand mal seizure.

Retro-bulbar block is still occasionally required but is much less frequently used than sub-tenon or peri-bulbar blocks.

TECHNIQUES OF FACIAL NERVE BLOCK

Van Lint technique*:* This blocks the terminal branches of the facial nerve.
Raise a weal 1 cm behind the point of intersection of the inferior and temporal orbital rims.
Insert a 1cm long needle into the deep tissues along the inferior orbital margin and inject 2-3 ml of local anaesthetic while withdrawing the needle.
Make a second injection through the same weal along the superior temporal margin of the orbit. Again inject 2-3 ml of local anaesthetic while withdrawing the needle.

O'Brien technique*:* This blocks the facial nerve proximally, anterior to the tragus of the ear below the posterior portion of the zygomatic process; directly over the condyloid process of the mandible which is identified by asking the patient to open and close the mouth.
Advance the needle until the condyloid process is reached.
Withdraw the needle slightly and then inject 2ml of 2% lignocaine with adrenaline.

SUB–TENON BLOCK

This block is becoming more commonly used as a safer alternative to retro and peri-bulbar blocks.

Method

- Apply topical local anaesthetic drops to conjunctiva.
- Retract lower lid.
- Lift the conjunctiva in the infero-nasal quadrant, with forceps, 5-7mm from the limbus.
- Make a small incision in the conjunctiva and dissect infero-nasally in the plane between the sclera and Tenon's capsule. (Tenon's capsule is white and avascular unlike the sclera).
- In this plane a blunt curved cannula (Southampton) is passed backwards beyond the equator of the eye and 3-5ml of local anaesthetic deposited.

Advantages of Sub–Tenon block

- Easy to perform
- Avoids complications of globe perforation, haemorrhage or optic nerve damage
- Technique of choice in anticoagulated patients
- Suitable for patients with axial length > 26mm

Disadvantages of Sub–Tenon block

- Dose not give such complete anaesthesia as retro and peri-bulbar blocks and does not provide akinesia. A mild anxiolytic premedication may be required.
- Occasional conjunctival haemorrhage. This is easily treated with cautery.

For a better understanding of ocular anatomy with illustrations and a more detailed description of these techniques consult a textbook of ophthalmic surgery

GENERAL ANAESTHESIA

Pre-operative management requires routine assessment and reassurance. Warn patients that they will not be able to see for a while after their operation. Premedication should be aimed at sedating patients. An anti-emetic, e.g. promethazine or prochlorperazine, should be incorporated in the premedication to reduce the incidence of post-operative vomiting.

Intra-operative management

- Induction. Thiopentone or propofol.
- Intubation. Spray cords with 10% lignocaine. Intubate using a non-depolarising muscle relaxant (suxamethonium is used if there is a risk of aspiration). An armoured or RAE tube is best, because this reduces the incidence of obstruction.
- Maintenance
  - Air or Nitrous Oxide/Oxygen/Volatile/Relaxant (non depolariser). Supplemented by fentanyl, pethidine or a non-steroidal suppository PR for analgesia if needed.
  - Monitor carefully and give fluids if required.
- Reversal

  Routine reversal with atropine 600 micrograms or glycopyrrolate 500 micrograms and neostigmine 2.5 mg (adult). Atropine 10-20 micrograms/kg or glycopyrrolate 10-15 micrograms/kg and neostigmine 50micrograms/kg (paediatric)

Technique using a laryngeal mask

In elective patients a LMA or reinforced LMA can be used as an alternative to intubation for short procedures. The patient can be allowed to breathe spontaneously or can be ventilated using a non-depolarising agent such as atracurium or vecuronium. IPPV via a LMA is usually satisfactory except in obese patients or those with a history of hiatus hernia.
This technique allows a smooth induction and awakening with less risk of coughing or straining.
Ensure a good placement and clear airway with good respiratory expansion.
A lighter level of anaesthesia can be used than would be necessary for a spontaneously breathing intubated patient.
Careful monitoring of heart rate, blood pressure, oxygen saturation and capnography (if available) is important.

Post-operative care following general anaesthesia
Try to prevent post-operative restlessness, coughing, nausea and vomiting.

COMMON EYE OPERATIONS (and potential anaesthetic problems)

Penetrating eye injury

The main problem is when the patient presents with a full stomach. This necessitates a rapid sequence induction with cricoid pressure and the use of suxamethonium which raises intraocular pressure.
If intubation is likely to be straightforward a *modified* rapid sequence induction can be used by giving rocuronium (if available) or vecuronium. Preoxygenate and apply cricoid pressure first as for standard RSI.
However if intubation is expected to be difficult it is preferable to ensure good relaxation by using suxamethonium. The theoretical risk to the eye is not as great as the risk to the patient's life. If thiopentone or propofol are used as induction agents their beneficial effect in lowering intraocular pressure will offset the potential rise following suxamethonium.
Two precautions may be taken to counteract the rise in intraocular pressure:

- Precede the suxamethonium by a small dose of non-depolarising muscle relaxant 3 minutes prior to induction (one third of intubating dose).
- Use diamox 3-5 mg/kg IV before the suxamethonium.

Strabismus or squint surgery

- Watch for the oculocardiac reflex.
  There is danger of sudden and profound bradycardia as a result of traction on the extra-ocular muscles via the oculocardiac reflex.
  When traction is released the heart rate normally returns to normal but a dose of atropine 0.02mg/kg may be required.
  In children a prophylactic dose of atropine is usually administered preoperatively.
- Watch for early signs of malignant hyperpyrexia.
  Malignant hyperpyrexia has been linked with muscle disorders such as squints.

Probing the naso-lacrimal duct

This is a short procedure, carried out under a mask anaesthetic using N_2O / O_2 and halothane. Ketamine anaesthesia is also useful for this procedure.

Cataract Extraction

This is usually performed under local anaesthesia but some patients require general anaesthesia eg those with difficulty in lying still or lying flat, in children and in patients with learning disabilities. For this relatively short procedure GA using LMA with spontaneous breathing is suitable.

CHAPTER 24

ANAESTHESIA FOR EAR, NOSE AND THROAT SURGERY

Outline:

Points of importance to the anaesthetist

Management
- Local anaesthesia
- General anaesthesia

Special procedures:
- Operations on the ear
- Operations on the nose
- Surgery in the mouth/pharynx and salivary glands
- Surgery on the larynx, trachea and bronchi
- Oesophagoscopy

POINTS OF IMPORTANCE TO THE ANAESTHETIST

Protect the airway from contamination by blood, secretion, or pus
Under general anaesthesia this is achieved by:

- Using a cuffed endotracheal tube.
- Using a pharyngeal pack.
- Ensuring the rapid return of reflexes at the end of the operation.
- Nursing the patient in the tonsillar position post-operatively.
- Avoiding use of local anaesthetic solutions in the respiratory tract as this interferes with the cough reflex post-operatively.

Prevent airway obstruction

- Use a RAE or non-kinkable armoured endotracheal tube.
- Choose the largest tube which can be used safely.
- Ventilate by hand, rather than mechanically, since the slightest degree of obstruction will be apparent.

Reduce bleeding to a minimum

- Good anaesthetic techniques such as a smooth induction and intubation, maintaining a clear airway and intermittent positive pressure ventilation help contribute to this.
- Use a slight head-up tilt.
- Use vasoconstrictor agents, e.g. adrenaline or felypressin.

Surgeon and anaesthetist share the same airway
This limits access to the airway for the anaesthetist intra–operatively

Post-operative complications are serious

- Airway obstruction
- Bleeding
- Aspiration into the respiratory tract of blood, pus or secretions
- Vomiting (common after middle-ear operations)

Other anaesthetic considerations

- Premedication must not be excessive, as a rapid return of the cough reflex is important.
- Vasoconstrictor drugs such as adrenaline must be used cautiously with halothane (as mentioned elsewhere). Halothane can be used if the amount of adrenaline is restricted to 5 micrograms/kg. (Adrenaline conc. not more than 1:100,000 and max adult dose 10ml in 10 mins or 30ml in 1 hour) However, if other anaesthetic agents are available, it is good practice to avoid the simultaneous use of adrenaline and halothane. If halothane is used it is best to turn off the halothane during infiltration and hand ventilate the patient, to avoid a rising carbon dioxide. Restart halothane immediately after infiltration.
- Cautery will preclude the use of flammable anaesthetics such as ether.

ANAESTHETIC MANAGEMENT

Local Anaesthesia can be used for many of these procedures. If there is no experienced anaesthetist it is the method of choice. Bleeding is reduced, the patient is awake and hence the protective cough reflex is present.

General anaesthesia

Pre-operative management

Make the usual pre-operative assessment. For premedication an oral benzodiazepine or IM opioid and atropine in the usual doses may be used depending on the proposed surgery.

Intra-operative management

- Induction. Use IV thiopentone or propofol in patients in whom there is no evidence of respiratory obstruction. An inhalational induction may be indicated in very young children or those with airway obstruction.
- Intubation usually follows a standard dose of suxamethonium chloride. Use a RAE or armoured tube and insert a pharyngeal pack. If intubation problems are likely it is safer to intubate the patient following an inhalational induction with an agent such as halothane or ether or under local anaesthetic with the patient awake.
- Maintenance. Air or Nitrous oxide, oxygen, volatile and non-depolarising relaxant. Halothane may be used as a supplement when there is no other agent available but with caution if adrenaline is used.
  An anti-emetic e.g. metoclopramide or cyclizine if available may be useful in reducing postoperative nausea.
- Reversal. Use atropine or glycopyrrolate and neostigmine.
  If a pharyngeal pack has been used make sure it has been removed
  Follow the usual rules for extubation but make sure the patient has adequate airway reflexes before extubation.

Post-operative management

Place the patient in the tonsillar position (left lateral, head down) and observe carefully for post-operative obstruction and bleeding.

SPECIAL PROCEDURES:

OPERATIONS ON THE EAR

Removal of foreign bodies
This can be done under local anaesthesia in adults. In children and in less co-operative adults, two techniques are commonly used:

- A spontaneous respiration technique using thiopentone induction, air or nitrous oxide, oxygen and volatile.
- In the absence of volatiles, ketamine may be used in a dose of 1-2mg/kg IV **or** 5-7mg/kg IM. Repeat 0.5mg/kg as necessary if the patient is moving.

Use diazepam / midazolam and atropine if required.

Myringotomy
The techniques as described for removal of foreign bodies are suitable.

Tympanoplasty
This operation is performed in more specialised units. It is a fairly long operation usually lasting more than one hour. Intubation is essential for keeping the airway clear. Bleeding must be reduced to a minimum. Nitrous oxide, if used must be turned off 15 minutes before a graft is applied. Nitrous oxide is 35 times more soluble in the blood than nitrogen. During anaesthesia, the nitrous oxide enters the air filled cavity 35 times faster than nitrogen leaves the cavity. This results in the graft being lifted.

Exploration of the facial nerve
This is performed under endotracheal anaesthesia. If the nerve is to be tested with a nerve stimulator, a reasonable technique would be to use suxamethonium to intubate, followed by spontaneous ventilation with oxygen, nitrous oxide and halothane.

OPERATIONS ON THE NOSE

Foreign body in the nose
This is common in children. The techniques described for removing of foreign bodies in the ear are suitable.

Reduction of nasal fractures
This usually requires general anaesthesia but does not often require to be done as an emergency and most patients can be left overnight or for 2-3 days.
If the procedure is likely to be quick it can be done using pre-oxygenation, IV thiopentone or propofol and head down position but the safest technique involves the use of a cuffed endotracheal tube and pharyngeal pack.

Note:

- Ventilating of the patient with a mask may be difficult if the nose is broken.
- When done as an emergency undigested food or the swallowed blood in the stomach means that these patients must be treated as having a full stomach and RSI should be used.
- Intra-operative bleeding requires the insertion of a pharyngeal pack.
- Early awakening with the return of good airway reflexes is important.
- If a nasal pack is inserted post manipulation it will prevent the patient breathing through the nose post-operatively. It is important to warn the patient of this beforehand.
- Blood loss should be assessed carefully. Intravenous fluids or blood may be required.

Nasal polypectomy

This can be performed under local anaesthesia. If you use general anaesthesia for nasal polypectomy or septoplasty you need to follow the principles mentioned at the start of the chapter. It is important for the surgeon not to use cocaine as a vasoconstrictor if a general anaesthetic is given but if cocaine is considered essential then ECG monitoring should also be used. Adrenaline, if used in a safe dose, is permitted. If adrenaline is used, halothane is best avoided (see above).

SURGERY IN THE MOUTH, PHARYNX AND SALIVARY GLANDS

Tonsillectomy and adenoidectomy

For these procedures use an oral RAE or preformed endotracheal tube.

This is appropriate for both the child and adult. It is important to be aware of the possibility of tube obstruction or kinking. The blood loss must be carefully assessed. Intravenous fluids are given to compensate for the pre-operative fasting, intra-operative blood loss and the low post-operative intake. At the end of the operation, the pharynx should be gently sucked out under direct vision including the nasopharynx behind the soft palate where clots can accumulate. This can be done by turning the end of the Yankauer sucker upwards.

Positive pressure on the bellows or bag during extubation may result in the patient exhaling immediately after extubation. Any blood clot in the larynx or trachea will therefore be coughed up.

Post tonsillectomy haemorrhage

This presents two major problems for the anaesthetist:

- The problem of blood loss and the "shocked" patient.
- The problem of the full stomach. Most of the blood lost is swallowed. The patient must be adequately resuscitated pre-operatively. Insert a large cannula for giving fluid and blood.
  Continue giving these until the patient is considered normovolaemic, as judged by the pulse, colour, skin turgor, tongue, urine output and blood pressure. Blood must be available for intra-operative use. Use a rapid sequence induction with pre-oxygenation and cricoid pressure and continue to monitor blood loss carefully.

Peritonsillar abscess

Draining such an abscess releases a large amount of pus that may flood the larynx and be aspirated into the lungs. Intubation may be difficult because the muscle spasm (trismus) is not always relieved by muscle relaxants. Local anaesthesia is the method of choice.

Cleft lip

The cleft lip may be as simple as a notch on the upper lip, or as serious as a cleft on the lip extending to the floor of the nose. It may be unilateral or bilateral. Repair is carried out at approximately 3 months of age. The usual principles of paediatric anaesthesia and the principles listed above for surgery in the oral cavity should be followed.

- A good intravenous line is necessary. It may be inserted into the foot.
- An oral tube such as a RAE tube can be used, with the connectors brought down over the chin in the midline.
- Unless the anaesthetist is very experienced, it may be advisable to give the patient an inhalational induction with air/nitrous oxide, oxygen and volatile and intubate while the child is breathing spontaneously. **No muscle relaxants should be given until the anaesthetist is able to ventilate easily with a face mask.**
- After intubation, the child is paralysed and ventilated.
- A pharyngeal pack should be inserted to absorb blood that trickles into the throat.
- Careful suction is necessary at the end of the operation after the pack is removed and the patient extubated.

Cleft palate
This may exist as a cleft or deficiency in the soft palate only, or as a defect involving both the soft and hard palates. The condition is repaired at about 12 months of age.

- Cleft palates are very often associated with other congenital abnormalities, e.g. the "Pierre Robin" syndrome in which the babies have micrognathia (poorly developed mandible) and a large tongue. These babies are difficult to intubate and an inhalational or local anaesthetic intubation is safest. The same rules apply as for anaesthesia for cleft lip above.
- Babies with cleft palates are prone to chronic upper respiratory tract infections. Infected nasal secretions, or fever associated with a running nose would be a contraindication to anaesthesia.
- Blood loss may sometimes be sufficient to require transfusion.
- Observe the same precautions outlined for cleft lip.

SURGERY IN THE LARYNX, TRACHEA AND BRONCHI

Laryngoscopy (inspection of the larynx)
This may be required for diagnosis, removal of foreign body or treatment of pathological conditions such as excision of tumours.
Indirect laryngoscopy is performed with a laryngeal mirror inserted into the mouth.

- Local anaesthesia is the best method in co-operative adults.
- Give general anaesthesia in children and in unco-operative adults.
  - if there is no pre-operative obstruction, inspection of the larynx may be carried out with:

    IV Ketamine 2mg/kg

    or

    Pre-oxygenation for 4 minutes followed by thiopentone, suxamethonium and further ventilation with oxygen before the laryngoscopy. A tube 1 or 2 sizes smaller than normal can be inserted and used to provide anaesthesia and/or ventilation if the procedure is prolonged.
  - If patients have pre-operative stridor, the larynx must be inspected under an inhalational anaesthetic while the patient breathes spontaneously. Surgical procedures on the larynx require very experienced anaesthetic skills and equipment. If there is no experienced anaesthetist, they are best not attempted. In an emergency, insert a small sized endotracheal tube (depending on the patient). The patient can be paralysed and ventilated through the tube, or breathe spontaneously. This gives the anaesthetist control of the airway and gives the surgeon access to operate around the tube.

Bronchoscopy
This is performed for diagnosis and for therapy.
Diagnostic bronchoscopies are performed in cases of lung or bronchial pathology, while therapeutic bronchoscopies are done to remove foreign bodies and mucus plugs, etc.

If the patient is very ill then the bronchoscopy is best performed under local anaesthesia, administered by the surgeon.

General anaesthesia for bronchoscopy (adult)

The following items must be available

- A means of ventilating the patient, either the anaesthetic machine or a bag/valve/mask "set-up".
- A source of oxygen
- Suction
- Guedel airway
- An endotracheal tube which fits the upper end of the bronchoscope
- Drugs: An induction agent and suxamethonium
- Laryngoscope.
- An endotracheal tube of the correct size for the patient.

Pre-operative management: assess as usual.
Premedication depends on the general condition of the patient.

Intraoperative management
Check the blood pressure. Insert an indwelling cannula. Preoxygenate the patient for four minutes and give a sleep dose of thiopentone or propofol. This is followed by 60-80 mg of suxamethonium. Ventilate the patient with 100% oxygen. Spray the cords with 4% lignocaine spray 2–3ml if available.

While the surgeon performs the bronchoscopy the anaesthetist is responsible for:

- Very careful observations of
  - Pulse
  - Colour
  - Oxygen saturation
  - Blood pressure
  - Evidence of the patient waking up.
- Intermittent oxygenation. Do this by using an endotracheal tube which fits into the upper end of the bronchoscope. The tube is connected to a ventilating device (anaesthetic machine or bag/valve) and the patient is ventilated intermittently with 100% oxygen.
- Supplementary doses of thiopentone (25-50 mg at a time) or propofol (10-20mg) if the patient shows evidence of waking up.
- Supplementary doses of relaxant (25 mg of suxamethonium) if there is evidence that muscle power is returning, e.g. swallowing.

Alternative methods of oxygenation during bronchoscopies are the use of a ventilating bronchoscope, or a venturi attachment.

Post-operative management

The bronchoscope should be left in place till the patient is breathing spontaneously. It is essential that the "cough reflex" is active at the end of the procedure.

After the bronchoscope is removed, perform suction on the patient very carefully. Give 100% oxygen by mask for a few minutes and turn the patient on his side.

Bronchoscopy in children

A variety of bronchoscopes may be available and the planned technique should be discussed with the surgeon.

There are two techniques:

- ***Inhalation anaesthesia using halothane*** (ether is explosive and best avoided). An appropriate dose of atropine should be given. Induce anaesthesia with halothane, gradually increasing the concentration of halothane as described in Techniques of Anaesthesia. (See Chapter 16) Do not start the bronchoscopy until the patient is deep enough. Spray the cords with local anaesthetic.

  The level of anaesthesia is maintained by intermittent insertion of an endotracheal tube through the bronchoscope or by attaching a T–piece and ventilating with oxygen/volatile through the side arm. If there is a foreign body in the respiratory tract, induction may be slower.
- ***Intramuscular ketamine with intermittent suxamethonium***. This technique is similar to that described for adult patients. It is important to select an endotracheal tube that fits into the upper end of the bronchoscope to ventilate the patient.

  Premedication: Give atropine in the usual dose.

  Give a small intramuscular injection of ketamine 5mg/kg. This provides about 20 minutes of dissociative analgesia.

  Pre-oxygenate for four minutes, then give suxamethonium 1mg/kg IV and ventilate the patient with 100% oxygen. The patient is now ready for the bronchoscopy. Give intermittent ventilation via the endotracheal tube inserted into the bronchoscope. If more suxamethonium is necessary, give half the original dose.

OESOPHAGOSCOPY

Oesophagoscopy can be performed for therapy or diagnosis. Therapeutic oesophagoscopy is indicated for removal of a foreign body in the oesophagus or dilation of an oesophageal stricture. Diagnostic oesophagoscopies are performed for examination and biopsy of lesions in the oesophagus.

Important points relating to anaesthesia for oesophagoscopy

- Partial obstruction of the endotracheal tube by pressure from the rigid oesophagoscope. An armoured tube is ideal. It is best to ventilate manually, so that you can detect even minimal obstruction of the airway.
- Relaxation of the post cricoid sphincter enables the oesophagoscope to be inserted with ease. Use muscle relaxants to do this.
- Coughing and straining may cause rupture of the oesophagus. A smooth anaesthetic is essential.
- Obstructive lesions in the oesophagus and even foreign bodies may cause secretion or food to accumulate. Patients needing an oesophagoscopy must therefore be treated as "full stomachs". A rapid sequence induction with cricoid pressure is necessary.
- Trauma and bleeding may occur especially after an oesophageal biopsy. A rapid return of reflexes at the end of the operation reduces the incidence of aspiration into the lungs. It is best to extubate when the patient is awake and positioned on his side.
- In the very young patient, an oesophagoscopy, especially if it is difficult or prolonged, is likely to increase the danger of laryngeal oedema.

Technique for general anaesthesia for oesophagoscopy

Pre–operative management

Premedication. Often unnecessary but an oral benzodiazepine or a small dose of IM opioid + atropine may be used.

Intra–operative management

- Induction (rapid sequence). Pre-oxygenation for four minutes, then give thiopentone followed by suxamethonium. Apply cricoid pressure. Intubate the patient using an armoured or PVC tube of the appropriate size. The cuff may have to be deflated temporarily to allow the scope to pass.
- Maintenance. Air or nitrous oxide, oxygen and volatile agent, or intermittent ketamine and oxygen may be used. Give suxamethonium (e.g. 25 mg IV) when the effect of the previous dose of suxamethonium has worn off. A non-depolarising relaxant may be used instead of intermittent suxamethonium.
- Reversal. Reverse the anaesthetic with atropine and neostigmine if a non-depolarising relaxant has been used.

Post–operative management

Perform extubation when the patient is fully awake and breathing adequately, with pharyngeal suction both before and after. Nurse the patient in the lateral position post-operatively.

CHAPTER 25

ANAESTHESIA for DENTAL SURGERY

Outline:

- Local anaesthesia
  - Dental blocks
  - Toxic side effects and treatment
- General anaesthesia
  - Indications
  - Potential anaesthetic problems
  - Technique for general anaesthesia in dental surgery
- Conscious sedation (or IV sedation)
  - Potential airway problems
  - Guidelines for using IV sedation in dental surgery
  - Equipment and drugs for conscious sedation
  - Technique of IV sedation
  - Complications of IV sedation and how to treat them
- Inhalational or relative analgesia

NB: Dental procedures should be carried out under Local Anaesthesia if possible.

LOCAL ANAESTHESIA

DENTAL BLOCKS

Nerve supply of the mandible

The inferior alveolar nerve (IAN or mandibular nerve) innervates the teeth and the mandibular bone from the molar area to the mandibular midline. The lingual nerve supplies the lingual gingiva in the molar region. The buccal nerve penetrates the buccinator muscle and innervates the buccal gingiva in the molar area. The mandibular (IAN), lingual and buccal nerves therefore must be blocked for dental work in the molar area of the lower jaw. The mandibular and lingual nerves can be blocked by one injection. The buccal nerve requires a separate injection.

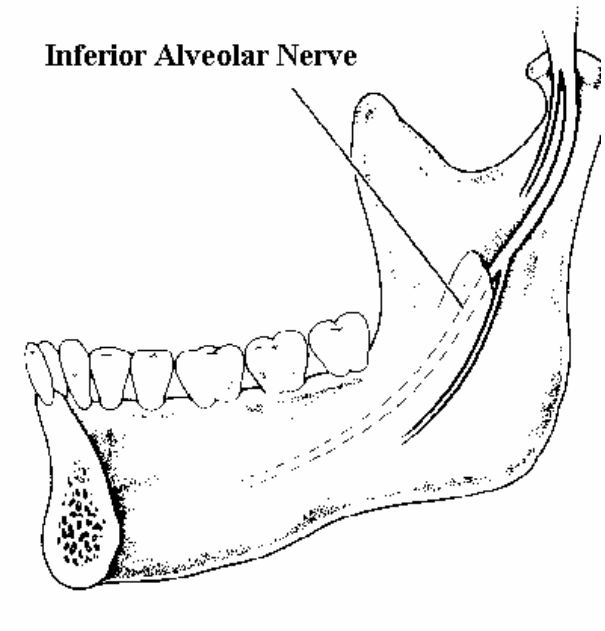


Fig 25.1 Anatomy of the inferior alveolar nerve

Method for mandibular block.

The patient's mouth is **widely** opened to make sure the landmarks are clearly visible.

- Palpate the coronoid notch with the left index finger.
- Find the injection site, just medial to the left index finger, lateral to the pterygomandibular raphe and 1cm above the occlusal plane of the molars.
- Direct the needle and syringe from the premolar region of the opposite side.
- After you insert the needle, ask the patient to reduce the degree of mouth opening. This reduces the tension in the pterygomandibular raphe.
- Advance the needle dorsally along the medial side of the ramus. During this procedure the needle must always be in contact with the bone of the ramus and held horizontally relative to the occlusal plane. Insert the needle 1.5 to 2 cm. Inject 2.0 ml of solution.

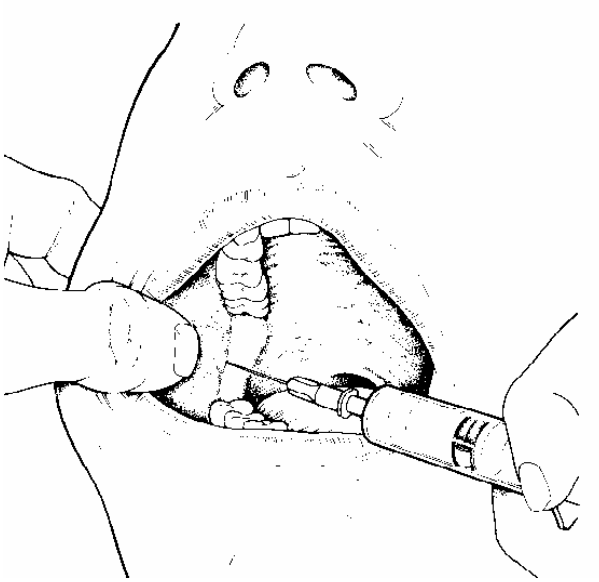


Fig 25.2 Blocking the inferior alveolar nerve

Method for lingual block.

- Block the lingual nerve by injecting 0.5 ml of solution at the mandibular temporal crest.
- Make the injection in conjunction with the mandibular block, while withdrawing the needle.

Method for the buccal nerve block

- The buccal nerve can be anaesthetised by infiltration. Puncture the mucosa just above the buccal fold, near the third molar and then guide the needle horizontally under the mucosa in a distal direction towards the mandibular ramus.
- Inject 0.5 ml.

MAXIMUM DOSES OF LOCAL ANAESTHETICS

Lignocaine with adrenaline 7mg/kg
Lignocaine alone 3mg/kg
Bupivacaine 2mg/kg

TOXIC SIDE EFFECTS OF LOCAL ANAESTHETIC DRUGS (see also Chapter 17)

Causes

- Inadvertent IV injection
- Exceeding the safe dose
- Rapid absorption because of increased vascularity
- Increased patient sensitivity
- Reaction to vasoconstrictor in local anaesthetic solution

Signs, symptoms and treatment

Central nervous system: Stimulation followed by depression. During stimulation the patient is talkative and restless, with tremor or convulsions. During depression the patient is unconscious.

Treatment

- For convulsions give oxygen by mask, then diazepam (0.1mg/kg) 5mg IV. Repeat if necessary (adult) 2.5mg IV. Place the patient in the left lateral position.
- For coma give oxygen by mask. Intubate (suxamethonium 1.5mg/kg) if there is a risk of aspiration or an inadequate airway with mask ventilation. Watch airway, respiration, CVS, BP, pulse.

Cardiovascular system: Bradycardia, hypotension

Treatment

- For bradycardia give atropine 0.6mg IV (dose 10micrograms/kg)
- For falling blood pressure give IV fluids, 10ml/kg.
- Give vasopressors, e.g. ephedrine (5-10mg IV for adults) or metaraminol 0.5mg IV.
- Give oxygen.

If anaphylaxis is suspected see Chapter 62

Respiratory system*:* Depression, apnoea

Treatment

- For respiratory depression with hypoventilation, give oxygen and assist breathing
- Apnoea. Give oxygen and IPPV

Reaction to vasoconstrictor in local anaesthetic: Pallor, tachycardia, palpitations, hypertension.
These signs and symptoms are usually transient

GENERAL ANAESTHESIA

Indications

A ***nasal mask*** or a ***laryngeal mask*** is commonly used for short procedures e.g. extractions especially in children. A gag and mouth pack should be inserted by the dentist.
For longer procedures ***endotracheal intubation*** and ***ventilation*** are usually required but if reinforced laryngeal masks are available and the anaesthetist is experienced some procedures can be performed with a spontaneously breathing technique.

Potential anaesthetic problems

Airway contamination. The airway is always in danger of being contaminated, by irrigation fluid, secretions, dental material, teeth, blood etc. Protect it by:

- Using an endotracheal tube, either nasal or oral
- Using a pharyngeal pack
- Encouraging reflexes to return rapidly post-operatively
- Placing the patient in the tonsillar position post-operatively
- Not using local anaesthesia in the respiratory tract
- Not using excessive premedication.

Airway obstruction. Watch for this constantly.

Shared airway. The anaesthetist and the surgeon share the airway.

The nasal tube needs careful attention

- Choose a soft tube, one size smaller in interior diameter than for oral intubation
- Measure the tube according to the chart, or at least 2 to 2½ times the distance between nostril and tragus. Fit the connector tightly into the endotracheal tube.
- Lubricate the tube well.
- Have an appropriate oral tube available in case it is needed.
- Have Magill forceps available.
- Never use force when passing a nasal tube.
- Be aware of the dangers of nasal tubes:
  trauma to nose, epistaxis and spread of infection to the lower respiratory tract.

***Contraindications to nasal intubation*:** purulent secretions and nasal obstruction.
Remember that a nasal tube is helpful but not essential. In most cases an oral tube is adequate.

The pharyngeal pack needs absolutely meticulous attention. It can easily cause a sore throat but that is minor. **Forgetting to remove the pack can be FATAL**. The pack should be included in the swab count. Both the surgeon and the anaesthetist must take responsibility for the situation.

TECHNIQUE FOR GENERAL ANAESTHESIA IN DENTAL SURGERY (endotracheal intubation)

Pre-operative management

- The patient should be fasted in accordance with guidelines for all general anaesthetics. (See Chapter 7 p 93)
- Assess the patient – take a history, examine as for other general anaesthetics, observe nostrils for purulent discharge or deviated septum. Check for history of epistaxis and if so from which nostril.
- Record the patient's weight.
- Give premedication: a benzodiazepine or small dose of opiate and atropine but avoid heavy sedation.

Intra-operative management

Check with the surgeon if a nasal tube is required

Check the equipment as for routine general anaesthetic, making sure you have:

- A nasal tube of the right size and length. Make sure the connector fits tightly (if the connector is dislodged, a plain ETT can be carried into a bronchus). Make sure the tube is well lubricated. If the patient is over 10 years old the tube must have a cuff, for younger children use an uncuffed one. Check the tube type: a PVC tube is acceptable but a RAE or preformed tube is the best. Have a smaller size tube available in case it is needed.
- An oral tube of the right size.
- Magill forceps.
- A pharyngeal pack, moistened with water and wrung out. Never use multiple swabs knotted together.

Induction.

- Pre-oxygenate.
- Give IV thiopentone 4 - 5 mg/kg **or** ketamine 2mg/kg **or** inhalational induction with oxygen and volatile.

Intubation. Give suxamethonium 1mg/kg IV. For the first minute ventilate with 100% oxygen. In the second minute, intubate as follows:

Step 1 Put the lubricated tube into the patient's right nostril. Pass it vertically downwards along the floor of the nasal cavity (towards the floor of the room). Don't force the tube in. If it sticks in the right nostril, try the left.

Step 2 Once the tube has been passed through the nose into the pharynx, do a routine laryngoscopy and observe the cords. The distal end of the tube should be visible in the pharynx.

Step 3 Lift the distal end of the tube with the Magill forceps and put it into the larynx between the vocal cords. The assistant applies gentle pressure on the proximal (top) end of the tube.

A further possible problem: The tube goes into the larynx between the vocal cords but gets stuck on the anterior wall of the larynx. If this happens, withdraw the tube slightly. Flex the head and advance the tube again. Pressure on the front of the larynx may also help. If nasal intubation is unsuccessful and the patient is still paralysed, ventilate with oxygen and try again. If still unsuccessful in the next 30 seconds, ventilate the patient again and intubate orally.

If an oral tube is used, position it on one side of the mouth. Half way through the operation remove the pack, change the tube to the other side and put in a fresh pack.

Maintenance. Give a ketamine infusion and muscle relaxant or oxygen with volatile and muscle relaxant and monitor closely, all as for a routine general anaesthetic.

Reversal. Give atropine or glycopyrrolate and neostigmine and extubate when the patient wakes, as in a routine general anaesthetic**. Remove pack.**

Post-operative management
Nurse the patient in the lateral position. Discharge the patient after 6 hours. Give the patient written postoperative instructions.

CONSCIOUS SEDATION (OR IV SEDATION)

This is suitable for conservative dental treatment especially in patients who are unwilling or unable to tolerate local anaesthesia.

Dangers of IV sedation in dentistry

Airway contamination. Contamination may be by blood, irrigation fluids, secretions or dental material. Since it is essential to keep the cough reflex active, it is essential to suction the pharynx meticulously. The whole team - surgical and anaesthetic - must be conscious of this problem.

Airway obstruction. The tongue may fall back and obstruct the airway after excessive sedation. There may be foreign matter in the pharynx, e.g. packs, fluid, secretions and dental material.

Hypoventilation, hypotension and ***loss of consciousness*** are all side effects of the drugs used.

Guidelines for using IV sedation in dental surgery

Patient selection. Intravenous sedation is **not ideal for small children** and should be restricted to those above 8 years.
Patients should be ASA 1 or 2. IV sedation is not recommended for patients with medical conditions such as asthma, hypertension, heart disease and sickle cell anaemia.

Pre-anaesthetic assessment is essential. It should be done before surgery, by the anaesthetist who will be giving the sedation. The aims are:

- To assess the patient's fitness for IV sedation, as for a general anaesthetic. The assessment will include history, examination and investigation if necessary
- To establish rapport with the patient (especially important with children)
- To explain the procedure to the patient
- To order any necessary pre-operative medication
- To give instructions about fasting to the patient (or the parents of a child patient). The patient must be fasted as per guidelines for GA surgery. (See Chapter 7 page 92)

Consent. This should be obtained before surgery, according to the rules of the hospital.

Duration. Set a time limit for the surgery. Intravenous sedation should be limited to 2 hours per session.

Weight. Record the patient's weight.

Premedication. It is not normal in dental surgery to give premedication but if a child is unusually anxious, the anaesthetist may arrange for premedication.

Position. The horizontal position is best for a patient having IV sedation. A sitting position aggravates any fall in blood pressure.

Local anaesthetic. The surgeon is recommended to give a local anaesthetic whenever possible. The IV sedation prepares the patient for the surgeon's local anaesthetic, which then provides the necessary analgesia.

Suction. Attend very carefully to the suction of the pharynx.

Packs. You will probably not need pharyngeal packs but if they are needed they must be positioned carefully, to absorb fluids but not to obstruct the airway.

IV access. An IV cannula must always be in situ.

Drugs. A single drug, e.g. midazolam (or diazepam) may be used, or a combination of midazolam with a narcotic such as fentanyl or midazolam with ketamine.

Monitor the following at least every 5 minutes*:*

- Pulse
- Blood pressure
- Consciousness level
- Ability to respond to verbal commands
- Colour
- Respiration
- Arterial oxygen saturation, using an oximeter.

Assistance. The surgeon should be willing to stop surgery and help the anaesthetist if any complication arises.

Discharge arrangements (to be discussed before sedation):

- The patient must be accompanied by a responsible person.
- Instruct the patient clearly **before sedation** not to drive a car or operate machinery for 24 hours and not to walk across a road unaccompanied.
- Tell parents to supervise children for 24 hours and warn them that the amnesic effects of the drugs will make it easy for their children to forget instructions.
- Keep the patient in the surgery room for at least 45 minutes after the operation, longer if the anaesthetist considers it necessary.
- Before discharge the patient should be fully conscious: the vital signs should all be in the normal range. The patient should be able to walk in a straight line and stand still with feet together and eyes closed.
- Transport home should be in a motor vehicle (if possible).

Equipment and drugs for conscious sedation in dental surgery

- Ventilating equipment: Self-inflating bag. The adult size can also be used for children weighing more than 15kg. For smaller children use a Paedivalve (non-rebreathing) and paediatric bellows with the Oxford Inflating Bellows.
- Oxygen source with attachments
- Oxygen catheter
- Airway (appropriate size for patient)
- Mask (appropriate size for patient)
- Endotracheal tubes (appropriate size for patient)
- Laryngoscopes (2)
- Suction machine and pharyngeal packs
- Yankauer suction head
- Tape
- Syringes: 3 each of 10ml, 5ml and 2ml
- Needles: 21G
- Scalp vein needles
- IV fluids, IV sets, cannulae. Make sure the blood pressure cuff is the right size.
- Monitoring equipment: Stethoscope
Oximeter
Sphygmomanometer
- Drugs for sedation: Midazolam or diazepam
Fentanyl if available
Ketamine

- Drugs for resuscitation: Atropine
  Adrenaline 1/1000
  Calcium gluconate
  Sodium bicarbonate
  Hydrocortisone
  Vasopressors: Ephedrine
  Metaraminol (Aramine)
  Thiopentone
  Suxamethonium
  Aminophylline
  GTN tablets or spray
  Water and 0.9% saline

Technique of IV sedation

- Check that all equipment is available, according to the above list.
- Check that the patient has fasted.
- Check that consent has been given.
- Draw up the drugs required. Label the syringes. Work out the doses for the patient. Use 0.9% saline for flushing the IV tubing.
- Place the patient in the dental chair in the horizontal position.
- Check blood pressure.
- Insert IV cannula
- **Slowly** inject the drug. If it is midazolam, inject over 2 minutes. Wait 2 minutes and watch for response. Give a further 0.1mg (1ml) at a time as necessary. Wait 2 minutes. Note slowing or slurring of speech and stop at this point.
- Give ketamine IV, 0.25mg/kg **slowly**.
- Monitor pulse, blood pressure, respiration, colour, conscious level and oxygen saturation.
- Ask the patient whether they are relaxed enough to let the surgeon look into their mouth.
- Proceed with local anaesthetic.

Suitable drugs and dosages			
	Preparation	**Initial dose**	**Maintenance**
Midazolam	In a 10ml syringe, draw up 1mg (1ml) midazolam and 9ml water. (Each ml thus contains 0.1mg midazolam). Prepare 2 such syringes for an average sized patient.	0.02mg/kg, so a 40kg patient will need 0.02x40 = 0.8mg = 8ml. Inject this slowly over 2 minutes, then wait 2 minutes.	Give further doses 1ml at a time.
Diazepam	In a 10ml syringe draw up 5mg diazepam and 9ml dextrose 5% or N saline. (Each ml thus contains 0.5mg diazepam).	0.04mg/kg, so a 40kg patient will need 0.04x40 = 1.6mg drug in 3.2ml diluting solution.	Give IV increments of 1 ml slowly.
Ketamine	In a 5ml syringe draw up 25mg (0.5ml) ketamine, plus 4.5ml water (25mg in 5ml)	0.25mg/kg, so a 40kg patient will need 0.25 x 40 = 10mg. This is a very small dose of ketamine, only intended to produce analgesia.	

Note: The above doses are very safe although careful vigilance is still necessary.

Complications of IV sedation and how to treat them

Respiratory complications

Airway obstruction caused by the tongue falling backward, packs, fluid in the pharynx and laryngeal spasm.
Treatment:

- Stop surgery.
- Treat the cause: suction the pharynx, support the jaw, extend the head, use a pharyngeal airway.
- Give oxygen by mask.
- If obstruction persists, perform endotracheal intubation (oral) with or without using suxamethonium 1mg/kg. Ventilate with oxygen.

Hypoventilation caused by shallow respiratory excursions, or the oxygen saturation being below 90%
Treatment:

- Encourage the patient to breathe more deeply.
- Give oxygen by nasal catheter 4L/min.
- If oxygen saturation is still below 90%, stop surgery. Help breathing with a self-inflating bag and mask, with oxygen entrained at 4L/min.

Apnoea
Treatment:

- Stop surgery.
- Insert a Guedel airway.
- Control ventilation with a self-inflating bag, mask and oxygen.
- If spontaneous respiration does not start, intubate and ventilate with oxygen.

Cardiovascular complications

Hypotension
Treatment:

- Give IV fluid quickly: 0.9% saline 500ml.
- If there is no response, for example, if blood pressure is less than 80mmHg in an adult, give vasopressors (e.g. ephedrine 5mg IV or metaraminol 0.5mg IV).

Bradycardia
Treatment:

- If the pulse rate is less than 50/min, give atropine 10 microgram/kg IV.

Cardiac arrest See Chapter 58 for information on cardio-pulmonary resuscitation.

Central nervous system complications
If the patient is unconscious:

- Stop surgery.
- Keep the airway clear.
- Administer oxygen by nasal catheter, 4L/min.
- Watch for respiratory complications such as hypoventilation and apnoea.
- Watch for cardiovascular complications such as hypotension.

INHALATIONAL OR RELATIVE ANALGESIA
This technique involves the use of a nitrous oxide / oxygen mixture at less than general anaesthetic concentrations i.e. < 50% nitrous oxide in oxygen. This can be used in conjunction with local anaesthesia in situations where nitrous oxide and oxygen are freely available.

CHAPTER 26

ANAESTHESIA FOR THYROID SURGERY

Outline:

Physiology of the thyroid gland

Indications for thyroidectomy

Potential anaesthetic problems associated with thyroid surgery
- Problems common to all head and neck surgery
- Problems associated with an enlarged thyroid gland
- Problems associated with a toxic gland
- Problems associated with a malignant gland

Anaesthetic management
- Local anaesthesia
- General anaesthesia

Thyroid crisis

Hypothyroidism and anaesthesia

PHYSIOLOGY OF THE THYROID GLAND

The thyroid gland normally secretes two hormones:
T4 (tetra-iodothyronine) and T3 (tri iodothyronine).

T3, the active hormone is only secreted in small quantities. T3 and T4 are synthesised in the thyroid gland from iodine and amino acids and stored in the gland as iodothyroglobulin. T3 is also synthesised in peripheral tissues such as liver and kidneys. When released from the thyroid gland they are carried in the blood stream and act on the heart and other organs.
The formation and release of T3 and T4 are under the control of the thyroid-stimulating hormone (TSH) released from the pituitary gland. When the level of circulating hormones T3 and T4 is low the pituitary is stimulated to put out more TSH and vice versa.
The hypothalamus also has a part to play in the production of the thyroid hormones. It secretes a thyrotropin releasing factor which acts on the pituitary and stimulates the output of TSH.

Tests of thyroid function commonly available are:

T3
T4
Serum TSH
Radioactive iodine uptake
Thyroid scan.

INDICATIONS FOR THYROIDECTOMY

- Toxicity, which must be controlled before surgery.
- Malignancy or potential malignancy.
- Cosmetic reasons
- Associated mechanical problems
  - Respiratory obstruction may follow vocal cord involvement or compression of trachea.
  - Retrosternal extension.

POTENTIAL ANAESTHETIC PROBLEMS ASSOCIATED WITH THYROID SURGERY

Problems common to all head and neck surgery

- Bleeding, which must be reduced to a minimum: take the following measures:
  - Prevent coughing and straining
  - Keep the airway clear
  - Use the largest suitable endotracheal tube
  - Avoid hypoxia or hypercarbia
  - Use a slight head-up tilt of the table. (Note: there is the possibility of air embolus).
- The inaccessibility of the airway. Secure the connectors (ETT), check the position of the tube carefully and secure it with tape.
- Respiratory obstruction is always possible, because of the underlying pathology and also because the surgical site is so close to the endotracheal tube. An armoured tube if available should be used. Care should be taken if using preformed tubes for thyroidectomies because the antero-posterior direction of the bevel may be obstructed when the head is flexed (at the end of surgery).

Problems associated with an enlarged thyroid gland

These are caused by the enlarged gland compressing the trachea, involving the recurrent laryngeal nerves or displacing the structures of the neck.

- If there is respiratory obstruction special techniques should be used for induction. If there is pre-operative obstruction, intubate under local anaesthetic spray and follow with a general anaesthetic. Occasionally a tracheostomy is required before a general anaesthetic is given.
- If there is no pre-operative obstruction but airway obstruction is possible after induction, then induce and intubate the patient with an inhalational anaesthetic. (Never paralyse a patient with a relaxant unless you are absolutely sure the patient can be ventilated with a mask if intubation is not possible).

Problems associated with a toxic gland

- Thyrotoxicosis or hyperthyroidism affects the cardiovascular system. The patient may have suffered from tachycardia, atrial fibrillation or cardiac failure, so the thyroid function must be controlled with drugs so that it is normal before surgery. This may take 2-3 months of treatment. Some of the drugs used are propyl thiouracil, carbimazole and propranolol (Inderal). Iodine preparation given 10 days before surgery reduces the vascularity of the gland. Occasionally, however, a thyrotoxic patient presents for emergency surgery (e.g. appendectomy). Propranolol 10–40 mg orally can be given in this situation for tachycardia. Choose a regional anaesthetic if suitable and combine it with sedation, since most hyperthyroid patients are restless and apprehensive.
- Exophthalmos may be a problem in a patient who has previously suffered from hyperthyroidism. The eyes require special protection during surgery.
- As mentioned before, thyrotoxic patients are very emotionally labile and may need benzodiazepine or opiate premedication.

Problems associated with a malignant gland

- Involvement of the nerves supplying the vocal cords
- Collapse of the trachea due to erosion into the tracheal cartilage
- The patient may have secondaries involving other organs
- The patient may be anaemic and in poor physical condition.

Fig 26.1 Patient with enlarged thyroid gland

Fig 26.2 Lateral view

ANAESTHETIC MANAGEMENT

Local anaesthesia

Thyroidectomies can be done under local analgesia. The disadvantages are:

- Patient discomfort
- The associated dangers of respiratory obstruction and pneumothorax, which can be dangerous in a conscious patient with no control of the airway.
- Surgical difficulty. It is difficult to identify properly the superior and inferior thyroid vessels and the recurrent laryngeal nerve.

General anaesthesia

Pre-operative management

It is important that the indication for thyroid surgery is clear in the anaesthetist's mind.

Assessment. The surgeon will see that a toxic thyroid is adequately treated and that Lugol's iodine solution is given pre-operatively to reduce the vascularity of the gland.

Always check for ***signs of airway obstruction:***

- History of dyspnoea, hoarseness
- Clinical signs of airway obstruction, e.g. stridor, intercostal muscle recession
- Deviation of the trachea, shown by x-ray of the neck and thoracic inlet
- Substernal extension, shown by chest x-ray
- Possible involvement of the cords. An indirect laryngoscopy (if it is possible to have one) will confirm this.

Premedication

Do not give premedication if there are signs of airway obstruction.

For other patients benzodiazepines or opioids +/– atropine in the usual dose can be given. The patient should not be presented for surgery if there is evidence of a resting tachycardia, so there is no need to reduce the dose of atropine. Treated thyrotoxic patients may need bigger doses of premedication.

Intra-operative management

Induction

- If there is definite pre-operative evidence of respiratory obstruction, intubate under local anaesthesia. If this is not possible, a tracheostomy will be needed.
- If there is no pre-operative evidence of respiratory obstruction but there is a chance the airway may become obstructed when the patient is unconscious, there are two alternatives:
  - Inhalational induction then intubate when the patient is deep enough under the inhalational anaesthetic, **OR**
  - Inhalational induction with ether or halothane. Then assist the patient's respiration and make sure ventilation is possible. If ventilation is possible paralyse the patient with suxamethonium and intubate.
- If the thyroid gland is small and there is no possibility that it is causing or will cause respiratory obstruction, then pre-oxygenate for 4 minutes and use a standard induction with thiopentone or propofol followed by a relaxant agent.

Intubation

- Use a standard dose of suxamethonium.
- Spray the cords with lignocaine. If you are using the usual pack of 10% Xylocaine remember that each 'puff' gives 10 mg. The toxic dose of 200 mg must not be exceeded. This prevents the patient straining on the tube and reduces congestion and bleeding.
- It is best to use an armoured endotracheal tube. If this is not available use a PVC tube.

Maintenance

- Air or nitrous oxide/oxygen/volatile/ relaxant or ether/air/oxygen/relaxant. There are no absolute contraindications but if alternatives are available avoid ether, ketamine and pancuronium as they all stimulate the sympathetic system
- IPPV: slight hyperventilation is recommended. This reduces the CO_2 level and reduces bleeding.
- Keep IV fluids running with a large cannula.
- A slight head-up tilt is helpful but note the possibility of air embolus. Use a footrest to make sure the patient does not slide off the table.
- An extension on the IV infusion set will avoid the necessity for an armboard.
- Protect the patient's eyes.
- Don't over-extend the head. (This leads to occipital headache post-operatively).
- Monitor all parameters very carefully.

Reversal. Use routine measures. Observe the movement of the cords if possible.

Post-operative management

Routine observation. Attend promptly to the earliest sign of respiratory difficulty.

Complications

- Haemorrhage, which may be due to venous or arterial bleeding. It results in pressure on the trachea, leading to respiratory obstruction, blood loss and shock.
  Treatment. Immediate evacuation of the haematoma. The stitches must be removed to allow the blood to escape. Replace blood loss.
- Respiratory obstruction caused by:
  - Haemorrhage (see above). In this situation collapse of the trachea which may require re-intubation or tracheostomy
  - Recurrent laryngeal nerve damage. This may be due to: *temporary bruising* which will heal within 2-4 weeks or *section of the nerve* (permanent)
  - If there is severe respiratory obstruction, re-intubation will be necessary.
  - Laryngeal oedema, which may occur in the first three post-operative days. If it is severe and stridor is present, intubation may be required.
- Tracheitis
- Thyroid storm (see below)
- Hypothyroidism (see below)

- Hypoparathyroidism (1-7 days). This may cause a transient hypocalcaemia (tetany) - treat initially with 10% calcium gluconate 20ml slowly IV.

THYROID CRISIS (THYROID STORM)

This occurs when a thyroidectomy has been performed on a patient whose thyrotoxic condition has not been controlled. Symptoms appear intra-operatively or in the early post-operative period. It is associated with an excessive release of thyroid hormone into the circulation and it is characterised by:

- CNS: delirium, restlessness, mania, stupor, coma.
- CVS: tachycardia, atrial fibrillation, heart failure.
- GIT: nausea, vomiting, diarrhoea.
- Dehydration: hyperpyrexia.

The mortality rate is high.

Treatment

Close attention to airway, breathing and circulation is necessary – if possible in a recovery area or high dependency ward:

- Cool the patient by means of sponging, fans etc.
- Give IV fluids
- Give antithyroid drugs, e.g. propylthiouracil 200mg orally or by NG tube, 6 hourly followed after 1 hour by sodium iodide 250mg IV or Lugol's iodine solution orally.
- Give beta-blocker e.g. propranolol (Inderal) for tachycardia. This is given IV 0.25-0.5mg in repeated increments (max 5mg).
- Give steroids - e.g. hydrocortisone 100mg 6 hourly.
- Sedation.

HYPOTHYROIDISM AND ANAESTHESIA

Hypothyroidism must be controlled before elective surgery.

Points of importance

Hypothyroidism leads to:

- Greater sensitivity to drugs such as premedicant drugs and drugs used intra-operatively.
- A lower cardiac output and reduced myocardial reserve. Blood loss and shock are poorly tolerated. Cardiac failure can ensue quite quickly.
- Respiratory depression can easily occur.

Also associated with hypothyroidism are:

- Hypothermia
- Hypoglycaemia
- Muscular weakness.

CHAPTER 27

ANAESTHESIA FOR CHEST SURGERY
(Thoracic Anaesthesia)

Outline: (This topic will be considered only briefly)

Introduction

Problems associated with chest surgery:
- The open chest
- Secretions
- Air leak
- General state of the patient
- Blood loss
- Cardiac problems
- Atelectasis

(Only the first two of these will be dealt with in the text following. For more detail consult a specialist text)

Anaesthetic management of a patient for chest surgery

The underwater drain

INTRODUCTION

Chest surgery may be elective or emergency surgery and may involve operations on the following structures:

- Chest wall: for instance for a tumour, or repair of chest wall injury
- Lungs, e.g. Lobectomy (removal of a lobe)
  Pneumonectomy (removal of a lung)
  Closure of a bronchopleural fistula (i.e. of a communication between the bronchus and the pleural cavity)
- Heart + great vessels
- Oesophagus
- Diaphragmatic hernia
- Others eg pleurectomy, drainage of abscesses, decortication.

PROBLEMS ASSOCIATED WITH CHEST SURGERY

The problems of the open chest

Collapse of the lung. When the chest is opened the negative pressure in the pleural cavity is replaced by atmospheric pressure. This positive pressure collapses the lung. If the chest is opened and the patient breathes spontaneously, two other strange phenomena occur, paradoxical respiration and mediastinal flap.

Paradoxical respiration. Here, air passes from the collapsed lung into the healthy lung during inspiration. During expiration air passes out from the healthy lung, not into the atmosphere but back into the collapsed lung. This cycle is repeated. It leads to hypoxia and hypercarbia, if the patient continues to breathe spontaneously.

Mediastinal flap. The mediastinum is the space between the two lungs. It is centrally situated and contains the heart, great vessels, oesophagus etc. It is kept central by the equal pressures on the two sides of the chest. If the chest cavity is opened on one side, the negative pressure is replaced by a positive pressure. During inspiration the negative pressure on the healthy side increases and this negative pressure draws the mediastinum towards the healthy lung. During expiration the negative pressure (on the healthy side) decreases and this pushes the mediastinum away from it. The mediastinum therefore moves to and fro with each respiration. This is called mediastinal flap.

Mediastinal flap has two effects:

- The pressure of the mediastinum against the sound lung during inspiration interferes with the exchange of gases in this lung. The patient therefore becomes even more hypoxic and hypercarbic.
- This intermittent movement causes obstruction of the great vessels (inferior and superior vena cava) at the opening into the heart. The patient therefore becomes hypotensive.

Respiration is affected by collapse, paradoxical respiration and mediastinal flap.

Circulation is affected by mediastinal flap and also by the absence of negative pressure in the chest and the position of the patient.

For all forms of chest surgery, where the chest is opened, IPPV must be used. IPPV will distribute the gases equally on both sides of the chest. This will eliminate the problems of paradoxical respiration and mediastinal flap. Further, it will prevent the collapse of the lung on the side of the open chest.

Secretions

This is another major problem with chest surgery. The affected lung may contain pus, blood or secretions and these may contaminate the unaffected side. Various techniques are used to reduce the danger of contamination:

- Position
- Suction
- An endobronchial tube (thus anaesthetising only the healthy lung)
- A bronchial blocker
- A double lumen tube.

These techniques are simply mentioned. A textbook of anaesthesia should be consulted for more details.

ANAESTHETIC MANAGEMENT OF A PATIENT FOR CHEST SURGERY

Consultation with the surgeon is necessary in planning the anaesthetic and choice of tube

Pre-operative care

Assess the patient's general condition. Especially check the patient for secretions and for pneumothorax/bronchopleural fistula.
Premedication: Oral benzodiazepine or opioids + atropine or glycopyrrolate.

Intra-operative care

Induction: Use thiopentone, propofol or ketamine IV depending on the state of the patient, followed by suxamethonium and intubation.
Use an ordinary PVC cuffed ETT.
If a special tube (e.g. double lumen) is required, it must only be used by someone experienced.

Maintenance:
Ether/Air/O_2/opioid/non-depolarising muscle relaxant. (EMO)

OR

Air or N_2O/O_2/ volatile/opioid/non-depolarising relaxant.

Analgesia can be supplemented by intercostal nerve blocks performed by the surgeon under direct vision.
Good analgesia helps the patient cough up secretions post-operatively.

Reversal: If the chest is closed without a drain, inflate the collapsed lung before the last few stitches are inserted.

Post-operative care

If an underwater drain is inserted, it must be checked regularly, in addition to other post-operative observations.

THE UNDERWATER DRAIN

This has been devised to prevent air entering the pleural space, at the same time allowing free drainage of fluid and air (if the intrapleural pressure rises above atmospheric level).

Apparatus

A large wide-mouthed bottle (Winchester bottle) with a rubber stopper through which two glass tubes pass.

The upper end of the longer tube is connected to the drainage tube or catheter by rubber or plastic tubing and a glass connection. The lower end of the tube should be below the level of the sterile solution in the bottle.

The shorter tube is open to the atmosphere and its lower end projects just beyond the rubber stopper. It allows air to escape naturally, or it could be connected to a suction pump.

There are also disposable commercially produced units available for chest drainage (e.g. Thoraseal).

If a clear passage exists between the bottle and the pleural space, the column of liquid will swing.

If the pressure in the intrapleural space is below atmospheric pressure, a column of liquid will be drawn up the tube. This column will swing during respiration.

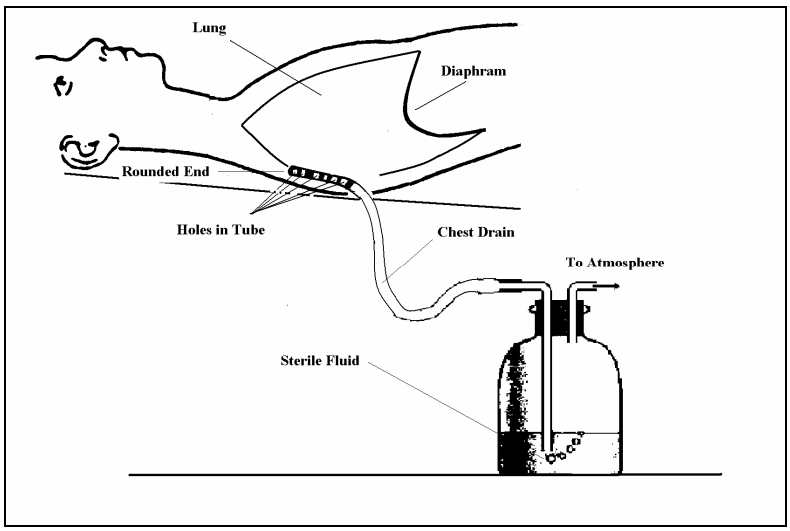


Fig 27.1 Underwater seal chest drainage system

If a **broncho-pleural fistula** exists the intrapleural pressure becomes positive at the end of each expiration and air will bubble through the end of the tube. If the patient coughs, a continuous stream of bubbles in the bottle suggests a fistula is present.

Obstruction of the drainage system

This is suggested by the absence of the respiratory swing in the tube.

Commonest sites of obstruction:

- In the dressings or bedding. These two sites of obstruction must be investigated first.
- In the tube from outside, by pressure from the chest wall, the tubing being too thin. In the lumen, a slough or a clot.
- At the end of the tube, debris causing occlusion against re-expanding lung.

Management of blocked tube:

- Check for the site of blockage.
- Milk the tube in either direction or readjust its position but be careful not to push it further into the chest as this may introduce infection.
- Irrigate with sterile saline.
- If these measures fail, remove the tube and replace it with a fresh one.

CHAPTER 28

ANAESTHESIA FOR ABDOMINAL SURGERY

Outline:

Points of importance associated with anaesthesia for abdominal surgery

Techniques for anaesthesia
- Regional
- General

Anaesthesia for Laparoscopy

POINTS OF IMPORTANCE ASSOCIATED WITH ANAESTHESIA FOR ABDOMINAL SURGERY

Good relaxation of the anterior abdominal wall muscles
The posterior sheath of the rectus and the transversus muscles are fused with the peritoneum in the upper abdomen. Hence when the peritoneum is sutured at the end of the operation the muscles need to be well relaxed. If the surgeon is unable to close the peritoneum due to inadequate muscle relaxation, a further dose of relaxant may be given.
It is, however, important to make sure that the problem is due to a lack of relaxant and not to some other factor, e.g. gastric distension. If the patient has not had any relaxant drugs for a period of 20 minutes and if there is no contraindication, then another small dose of relaxant may be given to assist the surgeon.

The maintenance of adequate ventilation
This is achieved by IPPV using a relaxant technique. However, ventilation may be impaired by the presence of packs and retractors in the upper abdomen.

The prevention of shock and cardiovascular depression
This may be caused by:

- Pre-operative hypovolaemia
- Intra-operative bleeding
- Loss of fluid from the gut, by prolonged exposure during surgery.
- Handling of the gut and traction on the mesentery.
- Compression of the inferior vena cava by packs, retractors and even abdominal tumours.

Cardiac arrhythmias
These may occur if there is excessive traction on the gut or mesentery.

Protection of the airway and prevention of aspiration.
This precaution is necessary in all types of surgery but especially in abdominal surgery. A naso-gastric tube must be used for aspiration of stomach contents if a full stomach is suspected. A rapid sequence induction is indicated if there is any danger at all of vomiting or regurgitation.

Hiccups
This may be encountered, especially in upper abdominal surgery. The topic is covered in Anaesthetic complications involving the Gastro-intestinal system in Chapter 46.

Respiratory complications
These are common in the post-operative period. Patients who have upper abdominal surgery are more prone to post-operative respiratory complications than others. Adequate ventilation during the operation and attention to adequate analgesia (e.g. opioids) to enable the patient to cough and take deep breaths post–operatively, is vital to prevent post–operative pneumonia. It is important to give opioids both during and after surgery. An appropriate regime would be:

- Intra–operatively boluses of up to 0.1–0.2 mg/kg of morphine IV.
- Post–operatively 0.1mg/kg IM 3 hourly.

Unless otherwise indicated, the patient is best nursed supported in a semi-sitting position in the post-operative period. Good preoperative and postoperative physiotherapy are very important in preventing chest complications. It cannot be stressed enough that adequate pain relief in the post-operative period is very important.

Deep vein thrombosis
This is more likely after lower abdominal and pelvic surgery. Prophylactic measures include:

- The use of calf stimulators
- Elastic stockings
- The peri–operative use of sub-cutaneous heparin preparations
- Avoidance of hypotension
- Early ambulation
- Physiotherapy in the post–operative period.

Post-operative nausea and vomiting
This is discussed under Anaesthetic complications in Chapter 46

TECHNIQUES FOR ANAESTHESIA

Regional techniques.

Spinal anaesthesia is usually the method of choice for surgery below the level of the umbilicus, provided there is no contraindication. The advantages of a spinal anaesthetic are detailed in Chapter 19.
Ensure adequate fluid replacement.

Local infiltration. If a general or spinal anaesthetic is contraindicated (e.g. in a moribund patient), then it may be necessary to resort to local infiltration by the surgeon.

General anaesthesia

The techniques of controlled ventilation are outlined in Chapters 14 and 16. A rapid sequence induction technique must be used for patients with a full stomach. For a full description of this technique see Chapter 16.

The depth of anaesthesia needs to be greatest during:

- The skin incision
- Initial abdominal exploration
- Closure of the peritoneum.

If the patient is very hypotensive or moribund, ventilate with oxygen and a very small dose of ketamine or N_2O to prevent awareness. At the earliest sign of the patient beginning to waken, increase these agents and/or give a small dose of opioid or halothane.

ANAESTHESIA FOR LAPAROSCOPY

Most anaesthetists favour general anaesthesia, although local and regional anaesthetics have been used. The following points should be noted:

- Endotracheal intubation (using a cuffed tube) is advisable:
  - For controlling ventilation. The insufflation of gas into the peritoneal cavity raises the diaphragm and interferes with breathing. It is important that the patient be paralysed and ventilated.
  - To prevent regurgitation of gastric contents. This is more likely with the high intra-abdominal pressure.

  In spite of these potential risks some anaesthetists will use a LMA and ventilation with a short acting muscle relaxant (eg mivacurium/ atracurium/ vecuronium) for elective surgery of short duration, (e.g. gynaecology) in fasted, non-obese patients.
- The introduction of gas into the peritoneal cavity can result in hypotension (secondary to inferior vena caval obstruction) and also cardiac arrhythmias. Carbon dioxide embolus can cause hypotension and cardiac arrest.

  It is important to limit intraperitoneal pressures to no greater than 15mmHg.

  Laparoscopy is often associated with bradycardia. Careful monitoring is essential. Atropine or glycopyrrolate is often required and should be readily available.
- The patient is placed in a steep Trendelenburg position (head down) which carries its own hazards. (See Chapter 30)

CHAPTER 29

ANAESTHESIA FOR UROLOGICAL SURGERY

Outline:

Types of surgical procedures

Points of importance to the anaesthetist

Anaesthesia for common urological procedures

TYPES OF SURGICAL PROCEDURES

Open surgery, e.g. surgery of the bladder, ureters and kidneys.

Endoscopic surgery

- Cystoscopy
- Trans-urethral resection of the prostate
- Resection of bladder lesions
- Urethral dilatations

POINTS OF IMPORTANCE TO THE ANAESTHETIST

Geriatric problems

Many patients presenting for urological procedures are older people. They therefore present with cardiovascular, respiratory and other problems common to their age. These problems are discussed under Anaesthesia for the Elderly in Chapter 36.

Renal failure

A proportion of patients will be in renal failure. The surgery performed will be for the purpose of correcting for example, obstructive lesions of the urinary tract. Some patients will present with hyperkalaemia which if > 6.5 needs to be corrected before surgery.

> ***A suggested regime would be:*** Insulin 15 units in 200ml 10% dextrose/water given over 30-60 mins. If only 5% dextrose/water is available add 20ml of 50% dextrose to 200ml of 5% dextrose/water + 15 units insulin and give over 30-60 mins.
>
> This may provide a temporary improvement and allow surgery to proceed.

The other problems encountered in patients with renal failure are discussed in Chapter 37.

Positioning the patient

Nephrectomy is performed with the patient in the lateral position, cystoscopies and TUR in the lithotomy position. These positions may affect the cardiovascular and respiratory systems. Special care is also needed to prevent injury to peripheral nerves.

Electrocautery

The use of electrocautery is a contraindication to the use of flammable anaesthetic agents.

Repeated anaesthetics

There can be a problem with repeated anaesthetics eg. with halothane, in patients who present for check cystoscopies for bladder tumours or urethral dilatations.

ANAESTHESIA FOR COMMONLY PERFORMED UROLOGICAL PROCEDURES

CYSTOSCOPY AND RETROGRADE PYELOGRAPHY

Local anaesthesia
1 or 2% Lignocaine jelly may be used but is contraindicated in cystitis or infection of the urethra, in the presence of trauma to the urethra or bleeding from the urethra.

Ketamine/diazepam technique
This has been discussed under "Anaesthetic Techniques" in Chapter 14. The patient must be medically fit for a general anaesthetic. Have an oxygen source, equipment for ventilating the patient and suction available. Record the blood pressure and leave the cuff in place. Insert an IV cannula.
Place the patient in the lithotomy position. Swab the operative field. Give ketamine 2 mg/kg IV (or 5-7 mg/kg IM) and diazepam 0.1 mg/kg IV. The patient may need supplementary doses every 20 minutes.

General anaesthesia using spontaneous ventilation
N_2O/oxygen and volatile would be suitable.

Regional anaesthesia
A spinal may be indicated in patients who are unfit for any of the above techniques. A spinal anaesthetic is not routinely justified because the procedure is such a short one.

CIRCUMCISION

This can be performed under

- Local infiltration (avoid vasoconstrictors in the local anaesthetic)
- Ketamine/diazepam
- General anaesthesia with spontaneous ventilation
- Regional techniques e.g. spinal
- Penile block

TRANS URETHRAL RESECTION OF THE PROSTATE

Spinal (or epidural block)

This is the recommended technique for the following reasons.

- Spinal anaesthesia is especially suitable for older patients, who may have medical problems.
- It provides good operating conditions with analgesia and relaxation.
- Bleeding is significantly reduced. This is helped by a head down tilt.
- Intra-operative complications are more easily detected if the patient is awake, e.g. pain may signify accidental perforation of the bladder or prostatic capsule. Restlessness may signify absorption of irrigating fluid into the circulation. It may also indicate cardiovascular overload.
- It reduces post-operative problems such as nausea, vomiting, dry retching or coughing. All these complications can increase bleeding.
- It reduces the risk of explosion.

Spinal anaesthesia should reach to the level of T10.

General anaesthesia

The use of the EMO with ether is not recommended for trans-urethral resection of the prostate gland because of the use of diathermy and the need for oxygen in this group of elderly patients. Special care must be taken with electrical equipment if this technique is used.

More suitable techniques would be:

- Spontaneous respiration with nitrous oxide, oxygen and volatile other than ether. This is suitable in the fit patient who is not obese.
- Controlled ventilation using nitrous oxide, oxygen, relaxant, supplemented with volatile, fentanyl, or pethidine **or** relaxant technique with ketamine infusion is useful in the less fit, the obese or bullnecked patient.

IV fluids for the patient should be 0.9% saline or Hartmann's.

Bladder irrigation fluid: 1.5% glycine is the fluid of choice for TURP intra-operative irrigation. Dextrose should not be used as the TURP syndrome is caused in part by hyponatremia (see below). 0.9% saline is used for irrigation post–operatively for 24 hours.

Intraoperative Complications

- Blood loss. This is difficult to assess because it is mixed with irrigation fluid and increases with operating time. It increases further if plasminogen activators are released from the prostate, causing fibrinolysis and platelet consumption. Hyperfibrinolysis and disseminated intravascular coagulation are discussed in more advanced textbooks.
- Bladder perforation. A conscious patient complains of abdominal discomfort and shoulder pain.
- **TURP Syndrome**
  This is due to reabsorption of large volumes of water from the prostatic bed. Reabsorption is normally at a rate of 1litre/hour and can be as great as 5 litres in total. This causes water intoxication leading to hyponatraemia, cerebral and pulmonary oedema, dyspnoea, hypoxia, confusion, cardiovascular collapse and death.
  Treatment:
  - Stop surgery
  - Restrict fluids
  - Supportive measures for respiratory and cardiovascular problems
  - Slow correction of hyponatraemia with isotonic saline (occasionally hypertonic saline may be required).

Surgery should be restricted to 1 hour as the incidence of TURP syndrome, hypothermia and heavy blood loss increases with duration of surgery.

SUPRAPUBIC PROSTATECTOMY

Relaxation of abdominal muscles is required. Considerable bleeding may occur. Suitable techniques would be:

- Spinal anaesthesia
- General anaesthesia with controlled ventilation, e.g. air, nitrous oxide, oxygen, volatile, relaxant and supplement with opioid.

NEPHRECTOMY

The principles outlined for abdominal surgery should be followed.

Additional problems

- The patient is placed in the lateral jack knife position. This is associated with hypoventilation, post-operative atelectasis and if the patient is in the right lateral position, obstruction of the inferior vena cava.
- Pneumothorax may occur as a result of technical difficulties. This is managed with an underwater drain.
- Thrombosis in the dependent extremity due to obstruction of the venous flow. This is prevented by flexing the operating table only moderately, using elastic bandages and encouraging early ambulation.
- Bleeding from the renal pedicle may be excessive.

Anaesthetic technique

Spinal anaesthesia is unsuitable.
General anaesthesia is necessary, with controlled ventilation using nitrous oxide, oxygen, volatile, opioid (e.g. morphine or pethidine) and a muscle relaxant.

UROLOGICAL SURGERY IN PATIENTS WITH SPINAL CORD INJURIES

Patients with spinal cord injuries often lose bladder control, so repeated cystoscopies may be required.

Autonomic imbalance

Paraplegics with spinal cord injuries at or above the level of the 5th thoracic vertebra may respond to distension of the bladder by irrigating fluid with hypertension and bradycardia. This may be encountered in cystoscopies and trans-urethral resection of the prostate. Occasionally hypotension may occur after induction of anaesthesia. A 16G cannula should be inserted before the anaesthetic starts. An IV infusion of e.g. Hartmann's solution should be commenced. Blood pressure should be monitored carefully, noting rises or falls. A rise in blood pressure may require an antihypertensive agent. Spinal anaesthesia reduces the complication of hypertension.

CHAPTER 30

ANAESTHESIA FOR GYNAECOLOGICAL SURGERY

Outline:

Abdominal gynaecological surgery

Vaginal surgery

Anaesthetic techniques

Special gynaecological procedures

ABDOMINAL SURGERY

This has been discussed in Chapter 28.
Most gynaecological abdominal surgery can be done under spinal anaesthesia.

VAGINAL SURGERY

Problems

Position: The Trendelenburg or head down position can cause:

- Impairment of respiration.
- Sudden increase in venous return, which may send the patient into cardiac failure if pre-operative cardiac disease exists.
- Headache.
- Regurgitation.

If the lithotomy position is used, then both legs should be moved together to avoid any strain on the pelvic ligaments. The knees can be inside or outside the metal support but should always be well padded as nerve damage can result due to pressure from the poles.
Respiratory impairment may occur especially in the obese patient.

Vasoconstrictors (e.g. adrenaline) are often used.

The danger of deep vein thrombosis is more likely in pelvic surgery.

Haemorrhage may occur and can often be difficult to see or measure accurately.

Post-operative vomiting is a common problem following gynaecological surgery.

ANAESTHETIC TECHNIQUES

Dilatation and curettage

Ketamine/diazepam technique as described under Techniques of anaesthesia Chapter 14

OR

Air or N_2O/Oxygen/volatile with spontaneous respiration.

Vaginal repairs)
Vaginal hysterectomy)
Spinal anaesthesia would normally be the method of choice.

If a general anaesthetic is required:

For short procedures: IV induction, LMA, spontaneous respiration using air or nitrous oxide/oxygen/volatile.
For longer procedures: IV induction, intubation, mechanical ventilation with air or nitrous oxide/oxygen/volatile and a non-depolarising muscle relaxant.

SPECIAL GYNAECOLOGICAL PROCEDURES

Laparoscopy

See under Abdominal surgery in Chapter 28

Vesico-vaginal fistula

Spinal anaesthesia is best used although this can be a long procedure and the type of anaesthetic should be discussed with the surgeon in advance. Some surgeons may require the reverse lithotomy position (i.e. prone with the legs supported in stirrups). The patient should not be placed in this position until the spinal anaesthetic is fixed. Careful padding under the chest and pelvis is necessary to allow free movement of the diaphragm and upper abdomen for adequate respiration.

CHAPTER 31

ANAESTHESIA FOR ORTHOPAEDIC SURGERY

Outline:

Points of importance to the anaesthetist

Anaesthetic techniques

Special anaesthetic problems in orthopaedic surgery

Management of a fractured jaw

GENERAL POINTS OF IMPORTANCE TO THE ANAESTHETIST

Orthopaedic operations may range from simple procedures such as closed reduction of fractures, to very long and complicated procedures such as hip replacements or spinal fusions.

SPECIAL PROBLEMS RELATED TO EMERGENCIES

Emergency orthopaedic operations may be complicated by:

Other injuries

- Head injuries
- Face, neck and upper airway injuries
- Chest injuries, pneumothorax or haemothorax
- Abdominal injuries.

These injuries must be specifically looked for and either ruled out or treated wherever possible before commencing the anaesthetic.

Blood loss may occur from open wounds, or the bleeding may be concealed within the thigh or pelvis. Blood loss must be replaced before surgery. Blood replacement must be guided by the vital signs discussed under Shock in Chapter 52.

Fractures and Blood Loss

Fracture of foot with moderate swelling	250-500 ml
Fracture of lower leg	500-1000 ml
Fracture of femoral shaft	500-2000 ml
Fracture of knee joint (up to)	2000 ml
Fracture of forearm	500 ml
Fracture of the humerus and shoulder (up to)	2000 ml.
Fracture of pelvis	3000- 4000 ml

A full stomach is a common problem associated with fractures and dislocations.

Points of importance for all orthopaedic procedures

The use of the tourniquet

- This is not recommended in patients with sickle cell disease (See Chapter 33) and peripheral vascular disease.
- A tourniquet is hazardous in old patients with cardiac failure. The sudden increase in venous return may embarrass the heart.
- The tourniquet produces ischaemia of the limb and a consequent acidosis.
- It may be associated with deep vein thrombosis.

- The time of application and release of the tourniquet must be noted. (This is usually the duty of the nursing staff).
- The surgeon should be notified when the tourniquet has been inflated for one hour. A maximum period of two hours should not be exceeded. The tourniquet time is less in older patients.

Posture. The patient may be placed in various positions. Abnormal positions carry certain hazards especially in patients with limited mobility.

Radiation hazards are present because of the use of image intensifiers and X-Ray machines. Take adequate precautions.

The need for repeated anaesthetics. Repeated exposure to halothane over a short period of time should be avoided if possible.

Wound infection is a very serious hazard in bone surgery. The anaesthetist must be especially conscious of this.

Restricted movement. Many patients have restricted movement or instability of cervical joints making intubation difficult.

ANAESTHETIC TECHNIQUES

Regional Anaesthesia

This provides good analgesia and relaxation and minimal disturbance of bodily functions. It requires a competent anaesthetist and a co-operative patient.

Suggested regional techniques:

- The upper extremities can be anaesthetised by an axillary block or Bier block.
- The lower extremities require a low spinal anaesthetic.

There are more specific blocks for wrist, hand, foot and ankle. See more specialized anaesthetic textbooks.

General anaesthesia

This is required when regional anaesthesia is contraindicated or when positioning for a regional anaesthetic causes too much pain.

Suggested techniques:

- Ketamine / diazepam may be used for short procedures such as reduction of fractures and dislocations. However, muscle tone is retained with ketamine and this may make reduction of fractures difficult. Supplement with opioids or a regional block.
- Mask or LMA anaesthetic with spontaneous respiration (Air or N_2O/O_2/halothane).

  This is suitable for surgery on the limbs. The patient must be supine. A ketamine infusion is useful.
- Controlled ventilation

  Air or N_2O/O_2/relaxant/volatile + opioids or regional block.

Patients should be intubated in all major emergency operations (unless a spinal or regional technique can be used).

SPECIAL ANAESTHETIC PROBLEMS IN ORTHOPAEDIC SURGERY

Fractured femur

- The patient with a hip fracture is usually old and may suffer from cardiovascular and/or respiratory disease.
- The patient may be hypovolaemic, having lost anything up to a litre of blood. Added to this is a period of low fluid intake associated with the pain and transport to hospital.
- The patient may be brought into the operating theatre in traction. This makes both regional and general anaesthesia difficult.
- These patients are more prone to deep venous thrombosis and pulmonary embolism.
- The orthopaedic table is hard, so pressure points must be carefully protected.

Total hip replacement

- Surgery for osteoarthritis or degenerative disorders of the hip joint is prolonged.
- Blood loss is large.
- The cement used is methyl methacrylate cement, prepared by mixing a liquid and a powder. When it is applied to the bone, hypotension and even cardiac arrest may follow.
  Prevent this complication by:
  - Maintaining adequate hydration
  - Replacing lost blood
  - Careful monitoring

Surgery on the vertebral column

This will not be discussed in detail as such operations will not be performed routinely in the hospitals for which this book is designed.

MANAGEMENT OF A FRACTURED JAW

A fractured jaw (mandible) is sometimes treated by wiring the jaws together. If it is at all possible this operation should be performed under local anaesthesia in order to avoid the dangers of general anaesthesia.

ANAESTHETIC MANAGEMENT

Pre-operative assessment

- A fractured jaw is often associated with other injuries. Examine the patient carefully for head, neck, chest, abdominal or orthopaedic injuries and assess blood loss.
- Check for airway obstruction, caused by:
  - Tissue injury and swelling of the face, pharynx and neck areas.
  - Material such as blood clots, dentures, teeth, food and vomitus in the upper airway.
  - The tongue falling back (in the semi-conscious or unconscious patient).
- Measure and record cardiovascular and respiratory parameters.
- Assess ability to open the mouth, as swelling and muscle spasm associated with the fracture may limit movement. Unless there is a bony mechanical obstruction, mouth opening improves with anaesthesia.
- A full stomach. The patient with a fractured jaw is in danger of regurgitation and aspiration of stomach contents. The stomach contents include swallowed blood and food. Severe trauma is associated with a delayed gastric emptying time.
- Preparation requires:
  - A nasotracheal tube (this is ***contraindicated*** if there is a drainage of CSF from the nose which may occur if the base of skull has been fractured).
  - A pharyngeal pack.
  - Wire cutters, to be available at the patient's bedside.
  - Expert post-operative nursing staff.

Premedication Avoid heavy sedation.

- The patient must be awake at the end of the operation.
- An anti-emetic (like promethazine) can be used in the premedication.
- A drying agent such as atropine or hyoscine is necessary especially if ether is used.

Intra–operative management

- Establish a good intravenous line. Adequate rehydration with fluids and blood is necessary before the anaesthetic is started.
- Carefully perform suction of the pharynx, using a disposable plastic or rubber catheter.

- If there is pre-operative evidence of airway obstruction, you may need to perform an awake intubation, blind nasal intubation or even a tracheostomy (under local anaesthetic) before anaesthesia is induced.
- If there is no pre-operative airway obstruction but the tissue injury and swelling in the facial and neck areas make it a likelihood when anaesthesia is induced, or in those patients who have a limited ability to open their mouths (because of the fracture), the following methods of anaesthesia are suggested:
  - An inhalational induction with air or N_2O/O_2 /volatile **OR**
  - Ether/air/O_2.

  Once the patient is deep enough, either intubate under deep inhalational anaesthesia (respiration may need to be assisted at this stage) or if the patient can be ventilated, then give a dose of suxamethonium 1mg/kg, apply cricoid pressure and intubate.

Maintenance

Air or N_2O/O_2/volatile/opioids/muscle relaxant, **OR**
Ether/air/oxygen/ opioids and muscle relaxant.
Monitor carefully.
Before the jaws are wired together:

- Remove the pharyngeal pack.
- Suction the upper airway very carefully (don't forget to look for blood clots behind the soft palate).

Reversal is routine, with atropine or glycopyrrolate and neostigmine.

Important points to remember

- Leave the tube in place until the patient is fully awake. This may mean that the patient is extubated in the recovery ward.
- Suction the pharynx both before and after extubation using a suction catheter.
- After extubation, a nasopharyngeal airway can be inserted if the patient can tolerate it.

Post-operative care

These patients need expert post-operative nursing.

- Observe the patient for the earliest signs of airway obstruction, which may be caused by the tongue falling back or by blood clots, secretions, vomitus, etc.
- Pay close attention to the patient's fluid intake (initially intravenous and later nasogastric or oral).
- Take steps to prevent infection. The use of antibiotics, the general nursing care of the patient and chest physiotherapy are all-important.
- Wire cutters ***must*** be available at the patient's bedside. Teach the nursing staff how and when to use them.

CHAPTER 32

ANAESTHESIA AND ANAEMIA

Outline:

Causes of anaemia

Important points about anaemia

Technique for anaesthesia

INTRODUCTION

Anaemia is a fall in the haemoglobin (Hb) concentration of the blood. It is a common clinical finding. Anaemia is only a sign of disease. It is not a complete diagnosis. The underlying cause must be found.

CAUSES OF ANAEMIA

There are three:

- Increased blood loss
- Decreased blood production
- Increased blood destruction

Increased blood loss

This may occur as a result of acute or chronic haemorrhage.
The haemorrhage may be in the:

Gastrointestinal tract accompanying:

- Peptic ulcer
- Cancer
- Haemorrhoids
- Parasites
- Ulcerative colitis

Respiratory tract from haemoptysis, as for example in tuberculosis.

Urinary tract from haematuria.

Decreased blood production i.e. lack of factors needed for Hb.

Microcytic anaemia (Iron deficiency). This can result from:

- Chronic blood loss
- Poor diet
- Poor absorption from gut
- Increased demand, as in pregnancy

Macrocytic anaemia. This can result from:

- Vitamin B_{12} deficiency
- Folic acid deficiency
- Dietary deficiency
- Pregnancy
- Gastric resection

Protein deficiency

Hormone deficiency e.g. lack of thyroxine or adrenal hormone.

Depression of bone marrow

- Aplastic anaemia e.g. toxins
- Lack of erythropoietin in chronic renal failure
- Infiltration of marrow as in leukaemia and carcinoma
- Disease of the marrow, e.g. myelofibrosis
- Chronic sepsis and infection

Increased blood destruction

Abnormal red blood cells in conditions such as sickle cell disease, thalassaemia and congenital spherocytosis.

Antibodies to red blood cells due to mismatched transfusions (of incompatible blood) or Rh factor incompatibility (erythroblastosis foetalis).

Medical conditions such as secondary carcinoma systemic lupus erythematosis and leukaemia.

Infectious diseases e.g. haemolytic streptococcus and malaria.

Chemicals and poisons e.g. lead poisoning.

Drugs e.g. sulphonamides
primaquine
quinidine
penicillin

IMPORTANT POINTS ABOUT ANAEMIA

- Anaemia is not a diagnosis but a *symptom* of an underlying disease. Find and treat the cause.
- If a patient is *dehydrated* the haemoglobin (Hb) may be artificially high.
- In some hospitals the *haematocrit* is the only available test for anaemia. Unless the patient is dehydrated, haematocrit gives a rough idea of Hb (approx. Hb x 3).
- When blood transfusions are needed to treat anaemia whole blood is not ideal. In the elderly patient whole blood may be dangerous because it can suddenly increase the blood volume. Packed cells may be safer.

  If only whole blood is available a small dose of diuretic (frusemide 10 mg IV) given with each unit helps to prevent overload.

Anaemia and the surgical patient

- In ***elective surgery*** aim to have the preoperative Hb as close to 10 gm/dL as possible. A minimum of 8gm/dL should be aimed at. A relatively fit patient with chronic anaemia may cope with surgery with a Hb as low as 7gm/dL whereas a patient with cardiac disease who may have a reduced cardiac output would not. The acceptable Hb level will depend on the type of surgery, the patient's general condition and the expected blood loss.
- In patients with Hb less than 5gm/dL ***operate only in emergencies*** where surgery is life saving, e.g. massive intra-abdominal bleeding. Blood should be given as soon as it is available.
- ***Pre-operative Transfusion.*** Packed cells (if available) can be given 24 hours before surgery. Watch for signs of overload.

1 unit of blood raises Hb by about 1gm Iron IM raises Hb by 1gm in 1 week Oral iron raises Hb by 1gm in 1 month

TECHNIQUE FOR ANAESTHESIA

These patients have minimum oxygen reserve. Hypotension or hypoxia can cause cardiac arrest. The possibility of high output cardiac failure must be considered.

- Transfuse or treat to correct anaemia.
- Avoid excess pre-operative sedation.
- Oxygenate before induction.
- Avoid myocardial depressants. Use ketamine instead of thiopentone and inject slowly.
- Use small doses of drugs.
- Have a good IV line in progress before you start.
- Use a high oxygen concentration.
- Intubate and ventilate except for very short procedures.
- Replace blood loss carefully with whole blood or packed cells.
- Keep the patient warm and warm IV fluids.
- Extubate only when the patient is making a strong ventilatory effort.
- Give the patient oxygen in the post-operative period – up to 100% O_2 if the patient is shivering.

See the information on Sickle cell anaemia in Chapter 33.

CHAPTER 33

ANAESTHESIA and DISEASES COMMON IN DEVELOPING COUNTRIES

Outline:

Sickle cell anaemia

Thalassaemia

Typhoid infection

Leprosy

Cancrum oris

Burkitt's tumour

Round worm infestation

Other common problems

SICKLE CELL ANAEMIA

Sickle cell disease occurs in West, Central and East Africa. It also occurs in people of African descent elsewhere. Patients with sickle cell disease have an abnormal haemoglobin. The formation of haemoglobin is controlled by specific genes, one inherited from the mother and the other from the father. If an abnormal gene is inherited from both parents the patient is said to be homozygous (SS) and presents with sickle cell anaemia. The patient who receives an abnormal gene from one parent is said to be heterozygous and presents with sickle cell trait. In sickle cell anaemia between 76% and 100% of the haemoglobin is HbS. The rest is haemoglobin F (Foetal Hb). There is no HbA (the normal adult haemoglobin) in these patients.

In sickle cell trait there is a mixture of HbA (normal haemoglobin) and haemoglobin S.

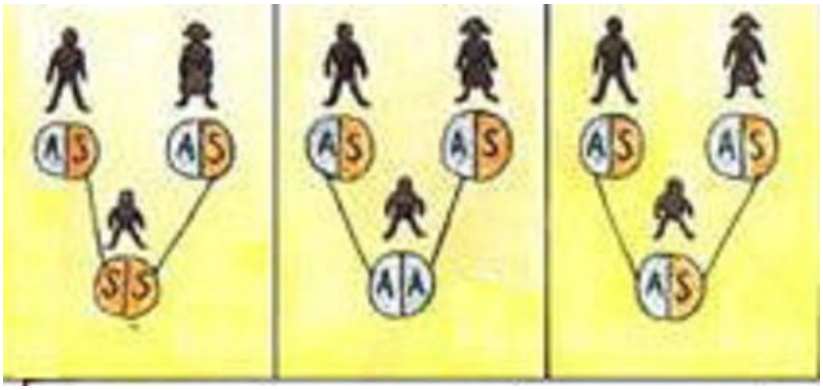


Fig 33.1 Genetic inheritance of Sickle cell anaemia (SS) and trait (AS)

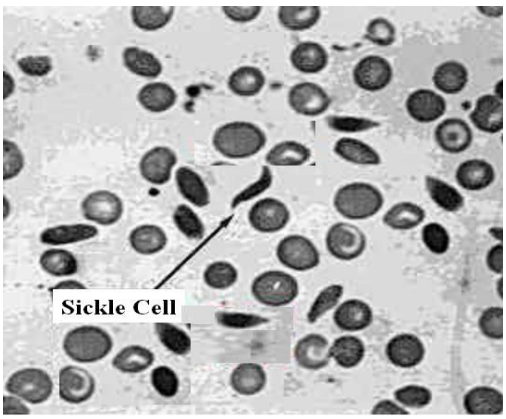


Fig 33.2 Blood film showing sickle cells

HbS is very much affected by changes in the oxygen tension of the blood. If the arterial tension falls below 45 mmHg in the case of sickle cell disease or below 25 mmHg in sickle cell trait, the haemoglobin within the red blood cells forms into crystals, called tactoids. These red blood cells have a sickle shape.

The liver, heart and spleen are enlarged as a result of this process.

Clinical manifestations

Sickle cell anaemia becomes clinically evident between the third and sixth months of life.

Haemolytic crises are characterised by anaemia, jaundice and fever.
Painful crises are produced by vaso-occlusive episodes, i.e. plugging of blood vessels.
Signs and symptoms depend on the organ involved, e.g. liver spleen, bone and joints.
Precipitating factors are infection, dehydration, acidosis and hypoxia.
Treatment consists of oxygen, analgesics, hydration and transfusion. The precipitating factors must also always be treated.
Aplastic crises. Complete cessation of RBC formation. This results in a rapid fall in Hb%.
Shock due to pooling of a large amount of blood in the liver or the spleen. (Shock may sometimes be cardiogenic).
Infection. Patients with sickle cell disease are prone to infection, especially pneumococcal and salmonella infections.
Gall stones. From high bilirubin levels secondary to liver dysfunction.

Surgical conditions associated with sickle cell disease

- Abdominal pain (acute abdomen)
- Osteomyelitis
- Leg ulcers
- Gall stones

Diagnosis

- History: anaemia, jaundice, abdominal pain
- Examination: anaemia, enlarged liver and spleen
- Laboratory diagnosis:
  - Haemoglobin/haematocrit
  - Special blood test using sodium metabisulphate. Sickling shows in 15 - 30 minutes. Microscopic examination of blood takes 48 - 72 hours.
  - Electrophoresis, a process which separates and identifies the haemoglobins.

Principles of anaesthesia in sickle cell disease

- All of the following can produce a crisis and must be avoided at all costs:
  - Hypoxia
  - Hypotension
  - Hypercarbia
  - Hypothermia
  - Dehydration

  The use of tourniquets is not recommended but is possible if special care is taken with exsanguination before inflating the tourniquet.
- Regional anaesthesia is the method of choice.
- It would be good practice to perform the "sickling test" on all African children and Africans with anaemia or a family history of sickle cell disease who present for surgery. Hb level would be useful.
- Small doses of drugs and a high inspired oxygen concentration are necessary. It is advisable to entrain oxygen at the rate of 4L/min throughout the procedure when using the EMO. A low ether concentration of 2 -3%, using the EMO, has been used in many cases of sickle cell disease without ill effects. Avoid excessive use of opioids because of the risk of respiratory depression.

Pre-operative management

Do not perform major surgery if the patient has:

- Haemoglobin lower than 7gm/dL
- Jaundice
- Hypovolaemia
- Infections

Pre-operative transfusion in a patient with a Hb% of 7g or over is best avoided because:

- These patients are more prone to reactions
- They are more prone to cardiac failure
- Transfusions may inhibit RBC formation.

Pre–medication: Routine -opioids in small doses, atropine (if ether is used).

Intra-operative management

Induction:

- Commence IV fluids (Hartmann's)
- Pre-oxygenate for four minutes
- Give thiopentone in small doses, e.g. 150mg for an adult male and then small increments till the eyelash reflex disappears. IV ketamine is an alternative.

Intubation: Give suxamethonium 1mg/kg. Ventilate with 100% oxygen for one minute and then intubate.

Maintenance:

- Air / nitrous oxide/oxygen (50%), volatile and muscle relaxant. Whichever anaesthetic circuit is used inspired oxygen concentration should not be less than 50%. This applies also to patients receiving ketamine only.
- Monitor the vital signs carefully.
- Fluid replacement: 0.9% saline. If needed replace blood loss with fresh blood.

Reversal: Atropine/ glycopyrrolate and neostigmine in the appropriate doses.

Post-operative management

- Give oxygen by mask until the patient is quite awake. For major surgery continue supplemental oxygen for 24 hours if possible.
- Continue IV fluids.
- Anticoagulants may be ordered by the surgeon to prevent thrombosis in the various organs.

THALASSAEMIA

Thalassaemia is associated with a defect in haemoglobin synthesis. It is found in sub-tropical regions such as the Mediterranean, the Middle East and parts of Asia. The patient who is homozygous inherits the abnormal genes from both parents and the disease is called thalassaemia major. The heterozygous form (i.e. inherited from one parent) is called thalassaemia minor. The deficiency of haemoglobin A (adult form) causes a compensatory increase in Haemoglobin F (Hb F). The bone marrow is hyperactive. Skeletal deformities such as a prominent maxilla, sunken nose and thickened skull are found.

The anaesthetist may be faced with a patient who presents for a splenectomy or any unrelated surgery. The normal principles of safe anaesthesia apply, plus:

- Optimal preoperative preparation of the patient is attained by transfusion until the haemoglobin level is at least 7- 8g/dL.
- Take care to avoid circulatory overload. Remember that the patient, though anaemic, is normovolaemic.
- The facial deformity mentioned earlier may cause problems with airway maintenance and intubation.
- A variety of systems such as the liver, kidneys and cardiovascular systems may be affected by the chronic anaemia.

TYPHOID INFECTION

Typhoid and paratyphoid fevers are caused by infections with the salmonella bacillus. The disease occurs where sanitation is poor and the water supply is contaminated by human waste. The organism infects the lymphoid tissue of the small intestine. The bacillus then enters the blood stream and is carried to other organs.

Clinical Signs

Incubation period is 10 to 14 days. The infection starts with a febrile episode in which the fever is highest at night. Headache, aching limbs and abdominal discomfort are features.

The classical rash, enlarged spleen and pea-soup stools develop after the 7th day. Toxic signs can develop rapidly. Between the 2nd and the 3rd week shock, perforation of or haemorrhage from the typhoid ulcers in the small intestine may occur. Other complications are myocarditis and meningitis. Diagnosis depends on serological tests.

The role of the anaesthetist in typhoid disease

The patient may present with a perforated ulcer. Two lines of treatment may be carried out.

- Treatment with chloramphenicol combined with drip and suction. No surgery is attempted.
- Surgical intervention with antibiotic treatment.

Problems

The patient presents for anaesthesia with some or all of the following problems in varying degrees:

- Dehydration
- Shock (septic, hypovolaemic, cardiogenic)
- Electrolyte imbalance
- Metabolic acidosis
- Very high temperature
- Delirium (altered mental state)
- Abdominal distension, with danger of regurgitation
- Chest infections. The typhoid perforation and the peritonitis predispose to chest infections. Other causes are abdominal distension, severe dehydration and toxic delirium.
- Myocarditis
- Anaemia associated with prolonged sepsis
- Renal failure

It is important to spend time resuscitating the patient before anaesthesia. This may take several hours, even up to 24 hours. The subject is covered in the section on Shock in Chapter 52.

Pre-operative measures

- Insert 2 large cannulae into the veins. Begin intravenous fluids with Hartmann's solution.
- Insert a urinary catheter and note the hourly urine output.
- Insert a CVP catheter if available.
- Monitor the following very carefully
  - Pulse
  - Blood Pressure
  - CVP and ECG if available
  - Respiratory rate
  - Pulse oximetry (if available) or colour
  - Level of consciousness
  - Temperature
  - Urine output
  - Electrolytes and acid base studies (if available)
  - Fluid balance charts
- Insert a nasogastric tube and note the drainage.
- Begin IV chloramphenicol.
- Use any other antibiotics indicated e.g. for secondary chest infection.
- Take specimens of stool and urine for examination.
- Take blood for routine tests:
  - Hb and Haematocrit
  - BUN, creatinine, electrolytes
- Take blood for special tests such as culture and serology (for antibodies). Note that in endemic areas serum antibodies may be raised due to chronic carrier status or previous immunisation.
- Arrange a chest x-ray to indicate any underlying pulmonary pathology.
- Prepare for post-operative ventilation if it is considered necessary. This involves:
  - Setting up a ventilator.
  - Ensuring a supply of O_2 or air to power the ventilator.
  - Organising personnel (doctors and nurses who will be responsible for the patient).
- Premedication. In most instances, no premedication is required.

Intra-operative Management

- Secure the airway with a tube.
- Give a higher oxygen concentration - at least 50% FIO_2.
- Give lower doses of drugs.
- IPPV is mandatory.
- Make sure fluid replacement is adequate.
- Monitor carefully.

Two methods of anaesthesia:

- A rapid sequence induction with cricoid pressure is indicated in all patients. This technique is discussed in detail under Techniques of anaesthesia in Chapter 16 and also in Shock in Chapter 52.
- An awake intubation followed by induction of light general anaesthesia might occasionally be accepted as the safest method in very sick patients with gross abdominal distension.

Post-operative care

Ventilation may be needed in addition to routine care.

LEPROSY

Leprosy is a complex clinical condition caused by infection with the *Mycobacterium leprae*. The total number of leprosy sufferers today amounts to some 15 million. Leprosy is transmitted by close contact with a leprosy patient. The bacilli are shed from the nasal mucosa and ulcerating skin lesions of patients. The epidermis of the skin is free of bacilli. A break in the skin is required for the spread of the disease.

There are two forms of the disease.

Lepromatous leprosy

The skin becomes atrophic, dry, hairless, hypopigmented and shiny. Localised nodules develop on the affected skin on the face and ears. The nodules contain aggregations of bacilli.

Tuberculoid leprosy

The essential lesion is a well-defined hypopigmented area of skin characterised by diminished sensation, sweating and hair growth. The nerves are affected:

- Damage to the sensory nerves results in anaesthesia in that area. Injury and secondary infection follow. Ulceration and repeated trauma result in the loss of extremities.
- Damage to the motor nerves results in weakness, paralysis and muscle contractures.

Fig 33.3 Skin lesions in a leprosy patient

Fig 33.4 Leprosy patient showing loss of extremities

Surgery in leprosy patients
Patients may present for surgery related to the leprosy e.g. tendon transplants, cosmetic surgery. Patients may also present for surgery unrelated to the disease, e.g. hysterectomies, vagotomies and abdominal emergencies.

Anaesthesia and the leprosy patient

- The social and psychological consequences of leprosy must be borne in mind and every effort made to show the patient that this is of no consequence where the anaesthetist / patient relationship is concerned. Every effort must be made to show the patient that the disease in no way detracts from his or her worth as a human being.
- Local infiltration can be used for tendon transplants.
- Spinals are considered contraindicated by some but in many instances may provide the best method of anaesthesia.
- Veins are often difficult to find in patients with leprosy.
- Contamination of equipment by nasal secretions will require the use of a disposable circuit or a circuit that is easily sterilised. Careful sterilisation of masks, airways, endotracheal tubes and laryngoscopes is essential.
- It is advisable to check the patient's previous medications. Patients with leprosy may sometimes have a history of treatment with steroids.

CANCRUM ORIS (Noma)

This is a gangrenous condition of the facial region. The suspected cause is Vincent's organism. There is rapid swelling of the affected facial area which becomes ischaemic and then gangrenous. This may follow any severe systemic disease in a child who is malnourished. Large areas of the lips, cheeks and jaws are destroyed and replaced by fibrous tissue. Surgery is required for reconstruction of the mouth and face. Intubation is difficult because of fibrosis around the mouth or jaws. Very often the aperture of the mouth is absent, so the laryngoscope cannot be inserted.

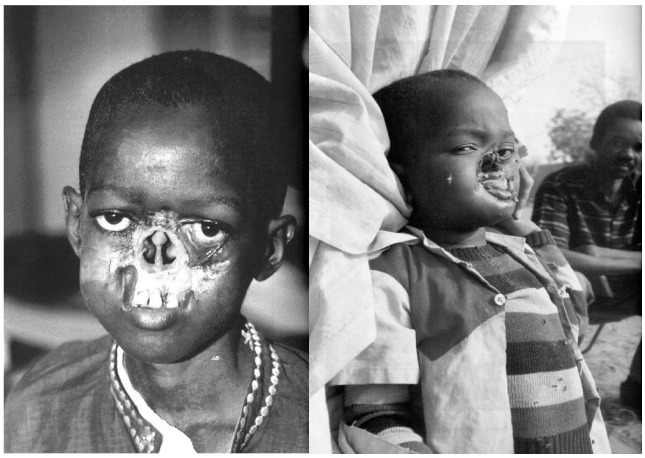


Fig 33.5 Facial destruction due to Cancrum Oris

- The problem must be very clearly discussed with the surgeon and the patient.
- A full general anaesthetic requires the insertion of an endotracheal tube. This may require a blind nasal intubation. A pre-operative tracheostomy may also be needed.
- The surgeon may decide to go ahead and operate with local anaesthesia and ketamine sedation.

BURKITT'S TUMOUR

This is the commonest malignant disease in children living in equatorial Africa. The disease occurs also in Papua New Guinea and in South America. It has been reported occasionally from the U.S.A., Western Australia and South Africa. Eighty percent of reported cases are in children.

Possible causes:

- Viral (frequent exposure to Epstein–Barr virus in early childhood).
- A chromosomal abnormality.
- Malarial changes in the reticulo-endothelial system.
- As a complication of AIDS.

The tumour affects the jaw and maxilla in 50% of patients. Other sites are ovaries, testes, thyroid, salivary glands and kidneys. Most of these tumours grow rapidly and relentlessly. Surgery is not indicated except for biopsy or removal of large ovarian tumours. The tumours are sensitive to chemotherapy e.g. cyclophosphamide, nitrogen mustard (methotrexate).

Problems for the anaesthetist

There are problems with intubation owing to involvement of the jaw. Laryngoscopy may be difficult or impossible. A blind nasal intubation or tracheostomy may be required if general anaesthesia is considered essential. Biopsy of the lesion under local anaesthesia is the safest approach if airway obstruction is likely.

ASCARIASIS OR ROUND WORM INFECTION

Ascariasis or round worm infection is caused by *Ascaris lumbricoides*. It is estimated that a quarter of the world's population suffers from the infection. The adult parasite is a large cylindrical worm 15 to 40 cm in length.

Infection follows the ingestion of the egg in food contaminated with faecal material or from contaminated hands. The larva is liberated in the small intestine, enters the blood stream and is carried to the lung. After about 10 days in the capillaries of the lung the larva passes up the bronchioles, the bronchi, the trachea and the pharynx and is swallowed with saliva back into the jejunum (small intestine). The larva develops into the adult worm in the small intestine.

The patient with ascariasis may need surgery for the following conditions:

- Abdominal pain which will need diagnostic laparotomy.
- Intestinal obstruction, intussusception and volvulus in the ileocaecal areas.
- Perforation of the intestine, especially at the site of a surgical anastomosis.

It is important to be aware of the possibility of:

- Bronchopneumonia, characterised by cough, fever and wheezing.
- Anaemia. This is much less common than in hookworm disease.

OTHER PROBLEMS COMMON IN AFRICA

Severe sepsis. This may follow advanced gangrene or gas gangrene. Patients with severe typhoid and typhoid perforation present with septic shock. Appendicitis, though rare, presents in hospital with the picture of septic shock.

Criminal abortions. Where infection has been introduced into the peritoneal cavity, patients often present with septic shock. Septic (bacteraemic) shock is described in Chapter 52.

Severe blood loss. This may follow an ectopic pregnancy or a traumatic rupture of an already enlarged spleen. The usual precautions taken for resuscitating and then anaesthetising a shocked patient must be followed. Treat all of these patients as having a full stomach and do a rapid sequence induction with cricoid pressure. Auto-transfusion may also be necessary, in addition to the use of blood from the bank.

Strangulated hernias are common. The patients are often frail and elderly. If the strangulation has already been present for a few days , the patients are generally grossly dehydrated. There is abdominal distension, a high temperature and electrolyte imbalance but a spinal is contraindicated on the grounds of hypovolaemia and intestinal obstruction. A general anaesthetic is hazardous. A field block as described in Chapter 18 (Local anaesthetic techniques) is the best choice. Pre-operative rehydration is essential.

CHAPTER 34

ANAESTHESIA AND HEART DISEASE

Outline:

Heart failure

Hypertension

Myocardial ischaemia

Arrhythmias

Other cardiac conditions

HEART FAILURE

Right heart failure

The signs are:

- Ankle oedema
- Elevated jugular venous pressure
- Enlarged and tender liver
- Third heart sound

Left heart failure

When the left side of the heart fails, there is congestion in the lungs. Therefore the symptoms are respiratory. Dyspnoea is the main symptom, first noted on exertion. Later attacks occur at night and finally the dyspnoea persists even at rest. On auscultation of the chest fine or coarse crepitations may be heard. It is important to find the underlying cause of the cardiac failure, e.g. ischaemia, hypertension, valvular heart disease.

Anaesthesia

Patients with uncontrolled congestive heart failure (left heart failure) should not be anaesthetised for routine surgery as there is a high perioperative morbidity. They should be treated pre-operatively with:

- Oxygen.
- Diuretics, e.g. frusemide.
- Potassium supplements as indicated.
- Digoxin only if there is rapid atrial fibrillation.
- ACE inhibitors (angiotensin converting enzyme inhibitors), if available.

Pre-operative management: Once the cardiac failure has been controlled, it is safe for surgery to proceed. Frusemide can also be given IM depending on the severity of the cardiac failure. The drugs for treatment of cardiac failure should be continued right up to the morning of operation

In the urgent case when it is not possible to spend time treating the cardiac failure, a regional anaesthetic may be preferable to a general anaesthetic. A spinal might be reasonable if aortic stenosis and unstable angina are excluded. If a general anaesthetic is essential it must be given very carefully.

Intra-operative management: If a general anaesthetic is used, the principles are:

- Give small doses of drugs
- Air/oxygen/volatile ± relaxant or a ketamine technique would be reasonable
- Give a higher inspired oxygen concentration to avoid hypoxia
- Avoid hypotension
- Avoid fluid overload
- Give intermittent positive pressure ventilation.

HYPERTENSION

This is an increasing problem in anaesthesia. Hypertension, when it occurs in younger people can be due to an underlying disease, e.g. renal, endocrine, etc. In all cases prolonged hypertension, especially if untreated, affects various organs, e.g. the heart, kidneys. These patients are prone to heart failure and renal failure, because they cannot compensate for hypotension, hypoxia or hypercarbia. Some may be on anti-hypertensive drugs. It is now accepted that:

- All hypertensive patients should be treated before elective surgery and their blood pressure stabilised to a diastolic pressure of 90-100 mmHg.
- Patients are kept on their anti-hypertensive drugs right up to the time of operation.
- Patients presenting for elective surgery with untreated hypertension with a diastolic pressure > 115 mm Hg on at least two readings should be cancelled and prescribed treatment.

Points of importance to the anaesthetist

Patients on anti-hypertensive drugs cannot compensate in the usual way for the stress of anaesthesia. Things to monitor closely are:

- Anaesthetic drugs and their side effects
- Blood loss
- Changes in position
- IPPV
- Avoid hypoxia
- Avoid hypercarbia.

The same precautions, as described for patients with myocardial ischaemia or CCF must be used for hypertensive patients.

MYOCARDIAL ISCHAEMIA

Myocardial ischaemia, or damage to the myocardium, results if the arteries supplying the heart muscle become narrowed or blocked. The blood supply to the heart muscle is therefore reduced and ischaemia results. Myocardial ischaemia must be carefully evaluated before surgery. An ECG should be done.

If this shows evidence of a myocardial infarct and the surgery is elective, then 6 months should elapse before the operation. If the surgery is semi-urgent and has to be done under a general anaesthetic then a minimum of 3 months should still elapse. Urgent surgery is carried out in the context of risk against benefit. Even 6 months following the infarct, the mortality rate is higher.

If the ECG shows evidence of myocardial ischaemia only, without damage or death of the heart muscle (i.e. no infarction) then anaesthesia still carries certain risks because these patients are very liable to go into heart failure and /or develop an arrhythmia, or progress to infarction.

Anaesthetic technique

The same principles and techniques as outlined for patients with congestive heart failure should be followed in patients with myocardial ischaemia.

- Stabilise the patient as much as possible preoperatively i.e. nitrates, beta-blockers
- Give small doses of drugs, especially myocardial depressants, e.g. thiopentone, halothane, etc.
- Use a high inspired concentration of oxygen.
- Have good intravenous access.
- Avoid hypoxia, hypercarbia and swings in blood pressure. These patients are more liable to hypotension on account of their drug treatment, e.g. beta-blockers.
- Avoid fluid overload.
- Use a relaxant technique with IPPV.

ARRHYTHMIAS

The dangers of arrhythmias under anaesthesia are:

- Myocardial ischaemia as a result of a rapid heart rate
- Ischaemia of the brain and kidney
- Myocardial infarction
- Cardiac failure
- Ventricular fibrillation (cardiac arrest)
- Thrombo-embolism.

The following are arrhythmias which should be treated before elective surgery:

- Sinus tachycardia. Find the cause and treat it first.
- Sinus bradycardia, especially if there is an associated fall in cardiac output and blood pressure.
- Atrial tachycardia)
Atrial flutter) if the rate is above 100/min.
Atrial fibrillation)
- Ventricular ectopics (if greater than 5 per minute, **or** multifocal, **or** close to the preceding T-wave with the risk of the R on T phenomenon).

OTHER CARDIAC CONDITIONS

- Rheumatic heart disease. In the acute stage, general anaesthesia is contraindicated. In rheumatic valvular heart disease, give pre-operative cover with antibiotics to protect against endocarditis and observe the safety principles outlined earlier.
- Congenital Heart Disease. Here too give antibiotic cover pre-operatively.

Suggested regime of antibiotic cover for patients at risk of endocarditis

Amoxycillin 3g orally 4 hours before surgery or 1g IV at induction followed by amoxycillin 3g orally 6 hours post-operatively

OR

(for patients who are allergic to penicillin)

Vancomycin 1g by IV infusion over 1-2 hours followed by gentamicin 120mg IV at induction.

Not all of these antibiotics will be available in many hospitals but should be used if possible.

CHAPTER 35

ANAESTHESIA AND RESPIRATORY DISEASE

Outline:

Acute chest infections

Chronic chest infections

Asthma

Pulmonary tuberculosis

Other respiratory problems complicating anaesthesia

ACUTE CHEST INFECTION

Any acute respiratory infection is a contra-indication to elective surgery. Anaesthesia will definitely worsen the problem, spreading the infection to the lower airways.
Treat the patient with antibiotics preoperatively and wait at least one week after recovery before contemplating surgery.

Emergency surgery is best done under a regional block. If a general anaesthetic is essential, then preoperative antibiotics are needed. Pre-operative bronchodilators may also be necessary. Ether and halothane may be useful if there is any bronchospasm.

Patients will need intensive preoperative and postoperative chest physiotherapy.

Upper respiratory tract infections in children

These are very common and a nasal discharge may be almost a permanent state in some children. It has been shown that a URTI within the six weeks prior to anaesthesia leads to an increase in respiratory complications in the peri-operative period.

A good history from the parents may establish whether the child has a permanent runny nose. If this is the case and there is no fever present and no evidence of lower respiratory tract disease then it is acceptable to proceed with anaesthesia for minor procedures.

If there is fever and/or abnormal signs in the lower respiratory tract surgery should be postponed for one month.

In the emergency situation the risks of postoperative complications against benefit have to be balanced.

CHRONIC CHEST INFECTIONS

Examples are chronic bronchitis, bronchiectasis and emphysema. These conditions are characterised by dyspnoea and chronic cough with sputum.

Pre-operative management

- Every effort should be made to reduce the volume of sputum by chest physiotherapy and postural drainage.
- Patients should not smoke for at least 48 hours before the operation.
- If there is any evidence of an infective process, e.g. yellow sputum, temperature, etc., test the sputum by culture and sensitivity if the facilities are available and use the appropriate antibiotic.
- Any pre-operative bronchospasm must be treated vigorously. Elective surgery must be postponed if it persists.
- If facilities are available, preoperative lung function tests should be done.

Intra-operative management

- Use a regional anaesthetic, if appropriate.
- If a general anaesthetic is used, use short acting drugs, avoid large doses of opioids and aim for a rapid return to good respiratory effort. Volatile agents are useful as they are bronchodilators. High inflation pressures may be needed for ventilation. Smaller tidal volumes with an increased respiratory rate will reduce airway pressures. A prolonged expiratory time is useful in severe bronchospasm to prevent gas trapping (i.e. 1:3 I/E ratio).
- The patient may need suction through the endotracheal tube at the end of the operation.

Post-operative care is very important

- An oxygen rich mixture given during or after anaesthesia may interfere with the hypoxic drive of the respiratory centre, so give oxygen with care. This is extremely rare (severe emphysema) and most patients require more oxygen than patients with normal lungs. This is discussed under Oxygen therapy in Chapter 54.
- These patients may be sensitive to central respiratory depressants such as opioids (morphine, pethidine). However good analgesia is very important in abdominal and chest surgery to help the patient cough and clear secretions.
- Patients will need intensive chest physiotherapy post-operatively.
- Occasionally patients with chronic chest disease require postoperative ventilation. This is more likely after prolonged surgery and if there is an upper abdominal incision.

ASTHMA

Elective surgery

This should be postponed until the attack of asthma (i.e. bronchospasm) is treated. Treatments include:

- Bronchodilators via aerosol e.g. Ventolin, Atrovent.
- Aminophylline
- Steroids
- Physiotherapy

A regional technique should be employed if possible but blocks with a risk of causing pneumothorax should be avoided.

Pre-operative management

Premedication

- Benzodiazepines are useful
- An antihistamine such as promethazine may be beneficial
- If an opioid is required pethidine (small dose) may be used

Intra-operative management

Induction: Propofol, etomidate or ketamine can be used for induction An inhalation induction with halothane is also suitable. Suxamethonium causes histamine release but this is not usually a problem and if rapid sequence induction is required it may be used. Good relaxation and rapid intubation are important in asthmatic patients and the risks in individual patients need to be balanced.

***Intubation*:** The presence of a tube in the trachea may spark off an attack of bronchospasm, so:

- Do not intubate the patient under a light plane of anaesthesia.
- Spray the vocal cords with a local anaesthetic solution.

Maintaining anaesthesia: Most volatile agents are bronchodilators and are good maintenance agents. Atracurium on account of the associated histamine released, may aggravate the bronchospasm. Pancuronium, vecuronium and rocuronium are the preferred relaxants if available. Pethidine and fentanyl are the preferred opioids for intraoperative and postoperative analgesia.

Reversal: Neostigmine may aggravate bronchospasm but this is rarely a clinical problem. It should be given in small doses (0.5 mg IV at a time), preceded by atropine or glycopyrrolate.

PULMONARY TUBERCULOSIS

Patients with open active tuberculosis should not have elective surgery until they have been treated with anti-tuberculous medication for a minimum of three weeks. If surgery is essential before this, then certain precautions must be taken to prevent spread of infection. Regional anaesthesia is the best choice.

- Use disposable equipment if possible.
- If non-disposable equipment is to be used then an easily sterilisable set-up such as the Magill circuit is best. After use, the circuit should be sterilised as described in Chapter 60. Discard the endotracheal tube and Guedel airway.
- Use bacterial filters if they are available, pore size 0.22.
- The patient should wear a mask in theatre.
- All personnel in theatre should change gowns, masks, shoes, etc. before the next case is brought in.

These precautions need not be taken in cases of inactive (old) T.B.

OTHER RESPIRATORY PROBLEMS COMPLICATING ANAESTHESIA

Pulmonary oedema, acute bronchospasm, pulmonary embolism and pneumothorax are problems which complicate anaesthesia and the postoperative period. They have been discussed elsewhere. See Chapters 46 & 62.

Pleural effusion

A pleural effusion implies the presence of underlying disease. Its cause needs to be investigated. The effusion may have to be tapped before anaesthesia if it is large or if it is producing cardiovascular or respiratory embarrassment. The aspirate needs to be examined and cultured for diagnostic purposes.

Cancer of the lung (Bronchial carcinoma)

Apart from the interference with lung function, bronchial carcinoma may be associated with a pleural effusion, anaemia and secondary deposits elsewhere such as in the spine. Many of these patients have pre-existing chronic obstructive airways disease. The association of bronchial carcinoma and myasthenic syndrome is discussed in Chapter 43.

CHAPTER 36

ANAESTHESIA AND THE ELDERLY PATIENT

Outline:

The ageing process and associated changes

Principles of geriatric anaesthesia

Anaesthetic management

THE AGEING PROCESS AND ASSOCIATED CHANGES

General considerations

- Ageing is associated with a reduction in the number of functioning cells and therefore a reduction in lean body mass.
- There is a reduction in total body water, mostly in the intracellular compartment.
- There is interference with the temperature regulating mechanisms in the body. This is partly due to the reduction in the number of skin capillaries and an atrophy of the sweat glands. These patients are more susceptible to hyper- and hypo-thermia.

Cardiovascular system

- Prolonged circulation time
- Myocardial ischaemia
- Cardiac failure
- Hypertension
- Arrhythmias

Respiratory system

- Chronic obstructive airways disease is more common.
- There may be limitation in chest movements due to ossification of cartilages.

Skin and the musculo-skeletal system

- Skeletal muscle wasting is common. Flabby cheeks, atrophy of the mandibles and loss of teeth make it more difficult to obtain an airtight seal with a mask. Any residual degree of neuro-muscular block becomes more significant if there is muscle wasting.
- Osteoarthritis of the joints is common. Stiffness in the neck and temperomandibular joints may interfere with intubation. Osteoarthritis in the neck may interfere with the blood flow to the brain. Osteoarthritis in the spine may interfere with positioning the patient for a spinal. This also makes the spinal block technically difficult. Positioning of patients with osteoarthritis must be done very carefully.
- The skin is fragile. Pressure areas must receive attention. Some peripheral nerves are liable to injury and must be protected. If stirrups, arm boards, tourniquets, restraining straps and ECG leads are used special care is needed. Intravenous cannulation is more difficult. Venepuncture sites must be carefully dressed.

Blood

Chronic anaemia and dehydration may be present.

Central nervous system

Cerebral atherosclerosis may result in a certain degree of dementia, which may interfere with the degree of co-operation received from the patient. The dose of drugs, both analgesic and anaesthetic, should be markedly reduced. The average elderly patient, however, is often placid and co-operative and hence suitable for regional techniques.

Renal system

A certain degree of renal impairment is common in the elderly. Drugs excreted by the kidneys should be used in smaller doses or avoided if possible.

Drug therapy

Intercurrent medical problems are more common in elderly patients, who are more likely to be on drugs such as antihypertensives, digoxin and diuretics. The effects of these drugs on anaesthesia are discussed in the section on Pre-operative care in Chapter 7.

PRINCIPLES OF GERIATRIC ANAESTHESIA

- Only very small doses of anaesthetic drugs are needed, because of:
  - Impaired renal function
  - Impaired liver function
  - Peripheral arteriosclerosis, channelling a greater proportion of the drug to vital centres, e.g. the heart and brain
  - A reduction in muscle mass
  - A decrease in the total volume of circulating blood, so the injected drug is distributed in a smaller blood volume.
- The circulation time is prolonged. Therefore it is important to pause after a small dose of drug is injected, to give the drug sufficient time to act.
- It is important to have good access to a vein, preferably through an intravenous infusion.
- It is important to supplement the anaesthetic technique with added oxygen.
- Hypotension, hypoxia and hypercarbia must be avoided.
- Careful monitoring of the patient is essential.
- Avoid hypothermia.

ANAESTHETIC MANAGEMENT

Any medical problem that can be corrected or improved must be treated pre-operatively. Then:

Regional anaesthesia
This is the method of choice in the elderly. The volume of local anaesthetic required for a spinal block is not dependent on age but the effect may last longer due to slower washout from the tissues. The dose of local anaesthetic should be at the lower end of the range or slightly less in the geriatric patient.

General anaesthesia

Pre-operative management

Pre-medication is best avoided in elderly patients as it can cause confusion. Atropine may be given if ether is used.

Intra-operative care

Induction: Preoxygenation should be carried out in all elderly patients. A very small dose of thiopentone, e.g. 125 - 150mg or propofol 1-2mg/kg **or** a small dose of ketamine (0.5 - 1.0mg/kg IV) may be used initially. Allow time for the drug to take effect.
Intubation: Suxamethonium 1mg/kg. Ventilate with 100% oxygen and intubate the patient.
Maintenance: Using the EMO technique, introduce oxygen at a minimum of 2L/min into the circuit + ether 2% - 3%, air and muscle relaxant if required.

or

Air/oxygen/volatile and muscle relaxant if required.
Reversal is routine, using atropine 1.2mg and neostigmine 2.5mg.

Post-operative care

- Routine observations are carried out.
- Post-operative oxygen and fluids are extremely important.
- Post-operative analgesia should be given in smaller doses because of the patient's increased sensitivity to these drugs.
- Restart pre-operative medication as early as possible in the post-operative period.

CHAPTER 37

ANAESTHESIA AND RENAL DISEASE

Outline:

Problems

Techniques of anaesthesia

PROBLEMS

Patients with renal disease require operations for procedures related to their renal disorder (e.g. nephrectomy, cystoscopy, etc.) or for procedures quite unrelated to these conditions. They may present the following problems:

- Metabolic acidosis
- Electrolyte imbalance, especially a high serum potassium. This may lead to cardiac arrhythmias and problems with relaxants.
- Uraemia
- Anaemia
- Abnormal response to drugs, especially drugs which are excreted by the kidneys. Gallamine (Flaxedil) must be avoided.
- Hypertension (common in patients with kidney disease).
- Drugs which are toxic to the kidneys must not be used.
- Extreme sensitivity to all anaesthetic drugs. Smaller doses should be used.

Anything that produces renal vasoconstriction, notably hypotension, hypoxia and hypercarbia, must be avoided.

TECHNIQUES OF ANAESTHESIA

Regional

Regional techniques are the methods of choice. Spinals are very suitable for transurethral resections (TUR) and suprapubic prostatectomies.

General

If a general anaesthetic is required:

- Drugs must be given in smaller doses.
- Blood transfusions must be freshly drawn as old blood with a high potassium content tends to raise an already high serum potassium.
- Care should be taken not to over transfuse as patients with chronic renal failure tolerate anaemia relatively well.
  Aim for a Hb of 8-10gm/dL.

Follow the usual principles for an ill patient:

- Small doses of drugs
- High inspired oxygen
- Careful monitoring
- Good IV access.

Using these guidelines, the following techniques would be suitable.

- If **controlled respiration** is indicated, as for nephrectomy and suprapubic prostatectomy, this technique will be safest.

  Pre-operative management

  Premedication: A small dose of benzodiazepine or opioid is suitable.

  Intra-operative management

  The aim should be to avoid hypoxia, hypovolaemia and hypotension. All common anaesthetic drugs reduce urine flow.
  Induction: Pre-oxygenate, give propofol or thiopentone, followed by suxamethonium and then intubate.
  Maintenance: Air or N_2O and oxygen with a volatile agent and relaxant

  or

  Ketamine/relaxant technique.

  Atracurium is the relaxant of choice as it is broken down in the blood stream and not excreted by the kidneys.
  Reversal: Atropine or glycopyrrolate and neostigmine.

- **Spontaneous respiration** with air/ N_2O /oxygen and halothane via a facemask or LMA may also be useful, as in a cystoscopy.

- **A ketamine technique** is also suitable for cystoscopies.

CHAPTER 38

ANAESTHESIA AND LIVER DISEASE

Outline:

Problems

Techniques

PROBLEMS

The liver is able to sustain a considerable amount of damage without much loss of function but operations and anaesthetics must be avoided when patients have serum or infective hepatitis.

Abnormal response to drugs:

- The response to drugs that are de-toxified in the liver can be prolonged or abnormal in patients with liver disease. These drugs must therefore be given in small doses. Examples are thiopentone, morphine and local anaesthetics.
- Drugs broken down by enzymes manufactured in the liver, e.g. depolarising relaxants such as suxamethonium have a prolonged effect.
- Certain drugs like pancuronium and vecuronium are bound to the globulin fraction of plasma proteins. In liver disease the albumin level falls but the globulins rise. Larger doses of pancuronium/ vecuronium may be needed.

Toxic drugs: Drugs that are toxic to the liver must not be used, e.g. chlorpromazine. In some regions many patients are Hepatitis C positive. Halothane is probably safe to use in these patients if there are no alternative agents**.**

Bleeding tendency: Patients with liver disease have a bleeding tendency.

- Due to decreased vitamin K absorption they have a low level of prothrombin, which is necessary for blood to clot. Vitamin K (the water soluble analogue) must be given pre-operatively (10mg IV slowly).
- In advanced liver disease the platelet count may be low due to hypersplenism.

Anaemia is often present.

Other general disorders: Patients with liver disease are usually in a poor physical state, e.g. chronic bronchitis is common.

Hypotension, hypoxia and hypercarbia must be avoided. Each of these can aggravate liver dysfunction and cause postoperative renal failure. In fact, avoiding them requires more attention than choosing the type of drug that will be used for anaesthesia.

Renal disease and hepatic disease often co-exist.

TECHNIQUES OF ANAESTHESIA

In liver disease a regional anaesthetic should be used whenever it is possible. It is advisable, however, to test the patient's coagulation studies before a regional anaesthetic is used, as a bleeding tendency is a relative contraindication to a regional technique. The amount of local anaesthetic used may need to be assessed carefully. If a general anaesthetic is needed then a very careful anaesthetic must be given, similar to that recommended for patients with renal dysfunction.

CHAPTER 39

ANAESTHESIA AND DIABETES MELLITUS

Outline:

The diabetic patient

Management of diabetes

Anaesthetic management of the diabetic patient
- Principles
- Peri-operative regimes

THE DIABETIC PATIENT

Diabetes mellitus is a clinical condition characterised by hyperglycaemia. It is caused by a deficiency, or diminished effectiveness of insulin in the body.

The action of insulin

The main action of insulin is to help the entry of glucose from the blood into the cells where it is stored as glycogen. If insulin is deficient then the following changes occur:

- The blood glucose is not converted into glycogen and stored. This results in hyperglycaemia. The high blood sugar spills over into the urine and therefore the patient has glycosuria.
- Fat metabolism is increased to supply energy. The products of fat metabolism (ketones) appear in the urine and blood.
- Protein metabolism. The synthesis of protein in the body is slowed down in diabetes and therefore muscle wasting occurs.

The symptoms and signs of diabetes

- Polyphagia (excessive hunger)
- Polyuria (excessive urine output)
- Polydipsia (excessive thirst)
- Ketones are acid in nature. An excessive accumulation of ketones causes the patient to develop a metabolic acidosis (or ketosis). This is associated with dehydration and electrolyte imbalance.
  The patient with uncontrolled diabetes may present with collapse and coma due to metabolic acidosis (ketosis).
- Other signs of diabetes mellitus include wasting and infections which will not heal. TB is more common in diabetics.

Symptoms of secondary involvement of other organs are

- Peripheral vascular disease
- Heart disease
- Renal disease
- Peripheral neuritis
- Eye disease

Diabetic coma

The differences between

coma from hyperglycaemia	coma from hypoglycaemia
General Condition	
Cold, dehydrated, having sunken eyeballs, breath smelling of acetone, sluggish reflexes	Pale, sweating, large pupils, hyperactive reflexes
Respiration	
Deep sighing	Normal
Circulation	
Low blood pressure	Normal blood pressure, tachycardia

In addition to the symptoms and signs given in the table, the patient's history aids diagnosis.

A history of gradual onset of coma, or a longer period of illness before the coma suggests hyperglycaemia. Hypoglycaemic coma has a sudden onset. A history of missing a meal, after taking the usual dose of insulin, suggests hypoglycaemia.

A coexisting secondary infection tends to upset the diabetic state and it is common to find patients developing hyperglycaemic coma.

MANAGEMENT OF DIABETES

Diabetes is usually treated by one of three methods:

Diet alone
About 30-35 cal/kg (125-145 J/kg) are divided as follows:
50% as carbohydrate
30% as fat
20% as protein

Oral hypoglycaemic agents
Older patients and milder diabetics can be treated with oral drugs. Tolbutamide, chlorpropamide and glibenclamide are examples.

Insulin The following categories of patients are treated with insulin:

- Juvenile or unstable diabetics
- Those not responsive to oral therapy
- Those presenting with severe infections or those presenting for major surgery.

There are various types of insulin

	Peak action	
Short acting	s/c 2-4 hrs	soluble insulin Actrapid
medium acting	4-8 hrs	isophane insulin
long acting	8-12 hrs	protamine zinc insulin, ultralente

Insulin preparations vary from country to country so it is best to become familiar with what is available locally.

ANAESTHETIC MANAGEMENT OF DIABETIC PATIENTS

PRINCIPLES

- It is always safer to have a patient hyperglycaemic under the anaesthetic than hypoglycaemic.
- Patients for elective surgery must not be anaesthetised unless their diabetic state is well controlled.
- A regional technique, if suitable, is the first method of choice.
- Place the diabetic first on the operating list, if possible, to minimise the period of fasting.
- Diabetes affects the heart, the blood vessels and the kidneys, so these systems should be investigated, especially in the case of long standing diabetes. The following tests are recommended: chest x-ray, ECG, serum electrolytes, random blood sugar, and urinalysis.
- Ether is associated with a rise in blood sugar, but a low concentration of ether is not contraindicated in diabetics.
- The scheme of control: textbooks describe various methods of management for diabetics undergoing surgery. It is best to get familiar with one or two methods. Two methods will be described below.

PERI – OPERATIVE REGIMES

The anaesthetic management depends on whether the diabetes is controlled or uncontrolled, whether the surgery is major or minor, and whether it is elective or emergency.

Controlled diabetes and major surgery

First method using sliding scale of soluble insulin

On the day of surgery withhold the usual dose of long acting insulin or oral medication and commence and stabilise the patient on a sliding scale of soluble insulin. The actual dose of insulin depends on the amount of glucose in the blood or urine. The following scales are practical and useful.

Sliding scale based on 4-hourly glucometer readings

mmol/l	*mg%*	*soluble insulin*
0 - 4	< 72	0 units
4.1 – 8	73-144	4 "
8.1 - 12	45-216	6 "
12.1 – 16	217-290	8 "
16.1 - 20	91-360	12 "
> 20	>360	Seek advice

On the morning of operation commence a 5% dextrose infusion running at 30 drops/min until the patient comes to theatre.
Add 10 mmols KCl to each 500mls of 5% dextrose.
Perform four-hourly blood sugar and urinalysis and give insulin treatment according to the scale. The bladder must be completely emptied after each sample of urine is taken for examination.

Second method using longer acting insulin

- Start a 5% dextrose infusion on the morning of the operation. The fluid runs in at the rate of 30 drops/min.
- Give half the usual dose of long acting insulin on the morning of the operation.
- Continue four-hourly blood sugar and urinalysis.
- Test blood sugar on the evening of the operation, and give a further dose of insulin if necessary.

For other regimes consult World Anaesthesia journal Update no 11 (2000)

Controlled diabetes and minor surgery

In the case of the patient with controlled diabetes who presents for minor surgery, the usual anti-diabetic treatment (i.e. insulin or tablets) is not given on the morning of the operation. A 5% dextrose drip is commenced in the OR, and normal medication is resumed the next day provided the oral intake of food is adequate.

Uncontrolled diabetes

Elective surgery: The diabetes must be treated and controlled before surgery is performed.
Emergency surgery: If ketones appear in the urine (even if the operation is an emergency) you must try to delay surgery until the diabetic state is treated. This may take 8-12 hours. The acidosis and electrolyte disturbance must be corrected first, if possible.

Regime for management of ketoacidosis

- ***Rehydration***
  Give 1 litre 0.9% saline over 30 mins
  Give 1 litre 0.9% saline over 1 hour
  Give 1 litre 0.9% saline over 2 hours
  Continue 2-4 hourly until blood glucose is controlled then continue with 5% dextrose 1 litre 2-4 hourly.
- ***Reduce blood sugar level***
  Give 20 units soluble insulin IM then 6 units IM hourly.
  Check blood glucose hourly.
  When blood glucose is < 15 mmol/l change to 6 units IM 2 hourly.
  When patient is eating and drinking change to S/C insulin.
- ***Potassium supplementation***
  Add 10mmol KCl to the first litre of saline. If potassium measurements are available this can be adjusted as required otherwise continue with 10mmol KCl / litre of fluid.
  Emergency surgery can start once this treatment regime is established.

CHAPTER 40

ANAESTHESIA AND ADRENAL INSUFFICIENCY

Outline:

General structure

Adrenal cortex

Guidelines for the use of steroid replacement therapy

Adrenal medulla

GENERAL STRUCTURE

The adrenal gland has two separate parts: the cortex (70%) and the medulla (30%). Each develops from a different type of embryological tissue and has its own distinct functions.

ADRENAL CORTEX

The adrenal cortex produces three types of hormones.

Glucocorticoids which have an effect on carbohydrates, fat and protein metabolism. They also have an anti-inflammatory action. Cortisol is the principal glucocorticoid and is essential for many physiological processes. It must be replaced if deficient. Hydrocortisone hemisuccinate, dexamethasone and prednisolone are examples of drugs used for steroid replacement therapy.

Mineralocorticoids which control sodium retention and potassium excretion. They are secreted in response to a reduction in plasma sodium concentration due to low intake or following surgery, trauma and haemorrhage. Aldosterone is the principal mineralocorticoid.

Sex hormones which are secreted by the adrenal cortex. Abnormalities of androgen secretion are usually genetic and interfere with normal growth and sexual characteristics in childhood.

Corticosteroid therapy (synthetic glucocorticoids and mineralocorticoids)

Steroids are used in the treatment of many disease states (e.g. asthma, ulcerative colitis). They are used for their anti-allergic and anti-inflammatory action. The output of the adrenal gland is controlled by the pituitary gland, which secretes the hormone ACTH (adrenocorticotrophic hormone). This is secreted in response to the circulating steroids. If the steroid level in the blood is low, then more ACTH is secreted and the adrenal gland is stimulated to put out more steroids. If the steroid level is high (for instance, if the patient is on steroid therapy) the output of ACTH falls and the adrenal gland can atrophy. In a stressful state (e.g. anaesthesia or surgery) this gland cannot increase its output of steroid as demanded, so the blood pressure falls and the patient may collapse. Adrenal reserve is diminished in Addison's disease, after bilateral adrenalectomy and during current or previous steroid therapy.

GUIDELINES FOR THE USE OF STEROID THERAPY PERI–OPERATIVELY

- Check carefully whether patient has a history of steroid therapy. Some diseases are associated with the use of steroids (e.g. rheumatoid arthritis or asthma).
- Patients who have had steroid therapy for longer than 2 weeks in the 3 months before major surgery or are currently taking steroids, should be given steroid cover peri-operatively. Hydrocortisone 25mg 1V is given at induction in the adult patient.

  Patients having minor surgery should not require any further supplement and should then continue with their regular preoperative dose.

  Patients having intermediate surgery. A total of 125mg hydrocortisone should be given on the first day of surgery. If the patient is not currently taking steroids this should be sufficient to cover previous use. Those who are currently taking steroids should then recommence their usual dose.

  Patients having major surgery involving no oral intake post-operatively. Patients currently taking steroid therapy should be continued on hydrocortisone 100mg / day (25mg 6hrly IV). When oral intake recommences they should be restarted on their previous dose of prednisolone.

ADRENAL MEDULLA

The adrenal medulla is responsible for the synthesis and excretion of catecholamines. (See Chapter 5 The Autonomic nervous system).

The output from the medulla is mainly adrenaline (80-90%) with noradrenaline (10-20%). It also produces dopamine. The medulla acts as a sympathetic ganglion and has no parasympathetic innervation.

CHAPTER 41

ANAESTHESIA AND OBESITY

Outline:

- Problems
  - Mechanical
  - Underlying disease
  - Other
- Anaesthetic Management

PROBLEMS PRESENTED BY THE OBESE PATIENT

Obesity is an increasing problem. The obese patient presents for two types of surgery:

- Surgery not necessarily related to the obesity e.g. appendicectomy, orthopaedic and trauma surgery. This group of patients is of practical interest as these conditions are more often seen in the smaller hospital.
- Surgery performed to correct obesity, e.g. gastric stapling.

MECHANICAL PROBLEMS

Mechanical problems are more common in the morbidly obese patient.

In the Ward: Weighing the patient.

Transport to theatre: The trolley may not be wide enough.

In the operating theatre:

- The table may not accommodate the patient.
- The blood pressure cuff may not be wide enough.
- Venepuncture is difficult.
- Regional techniques are difficult because landmarks are not palpable, the needles used are not long enough and positioning the patients for regional anaesthesia is more difficult.
- Maintaining a clear airway is difficult.
- Ventilation by mask is difficult.
- Intubation is more difficult.
- Positioning the patients for surgery is more hazardous. The supine position interferes with the movement of the diaphragm.
- The prone, lithotomy and Trendelenburg positions aggravate this further.

PROBLEMS RELATED TO UNDERLYING DISEASES

- Hypertension, ischaemic heart disease, cardiac arrhythmias and cardiac failure.
- Chronic respiratory disease, chest infections.
- Hiatus hernia, which increases the tendency to regurgitation and aspiration of gastric contents.
- Diabetes mellitus.
- Deep vein thrombosis and pulmonary embolism.
- Endocrine problems, e.g. thyroid, or adrenal disease.
- Psychological problems are common. Obese patients can suffer from severe depression and loss of self-esteem, behind a happy exterior. Their feelings must be handled sensitively.

OTHER PROBLEMS

- Post-operative complications are more common, especially respiratory complications.
- The surgeon encounters problems operating on an obese patient.
  - Longer operating time
  - Large incisions
  - Poor visibility
  - More bleeding

ANAESTHETIC MANAGEMENT

General guidelines

If possible a regional technique, although technically more difficult, is ideal. Long spinal needles may need to be used.
If a general anaesthetic technique must be used, follow these principles:

- A good IV line
- Strict maintenance of the airway, so intubation is advisable even for brief surgery
- Adequate muscle relaxation
- Adequate ventilation both during and after surgery
- Adequate oxygenation
- Complete reversal of the relaxant, plus minimal post-operative respiratory depression
- Adequate post-operative analgesia so that respiration is not inhibited by pain
- Post-operative chest physiotherapy
- Post-operative oxygen
- Post-operative monitoring
- Early post-operative ambulation

Summary of anaesthetic technique

Pre-operative

- Carefully assess and treat any medical problems associated with the obesity.
- Attempt to help the patient lose weight.
- Arrange pre-operative chest physiotherapy.
- Give pre-operative medication: subcutaneous heparin, benzodiazepines, opiate or atropine where appropriate.
- Ranitidine or omeprazole, if available, should be given pre-operatively and/or 30ml sodium citrate 0.3M orally in the anaesthetic room.

Intra-operative

- Preoxygenate, then induce with thiopentone, ketamine or propofol.
- Give suxamethonium and while it takes effect ventilate with 100% oxygen. Cricoid pressure should be used if possible to help avoid aspiration.
- Intubate. Have an introducer or a gum elastic bougie available for the endotracheal tube. Many obese patients will be difficult to intubate so an alternative method would be to use an inhalational induction and intubate under deep inhalational anaesthesia.
- The LMA is not suitable for obese patients except for very short procedures as a good seal cannot be achieved.
- Maintain adequate ventilation using a non-depolarising agent. The dose of non-depolarising agents and opiates should be calculated on an estimated lean body weight rather than actual weight.
- Give careful attention to positioning and monitoring. Protect limbs and parts of the body which may fall over the edge of the theatre table.
- Perform complete reversal and extubate when the patient is fully awake. Extubate in sitting or semi-recumbent position.

Drug dosage can be difficult to calculate accurately on a weight basis. The dose of water-soluble drugs needs to be reduced on a mg/kg basis and prolonged use of fat-soluble drugs can delay recovery.

Post-operative

- Check to see if ventilation is adequate. If not, continue mechanical or assisted ventilation post-operatively.
- Supervise chest physiotherapy carefully.
- Subcutaneous heparin is advised to prevent deep venous thrombosis.
- Early ambulation.

CHAPTER 42

ANAESTHESIA AND BURNS

Outline:

Points of importance to the anaesthetist

Indications for surgery in the burned patient

Initial management of the burned patient

Anaesthesia for the burned patient

POINTS OF IMPORTANCE TO THE ANAESTHETIST

- Reduced circulating blood volume. There is a loss of water and protein from the circulation, so IV fluids, both crystalloids and colloids, may be required.
- A fall in body temperature due to the loss of a large amount of skin.
- Anaemia. Deep burns result in haemolysis of red blood cells.
- Hypotension may be associated with the hypovolaemia and may also be due to the toxins released from the damaged tissues.
- Renal failure may follow hypovolaemia and hypotension.
- There is a constant danger of infection in the burned patient. If infection occurs the patient becomes debilitated.
- Airway damage, which may involve the upper or lower airways. It may be due to the inhalation of hot gases, fumes, steam, smoke, etc. Upper airway obstruction will increase over 6 to 24 hours, so intubate the patient or consider early tracheostomy depending on nursing care facilities.
- Pulmonary oedema may be caused by inhalation of noxious fumes especially if the patient was in a closed space. Face, neck, nose and mouth burns are commonly associated with this type of trauma. Pulmonary oedema may also occur during resuscitation from fluid overload. Dyspnoea and tachypnoea are early signs.
- Respiratory infection.
- The danger of hyperkalaemia. Burned patients should not be given suxamethonium compounds after 48 hours and for up to 1 year after a significant burn injury. The rapid rise in serum potassium during the depolarisation of the muscle with suxamethonium has been known to produce cardiac arrest.
- Venepuncture is difficult.
- Using a mask is difficult in patients with facial burns.
- Maintaining a clear airway is difficult in patients with neck contractures.
- Monitoring is difficult in patients with burns to the upper half of the body.
- The problem of multiple anaesthetics and the danger of repeated use of the same anaesthetic agents.
- Fear and depression. The anaesthetist needs to establish rapport with the patient and reassure them.

INDICATIONS FOR SURGERY IN THE BURNED PATIENT

- Debridement
- Tracheostomy
- Fasciotomy
- Skin grafts
- Reconstructive surgery to improve function and cosmetic appearance.

INITIAL MANAGEMENT OF THE BURNED PATIENT

- **Assess the depth of the burns**

  First degree burns affect the epidermis (superficial tissues). The burns are painful, red and dry but they do not blister.

  Second degree burns blister and are painful, mottled, red and moist.

  Third degree burns reach the subcutaneous tissue. Fat, muscle and even bone may be destroyed.

  The critical burn area is that percentage of the body surface above which the patient cannot compensate for fluid loss. When the burn area is greater than 15% in an adult or 10% in a child the patient can no longer compensate and IV fluids will be required.

- **Assess the extent of the burns**

  The "Rule of Nines" is commonly used to obtain the percentage of the surface area affected.

Area	Adults	Infants and young children
Whole head	9%	18%
Each upper limb	9%	9%
Front of trunk	18%	18%
Back of trunk	18%	18%
Each lower limb	18%	14%
Perineum	1%	1%

In an adult the palm of the hand is approximately 1% of surface area.

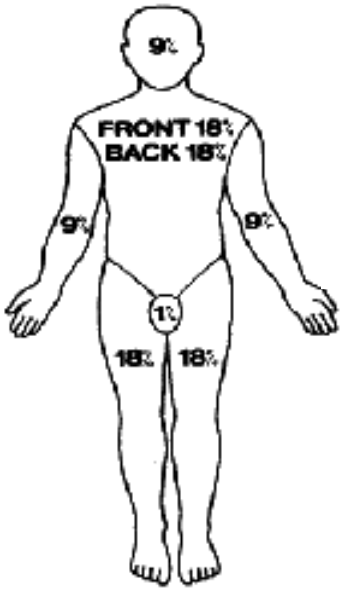


Fig 42.1

The "rule of nines" for rapid assessment of % body surface area

- **Weigh the patient if possible**, otherwise estimate weight.

- **Calculate intravenous fluids** according to the formula 2ml x body weight in kilograms x percentage of surface area burnt. This gives the requirement for the first 24 hours. Half of the calculated volume can be given as colloid and half as crystalloid solution. Half the calculated volume can be given in the first 8 hours and the rest in the next 16 hours.

  In addition to these, the volume lost by nasogastric suction is replaced with 0.9% saline or 0.45% saline. Give extra fluid to compensate for fluid loss from diarrhoea, vomiting and pyrexia.
  Use a large bore cannula, inserted under sterile conditions, for administering the fluid.

  Re-assess fluid requirements at regular intervals.
  Maintain a urine output of 1ml /kg /hour. After 24 hours the volume of fluid given may be reduced to about half that given during the first 24 hours.
  Anaemia may be corrected by the use of blood after the blood volume is first restored with other fluids.

- **Give analgesics if the patient is in pain:**
  - Give the drugs IV, not intramuscularly. The muscle blood flow is reduced in the shocked patient.
  - Use adequate doses of drugs. These patients often develop tolerance and have increasing requirements for opioids, which should not be withheld. Sedatives and anxiolytics may also be useful.
  - Clean and manage the burn with a dressing or by open exposure. Ketamine 0.5- 1.0 mg/kg IV is useful for dressing changes.
  - Use silver sulphadiazine cream to reduce bacterial contamination.

- **Continue to monitor the following:**
  - Pulse
  - Blood pressure
  - Temperature
  - Urine output
  - Serum electrolytes
  - Haemoglobin and haematocrit

ANAESTHETIC TECHNIQUES FOR BURNS PATIENT

General Anaesthesia

Intubation with a muscle relaxant and IPPV. Endotracheal anaesthesia is useful for surgery in the area of the head and neck. It is also useful for prolonged surgery and surgery performed in abnormal positions. It ensures adequate ventilation. Care is needed however with the use of muscle relaxants such as suxamethonium.
Rapid sequence induction is not advised because of the dangers of suxamethonium.
Once healing of burns has begun fibrosis and strictures may make laryngoscopy impossible.
Tracheostomy is not desirable because of the danger of infection and awake intubation using local anaesthesia may be necessary.

Mask (inhalational) anaesthesia. Inhalational anaesthesia is useful for short procedures performed in the supine position, in a patient with a secure airway but may be difficult in patients with facial burns.
Remember that peripheral vasodilatation increases heat loss and post-operative shivering may cause movement of the graft. However, peripheral vasodilatation may help with venepuncture.

Ketamine (Bolus or Infusion)

The choice of the technique will depend on the site of the burn, the posture required for surgery and the nature and duration of surgery. Absorption of the ketamine (IM) may be delayed in burns resulting in a slower than normal recovery.
The use of ketamine has been described under Techniques of anaesthesia in Chapter 14. Ketamine is useful for debridement and grafting of facial burns, where the use of a mask is impossible. It is also useful for the division of neck contractures prior to intubation. The burned patient with neck contractures and therefore possible airway obstruction will have to be treated with special care. This is discussed under anaesthesia in the presence of respiratory obstruction in Chapter 8 (The Airway and its maintenance).

CHAPTER 43

ANAESTHESIA AND SOME UNCOMMON DISEASES

Outline:

Tetanus

Acute intermittent porphyria

Poliomyelitis

Myasthenia Gravis

TETANUS

Tetanus is a disease caused by a gram-negative organism, Clostridium tetani, found in soil and faeces. These organisms release toxins, which act on the anterior horn cells of the brain stem and spinal cord. The incubation period for tetanus is 5 days to 15 weeks.
Spores can infect wounds, cuts or any break in the skin which comes into contact with the soil.
Neonatal infection occurs when the umbilical cord is cut with a dirty knife or scissors or when the stump is coated with soil or dung.

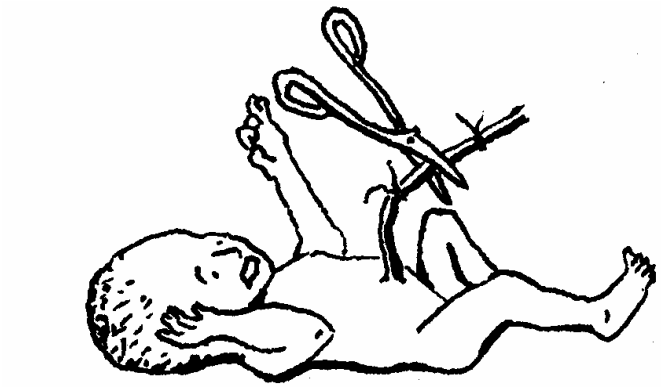


Fig 43.1 Cutting the umbilical cord

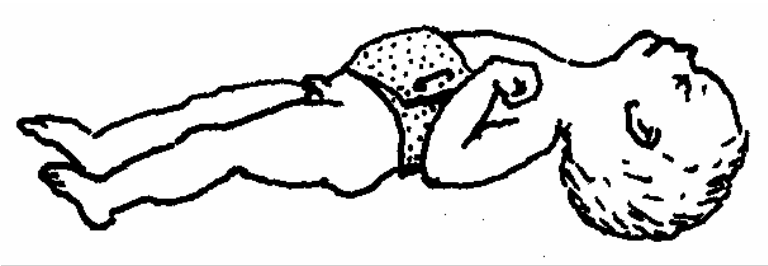


Fig 43.2 Neonate with muscle spasm (Opisthotonus)

Signs and Symptoms

- **Muscle Spasm**
  - Jaw stiffness (trismus)
  - Spasm of facial muscles (risus sardonicus)
  - Convulsive body spasms
  - Dysphagia
  - Respiratory distress if the spasm involves the respiratory muscles
  - There may be a history of injury.

- **Autonomic**
  - Labile hypertension
  - Tachycardia
  - Sweating
  - Vasoconstriction

PREVENTION OF TETANUS.

Patients who have already been immunised with a course of tetanus toxoid

- Give a booster dose (0.5ml) of tetanus toxoid if >5 years since immunisation.
- Debride the wound.
- Give procaine penicillin.

Patients who have not been immunised

- Give human tetanus immune globulin (if available) 200-500 units IM.
- If human tetanus immune globulin is unavailable, give anti-tetanus serum, 3000-5000 units IM after a sensitivity test.
- Start active immunisation with tetanus toxoid as well.
- Debride the wound.
- Give penicillin 1.2 mega-units 6 hrly for 10 days
  and metronidazole 500 mg IV 6 hrly for 10 days.

TREATMENT OF TETANUS

Prompt diagnosis and treatment are essential

Principles of management

- Control the muscle spasm
- Attend to the airway and ventilation
- Debride the wound to prevent the release of further toxins
- Neutralise toxins
- Control autonomic manifestations
- Prevent complications.
- Reduce external stimuli

Here are some details for practising these principles.

Controlling muscle spasm

Patients fall into two broad groups:

Mild spasm These patients respond to Valium (diazepam) 0.1 mg/kg IV in repeated doses. Other drugs that can be used are chlorpromazine or thiopentone, in small doses.

Severe spasm

- Those with respiratory embarrassment due to muscle spasm.
- Those with laryngeal and pharyngeal muscle involvement.
- Those whose muscle spasm cannot be controlled with anticonvulsant drugs such as diazepam. These patients require to be paralysed, so that a nasotracheal tube can be inserted. The relaxant used will depend on what is available. Pancuronium or other non-depolarising agent in the usual doses may be used, along with the necessary sedative drugs, e.g. diazepam.

Paralysis of the patient requires the following

- The use of a ventilator.
- Adequate medical gas or an air generator to "run" the ventilator.
- 24 hour supervision by personnel who understand the working of the ventilator and the dangers of endotracheal intubation.
- In the absence of a ventilator, it may be necessary to hand ventilate the patient using a self-inflating non-rebreathing valve and bag. This has been done in places where ventilators have not been available and the ventilation has saved lives. It requires careful planning. Entrust the task to a team of two nurses working together over a six-hour shift. They must be fully aware of the dangers and possible complications, the monitoring requirements and the nursing care. It is important that free communication and advice is available from the person co-ordinating treatment. At the end of the six-hour shift another two nurses take over.

This is only possible if short-term ventilation, e.g. up to 48 hours, is contemplated. Those on duty must keep careful charts of the frequency and duration of muscle spasm. When the patient is free of spasm for 24 hours the dose of drugs can be reduced.

Attention to the airway

- In the mildly affected patient, keep the airway clear either with or without an oropharyngeal airway.
- In the more severely affected patient use endotracheal intubation.
- Perform tracheostomy early, as a non-urgent procedure.

Management of the wound

- Debride the wound.
- Give antibiotics : Metronidazole 500mg 6 hourly or benzyl penicillin, 2 million units 4-hourly for 10 days. In penicillin-sensitive patients use erythromycin or tetracyclines.
- If the patient is in pain as a result of the wound, small doses of analgesics may be given.

Neutralisation of toxins

- Give tetanus immune globulin (human antitoxin) 3000-6000 units IM or slow IV.
- If this is unavailable use anti-tetanus serum (equine), in a dose of 10,000 units IV and 10,000 units IM. Human antitoxin has the advantage of being virtually reaction free and it persists in the circulation for much longer (over 30 days).
- Active immunisation with the toxoid should be started at the same time. Tetanus does not confer immunity.

Control autonomic manifestations

- Variations in blood pressure. Severe hypertension or hypotension may occur.
- Hyperpyrexia
- Variations in pulse rate. Tachycardia, bradycardia or various arrhythmias may occur.

Decrease external stimuli

As far as possible reduce noises and lights. Keep procedures to a minimum.

Prevention of complications
Monitor the patient carefully, giving meticulous nursing care and physiotherapy. Complications need to be recognized early and treated immediately:

- Chest complications such as sputum retention, atelectasis, bronchopneumonia.
- Paralytic ileus and gastric distension usually last for 2 or 3 days and are treated by nasogastric drainage and replacement of fluid loss. The needs of the unconscious patient must be attended to. This includes IV fluids and nasogastric feeding,

ANAESTHESIA FOR THE PATIENT WITH TETANUS

Indications
Tetanus patients need anaesthesia for debridement of the wound, treatment of any primary injury such as fracture and for tracheostomy.

Assessment
The patient must be assessed carefully to ascertain:

- The degree of respiratory embarrassment, secondary to the muscle spasm
- The presence of any respiratory complication, e.g. bronchopneumonia
- The presence of gastric distension
- The presence of cardiovascular complications
- Drugs which may already have been administered, e.g. chlorpromazine and diazepam, potentiate anaesthesia.

Management

- Secure a good intravenous line.
- Pre-medication: Give chlorpromazine or diazepam intravenously with a small dose of atropine, (e.g. 0.4 mg) in the adult patient.
- In the mildly affected patient with no gastric distension a mask anaesthetic may be used for wound debridement e.g. thiopentone or diazepam induction followed by air or nitrous oxide, oxygen and volatile. In the absence of volatile and nitrous oxide, IV diazepam and ketamine may be used.
- The severely affected patient may be brought to theatre intubated and paralysed. If the patient is not intubated and there is gastric distension or bleeding through the nasogastric tube, an endotracheal tube should be inserted. A rapid sequence induction is required with cricoid pressure. Thiopentone or diazepam should be used as an induction agent and suxamethonium as the relaxant. In later stages of the disease suxamethonium has been known to cause cardiac arrhythmias secondary to hyperkalaemia. Maintain the anaesthetic with a non-depolarising relaxant and air or N_2O /oxygen/volatile.

ACUTE INTERMITTENT PORPHYRIA

This is a congenital metabolic defect resulting in an overproduction of porphyrins which are iron containing compounds involved in biosynthesis of haem. In porphyria the excess porphyrins accumulate in tissues particularly neurological tissue. The liver is affected in many forms of porphyria and crises can be precipitated by a number of drugs commonly used in anaesthesia. In the absence of full biochemical and urine testing the condition is characterised by urine that turns red on standing. It is possible that a patient who is known to have the disease will require an anaesthetic. Acute intermittent porphyria may also present as acute abdominal pain and the patient may need a laparotomy to rule out a surgical condition.

When a patient presents with acute abdominal pain and also has signs of a neurological disorder, it is wise to think of acute intermittent porphyria. The main danger of this condition from the anaesthetist's point of view is that anaesthetic drugs can lead to partial or total paralysis.

Barbiturates (e.g. thiopentone) are strictly contraindicated. **They must never be used under any circumstances in these patients**.

Propofol and ketamine have been used without problems.
Opioids, suxamethonium and vecuronium are also considered safe. Pancuronium should be avoided.
Adverse reactions have been reported with diazepam, phenytoin, ergotamine and sulphonamides but volatile anaesthetics can be given with the exception of enflurane.

Regional techniques are avoided by many anaesthetists in these patients because of a risk of peripheral neuropathy but spinal anaesthesia has been used. Bupivacaine is the recommended local anaesthetic agent. Lignocaine should be avoided.

The operation itself may precipitate an attack.
Acute porphyria should be considered in the differential diagnosis if a patient remains paralysed or comatose after surgery.

Drugs which are considered safe to use

For ***induction***	Propofol
For ***maintenance***	Nitrous oxide Oxygen Ether
For ***muscle relaxation***	Suxamethonium Vecuronium
Reversal agents	All
For ***analgesia***	Aspirin Morphine Pethidine Fentanyl
For ***local anaesthesia***	Bupivacaine

Probably safe	**Drugs which should be avoided**
Ketamine Lignocaine Halothane Isoflurane Sevoflurane Pancuronium Lignocaine Diazepam	Thiopentone Etomidate Pentazocine Enflurane

POLIOMYELITIS

The help of the anaesthetist is important in managing polio patients.

Polio can be acquired by droplet infection through the respiratory tract or through ingestion. It has an incubation period of 5 to 35 days.

Symptoms

- Fever, nausea and vomiting, sore throat.
- Headache, stiff neck, muscle pains and stiffness.
- Paralysis associated with muscle wasting. Depending on the area of the spinal cord involved, the muscle weakness will give rise to different symptoms. If the spinal cord alone is affected, the patient may have asymmetrical weakness of the limb muscles and also the intercostal muscles. If the brain stem is affected, then the patient will have dysphagia (difficulty in swallowing), dysphonia (difficulty in speech), regurgitation of fluids, facial weakness and paralysis of the soft palate.

There is no sensory loss.
Paralysis when present is established within 2-3 days and does not progress after the temperature has come down.

TREATMENT OF POLIOMYELITIS

General measures

- Rest.
- Analgesics for muscle pain.
- Antibiotics and physiotherapy for upper respiratory infections.
- Specialised treatment depending on the muscles involved.
- Rehabilitation.

Patients can be classified into three categories:

- **Spinal** These patients can swallow but cannot breathe because the intercostal muscles are affected.

  Treatment: Iron lung or any other available ventilation.

- **Bulbar** These patients can breathe but cannot swallow, because the brain stem is affected, not the spinal cord.

  Treatment: Endotracheal tube or tracheostomy. Head down position and suction.

- **Bulbospinal** Here the patients can neither swallow nor breathe, because both the spinal cord and the brain stem are affected. They will need both intubation and ventilation.

Drugs used for ventilating a polio patient

Try sedatives first, e.g. morphine, diazepam. If they fail then use non-depolarising muscle relaxants.

Recovery occurs over weeks or months with some return of function but many patients will be left with weakness. If there is no recovery by 6 – 18 months, permanent paralysis is likely.
There is a mortality rate of 5-20% in paralysed patients.

MYASTHENIA GRAVIS

Myasthenia gravis is a disorder affecting muscles, specifically the neuro-muscular junction, at the level of the motor end plate of the skeletal muscles. It is an auto-immune condition with antibodies to acetylcholine receptors resulting in a decreased number of active receptors at the neuromuscular junction. The patient presents with weakness and easy fatigue of the voluntary muscles. The disease involves the limb muscles, the facial and eye muscles. Later, swallowing and speech are affected. The biggest danger of this condition is the development of respiratory failure.

Diagnosis

This is usually made on clinical grounds but can be confirmed by the observation of an improvement in muscle power within 10-30 seconds of giving edrophonium 2-5 mgs IV. The improvement should last for about 5 mins. If edrophonium is not available neostigmine 1-2mg IM may also produce a positive response. Before the test a small dose of atropine should be given to counteract the muscarinic side effects of edrophonium.

Treatment of myasthenia gravis

A life of restricted activity is necessary. During attacks, complete rest is essential. Treat any infection, especially respiratory infections, rigorously and watch for involvement of the respiratory muscles. Drug treatment is pyridostigmine (an anticholinesterase) and steroids. Surgical removal of an enlarged thymus gland has been helpful in some cases.

ANAESTHESIA AND THE MYASTHENIC PATIENT

Patients with myasthenia gravis can present for surgery for a variety of reasons. A frequent reason is an enlarged thymus gland, so they may present for thymectomy. They may also present for surgery which is quite unrelated to the disease, e.g. for an appendicectomy.

Potential anaesthetic problems

- Abnormal sensitivity to muscle relaxants. Both depolarising and non-depolarising relaxants must be avoided.
- Narcotics (e.g. pethidine and morphine) and sedatives (e.g. barbiturates) must be used very carefully and in small doses.
- Ether is best avoided because of its neuromuscular blocking acting and its tendency to cause secretions.
- These patients, when treated with anti-cholinesterase drugs, can produce a lot of secretions which interfere with anaesthesia.
- Some myasthenics may have involvement of the oropharyngeal muscles (bulbar symptoms). Myasthenics are thus at risk of reflux and cricoid pressure should be used on induction.

Anaesthetic technique

Regional anaesthetics are the method of choice and should be used in preference to a general anaesthetic whenever possible although the dose of local anaesthetic drugs should be reduced due to their neuro-muscular blocking action.
If a general anaesthetic is required, use the following technique:

Pre-operative management

- Careful assessment of respiratory function and severity of muscular weakness. Any respiratory infection should be treated before surgery. Check that serum K+ is within normal limits as hypokalaemia may exacerbate muscle weakness.
- ***Premedication:*** Atropine. A small dose of opiate may also be used, especially if the patient is in pain.

Intra-operative management

- ***Induction:*** A small dose of thiopentone or propofol may be used and the anaesthetic deepened with nitrous oxide, oxygen and volatile.
- ***Intubate*** while the patient is breathing spontaneously under a volatile agent.
  Note: If suxamethonium has to be used for intubation, half the usual dose should be given although some patients may be resistant to suxamethonium. It is best avoided. If muscle relaxation is required a small dose of short acting non-depolarising agent can be given e.g. vecuronium or atracurium.
- ***Maintenance:*** Use nitrous oxide/oxygen and halothane.

Post-operative management

- Observe respiration. Respiratory support in the form of assisted or controlled ventilation may be necessary.
- Support the circulation with fluids.
- Monitor the patient's progress carefully.
- A small dose of opiate may be given.

MYASTHENIC SYNDROME

This is found in patients who have carcinoma of the bronchus or secondaries from carcinoma elsewhere. These patients are extremely sensitive to both depolarisers and non-depolarisers.

CHAPTER 44

ANAESTHESIA AND AIDS

Outline:

Introduction

Tests for AIDS

Management of the HIV +ve and AIDS patient presenting for anaesthesia

Example of a hospital protocol for health care workers

INTRODUCTION

Acquired immunodeficiency syndrome (AIDS) is a disease which indicates an underlying cellular immune deficiency.

AIDS is caused by a group of retroviruses, the human immunodeficiency viruses (or HIV) the two most common of which are HIV 1 and 2.
The viruses attach to the T-lymphocytes (CD4 protein bearing cells) which are responsible for the body's immune response. The reduction in numbers of T-lymphocytes results in a significant immuno-suppression leading to a variety of infections during the later stages of the disease.

Clinical picture

The natural history of the disease is poorly understood. A few weeks after infection, the virus causes an acute illness of short duration similar to glandular fever or 'flu'. This is followed by a period with no symptoms, lasting months or years. This may then be followed by one of two clinical courses:

- a period of generalised lymph gland enlargement, weight loss, fever, diarrhoea, progressing to encephalopathy **or**
- a state of immune deficiency resulting in unusual malignancies (e.g. Kaposi Sarcoma) or unusual infections (e.g. Pneumocystis carinii pneumonia).

The virus infects the T-lymphocyte cells in all lymphoid tissues of the body such as the peripheral blood, lymph nodes, spleen and brain.

Transmission

The transmission of AIDS is by blood and semen and from mother to child through breast milk. Apart from sexual transmission, needle injuries, contamination of cuts, sores and abrasions, may be responsible for the spread of AIDS. Transmission by saliva, airborne droplets or through contamination of intact mucous membrane is unlikely but is not yet disproved.
The incubation period is not known and the carrier state may last for life.

It is believed that 4%-19% of sero-positive patients develop AIDS within 5 years. The following groups of patients have the highest risk of contracting AIDS:

Homosexuals and bisexuals
IV drug users
Haemophiliacs
Female sexual partners of men at risk
Children of affected mothers
Patients of Haitian or Central African origin

TESTS FOR AIDS

No test is yet available for the antigen or virus. Antibody tests are available for HIV-1 + HIV-2 and antibodies are usually present by 3 months post infection. AIDS can be diagnosed by a +ve antibody test and a falling CD4 count along with some clinical conditions characteristic of the disease. Not all sero-positive patients will progress to AIDS. It is important to remember that there will always be a certain percentage of patients who appear negative to the antibody test but who are infected with the virus and have not yet developed the antibody (called false negatives). Some workers also suggest that false positives can be found in patients suffering from other non-HIV debilitating infections and repeat testing is recommended before diagnosis is confirmed.

MANAGEMENT OF THE HIV +ve OR AIDS PATIENT PRESENTING FOR ANAESTHESIA

Patients with a positive antibody test and patients with frank AIDS should be managed in the same way.

Pre-operative visit

There is no risk from casual contact except in patients suffering from frank haemorrhage from any cause.

The patient's respiratory, gastro-intestinal and neurological function should be checked.

All patients should be treated as infection risks and "universal precautions" should be taken peri-operatively.

- Avoid contact with blood, sputum, urine, etc. Use protective covering, e.g. gowns, gloves, eye protection (goggles).
- After contact wash hands thoroughly with soap and water.
- Use the AIDS barrier nursing techniques for severely affected AIDS patients, to avoid the spread of infection to and from the patient.

Intra-operative management

- Keep equipment to a bare safe minimum.
- Use disposable equipment wherever possible.
- Sterilise all other equipment according to the recommended procedure (see Chapter 60).
- The personnel in theatre should wear protective gowns, gloves, caps, boots and goggles. Consider plastic aprons as well.
- Take great care with inserting intravenous needles to avoid spillage.
- Dispose of contaminated sharps into rigid-walled, puncture resistant containers.
- Take care to minimise contamination with saliva and blood during intubation and extubation.

- Don't replace equipment, once used, on the clean anaesthetic trolley. Put it in a tray kept for the purpose.
- Contaminated linen should be double bagged. Wear gloves for this procedure.
- Specimens from theatre should be taken out of theatre in a waterproof bag or container with an appropriate warning label.
- Wash hands thoroughly with soap and water.

It is important to note that the AIDS virus attacks the cells of the central nervous system. About 20% of patients with AIDS develop spinal cord involvement. This calls for extra caution when spinal anaesthetics are used in AIDS patients.

The use of blood and blood products still carries a definite risk. All donors are tested for HIV1 and 2 antibodies. Some products (e.g. immunoglobulin, factors VIII and IX) are pretreated in an effort to inactivate the virus.

Post-operative care
Take the same precautions as for intra-operative management.

EXAMPLE OF A HOSPITAL PROTOCOL FOR HEALTH CARE WORKERS

Should accidental contamination occur the hospital should have a policy for post-exposure prophylaxis for its staff.

Exposure may be with blood or body fluids from needle-stick injury, cuts, broken skin and splashes in eyes, nose and mouth.
Remember that even significant exposure only carries a 1:200 risk of infection.

- Rinse/ wash with water or soap and water.
- Worker should be advised that taking anti-retroviral drugs for 1 month further reduces the risk of infection.
- Before treatment both patient and worker should have an HIV test. Depending on the result prophylaxis may not be necessary.
- If treatment is being given the health care worker should be advised to abstain from or use protection during sexual intercourse for 6 months until confirmation of a negative test.
- Post-exposure prophylaxis should be started as soon as possible ideally within 2 hours of exposure but not after 72 hours.
- Follow-up testing should be carried out at 6 weeks, 3 months and 6 months and if negative the worker can be assumed to be clear of infection.

CHAPTER 45

ANAESTHESIA FOR SURGICAL EMERGENCIES INCLUDING MAJOR TRAUMA

Outline:

Assessment of a patient presenting with major trauma

- General assessment: airway, breathing and circulation
- Systematic assessment

Summary of anaesthetic problems

Anaesthesia for the patient with multiple trauma

Special problems associated with spinal cord injuries

INTRODUCTION

The term "emergency surgery" implies that the time available to prepare the patient for surgery is limited i.e. the outcome of the surgical procedure depends on how soon it is done. Acute internal bleeding (especially into the abdomen or chest), a rising intracranial pressure, visceral injury, foetal distress and open wounds often associated with compound fractures are the kinds of emergencies we are considering.

ASSESSMENT OF THE PATIENT PRESENTING WITH MAJOR TRAUMA

General Assessment

Before starting a detailed assessment of the underlying injuries, it is essential to make a swift assessment of the patient's airway, breathing and circulation.

The airway: Any obstruction of the airway must be detected and corrected. This has been dealt with in detail in Chapter 8.
At a pharyngeal level, the tongue falling back, blood clots, secretion, food, dentures and foreign bodies are the usual causes. Marked surgical emphysema of the face and neck may suggest a ruptured trachea.
Similarly, bleeding into the neck and severe tenderness over the thyroid cartilage may suggest a ruptured larynx.

Breathing: The patient may be given oxygen by mask until the general assessment is finished. If the patient is not breathing, intubate and ventilate with 100% oxygen. If the patient is breathing but the breathing is shallow, respiration may be assisted with a mask or endotracheal intubation may be required.

Circulation: Note the pulse and blood pressure. Insert a 14G or 16G cannula and start an initial infusion with Hartmann's solution followed by a colloid solution.
Send blood for grouping and cross-matching.
If the facilities are available, measure arterial blood gases. This assesses the respiratory and metabolic state of the patient.

Systematic assessment

Evidence of intracranial injury: Careful neurological observation is essential, especially of the level of consciousness and the size of the pupils. All precautions must be taken to avoid a rise in intracranial pressure, as outlined in Neurosurgical anaesthesia in Chapter 22.
Record the patient's Glasgow Coma score

Spinal cord injury: Acute compression of the spinal cord requires very careful positioning. In trauma patients a cervical collar should always be used until clinical and X-ray evidence confirms that there is no cervical cord injury. A lateral x-ray of the neck is essential in patients with multiple trauma.

Facial and neck injuries: Airway obstruction is the major problem. An awake intubation, or tracheostomy may be needed before anaesthesia is started. If airway obstruction has not been evident in the general assessment but facial and neck injuries make it likely - then an inhalational induction and intubation will be safer than using relaxants.
Bleeding into the airway requires careful suction.

Have a range of tubes available. A smaller tube than usual may be necessary.

Injuries to the structures of the chest:

- Tears to the trachea are associated with subcutaneous emphysema of the face and neck. The airway may be secured by passing an endotracheal tube beyond the tear and inflating the cuff. If this is not possible, surgical exploration is indicated.
- Injury to the chest wall may result in a flail chest which needs to be treated by intubation and prolonged IPPV or if this is not available some other means of immobilizing the segment. Local anaesthesia and epidural anaesthesia where available are also used and may avoid the need for artificial ventilation in less severe cases.

  Flail chest occurs when there are multiple fractures of adjacent ribs causing a segment of the chest wall to become free. This segment will move paradoxically with respiration and this movement along with underlying lung contusion will usually result in pain and hypoxia.

- Bleeding into the pleural cavity (haemothorax) may require drainage.
- Air in the pleural cavity (pneumothorax) may require under water drainage. An x-ray will confirm the diagnosis. See Chapters 27 and 46 for water-seal drains.
- Direct lung trauma may result in contusion of the lung or rupture of a bronchus.
- Widening of the mediastinum seen on x-ray may suggest that the aorta or pulmonary artery has been damaged.
- Crush injuries of the lower chest may be associated with rupture of the diaphragm, especially on the left.

- Cardiac tamponade is associated with a low blood pressure, jugular distension and muffled heart sounds. If tamponade is severe, the pericardium should be tapped before the anaesthetic is administered. Avoid myocardial depressants.

Abdominal injuries: Explore any penetrating injuries in relation to the abdomen. The following may lead to suspicion of an abdominal injury:

- Persistent hypotension despite apparently adequate resuscitation. In this situation bleeding may occur from the liver, spleen or a major blood vessel.
- Increasing size in the abdomen after resuscitation may suggest intra-abdominal bleeding.
- Guarding or re-bound tenderness may be associated with intra-abdominal bleeding or soiling of the peritoneal cavity from a ruptured bowel.
- Haematuria suggests kidney or renal tract injury.

Orthopaedic injuries: Orthopaedic injuries that constitute an emergency are acute fractures, especially those associated with arterial obstruction and open wounds.
Orthopaedic injuries, both bony and soft tissue, can be associated with a large but relatively hidden blood loss.
This is discussed under Anaesthesia for Orthopaedic surgery in Chapter 31.

Resuscitation: This may involve care of the airway, artificial ventilation and cardiac compression and treatment of the shocked patient, which are described in detail in Chapters 52 and 58

SUMMARY OF ANAESTHETIC PROBLEMS

All of the following problems have been discussed in detail elsewhere.

- Airway problems. See Chapter 8.
- Hypovolaemia/shock. See Chapter 52.
- Hypoxia - especially due to chest injuries. See Chapter 8.
- Raised intracranial pressure. See Chapter 22.
- Full stomach - danger of regurgitation. See Chapter 16 for RSI and Chapter 46
- Massive blood transfusion. See Chapter 49
- Fat embolism. See Chapter 46

ANAESTHESIA FOR THE PATIENT WITH MULTIPLE TRAUMA

The subject is dealt with extensively under the subject "Shock" in Chapter 52 but note these points:

- Regional anaesthetics, if not otherwise contraindicated, can be used provided that there has been adequate preoperative fluid resuscitation. Surgery should be delayed until this has been achieved unless there is uncontrolled bleeding.
- Spinal anaesthetics are unsafe in hypovolaemic patients whose blood loss cannot be controlled.
- Whenever possible patients should be normovolaemic. Two infusions (14 - 16 G cannulae) should be in progress before anaesthesia is commenced.
- A higher concentration of oxygen (50%) than normal may be needed.
- If there is air or blood in the pleural cavity it should be drained before anaesthesia/surgery. Chest and head injuries must always be looked for in patients with multiple trauma.
- Do not use ketamine if there is evidence of a head injury. If there is no head injury ketamine is an excellent induction drug for the shocked patient.
- IPPV may be necessary after surgery.
- Large volumes of blood will be required. The problems associated with massive transfusions need careful attention. See Chapter 49
- If there is airway obstruction pre-operatively, an “awake intubation” or a tracheostomy may be necessary before the start of anaesthesia.
  If there is no airway obstruction but there is a likelihood of obstruction due to facial and neck injuries, then induction and intubation under inhalational anaesthesia is safer than using relaxants.
- Every case of major trauma must be treated as having a full stomach, because of swallowed blood, delayed gastric emptying time and recent food.
- The hourly urine output, serum electrolytes and acid base status should be monitored if the facilities are available.
- The usual principles of light anaesthesia, using small doses of drugs and high oxygen concentrations, are used.

SPECIAL PROBLEMS ASSOCIATED WITH SPINAL CORD INJURY

- A recent quadriplegic may have an unstable cervical spine and pose intubation problems.
- Muscle spasms may be a problem. Even if the patient has sensory loss at the site of the operation they may still need some form of anaesthesia to control the muscle spasm.
- There is also a tendency to hypertension.
- Temperature regulation is interfered with in quadriplegics.
- Positioning may be difficult.
- Suxamethonium can cause severe hyperkalaemia 48 hours after a spinal cord lesion. It is safe to use within 48 hours of injury but unsafe to use thereafter because of the possibility of a rapid rise in serum potassium. This response can persist for up to 1 year. The increase in serum potassium in these patients may be responsible for cardiac arrhythmias when suxamethonium is given.
- Respiratory problems are more common due to intercostal paralysis and partial paralysis of the diaphragm.
- Chronic infection and nutritional deficiencies lead to anaemia.
- Pressure sores are more common.
- Psychiatric disturbance is likely.

CHAPTER 46

COMPLICATIONS OF ANAESTHESIA

Outline:

Anaesthetic complications are classified according to the system most affected. If the problem mainly involves the heart and circulation it is classified as cardiovascular. Problems involving the respiratory tract or the lungs are classified as respiratory and so on.

- Cardiovascular complications
- Respiratory complications
- Gastrointestinal complications
- Urinary complications
- Neurological complications
- Complications in eye surgery
- Other complications
  - Shivering
  - Awareness during anaesthesia
  - Malignant hyperpyrexia

The following are dealt with in specific chapters:

- Complications of endotracheal intubation: see Chapter 9
- Complications of anaesthetic drugs, including anaphylaxis: see Chapters 6 and 62
- Complications of blood transfusions: see Chapter 49
- Complications of regional techniques: see Chapters 18 and 19

CARDIOVASCULAR COMPLICATIONS

Hypotension

Hypotension under anaesthesia may have several causes. The easiest way to consider these causes is to group them under three headings:

Anaesthetic causes:

- Drugs
- Premedicant drugs, e.g. opioids
- Induction agents, e.g. thiopentone
- Inhalational agents e.g. halothane and ether
- Muscle relaxants, e.g. pancuronium, atracurium
  An overdose or hypersensitivity reaction can produce hypotension.
- Over inflation of the lungs (excessive positive pressure)
- Pneumothorax (see under respiratory complications)
- Hypoxia and hypercarbia in the later stages
- Incompatible blood transfusions
- Spinals or epidurals

Surgical causes

- Position, e.g. reverse Trendelenburg or lateral position
- Blood loss with inadequate fluid replacement
- Vagal stimulation- reflex bradycardia
- Following the release of a tourniquet or clamp
- Embolism, e.g. air or amniotic fluid
- Packs or retractors obstructing the inferior vena cava.

Patient causes (related to the general medical state of the patient)

- Hypovolaemia i.e. blood loss or dehydration
- Heart disease (ischaemic) and heart failure. Arrhythmias: tachycardia and bradycardia
- Pre-operative medication e.g. hypotensive agents, recent steroid therapy
- Supine hypotensive syndrome – see Chapter 21
- Spinal shock, quadriplegia, which often causes variations in blood pressure
- Septic shock.

Marked hypotension (a fall in blood pressure more than 25 mm below the resting blood pressure) and also a hypotensive trend (a gradual decline in the blood pressure) must be treated very seriously. Hypotension if untreated may be followed by cardiac arrest. Even if cardiac arrest does not result, marked hypotension carries the risk of cerebral, myocardial and renal damage following ischaemia or thrombus formation.

There are other factors besides blood pressure involved in delivery of oxygen to the tissues e.g. blood flow in the organ and the oxygen content of the blood but every effort must be made to correct the hypotensive state. This is especially so for the elderly, severely ill and patients with a history of hypertension.

Treatment of hypotension

- Find and treat the cause.
- Start a rapid infusion of intravenous fluids (Hartmann's, saline or colloid) e.g. 10ml/kg stat.
- Increase the concentration of oxygen and reduce the concentration of anaesthetic agent. If the blood pressure is below 80mmHg, then turn off the volatile and give the patient 100% oxygen.
- Use vasopressors to raise the blood pressure when it is dangerously low (below 80mmHg in spite of measures mentioned above). Vasopressors are of most use if the hypotension is due to peripheral vasodilation, e.g. after a spinal or after certain anaesthetic agents. If the low blood pressure is due to haemorrhage or dehydration then they are of temporary use (while fluid is given to replace deficits) as the vessels are already constricted. Similarly, if the hypotension is due to cardiac failure they do not help much.
- The patient's feet can be raised above the level of the trunk to help venous return.

Hypertension

The causes can be classified as follows:

Anaesthetic

- Inadequate anaesthesia and/or intra-operative pain relief.
- Inadequate ventilation resulting in the retention of carbon dioxide. Both hypoxia and hypercarbia may initially present with a rise in blood pressure.
- Certain anaesthetic agents, e.g. ketamine or pancuronium.
- Other less common causes
  - Over transfusion
  - Malignant hyperpyrexia
  - Rare endocrine causes i.e. phaeochromocytoma.

Surgical

- Infiltration with adrenaline. The maximum dose of adrenaline that should be used is 200 micrograms in a concentration of 5 micrograms/ml solution (1 in 200,000).
- Traction on viscera (may cause bradycardia and also hypotension).
- Use of oxytocics, e.g. ergometrine
- Posture: Trendelenburg position
- Clamping of major blood vessels

Patient causes (related to the past history of the patient)

- Pre-existing known hypertension
- Undiagnosed hypertension, e.g. phaeochromocytoma.
- Patients on a group of tranquillisers termed the *monoamine oxidase inhibitors*. (MAOI's)
- Pre-eclampsia
- Full bladder
- Quadriplegia
- Head injury with raised intracranial pressure

Dangers of persistent hypertension during anaesthesia are:

- Cardiac failure – leading to pulmonary oedema
- Cerebrovascular accident (stroke)
- Cardiac arrest
- Myocardial hypoxia
- Cardiac arrhythmias.

Treatment of hypertension during anaesthesia:

- Correct and treat the cause e.g.
  - Deepen anaesthesia
  - Relieve pain
  - Increase ventilation
- Elevate the head of the table
- Drug treatment

If the above measures do not reduce the blood pressure and the diastolic blood pressure persists above 100 mmHg, then a hypotensive agent such as hydralazine (5mg IV) or propranolol (1mg IV) may be used and repeated as necessary. Increase in concentration of volatile agents should be tried first.

ARRHYTHMIAS

Bradycardia

Anaesthetic causes

- Drugs: Suxamethonium
  Neostigmine
  Halothane
  Local anaesthetics
- Reflex bradycardia, e.g. during intubation under light anaesthesia.
- Hypoxia in the late stages (the initial response to hypoxia is tachycardia which may be rapidly followed by bradycardia if the hypoxia is not corrected).
- High spinals

Surgical causes

- Traction on the mesentery
- Traction on the eyeball or carotid sinus
- Neurosurgery
- Anal stretch
- Dilatation of cervix

Patient causes

- Pre-existing heart disease associated with a slow pulse.
- Idiopathic bradycardia - especially in athletes.
- Drugs (pre-operative medication) may give the patient a bradycardia, e.g. digoxin, beta-blockers.
- Hypothermia
- Patients with increased intracranial pressure (late sign).

Treatment of bradycardia

- Find and treat the cause.
- If the pulse rate is less than 60/min and the patient is hypotensive give atropine 0.6mg IV in divided doses. The indication to treat the bradycardia would be its effect on the cardiac output and therefore on the blood pressure. If the bradycardia is associated with a fall in blood pressure, treatment is needed more urgently.

Tachycardia

Anaesthetic causes

- Drugs, e.g. atropine, pancuronium
- Hypercarbia from any cause
- Hypoxia from any cause
- Hypotension
- Inadequate depth of general anaesthetic

Surgical causes

- Infiltration with adrenaline
- Traction on viscera
- Neurosurgical and cardiac surgery

Patient causes

- Cardiac failure
- Thyrotoxicosis
- Fever
- Hypovolaemia
- Pre-existing arrhythmia
- A patient who is very ill or moribund.

The above list refers predominantly to sinus tachycardia. Atrial fibrillation and atrial flutter need to be excluded or diagnosed and treated.

Treatment of tachycardia and other arrhythmias

Find and treat the cause. An ECG is necessary to diagnose the type of arrhythmia. Treatment of the specific arrhythmia must be left in the hands of the doctor.

Cardiac arrest

See Chapter 58 under Cardiopulmonary Resuscitation.

Air embolism

An embolus is any foreign matter in the blood stream. Emboli may be:

- Blood clots (e.g. pulmonary embolism)
- Air
- Fat
- Tumour
- Amniotic fluid

Causes of air embolism:

- Head and neck surgery, especially if performed in the sitting or steep reverse Trendelenburg (head-up) position.
- Pelvic surgery
- Laparoscopy
- Open heart and chest surgery
- Maxillary antrum wash out
- Insufflation of the fallopian tubes
- Delivery in the presence of placenta praevia
- Criminal abortions
- Pneumo-encephalograms
- Infusions and CVP lines.
- Hip surgery

The volume of air required for clinical signs to present themselves is 10 to 15ml.

Signs and Symptoms

- CVS
  - Marked hypotension
  - Arrhythmias
  - Myocardial ischaemia
  - O/E a “mill-wheel murmur” at the apex. This has been described as (on auscultation) "waves lapping against the shore".
  - Cardiac arrest
- RS
  - Respirations may become irregular (if the patient is breathing spontaneously). On examination the patient will be cyanosed and the blood dark.
- CNS
  - Convulsions followed by coma.

Treatment

- Speedy recognition is essential.
- Prevent further entry of air into the blood stream by jugular compression and by flooding the wound with saline.
- Place the patient in the left lateral head-down position. This will help trap the air bubbles in the right atrium and prevent entry into the lungs.
- Discontinue N_2O.
- Give oxygen by mask. 100% oxygen if intubated.
- Treat hypotension and arrhythmias.
- Aspirate the air from the right heart by means of a catheter. If a CVP line is in place this can be used to aspirate air.

Fat embolism
This is usually associated with fractures of the lower limb long bones. Particles of fat are carried in the blood stream and deposited in the lungs, brain and skin. The symptoms may appear soon after the injury or up to 2-3 days post injury.

Signs and symptoms
- Dyspnoea
- Petechiae – usually in neck, axillae and conjunctivae
- Mental confusion

These three signs in the presence of a long bone fracture are diagnostic of fat embolism.
Other signs are fever and tachycardia, hypoxia, pulmonary oedema with bilateral shadowing on chest x-ray, reduced haematocrit and platelet count.

Treatment
This is mainly supportive – respiratory, cardiovascular and renal. The fracture should be stabilized.
High dose steroids have been advocated but their use is not supported by clinical data.

Prognosis
Prognosis is variable and in severe cases mortality is high.

Cardiac failure
This complication is dealt with in Chapter 49. Rapid transfusion and overloading the circulation with fluid can result in pulmonary oedema.

RESPIRATORY COMPLICATIONS

Respiratory obstruction
- Spasm of jaw muscles
- Tongue falling back
- Laryngeal spasm
- Bronchospasm

The causes, symptoms and signs are dealt with in Chapter 8

Hypoventilation from any cause
See Chapter 57 under Respiratory failure

Apnoea
See Chapter 57 under Respiratory failure

Coughing
This may occur under the following conditions:

- Induction of anaesthesia with an inhalational agent. This delays the uptake of the vapour and therefore the process of induction. It is more likely to occur when the inspired concentration is increased too rapidly.
- Induction with an intravenous agent.
- Irritation of the larynx by:
  - An oropharyngeal airway, e.g. Guedel airway.
  - Laryngoscope, e.g. attempting intubation when the patient is not deeply asleep.
  - Secretions.
  - Regurgitated gastric contents.

Treatment: Remove the airway and suction the pharynx. Give oxygen if required. Deepen the anaesthesia.

Tachypnoea (rapid respiration)
Fast respirations are shallow. They tire the patient. The tidal volume is small and therefore the exchange of gases is poor.

Causes

- Inadequate depth of anaesthesia
- The stimulant effect of the anaesthetic agent used: ether, halothane
- Hypoxia
- Hypercarbia
- Shock
- Hyperpyrexia

Treatment

- The cause must be found and treated first. For instance, if the patient is light the anaesthetic must be deepened.
- Assist ventilation. Squeeze the bag or press down the bellows as the patient takes a breath and so increase the tidal volume.
- Control ventilation. This may be necessary depending on how rapid and shallow the respiration is. The patient is ventilated at a rate decided by the anaesthetist using a non-depolarising muscle relaxant if necessary.

Carbon dioxide Retention

Hypoventilation and apnoea generally result in hypoxia and carbon dioxide (CO_2) retention. However, it is important to remember that if the inspired oxygen concentration is very high, then CO_2 retention may occur unaccompanied by hypoxia. This may occur during the operation and post operatively in the recovery room.

Causes of CO_2 excess

- Hypoventilation from any cause. i.e. excessive use of opioids causing respiratory depression. In the presence of a high inspired oxygen concentration hypoxia may not occur.
- Respiratory obstruction.
- Misplacement of the endotracheal tube down one main bronchus (usually the right).
- Exhaustion of the soda lime canister, especially at low gas flows.
- The presence of a large dead space, as when large masks or connectors are used for children.
- The use of a T piece (paediatric anaesthesia) with inadequate gas flows.
- Defective exhalation valves on an anaesthetic machine.
- A high oxygen concentration in patients with chronic obstructive airway disease can result in CO_2 retention. This is rare and hypoxia from inadequate oxygen administration is a more common problem. Careful monitoring is required.

Pneumothorax

Pneumothorax is the term used to describe air in the pleural cavity. The lung on that side then collapses and gaseous exchange does not occur.

Types of pneumothorax

- Closed: where there is no communication with the atmosphere. Air may be trapped in the pleural cavity when the chest wall is intact
- Open: here there is free communication with the atmosphere through an opening in the chest wall or through a broncho-pleural fistula.
- Tension: a flap of pleura may act like a valve. This will enable air to enter the pleural cavity during inspiration but prevent it from leaving during expiration. The condition is made worse by I.P.P.V. This is very dangerous. The mediastinum gets pushed to the opposite side and compresses the normal lung.

Causes of pneumothorax in the surgical patient

- Rupture of an emphysematous bulla. This may occur spontaneously or after IPPV.
- Pneumothorax may follow certain surgical procedures:
  - Thyroidectomy
  - Tracheostomy
  - Surgery on the neck, e.g. neck dissection
  - Kidney surgery
  - Gall bladder surgery
  - Insertion of a subclavian or internal jugular catheter (CVP)
  - Certain regional techniques such as intercostal block or a supraclavicular brachial plexus block.

Diagnosis: A pneumothorax must be suspected if it becomes increasingly difficult to ventilate the patient and the patient's condition rapidly deteriorates.

Early signs are

- A fall in oxygen saturation
- Cyanosis / dyspnoea
- Tachycardia
- Hypotension

Exclude other causes of difficulty in ventilation e g respiratory obstruction in the tube or upper airways, right main bronchus intubation, bronchospasm and inadequate relaxation of the muscles of the chest wall.

The diagnosis is made by listening to the chest. The air entry on the side of the pneumothorax will be decreased or absent. There may also be displacement of the trachea to the unaffected side and later, crepitus. The diagnosis is confirmed by CXR. However treatment is always on the basis of clinical findings. In the case of a tension pneumothorax there is not time for a chest X-Ray.

Treatment: (open and closed pneumothorax)

- Discontinue nitrous oxide and give 100% oxygen.
- Once diagnosis is confirmed an underwater-seal drain should be inserted.

Treatment: (tension pneumothorax) As soon as the diagnosis is suspected a 14G or 16G needle must be inserted into the pleural cavity on the affected side in the 2nd intercostal space in the mid-clavicular line. Aspiration of air will confirm the diagnosis. A chest tube connected to an underwater seal drain must also be inserted as quickly as possible.

POST-OPERATIVE CHEST COMPLICATIONS

Such complications occur in 5% of all operations. At least 10% of all abdominal surgery is followed by some degree of complication.

Causes of post-operative complications:

Patient causes

- Age. Chest complications are more frequent in older people.
- Sex. Such complications are three times more common in males.
- Smoking. Complications are six times more frequent in smokers than in non-smokers.
- Acute upper respiratory infection may result in chest infections.
- Pre-existing lung disease.

Surgical causes

- Chest complications are most common after upper abdominal surgery. Pulmonary embolism is more common after pelvic surgery and lower limb orthopaedic surgery.
- The longer the duration of surgery, the greater the chance of complications. Prolonged anaesthesia inhibits ciliary activity and delays return of airway reflexes.
- Steep Trendelenburg and lithotomy positions increase the incidence of complications.
- Surgery which involves handling the bowel and retraction of organs.
- Surgery which involves prolonged post-operative bed rest.

Anaesthetic causes

- Excessive premedication. Opiates can predispose to chest complications if given in excess.
- Inadequate pre-operative preparation. Patients with acute chest infections should not be subjected to routine surgery. Those requiring emergency surgery will do better with a regional anaesthetic. If appropriate, regional anaesthesia may be a better choice for patients with acute chest infections requiring emergency surgery.
  If time permits those with chronic chest infections should be treated with physiotherapy and antibiotics if indicated, in an attempt to optimise their condition.
- Hypoventilation from any cause.
- Aspiration of stomach contents or secretions whilst under anaesthesia.
- Prolonged shock.
- Contaminated equipment.
- Excessive post-operative sedation.

Bronchitis

This vague term is used to describe cough, sputum, fever, dyspnoea and wheezing. It is more common in those with pre-existing chest disease. The pathological change is inflammation of the bronchioles.

Treatment consists of antibiotics, supportive therapy (i.e. oxygen therapy, physiotherapy, etc.) and bronchodilators.

Pneumonia and less commonly lung abscesses may occur in addition to bronchitis. The infective organism may reach the lung from many sources, examples being:

- The upper respiratory tract, as from dental sepsis.
- Contaminated equipment, e.g. endotracheal tubes.
- Disease in the abdomen, e.g. peritonitis or subphrenic abscess.
- The aspiration of gastric contents.
- Previous infection, which can be the source of bacteria responsible for post-operative infection.

Collapse of the Lung

Collapse of the lung may be classified depending on the extent of lung involvement:

- Entire lung
- Lobular collapse
- Segmental collapse /atelectasis

Lobular collapse or atelectasis

This usually develops in the first 48 hours after the operation with:

- Fever
- Increased difficulty in breathing
- Dry cough
- Rapid heart rate
- Dilatation of the alae nasae
- Cyanosis
- Restricted chest movements on the affected side
- Diminished breath sounds on the affected side.

The danger of atelectasis is that secondary infection may develop and this can lead to pneumonia, bronchiectasis, lung abscess and pleural effusions. The most important aspect of treatment of atelectasis is *prevention*. All the signs and symptoms mentioned above require attention.

Pre-operative preventive measures

- Treat all pre-existing chest disease before anaesthesia.
- Treat oral sepsis.
- Instruct the patient to stop smoking for at least 48 hours pre-op, preferably longer.
- Organise preoperative physiotherapy (deep breathing exercises) for all patients scheduled for major surgery.
- Avoid excessive pre-operative sedation.

Post-operative preventive measures

- Start chest physiotherapy and encourage early mobilisation.
- Give adequate pain relief. If the patient has inadequate pain relief, breathing will be shallow.

Treatment of atelectasis once it has occurred

- Chest physiotherapy:
  - Turn the patient regularly.
  - Start deep breathing exercises.
  - Encourage coughing.
  - Perform percussion of the chest (to dislodge a plug of mucus) and postural drainage.
  - Mobilise as soon as possible.
- Treat the infection with an appropriate antibiotic.
- Treat any bronchospasm with bronchodilators, e.g. Ventolin or aminophylline.

Segmental or lobular atelectasis is the commonest complication after anaesthesia.

Aspiration pneumonitis (Mendelson's Syndrome)
Aspiration of stomach contents, as a result of vomiting or regurgitation, is a dangerous problem. The gastric contents are acid. If the pH is less than 2.5, (which means the contents are very acid) and if the stomach contents reach the lungs then a pneumonitis results. This was originally described by Mendelson in obstetric patients where the risk of aspiration was high and the stomach contents had a lower than normal pH.

Signs and symptoms

- Dyspnoea
- Tachycardia
- Tachypnoea (rapid respirations)
- Bronchospasm
- Pulmonary oedema
- Cardiovascular collapse i.e. hypotension
- CXR mottled opacities.

Prevention: The patients likely to vomit or regurgitate will be discussed under gastrointestinal complications on the next page.

- Use a regional technique if anaesthesia is needed, provided there is no contraindication.
- Attempt to empty the stomach by using a wide-bore orogastric or nasogastric tube.
- Use a non-particulate (i.e. sodium citrate) antacid within 30 minutes of induction. Dose 30ml of 0.3M solution orally. (Antacids make gastric contents less acid).
- An awake intubation may be attempted in the very ill and poor risk patient.
- A rapid sequence induction with cricoid pressure, described in Chapter 16 must be used.
- Other drugs (Histamine H_2-receptor antagonists) decrease the volume and acidity of gastric contents. Examples are cimetidine and ranitidine. Omeprazole is another useful drug. Metoclopramide 10mg reduces gastric volume if given at least one hour before surgery.

Treatment

- Repeated tracheal suction, **preceded by** oxygen administration.
- Further oxygen by mask.
- Bronchodilator drugs for treatment of bronchospasm, e.g. aminophylline or salbutamol.
- Give antibiotics if the aspiration is likely to be infected fluid, for example in bowel obstructions; in this case immediate broad-spectrum antibiotics are indicated. However, if the aspiration is of gastric contents only (most likely sterile) it is best to withhold antibiotic treatment until signs of infection, with positive cultures, have been established.
- IPPV may be required. PEEP is usually indicated to maintain oxygenation.
- Cardiovascular support. In severe aspiration a shock-like syndrome develops which requires aggressive fluid management with possible inotrope support.
- Bronchoscopy. Therapeutic bronchoscopy is indicated if there is particulate aspiration, focal pulmonary collapse suggesting large airway obstruction, or chest x-ray evidence of foreign bodies.

Steroids have no proof of benefit in aspiration syndrome and may be harmful causing delayed healing and a tendency to infection.

Pulmonary Embolism

This occurs when a clot from a vein in the lower limb or the pelvis is detached and carried to the lung. It usually happens 3-21 days after the operation and is more common in older patients who have had prolonged bed rest.

Symptoms: The symptoms will depend on the size of the clot. If it is large, it may obstruct the pulmonary artery which conducts the blood from the right ventricle to the lungs and sudden death may result. If the clot is small, then the patient presents with fever, cough, haemoptysis, dyspnoea and chest pain. Pulmonary infarction is said to have occurred. It may be difficult to differentiate pulmonary infarction from a chest infection. A chest x-ray and ECG may help. It is important to remember that pulmonary embolism may occur without any warning sign of deep vein thrombosis.

Treatment:

- Give oxygen
- Give analgesia for pain
- Treat any arrhythmias which may occur.
- Start on anticoagulants, e.g. Heparin
- Surgery. If the patient continues to have pulmonary emboli in spite of anticoagulant therapy, then in hospitals with specialised facilities a filter can be inserted into the inferior vena cava to prevent clots travelling to the heart. In rare cases surgical embolectomy is necessary.

Prevention: The most important measure is to prevent deep vein thrombosis.

- Give prophylactic anticoagulants: Heparin 5000 units SC, 12 hourly.
- Use calf stimulators during surgery.
- Use regional techniques if possible – this can reduce the incidence of DVT by as much as 20%.
- Encourage patients to move the legs, feet and toes frequently.
- Teach deep breathing exercises.

Examine the usual sites for DVT in the calf, the groin and feet.
Commence anticoagulants if there is any evidence of deep vein thrombosis.

Patients at high risk of DVT and pulmonary embolism

- Obese
- Diabetics
- Marked peripheral arterial disease
- Elderly
- Varicose veins
- Those who have been in shock
- Congestive cardiac failure
- Prolonged surgery
- Pelvic surgery
- Orthopaedic surgery, especially of the hip or knee.

Pulmonary oedema
The most common causes of post-operative pulmonary oedema are fluid overload, cardiac failure, prolonged airway obstruction and trauma
These are discussed in Chapters 34 and 62.

GASTROINTESTINAL COMPLICATIONS

Vomiting and aspiration

Differences between vomiting and regurgitation.
Stomach contents can reach the lungs by two mechanisms:

- Vomiting which is an active process. It is the expulsion of the material from the alimentary tract by muscular contraction.
- Regurgitation which is passive. It does not involve any muscle action. It occurs silently and is more dangerous than vomiting.

Dangers of Vomiting and aspiration
Vomiting and aspiration of gastric contents can occur during the induction and maintenance phases of the anaesthetic or during recovery.
The dangers are:

Hypoxia. Large volumes of liquid can flood the lungs. Solid particles of food can obstruct the inlet. The aspirated material can cause ***laryngeal spasm***.

Aspiration pneumonitis (Mendelson's Syndrome) - discussed earlier in this Chapter. This is more likely if the gastric contents are very acid (pH< 2.5).

Cardiac arrhythmias secondary to hypoxia.

Respiratory infections, e.g. bronchopneumonia, atelectasis.

Patients at risk of vomiting or regurgitating under anaesthesia
The following patients should always be anaesthetised using the technique of rapid sequence induction with cricoid pressure to help reduce the risk of regurgitation.

- Those with material in the gastrointestinal tract. This material may be food or blood, if there has been bleeding into the gastrointestinal tract.
- Those with obstructions in any part of the gastrointestinal tract.
  - Oesophagus: stricture, tumour, pouch
  - Stomach: pyloric stenosis, hiatus hernia
  - Small or large intestine: adhesions, volvulus, tumours, hernia or intussusception, gallstones, worms and foreign bodies.

  In intestinal obstruction the absorption from the gut is impaired but the secretions continue to be poured into the intestine.
- Those who have fasted for the 6-hour period required by anaesthetists but who have a delay in the gastric emptying time:
  - Pregnant women
  - Seriously ill patients
  - Those with head injuries
  - Patients who have received opiate drugs.
- Those who have an incompetent lower oesophageal sphincter (the sphincter between the oesophagus and the stomach).
- Another risk factor is raised intra-abdominal pressure, which may be caused by:
  - Peritonitis. For example after a perforated typhoid or duodenal ulcer.
  - Obesity
  - Pregnancy
  - A tumour (for instance, ovarian)
  - Ascites
  - Position, e.g. lithotomy.

Intra operative nausea and vomiting after a spinal or epidural
Before any medication is given for the symptoms, the underlying cause must be sought.

- Hypotension is a major cause of nausea after a spinal. It is a good practice to check the patient's blood pressure immediately when a complaint of nausea is made. This is particularly important in patients for caesarian section.
- Hypoxia from hypoventilation
- Surgical traction on the intestines
- Increased intestinal peristalsis as a result of the spinal block.
- Narcotic analgesics given as premedication
- Anxiety on the part of the patient.

Treatment

- Treat the cause. Correct hypotension and hypoxia and reduce surgical traction.
- Reassure the patient
- The following drugs may be used:

  Prochlorperazine (Stemetil) - 12.5mg IM
  Metoclopramide (Maxolon) -10-20 mg IM or IV
  Promethazine (Phenergan) - 25mg IM.
  Cyclizine - 50 mg IM or IV
  Ondansetron - 4 mg IV

Management of vomiting during anaesthesia

If vomiting does occur take the following measures:

- Position: Head down, lateral position. (This minimises the chance of the vomited material being aspirated).
- Suction. Clear the airway of any vomitus.
- Give oxygen.

Always watch for signs of aspiration. The patient may show evidence of dyspnoea, wet or moist breath sounds, wheezing, either at the time of vomiting or some hours after the incident. A CXR must be done if this is suspected.

Treatment of aspiration during anaesthesia

- Oxygen therapy
- Bronchodilators:
  - Ventolin is the mainstay of treatment given via nebules, IV (250micrograms over one minute) or IM (500micrograms four hourly for adults).
  - Aminophylline 250 mg stat IV over 10 mins followed by an infusion if necessary.
- IPPV with oxygen - if severe i.e. refractory hypoxia.
- Bronchoscopy
- Active chest physiotherapy

It requires special expertise to anaesthetise patients with full stomachs or those likely to vomit under anaesthesia. These techniques are discussed in Chapter 16.

Post-operative nausea and vomiting
Consider the following:

- The sex and age of the patient. Vomiting is more likely in young females.
- The condition of the stomach. Gastric distension or the presence of gastric contents predispose to vomiting.
- Premedication. Opiates, e.g. pethidine, morphine, etc. can cause vomiting.
- The type and concentration of anaesthetic agent used e.g. ether / N_2O.
- The surgical procedure performed. Upper abdominal surgery, especially surgery on the biliary tract and pelvic surgery are associated with vomiting.

Treatment

- Prevention is the most important.
- Drug treatment is outlined above.
- A combination of anti-emetic drugs, if available, is thought to improve treatment.

Hiccups
This is a state of intermittent spasm of the diaphragm, caused by stimulation of the sensory nerve endings in the diaphragm, as occurs with upper abdominal or thoracic surgery. Hiccups may occur during gastrectomy, vagotomy etc and may also be associated with gastric distension. Hiccups may sometimes be seen in uraemic patients, as a result of central stimulation of the medulla.

Treatment (often no treatment is required)

- Gastric decompression using a nasogastric tube.
- Minimise irritation or stimulation of the diaphragm.
- Deepen anaesthesia.
- Use a muscle relaxant and IPPV.

Gastric distension
The stomach may become distended in the following situations:

- During IPPV when a mask is used. This is more likely if the airway is partially obstructed or if high gas flows have been used.
- When an air leak occurs around the endotracheal tube. This may occur if too small a tube has been inserted into the patient's larynx or if the cuff of the endo-tracheal tube has ruptured in situ.
- Accidental oesophageal placement of the endotracheal tube. **It is vital to visualise the vocal cords during intubation and to auscultate the chest for breath sounds after intubation. Capnography is valuable.**
- Surgical manipulation of stomach, intestine and mesentery.

Dangers

- Respiratory embarrassment, during and after surgery, due to splinting of the diaphragm.
- Increased risk of aspiration
- Post-operative vomiting
- Hiccups
- Interference with surgical procedure.

Treatment: Relieve gastric distension by passing a nasogastric tube.

Liver damage

The main cause of liver damage under anaesthesia is hypoxia especially in association with hypotension.
Other causes are:

Halothane hepatitis
The incidence of post-operative liver damage after halothane anaesthesia is very rare, 1 in 10,000 in adults, even more rare in children.
Halothane hepatitis is believed to be a hypersensitivity reaction.
It is identical to infectious hepatitis, both clinically and biochemically.

Some Important Points

- Allow at least 12 weeks between administrations of halothane, especially in obese middle-aged women, unless the indications are clinically overriding.
- Do not use halothane if the previous administration was associated with a fever of unknown origin. The fever, nausea and vomiting appear 2-5 days after the halothane anaesthetic. This is also associated with abnormal liver function tests, indicating hepatitis.
- A single administration of halothane is unlikely to be associated with severe liver damage.
- Pre-existing liver disease (if not due to halothane hepatitis) is not a contraindication to the use of halothane provided the patient is considered otherwise fit for surgery and anaesthesia.

Ether anaesthesia
Like most general anaesthetic agents, ether is associated with a reduction in blood flow to the liver. It has no hepatotoxic action.

Enflurane (Ethrane)
Hepatitis has been reported following the use of enflurane, however, it is extremely rare.

URINARY COMPLICATIONS

Difficulty in passing urine

This is more common after a spinal anaesthetic but may also occur after a general anaesthetic. It is more common in anxious patients, those who have had abdominal, pelvic or perineal surgery, those who have had heavy sedation and in those patients with enlarged prostates. Every means of encouraging a patient to urinate should be tried. If all else fails catheterisation is necessary.

Reduction in output (oliguria or anuria)

The normal urine output is about 1 ml/kg/hr, i.e. about 60 ml/hr in the adult patient. The minimum acceptable urine output is 0.5ml/kg/hr. A fall in the urine output can be due to:

Pre-renal causes: Usually associated with volume depletion (dehydration or blood loss). The fall in urine output can be corrected by a fluid load, e.g. 1 litre of Hartmann's/ saline solution administered over half an hour. The usual signs of dehydration such as dry tongue, loss of skin turgor, tachycardia, fall in blood pressure plus a low central venous pressure (CVP) would also suggest pre-renal oliguria.

Renal causes: The renal tubules are damaged by hypoxia, hypotension, bacterial toxins, mismatched blood transfusions and drugs (e.g. gentamicin, non-steroidal anti-inflammatory drugs). If a diagnosis of renal oliguria or anuria is made then careful fluid management to avoid fluid overload is necessary. However, it is important to maintain adequate renal perfusion. Maintaining electrolyte balance is important and in more severe cases renal dialysis is necessary.

Post renal causes: In this situation oliguria or anuria is due to some obstruction in the urinary tract or catheter, e.g. kinking or obstruction of the catheter, prostatic enlargement, urethral stricture. There will be evidence of an enlarged bladder if the obstruction is distal to it.

NEUROLOGICAL COMPLICATIONS

Complications involving the central nervous system:

Coma and convulsions

These may follow the use of regional techniques or general anaesthesia.

- Convulsions and coma as a result of local anaesthetic drugs or regional techniques have been discussed in the appropriate chapters. Overdose of local anaesthetic drugs is one of the more common causes.
- Convulsions may occur in known or latent epileptics especially those who are poorly controlled. Ether convulsions are discussed in Chapter 6. Enflurane may cause convulsions in epileptics and propofol has been associated with convulsions (pseudo-seizures) often occurring some time after administration.
- Convulsions and coma may also occur during or after general anaesthesia, perhaps after a period of acute hypoxia (e.g. associated with a cardiac arrest) or a period of chronic hypoxia (e.g. associated with a partially obstructed airway or hypoventilation, etc.). Recovery may be delayed: the patient may regain consciousness only to lapse into coma again. The cerebral oedema associated with hypoxia must be treated. Generally the prognosis is poor.
  - Maintain normotension and oxygenation.
  - Tilt the head up at 30°.
  - Assist or control ventilation as required.
  - Support the circulation.
  - Mannitol 20% 0.5gm/kg
- Coma can also be due to many other causes, for example, overdose of anaesthetic agents, retention of carbon dioxide, cerebrovascular accident or myocardial infarction with reduced cardiac output and as a consequence of hypotension, shock, diabetic state, liver or renal disease. Convulsions may follow neurosurgery. Acute porphyria and preoperative treatment with monoamine oxidase inhibitors may also cause coma.

Treatment of convulsions

- Treat the cause
- Give oxygen
- Give anticonvulsants, e.g. diazepam, midazolam or thiopentone. Again, treat any associated cerebral oedema.

Peripheral nerve injuries may result from faulty positioning
Injury to nerves can be avoided by:
- Padding the shoulder braces if they are used.
- Abducting the arm no more than 90 degrees at the shoulder joint.
- Padding the arm board so that it is level with the mattress on the table.
- Protecting the ulnar and lateral popliteal nerves by foam rubber to avoid excessive pressure.

Other causes of peripheral nerve injuries
- Extravasation of injected drugs (e.g. thiopentone) in the region of the peripheral nerves.
- Spinal anaesthesia .The neurological consequences of spinal anaesthesia have been discussed in Chapter 19

OPHTHALMIC COMPLICATIONS

Corneal abrasions
These can occur very easily under anaesthesia if the eyes are left open. The cornea dries very quickly and is easily injured.
Prevent this from happening by:
- Using a sterile ointment in the eyes during anaesthesia.
- Closing the eyes carefully with a piece of adhesive tape.

Blindness
Excessive pressure of the mask on the eyeball, especially if the patient is hypotensive, can result in serious damage or blindness by occluding the blood supply to the eyes. Careless positioning when the patient is prone can also cause serious eye damage.

OTHER COMPLICATIONS

Shivering:
This is seen after general anaesthesia with halothane, enflurane, ether and even thiopentone. It may be the body's response to heat loss following vasodilation that accompanies general anaesthesia. A further loss of heat occurs from the respiratory tract when dry gases are breathed in through an endotracheal tube. Prolonged surgery and cold IV fluids also contribute to hypothermia. A cold operating room may also cause shivering postoperatively.

Treatment
- Warm blankets
- Oxygen by mask as long as the shivering continues.
- Sedation if shivering is excessive e.g. pethidine 15-25mg IV.

See also Chapter 51 (Hypothermia)

Awareness during anaesthesia

Many reports of awareness have come from patients receiving nitrous oxide/oxygen, relaxant anaesthetics. This occurs when ether, halothane or other volatile is not used. It may also occur in obstetrics where a narcotic premedication is avoided and a smaller dose of thiopentone is often used for fear of depressing the baby. (Note that patients with pre-eclampsia should have the normal induction dose of thiopentone to help reduce the hypertensive response to intubation).

The problem of awareness can normally be avoided by using a "supplement" with nitrous oxide and oxygen (i.e. volatiles) and by carefully monitoring the patient's pulse and blood pressure.

Malignant hyperpyrexia

This condition is rare but very dangerous. It runs in families being an inherited disorder of skeletal muscle triggered by some common anaesthetic drugs. The patient may show some or all of the following features.

- A family history of anaesthetic problems.
- Raised CPK enzyme level.
- Malignant hyperpyrexia is thought to be more common in patients with muscular dystrophies and related disorders. There may be an association with squint surgery. The only proven associations are with rare genetic muscle disorders (i.e. King/Denborough disease and Central Core disease).
- The reaction may be triggered by either a volatile agent (i.e. halothane, enflurane, isoflurane), or by suxamethonium. These are the most potent triggers of malignant hyperpyrexia.
- A previous uncomplicated general anaesthetic does not exclude the development of malignant hyperpyrexia.

Clinical signs under anaesthesia

- Spasm of the masseter muscle of the jaw and a general increase in muscle tone in spite of neuro-muscular blockade.
- Unexplained tachycardia
- Hypercapnia in ventilated patients
- Tachypnoea in spontaneously breathing patients
- Cyanosis
- Arrhythmias
- Rise in temperature
- Hyperkalaemia

Later signs include renal and cardiac failure.

Management of malignant hyperpyrexia:

- Stop the anaesthetic and surgery.
- Give 100% oxygen via endotracheal tube. Hyperventilate the patient.
- Dantrolene if available: initial dose 2.5mg/kg. Then 1mg/kg (up to 10mg/kg) repeated every 10-15 minutes. Dantrolene is difficult to prepare and therefore requires a dedicated person. It is prepared with sterile water and as each ampoule contains 20mg of Dantrolene this can be quite labour intensive. (60ml of sterile water for each ampoule).
- Treatment of any arrhythmias as they occur.
- Correction of acidosis with bicarbonate (0.5-1 mEq/L) is controversial. Unless there is adequate ventilation or the patient is aggressively hyperventilated this will cause an intracellular acidosis.
- Cool the patient: Insert core temperature probe e.g. nasal, oesophageal.
  - Pack patient in ice or immerse in a cooling bath.
  - Give IV infusion of cold fluids (cooled saline solution, 1000ml/10 minutes for 30 minutes).
  - Gastric, wound and rectal lavage with cold saline solutions.
  - Cooling fans.

  Stop cooling when central temperature falls to 38°C.
- Monitor ECG, temperature, pulse and blood pressure.
- Treat electrolyte imbalance
- Support circulation
- Maintain urine output with fluids, frusemide and mannitol.
- Keep patient sedated throughout with IV midazolam or diazepam.

Other measures: The rest of the family should be tested (if possible) and warned of possible anaesthetic problems.

The side effects of Dantrolene include muscle weakness, sedation and thrombophlebitis with tissue necrosis following extravasation. The muscle weakness may require postoperative ventilation until muscle strength returns to allow spontaneous breathing.

Anaesthesia for a malignant hyperpyrexia susceptible patient

- A regional technique, if appropriate, would be safest
- Ketamine techniques are also useful.

If a general anaesthetic is essential:

- Monitor ECG and temperature meticulously and end-tidal carbon dioxide if available.
- IV induction with thiopentone, propofol or ketamine then muscle relaxation with a non-depolarising agent.
- Maintain general anaesthesia with nitrous oxide/ oxygen and IV opiate or with ketamine or propofol infusion.

The anaesthetic machine should be free of vapourisers and have been flushed with oxygen for 20- 30 mins before use.

CHAPTER 47

VENEPUNCTURE AND ITS TECHNIQUE

Outline:

Choice of veins

Measures to aid venepuncture

Technique of venepuncture

Fixing the cannula

Equipment

Venepuncture is a technique practised most frequently by the anaesthetist. It is used for administering drugs (anaesthetic and non-anaesthetic), also for administering fluids including blood.

CHOICE OF VEINS

- Use the non-dominant arm if possible.
- The injection site should be accessible to the anaesthetist during the operation.
- Don't use varicose veins.
- Avoid lower limb veins also if possible.
- Inserting a needle at the junction of 2 veins is easier than in other positions.

The following sites should be inspected before venepuncture:

- The radial aspect of the wrist joint
- The back of the hand
- The ante-cubital fossa
- The front of the wrist joint
- The saphenous vein anterior to the medial malleolus
- Others: The external jugular vein
  The subclavian veins
  In infants, a scalp vein.

Good lighting is important. The light should come from the side and not from directly overhead.

MEASURES FOR DILATING VEINS

- Use a tourniquet
  Various types of tourniquets are available.
  - Elasticised fabric fastened with velcro
  - Elasticised fabric fastened with a slip catch
  - A blood pressure cuff, inflated to midway between systolic and diastolic.
  - A piece of rubber tubing.
  - The simplest tourniquet of all – the hand of an assistant. This is especially useful with children.

  The tourniquet must occlude only the venous circulation. The arterial pulsation must be felt. The arterial filling results in greater distension of the veins. If an arterial pulse can be palpated the risk of accidental arterial injection is minimised.
  Tourniquets should not be used on patients with sickle cell disease.

- Ask the patient to "open and close" the hand a few times, as if making a fist.
- Gently tap the region of the vein.
- Pad arms and apply a covered hot water bottle. This is useful in patients with few visible veins.
- Light general anaesthesia produces peripheral vasodilatation. This is useful if an intravenous cannula has to be inserted. Start a light general anaesthetic with a scalp vein needle in the peripheral vein and after anaesthesia is induced, insert a cannula. If, however, the patient is a poor risk or there is preliminary bleeding, then it is essential to insert a cannula before anaesthesia starts.

TECHNIQUE OF VENEPUNCTURE

- Choose the vein and (for children) apply EMLA cream 45 minutes before the procedure.
- Apply a tourniquet.
- Remove the EMLA cream, wash hands and wear disposable gloves.
- Clean the skin with antiseptic. Avoid contaminating the point or shaft of the cannula before insertion.
- Use the left thumb to stretch the skin, distal to the point at which the needle will be inserted. This steadies the vein.
- Insert the needle so that it enters the skin lateral to the vein.
- Advance the point of the needle so that it enters the vein at a different point.
- Once blood is aspirated, make note of the colour and the pressure with which the blood enters the needle. Arterial blood is bright red and pulsates. Venous blood is generally darker but in patients breathing a high oxygen concentration, venous blood can be bright in colour.
- Thread the needle up the vein and fix it as described below, with tape. When using an intravenous cannula – once needle and cannula tip are within the vein, hold needle steady, advance the cannula up the vein and then withdraw the needle. Fix as for a needle.
- In anxious patients or when using a large (e.g. 16G) cannula it may be kinder to use a local anaesthetic solution at the site of the skin entry. Inject 0.5ml of 0.5% lignocaine intradermally with a 25G needle before inserting the cannula.

FIXING THE NEEDLE OR CANNULA

- Put a band-aid type dressing over the puncture site.
- Put a piece of adhesive plaster lengthwise over the cannula and two pieces transversely across the cannula.
- The tubing of the intravenous giving set is looped so that any "pull" on the tubing is not transmitted to the cannula. The loop must be fixed independently of the cannula and must not get entangled.
- An 'opsite' dressing, if cost permits, is ideal over the site of venepuncture.

Insertion of a scalp vein needle

During insertion bend the two plastic wings upwards and use them to grip the needle while it is inserted.

Fixing scalp vein needles

Scalp vein needles are secured in place by a piece of adhesive tape attached to the wings.

Removal of the needle or cannula

Withdraw the cannula and apply firm pressure for a few minutes. Cover with a sterile dressing.

EQUIPMENT AVAILABLE FOR VENEPUNCTURE

Needles

Scalp vein needles are available in varying sizes from 21G to 25G.
They are easy to fix and the larger needles can be used for giving intravenous infusions.

Cannulae

Cannulae are necessary for administration of intravenous fluids for several hours or even days. A variety of cannulae are available. Some points to note are:

- The sharpness of the introducing stylet.
- The smoothness of the tapering end of the cannula, from where the metal stylet protrudes.
- The stiffness of the plastic cannula.

  All of these make it easier to insert the cannula.

Flow rate is important when IV fluids are required rapidly

Size	*Flow rate* (ml/min through 110cm tubing)
20 Gauge	40-80
18 Gauge	75-120
16 Gauge	130-220
14 Gauge	250-360

Different makes of cannula of the same gauge can have different flow rates.

Infusion Sets

These usually incorporate a drip chamber and a filter, a delivery tube to the patient and a flow regulator. Occasionally it is necessary to use a microdrip set or to incorporate a burette into the drip set particularly when giving IV infusions to babies and small children. A standard drip set will deliver 15 drops = 1ml and a paediatric buretrol 60 drops = 1ml but makes of drip sets may vary so check instructions in the packaging.

Splints

It is useful to have splints of various lengths available in the operating theatre and the wards. Use splints if the site of venepuncture is close to a joint and the joint needs to be immobilised.
Use splints also if the loop of tubing protrudes a long way from the end of the limb. The splint acts as a support for the loop.
The joint near the insertion site of the cannula must be immobilised with the splint. Use adhesive tape or a crepe bandage for this purpose.

Extension Sets

Extension sets are made of plastic tubing. They can be interposed between the cannula and the giving set and allow access to the injection port when distant from the cannula. Warming coils can be inserted easily if an extension set is used. Three-way taps can be attached to gain access where needed.

Blood Warmers

Blood may be warmed by several methods. The aim is to transfuse the patient with blood at 37°C.

- Place the blood container in lukewarm water.
- Run the blood through a coil placed in warm water, maintained at temperature between 37 and 41°C.
- For children, put the transfusion set tubing under the warming blanket on which the patient is lying.

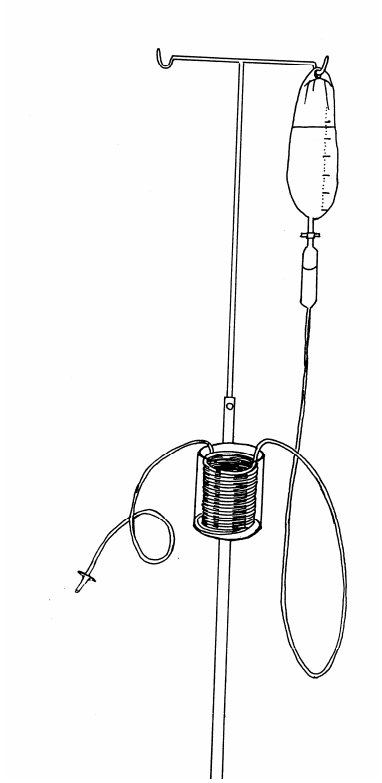


Fig 47.1 Example of a blood warmer using a coil in warm water

CHAPTER 48

INTRAVENOUS FLUID THERAPY

Outline:

Normal distribution of body water

Normal fluid balance

Assessment of fluid depletion

The aims of fluid therapy

Monitoring the dehydrated patient

Management of the surgical patient

Complications of intravenous therapy

Enteral feeding

Total parenteral nutrition

DISTRIBUTION OF BODY WATER

The total body water in the adult is 60 - 65% of the body weight. Two thirds of the body water is intracellular. One third is extracellular i.e. outside the cells. This takes the form of interstitial fluid and also the circulating blood volume.

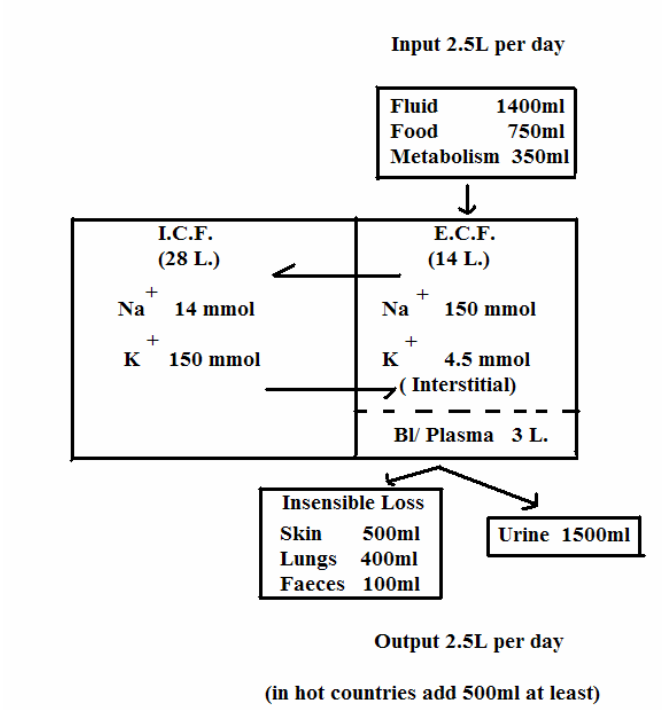


Fig 48.1 Distribution of body water and average input and output

Fig. 48.1 shows the relative volumes of intra and extra cellular fluid. Note the relatively small compartment of the blood volume and remember that in most emergency and surgical situations this is the only compartment to which there is access for making adjustments in the patient's hydration.

There is movement between the compartments both active and passive.

In the ECF: if the vascular endothelium is intact the circulation retains colloid and large molecules. The interstitial spaces have electrolyte containing fluids.

In the ICF: the cell walls are impermeable to Na^+ but highly permeable to H_2O. Cell damage upsets this balance.

NORMAL FLUID BALANCE

The fluid intake must be equal to the output for proper fluid balance to be maintained. If the intake is more than the output the patient will suffer from fluid overload and pulmonary oedema. If intake is less than required the patient will become dehydrated. The output will also begin to fall but not sufficiently to prevent the patient suffering the symptoms and signs described below.

In hot climates and in febrile patients insensible loss is significantly increased - therefore in such situations, the daily fluid intake must be increased.
Infants are at even greater risk of dehydration than adults.

ASSESSMENT OF FLUID DEPLETION

History

Vomiting, diarrhoea, reduced intake of food and fluid - will suggest fluid depletion. Thirst is very frequently a symptom of fluid lack.

Examination

- The patient may be weak and show a loss of interest in the surroundings.
- The respiratory rate may be rapid, with shallow breathing.
- The tongue is dry and may be furrowed.
- The skin is cool to the touch and may be mottled. There is a loss of skin elasticity - (if the skin is pinched it stands up in folds).
- The eyeballs may be sunken in severe dehydration.
- The body temperature may be elevated.
- The pulse is fast and thready.
- The blood pressure is initially maintained by peripheral vasoconstriction - but falls in the later stages. There may be postural hypotension.
- The urine output falls.
- The central venous pressure is low.

Laboratory tests

There is gradual increase in the following:
- Serum sodium, potassium, chloride, blood urea nitrogen and creatinine.
- Haemoglobin and haematocrit levels.
- The specific gravity of the urine.

These changes are due to haemo-concentration.

Assessing the degree of dehydration (% of body weight)

Patients come into hospital having lost much fluid and electrolytes from diarrhoea, vomiting, fistulae or accumulation of fluid in the obstructed gut e.g. intestinal obstruction.

Degree of dehydration

- **Mild (5% total body water or 3L in 70kg adult)** - A history indicating dehydration e.g. an adult who has fasted 24 hrs or a child 12 hrs but without clinical signs except thirst and a dry mouth.
- **Moderate (5-10% TBW or 5L in 70kg adult)**
  Mouth- dry
  Eyes - moderately sunken
  Skin - moderate reduction in skin elasticity. Reduced peripheral perfusion. Depressed fontanelle in the neonate.
  CVS - B.P. normal due to peripheral vasoconstriction. However there is often postural hypotension. Decreased JVP/CVP. Increased heart rate.
  CNS - restless, irritable or drowsy.
  Urine output - oliguria.
- **Severe/Shock (10%-20% TBW or 8L in 70kg adult)**
  Mouth- very dry
  Eyes - very sunken
  Skin - gross loss of elasticity
  CVS - B.P. low, pulse very fast, peripheral vasoconstriction
  CNS - confused/delirious
  Urine output - oliguria/anuria.

THE AIMS OF FLUID THERAPY

- Restoration of a normal circulating blood volume
- Maintenance of an adequate urine output
- Maintenance of a normal electrolyte composition of the plasma
- Provision of adequate nutrition

Fluid requirements

Normal maintenance for a patient not taking any oral fluids

- Replacement of normal losses of 2.5L/day +500ml in hot climate
- Na^+ and K^- – 1mmol/kg/day
- Some calories

This can be provided by the equivalent of 2L 5% dextrose + 1L 0.9% saline in any combination + KCl in every bag (13.4mmols/10ml)

Additional needs must be calculated or estimated from history and clinical observation as detailed above.
Potential losses may be some or all of the following:

- Water and electrolytes. These are both lost together. There will be an increase in osmolarity across all compartments.
  Insensible losses from skin and respiratory tract will increase due to sweating with loss of H_2O, Na^+ and Cl^- and hyperventilation.
  Pyrexia or a hot climate can increase losses by as much as 2-3 Litres.
  Gut losses such as vomiting (with loss of H^+**,** Cl^- and water), diarrhoea or suction from naso-gastric tube or fistula.
  Bowel prep before surgery can also result in loss of H_2O, K^+ and Cl^-.
  Surgical and trauma cases: fasting for 4-12 hrs can result in losses of up to 5ml/kg/hr.
  During open abdominal surgery insensible loss from the gut can be as much as 10ml/kg/hr.
  In post-op and trauma patients there can also be "third space losses". This is sequestered fluid in the tissues (at the site of injury) which doesn't play any part in the metabolic process. It will be reabsorbed after 2-3 days.
- Plasma is lost in burns and sepsis from the disrupted cell membrane. There will be a fall in plasma proteins and in capillary oncotic pressure leading to leakage of water and electrolytes from the vascular compartment to the ECF.
  The loss of protein affects the binding of certain drugs and there can be an increase in free plasma concentration leading to overdose and increased side effects.
  Plasma and other colloid solutions can increase the intravascular volume.
- Blood may be lost due to haemorrhage or intraoperatively. During surgery blood loss should be carefully measured.
- Pre-existing deficits should be estimated according to the assessment procedure detailed above.

Fluid resuscitation for the dehydrated patient

This should consist of maintenance plus all measured or calculated additional losses.

- Use dextrose / saline for maintenance.
- Use 0.9% saline for tissue fluid losses with additional K^+ if indicated. Laboratory measurement of electrolytes is recommended if available.
- Use colloid solution when available to try to expand plasma volume.

Rate of correction

The rate of infusion of fluid will depend on the degree of dehydration, the urgency of surgery required and the cardiac state of the patient.
The body has a homeostatic mechanism by which losses from the vascular compartment are replaced by other extra-cellular fluids and then by intra-cellular fluids. Equilibration will take a number of hours. Every patient is different. A young previously fit patient will tolerate infusion of 3L in 4 hrs whereas in an elderly or frail patient infusion of this volume should be over 12-24 hrs.
Careful observations of the urine output, pulse and blood pressure, will indicate at what rate the infusion should be continued. Ongoing losses will also need to be replaced. Auscultation of the chest must be done at regular intervals to detect circulatory overload although under hydration is a far greater problem than over hydration.

MONITORING THE DEHYDRATED PATIENT

The following indices will show the effectiveness of rehydration and guide ongoing rehydration.

- Pulse rate - a gradual decline.
- CVP - a rise of 2-3 cm with rehydration.
- Urinary output -
  A good urine output = 1ml/kg/hr = 60ml/hr in the adult.
  The minimum acceptable output = 0.5ml/kg/hr = 30ml/hr in the adult.
- Other signs:
  - Urine SG decreases
  - B.P may rise
  - Mouth less dry
  - Eyes less sunken
  - Skin turgor increased
  - CNS - patient more rational

Points of importance

- Severe dehydration falsely elevates haematocrit and haemoglobin.
- Severe dehydration is associated with electrolyte imbalance.
- Check Na^+ and K^+ if possible.

IV fluids are given as replacement therapy i.e. to replace and restore sustained losses or as maintenance therapy i.e. to maintain the state of cellular hydration during a period of fasting.

Pre-operative management

Replacement Therapy

- Every patient presenting for surgery should be adequately hydrated - postponement of surgery may be required unless it can be proved that delay in operating will further increase the threat of death or worsen the patient's condition. The fluid deficit, electrolyte abnormalities and acid base disturbances must be corrected preoperatively. Neonates and small infants are prone to hypoglycaemia with prolonged fasting so clear fluids are continued orally up to 2 hours preoperatively or dextrose is given IV if the child is unable to tolerate oral fluids.
- Induction of anaesthesia in the hypovolaemic patient can result in profound collapse - spinal anaesthesia is contraindicated in the hypovolaemic patient.
- The patient must be carefully assessed for signs of dehydration. The following parameters must be monitored: level of consciousness; condition of tongue and elasticity of skin; respiration; pulse; blood pressure. Hourly urine output after catheterisation - a urine output of 50ml/hour is desirable before surgery. Central venous pressure - this should be 8 –10 cms of water preoperatively. Clinical parameters are far more useful than relying on central pressures.
- The fluids used in replacement therapy will depend on the actual fluid lost.

  Fluids may be lost due to the following (see above):
  - Gastro-intestinal losses: vomiting; diarrhoea; fistulae; intestinal obstruction
  - Renal losses: diuretic therapy; chronic renal failure; diabetes mellitus
  - Skin losses: excessive sweating; hyperpyrexia; burns
  - Other causes of dehydration eg deficient intake.

For guidelines for replacement therapy: see page 539

Maintenance Therapy The patient may be in fluid balance preoperatively but the period of fasting superimposed before surgery may render the patient "dry" - this is more likely in:

- The very old patient
- Those who have been prepared for morning surgery but who have their surgery postponed till later in the day.

Maintenance fluids would include dextrose/water and perhaps electrolytes e.g. 5% dextrose/0.45% normal saline at the rate of 2ml/kg/hour.

Peri-operative management

Replacement therapy: Blood loss must be assessed carefully. The volume of blood in the suction unit and the weight of blood in the sponges must be determined. The blood on the drapes and floor should be taken into account. The volume of irrigation fluid used by the scrub nurse should be noted and deducted from the contents of the suction bottle. Blood loss is replaced with crystalloid solutions. 0.9% Saline or Hartmann's solution may be used in quantities of 3 - 4 times the blood lost. 5% Dextrose must not be used as it is dissipated rapidly into the tissue spaces and can result in water intoxication. Colloid solutions if available, are useful. Blood is needed when the loss reaches 15ml/kg (approximately 1 litre in an adult) provided the patient was not anaemic preoperatively. Auto transfusion may be considered. In cases of intestinal obstruction fluid aspirated from the gut must also be replaced by electrolyte solutions.

Maintenance therapy: Hartmann's solution if available or saline is used - 10ml/kg/hour in the first hour, 2-5 ml/kg/hour thereafter depending on the type of surgery. The larger volume in the first hour compensates for the period of fasting before surgery. Maintenance fluid makes up for the insensible water loss from the respiratory tract and also the operative site. Large wounds and large areas of exposed gut may produce insensible losses of up to 10 ml/kg/hour. The use of dry anaesthetic gases increases water loss.

Post-operative management

In the past it was common to restrict fluids in the post-operative period on the grounds that the kidney could not cope with the fluid load. Now it is believed that the body must be provided with an adequate amount of fluid in the post-operative period if the kidneys are to function properly.

Maintenance therapy:

- Water - the obligatory (i.e. minimal) water loss is 1500ml. (800 - 1000ml insensible loss and 500ml in urine).
- Carbohydrate is necessary to prevent the breakdown of fat and protein and provide energy.
- Electrolytes are required to avoid hyponatraemia and hypokalaemia. For daily requirements see p 538.

Daily input - 3 litres of fluid (e.g. 1 litre of 5% dextrose in 0.45% saline IV; 1 litre of Hartmann's IV or saline; 1 litre of 5% dextrose in 0.45% normal saline IV). In addition to this, abnormal losses should be replaced.

	Na^+ mmol/L	K^+ mmol/L	Cl^- mmol/L	CHO	Protein MW(da)	Half Life
Saline 0.9%	154		154			
Dextrose 5%				50		
Saline 0.45 / Dextrose 5%	77		77	50		
Hartmann's	131	5	112			
Haemaccel	145	5.1	145		35,000	5 hrs
Starches						17 days

Fig 48.2 Some common IV fluids

Potassium

The normal diet contains 1mmol/kg/day of potassium. Excretion of potassium is mainly through the kidney. Normal plasma potassium is 3.5 - 5.0mmol/L. A minimal amount is lost through the faeces and skin.

Hypokalaemia - a fall in the level of potassium in the blood may be due to

- Increased loss:
  - In the urine e.g. following diuretics
  - Through the gastrointestinal tract e.g. following diarrhoea
  - Sweating
- Decreased intake:
  - Fasting
  - The administration of potassium free IV fluids
- Shift of potassium into cells:
  - Insulin therapy
  - Administration of antacids.

Symptoms and signs of hypokalaemia

- Neuromuscular weakness
- Irritability - stupor
- ECG changes. Flat or inverted T wave
- Cardiac arrhythmias - especially in patients on digoxin treatment
- Paralytic ileus

Treatment:

- Intravenous potassium supplements
- KCl tablets
- Diet

If the serum potassium is between 2.5 – 4.5mmol then 30mmol/L of potassium is given per litre - 90mmol/day. If the serum potassium is less than 2.5mmol - 60mmol/L of potassium is given - 180mmol/day. Potassium should only be given if the patient has an adequate renal output. IV KCl should not be given rapidly. Maximum is 0.5mmol/kg/hour to allow equilibration with intracellular fluid.

Hyperkalaemia is associated with:

- Acute renal failure
- Severe acidosis
- In conditions of tissue break down eg. massive trauma

Signs and symptoms:

- Neuromuscular manifestations
- Cardiac irregularities.

Treatment:

- Calcium gluconate 5-10 ml of 10% IV
- Sodium bicarbonate 1.5-2mmol/kg IV
- Glucose insulin infusion 50 gm glucose + 20 units soluble insulin IV bolus then infusion. The blood sugar should be monitored.
- Ion exchange resin

Further details are available from a medical textbook.

COMPLICATIONS OF IV THERAPY

- Sepsis - thrombophlebitis - bacteraemia. Intravenous catheters and cannulae are a common source of infection. 33% of the IV catheters left in for 2 days become colonised with bacteria.
- Haematoma formation
- Pain at the site of venepuncture
- Fluid overload

ENTERAL FEEDING

Post-operative fluids do not always have to be given intravenously. They may be given by means of a naso-gastric tube or gastrostomy provided the gut is functioning normally. This is unlikely in abdominal surgery and following major surgery where opioids are used.

Naso-gastric feeding. This may be preferable to intravenous feeding long term because the needs of the patient can be provided without the hazards of an intravenous infusion (e.g. leakage around the vein, thrombosis, circulatory overload, fever, rigors, etc.). Again the gut must be beginning to work normally - some bowel sounds and the passage of flatus. The tubes used may be polyurethane or PVC. Polyurethane tubes can be left in place with minimal danger of irritation to the mucous membrane. A few practical points with regards to the passage of the tube:

- Lubricate the tube with a local anaesthetic ointment.
- Ask the patient to breath deeply and swallow when the tube reaches the back of the throat.
- Always check, (especially in the unconscious patient) that the tube has been inserted into the oesophagus and not into the trachea.
  - Aspirate the tube. Gastric contents confirm the placement of the tube in the stomach.
  - Dip the end of the tube in water. Ask the patient to exhale. If air leaves the tube with expiration, it is in the lungs.
  - Inflate air into the tube and listen for a gurgling noise in the epigastrium.
  - Direct visualisation of the position of the tube with a laryngoscope.

Enteral feeds are commercially produced containing the average daily nutritional requirements. Feed should be sterile. Liquidised food could be substituted if commercially prepared feed is unavailable but care must be taken in the preparation.

Dangers of enteral feeding

- Aspiration of gastric contents into the lungs - especially in comatose patients with an absent cough reflex.
- Diarrhoea
- Dehydration. The patients tend to receive diets high in carbohydrate and there is a relative dehydration.
- Nausea and vomiting
- Hyperglycaemia and other metabolic abnormalities
- Mechanical problems relating to the tube
  - Blockage
  - Migration
  - Oesophageal perforation

TOTAL PARENTERAL NUTRITION

This is the provision of food intravenously. Parenteral nutrition is indicated in the following cases:

- After major abdominal surgery where gastro - intestinal failure may follow
- In debilitated patients who are being prepared for surgery
- In burns, trauma, sepsis

Parenteral nutrition can be delivered via peripheral or central veins.

The following nutritional factors are provided:

- Water - 30ml/kg
- Protein 1gm/kg (amino acid mixture)
- Carbohydrates 2gm/kg (glucose in the form of a 10% solution)
- Fats 2gm/kg - (intralipid)
- Calories – 30kcal/kg
- Vitamins - A, B, C, D, E and K
- Minerals - further details can be obtained from more advanced texts.

CHAPTER 49

BLOOD TRANSFUSION

Outline:

History

Blood groups

Complications of blood transfusions

Blood and plasma products

Plasma expanders

Autotransfusion

Microfiltration of blood

Blood coagulation

Coagulation disorders

HISTORY

1666 Transfusions were performed in animals.
1667 Transfusions were performed in man using lamb's blood.
1818 The first successful 'man to man' transfusion took place (given to alter the patient's temperament).
1900 Landsteiner described the ABO blood groups.
1939 The Rh system was first described.

BLOOD GROUPS

There are several blood group systems, the two most important being the ABO system and the Rh system.

The ABO System

There are 2 antigens and 2 antibodies in the blood, all naturally occurring but not all of them occur in all people. The antigens are found in the red blood cells and are referred to as the A antigen and the B antigen. The antibodies occur in the serum and are referred to as the a and b antibodies.

There are therefore 4 kinds of blood, or 4 blood groups as they are called, according to their antigens and antibodies. The chart below sets them out.

Group A	Red blood cells have antigen A	Serum has antibody b
Group B	Red blood cells have antigen B	Serum has antibody a
Group O	Red blood cells have no antigens	Serum has antibodies a and b
Group AB	Red blood cells have antigens A and B	Serum has no antibodies

When a patient's blood is cross-matched, the donor's red blood cells are matched with the recipient's serum.

Group O was formerly classified as the universal donor, because the red cells of the group O donor had no antigens to react with the antibodies in the recipient's serum. O Rh negative blood is used in an emergency when there may be a delay in obtaining group-specific blood.

Group AB was referred to as the universal recipient, because the serum of the group AB patient had no antibodies which would react with the donor's antigen.

Unmatched group-specific blood (i.e. blood of the same ABO and Rh group) can be used in an emergency when the patient’s blood group is known. It has a very low risk of transfusion reaction and can be used while definitive cross-match is being undertaken.

The Rh system

The Rh antigen is called the D antigen. If this D antigen is present, the patient is said to be Rh positive. If it is absent, the patient is said to be Rh negative. The antibodies to the D antigen do not occur naturally, so if the patient lacks the D antigen it does not always follow that the patient has an (anti D) antibody. The antibody to the D antigen (anti D) occurs only if Rh-positive blood is transfused into an Rh-negative patient.

The antigen can also pass from an Rh-positive foetus to a Rh-negative mother and cause the production of d antibodies. These antibodies can return to the foetus and affect it adversely. Such effects on the foetus increase with each subsequent pregnancy as the antibody level in the mother builds up.

In earlier pregnancies the baby will be jaundiced, a condition referred to as erythroblastosis foetalis. In later pregnancies, death in utero may result (Hydrops Foetalis).

Rules

- No woman of child-bearing age should receive a Rh-positive transfusion if she is Rh negative.
- No Rh-negative patient at any age should receive a Rh-positive transfusion if a previous Rh-positive blood transfusion has been given.
- If available, anti-Rh immunoglobulins should be administered to Rh-negative mothers at delivery if the baby is Rh positive.

Rh-negative blood can be given if uncross–matched blood is required in a hurry because it contains no antibodies. Therefore, if uncross-matched blood is required, group specific Rh-negative blood is safest.

Techniques of blood grouping

Grouping and full cross-matching takes about 1 hour. A rapid cross-match can be done in half an hour to allow blood to be released for patients with severe haemorrhage while cross-matching continues to confirm compatibility.

Direct cross-match. One drop of the recipient's serum and one drop of the donor's blood are placed on a slide and then observed after 10 - 15 minutes. If there is any evidence of clumping, then the two bloods are not compatible.

COMPLICATIONS OF BLOOD TRANSFUSIONS

Topics covered

- Haemolytic reactions: intravascular and extravascular
- Contamination of blood
- Circulatory overload
- Air embolism
- Disease transmission e.g. malaria, syphilis, hepatitis, AIDS
- Febrile reaction
- Allergic reactions
- Hyperkalaemia (high potassium levels)
- Citrate intoxication
- Hypothermia
- Metabolic acidosis
- Coagulation problems
- Septicaemia
- Depression of reticulo-endothelial function
- Shocked lung syndrome
- Massive or large volume transfusion

HAEMOLYTIC REACTIONS

Intravascular haemolysis. The red blood cells are haemolysed or broken down after they are introduced into the patient's circulation. The cause of this haemolysis is incompatibility of the transfused blood with the patient's blood.

ABO incompatibility usually produces a violent reaction. The donor's red cells are destroyed by the antibodies in the recipient's serum. The signs of an incompatible blood transfusion may be masked by general anaesthesia.

The blood pressure and the pulse rate must be checked every 5 minutes for the first 15 minutes after each new bottle of blood.

A haemolytic reaction can be produced by just a small quantity of mismatched blood. Human error is frequently involved. Incorrect laboratory work; mislabelling of blood and misreading of the labels on blood bags and bottles, are frequently responsible.

Take maximum care in checking the name of the patient, the blood group, the bottle number and expiry date as shown on the blood product, with the slip issued from the laboratory. The patient's name and hospital number on the label of the blood pack should match those on the identification tag worn by the patient. Checking must be done by the anaesthetist and another responsible hospital staff member.

Signs of incompatible blood transfusion in the conscious patient

- Feeling of pressure in the head
- Tingling of limbs
- Precordial pain
- Dyspnoea
- Lumbar pain
- Restlessness
- Pyrexia
- Circulatory collapse (hypotension, tachycardia)
- Nausea and vomiting
- (Later) oliguria, haemoglobinuria.
- Skin rash

Some of these signs may be masked by anaesthesia and hypotension and oozing may be the only indication of an incompatible transfusion.

Signs of incompatible blood transfusion in the anaesthetised patient

- Hypotension
- Tachycardia
- Cyanosis
- Skin rash
- General oozing from the wound
- Later jaundice and oliguria (5 - 10% incidence).

Acute circulatory collapse and renal failure are the most serious complications and may lead to death.

The effects vary with the amount of blood transfused, the degree of incompatibility and the physical state of the patient at the time of transfusion.

The mortality from major incompatibility is 50%.

Mild reactions, particularly febrile ones, are due to antibodies to white cells and occasionally to platelets.

Treatment of a transfusion reaction

- Stop the transfusion.
- Treat hypotension appropriately (as for anaphylaxis).
  - Give fluids (crystalloid / colloid)
  - Give inotropes (e.g. adrenaline, 1-5ml of 1:10,000 solution IV, or dopamine)
  - Give other compatible blood, if necessary.
- Give oxygen
- Corticosteroids for severe reactions: Hydrocortisone 100-300mg.
- Establish the diagnosis with blood bank assistance. (See below)
- Treat renal vascular ischaemia with:
  - Saline, to keep urine output above 1ml/kg/hr (insert urinary catheter).
  - Frusemide 20 mg IV
  - Mannitol 0.5g/kg IV
- Evaluate and treat disseminated intravascular coagulation:
  - Coagulation screen if available (platelet count, activated partial thromboplastin time, APGT, fibrinogen, fibrin degradation products).
  - Platelet concentrate and fresh frozen plasma as indicated.
- Manage bronchospasm as for anaphylaxis:
  - Adrenaline 1-5ml of 1:10,000 or 0.1-0.5 ml of 1:1000 if necessary.
  - Salbutamol 250 - 500 micrograms IV.
- If patient has rigors and fever with no evidence of haemolysis:
  - Start antibiotics
- Try to distinguish between a transfusion reaction due to incompatible blood and an anaphylactic reaction to another drug, acute septicaemia and extravascular haemolysis. This can be very difficult as the clinical presentations can be similar.

Diagnosis of transfusion reaction

Send to the laboratory:

- The remaining blood in the bottle or bag
- A sample of the patient's blood
- A sample of the patient's urine

The diagnosis is established by haemoglobinaemia (haemoglobin in the plasma) and haemoglobinuria.

Extravascular haemolysis

This is where the haemolysis or breakdown of the red cells occurs before the cells enter the patient's circulation. The red cells break down in the bottle or bag.

Causes of extravascular haemolysis

- Old blood (more than 3 - 4 weeks after collection)
- Cold blood (stored at less than 2° C)
- Hot blood (heated above 38° C)
- Septic blood

CONTAMINATION OF BLOOD

Organisms may enter the blood by contamination through carelessness in collection or cross-matching. They will proliferate if blood is allowed to remain out of the refrigerator at room temperature. Blood that is left out of the refrigerator for more than 30 minutes should not be put back into storage.

CIRCULATORY OVERLOAD

During rapid transfusions there is always a risk of pulmonary oedema and cardiac failure. This is referred to as circulatory overload. This danger is more common in:

- Elderly patients
- Patients with severe anaemia
- Patients with heart disease, either ischaemic, valvular or hypertensive
- Patients receiving massive or large volume transfusions.

Precautions

Slow the transfusion if the patient's blood pressure is normal.

Check the central venous pressure. If it is more than 12cm of water with IPPV, or 5cm of water with the patient breathing spontaneously, then the infusion should be slowed because it shows that the patient is not hypovolaemic any more. Alternatively it may indicate that the patient is going into heart failure and this requires treatment.

In patients who are likely to go into pulmonary oedema it is advisable to give 20 - 40mg of frusemide IV at the commencement of the transfusion. This will promote a diuresis and prevent overload.

In patients likely to go into heart failure, use packed cells instead of whole blood. However, when there is acute haemorrhage then whole blood may be used to replace the blood lost, as it contains platelets and clotting factors. When massive transfusion is required, coagulation disorders are common and if facilities for obtaining FFP and platelet concentrates are not available, fresh or recently withdrawn whole blood may be the best alternative.

Check very carefully for signs of circulatory overload. Unanaesthetised patients can be propped up during transfusion.

Signs of circulatory overload

In the conscious patient

- Dyspnoea
- Cyanosis
- Engorgement of neck veins
- Basal crepitations
- Pulmonary oedema

In the anaesthetised patient (many of the above signs will be masked)

- Cyanosis
- Basal crepitations
- Reduced lung compliance (stiff lungs)
- Pink frothy secretions in ETT (pulmonary oedema)

Treatment of circulatory overload

- Stop the transfusion
- Give oxygen
- Prop up the patient or raise the head of the table
- Give diuretics: frusemide 40mg IV. This can be repeated.
- Give morphine (small doses, i.e. 1-2mg IV)
- Other measures include the application of tourniquets at a pressure less than the systolic and maintained for 15 minutes at a time.
- IPPV with 100% oxygen.

AIR EMBOLISM See Anaesthetic Complications in Chapter 46.

TRANSMISSION OF DISEASE

Some important viral diseases may be transmitted in transfused blood especially where screening tests are not available.

- Viral hepatitis (jaundice may occur between 25 and 180 days)
- HIV
- Malaria
- Syphilis

FEBRILE REACTIONS (3 - 6% incidence)

This is evidenced by a rise in temperature, shivering and malaise. It may be due to pyrogens which are high molecular weight lipopolysaccharides of bacterial origin. A febrile reaction can also be due to white blood cell antigens following previous transfusions or pregnancy.

ALLERGIC REACTIONS

These may be caused by antibodies to white blood cells, platelets or immunoglobulin reactions between patient and donor.
Mild urticaria is the commonest manifestation. More serious allergic reactions are:

- Asthma
- Angioneurotic oedema
- Laryngeal oedema
- Cardiovascular collapse
- Anaphylactic reaction (rare).

Some allergic reactions may be difficult to distinguish at an early stage from haemolytic reactions.

Treatment

Mild allergic reactions: give antihistamine e.g. chlorpheniramine (Piriton) 10-20mg IV.

Severe reactions:

- Stop the transfusion
- Give adrenaline 0.1-0.5mg IV i.e. 0.1- 0.5ml of 1:1000 solution or 1-5ml of 1:10,000 solution (note: the dose of adrenaline should be titrated to effect and side-effects)
- Give hydrocortisone 100mg IV and repeat
- Treat bronchospasm if it occurs.

See section on Anaphylaxis (Chapter 62).

HYPERKALAEMIA (high potassium levels)

Red blood cells contain a very high concentration of potassium (about 100mmol/L). Plasma contains 3.5 - 5 mmol/L. Old blood (after 3 - 4 weeks) has about 25mmol/L of potassium in its plasma. The potassium from the red blood cells diffuses out into the plasma. If patients have a high serum potassium **or** there is renal failure (which is usually associated with a high serum potassium), fresh blood or blood stored for less than 5 days should be given. However after transfusion potassium is taken back into the red cells and hyperkalaemia is rare. Patients may even develop hypokalaemia.

CITRATE INTOXICATION

Whole blood is prevented from clotting (after collection), by the use of citrate. The citrate forms a complex with the calcium ions in the blood. Since calcium is necessary for the blood to clot and the calcium is not available (being tied up in a complex with citrate), the blood remains fluid. When the citrated blood is introduced into the patient's circulation, the citrate is quickly broken down into water and carbon dioxide and the calcium is set free. If for some reason the citrate persists in the circulation, signs of citrate intoxication are seen. These signs are essentially the signs of lack of calcium in the blood.

Signs of citrate intoxication (and therefore calcium lack)

- Cardiac depression
- Increased oozing
- Tetany (carpal spasm and contraction of facial musculature on tapping the facial nerve)

Citrate intoxication is rare after transfusion but it may be seen in:

- Massive transfusions,
- Transfusions in the presence of:
  - Liver disease
  - Renal disease
  - Hypothermia
  - Hypotension

Treatment

Give 10ml of 10% calcium gluconate or chloride for every 6 units of blood transfused.

HYPOTHERMIA (for more detail on this topic see Chapter 51)

If the patient receives a significant transfusion of cold blood, then the body temperature will fall. This can be prevented in the case of large transfusions by using a blood warmer, which warms the blood as it passes through the tubing to the patient.

Hypothermia is associated with:

Cardiovascular effects

- Reduced cardiac output
- Arrhythmias
- VF at 28°C
- Vasoconstriction
- Increased blood viscosity
- Reduced peripheral perfusion

Respiratory effects

- A shift of the oxygen dissociation curve to the left, resulting in a reduced tissue oxygen (O_2) delivery, especially in combination with reduced cardiac output and vasoconstriction
- Reduced oxygen demand and CO_2 production

Neurological effects

- Confusion below 35°C
- Unconsciousness at 30°C

Other effects

- Diuresis due to inability to re-absorb sodium and water
- Respiratory metabolic acidosis
- Increased blood glucose and potassium
- An initial increase in metabolic rate followed by a decrease
- Decreased drug metabolism with prolonged drug effects
- Increased platelet aggregation and thrombocytopenia

Hypothermia due to blood transfusion is rarely so severe unless in combination with other causes.

METABOLIC ACIDOSIS

Stored blood is more acid than blood in the circulation. It has a lower pH i.e. more hydrogen ions.

This acidity is due to the accumulation of metabolic acids, e.g. lactic and pyruvic acids and also citric acid. If large quantities of the acid blood are introduced into the patient's circulation, a state of metabolic acidosis may result. This is usually only temporary as the citrate in stored blood is metabolised in the circulation to bicarbonate.

COAGULATION PROBLEMS

These may be caused by:

- A reduction in the number of functioning platelets.
- A reduction in the coagulation factors in old blood. These can both be corrected by giving fresh frozen plasma and platelet concentrate.
- A reduction in the available calcium, especially in citrate intoxication
- D.I.C. (disseminated intravascular coagulation) or consumption coagulopathy leading to a fall in platelets and fibrinogen and an increase in fibrin degradation products (FDP).
- Depletion of inhibitors of coagulation.

These problems may begin to occur after 6 units of packed cells or stored blood have been administered.

SEPTICAEMIA

Contaminated blood or a contaminated IV cannula tip can introduce organisms into the blood stream.

DEPRESSION OF RETICULO-ENDOTHELIAL FUNCTION

Massive transfusion is associated with a depression of the reticulo-endothelial system (RES). This is a system whereby leucocytes, mainly monocytes, are involved in fighting and digesting foreign organisms in the body.

SHOCKED LUNG SYNDROME See Microfiltration of Blood later in this chapter.

MASSIVE TRANSFUSION

This is defined as the replacement of the total blood volume in less than 24 hours or of 1.5 times the circulating volume over a longer period. Coagulation problems commonly result. Platelets and fresh blood or plasma are required.

Precautions when transfusing large volumes of blood:

- Use 2 units of fresh frozen plasma for every 6 units of CPD bank blood. Fresh frozen plasma contains all the clotting factors except platelets. Where fresh frozen plasma or platelets are not available, fresh blood should be used if possible to provide clotting factors and platelets.
- Do platelet counts after every 6 units of blood. Give platelets if the count is less than 75,000/ mm^3.
- Give calcium gluconate 10% (or chloride) at the rate of 10ml/6 units of bank blood. Give calcium solutions slowly over a period of 5 minutes. If ECG monitoring is available it should be used during calcium administration.
- Monitor: pulse, blood pressure, ECG, CVP, urine output, acid base state and serum electrolytes if possible.

Blood loss of **less than 1 litre** can be replaced with normal saline or Hartmann's solution. Because of redistribution of electrolyte solutions, it may be necessary to give up to 3 times the volume of blood lost.

If blood loss is **greater than 1 litre**, replace the blood loss with at least 2 litres of normal saline or Hartmann's solution then with blood or colloid solutions, depending on the clinical situation. If surgical bleeding is not corrected then blood is necessary until the patient is surgically stabilised. Blood may often be needed after only 1 litre blood loss if the patient is anaemic.

Always have blood available in any situation where this degree of blood loss is anticipated. Fit adults will tolerate Hb levels of 7 g/dL as long as bleeding is controlled and blood volume is restored. Seriously ill patients may need Hb levels maintained around 9 g/dL.

BLOOD AND PLASMA PRODUCTS

Whole blood (stored blood)

A unit of whole blood consists of 450ml of donor blood mixed with 63ml of citrate phosphate dextrose anticoagulant (CPD). CPD blood when stored at $4^{o}C$ has a shelf life of 28 days.
Adenine is now usually added to CPD (CPDA) extending the shelf life of stored blood to 35 days and other additive solutions such as mannitol can extend this to 42 days.
Whole blood is low in platelets and factors V and VIII unless it is transfused within 6 hours of collection. Whole blood when stored contains a high concentration of potassium and hydrogen ions.
The main use of whole blood is in the restoration of blood volume in patients in hypovolaemic shock. This may follow haemorrhage due to trauma or gastrointestinal bleeding.
One unit of blood raises the Hb by about 1g /dL. Before major surgery it is desirable that the Hb is at least 8g /dL (preferably higher).

Fresh whole blood

This is used as soon as possible after collection. It is indicated in those patients who have a low haemoglobin and also a low platelet count, less than $40,000/mm^3$. Such patients are:

- Those with thrombocytopenia (low platelet count) who are bleeding.
- Those who have received a massive transfusion with stored blood and who are low in platelets and labile clotting factors.

Packed Cells

These are prepared by centrifuging whole blood and removing 200ml of plasma per unit. Packed cells have a volume of 300ml and a shelf life of 21 days at 4°C.
Each unit of packed cells raises the Hb by 1-1.5g /dL.

Indications for packed cells

- Chronic anaemia in the normovolaemic patient whose circulation must not be overloaded
- Patients with cardiac failure
- Very old patients
- Those with chronic sepsis

Packed cells can be transfused along with normal saline or colloid and used in the treatment of shock. Since packed cells contain only about 80ml of plasma (as compared to 250ml of plasma in whole blood) the amount of antibodies introduced into the patient and hence the incidence of adverse reactions is reduced.

Fresh frozen plasma

This is prepared from whole blood within 6 hours of collection. It is frozen immediately and hence carries the labile clotting factors V and VIII. It has a shelf life of 3 months at -20°C and 1 year at -30°C.
Thawing of fresh frozen plasma must be done gradually. It takes 30 minutes at 37°C to thaw and it must be infused soon after thawing.

Indications for the use of fresh frozen plasma: Replacement of clotting factors:

- After massive transfusions
- In haemophilia
- Following anticoagulant overdose
- DIC (Disseminated Intravascular Coagulation)
- Bleeding from liver disease

Cryoprecipitate

This is obtained by centrifuging the refrigerated plasma. It has a very high concentration of fibrinogen and factor VIII. It carries a risk of hepatitis.

- Useful in the treatment of haemophilia and other deficiency states.
- After massive transfusion if fibrinogen is < 1.5g/L.

Platelet concentrates

The normal platelet count is around 250,000/mm^3. Bleeding during surgery occurs if the platelet count is less than 50,000/mm^3.
Spontaneous bleeding (in the non-surgical patient) occurs with levels less than 10,000/mm^3.
A low platelet count can be detected on a peripheral blood smear. A knowledge of the platelet count and platelet function is important. Platelet deficiency can be treated with one of the following:

- Platelet concentrate
- Whole blood within 6 hours of collection
- Platelet rich plasma.

Human albumin solutions (HAS)

These solutions contain 4.5% or 20% of human albumin. They are heat-treated and carry little risk of hepatitis but contain no clotting factors.
They have little role in acute replacement management during surgery.
The 4.5% preparation can be used as a colloid following high albumin loss e.g. in burns.
The 20% preparation is used in the treatment of hypo-proteinaemia eg in liver failure.
These solutions are very expensive.

PLASMA EXPANDERS

These are solutions which contain high molecular weight proteins which are unable to diffuse through normal capillary membranes and therefore stay within and expand the intravascular volume.
These include plasma protein preparations and artificial plasma substitutes.

Indications

- Providing intravascular volume expansion where blood is not available.
- Allowing time for full cross-matching of blood to be performed.
- Replacement of plasma losses in
  - Anaphylaxis
  - Plasmapheresis
  - Burns
- Conserving donor blood, by providing volume replacement.

These are dealt with in detail in Chapter 52 under the heading of Shock. It is important to be thoroughly familiar with the plasma expanders available.

Adverse effects of plasma volume expanders

- Fluid overload, so careful monitoring is essential
- Contamination and infection, though the risk of infection is less than with whole blood
- Allergic and anaphylactoid reactions
- Effects on haemostasis: dextran 70 interferes with coagulation
- Effect on renal function: dextran 40 may be associated with renal failure
- Effect on cross-matching: dextran 70 may interfere with cross-matching of blood
- Incomplete metabolism and storage: HES is found in the body years after administration

Solution Content	Half-life	Distribution *extra vascular*	Distribution *intra vascular*	Osmotic Activity	Na (mmol/L)
Albumin 4.5%	5-10 days	20%	80%	iso-osmotic	100-160
Albumin 20%	5-10 days	20%	80%	hyperosmotic	50-120
should be reserved for use where colloid osmotic pressure is low					
Dextran 40 (rheomacrodex) in 10% saline or dextrose	6-9 hours		100%	hyperosmotic	154
Dextran 70 (macrodex) 6% saline or gelatin	12 hours		100%	hyperosmotic	154
do not exceed 1.5L in adults					
Modified gelatin (haemaccel) (gelofusine	4 hours	50%	50%	iso-osmotic	145
Hydroxy ethyl starch (HES) used in UK/USA	24 hours		100%	iso-osmotic	154

AUTOTRANSFUSION

Autotransfusion is a procedure in which blood is collected from a patient and later reinfused i.e. donor and recipient are the same person.
These techniques are commonly used in situations where blood from the blood bank is either limited in quantity or unavailable.
The technique of autotransfusion has been used frequently, in volumes up to 1.5 litres.

Advantages of autotransfusion

- Fresh blood is transfused at a normal temperature
- Platelets and coagulation factors are present. The red blood cells that survive autotransfusion are said to have a normal life span.
- The blood is truly compatible
- There is no chance of disease transmission

Disadvantages of autotransfusion

These are proportional to the volume used.

- There may be damage to red blood cells, fibrinogen and platelets, caused by mechanical injury associated with collection and also by extravascular contact. Platelet transfusions and fresh frozen plasma may still be required after large volumes of autologous blood.
- Post-traumatic pulmonary insufficiency, which may be caused by the presence of micro-aggregates in the lung. Furthermore, the platelets may release vasoactive substances that may produce vasoconstriction and bronchospasm.
- Occasionally renal failure has been encountered.

Indications for intra-operative auto-transfusion

- Ruptured ectopic pregnancies
- Blunt or penetrating trauma to chest and abdomen eg ruptured spleen, haemothorax.

Contraindications to intra-operative autotransfusion

- Infection in the peritoneal cavity
- Malignancy
- Contamination of the peritoneal cavity from the gastrointestinal tract
- Long delay since onset of bleeding. Acute blood loss must be recent (within a few hours).

Methods used:

- Salvaging at operation
- Pre-donation
- Haemodilution at induction of anaesthesia

Salvaging at operation

Autotransfusion is performed during surgery in patients who are bleeding profusely.
The blood is collected from the surgical field into a sterile receptacle containing citrate as an anticoagulant (63 ml in a 500ml bottle). Heparin may also be used.
The Bentley autotransfusion unit aspirates blood, defoams and filters it through a 125 micropore nylon filter. In the absence of this expensive piece of equipment, the blood is filtered through 3 - 4 layers of sterile gauze on a sterile glass funnel into a second sterile bottle. A sterile rubber bung is placed in the mouth of the bottle and the blood handed to the anaesthetist for transfusion. The collection and filtering are performed by the nurse assisting the surgeon.

Fig 49.1 Making a small opening in the peritoneum to collect the blood

Fig 49.2 Filtering the collected blood to remove small clots and tissue

Fig 49.3 Transferring filtered blood to a transfusion bottle containing anti-coagulant solution

Fig 49.4 Blood being re-transfused into the patient

Pre-donation

Blood may be collected 2-3 weeks before surgery and the patient given iron medication.
The deficit is corrected within 3-5 days

Preoperative haemodilution

This is performed immediately prior to induction of anaesthesia.
One or two units of blood are withdrawn into bottles containing anticoagulant and replaced by an equal volume of colloid, or 3 times the volume of normal saline. Once surgery is complete and haemostasis is achieved the blood is returned to the patient.

Indications for pre-donation and preoperative haemodilution

- Elective surgery if massive blood loss is anticipated.
- Rare blood types with antibodies which preclude cross-matching.
- Patients who are Jehovah's Witnesses refuse blood transfusion. Autotransfusion may be performed by bleeding the patient 2 weeks before surgery and transfusing the blood during surgery though many will not accept this method.

MICROFILTRATION OF BLOOD

Micro-aggregates are composed of

- Degenerated platelets
- White blood cells
- Fibrin strands
- Denatured proteins
- Damaged red blood cells (present especially after the first week of storage).

The rate of formation of micro-aggregates depends on length of storage, platelet and WBC count at time of collection and the anticoagulant used. Heparin / CPD / ACD.

Effect of micro-aggregates:

They are filtered by the pulmonary circulation and may cause histamine release and pulmonary insufficiency from increased pulmonary resistance and ventilation-perfusion mismatching.
These pulmonary microemboli can be prevented by transfusing blood that is as fresh as possible and by using membrane filters with a small pore size of 20 - 40 microns although this slows the rate of infusion.
(Standard blood transfusion sets have a pore size of 170-260 microns).

BLOOD COAGULATION

Mechanism

Blood coagulation is a complex process. Thirteen clotting factors have been identified. Medical textbooks will give more details.

Factor I is fibrinogen, a plasma protein.
Factor II is prothrombin, formed in the liver. It decreases in hepatic disease. It is dependent on vitamin K for synthesis.
Factor III is the tissue factor. Tissues extract thromboplastin.
Factor IV is calcium.
Factors V – XIII

The main reaction in the coagulation mechanism is the conversion of prothrombin to thrombin. Thrombin converts fibrinogen to form fibrin. Two pathways lead to this event:

- The intrinsic blood clotting system. This is not associated with tissue injury. Clotting occurs as a result of interaction of blood constituents and is activated by surface contact or vascular injury.
- The extrinsic system, activated by tissue damage. A simplified version is shown in the following chart.

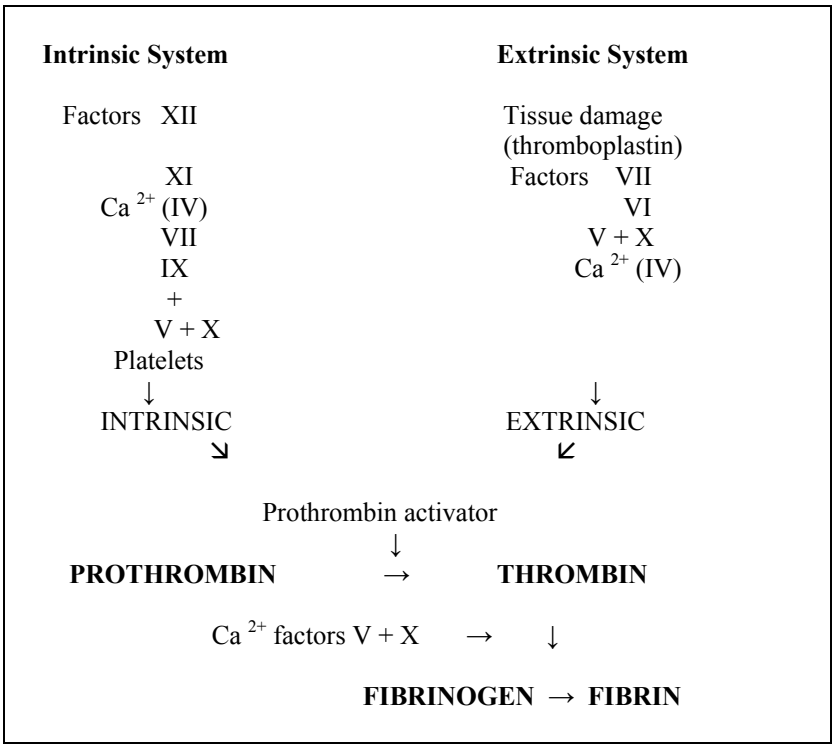


Fig 49.5 Simplified scheme of the coagulation mechanism

Importance of the coagulation process to the anaesthetist:

- A deficiency of the clotting factors will result in a bleeding tendency. Specific diseases are caused by the absence of specific clotting factors. Patients may present for surgery with these diseases.
- Massive transfusions may result in a deficiency of some clotting factors, which are absent in stored blood. These deficiencies may have to be replaced.
- Disseminated intravascular coagulation may be seen in the surgical patient. The intravascular clotting uses up the clotting factors, resulting in a haemorrhagic tendency. The clotting factors may have to be replaced.

Factors limiting coagulation

- Fibrinolysis (breakdown of fibrin) or clot dissolution takes place by the action of the enzyme plasminogen. The fibrin fragments thus formed inhibit further clot formation.
- The smooth lining of the endothelium of blood vessels prevents the intrinsic thromboplastin formation.
- A rapid flow of blood in the blood vessels keeps the blood fluid.
- A very low concentration of heparin is found naturally in the blood and this may make the blood more fluid.

Factors predisposing to coagulation

A thrombus is a solid mass, formed in the living heart or blood vessels from the constituents of blood. Initially there is local damage to the vascular endothelium or a slowing of the blood flow. This leads to platelet accumulation at the site. Thromboplastin is liberated and further clot formation occurs at the site.

The following factors predispose to post-operative thrombosis.

- Slowing of blood flow
- Changes in the composition of blood after trauma: i.e. increase in platelets and increase in fibrinogen
- Damage to the lining of blood vessels
- Sepsis – organisms can produce a toxin which leads to clotting.

Drugs inhibiting coagulation (Anticoagulant drugs)

Heparin

This occurs in the circulation in low concentrations.
It can be given IV, IM or subcutaneously and inhibits coagulation in the following ways:

- It potentiates the action of antithrombin
- It neutralises the action of thrombin in converting fibrinogen to fibrin
- It inhibits the platelet aggregation effect of fibrin

Heparin is neutralized by protamine sulphate.

Vitamin K antagonists

Dicoumarol (Warfarin) is the most commonly used example.
These agents interfere with the action of vitamin K in the synthesis of several clotting factors. They therefore prevent prothrombin synthesis. They are used in the treatment of deep vein thrombosis and coronary thrombosis.

Citrate

This is added to blood collected for transfusion and is only active in stored blood. The citrate forms a complex with the calcium, which is thus not available for clotting. The blood therefore remains fluid. After the blood is transfused the citrate is rapidly metabolised in the liver, the calcium is set free and is then available for use in the clotting process.

Bleeding disorders

Clotting plays an important role in haemostasis and the clotting process depends on several factors.
Defects of coagulation can be identified more specifically by the following tests.

Coagulation tests (check normal values with your lab):

- Bleeding time 2 - 10 minutes
- Clotting time 6 - 12 minutes in glass
- Platelet count (150,000 - 250,000). If the platelet count is low it is termed thrombocytopenia.
- Prothrombin index. This estimates defects in the extrinsic system (i.e. Factors VIII and X and V and prothrombin concentrations)
- PTTK or APTT. These test the intrinsic and common pathways

COAGULATION DISORDERS

A lack of clotting factors results in coagulation disorders. These disorders can be congenital or acquired.

Congenital coagulation disorders

- Haemophilia is an inherited disorder associated with a lack of Factor VIII.
- Christmas disease is a bleeding tendency associated with a lack of Factor IX.

Acquired coagulation disorders

- Multiple transfusion. After multiple transfusions of stored blood there will be a deficiency of clotting factors, which can be treated with:
  - Fresh blood
  - Fresh frozen plasma
  - Cryoprecipitate.
- Vitamin K deficiency. Vitamin K is found in green vegetables and in meat. It can also be synthesised by the bacteria in the intestine. It is absorbed from the intestine (with the help of bile salts) and conveyed to the liver. There it helps in the production of prothrombin, which is necessary for clotting. The following conditions are associated with vitamin K deficiency:
  - Obstructive jaundice
  - Haemorrhagic state in infants. Here the lack of vitamin K may be associated with a lack of bacteria in the gut.
  - Chronic diarrhoea, which may result in defective absorption of vitamin K or a change in the bacteria of the gut.
  - Liver disease. There may be a failure in prothrombin production. This condition does not respond to vitamin K administration.
- Disseminated intravascular coagulation (DIC)
  Coagulation and fibrinolysis are usually in equilibrium. If the coagulation mechanism is activated and the clotting factors are used up, intravascular thrombosis occurs with bleeding i.e. there is inappropriate triggering of coagulation in flowing blood by disease processes.

Some causes of DIC

- Shock, resulting in inadequate capillary perfusion, haemolysis, trauma, burns, septicaemia and anaphylaxis
- Haemolytic reactions such as those from snake venom or after blood transfusion
- Obstetric problems such as concealed ante-partum haemorrhage, abortion, amniotic fluid embolism and pre-eclampsia
- Cancer of the prostate
- Extra-corporeal circulation (heart-lung bypass)
- Massive transfusion

Diagnosis of DIC

- Low platelet count
- Afibrinogenaemia or low plasma fibrinogen level
- Increased fibrinogen degradation products

Treatment

- Treat the cause e.g. treat septicaemia, empty uterus, expand blood volume
- Supportive: respiratory, renal and cardiovascular systems
- Replace sufficient clotting factors with
  - Fresh frozen plasma
  - Platelets
  - Possibly cryoprecipitate
  - Fresh blood

CHAPTER 50

MONITORING DURING ANAESTHESIA

Outline:

Cardiovascular system:
- Pulse
- Blood pressure
- Central venous pressure
- The electrocardiograph
- Blood loss

Respiratory system:
- Tidal volume
- Respiratory rate
- Colour of blood
- Arterial gases
- Oximetry
- Capnography

Urinary system

Temperature

Neuromuscular blockade

Other parameters monitored in special units

Monitoring equipment should never replace sound clinical judgment and common sense. The anaesthetist must always remain with the anaesthetised patient and must be aware of every problem, surgical and otherwise, to which the patient is exposed. Good communication with the surgeon is therefore essential.
The colour of the blood, mucous membrane of the mouth, skin temperature and the patient's depth of anaesthesia must be constantly monitored.

CARDIOVASCULAR SYSTEM

Pulse

Always make sure there is access to the pulse when the patient is draped. The radial or the brachial pulse is usually palpated. Note the rate and the regularity of the pulse, every 5 minutes in the average patient and even more often in the very ill patient. The volume of the pulse is important. It is a good habit to place a hand that is free on the patient's pulse whenever possible. The normal pulse rate in adults varies between 50 and 90 beats per minute.

Blood pressure (BP)

Measure blood pressure is normally measured with the use of a sphygmomanometer. Monitor and chart it every five minutes in patients who are stable and more often in ill patients. Both systolic and diastolic blood pressures must be recorded. The blood pressure cuff must be wide enough to cover two-thirds the length of the upper arm.

Palpation method The pressure in the cuff is increased to a point above which the radial artery is impalpable, then decreased at a rate of 2-3 mmHg per second. The point at which the radial pulse re-appears is the systolic blood pressure.

Auscultation method Both systolic and diastolic blood pressure are obtained by this method. The systolic blood pressure is about 10 mmHg. above that obtained by the palpation method.

Oscillotonometer. The blood pressure may be recorded by noting the oscillations of a needle in the oscillotonometer.

Automatic electronic devices are commonly used today.
The normal blood pressure varies. For an adult it is 100/70 to 140/90, while for babies and small children it is 80 or 90 systolic and increases gradually with age.

Central venous pressure (CVP)

The CVP is the pressure in the venous side of the circulation measured by a catheter inserted into the large veins opening into the heart. Normal CVP is from 5 to 10 cm of water.

About 80% of the blood volume is contained in the venous circulation, so when the blood volume falls, the venous pressure falls. Venous pressure falls before arterial pressure. The measurement of the venous pressure gives us an early indication of blood loss. If the patient has been transfused and the blood volume has been increased, the venous pressure rises. The venous pressure also rises if the heart is failing. When the heart action fails there is a banking up of blood on the venous side of the circulation. The central venous pressure should always be interpreted together with the other parameters monitored - arterial blood pressure, pulse, peripheral circulation, etc.

Guide to CVP measurements

CVP	***BP***	***Diagnosis***	***Fluids***	***Drugs***
Low	Low or normal	Fluid Lack	Increase	
High	Normal	Fluid overload	Reduce	Diuretics e.g. frusemide
High	Low	Cardiac Failure	Restrict	Diuretics Digoxin- (for rapid AF) IV inotropes eg dobutamine

Indications for CVP monitoring

- Whenever massive infusions or transfusions are given
- In severe shock
- In patients with heart failure receiving fluid therapy
- In acute cardiac failure of obscure origin.

The Electrocardiograph (ECG)

The ECG gives an accurate assessment of:

- Rate
- Rhythm of the heart
- The degree of oxygenation of the heart muscle. Hypoxia may show as a change in the ST segment. Ideally, all patients anaesthetised should be monitored electrocardiographically.

The ECG is especially useful in:

- Patients with heart disease such as arrhythmias, ischaemia, hypertension, heart failure
- Patients who have a previous history of anaesthetic problems
- Patients who are shocked, hypotensive or bleeding
- Patients who are poor risks due to underlying medical conditions, e.g. thyrotoxicosis, diabetes, renal or liver disease.

Blood loss

Blood loss can be measured by several methods:

The gravimetric method: The swabs, sponges and packs are weighed dry and then with the absorbed blood. The difference in weight gives the weight of the blood absorbed. One gram of blood is then taken to be equal to 1ml of blood. To this weight is added the volume of blood in the suction bottles. A further 25% should be added to the total to account for the blood in the drapes, etc.

Colorimetric method: The swabs and sponges are mixed with a large known volume of liquid and the haemoglobin level of the resultant solution is estimated. By using a formula the blood loss may be worked out.

RESPIRATORY SYSTEM

Tidal volume

To estimate the tidal volume you need a spirometer.
A Wright's respirometer is commonly used as it is small, portable and does not require any power supply. The gas flow drives a spinning vane in one direction only and the speed of rotation is converted to volume which is then displayed on a dial.

The Respiratory Rate

This should be noted, especially in the case of spontaneous respiration.

Colour of blood

This must be constantly observed. It shows how well oxygenated the patient is and therefore gives an idea of the respiratory and cardiovascular systems but is not nearly as accurate as pulse oximetry.

Pulse oximetry

Arterial oxygen saturation (SaO_2) is the measure of oxygenation of the blood. The relationship between oxygen saturation of haemoglobin (Hb) and the partial pressure of oxygen in the blood is not linear (See Respiratory physiology in Chapter 3). As saturation drops below 80% partial pressure drops dramatically. Any value below 90% is clinically dangerous.

A small sensor is placed on the end of the finger, toe or ear lobe. The sensor consists of 2 light-emitting diodes one red and one infrared illuminating in turn. They emit light of 650 nanometers for oxyhaemoglobin (HbO_2) and 900 nanometers for reduced or de-oxyhaemoglobin (Hb). The blood absorbs this light depending on the content of HbO_2 & Hb. The photo diode detects the residual light energy. This is interpreted as a Hb/HbO_2 ratio and the resultant SaO_2 is calculated and presented on the machine. The pulse oximeter depends therefore on the pulsatile perfusion of the tissues and so doubles up as a pulse meter. Pulse oximetry must be interpreted with caution in the following conditions:

When abnormal Haemoglobins are present

- Carboxyhaemoglobin (smokers may have 10-20 % carboxy Hb). The pulse oximeter does not distinguish between oxyhaemoglobin and carboxyhaemoglobin and in heavy smokers the reading will be falsely high.
  Following poisoning with carbon monoxide a pulse oximeter is very inaccurate.
- Methaemoglobin. This is normally only 1% but it rare cases can reach levels of over 20%. Methaemoglobinaemia can be caused by certain drugs e.g. nitrites, sulphonamides and prilocaine and some poisons. It can also occur spontaneously. It is of importance to the anaesthetist as it reduces the oxygen carrying capacity of the blood and shifts the oxygen dissociation curve to the left. Treatment is with methylene blue 1% given IV at a dose of 1–2mg / kg over 5 mins.

When limb blood flow is low

- Low blood pressure
- Hypothermia
- Vasoconstriction from any cause
- Peripheral vascular disease.

Miscellaneous causes

- High intensity light
- Heat lamps
- IV dyes such as methylene blue
- Electrocautery
- Venous congestion.

Capnography

A capnograph measures carbon dioxide in inspired and expired gases using infra–red absorption. The information can be displayed in numerical form or as a waveform or capnograph tracing.
Hypercapnia (also called hypercarbia) means an abnormally high carbon dioxide.
Hypocapnia (also called hypocarbia) means an abnormally low carbon dioxide.

Carbon dioxide is produced in the tissues as a result of cellular metabolism. It is carried from the tissues in the venous blood to the right side of the heart and from there into the lungs via the pulmonary artery. The pulmonary arterioles perfuse the alveoli of the lung. The carbon dioxide diffuses from the arteriole into the alveolus and via the expiratory gases through the upper airway into the atmosphere.
If this process of elimination is interfered with, carbon dioxide accumulates in the blood stream (See Chapter 3 Respiratory failure).
The closer to the alveoli we sample the gas, the more accurately will the sample reflect the changes in the alveoli and in the blood stream.

Methods of sampling of end-tidal CO_2

Mainstream The gas is analysed as it passes through the circuit.
Sidestream Here the gas is led through a small capillary tube to a capnograph and then analysed. Hence a slight delay in analysis.

Principle of carbon dioxide measurement

This depends on infra-red absorption. An infra-red light beam is projected through a gas source and the intensity of transmitted light is measured. The difference will indicate the amount of carbon dioxide absorbed.

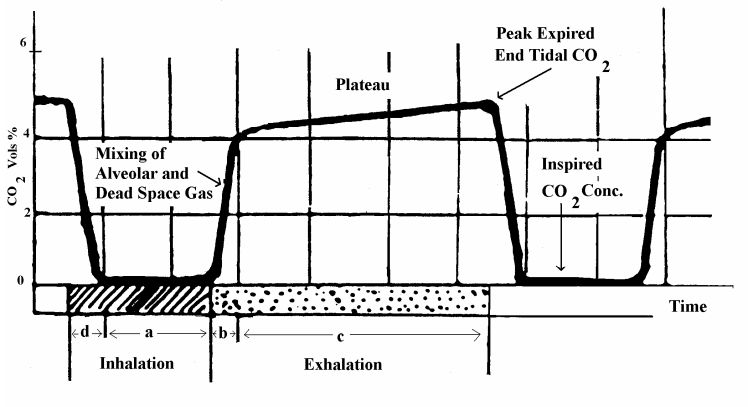


Fig 50.1 Capnograph tracing

The normal capnograph is shown in the diagram. Any significant change indicates respiratory abnormality, a mechanical defect in the circuit or a complication.

Description of the capnograph

Phase b (2nd phase ascending part) represents exhalation. Carbon dioxide builds up during exhalation as the gases containing it are expired from the alveoli into the atmosphere via the sensor.

Phase c (3rd phase plateau) This remains nearly constant until the next inspiration sweeps the carbon dioxide away.

Phase d (4th phase descending part) The fresh gas flow entering the lungs during inspiration contains no carbon dioxide. The carbon dioxide level falls rapidly to the base line, indicating the start of inspiration.

Phase a (1st phase) The capnograph remains steady at zero, indicating inspiration.

How to interpret the capnograph

Phase b abnormal A slanted upstroke means

- Uneven emptying of the lungs. Carbon dioxide rises at different times (a gradual rise).
- The sampling mechanism is too slow.

Phase c The Plateau.

Normal peak values are 36 - 44 mmHg. The highest carbon dioxide is found at the end of expiration and is called the End tidal carbon dioxide ($EtCO_2$)

High plateau (greater than 44 mmHg) may be caused by

- Hypoventilation:
  - The effect of anaesthesia and opioids on the spontaneously ventilating patient
  - Respiratory failure (see Chapter 57)
- Increased production of carbon dioxide, secondary to malignant hyperpyrexia, fever.
- Transient increase in carbon dioxide:
  - Release of limb tourniquets after surgery
  - IV sodium bicarbonate ($NaHCO_3$)
  - Insufflation of carbon dioxide – laparoscopy

Low plateau (less than 36 mmHg)

- Hyperventilation (normal minute volume 100 - 120 ml/kg)– caused by hypoxia, fever, (hysteria in awake patient).
- Low carbon dioxide delivery to the lungs:
  - Hypotension
  - Pulmonary embolus
  - Decreased cardiac output from any cause.

Irregular plateau indicates a physiological cause - a dip showing an attempt to breathe spontaneously e.g. muscle relaxant wearing off.
Slanted plateau indicates COAD or asthma.
Shorter plateau indicates a leak in the system.

Phase d Slanted downstroke
Normally the downward slant is very brisk (almost vertical). If it is slow it may mean there is carbon dioxide in the inhaled gases, or the inspiratory valve is not functioning properly.

Phase a
An abnormal base line means the inspired air contains carbon dioxide.

Uses of Capnography

- Confirming intubation of the trachea. A normal capnograph indicates tracheal intubation. There is no carbon dioxide detected with oesophageal intubation.
- Apnoea. The capnograph alarms when the next wave of carbon dioxide fails to arrive. This reveals apnoea from whatever cause (i.e. airway obstruction, drugs such as excessive opioid usage or inadvertent muscle relaxant etc.).
- Warning of a disconnection in the circuit.
- Measuring the adequacy of mechanical ventilation.
- Diagnosis of rebreathing.
- Measuring the adequacy of cardiac output (i.e. PE or cardiac event such as acute myocardial infarction).
- Early detection of malignant hyperpyrexia.

URINARY SYSTEM

The urine output is measured every hour if the patient is catheterised.
A good urine output is 60 ml/hr in an adult or 1 ml/kg/hr in a child.
Minimum urine output should be 30 ml/hr or 0.5ml/kg/hr.

TEMPERATURE

This should be checked routinely in very young patients. The temperature should be monitored in several other groups of patients, e.g. febrile patients, those having major or prolonged surgery.

- Rectal temperature. This only poorly reflects central body temperature.
- Oesophageal temperature. A probe is inserted into the naso-pharynx or into the upper oesophagus between the heart and the descending aorta. This reflects the temperature in the central circulation.

NEURO-MUSCULAR BLOCKADE

A peripheral nerve stimulator is used for monitoring neuromuscular function, the aim being to provide optimum recovery from paralysis after anaesthesia. A nerve stimulator applies a current to a peripheral nerve and measures the response in the muscle that the nerve supplies.
The features of the nerve stimulator are:

- The pulse waveform is a unipolar square wave of 0.2 - 0.3 ms duration.
- The output from the nerve stimulator should provide supra-maximal stimulation (more than 50 mA). That means a current is applied which guarantees every muscle fibre to be stimulated with each pulse.
- Stimulus patterns Single twitch (1.0 Hz)
  Tetanus (50 Hz)
  Train of four
  Double burst.

These are discussed a little later. A digital print-out is useful.

Site of stimulus
The ulnar nerve is commonly used. The muscle observed is the adductor pollicis which is innervated solely by the ulnar nerve.

Setting up

- The electrodes should be positioned as close as possible to the nerve.
- There are two types of electrodes - surface and needle. Surface electrodes are generally used. Needle electrodes are useful in the obese, those with thick skin and burns patients.
- Preparation of skin involves:
  - Removing excess hair using a razor.
  - Light abrasion of the skin.
  - Cleansing with alcohol.
- Placement of electrodes:
  - Place the distal electrode, which is connected to the negative (black) terminal, 1-2 cm proximal to the proximal skin crease, just lateral to the tendon of the flexor carpi ulnaris.
  - Place the proximal electrode as close to the distal electrode as possible along the line of the ulnar nerve (3 - 4 cm apart).

Baseline adjustment

Before administering the muscle relaxant, adjust the nerve stimulator to provide ***supramaximal stimulation***, which means the level of stimulation at which all the muscle fibres are depolarised. Increasing the current above this level does not increase the twitch response. Once this setting has been found for the patient, it should be kept for the entire procedure.

Monitoring the neuromuscular function

Twitch response The stimulus is delivered at a rate of 1 per second. After the relaxant is given the twitch response starts to fade. The twitch may completely disappear, which means more than 90% of receptors are blocked. However, it is better not to abolish the twitch completely under anaesthesia but to give just adequate amounts of relaxants to maintain a faint visible muscular contraction.
If the twitch has been abolished by an overdose of relaxant, wait until it reappears before giving a subsequent dose or reversing the patient.

Tetanic stimuli When the simple twitch returns to normal one can say that 20% of the receptors are free. But this also means that 80% of the receptors are blocked and this is dangerous, so we cannot rely on the twitch response to tell us if recovery is adequate. The tetanic stimulus was designed for this purpose: 50 or 100 stimuli per second (Hz) are delivered for 5 seconds. This rapid stimulation depletes the neuromuscular junction of acetylcholine when the number of free (unblocked) receptors is decreased. The tetanic response gets less and less, that is, it fades.
The higher the rate of stimulation the more obvious the 'fade'. Although tetanic stimulation is a means of assessing recovery it is painful and therefore of limited value in the unanaesthetised patient.

Train of four (TOF) stimuli The nerve is stimulated with 4 supramaximal currents 0.5 of a second apart. The response can be assessed in the form of the TOF count (TOFC) or the TOF ratio (TOFR). The TOFC indicates the number of palpable or visible twitches after the TOF stimulation. When a patient is paralysed with a muscle relaxant the twitches are lost in the order 4-3-2-1.
During recovery from the relaxant the 1st twitch reappears first and the others follow. When only 1 twitch is present after TOF stimulation it means there is 90% block. This is a good level of relaxation to maintain for surgery.
It is important not to completely abolish the thumb twitch (TOFC = 0). This would indicate too much relaxant.
Recovery from the block occurs when the TOFC is 4 and all the twitches are of equal height.

Double burst stimulation (DBS) This pattern of stimulation is available on the newer nerve stimulators. It consists of 2 short bursts of tetanus (50Hz for 60ms) separated by 0.75 of a second. It is easier to see fade on DBS than using TOF. DBS is more useful in the post-operative and recovery phase than TOF.

The Post-tetanic count (PTC) is sometimes used to assess deeper levels of block when all response to TOF stimulation is absent (no twitching at all). It consists of 50Hz for 5 seconds, (like the tetanic stimulus mentioned earlier), a 3 second pause and then a single twitch. The PTC is the number of visible twitches. One (1) means deep block; 2-8 moderately deep.

Clinical application

Induction	TOFC 1 PTC 0-1	Good relaxation sufficient for laryngoscopy. Paralysis of diaphragm if required in special conditions.
Maintenance	TOFC -1	(90% depression of single twitch) Stimulate with TOF every 5 – 15 minutes and seek to maintain a TOFC 1. If there is no response to TOF a PTC is performed.
Reversal	Aim to have the TOF at 3 - 4 by the time of reversal. If before reversal the TOFC is 0 the patient should not be reversed but simply ventilated and kept anaesthetised. If the TOFC is 1 - 2, neostigmine plus atropine (or glycopyrrolate) is used and the patient must be carefully observed for up to 30 minutes for complete recovery.	
Recovery	The simplest test for adequate recovery is the sustained head lift for 5 seconds. Using the nerve stimulator: • A TOFC of 4 and all 4 twitches are the same height. • A sustained response to tetanus. If you cannot detect a fade, use a DBS or 100Hz tetanus for 3-5 seconds. Any detectable fade will imply residual muscle paralysis.	

Possible errors in monitoring blockade

- Technical error
- The presence of nerve and muscle disease

OTHER PARAMETERS MONITORED IN SPECIALISED UNITS

Cardiac output, pulmonary artery pressure, arterial blood gases, electroencephalogram (EEG) and blood volume can all be measured using electronic equipment. Monitoring of these parameters is not routine but there are specific conditions where they are useful. This type of monitoring would normally be carried out in an Intensive Care Unit. For more detailed information see an advanced textbook of anaesthesia or intensive care medicine.

CHAPTER 51

HYPOTHERMIA

Outline:

Definition

Temperature control

Pathophysiology

Causes of intra-operative heat loss

Methods of prevention

Methods of warming blood and IV fluids

DEFINITION

Hypothermia describes subnormal body temperatures and an inability to generate heat or compensate for heat loss.

It may be categorised as **mild** with temperatures ranging between 32 - 35 degrees centigrade, **moderate** at 27 - 32 degrees centigrade or **severe** when the temperature is less than 27 degrees centigrade.

Body heat is lost through:

- Radiation (40%)
- Convection (30%)
- Evaporation (30%)
  - 20% is lost through the skin
  - 10% through respiration of which 8% is through evaporation and 2% through warming the inhaled air
- Conduction (minimal)

Induced hypothermia is sometimes used in neurosurgical, cardiac and vascular surgery to reduce damage to vital organs during periods of hypoxia.

Accidental hypothermia is associated with

- Prolonged surgery
- Elderly and hypothyroid patients
- Exposure
- Near drowning

Signs of hypothermia

Signs of ***mild hypothermia*** are peripheral vasoconstriction, shivering, decreased motor activity and CNS depression resulting in apathy or amnesia.

Moderate hypothermia leads to further depression of the CNS resulting in unconsciousness, cardiac arrhythmias and water diuresis with ensuing dehydration.

Severe hypothermia decreases blood pressure, heart rate and may cause ventricular fibrillation. At these temperatures the person will be in a coma and without reflexes.

TEMPERATURE CONTROL

The temperature control mechanism is situated in the hypothalamus where temperature information from skin, central and neural tissues is processed.

- Two types of thermostatic neurons are located in the hypothalamus. One type is affected by increased temperature, the other by decreased temperature of the blood.
- Descending pathways connect mainly to cardiovascular and respiratory centres in the brain stem and initiate heat loss or heat production responses in an attempt to bring temperature back within normal levels.

A rise in the temperature of the blood causes an increase in the respiratory rate, peripheral vasodilation (which increases radiation) and increased perspiration.
Decreased temperature causes conservation of heat by vasoconstriction, which reduces loss from radiation and by stimulating shivering. Heat production is also raised by the increase of the thyrotropic function of the anterior pituitary and a resulting increase in thyroid activity.
Temperature changes also cause obvious behavioural adjustments in conscious adults eg movement to or from a heat source and the adding or removing of clothing.

PATHOPHYSIOLOGY OF HYPOTHERMIA

Metabolic effects

There is a decrease in oxygen consumption and carbon dioxide production. Serum glucose levels are increased as a result of catecholamine release and a decrease in insulin production. Metabolic acidosis occurs resulting in an increase in potassium to which the hypothermic heart is very sensitive.

Central nervous system

Cerebral blood flow is reduced, oxygen consumption is reduced, the victim becomes sleepy at 33° C and cold narcosis follows at 30° C. The minimum alveolar concentration of volatile agents is decreased therefore a smaller concentration is needed and emergence from sleep may be delayed.

Blood

There is a decrease in plasma volume with an increase in viscosity. Aggregation of platelets decreases their function and leads to thrombocytopenia.

Respiratory system

Shivering increases oxygen consumption 3-5 times and may hinder the reading of the pulse oximeter.
There is reduced availability of oxygen to the tissues as a result of respiratory depression, a fall in cardiac output, peripheral vasoconstriction and increased blood viscosity. Increased solubility of carbon dioxide lowers the arterial pressure of carbon dioxide further decreasing ventilatory drive. The mechanism of hypoxic vasoconstriction is impaired resulting in an increase in ventilation/perfusion mismatch and hypoxaemia. The oxygen dissociation curve is shifted to the left decreasing oxygen delivery to the tissues.

Cardiovascular system

Hypothermia can induce ventricular ectopic beats leading to unresponsive ventricular fibrillation or to bradycardia leading to unresponsive asystole.
Vasoconstriction increases systemic vascular resistance increasing after load and myocardial oxygen demand, causing tissue hypoxia and acidosis although coronary blood flow is well maintained initially.

Urinary system

Renal blood flow and the glomerular filtration rate are decreased.
Decreased sodium reabsorption causes impairment of concentration of urine leading to cold diuresis and hypovolaemia.

Liver

The blood supply to the liver is diminished slowing down liver function and the metabolism of drugs.

CAUSES OF INTRA-OPERATIVE HEAT LOSS

For the anaesthetist, hypothermia as a result of prolonged surgery is the most important and most common cause.

Patient factors

There is loss of movement, a reduced capacity to shiver, exposure and an increased surface area with increased evaporation. The introduction of cold fluids, either intravenously, as peritoneal lavage or bladder washout, also contributes to heat loss. (For methods of warming infused fluids see below).
Infants have an increased surface area to body mass ratio and therefore loose heat more rapidly than adults. Patients with burns, severe injuries and those who are hypothyroid are predisposed to greater heat loss, as are the elderly who have decreased sympathetic activity.

Anaesthetic factors

Dry anaesthetic gases cause extra heat and moisture loss.
Many anaesthetic drugs, such as thiopentone and halothane, cause vasodilatation. Opiates decrease vasoconstriction and volatile agents interfere with thermoregulation in the hypothalamus. Subarachnoid blocks also cause vasodilatation, inactivate muscular movement and block sensory input to the thermoregulatory centre.

Surgical factors

Prolonged exposure of abdominal organs, abdominal lavage and bladder washouts all lead to a significant drop in body temperature.

METHODS OF PREVENTION OF HEAT LOSS DURING SURGERY

- The use of heat and moisture exchange filters (HME) helps to reduce heat loss from the respiratory tract.
- Warming the fluids given IV or used for lavage reduces further heat loss from exposed tissues and cold fluids.
- Some form of external body warming such as a hot air blanket, a covered hot water bottle or covering of head and exposed limbs by drapes or even woollen garments.
- Raising the ambient temperature of the operating room even if this may become uncomfortable for staff.

SIMPLE METHODS OF WARMING BLOOD AND IV FLUIDS

- Put the container of blood or plasma in lukewarm water before administering it.
- The infusion set tubing can be passed through a warm bath or under the warming blanket.
- Use a blood warmer. This consists of a very long plastic coil which is immersed in a water bath maintained at about 37°C. The aim is to deliver blood to the patient at near body temperature without causing blood damage. (See Chapter 47)

CHAPTER 52

SHOCK

Outline:

Definition

Physiology

Classification

Signs and Symptoms

Immediate treatment for shocked patients

Specific treatment for hypovolaemic shock

Anaesthetic management of the shocked patient

Other forms of shock

DEFINITION

Shock is a state of inadequate tissue perfusion which may be caused by a reduction in the volume of circulating fluid, cardiac failure, neurogenic injury, sepsis, or a number of other causes. The danger of shock is that tissue perfusion (or the blood flow to the tissues) is severely reduced and the tissues are therefore damaged or die from lack of oxygen and nourishment.

PHYSIOLOGY

Three things are needed to maintain a normal blood pressure.

A pump (in this case, the heart). The heart must contract efficiently.

A circulating blood volume. The blood is pumped by the heart into the arteries and thence to the tissue capillaries. The veins collect the blood and return it to the heart. If the blood volume falls then the patient can go into shock.

The peripheral resistance. This is the state of the small arteries (arterioles) and capillaries. If the peripheral resistance is high, it means that these small vessels are constricted. If the peripheral resistance is low, these vessels are dilated. A low peripheral resistance can result in low blood pressure. The blood tends to stagnate or pool in the dilated peripheral vessels. The volume of blood returning to the heart is reduced, so the blood pressure falls. Certain drugs, e.g. anaesthetic agents thiopentone and halothane, tend to lower blood pressure, mainly by causing peripheral vasodilatation. Sepsis may also induce shock by vasodilatation.

CLASSIFICATION OF SHOCK

Shock can be classified as follows:

Hypovolaemic shock (a reduction in the circulating blood volume) caused by:

- Loss of blood as in haemorrhage
- Loss of plasma, as from burns
- Dehydration, caused by:
  - Decreased input, e.g. fasting, especially in the very young and the very old.
  - Increased output from vomiting, diarrhoea, fistula, intestinal obstruction with accumulation of fluid in the gut.

Septic (bacteraemic) shock caused by

- Systemic infection (usually by gram-ve organisms) and characterised by hypotension and altered organ function

Neurogenic shock (reduction in peripheral resistance) caused by

- Spinal cord or brain stem injury

Anaphylactic shock caused by

- A hypersensitivity reaction to an antigen usually a drug

Cardiogenic shock (failure of heart action) caused by

- Heart disease, e.g. infarct, valve disease etc.
- Drugs or toxins depressing the heart

Very often more than one factor is involved, e.g. in septic shock there may be heart failure, hypovolaemia and a loss of peripheral resistance.

SIGNS AND SYMPTOMS

Cardiovascular

- Poor peripheral circulation seen as pallor and cold extremities.
- Poor filling of peripheral veins and nail beds is a better sign than a falling blood pressure (BP).
- Rapid and thready pulse.
- Fall in blood pressure. This is the **least** reliable sign. The blood pressure remains normal till all the compensatory mechanisms fail, then there is a dramatic fall after one third of the blood volume is lost.
- Collapsed peripheral veins. The veins in the neck are the best guide. In severe shock, the veins collapse. They reappear as resuscitation progresses. The central venous pressure (CVP) is low.

Respiratory

- Respirations are rapid and shallow.

Central nervous system

- The mental state of the patient in shock varies. Often the patient is mentally alert. If the blood pressure is low enough to cause cerebral hypoxia, restlessness and confusion can occur. Don’t give sedatives or analgesics until you are sure that the patient's restlessness is due to pain.

Gastro-intestinal

- Nausea and vomiting may occur.

Genito-urinary

- A fall in the urine output. A good urine output in the average adult patient is about 60ml/hr. A minimum acceptable output is 30ml/hr (i.e.0.5ml/kg/hour).
  In shock the urine output per hour is very low.

IMMEDIATE GENERAL TREATMENT (AIRWAY, BREATHING, CIRCULATION) FOR ALL SHOCKED PATIENTS

Maintain respiration

- Clear the airway. Suction may be required to remove any vomitus or secretions.
- If the airway is obstructed, extend the head, lift up the chin and insert an airway if the patient will tolerate it.
- Give oxygen by mask 6 L/min.
- If the patient is not breathing adequately, ventilate with oxygen, using a mask or an ETT.

Maintain circulation

- An intravenous infusion is commenced. More than one infusion may be required. Give fluids - crystalloid or colloid (1-2 litres in an adult, 10-20ml/kg in a child) initially stat. while the cause is established.
- Monitor the pulse, blood pressure, colour, urine output and CVP. CVP (if available) gives information on right ventricular filling pressures and also provides a means of giving inotropes many of which should be given centrally. CVP gives a rough guide of central blood volume.
- Repeat fluid bolus if no improvement or inadequate improvement while the diagnosis is being made. Continue crystalloid infusion titrating rate to clinical setting.

SPECIFIC TREATMENT FOR HYPOVOLAEMIC SHOCK

This is a common form of shock but much of the treatment is relevant to other forms of shock.

Insert two 16-gauge cannulae and begin two infusions. Use Hartmann's solution or 0.9% saline until blood is available. The first few bottles can be given quickly (depending on the degree of shock). Monitor clinical parameters as above.

In order to increase the rate of infusion:

- Use a large needle or cannula
- Elevate the bottle as high as possible
- Use a pump set
- Use a pressure bag

Fluids available

Crystalloid solutions. These are synthetic salt or dextrose solutions. Crystalloids distribute themselves readily throughout the extravascular space, therefore large volumes have to be given to expand the circulating blood volume.

- Saline. Normal saline contains more chloride ions than blood.
- Hartmann's (Ringer lactate) solution contains sodium, chloride, lactate, potassium and calcium. It is considered a more physiological solution than normal saline, as the concentration of electrolytes resembles interstitial fluid. Hartmann's solution is generally used in "shocked" patients until blood is available. If Hartmann's solution is not available, normal saline can be used.

Dextrose solutions must not be used in the treatment of hypovolaemic shock.

Colloid solutions (plasma expanders). Colloid solutions contain large molecules which are not filtered by the kidney and do not diffuse through the capillary membrane but are retained in the circulation and therefore expand the circulating volume.

Protein colloids. (Human plasma products). For a more detailed description see Chapter 49 (Blood transfusion).

- Albumin (4.5% and 20%) is very expensive. Both these solutions have a prolonged half-life 5-10 days.
  They are both heat treated and carry a very small risk of hepatitis.
  5% solutions are preferred for rehydration and intravascular volume expansion but are not ideal for rapid resuscitation and are more commonly used in hypo-proteinaemic states.

Synthetic colloids

- Haemaccel
- Gelofusine
  These are gelatin preparations. They do not interfere with blood cross matching, nor is there any disturbance of haemostasis with their use but allergic reactions may occur. They have a half-life of 4-5 hours and are suitable for use in rapid resuscitation.
- Hetastarch
- Pentastarch (isotonic)
  Synthetic starches give longer plasma expansion with a half life of up to 17 days.
- Dextran 70 (macrodex). This is a plasma substitute available as a 6% solution in either saline or dextrose. The particles may remain in solution for up to a week. It is associated with certain side effects and it is advisable not to use more than 1.5 L in any one patient.

Disadvantages of Dextran

- Dextran interferes with blood cross-matching. Any samples of blood for cross matching must be taken before the dextran is administered.
- Anaphylactic reactions may occur.
- It may interfere with platelet function and therefore haemostasis.
- The danger of fluid overload and infection is common to all plasma expanders.

Blood (See Chapter 49)

Blood may be available in the following forms:

- ***Fresh whole blood***. This is the ideal as it provides clotting factors but it rarely available.
- ***Stored bank blood.*** This is the commodity usually available.
- ***Packed red cells.*** Volume replacement must be made at the same time.
- ***Un-cross matched blood.*** Group O negative. This should not be used except in life- threatening conditions.
- ***Type specific.*** This is considered much safer than using Group O negative blood. It takes 15-20 minutes to group the patient and get type specific blood (i.e. blood of the same ABO and Rh group as the patient).

Rate of transfusion of fluids

In the patient with moderate to severe shock, the rate of transfusion must be rapid, e.g. 500ml in ten or fifteen minutes. As soon as a real improvement in the patient's pulse and blood pressure are noted then the transfusion must be slowed down. The danger of rapid transfusion is that of pulmonary oedema, especially in older patients. Generally speaking the dangers of inadequate volume replacement far outweigh the small risk of over hydration. Careful clinical monitoring is the key.

To determine the rate of transfusion, re-evaluate frequently:

- ***Pulse:*** Rapid pulse rate may indicate hypovolaemia.
- ***Blood pressure:*** If the blood pressure is less than 90 mmHg in a normotensive patient or if it is significantly lowered in a hypertensive patient, further transfusion is necessary.
- ***Urine output:*** A urine output of at least 0.5ml/kg/hour must be maintained. A smaller urine output may signify hypovolaemia. Urine output is an excellent guide to fluid resuscitation and the insertion of urinary catheter with an hourly measurement bag should always be considered.
- ***Central venous pressure:*** The normal central venous pressure is 8-12 cm water. See previous comments.

- ***General condition of the patient:*** Look for restlessness, thirst, dyspnoea, faintness, pallor and cold extremities which may suggest the need for further transfusion. Haematocrit is not useful in estimating the precise amount of blood lost until 12-24 hours have passed. However it is a useful guide in determining how much blood may be needed.

The use of drugs in hypovolaemic shock

The main aim in treating shock is to restore tissue perfusion and oxygenation. If after adequate volume replacement there is still inadequate tissue perfusion a second diagnosis may be considered (e.g. cardiac failure, sepsis, etc.). Ongoing bleeding or volume loss must always be excluded. The use of drugs to support blood pressure in hypovolaemia is only a temporary measure until volume replacement occurs or there is a diagnosis of a secondary cause of the shock (e.g. cardiac failure, sepsis, tension pneumothorax, or neurogenic shock). Agents that can be used to restore blood pressure temporarily while replacement is taking place include metaraminol, noradrenaline, adrenaline, or dopamine. Noradrenaline and adrenaline require central access (CVP line).

ANASTHETIC MANAGEMENT OF THE SHOCKED PATIENT

- Carefully assess the degree of hypovolaemia.
- Use the IV route for any drugs given to the shocked patient. Drugs given IM are poorly absorbed.
- Treat for shock as already outlined.
- Cross match blood and have it available for intra-operative use.
- The presence of head and neck injuries, chest and abdominal injuries, must be ruled out in traumatic shock.
- Treat shocked patients as you would a patient with a full stomach i.e. rapid sequence induction with cricoid pressure.
- Severely shocked patients may need ventilation after surgery.

Unless the patient requires life-saving surgery it is beneficial to delay surgery while resuscitation is undertaken and hypovolaemia, hypoxia and hypothermia treated.

Technique of anaesthesia

Use a general anaesthetic with a rapid sequence induction.

- Pre-oxygenate for four minutes, then perform induction (awake intubation may be performed in moribund patients). In patients who have been adequately resuscitated use IV ketamine (1 mg/kg) or etomidate (150 micrograms/kg), then suxamethonium 1 mg/kg and cricoid pressure.
- Intubate using an appropriate cuffed endotracheal tube.
- Maintaining anaesthesia will depend on the patient's condition. If the patient is moribund, ventilate with 100% oxygen and a small dose of ketamine. If the patient shows evidence of being too lightly anaesthetised then nitrous oxide, a low concentration of ether (2- 3%) or a ketamine infusion may be introduced. The patient who is well resuscitated is ventilated with 50% oxygen, together with opiate and a volatile agent in low concentrations. Again, if there is evidence that the patient is light, the anaesthetic can be further supplemented. Shocked patients need small doses of drugs but a higher concentration of oxygen. The ketamine infusion and relaxant technique is an excellent one for the shocked patient.
- Reversal using the routine reversal procedure. Patients who will require post-operative ventilation need not be reversed.

OTHER FORMS OF SHOCK

Specific treatment for septic or bacteraemic shock

- General airway measures and oxygen as described for immediate resuscitation.
- Restore the circulating blood volume as in hypovolaemic shock.
- Give antibiotics, **after** blood cultures have been taken.
- Give vasopressors/ inotropes for resistant hypotension.
- Surgical treatment if indicated to remove the source of infection.

Many patients with septic shock will require laparotomy for diagnosis and treatment of acute abdominal conditions. Many will also be hypovolaemic and pre-operative resuscitation should be attempted before anaesthesia is commenced.

Management of anaesthesia should be as discussed for hypovolaemic shock with particular care being taken to avoid hypotension and hypoxia.

Specific treatment for anaphylactic shock

This occurs after administration of a drug or after the intake of food, to which the patient is sensitive. Drug anaphylaxis is further discussed in Chapter 6 and Chapter 62.

- Stop administration of drug or food causing the reaction.
- Maintain circulating blood volume. You may need large volumes of plasma expanders.
- Give adrenaline 0.1-0.5 mg IV (0.1-0.5ml of 1:1000 solution or 1-5ml of 1:10,000 solution.) Adrenaline is the first line treatment in anaphylaxis. An infusion may need to be commenced.
- Give salbutamol (250 micrograms IV) as a second line treatment of bronchospasm associated with anaphylaxis. Give aminophylline as a third line treatment for bronchospasm, although it is not nearly as effective as adrenaline or salbutamol.
- Give an antihistamine- chlorpheniramine (Piriton) 10-20mg slowly IV.
- Give steroids hydrocortisone 100- 300mg IV. These are of use in anaphylaxis, however there is a delayed onset of action (several hours) and therefore it is not a first line treatment.

When anaphylactic shock occurs during anaesthesia any drugs which might have been responsible for the collapse should be discontinued and surgery abandoned or completed as quickly as possible. Oxygenation should be maintained by whatever method is already being used and the above measures instigated where appropriate.
An attempt should be made after stabilization to identify the cause of the reaction even if no specialized laboratory testing is available. The patient should be informed about his condition and the occurrence and treatment documented in the medical notes.

Specific treatment for cardiogenic shock

This is most commonly seen after myocardial infarction, when the force of cardiac contraction has been reduced. The aim of the treatment is to increase cardiac output.

- Maintain airway and breathing with supplementary oxygen.
- Exclude and treat arrhythmias.
- Begin treatment of myocardial infarction i.e. aspirin, thrombolysis (if available and indicated), heparin and nitrates.
- Give dopamine, in a dose of 2-10 micrograms/kg/min, or dobutamine 2.5-10 micrograms/kg/min to increase the force and rate of cardiac contraction and also to increase renal blood flow.
  Adrenaline may also be used in severe cases or where other inotropes are unavailable.

- In the case of complete heart block, isoprenaline 0.5-10 micrograms/min by infusion may be used before the correction of the underlying cause with a pacemaker (if available).

The use of these drugs has been discussed under the subject of management of anaesthetic complications in Chapter 46 and in Chapter 6 (Pharmacology)

CHAPTER 53

THE RECOVERY ROOM

Outline:

Principles of recovery room care

Equipment and staffing

Patient care in the recovery room

PRINCIPLES OF RECOVERY ROOM CARE

The recovery room should be close to the operating theatre.
Every patient who has had a general or a regional anaesthetic must be observed in the recovery room before being discharged to the ward.
The patient should be on a trolley which can easily be tilted head down.

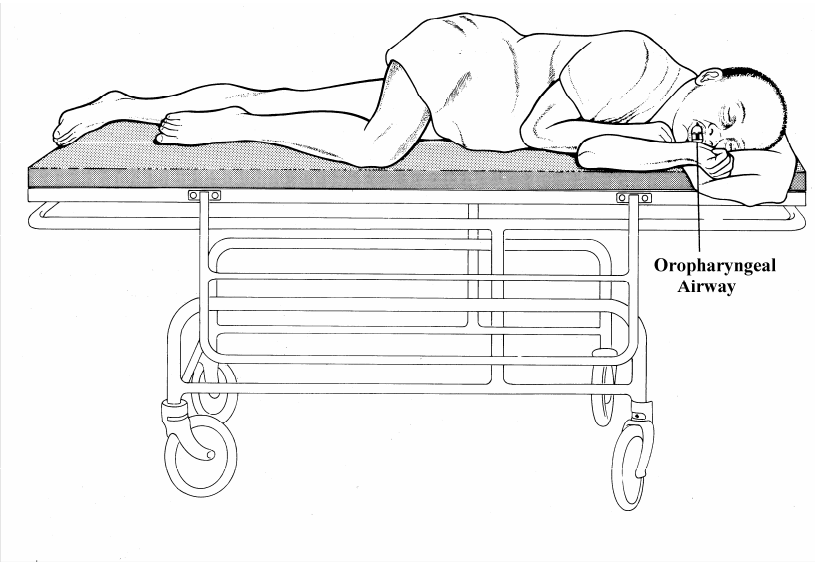


Fig 53.1 Post-operative patient in recovery position

EQUIPMENT AND STAFFING

The recovery room should have:

- Good lighting
- Adequate oxygen and suction equipment
- Adequate ventilation equipment:
  - Self inflating bag and mask
  - Face masks and airways
- Equipment for monitoring blood pressure
- A reliable clock with a clearly visible second hand
- Equipment for intravenous therapy
- Drugs for resuscitation

The recovery room should be supervised by a trained nurse, who must be able to communicate efficiently and easily with the anaesthetist. It is the anaesthetist who should personally hand over the patient to the recovery room nurse and discuss with the nurse any problems that have arisen in the operating room, for instance blood loss or a fall in blood pressure.

The anaesthetist must not leave the patient until respiration, colour, pulse and blood pressure are all satisfactory.

If any anaesthetic complications such as vomiting, bronchospasm, laryngeal spasm or convulsions have developed, the anaesthetist must not leave until the problem has been treated.

PATIENT CARE IN THE RECOVERY ROOM

- Careful records must be kept.
- All vital signs should be recorded every 10 minutes until discharge.
- Patients who have had spinal anaesthesia should remain in recovery for 1 hour since insertion of the spinal and ideally show signs of diminishing level of block before discharge.
- If IV or IM opiate analgesia has been given patients should remain in recovery for a further 20 minutes post injection.

Conditions for discharge from the recovery room

- The patient must be awake and responding fully to verbal stimuli.
- Breathing must be regular and deep.
- The colour of the mucous membrane of the lips should be pink. Oxygen saturation if available should be normal (>95%) breathing air.
- The pulse should be regular and of acceptable rate.
- The blood pressure must be stable.
- If the patient is catheterised the hourly urine output should be acceptable.
- Patient should have received adequate analgesia.

CHAPTER 54

OXYGEN THERAPY

Outline:

The aim of treatment

Indications for oxygen (O_2) therapy

Methods of administration

Choice of correct inspired O_2 concentration

Side effects of administration

Oxygen can be seen as a drug and like other drugs it has indications and undesirable side effects. Like all drugs oxygen must be prescribed in the right dose (concentration or flow rate), for a prescribed length of time.

THE AIM OF THE TREATMENT

To make sure there is enough oxygen in the inspired air to ensure adequate tissue oxygenation.

INDICATIONS FOR OXGEN THERAPY

Oxygen is indicated in states of hypoxia. Hypoxia means lack of oxygen in the tissues and is discussed in detail under Respiratory physiology in Chapter 3.
Oxygen is indicated in hypoxic, anaemic and stagnant hypoxia.

METHODS OF ADMINISTRATION

Mask
There are two types of mask:

Variable performance masks which use a low flow of oxygen,
e.g. 2-4 L/min. Air is entrained and a large volume of air and oxygen reaches the patient. Examples are the Hudson mask and Edinburgh mask. The Edinburgh masks used with 2 - 3 L/min of oxygen delivers 27 - 30% oxygen to the patient.
The inspired concentration depends on several factors one of which is the tidal volume.

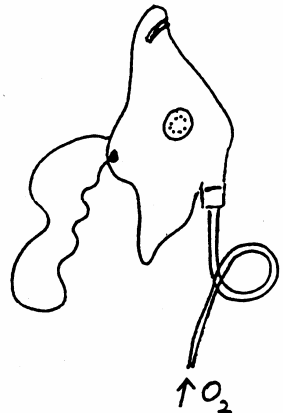


Fig 54.1 An example of a variable flow mask

A variable performance mask with an oxygen reservoir can increase inspired oxygen to almost 80%.

Fixed performance masks. These need about 6-8 litres of oxygen per minute to wash out the carbon dioxide (CO_2) and provide a fixed and constant inspired oxygen concentration. The oxygen passes through a jet which entrains air in such a way that the total flow rate exceeds the peak inspiratory flow rate (PIFR) of most patients. Venturi masks are used to deliver 24% oxygen, 28%, 35% or 40% oxygen depending on the mask. A different mask must be used for each range of oxygen concentration required.

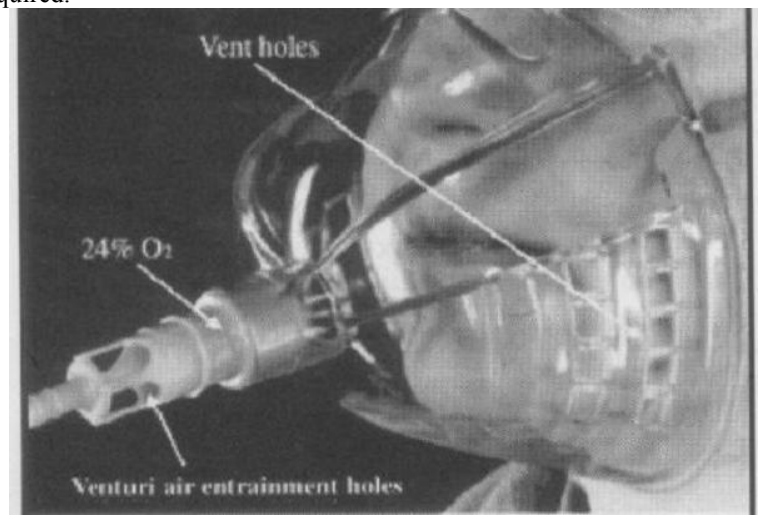


Fig 54.2 Example of a fixed performance mask with a 24% oxygen attachment

Disadvantages of masks:

- Eating, speaking, etc. are difficult
- They are dangerous if the patient vomits
- They can be uncomfortable and hot
- They increase dead space.

A non-rebreathing valve plus a mask enables 100% oxygen to be given.

Nasal cannulae or prongs

These are either PVC or plastic and consist of a tube with 2 soft tips that fit into the nostrils. They have the following advantages:

- There is no dead space.
- Vomiting or respiratory obstruction is easily seen.
- There is no interference with eating, speaking, etc.

Nasal catheters

These have similar advantages to nasal cannulae but also have dangers:

- If a nasal catheter is inserted too far then the oxygen may be delivered to the oesophagus instead of to the lungs and rupture of the oesophagus or the stomach can result. Avoid this by carefully measuring the distance between the side of the nose and the tragus of the ear and making sure the catheter is not inserted further than this.
- Prolonged use may cause nasal irritation. Four to six litres of oxygen per minute gives the patient a concentration of 30 to 40%.

Tent

This is commonly used with young children who will not tolerate a mask or a catheter. Tents are cool, moist and comfortable but leakage is common especially while the child is being nursed or monitored. The tent is an inefficient means of providing oxygen. It also limits patient care.

Incubator

This provides a controlled environment for the infant.

Endotracheal Tube

The patient may be intubated and placed on a ventilator with a selected concentration of oxygen, or intubated and allowed to breathe spontaneously while a selected concentration of oxygen is given.

Hyperbaric oxygen

This is used to treat symptoms of severe tissue hypoxia. This is oxygen administered at a pressure greater than atmospheric. The oxygen dissolved in the blood increases with an increase in oxygen pressure. Hyperbaric oxygen is used to treat conditions of severe hypoxaemia that cannot be treated with normal pressure oxygen. It is used in the treatment of carbon monoxide poisoning, air embolism, gas gangrene and decompression sickness.

Hyperbaric oxygen is administered in specialised units.

Humidifiers

Ideally when oxygen is administered for longer than 24 hours the inspired oxygen should be humidified by passing it through warmed water.
Humidification of gases with warm, moist air prevents drying of the respiratory mucosa and may help prevent hypothermia.
In practice humidifiers can be the source of serious hospital borne infections and must be used with meticulous care.
Disposable heat and moisture exchange (HME) bacterial filters are now routinely used during anaesthesia but they are expensive and therefore not readily affordable.

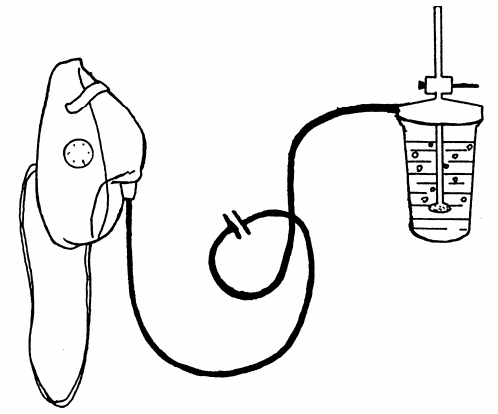


Fig 54.3 Method of humidifying oxygen

The oxygen concentrator

This provides oxygen from room air. It is described in detail in Chapter 13.

CHOICE OF CORRECT INSPIRED CONCENTRATION OF OXYGEN (FiO_2)

Patients can be classified into two broad groups with regard to oxygen therapy:

Those with normal respiratory control. The normal respiratory centre is stimulated by a high carbon dioxide level. As the level of carbon dioxide rises there is an increase in the rate and depth of breathing. It is also stimulated to a lesser extent by a fall in the oxygen level.

These patients may require oxygen because of injury or disease of a more acute nature, e.g. chest infection, fractured ribs or polio. In these cases oxygen can be given until the cause of the hypoxia is cured.

Those with abnormal respiratory control. The respiratory centre in COAD, on the other hand, may be stimulated not by the high carbon dioxide level but by the low oxygen level only. This is because a high carbon dioxide level already exists in these patients and it ceases to have an effect on the respiratory centre. The low oxygen level keeps the respiratory centre going. This is referred to as the patient's "hypoxic drive" (It is the hypoxia that "drives" the respiratory centre).

These patients require careful and controlled oxygen therapy. Therefore start with a low concentration of oxygen, about 24% and observe patients very carefully for signs of depression of the respiratory centre (shallow breathing and evidence of carbon dioxide accumulation). If 24% does not depress breathing then gradually increase the concentration noting respiratory rate and oxygen saturations. Those with hypoxic drive will reduce their respiratory rate as the oxygen concentration is increased.

Normally COAD patients with an acute exacerbation need high flow oxygen.

If a high oxygen concentration is required and the patient is showing signs of respiratory depression, then the patient's respiration may have to be controlled using IPPV.

Assessing the adequacy of oxygen therapy

- Clinical signs: colour, pulse, blood pressure, mental state
- The oximeter, to measure oxygen concentration in the blood
- Arterial blood gases

SIDE EFFECTS OF OXYGEN THERAPY

Respiratory depression

As mentioned above this can occur in certain types of patient, particularly those with chronic obstructive airway disease (COAD). These patients may be dependent on their "hypoxic drive" to keep them breathing.

When high concentrations of oxygen are given to some patients with COAD this hypoxic drive is abolished and the respiratory centre may slow down or even stop.

This condition is rare and often over-diagnosed and patients are denied adequate oxygen and allowed to remain hypoxic when they need high concentrations of oxygen and/or assisted or mechanical ventilation.

Arterial blood gas measurement if available will reveal an elevated CO_2 and decreased PaO_2.

Atelectasis

Prolonged administration of oxygen tends to result in atelectasis. This is due to the rapid absorption of oxygen into the blood stream.

Substernal pain

Tracheitis occurs if 70-100% oxygen is inhaled for more than a few hours.

Pulmonary damage and oedema

This will result if 100% oxygen is inhaled for long periods eg 1-3 days. Inspired oxygen concentration should always be kept at the lowest level that allows adequate tissue oxygenation.

Retrolental fibroplasia

This is the formation of a fibrovascular membrane behind the lens of the eye. It results in blindness. It occurs especially in premature babies who have been exposed to high concentrations of oxygen at birth. Premature babies should only receive oxygen if they are hypoxic and then preferably to maintain oxygen saturation around 92%.

CHAPTER 55

ACUTE PAIN RELIEF

Outline:

Definition

Introduction

Pain pathways

Pain assessment

Commonly used drugs

Pre-operative pain relief

Intra-operative pain relief

Post-operative pain relief

Monitoring

Other specialised techniques for parenteral opioid administration

Paediatrics

DEFINITION

Pain is defined as:

‘ *An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage.’*

In other words it indicates the potential for damage to the organism and is usually associated with behavioural responses designed to minimise the impact of any painful stimulus.

INTRODUCTION

Many myths have been associated with pain management and this has led to poor analgesia in the past. New understanding and techniques have allowed us to develop improved strategies for analgesia and to promote education in their use.

Adequate pain relief is not only humane but improves recovery. With good quality analgesia following upper abdominal surgery for example, patients can cough and clear pulmonary secretions effectively and mobilize more readily thus preventing deep venous thromboses, chest infections and other post operative complications. This may reduce the length of hospital stay.

In addition knowledge of pain and pain management may aid diagnosis when the colic of bowel obstruction changes to a diffuse peritonitis following perforation or after surgery as a change in the intensity of pain post-operatively signifies a surgical complication until proven otherwise.

For these reasons education in pain management should be part of the training of all health care staff.

PAIN PATHWAYS

Pain receptors are naked nerve endings in the skin. They are stimulated by many different modalities all of which share the common role of being potentially harmful. These pain receptor fibres amalgamate into nerve axons which run from their peripheral site in the skin to the spinal cord where they synapse with second order nerves in the substantia gelatinosa of the dorsal column of the cord. Two different types of peripheral pain nerves exist. One is myelinated (Aδ fibre) and transmits quick and focused pain information, the second is unmyelinated (C fibre) and conducts more slowly. This second nerve type is responsible for the vague and more generalized pain that develops after initial injury.

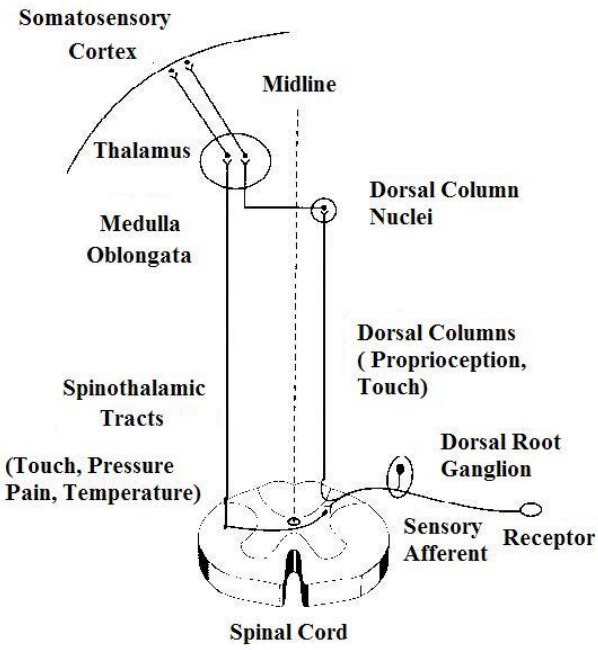


Fig 55.1 Pain pathways

After entering the spinal cord these nerves cross over and ascend to the thalamus in the controlateral, lateral spinothalamic tract, eventually radiating from here to the sensory cortex. Pain is consciously perceived initially at the level of the thalamus.

The transmission of pain is complex involving multiple mediators/neurotransmitters and is only beginning to be understood. Hence pain management is far from straightforward and may require complex multi-factorial strategies to achieve adequate relief. This is the concept behind modern pain management in big hospitals but there is still much that can be done to improve the care of the patient in pain even where facilities and drugs are limited.

(For more detailed information on pain transmission consult a specialist textbook).

PAIN ASSESSMENT

All patients are different and will respond differently to the same surgical process and subsequent analgesic regimen. We must recognise this and tailor our analgesia appropriately. In order to do this properly we require a reliable method of pain assessment.
Pain can be assessed either objectively or subjectively.

Objective assessment
This is based on the interpretation of a patient's pain and behaviour by a third party. It therefore tends to reflect that individual's prejudices and understanding of pain management rather than truly assessing patient discomfort. As a result it is of limited value but may be the only tool available in situations of poor communication – confused patient, different language or dialect etc.

Subjective assessment.
A much more valid tool is the subjective pain score. This is a direct assessment, from the patient about their pain and its significance. Many methods are available to perform subjective pain scoring and amongst the simplest is a Verbal Numerical Rating Scale:

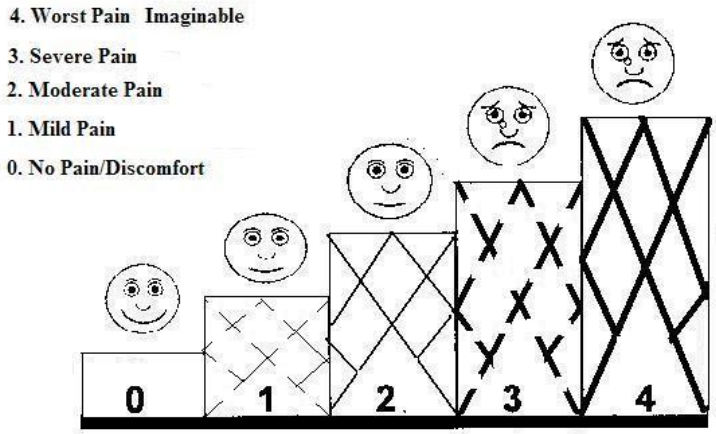


Fig 55.2 Example of a verbal Numerical Rating Scale

The patient is told that a score of zero represents no pain and a score of 4 represents the worst pain they can imagine. The patient then picks a number on this scale which best describes their pain. If they find this difficult, simply ask if the pain is mild, moderate or severe.

These pain scores are charted regularly and used to gauge the effectiveness of analgesic interventions. It is important to stress that these scores are charted in response to movement e.g. coughing or deep breathing as this gives a much more reliable indicator of the effectiveness of any pain relief. Also it is important to establish acceptable pain scores indicating effective analgesia. It is unrealistic to assume that patients will be pain free postoperatively without the use of invasive local anaesthetic based techniques such as epidural analgesia but using the above system a pain score of less than 2 on movement would indicate effective analgesia. This kind of pain relief should facilitate coughing/mobility and thus contribute to recovery.

Ideally the pain score should be assessed and charted at the same time as other vital signs are assessed.

Using this type of assessment, postoperative pain can be classified into two broad groups depending on the type of surgical insult:

- **Mild to moderate pain** (Pain score < or = 3). These patients will often achieve effective analgesia with simple oral analgesic formulations e.g. paracetamol, co-codamol or non-steroidal anti-inflammatory drugs (NSAIDS) such as Voltarol and Brufen (see below). Such patients would be undergoing relatively minor surgery (varicose vein surgery, laparoscopy, hernia repair etc.).
- **Moderate to severe pain** (pain score > 3) usually requires the use of parenteral (intravenous/subcutaneous/intramuscular) opiates.

This scale will be utilised to explain analgesic strategies throughout this chapter.

COMMONLY USED DRUGS IN PERI–OPERATIVE PAIN RELIEF
(see also Chapter 6 Pharmacology)

- Local anaesthetic agents either by regional block or infiltration e.g. lignocaine, bupivacaine
- Oral analgesics e.g. aspirin, paracetamol, non–steroidal anti–inflammatory drugs
- Weak opioids and combinations of oral analgesics e.g. codeine phosphate plus paracetamol (Panadeine, Co-codamol)
- Strong opioids e.g. morphine, pethidine, fentanyl.

For severe pain a combination of all of the above may need to be used.

Local Anaesthetics

See Chapters 17, 18 and 19

Paracetamol

A mild analgesic but surprisingly effective when administered regularly. Maximum dose is 4gm/24 hours in adults, 90mg/kg/day in children. Care is required to make sure that multiple paracetamol containing drugs are not co-prescribed e.g. paracetamol and co-codamol. Above the maximum dose it can cause serious liver dysfunction. It should be avoided in patients with underlying liver disease.

Non-Steroidal Anti-Inflammatory Drugs (NSAIDS)

NSAIDS are excellent analgesics with NNT of around 2 (see page 621). They are also a very versatile class of drugs administered in many different ways – topical, orally, rectally etc. If no contra-indication exists they should be added to any analgesia regime. However serious side effects such as renal failure and platelet dysfunction can limit their use. They are contra-indicated in:

- Dehydration and/or hypovolaemia
- Pre-existent renal impairment
- Congestive cardiac failure
- Peptic ulcer disease or past history of a GI bleed.
- Co-administration of nephrotoxins i.e. gentamicin.
- Surgery where platelet function must be normal i.e. neurosurgery.
- Known allergy or aspirin sensitive asthma
- Caution in the elderly
- Patients on Warfarin

Weak Opiates

Examples include tramadol, codeine and combination codeine and paracetamol products. The most efficacious drugs in this class are the high dose codeine/paracetamol combinations (codeine 30mg/paracetamol 500mg per tablet). Rare adverse effects include drowsiness, confusion and constipation – laxatives can be used if necessary.
Care is required in co-prescribing with other paracetamol containing preparations.

Potent Opiates

Partial agonist drugs such as pentazocine are best avoided for the management of acute pain as they are limited in their effectiveness by a ceiling dose preventing adequate titration of analgesia. They also act as an opiate antagonist when any pure agonist drug such as morphine is introduced resulting in further pain.

Morphine is the gold standard opiate agonist and should be used first in preference to any other opiate. Pethidine is a weaker drug, is limited in its use by a maximum 24 hour dose preventing effective titration and it can cause seizures.
Both morphine and pethidine have metabolites which can accumulate in renal impairment and cause complications.
Recognised side effects of opiates include:

- Nausea and vomiting
- Itch
- Respiratory depression
- Urinary retention
- Constipation

PREOPERATIVE PAIN RELIEF

A regional block is useful pre-operatively and may be used as the sole form of anaesthesia, if appropriate. Alternatively, it may be used to supplement general anaesthesia.

If the patient is in pain pre-operatively then 0.1 mg/kg morphine 3hourly IM or pethidine 1mg/kg 2 hourly IM is appropriate. An anti-emetic may be needed.

INTRAOPERATIVE PAIN RELIEF

- A regional technique or spinal technique has the advantage of avoiding general anaesthesia and opioids. It also provides good initial post-operative analgesia.
- Local infiltration either alone or in combination with general anaesthesia. It is important to be aware of safe doses of local anaesthetics.
- For painful procedures under general anaesthesia intravenous opioids should be given in increments during the procedure. All opioids are respiratory depressants and must be carefully titrated to provide good pain relief and allow the patient to breathe adequately at the end of the procedure. It is probably best to under-dose and give more in the recovery room.

Patients vary in their response to opioids. If a patient is very sensitive to opioids and remains apnoeic at the end of the procedure a small dose of naloxone (40 micrograms) can be given which will reverse the respiratory depressant effect while preserving analgesia.
Naloxone lasts about 20 minutes so occasionally further doses are required.

Non-steroidals can be very useful to supplement opioids, especially for gynaecology, orthopaedic and thoracic surgery i.e. diclofenac (Voltarol) 100mg. PR. Many oral analgesics are now available as suppositories which can be given PR before or during anaesthesia and in the post–operative period while the patient is unable to take oral drugs.

POST–OPERATIVE PAIN RELIEF

This is an important part of the anaesthetic and often needs to be carefully managed for several days.

Two important concepts underline the use of oral analgesics in modern medical practice.

- **Escalation of analgesia**

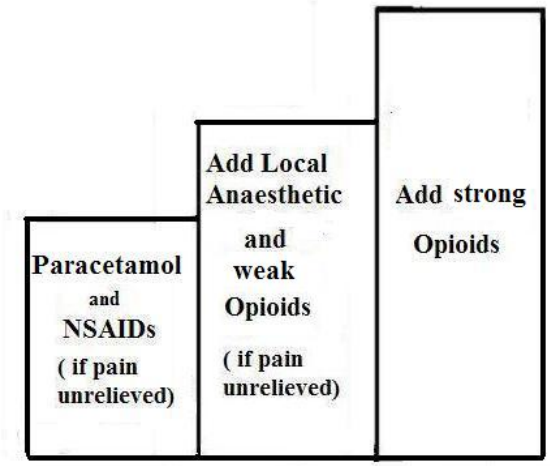


Fig 55.3 Analgesic Ladder

Level 1	**Level 2**	**Level 3**
Aspirin/paracetamol/ NSAIDs	Add LA and codeine/ paracetamol combinations	Add strong opioids e.g. morphine

This serves to remind us to escalate the potency of analgesic drug interventions as pain increases starting with the simplest and often safest interventions first.

Although initially designed for the management of chronic pain, the concept can equally apply to the post-operative patient.

- **The concept of Balanced Analgesia**
  This is an idea put forward to enhance the quality of pain relief at the same time as minimising adverse effects. It seeks also to recognise that pain is multi-factorial in nature involving multiple receptors/mediators and pathways (see above) and that using a combination of drugs with different modes of action will not only be more effective but the absolute dose of each drug can be limited to minimise the potential for adverse effects. This can be seen most effectively in the postoperative environment following major surgery. Patients receiving morphine or any other opiate alone can achieve the same quality of analgesia as a patient receiving a balanced analgesic technique (usually a combination of paracetamol, NSAID, opiate and local anaesthetic) but the morphine only group will use 30% more opiate to achieve adequate analgesia on average. This has been shown to result in a higher incidence of opiate related side effects in this group which can significantly impair their recovery e.g. sedation, nausea and vomiting etc.

Parenteral and oral techniques
Parenteral and oral techniques can be combined as indicated below to provide an optimum level of analgesia.

In the recovery room depending on which drugs are available intravenous increments of either:
- Morphine 1mg IV every 10 mins
- Fentanyl 20 micrograms every 10 mins
- Pethidine 10mgs IV every 10 mins

May be given with careful monitoring of vital signs. Some patients, especially after major surgery will require supplemental oxygen post-operatively.

On the ward Parenteral combined with oral.
- Oral analgesia - suitable for patients with mild to moderate postoperative pain (Pain Score 1-3).
- Parenteral analgesia - For painful procedures intramuscular opioids should be given regularly with regular paracetamol and non-steroidals O/PR if appropriate (see below). On day 2 or 3 this may be scaled down if appropriate according to the recommendations for follow-on analgesia.

Examples of opioid doses are:
- Morphine 0.15mg/kg IM or SC. 3 hourly~ 10mg 3 hourly for a 75kg adult.
- Pethidine 1mg/kg IM or SC. 2 hourly~ 75mg 2hourly for a 75kg adult.

Follow-on analgesia

When patients are eating and drinking normally and their pain intensity has decreased (usually around 48-72 hours after major surgery) it is time to consider stepping down the analgesic regime from potent parenteral opiate drugs plus adjuvants (paracetamol/NSAIDS) to an oral analgesic regime. Oral paracetamol or paracetamol/codeine combinations may be adequate. Paracetamol (including paracetamol/codeine preparations) can be given 1 gram O/PR 4 hourly or 20 mg/kg for paediatric patients.

This is a period where pain relief often fails as follow on analgesia is often not structured to reflect previous opiate use. Another common mistake is to replace regular administration of analgesia with as required pain relief, which leads to periodic analgesia and recurrent breakthrough pain.

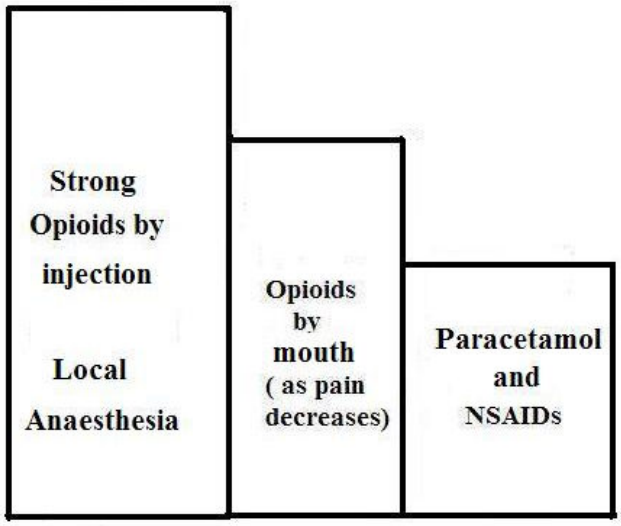


Fig 55.4 Step-down analgesia

Follow on analgesia should be a regular prescription for at least the next 24-48 hours subsequently stepped down to as required analgesia as pain settles and patients recover. The ultimate aim is to discharge patients on simple painkillers, no painkillers or the same analgesics that they were taking on admission.

Choice of drugs for oral analgesia

There is now evidence available to suggest which oral analgesic drugs are most effective in the management of postoperative pain. The number needed to treat (NNT) is an evidence based medicine concept to indicate the effectiveness of various therapeutic interventions. Analgesic drugs have been subsequently ranked with those drugs with smaller NNTs at the top of a **league table of analgesics**. This allows us to confidently recommend their use and move away from less effective drugs. In particular, all NSAIDs have NNTs around 2 and are thus very effective. Paracetamol has an NNT of around 4 and is surprisingly effective in the management of significant pain post-operatively. Also codeine 30mg with paracetamol 500mg preparations are very effective. All this information can be collated into an oral analgesic regime using the best drugs available in the hospital.

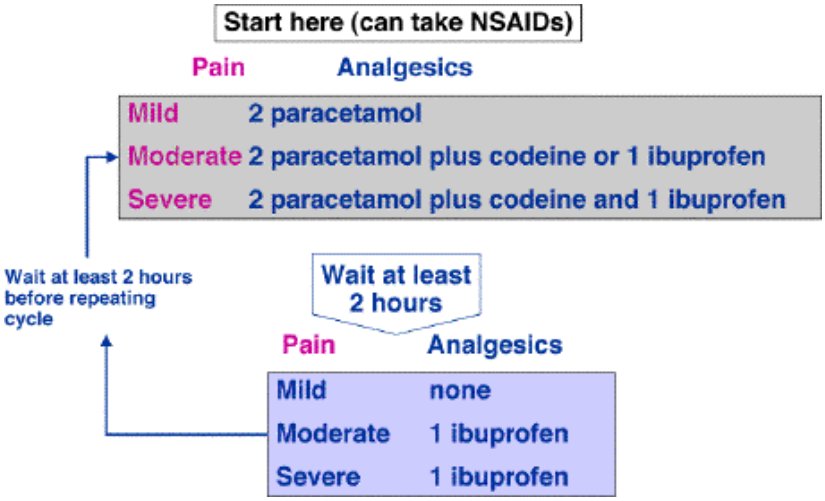


Fig 55.5 A regime for oral analgesia

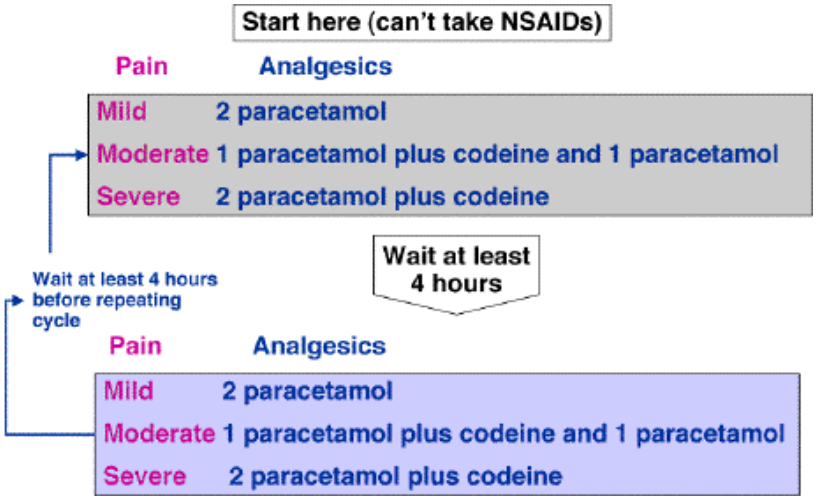


Fig 55.6 An oral regime for patients unable to take NSAID's

MONITORING FOR ADVERSE EFFECTS

Patients differ in their pain threshold and response to analgesia. Older patients and young children may be more sensitive to analgesic drugs, although this must be carefully assessed and monitored closely. The greatest area of concern in the management of acute pain is the potential to induce respiratory depression with the use of parenteral opiate drugs e.g. morphine/pethidine. It is always safer to titrate these drugs to effect particularly when establishing analgesia. This allows careful assessment of the effects of the drugs as there is always a lag time from administration to peak effect.

The drugs should be diluted to a suitable concentration with water for injection e.g. morphine 1mg/ml or pethidine 10mg/ml. Small volume injections can then be administered to optimise analgesia e.g. 1-2mg morphine boluses every 5 minutes in younger adult patients and smaller doses e.g. 0.5-1mg morphine boluses in patients over 70 years old. Vital signs should be monitored regularly during administration as well as pain score and respiratory rate. Patients should also be routinely questioned about other common opiate related adverse effects such as nausea and vomiting, itching etc.

- **Respiratory Depression:**
  The traditional teaching of counting respiratory rate to indicate potential respiratory depression is over simplified. A much more subtle way of identifying problems with respiratory depression is by using a **sedation scoring** system. Sedation scoring is charted on a scale of 0-3 as shown:

  0 Patient not sedated, wide awake
  1 Patient reports feeling tired but still fully alert
  2 Patient drowsy and readily falls 'asleep' when not stimulated
  3 Patient unresponsive to deep stimulus

  If sedation score is rising this should lead to caution concerning the continued administration of opiate drugs.
  A sedation score of 3 or a sedation score of 2 with a low respiratory rate requires urgent intervention to prevent a respiratory arrest and mandates the use of intravenous naloxone.

  In order to reverse the respiratory depression but not all of the analgesia, naloxone can be titrated to effect. Four hundred micrograms of naloxone are diluted with water for injection to a total of 4 ml giving a concentration of 100 micrograms of naloxone per ml. Naloxone is titrated in 1 ml doses until sedation score has returned to less than 2.

Caution: Naloxone is shorter acting than morphine/pethidine and its effects may wear off resulting in re-sedation. Any patient receiving naloxone should be monitored closely for the next 30-40 minutes and no further opiate should be given during this time.

In respiratory arrest or extreme depression a full dose of naloxone (400 micrograms) should be administered as a single bolus immediately.

As well as regular assessment of vital signs, pain and sedation scores, it is recommended that patients receiving regular opiates should receive supplemental oxygen where available. Patients should also be regularly assessed for complications or side effects of treatment and offered appropriate treatment as required.
With adequate analgesia, regular physiotherapy, early return to oral intake and mobilisation should be possible to aid recovery.

Aim

Using the fundamental tools of ***pain assessment*** and ***sedation scoring*** we can aim to provide effective analgesia (a pain score of less than 2 on movement) and allow regular and aggressive delivery of opiate drugs without excessive respiratory depression (sedation score of greater than or equal to 2). The idea is that every time a patient has their pain assessed, they also have their sedation score assessed. Pain should indicate the delivery of additional analgesia as long as there are no concerns with sedation. At all times a high sedation score overrides a high pain score to prevent worsening of the problem with the delivery of additional opiate drugs.

- **Post-Operative Nausea and Vomiting (PONV):**
  Anti-emetics may be required for the management of sickness related to opiate use or general anaesthesia. Good evidence exists to guide the use of these drugs but other general points are also worth highlighting:
  - Certain sub-groups of patients are at particular risk: including patients with a past history of PONV, patients with a history of travel sickness and women have a much higher incidence than men.
  - Certain types of surgery are associated with a higher incidence of PONV including emergency surgery, ENT and gynaecological procedures and bowel surgery.
  - Certain anaesthetic agents are also associated with a higher incidence of PONV e.g. nitrous oxide and ether.

  There are generally 5 different types of anti-sickness medicines (anti-emetics):

  Anticholinergics e.g., hyoscine
  Antihistamines e.g. cyclizine
  Dopamine blockers e.g. metoclopramide (Maxolon), prochlorperazine (Stemetil)
  Serotonin (5HT3) blockers e.g. ondansetron and granisetron
  Steroids e.g. dexamethasone

Some of these drugs have been proven to be more effective than others and we would currently recommend the use of **Stemetil** (prochlorperazine) or **cyclizine** as first line drug followed by the use of **ondansetron or granisetron**, if available, for resistant PONV. As these drugs act in different ways they can be co-prescribed. Dexamethasone is a useful long acting anti-emetic.

The use of a balanced analgesia protocol post-operatively (paracetamol/voltarol, if no contraindication, in addition to an opiate) will significantly reduce the requirement for potent parenteral opiate analgesia. This opiate sparing effect will therefore tend to reduce the incidence of opiate related nausea and vomiting.

Points to note

- Do not mobilise patients too quickly post operatively as this may trigger sickness.
- Try to recognise and target specific treatments for at risk groups. Once identified, prescribe an anti-emetic regime.
- Start oral intake slowly and keep patient well hydrated. Offer mouth care where appropriate.
- Administer anti-emetic medication early when the patient is nauseated instead of waiting until they vomit and encourage patients to communicate if they feel sick.
- Intravenous/intramuscular administration of anti-emetics is most effective after surgery. Consider prescribing the drugs regularly in resistant cases.

OTHER SPECIALISED TECHNIQUES FOR PARENTERAL OPIOID ADMINISTRATION

There are a number of other specialised techniques for the administration of opioids now used in many bigger and well-equipped hospitals. They will be mentioned briefly here but for more detailed information consult a specialised anaesthetic/ pain textbook

- **Subcutaneous morphine. S**uitable for patients with moderate to severe pain (Pain Score > 3).

  This technique is increasingly used instead of traditional intra-muscular (IM) analgesia. The characteristics of absorption and subsequent analgesia are no better with this technique but due to the frequency of injections it is more comfortable for patients to use an indwelling subcutaneous catheter if possible. The real benefit of this technique requires the rigid use of a protocol.

- **Intravenous morphine infusions.** These should only be utilised in critical care areas such as the general HDU/ICU because of the increased potential for respiratory depression.

- **Patient controlled analgesia (PCA).** This technique is the cornerstone of any acute pain service and has revolutionised modern pain management. Many different devices exist which can be programmed to varying degrees. All have a reservoir of drug (usually morphine 1mg/ml) connected to the patient's intravenous drip by means of a one way antisyphon valve and the administration of analgesia is controlled by a device for activating morphine delivery, usually a button of some description. Essentially the technique allows patients to take total control of their own analgesia and therefore allows for individual variability in pain perception and management.

The machines are programmed in a standard fashion according to local protocol with various safety features providing a more controlled administration of morphine than a continuous infusion.
PCA is a very effective method of maintaining analgesia but patients must be appropriately 'loaded' with morphine prior to its introduction. As most PCA machines are introduced post operatively this has usually been achieved by administering intravenous morphine in theatre and in the recovery area until the patient has a pain score of less than 3. As PCA allows patients to control their own pain by pressing a button to demand analgesia it also has a significant placebo effect which helps with pain control.
A full description of this technique is outwith the scope of this chapter and readers are referred to other texts in the list of further reading.

- **Epidural analgesia.** This is a highly specialised form of analgesia involving the insertion of a small catheter into a potential space around the dura of the spinal cord. The catheter allows the intermittent or continuous administration of pain control medication directly to the central nervous system. A mixture of local anaesthetic and opiate has been shown to be the most effective to control pain, particularly on movement by preventing the transmission of painful impulses through peripheral nerves to the brain. This allows us to maintain an area of 'numbness' over the surgical incision to allow the patient to mobilise completely free of pain post operatively.
  This technique can be very effective but because of its specialised nature can only be offered to specific patients who will be nursed post operatively in a critical care environment e.g. HDU/ICU.
  Again a full description of this technique is outwith the scope of this chapter and readers are referred to other texts in the list of further reading.

- **Peripheral Nerve Blocks.** These techniques have the capacity to produce profound analgesia and can be used as alternatives to general anaesthesia for surgery in the distribution of the block. They are particularly well suited for the management of peripheral limb injuries and trauma. However use of these techniques requires skill and expertise which may not be available locally.
  Local anaesthetic infiltration can also be useful for the management of pain related to small wounds/incisions e.g. hernia repair, caesarian section wound etc. but are of unproven benefit in larger laparotomy type wounds.
  When performing regional anaesthetic techniques such as peripheral nerve blocks it is important to be aware of the safe maximum doses of the various local anaesthetics – See Chapter 17.

- **Other techniques** – Entonox, Transcutaneous Electronic Nerve Stimulation (TENS) and acupuncture are also available and used occasionally in specific situations.

PAEDIATRICS

All the principles laid out before can be applied to paediatric practice with the following warnings:

- Children tend to be more sensitive to the effects of potent analgesic drugs such as morphine and smaller doses are required to achieve an effect. Their ability to metabolise and eliminate drugs may also be impaired resulting in longer duration of analgesia for a given dose.
- Children experience pain vividly and should be given analgesia readily.
- Pain assessment:
  A separate assessment tool may be required for very young children. Many examples exist based primarily on the assessment of facial expression.
- Sedation scoring may be difficult to use in very young children.
- Drug Doses:
  In children these are still based on weight:
  Paracetamol 20mg/kg 4-6 hourly orally or as suppository. Only 4 doses per day. Maximum (4gms per day).
  Voltarol 1mg/kg 8-12 hourly orally or as suppository
  Morphine 0.1mg/kg either intramuscularly or subcutaneously 2-3 hourly as required.
  Ondansetron 0.1mg/kg slowly intravenously (Maximum 4 mg per dose).
- **Oromorph** (immediate release oral morphine) has been used with success to manage severe pain in children particularly for fractures or to manage dressing changes in burns victims. Single doses of 100-400 micrograms per kg are recommended if available. Patients should be monitored closely for excessive sedation/respiratory depression.

Further Reading

1. **Acute Pain Management, A Practical Guide. PE McIntyre, LB Ready.**
   1996 W.B. Saunders Company Ltd. (ISBN 0-7020 1990-9).

2. **Oxford Pain Internet Site.** www.jr2.ox.ac.uk/bandolier/painres/

3. **Acute Pain Management: Scientific evidence (second edition 2005)**
   Australian National Health and Medical Research Council.
   www.anzca.edu.au

CHAPTER 56

CHEST PHYSIOTHERAPY

Outline:

Aims

Pre-operative

Post-operative

Other indications for chest physiotherapy

Methods

AIMS

- To help remove secretions.
- To ensure that both the normal and the accessory muscles of respiration are used properly. This provides adequate ventilation and also reduces the incidence of chest infections.

PRE–OPERATIVE

Physiotherapy is indicated in patients with chest disease who are presenting for surgery. Ideally any elective surgery should be postponed till these patients have had a pre-operative course of chest physiotherapy and the condition of their lungs has been optimised. It is also indicated in obese patients and those scheduled for prolonged or major surgery. In addition to chest physiotherapy, there may be a place for antibiotics and bronchodilators in some patients.

POST–OPERATIVE

This is indicated in patients who have had previous chest disease and in those who have had respiratory complications during or soon after the anaesthetic, for example those who have vomited and aspirated. It is also indicated in patients who have had no previous chest disease or complications but who have had major and very prolonged surgery which will entail a degree of immobility for some time post–operatively.
Adequate analgesia is essential in this situation.

OTHER INDICATIONS FOR CHEST PHYSIOTHERAPY

The unconscious or bedridden patient will benefit from regular chest and limb physiotherapy.

METHODS

Vibration

Here vibrations are applied to the chest wall with the hands. This is done during expiration at a frequency of 4-5 vibrations per second. The purpose of this is to loosen secretions.

Percussion

This is the rhythmic clapping of the chest wall using the air pocket between the hand and the chest to transmit the impulse. This tends to loosen secretions.
Both percussion and vibration must be done while the patient is in the position for postural drainage.

Coughing

Encourage patients to cough in order to expel the secretions that have been dislodged by the first two methods.

Suction

If coughing is impossible, for example if the patient is unconscious, then suction is the method to use. If a patient has an endotracheal tube or a tracheostomy tube in place, then suction must be done through the tube, with meticulous care using a sterile suction catheter and "no touch" technique.
Suction must be effective and at the same time safe for the patient. If suction is excessive there is a danger of airway collapse.
Nasopharyngeal suction is sometimes useful if the patient cannot cough up secretions. This can be done gently with a catheter or through a naso-pharyngeal airway.

Postural drainage (to be carried out **along with** vibration and percussion)

Postural drainage is the removal of secretions from the lung by using posture. The affected part of the lung is positioned at the highest level so that the secretions can drain out of the lung. A chest x-ray will show which part this is. However, many very sick patients cannot tolerate these postures and modified positions (for each lobe of the lung) may have to be used.

The lung has five lobes, three on the right and two on the left. Each lobe is divided into segments (See Chapter 2 Anatomy of respiratory tract). The positions that are employed for postural drainage of each of these segments are shown in manuals of physiotherapy. There are 8 positions for each lung. Postural drainage is done for 3 minutes, three times a day.

IPPV

This is used with bronchodilators as a form of chest physiotherapy.
"Bagging" is often used in patients in intensive care units on long–term ventilation.
It requires two people. One ventilates the patient by hand using a self-inflating bag and the other will perform vibration as the lung deflates.

Breathing exercises

These are designed to increase the vital capacity and help drainage.
This is done by:

- Encouraging diaphragmatic movement so that the lung bases are ventilated.
- Promoting good and controlled movements of all parts of the chest.
- Teaching the patient to cough.

CHAPTER 57

RESPIRATORY FAILURE

Outline:

Definition

Causes of respiratory failure

Diagnosis

Treatment of respiratory failure

DEFINITION

Respiration is a complex process. It is not simply taking atmospheric air into the lungs and delivering expired air into the atmosphere. Respiration is the process whereby the air in the atmosphere is taken into the lungs and the oxygen in the inspired air transferred to the blood. The oxygen in the blood is then carried to the tissues where it is used for metabolism. Simultaneously, the carbon dioxide that is manufactured in the tissue is carried from the tissues through the blood stream to the lungs and then from the lungs to the atmosphere. Respiration is therefore gas exchange. Oxygen is exchanged for carbon dioxide at the level of the lungs and again at a tissue level. (For more detail see Chapter 3 Respiratory physiology).

Many organs are involved in this process of gas exchange. The respiratory centre and the spinal cord, the peripheral nerves, the neuro muscular junction, the muscles, the chest wall, the pleural cavity, the lung tissue and the airways - all help to maintain this process of respiration. If one of the members of this team does not function properly, then the process of respiration is interfered with and respiratory failure begins to develop.

Consider a patient going into respiratory failure. Estimations of blood gases (e.g. the levels of oxygen and carbon dioxide in the arterial blood) will show the oxygen tension or pressure to be lower than normal and the carbon dioxide tension or pressure to be higher than normal. The normal arterial oxygen level is 100 mmHg in the average fit patient. In respiratory failure, this would be less than 60 mmHg. The normal arterial carbon dioxide level is 40 mmHg. This would be over 60 mmHg in respiratory failure.

CAUSES OF RESPIRATORY FAILURE

- Central causes in which the respiratory centre is affected: intracranial haemorrhage, drugs, tumours, infection
- Spinal cord: trauma, poliomyelitis, tetanus, Guillain Barre syndrome
- Nerve roots, e.g. high spinal or epidural block
- Peripheral nerves: acute polyneuritis
- Neuromuscular junction: muscle relaxants, myasthenia gravis, organic phosphate poisoning, snake bite
- Muscles: muscular dystrophies
- Chest wall: rib fractures, flail chest, scleroderma, post thoracotomy (a surgical incision may interfere with breathing)
- Pleural cavity: pneumothorax, haemothorax
- Lung parenchyma: collapse of the lung, consolidation, e.g. pneumonia
- Airway obstruction:
  - ***Upper***- tumour, scarring, external pressure from neck swellings, oedema, inhaled foreign body
  - ***Lower*** - asthma, COAD

DIAGNOSIS

There are two elements to the diagnosis of respiratory failure.

- The clinical diagnosis based on the history of the illness and the evidence of hypoxia and hypercarbia.
- The laboratory diagnosis. This can be made only if there are facilities to measure the levels of oxygen and carbon dioxide in the blood.

Clinical Diagnosis of Respiratory Failure

- ***History.*** There may be a history of lung problems, e.g. chronic obstructive airway disease, pneumonia, trauma, drugs, recent surgery, particularly major abdominal surgery, thoracic surgery.
- ***Clinical evidence of hypoxaemia.*** This is detected by low oxygen saturation on pulse oximetry or by clinical observation of cyanosis eg in the mucous membranes of the mouth. Cyanosis may be difficult to detect in anaemic patients.
- ***Clinical evidence of hypercapnia***. Sweating, confusion, even coma. An initial rise in blood pressure and pulse rate is followed by a fall.

Laboratory diagnosis of respiratory failure

Measurement of arterial blood gases if available.

In patients with chronic lung disease:

- Arterial oxygen level < 60 mmHg (8 kPa)
- Arterial carbon dioxide level. > 50 mmHg (7 kPa)
- pH changes showing respiratory acidosis and retention of bicarbonate
- There may be electrolyte changes with a rise in serum potassium

In patients with acute respiratory failure urgent treatment will be required at less severe levels of blood/gas derangement.

TREATMENT OF RESPIRATORY FAILURE

Facilities for mechanical ventilation may be required

- Check the airway. Infection, swellings, secretions and foreign bodies may cause obstruction.
- Maintain ventilation
  - Oxygen
  - Assisted ventilation
  - Controlled ventilation

  For all of these see the notes on Artificial Ventilation in Chapter 10.
- Intubation and later tracheostomy. See notes on Maintaining a clear airway in Chapter 9.
- Maintain circulation: monitor the patient, checking fluids and drugs.
- Treat the cause: asthma, pneumothorax, drug overdose, infection.

CHAPTER 58

CARDIOPULMONARY RESUSCITATION

Outline:

Diagnosis of cardiac arrest

Factors precipitating cardiac arrest

Immediate management (Note the ABCDEF to help memory.)

- **A**irway
- **B**reathing
  - Mouth to mouth respiration
  - Ventilation with a mask, valve and bag
  - Endotracheal intubation and ventilation
- **C**ardiac massage
  - External
  - Internal
- **D**rugs and IV access
- **E**CG and defibrillation
- **F**actors precipitating cardiac arrest (see above)

Later management

DIAGNOSIS OF CARDIAC ARREST

Cardiac arrest may or may not be witnessed.
The patient:

- Is unconscious
- Is not breathing
- Has no pulse in a major peripheral artery (the carotid or femoral artery). The carotid artery is palpated between the anterior border of the sternomastoid muscle and the thyroid cartilage.

These signs indicate that the diagnosis is cardiac arrest. Start resuscitation immediately.

Other signs: colour (cyanosis or ashen pallor)
pupils – usually dilated (unreliable sign)
heart sounds (absent)

But don't waste time looking for these signs.

During cardiac arrest circulation ceases, so there is an absence of oxygen in the tissues. Anaerobic tissue metabolism continues, resulting in a build up of acid waste products. These waste products are poisons. If they persist in the circulation the following effects may occur.

- Depression of the myocardium, the patient remaining hypotensive and unresponsive to all efforts at resuscitation.
- A lowered threshold for ventricular fibrillation, so arrhythmias are more likely.
- Pulmonary and renal vascular resistance may increase.
- Bohr effect. The haemoglobin loses some of its affinity for oxygen, so it carries less.

FACTORS PRECIPITATING CARDIAC ARREST

Hypoxia: inadequate inspired oxygen.

Hypercarbia: hypoventilation from any cause, respiratory obstruction, severe lung disease, etc.

Myocardial infarction

Pulmonary embolism

Haemorrhage leading to hypovolaemia.

Drugs

- Anaesthetic drugs may lead to cardiac arrest by causing:
  - Myocardial depression
  - Hypotension, due to peripheral venous pooling
  - Hypoxia or hypercarbia (which may be caused by central respiratory depressants or neuromuscular blocking agents)
  - Vagotonic action
  - Sympathetic stimulation.
- Non-anaesthetic drugs
  - Large doses of digitalis or procainamide
  - Anaphylaxis to IV contrast used in X-ray studies.

Vagal reflex mechanism: stimulation of organs e.g. dilatation of rectum, cervix, uterus, etc. in an awake or lightly anaesthetised patient.

Electric shock

Electrolyte imbalance, especially potassium (K^+) imbalance.

Tension pneumothorax, cardiac tamponade

Drowning

Hypothermia

Air embolism

IMMEDIATE MANAGEMENT – BASIC LIFE SUPPORT – CARDIOPULMONARY RESUSCITATION (CPR)

Airway

Clear the airway using the most appropriate measures:

- Suction, in the case of vomitus, secretions or blood.
- Manual clearance. Under direct vision insert your fingers into the pharynx and remove food, dentures or foreign bodies. Avoid "blind" finger sweeps.
- Posture. Extend the head and lift the chin and jaws.
- Insert a Guedel airway.

Breathing

Mouth-to-mouth respiration. Do this if there is no apparatus available to ventilate the patient, e.g. if the cardiac arrest occurs outside the hospital.

Clear the airway as explained above. Extend the head and lift the jaw up. Kneel beside the patient's head. Take a deep breath. Pinch the patient's nose with your free hand. Apply your lips to the patient's mouth to provide a seal. Blow into the patient's chest. Note the rise and fall of the chest. Do this 12-15 times per minute. Allow approximately 1-2 seconds for inspiration and 3 seconds for expiration. In infants, mouth to nose breathing works well. Give only gentle puffs to avoid lung rupture.

Disadvantages:

- It is not a very pleasant procedure to carry out.
- The patient is ventilated with expired gases which contain only 16% oxygen.
- The rescuer tends to get "dizzy" after a few minutes because of low carbon dioxide. Mouth-to-mouth resuscitation cannot be carried out for long.

Ventilation using a bag, valve and mask. A non-rebreathing valve, self–inflating bag and mask may be used. This enables the patient to be ventilated more effectively.

- Clear the airway completely, as described previously.
- Make sure the chest moves every time the bag is squeezed.
- An air-tight seal between the face and the mask is necessary to inflate the lungs. A self- inflating bag fills during expiration regardless of the seal and a one-way valve allows ventilation of the patient with 100% oxygen. A flow rate of 10L/min is necessary to remove carbon dioxide.

(Endotracheal intubation is not an emergency measure. Ventilation can be quite effective using a mask and bag provided the rules are followed. Intubation can be postponed until someone experienced in intubation is available).

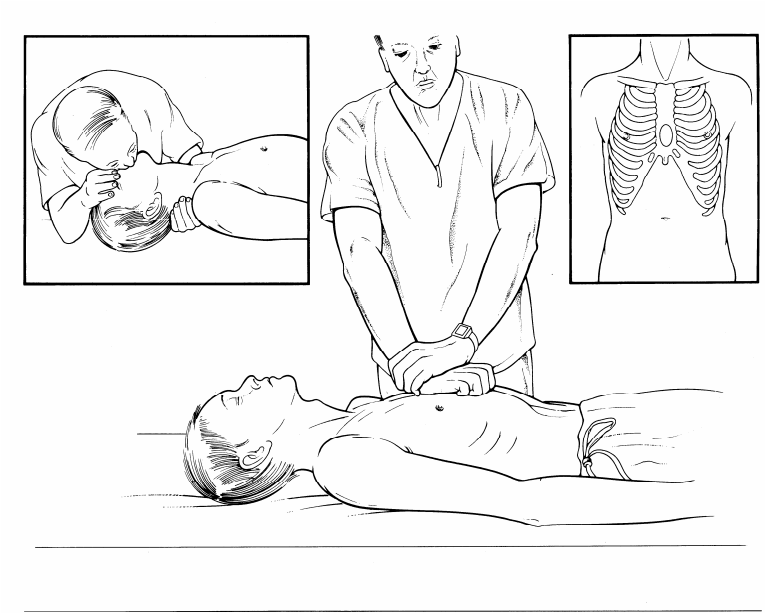


Fig 58.1 Mouth to mouth respiration and external cardiac massage
(oval drawn on the sternum shows where pressure should be applied)

Cardiac massage

External cardiac massage (ECM) The heart is compressed between the sternum and the vertebral column.

Technique
The patient must lie on a rigid surface. The operator should stand or kneel beside the patient so that his shoulders are directly in line with his hands when placed on the patient's sternum.

Put the heel of one hand on the lower half of the sternum. Put the other hand directly on top of the first. Press down on the sternum rapidly and forcefully for a depth of 2.5 - 5 cm. Apply pressure backwards towards the vertebral column. Keep the arms extended (not bent at the elbows) and the weight of the shoulders will provide the necessary force. Press down approximately 100 times per minute.

ECM must be synchronised with ventilation but they must not be carried out at the same moment. A good system is to give the patient two inflations of the lungs for every 30 cardiac compressions in an adult, two inflations for every 15 compressions in a child.

Always check the effectiveness of ECM by:

- Palpating the carotid or femoral artery
- Noting improvement in colour
- Noting change in pupils.

In children under ten, use one hand for ECM. In infants, use 2 fingers and compress the lower half of the sternum or encircle the chest with the hands, pressing on the sternum with the thumbs.

Advantages

- ECM can be performed by anyone
- No equipment is required
- No risk of infection
- Ventilation is not interfered with.

Dangers

- Fractured ribs
- Pneumothorax
- Injury to liver

Internal Cardiac Massage (ICM)

This approach is rarely necessary but may be indicated:

- When cardiac arrest occurs intra operatively, the chest or abdomen is already opened and the surgeon is present.
- In trauma arrests, e.g. penetrating chest wounds, severe chest crush injuries, haemothorax.
- If ECM is ineffective because of underlying pathology, e.g. pericardial tamponade, severe scoliosis.

Transthoracic approach Abduct the left arm. Make the incision in the 4th or 5th left intercostal space (i.e. between the ribs). Stop the incision 2.5cm from the sternal edge in order to spare the internal mammary artery. Avoid injury to the lung by making a small hole in the pleura first. Use a rib spreader to separate the ribs. Insert the whole hand and lift up the heart against the sternum. Compress the heart against the sternum at the rate of once per second.

Transabdominal approach This may be tried if the cardiac arrest occurs while the abdomen is opened. Place one hand on the sternum and the other under the diaphragm and compress the heart between the two.

Drugs and IV access

It is vital to establish an intravenous line as soon as possible.

Drugs

There are very few drugs that are of clinical use in the setting of a cardiac arrest. The main drug of use is

- Adrenaline (see management of cardiac arrest algorithm). Dose 1mg (i.e. 1ml of 1 in 1000 solution or 10ml of 1 in 10,000 solution) as required at 3 minute intervals. Adrenaline may be given through the endotracheal tube at twice the IV dose (i.e.2mg).
  If there is no IV access adrenaline is sometimes given by intra-cardiac injection.

Other drugs that may be of use are:

- Atropine (occasionally used in the setting of asystole). Dose 3mg.
- Lignocaine (this may be considered in the treatment of ventricular tachycardia (VT) in which there is no hypotension. Dose 50-100mgs then infusion or repeat bolus every 20 mins. VT associated with hypotension is treated with defibrillation.
- Calcium chloride (if there is a possibility of severe hyperkalaemia, known hypocalcemia, or overdose of calcium channel blockers). Dose 2-5ml 10% CaCl injected slowly, observe and monitor at the same time.
- Amiodarone 300mg may be considered for treatment of arrhythmias.
- Isoprenaline (only of use in the setting of complete heart block; it is used as a temporary measure until some form of pacing, either external or internal, is established.
- Sodium bicarbonate is rarely used except for severe acidaemia where pH < 7.1 or base excess > –10.

ECG and defibrillation

The ECG may show:

- Asystole – indicated by a straight line with no electrical activity. Defibrillation is of no value in the treatment of asystole.
- Ventricular fibrillation (VF). This is the commonest primary arrhythmia indicated by a wavy, irregular, chaotic tracing.
- Elecromechanical dissociation (EMD). There is no cardiac output, i.e. there is no palpable pulse and no oxygen delivery to the tissues, despite recognisable QRS complexes.
- Pulseless ventricular tachycardia (VT). There are regular, rapid, broad ventricular complexes but no detectable pulse.

Management

If the pulse has stopped, begin CPR immediately.

Defibrillation

The defibrillator passes a current (high energy impulse of short duration) through the heart. The heart muscle is stimulated to contract, then it passes into the refractory period after which it may resume sinus rhythm. The current is passed across two paddles located either on the chest wall (external defibrillation) or on the surface of the heart (internal defibrillation).

DC defibrillation (as opposed to AC defibrillation) is not only more effective, it is also less dangerous. The impulse is short, 3 milliseconds as opposed to 200 milliseconds for AC).

The DC External defibrillation energy level for adults is 400 joules for a monophasic defibrillator, 150 joules for a biphasic defibrillator.
Child: 4 joules/kg.
Internal defibrillation. 20-100 watt-secs or joules.

The defibrillator can be used for the correction of rhythm disturbances other than ventricular fibrillation, e.g. VT or atrial arrhythmias. In these cases the impulse should be synchronised with the ECG so that the vulnerable period of the cycle (start of the T complex) is avoided. In the case of an acute rhythm disturbance such as VF, this is not important. Steps in using the defibrillator for VF are:

- Apply a conducting paste on the paddles.
- Position the paddles, one at the right border of the sternum, the other at the apex of the heart, that is the 5th ICS mid axillary line.
- Make sure there is no contact between the defibrillator and the bed and that all personnel are standing clear.
- Select the energy level, e.g. 200 joules.
- Press the charge button.
- When the charge is built up, press the discharge button.
- Continue external cardiac massage and ventilation as soon as possible.

During CPR always consider reversible causes of cardiac arrest (4 H and 4 T) and correct any found. See algorithm on page 646.

LATER MANAGEMENT (In brief)

There is no specific late management of cardiac arrest (apart from that directed at the underlying cause) except supportive therapy for cardiovascular, respiratory and central nervous system failure often requiring treatment in an intensive care unit.

The presence of cerebral oedema is a particularly poor prognostic sign and is usually fatal. Treatment for cerebral oedema secondary to cardiac arrest would include avoidance of hypoxia and hypercapnia (endotracheal intubation and ventilation if necessary), mannitol / diuretics to reduce intracranial pressure and sedation to facilitate ventilation and reduce seizures.

ADULT BASIC LIFE SUPPORT

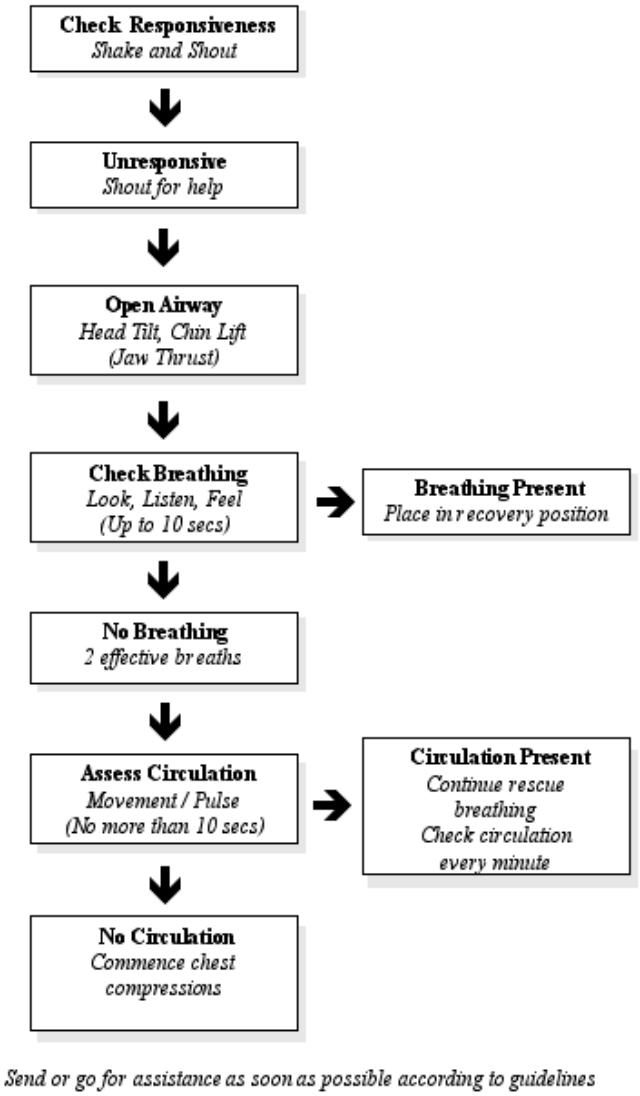


Fig 58.2 Adult basic life support algorithm

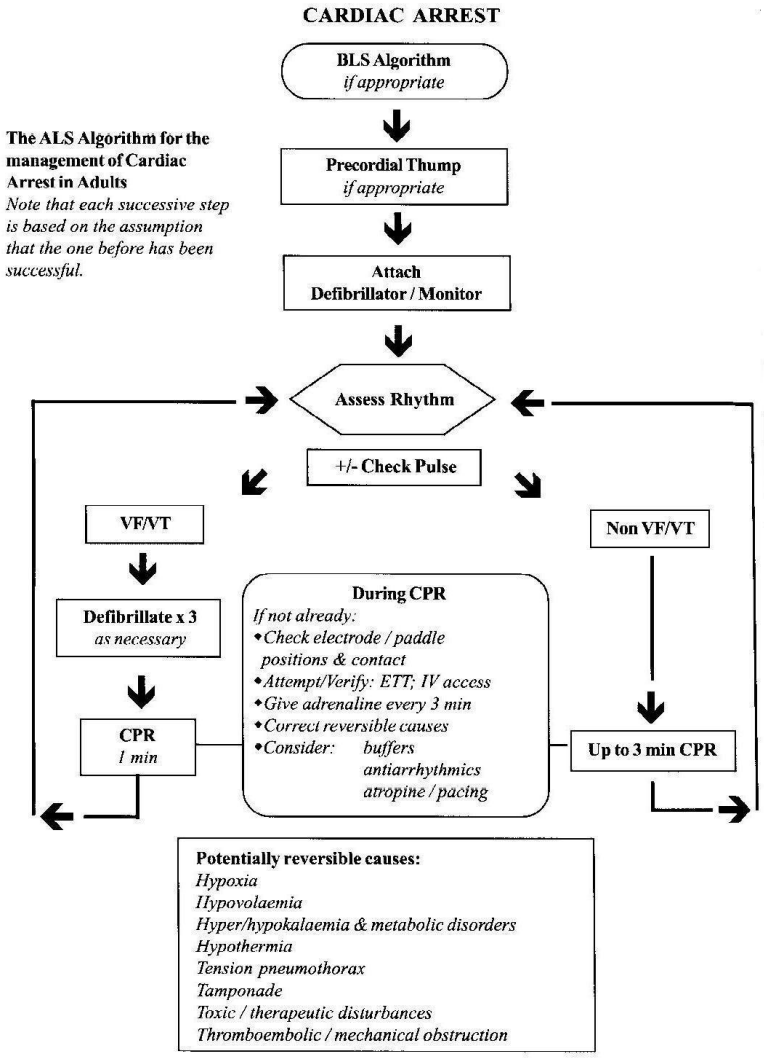


Fig 58.3 Adult advanced life support algorithm

CHAPTER 59

ACID-BASE BALANCE (SIMPLIFIED)

Outline:

Introduction

Definitions

Respiratory disturbances

Metabolic disturbances

Compensatory changes

INTRODUCTION

Complete oxidation of carbohydrates and fats yields carbon dioxide and water. 13,000 mmols of carbon dioxide are produced in this way every day and excreted through the lungs. In the normal person, this has no effect on the acidity of the blood.

Carbon dioxide is a volatile acid. The body also produces certain other acid substances which are referred to as non-volatile acids. These acids result from the metabolism of sulphur-containing amino acids, fats, etc. These substances are not completely oxidised to carbon dioxide and water. Therefore they cannot be excreted through the lungs and require renal excretion. If they are not excreted adequately then they can cause acid-base disturbances.

The lungs, kidneys and the "buffers" in the blood provide defence against acid-base disturbances.
These buffer bases are like sponges that soak up the hydrogen ions (H^+) and minimise the change in H^+ concentration and therefore the changes in the acid-base state of the blood. When the buffer base reacts with the H^+, the H^+ ion is removed from the blood. There is also a reduction in the buffer base concentration. For every H^+ ion neutralised, there is a fall in the buffer base concentration by an equivalent amount.

DEFINITIONS

An acid is a substance which when in solution donates H^+. Acids are therefore H^+ donors.

An alkali is a substance that donates anions which react with the H^+ and remove them from solution.

pH The acidity or the alkalinity of the blood is measured in terms of pH. The standard definition of pH is as follows: The negative logarithm to the base 10 of the H^+ concentration. The pH is inversely proportional to the H^+ concentration. Therefore when the pH is low the H^+ ion concentration is high (acidaemia). When the pH is high the H^+ ion concentration is low (alkalaemia).

Acidosis is a condition where the pH is lower than normal.

Alkalosis is a condition where the pH is higher than normal.

The causes of acid base disturbance are classified into respiratory and metabolic. The respiratory changes are related to the arterial carbon dioxide tension. The normal arterial PCO_2 ($PaCO_2$) is 36-44mmHg (average of 40mmHg).
Carbon dioxide dissolves to form carbonic acid (H_2CO_3). Carbonic acid dissociates (breaks down) into H^+ and HCO_3^- (bicarbonate ions), thus

$$H_2CO_3 \Leftrightarrow H^+ + HCO_3^-.$$

RESPIRATORY DISTURBANCES

Respiratory acidosis

This exists if the carbon dioxide tension is greater than 44mmHg.
Respiratory acidosis may result from lesions at the following sites. (See notes on Respiratory failure in Chapter 57)

- ***Central causes:*** in which the respiratory centre is affected: Stroke, intracranial haemorrhage, drugs, tumours, infection
- ***Spinal cord:*** Trauma, poliomyelitis, tetanus, Guillain-Barre syndrome.
- ***Nerve roots:*** high spinal or epidural block
- ***Peripheral nerves:*** acute polyneuritis
- ***Neuromuscular junction:*** muscle relaxants, myasthenia gravis, organo-phosphate poisoning, snake bite
- ***Muscles:*** muscular dystrophies
- ***Chest wall:*** flail chest, scleroderma, post thoracotomy. (A surgical incision may interfere with breathing)
- ***Pleural cavity:*** pneumothorax, haemothorax
- ***Lung parenchyma:*** collapse of the lung, consolidation e.g. pneumonia
- ***Airway, upper:*** Tumour, scarring, external pressure from neck swellings, oedema
- ***Airway lower:*** obstruction such as asthma or COAD

In addition to the causes shown the following may contribute to **respiratory acidosis during anaesthesia.**

- Non functioning soda lime
- Inadequate fresh gas flow resulting in rebreathing of expired air.

Treatment: Reverse underlying pathology and give mechanical ventilatory support if required.

Respiratory Alkalosis

There is a fall in the H^+ concentration in the blood. This is associated with a reduction in the arterial PCO_2.

Causes of respiratory alkalosis

- Hyperventilation, e.g. voluntary (Hysteria).
- Excessive intermittent positive pressure ventilation. This occurs frequently under anaesthesia.

Respiratory disturbances are not difficult to diagnose:

- The pH will be low in acidosis and high in alkalosis.
- Carbon dioxide changes: the $PaCO_2$ (arterial pressure) will be high (above 44mmHg) in respiratory acidosis and low (below 36mmHg) in alkalosis.

METABOLIC DISTURBANCES

Metabolic disturbances are caused by changes in the concentration of non-volatile acids, i.e. acids other than CO_2. If these acids accumulate then there is a rise in the H^+ in the blood. The buffers quickly soak up this H^+ by reacting with it, thus:

$$H^+ + HCO_3^- \text{ (buffer)} \Leftrightarrow H_2CO_3$$

As the H^+ ions are removed from solution (by the buffers) to form H_2CO_3, the buffer base is used up too. Therefore the reduction in the concentration of buffer base (mainly HCO_3^-) will give an indication of how much metabolic (or non–volatile) H^+ has been neutralised.

The HCO_3^- is the main buffer. Others are phosphate (HPO_4^-) protein and haemoglobin. In estimating the metabolic changes our measurements are confined to changes in the HCO_3^- buffer.

To summarise:

- Metabolic disturbances are associated with an increase or decrease of non-volatile or metabolic acids.
- When these H^+ ions are added to the blood, the buffer base reacts with it, soaks up the H^+ and minimises the change in H^+ concentration thus:
$$H^+ + HCO_3^- \Leftrightarrow H_2CO_3$$
- For every H^+ neutralised, one HCO_3^- is used up.
- Therefore, by measuring the fall in the concentration of HCO_3^- (the main buffer), we can get an idea of the increase of acid in the blood.

However:

The volatile acid in the blood, i.e. the carbon dioxide, can also have an effect on the HCO_3^- , thus carbon dioxide dissolves in water to form carbonic acid.

$$CO_2 + H_2O \Leftrightarrow H_2CO_3$$

Carbonic acid dissociates to form $H^+ + HCO_3^-$

$$H_2CO_3 \Leftrightarrow HCO_3^- + H^+$$

It can be seen that the HCO_3^- level in the blood can be affected by both metabolic changes and respiratory changes.

Estimation of plasma bicarbonate

Respiratory disturbances are easy to diagnose using the $PaCO_2$. If it is possible in some way to maintain the $PaCO_2$ at a normal level, any changes in the HCO_3^- must then reflect a metabolic disturbance.
If the sample of blood is exposed to a $PaCO_2$ of 40mmHg, the blood will acquire that $PaCO_2$. If the HCO_3^- level is measured, in the presence of a normal $PaCO_2$ (in other words eliminating any changes in HCO_3^- due to a respiratory disturbance), then the changes in HCO_3^- will reflect a metabolic disturbance.

There are several estimations made of plasma HCO_3^- all made or calculated by using a blood gas analyser.

- The standard HCO_3^- is the bicarbonate level in the plasma, at a $PaCO_2$ of 40 mmHg and a temperature of 38°C. Normal is 24.5mmol/L. The standard bicarbonate therefore eliminates any changes in HCO_3^- caused by a respiratory disturbance and an abnormal standard bicarbonate indicates a metabolic disturbance
- The base excess or deficit. This is calculated from pH, $PaCO_2$ and Hb and is a more accurate measurement of the metabolic component of the acid-base disorder. The base excess is normally zero.
  Base excess is less commonly used now but can be a clinically useful measurement.
- The total CO_2 measures the HCO_3^- and the dissolved CO_2 in plasma. (Only about 10% of the total CO_2 is dissolved CO_2 so this figure gives a reasonably accurate figure for HCO_3^-).
- The serum HCO_3^- measures the HCO_3^- level in the plasma. This is influenced by both the metabolic and respiratory state of the patient. This is also referred to as the CO_2 combining power.

The serum bicarbonate, the standard bicarbonate and the base excess or deficit are all calculated values, not direct measurements so of less value than $PaCO_2$ and H^+.

Metabolic acidosis (increased H^+, low pH, low standard HCO_3^-)

This is caused by an accumulation of fixed (non-volatile) acids **or** a loss of alkali.

Causes of metabolic acidosis

- Diabetic ketoacidosis
- Renal acidosis
- Prolonged hypotension (shock)
- Cardiac arrest
- Prolonged exercise
- Starvation
- Prolonged intestinal obstruction
- Gangrene of an extremity
- Occlusion of a large vessel
- Salicylate poisoning
- Infantile diarrhoea.

Treatment of metabolic acidosis: The treatment of metabolic acidosis is aimed at the cause, i.e. correcting hypovolaemia etc. Sodium bicarbonate is used rarely and only occasionally for low bicarbonate metabolic acidosis, such as in GIT loss or renal loss. With adequate treatment of the underlying cause the acidosis will resolve. The use of sodium bicarbonate is controversial and may worsen the situation as it may cause intracellular acidosis. Its use is contraindicated if inadequate ventilation exists leading to a large rise in CO_2 and also in lactic acidosis**.**

If sodium bicarbonate has to be used in an emergency situation calculate from the following formula (infusion in mmols)

0.33 x Base Excess x body weight (kg)

The 8.4% formulation has 1 mmol/ ml of Na and bicarbonate. Half of this dose should be given initially and arterial blood gas analysis repeated and treatment re-assessed.

Metabolic alkalosis (decreased H^+, increased pH, increased standard HCO_3^-)

This is associated with the loss of fixed or non-volatile acid, e.g.

- Pyloric stenosis with loss of hydrochloric acid
- Prolonged nasogastric suction.

Acid-base disturbances must be corrected because if they are severe, they are not compatible with life.

COMPENSATORY CHANGES

In the presence of chronic acid-base disturbances the body tries to minimise the changes in H^+ and pH. The following compensations occur:

Problem	Compensation
metabolic acidosis	respiratory alkalosis
metabolic alkalosis	respiratory acidosis
respiratory acidosis	metabolic alkalosis
respiratory alkalosis	metabolic acidosis

The main underlying problem is referred to as the primary disorder and this is shown by the direction of change in the pH.

Example:
A patient with chronic chest disease has the following:

pH = 7.34 — The pH is low (therefore H^+ ions are increased)

$PaCO_2$ = 50mmHg — The primary disorder is a respiratory acidosis

Std HCO_3^- = 28 — The high std HCO_3^- shows a metabolic alkalosis. This is a compensation for the respiratory acidosis.

A summary of cause, treatment and compensation for acid-base disturbances

Disturbance	**Respiratory acidosis**	**Respiratory alkalosis**	**Metabolic acidosis**	**Metabolic alkalosis**
Defect	Retention of CO_2	Excessive wash out of CO_2	(a) Retention of 'fixed acids' (b) Loss of base HCO_3	Loss of acids Excess HCO_3
Cause	Hypo-ventilation from any cause Obstructed airway, inadequate gas flow	Hysteria, excessive IPPV	(a) Diabetes mellitus, lactic acidosis, shock, cardiac arrest, gangrene, etc. (b) Diarrhoea, fistulae, intestinal obstruction	Pyloric stenosis NG suction
Treatment	Increase ventilation. Treat cause	Decrease ventilation	(a) Treat the underlying cause. (b) (Give $NaHCO_3$ only for HCO_3^- losing met. acidosis)	Treat cause
pH	Low	High	Low	High
$paCO_2$			Normal	Normal
Std HCO_3^-	Normal	Normal		
Compensation	Metabolic alkalosis	Metabolic acidosis	Respiratory alkalosis	Respiratory acidosis

CHAPTER 60

CLEANING AND STERILISATION OF ANAESTHETIC EQUIPMENT

Outline:

Introduction

Definitions

Methods of disinfection and sterilisation

Sterilising specific items of equipment

Special cleaning problems:
- Tubercle bacilli
- Tetanus
- AIDS and hepatitis

INTRODUCTION

Attention must always be given to the general cleanliness and sterility of anaesthetic equipment. Absolute sterility is not always essential but infections have been spread through the contamination of anaesthetic equipment. All equipment that enters the respiratory tract should be kept as clean as possible. This applies to endotracheal tubes, masks, airways and laryngoscopes.

DEFINITIONS

Decontamination (basic cleaning) removes unwanted matter e.g. blood, vomit, mucous and dirt by washing or scrubbing.

Disinfection does not kill all micro–organisms but will reduce their numbers to a level not normally harmful.

Sterilisation destroys all bacteria and viruses including bacterial spores and is essential for all pieces of equipment which come into contact with normally sterile body tissues or with wounds in skin or mucous membranes.

METHODS OF DISINFECTION AND STERILISATION

Disinfection

- ***Boiling for 15 minutes***. This does not kill all organisms. It kills all bacteria including the tubercle bacillus but it does not kill the spores of certain organisms. It is assumed that in the operating theatre these spores are not normally present because of the care and cleanliness of the area.
- ***Pasteurisation*** is a process that reduces the number of micro-organisms by mild heat through washing, rinsing and drying cycles. Washing cycles vary from 5 minutes at 100° C, 10 minutes at 80° C or 20 minutes at 70°. It is suitable for rubber items.

- ***Chemical methods***
  - Chlorhexidine (Hibitane). Available in various strengths from 0.05% to 5% aqueous solution.
  - Glutaraldehyde 2% freshly prepared solution for 1 hour. (1% solution used routinely for instruments has been found effective).
  - Sodium hypochlorite (bleach)
    0.5% for porous surfaces, bench surfaces, floors and walls. (Any surfaces contaminated by spillage).
    0.05% for general cleaning of uncontaminated surfaces. Leave the solution in contact for at least 30 minutes.
    (Note: this solution may corrode metal objects).
  - Ethanol 70% for 1 hour. This is used after decontamination. Methylated spirits (95% ethanol) suitably diluted may be acceptable.
    Use with great caution near electrical appliances and naked flames because it is highly flammable. Commercial preparations of alcohol and antiseptic (e.g. Savlon or Chlorhexidine) may also be used.
  - Iodine 0.5% in 70% ethanol for 30 minutes.

Sterilisation

- ***Autoclaving.*** The equipment is wrapped and sterilised in a steam chamber at a temperature of 122°C (250°F) and a pressure of 15psi for 15-30 minutes. This method destroys bacteria, spores and viruses.

Operating temperature (°C)	Holding time
121°	15 minutes
126°	10 minutes
134°	3.5 minutes

- ***Dry heat. (Dry air oven)*** A temperature of 170°C for one hour destroys all spores and organisms. This is useful for powders, oils, greases and glass syringes but is slow.

121° C	16 hours
140° C	3 hours
160° C	2 hours
170°C	1 hour

- ***Boiling.*** 10-30 minutes at 100°C depending on the size of the instruments. This may require to be adjusted for altitude.

- ***Chemical sterilisation.*** Chemicals are useful for things that will not withstand heat sterilisation but they may destroy some metals, rubber and plastics.
  - Ethylene oxide. This does not destroy rubber or plastic and does not corrode metals. It destroys all bacteria and spores and penetrates paper, plastic and cellophane. However, it is expensive, explosive and slow (takes up to 12 hours). It is necessary to remove all absorbed ethylene oxide from the article before use. This may take several days.
  - Glutaraldehyde (Cidex) kills both bacteria and spores.

DISINFECTING OR STERILISING SPECIFIC ITEMS OF EQUIPMENT

The following items only require disinfecting

Endotracheal tubes Clean and wash thoroughly both inside and outside with soap and water and a bottle brush. Disinfect by pasteurising or placing for 10 minutes in instrument Savlon and rinsing. Store in a clean container. Autoclaving is ideal for sterilising endotracheal tubes, though the life of the tube is shortened.

Face masks Wash thoroughly with soap and water. Disinfect by pasteurising or soaking for 10 minutes in instrument Savlon and rinsing.

Laryngoscope blades If only one laryngoscope is available then wash it between cases with soap and water. Soak for 10 minutes in instrument Savlon at the end of the day.

Corrugated tubing Rinse daily in water, soak in Eusol or pasteurise. Ensure all water is removed from the tube. Soak once a week in instrument Savlon.

Ventilators These can be protected by bacterial filters if available. Follow the manufacturer's instructions on cleaning and maintenance.

These items require sterilisation

Spinal packs These should be autoclaved. The spinal anaesthetic solutions are best excluded from the packs, because they must not be re-autoclaved. The packs should be re-autoclaved if not used within a certain time (28 days).

Glass syringes and needles Wash these with cold water and a brush. Remove the plunger from the syringe. Syringe the needle through to make sure that it is not blocked. Sterilise by autoclaving or hot air oven.

SPECIAL CLEANING PROBLEMS

Cleaning and sterilising anaesthetic equipment contaminated by tubercle bacilli

- Choose an open circuit in tuberculosis cases in preference to the circle absorber, which is very difficult to sterilise.
- Wear a gown and gloves if the patient has "open" tuberculosis.
- Endotracheal tubes, masks, airways, corrugated tubing and reservoir bags can all be soaked in Cidex solution. Cidex solution destroys all vegetative organisms in 10 minutes and spores (e.g. tetanus) in 3 hours. The shutters of the non-rebreathing valve can be autoclaved according to the manufacturer's instructions.
- If you have to use the circle absorber
  - Discard the soda lime
  - Autoclave the metal parts
  - Autoclave the rubber tubing for 15 minutes if possible. Discard plastic tubing.
- Scrub the anaesthetic machine with 70% alcohol or isopropyl alcohol. Do this after any case in which a bad chest infection is known to have existed pre-operatively.

Sterilising equipment contaminated by tetanus

- Soak the equipment in Cidex solution for three hours. If Cidex is not available, then any equipment that can be autoclaved should be. The rest must be soaked in instrument Savlon or chlorhexidine.

  Cidex is a solution of glutaraldehyde and does not affect rubber or plastic. It does not interfere with the conductivity of rubber. It washes off easily and is non-corrosive. To sterilise equipment contaminated with bacteria, soak in Cidex for 10 minutes (this applies to cases of tuberculosis too). To sterilise equipment contaminated with spores, e.g. tetanus, soak for 3 hours.

Disinfecting and sterilising procedures for equipment used for patients with AIDS (or known to be HIV + ve) and hepatitis

The HIV virus is unstable at room temperatures of 20–22°C although hepatitis B is stable. Since the secretions and tissues that contain the HIV virus may also contain hepatitis B virus (and hepatitis non A, non B virus), use disinfecting procedures that are effective against both agents.

- Use disposable equipment wherever possible.
- Burn swabs and other combustible material.
- Before you deal with contaminated equipment, put on a gown, mask, goggles and heavy-duty gloves.
- **Before** disinfecting, clean instruments thoroughly:
  - Use cold water and detergent.
  - Don't splash.
  - If prompt physical cleaning is not possible, soak the instruments in the appropriate disinfectant solution and follow the recommended procedures later.
  - Then sterilise by one of methods described above.

(A protocol for health care workers accidentally exposed to contamination with body fluids from an infected patient is described in Chapter 44).

CHAPTER 61

HAZARDS IN THE OPERATING THEATRE

Outline:

Fires and explosions

Static electricity

Electrical hazards

Radiation injury

Air pollution

Power failure

FIRES AND EXPLOSIONS

Both of these can cause death or injury to the patient in the theatre. A fire can only occur if we have three things:

- A spark or a hot surface
- A flammable substance
- A source of oxygen

Sources of sparks or heat

- Static electricity (discussed in detail below)
- Faulty electrical switches and apparatus, e.g. saws, plaster cutters, drills, x-ray apparatus, endoscopy instruments
- Foreign matter, e.g. dirt or grease in the oxygen or nitrous oxide cylinders
- Diathermy
- Open flames.

Flammable substances

- Ether
- Ethyl chloride
- Solutions in spirits.

The concentrations of the agents used in anaesthesia are well within the explosive range and the addition of oxygen makes them even more dangerous. (Some authors claim that the EMO machine used with ether and air only and with no oxygen added, is safe for use with the diathermy machine. However explosions have been reported by others and it is wise not to use diathermy at all if ether is being used, even in the smallest concentrations).

STATIC ELECTRICITY

Electricity is present in the atmosphere. A static electric charge occurs if two materials which conduct electricity poorly are brought into contact and then separated. If there is friction or movement between the two, a spark is produced and a spark, of course, can produce an explosion. Examples of non-conductors which can spark if they touch each other.

- Plastic
- Woollen fabrics
- Non-conducting rubber
- Synthetic materials such as nylon and terylene.

These should always be avoided in the operating theatre, using special conducting rubber instead. This rubber has graphite impregnated in it. It is black and has a yellow coding (either a yellow line or a stamp) to show it is antistatic.

Other precautions which can be taken to reduce the incidence of static electricity

- ***The floors in the operating room*** should have a conductivity of the same order as the other items resting on them. Terrazzo, concrete, or conductive rubber or plastic, placed on existing floors tend to give the desired conductivity (25,000 - 500,000 ohms when measured by two electrodes placed three feet apart).
- ***Clothing.*** If in the operating room, don't wear clothing that could acquire a static charge. Avoid wool, plastic and nylon fabrics and wear cotton or other anti-static outer clothes instead. Wear aprons of conductive rubber. Wear anti-static boots or conductive canvas overshoes.
- Maintain a minimum relative ***humidity*** of 60% in the operating room. Static sparks are more frequent when the air is dry.
- ***Ventilation***. Anaesthetic gases are heavier than air and tend to collect at ground level. Air within one foot of the ground is the most dangerous. Fresh air should enter at the top and stale air should be drawn out at the bottom. For the same reason switches should not be placed close to the ground.
- ***Chains*** are sometimes attached to the anaesthetic machines and trolley to disperse the electric charge to the earth.
- ***Electric switches and electric apparatus*** should be inspected regularly by the electrician.
- ***Fire fighting equipment*** should always be available outside the operating theatre.

Smoking and open flames must be forbidden in the operating room.

ELECTRICAL HAZARDS

There are electrical hazards associated with the operating theatre and similar situations where anaesthesia is used. They may occur when patients are:

- Connected to or in contact with faulty electrically-operated medical equipment
- Accidentally connected to electric circuits by spillage of blood or saline into equipment being used
- Dependent on electrical equipment to replace or support vital organ functions
- Exposed to fire or explosions
- Undergoing treatment when safe levels of electrical energy are exceeded.

Additional care in keeping patients away from these hazards is needed when they are unattended or unconscious.

Electric shock

This occurs when the body actually becomes part of an electrical circuit with significant current flowing in it. Wiring defects, faulty equipment components and deteriorated insulation all allow abnormally high currents to flow in otherwise safe systems. ***Lack of maintenance*** and ***misuse*** are the usual causes. Sensing devices applied during treatment, especially to the heart, introduce the danger of electric shock even at low current levels when sweating, electrode paste or the moist environment of a hospital operating bed lowers the resistance of contacts to the patient's body and permits more current to flow.

Individual susceptibility is variable and extra care is needed if the patient is strapped to the equipment or is unconscious.

Macroshock is the most common form and occurs when the body conducts an electric current which does not pass directly through the heart. It varies in intensity from mild sensory stimulation at 5 to 10 milliamperes (mA), becoming more painful as the current increases. At 50 to 60mA muscular contraction occurs and as the current increases towards 100mA the victim cannot release his grip and breathing becomes extremely difficult. Somewhere above this level of current respiratory paralysis, cardiac arrest and severe burning occur.

Microshock may occur when very tiny currents, such as 100μA, are intentionally or otherwise passed directly through the heart muscle; e.g. direct cardiac catheterisation, measurement of cardiac output, etc.

High frequency currents i.e. alternating currents (AC) above 50 hertz are less likely to produce electric shock but can cause burns and interference with other devices such as pacemakers.

Direct current (DC) is less likely to cause ventricular fibrillation than high frequency alternating currents (above 50Hz) but can cause a more pronounced skeletal muscle contraction. DC passing through any body tissue for more than very short periods can cause injury by electrolysis. Low voltages (3V) as used in endoscopies can do this.

Nerve damage often occurs in electric shock especially with high currents. The spinal cord may be injured by large currents passing from head to foot or from arm to arm.

Electrical burns and electrically initiated burns

There are three types of electrical burn injury:

- Carbonisation of skin (from burns at **very** high temperatures of 1,000°C)
- Flame burns
- Direct heating of tissues by electric current producing coagulation and necrosis at entry and exit points and associated injury in muscle and blood vessels etc. through which current passes. The associated injury occurs at some distance from the body surface and results in much greater tissue destruction than is apparent at first inspection.

Note that patients suffering impaired circulation and prolonged contact may be burnt under conditions which would not hurt a healthy person, while those insensitive to pain or under anaesthesia may not realise that they are being burnt.

Electrosurgical units e.g. diathermy are arranged so that current from the active electrode flows through the patient and back to the generator via the dispersive cable. If other paths are available, the current will divide and flow in them. Burns to both patient and surgeon may occur through small areas of body contact. For example, with a broken dispersive cable, the current may return via ECG electrodes.

Electric blankets have large surface areas but also many pressure points through which high current levels may pass, generating heat. Don't use electric blankets in conjunction with electrosurgery. Use a water blanket instead.

Some ***other electrical burn hazards*** include:

- Poor siting of return electrode. It should be near the operation site and where sufficient soft tissue provides a large contact area.
- Damaged leads, electrodes, etc.
- Skin preparation materials may impair the function of the skin as a return electrode region. If these preparations are flammable they may ignite.

Electrically initiated burns may occur when patient heating pads, lamps, humidifiers etc. are used to excess.

Unintentional overheating or overcooling of blood before or during transfusion may damage the blood or even result in cardiac arrest.

RADIATION INJURY

Radiant energy (radiation) may cause damage to eyes or skin, dehydration, ultrasound damage as a result of excessive rises in temperature at tissue interfaces and laser damage.

Types of radiation include:

Ionising radiation from X-rays or radio-isotopes.

Electromagnetic radiation which may interfere with other equipment: power mains, electrosurgical units, ECG and some types of pacemaker.

Microwave fields which may cause pathological tissue damage.

AIR POLLUTION IN THE OPERATING THEATRE

It is not surprising that in places where the air is highly polluted, certain medical conditions occur more frequently than they do in places where the air is cleaner. Three of those conditions are spontaneous miscarriage, congenital abnormalities and liver disorders.

It is especially important, then, that in a hospital, particularly in a hospital operating theatre, we provide the cleanest air possible for patients and particularly for staff. The patient spends only a short time in the operating theatre whereas nursing and medical staff spend many hours each day in this atmosphere and are at risk from health problems.

The operating theatre has its own particular sources of air pollution.

Pollution from waste anaesthetic gases

Waste anaesthetic gases can escape from:

- Faulty valves
- The ventilator
- Poorly fitted components in the breathing circuit
- Spilt anaesthetic drugs
- Expired gases from the spill valve of the anaesthetic breathing system
- Gases exhaled by the patient after anaesthesia.

This pollution can be reduced by

- Regular thorough inspection of all anaesthetic equipment
- Employing anaesthetic techniques which limit or avoid the use of inhalational gases and agents e.g.
  - Circle system
  - Total intravenous anaesthesia
  - Regional techniques
- An efficient scavenging system.

Scavenging devices

The scavenging device works by collecting the expired gases from the exhalation valve or the spill valve in the machine or ventilator. Special valves and other devices have been designed for these purposes. From these valves a lightweight tube takes the gases to a disposal system.

In the ***passive disposal system*** the gases are conducted to a wall outlet and piped to an area outside the theatre. This area must be a place where people do not normally go and where there is no risk of cigarette ends or lighted matches being thrown. This is the disposal system recommended for the EMO vaporiser.

The ***active disposal system*** is used in many operating theatres with the conventional gas machine. Gases from the exhalation valves of the machine (or ventilator) flow into a reservoir, which is continuously emptied by a suction device.

No system is one hundred percent effective but the active system is more efficient than the passive one.

POWER FAILURE

Critical areas employing electrically driven equipment such as some respirators and dialysis machines require standby equipment (i.e. generators) to deal with this emergency.
Uninterrupted power supply units (UPS) are recommended for equipment in such areas.

CHAPTER 62

DOs AND DON'Ts AND SOME COMMON PROBLEMS

DOs AND DON'Ts

DO

- See every patient before surgery if possible. Plan a suitable anaesthetic and decide on whether premedication requires to be ordered.
- Inform the surgeon both before and during the surgery if you are concerned about the patient's general condition.
- Whenever you have a poor risk patient, always have a large bore cannula in place and a good intravenous infusion running.
- Have a pumping device available for intravenous fluids if you expect major blood loss.
- Remember that large volumes of blood are lost into the sites of fractures.
- Monitor anaesthetised patients meticulously and chart the readings immediately.
- Choose a regional anaesthetic technique wherever possible in preference to general anaesthesia, provided there is no contraindication to a regional technique and it is appropriate for the surgery which is to be performed.
- Check the anaesthetic machine before **every** anaesthetic.
- Pre-oxygenate (before induction) every poor risk patient, every potentially difficult intubation and every patient for a rapid sequence induction.
- With poor risk patients, reduce the prescribed dose of anaesthetic drugs and increase the inspired oxygen concentration.
- Remember: elderly patients have a very prolonged circulation time. You need to wait much longer before the drugs administered take effect.
- Have an assistant to help monitor and/or ventilate the patient, especially if you don't have a mechanical ventilator.

- In thyroidectomy patients, watch for respiratory difficulties following extubation. Always be ready to re-intubate if necessary.

- Note when pharyngeal packs are inserted and when they are removed.

- Observe aseptic (sterile) technique for spinals and for insertion of intravenous cannulae.

- Before a spinal anaesthetic, check the patient's blood pressure and insert an intra-venous cannula.

- If patients have had anaesthetic complications, make sure you see them after surgery. Try to see all patients post-operatively - even routine cases.

- Take time to prepare everything that may be necessary before commencing.

DON'T

- Don't give a general or a regional anaesthetic (e.g. spinal) unless oxygen, a means of ventilation, suction and resuscitation drugs are available in the operating theatre.

- Don't premedicate the following patients with narcotic analgesics
  - Moribund patients
  - Babies less than 6 months old
  - Patients for caesarean section or forceps delivery
  - Patients with head injuries, with a raised intracranial pressure

- Don't concentrate on the surgical procedure and neglect to monitor the patient.

- Don't anaesthetise a patient with a pre-existing medical problem unless an attempt has been made to correct it. This applies especially to elective surgery.

- Don't leave an unconscious patient **for any reason at all** unless you have asked someone responsible to watch the patient for you.

- Don't hesitate to intubate a patient if a clear airway cannot be maintained with other means (e.g. re–positioning of head or jaw, the use of a Guedel airway or LMA).

- Don't let a pregnant mother at term lie on her back (supine).

- Don't give a hypotensive mother at term either a general or a spinal anaesthetic until the cause is diagnosed and treated. Uncontrolled bleeding is an exception where general anaesthesia with aggressive fluid resuscitation occurs concurrently with surgery to correct the problem.

- Don't hesitate to refuse to anaesthetise a patient if you feel you haven't had enough experience.

- Don't hesitate to ask for help if you need it, even if it means the surgeon has to get unscrubbed.

- Don't let any one force you to do something which in your view is not safe.

- Don't let experience in the field of anaesthetics make you feel you can bypass the safety measures you have learned.

- Don't use relaxants and paralyse patients unless you are sure of intubating them or ventilating them with a mask. Don't paralyse patients with an obstructed airway, or those with anatomical or pathological problems that will make intubation difficult.

- Don't use suxamethonium any later than 48 hours after moderate to major burn injuries.

- Don't use force when intubating a patient. Don't use a big tube, especially in children. Don't over-inflate the cuff.

- Don't discharge a child who has had an endotracheal intubation until at least 6 hours after extubation.

- Don't extubate a patient until you are sure that reversal is complete.

- Don't anaesthetise a child without first working out the doses of drugs and the volumes of maintenance fluid that you will need and preferably checking with another anaesthetist.

- Don't use adrenaline for local anaesthetic blocks on the fingers, toes or penis.

COMMON PROBLEMS AND HOW TO MANAGE THEM

DIFFICULTY IN CANNULATING A VEIN

- Proceed with an inhalational induction using ether or halothane if there is no risk of aspiration and you are confident that the airway will be easy to manage. The resulting vasodilation may help you cannulate.
- Induce anaesthesia with IM Ketamine. Allow the patient to breath oxygen and halothane or ether. Proceed with cannulation.
- Cut down on to a vein.
- Cannulate a central vein.
- Consider intraosseous access in a child.

ACCIDENTAL INTRA-ARTERIAL INJECTION OF THIOPENTONE (see Chapter 6 Pharmacology)

- Postpone surgery if possible.
- Leave the needle in the artery.
- Flush with anticoagulant e.g. heparin/saline (10 units/ml) 500 units
- Inject procaine 0.5-1% 10-20ml or lignocaine 1% 5-10 ml or papaverine 10-40mg in 10ml of normal saline intra-arterially.
- Heparin 10,000 units may be given intra-arterially.
- The patient may be allowed to breathe oxygen/ether or oxygen/halothane in order to vasodilate the collateral circulation.
- Elevate the arm.
- Surgery to restore the blood supply through the artery may be required.

Other measures

  - A supraclavicular brachial plexus block or a stellate ganglion block may increase the collateral circulation to the limb. This must be done by someone with the necessary expertise, **before** the heparin is administered.
  - Support the patient's circulation and respiration.

WHEN THE AIRWAY IS OBSTRUCTED by the tongue falling back

- Extend the head
- Lift the chin and perform jaw thrust
- Insert a Guedel airway. If there is no relief try inserting a LMA.
- If there is still no relief perform endotracheal intubation.

WHEN THE AIRWAY IS OBSTRUCTED by jaw muscle spasm and the tongue falling back

- Use a mouth gag to force open the jaws
- Insert a Guedel airway and continue the anaesthetic
- Consider deepening anaesthesia

LARYNGEAL SPASM

- Stop surgical stimulation
- Give the patient 100% oxygen to breathe via the mask
- Deepen the general anaesthesia
- If there is no relief, give suxamethonium 0.1-0.2mg/kg IV. This will correct the problem if it is true laryngospasm. Continue to ventilate with 100% oxygen.
- Endotracheal intubation if the airway is still not secured (give suxamethonium 1mg/kg).

The spasm may recur on extubation.

BRONCHOSPASM

- Increase inspired oxygen concentration.
- Deepen the general anaesthesia using halothane or ether.
- Salbutamol, either via a nebuliser through an adapter in the circuit or 0.25mg IV over 1 minute (=250 micrograms).
- If salbutamol is unsuccessful give adrenaline 50–100 micrograms IV (0.5 – 1 ml of 1:10,000 or 0.05 – 0.1 ml of 1:1000)
  or
  adrenaline 0.5-1mg IM (0.5 – 1ml of 1:1000) titrated to effect.
- Give hydrocortisone 100mg IV (this will take a number of hours to work).
- Aminophylline 250mg slowly IV.

THE PATIENT FAILS TO RELAX

The cause must be found. **Don't** attempt intubation until the patient is relaxed. Possible causes:

- The tourniquet is still on.
- The needle or cannula is in the tissues.
- The dose of relaxant was inadequate.
- The drug was defective.
- Less common causes:
  - An early sign of malignant hyperpyrexia.
  - Contractures, not relaxed by muscle relaxant.
  - Underlying myotonia dystrophica.

ANTICIPATED DIFFICULT INTUBATION (See also Chapter 21)

- If you suspect anatomical reasons, e.g. bull neck or receding chin:
  - Pre–oxygenate for 3-4 minutes.
  - Make sure an introducer, bougie and a laryngeal mask are available.
  - Position the patient carefully (sniffing position).
  - Use suxamethonium rather than a long-acting relaxant, (inhalational induction in children).
  - Apply external backwards pressure on the larynx.
- If you suspect pathological reasons, e.g. tumour of the neck, jaw, face, contractures, tumours or inflammation of the pharynx or mouth:
  - Don't paralyse the patient unless you can guarantee ventilation.
  - Use an inhalational induction.
  - Occasionally you may need an awake intubation or tracheostomy before the anaesthetic is induced.

FAILED INTUBATION (See also Chapter 21 pages 322-323)

Hypoxia must be avoided at all costs. In patients with a full stomach aspiration of gastric contents is very likely.

- Call for help and failed intubation equipment.
- Mask ventilate with 100% oxygen. Continue applying cricoid pressure if there is a risk of aspiration. Assess adequacy of ventilation and oxygenation.
- Decide whether it is essential to proceed with surgery or not. **If not**, wake the patient up and postpone the procedure.

In elective surgery

- Plan an alternative method using a regional technique or awake intubation.

In emergency surgery (e.g. caesarean section for foetal distress)

- Continue with mask ventilation and cricoid pressure. Consider the use of a laryngeal mask airway with cricoid pressure.
- Allow the short acting muscle relaxant to wear off and get the patient breathing with either a face mask +/- Guedel airway or laryngeal mask.
- If a long-acting muscle relaxant has been administered, optimise attempts at intubation by improved positioning and by using intubation aids e.g. stylet or bougie, other laryngoscope blades and call for a more experienced anaesthetist.

YOU CAN'T VENTILATE OR INTUBATE THE PATIENT

If all the above methods have failed

- Cricothyroid puncture with a 12 or 14 gauge IV cannula.
  (See page 324 Chapter 21).
  Note: the 12G or 14G cannula is inserted into the trachea through the cricothyroid membrane. Confirm correct placement by air aspiration through the cannula using a syringe.
  Connect a 2 ml syringe to the cannula. Insert a 7.5mm ETT connector into the syringe barrel. This will then provide the 15/22 mm connection for a self-inflating bag or the anaesthetic breathing circuit. It is difficult to give big tidal volumes but use 100% oxygen and you will keep the patient alive.
- Tracheostomy.

INCREASING DIFFICULTY IN VENTILATING AN ANAESTHETISED PATIENT

Possible causes:

- Airway obstruction
  - The patient's upper airway (including laryngeal spasm)
  - The endotracheal tube blocked or kinking
  - The machine
- Bronchospasm
- Returning muscle tone in the chest wall and diaphragm
- Pneumothorax
- Abdominal distension interfering with ventilation

Management

- Take the patient off the ventilator. Ventilate manually.
- Suction the ETT.
- Increase the oxygen concentration.
- Treat the cause.

TEARS

Inadequate depth of anaesthesia.

RASHES

- Drug reaction. Avoid any further doses.
- If associated with hypotension and bronchospasm, a rash may indicate a transfusion reaction or anaphylaxis - see below.

SWEATING

Possible causes

- Light anaesthesia
- Carbon dioxide retention, because of:
  - Hypoventilation
  - Respiratory obstruction
  - The carbon dioxide absorber is not working
  - Increased dead space
- Hypoxia
- Increased temperature
- Shock
- Hypoglycaemia
- Malignant hyperpyrexia.

HICCUPS

- Deepen anaesthesia
- Stop surgical stimulation in the region of the diaphragm
- Give an additional dose of muscle relaxant.

ANAPHYLACTIC REACTION TO DRUGS

- Stop administration of suspected drug or drugs
- IV infusion of crystalloid and colloid solution
- Give 100% oxygen by mask or endotracheal tube
- Adrenaline IV 0.1-0.5ml of 1:1000 or 1-5ml of 1:10,000 solution. Repeat as necessary.
  NB. Increase dose of adrenaline up to 1mg if intractable hypotension develops. Commence adrenaline infusion.
- Hydrocortisone 200mg IV
- Salbutamol 250 micrograms IV (second line for bronchospasm)
- Aminophylline 250mg IV for bronchospasm (third line for bronchospasm)
- Antihistamines such as promethazine (Phenergan) 25mg slowly IV or chlorphenamine maleate (Piriton) 10mg IV.
- Check arterial blood gases and consider Na bicarbonate if pH < 7.1.

CENTRAL CYANOSIS

Management:

- Turn off all anaesthetics.
- Ventilate with 100% oxygen.
- Monitor pulse and blood pressure.
- Watch for cardiac arrest. Treat the cause.

Always ventilate the cyanosed patient by hand.

Some causes of cyanosis:

- Inadequate oxygen supply
  - Empty cylinders
  - Defective flow meters
  - Disconnection of apparatus
  - Doubtful quality of oxygen
  - A malfunctioning ventilator
- Respiratory obstruction:
  - Upper airway
  - The endotracheal tube
  - Bronchospasm.
- Pneumothorax.
- Hypoventilation of lungs, for example pulmonary oedema, pneumonia, collapse.
- Inadequate blood flow to the lungs due to:
  - Hypotension.
  - Cardiac failure.
  - Pulmonary embolism.
- Other causes of cyanosis
  - Air embolism
  - Malignant hyperpyrexia
  - Anaphylaxis.

PULMONARY OEDEMA

- Stop the fluid load.
- Give oxygen and IPPV (IPPV if severe and unresponsive to therapy).
- Give diuretics - frusemide 20- 40mg IV.
- Give Digoxin (only for rapid atrial fibrillation).
- Prop up the patient.

TRANSFUSION REACTION

- Stop blood transfusion
- Give fluids - Hartmann's solution or colloids to maintain urine output
- Give adrenaline if hypotension develops which would indicate a severe reaction. Start at low doses such as 50-100 micrograms, repeated as necessary. High doses (i.e. 1mg) such as for cardiac arrest may be required if cardiovascular collapse occurs. Clinically this can be similar to anaphylaxis.
- Give oxygen: intubate and ventilate if severe reaction.
- Give Mannitol 25G IV or Lasix 40mg IV.
- Give steroids: Hydrocortisone 100mg IV.
- Dialysis may be required if acute renal failure develops. (Send the remaining donor blood, a fresh sample of the patient's blood and urine to the laboratory).

INCREASED BLEEDING FROM A WOUND

- Surgical bleeding from an artery or vein must be treated by the surgeon.
- Capillary bleeding. Check the following possible causes:
  - Carbon dioxide retention
  - Oxygen lack
  - Increased venous pressure, (in head and neck surgery this may be caused by coughing or straining)
  - Bleeding problems
    - Lack of platelets
    - Lack of clotting factors
    - Lack of calcium
    - D.I.C.
    - Continued use of aspirin
  - Mismatched transfusion.

SEVERE HYPOTENSION

- Treat any underlying cause, e.g. blood loss, fluid loss, arrhythmias, myocardial infarction, pressure from retractors, traction on the gut, anaphylaxis, tension pneumothorax, anaesthetic drugs.
- Turn off anaesthetic agents and ventilate with 100% oxygen.
- Give IV fluids rapidly. If necessary insert a second cannula.
- Watch for cardiac arrest. Investigate and treat the cause. e.g. hypoxia. If there is no response, try vasopressors such as
  - Metaraminol (Aramine) 1 mg IV p.r.n.
  - Adrenaline 0.1-1mg IV, titrate to response. If diagnosed cardiac arrest, give 1mg of adrenaline and proceed with CPR.

CARDIAC ARREST (See Chapter 58)

- Airway – Clear
- Breathing – Give mouth to mouth ventilation
  – Ventilate with a bag, valve and mask
  – Insert LMA or
  – Endotracheal intubation
- Cardiac massage
- Drugs and drip – Adrenaline 1mg.
- ECG – Ventricular fibrillation : Defibrillate
  – Ventricular tachycardia : Lignocaine 1% 1mg/kg IV or amiodarone 300mg IV (if available)
  – Asystole : Adrenaline 10ml of 1:10,000, or 1ml of 1:1000
- Find and treat the cause.

AIR EMBOLISM

- Place the patient in the head down, left lateral position.
- Give oxygen (100%) via mask or endotracheal tube.
- Treat arrhythmias and hypotension.
- Prevent further emboli by flooding the wound with saline and jugular compression.
- Aspirate air via a catheter in the right atrium or ventricle.

MALIGNANT HYPERPYREXIA (See Chapter 46)

- Stop volatiles and keep the patient sedated with IV benzodiazepines e.g. diazepam or midazolam.
- Ventilate with 100% oxygen.
- If it is available give dantrolene 1mg/kg IV, with further doses of 1mg/kg up to a maximum of 10mg/kg. (This is a very expensive drug).
- Give IV fluids, i.e. a rapid infusion of crystalloid.
- Cool the patient with ice, fans, wet sheets, ice-cold fluids (saline) and an ice-cold saline gastric lavage. Cease when temperature has come down to 38°C. Continuously monitor core temperature.
- Monitor temperature, urinary output, pulse, blood pressure and if possible arterial blood gases, electrolytes.
- Give sodium bicarbonate 50mmol IV, initially.
- Maintain urine output of 2ml/kg/hr
  Lasix 40mg IV and Mannitol 12.5g IV may be required.
- Glucose/insulin infusion if the potassium (K^+) level is high.
- Treat any arrhythmias which develop.
- Investigate the patient and the patient's family further.

RESIDUAL NEUROMUSCULAR BLOCK

- Treat the cause if possible. Some causes are:
  - Inadequate dose of neostigmine.
  - Electrolyte imbalance and metabolic acidosis.
  - Hypothermia.
  - The use of certain antibiotics e.g. gentamicin.
  - Magnesium sulphate.
  - Underlying medical problems, e.g. a very ill patient, myasthenia gravis, myasthenic syndrome.
- Don't extubate the patient.
- Continue the anaesthetic until spontaneous respiration returns.

POST-OPERATIVE APNOEA

Leave the endotracheal tube in place. Ventilate the patient with oxygen (and nitrous oxide if available).

Treat the cause, which could be:

- Overdose of drugs:
  - Premedication drugs
  - Induction agents
  - Maintenance drugs
  - Analgesics.
- Hypocarbia
- Abnormal blood glucose
- Hypoxia
- Residual neuro-muscular block
  - Depolarising relaxant
    - Low or abnormal pseudocholinesterase
    - Dual block
  - Non-depolarising relaxant
    - Inadequate neostigmine
    - Electrolyte imbalance
    - Metabolic acidosis
    - Myasthenia gravis or myasthenic syndrome
    - Very ill patient
- Hypercarbia
- Hypothyroidism.

DELAYED RETURN OF CONSCIOUSNESS

- Maintain ventilation
  - Oxygen
  - The airway must be clear
    - Lift chin and jaw
    - Insert Guedel airway
    - Insert LMA or endotracheal tube if required
  - IPPV may be required.
- Maintain circulation
  - Monitor pulse and blood pressure
  - Give IV fluids
- **Never** leave the unconscious patient unattended.
- Treat the cause. Some causes are:

  Respiratory problems
  - Hypoventilation
  - Airway obstruction
  - Pneumothorax
  - Lung disease: collapse, pneumonia
  - Decreased blood flow through lungs:
    - Cardiac failure
    - Pulmonary embolism
  - Carbon dioxide absorber not working

  Central depression (drug action)
  - Premedication
  - Induction agents
  - Maintenance agents, e.g. ether or halothane
  - Analgesic drugs.

  Underlying medical problems
  - CVA
  - Hyperglycaemia
  - Hypoglycaemia
  - Sickle cell disease
  - Fat embolism
  - Monoamine oxidase inhibitor drugs
  - Porphyria.

ABNORMAL MUSCULAR MOVEMENTS AFTER ANAESTHESIA

Find the cause:

- Inadequate reversal of muscle relaxant
- Shivering
- Ketamine hallucinations
- Convulsions:
  - Find the cause: hypoxia, hypoglycaemia, low calcium level, withdrawal of anticonvulsants before surgery, pyrexia, central nervous system disease (e.g. cerebral oedema), water intoxication, eclampsia.
  - Maintain a clear airway and give oxygen
  - Give anticonvulsants
    Diazepam: 100 micrograms/kg IV until the convulsion stops, or
    Thiopentone: 50-100mg IV.
  - Give suxamethonium 1mg/kg IV and ventilate with 100% oxygen.
  - Intubate if necessary.

ASPIRATION DURING ANAESTHESIA

- Position the patient in the head down lateral position if airway unprotected.
- Suction
- Give oxygen
- Auscultate the chest for signs of aspiration. If significant aspiration with persistent hypoxia then intubate the patient in the supine position with cricoid pressure.
  then
  - Suction via endotracheal tube
  - Antibiotics (if the gastric aspirate is thought to be infective, i.e. bowel obstruction)
  - Bronchodilators, e.g. salbutamol 250 micrograms slowly IV, adrenaline 50-100 micrograms IV, aminophylline 250mg IV.
  - IPPV
  - Bronchoscopy.

POST–OPERATIVE LARYNGEAL OEDEMA

This is more common in children (See Chapter 20).

- Humidified oxygen
- Give adrenaline nebules, 1mg (with 1ml of normal saline) up to 5mgs until relief is achieved.
- Give steroids (dexamethasone up to 8mg IV or hydrocortisone up to 100mg IV).
- Perform an endotracheal intubation using a very small tube, or tracheostomy if the situation deteriorates in spite of above measures.

WHAT TO DO IF

THE LARGE OXYGEN CYLINDER RUNS OUT DURING A GENERAL ANAESTHETIC

This should **not** happen. Check the cylinder and check with the surgeon the expected duration of surgery. A rough guide: 500 ml of oxygen in a large cylinder lasts for 1 hour at most.

If you do have to change a cylinder

- If the patient is breathing spontaneously (with a face mask or laryngeal mask)
  - Disconnect the machine
  - Give IV ketamine 0.5 - 1 mg/kg and breathe air

    or
  - Connect to EMO - breathe ether starting with 3%. Gradually increase the concentration.
- If you are controlling respiration
  - Use a self inflating bag to ventilate the patient
  - Give IV ketamine 0.5 - 1 mg/kg.

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