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GENERAL QUESTIONS OF ANAESTHESIOLOGY

Study Guide

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This book covers information about basic principles and methods of the modern anesthesiology.

For English-speaking students of higher educational institutions III-IV levels of accreditation, postgraduates.

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CONTENTS

Topic 1. PREOPERATIV PREPARATION

The main aim: to be able to prepare the patient for surgery, to assess the risk of anesthesia, choose the appropriate type of anesthesia, premedication appoint, prepare equipment and instruments for anesthesia.

The student must know:
-The components of modern anesthesia.
-Features of anesthesia preoperative history and physical.
- The risk assessment.
-Classification of modern anesthesia.
-Principles of choice of anesthesia technique.
-Pharmacokinetics of drugs for premedication.
-Mein components of the anaesthetic machine.
-Preparation of the anaesthetic machine.
- Mein components and preparation of monitors.

The student must be able:
-Collect anamnesis and examine of the patient in anestesiological aspect.
-Prognosticate the difficult intubation.
-Evaluate risk of anesthesia according ASA Physical Status Classification.
- Select the appropriate for patient anesthesia technique.
- Prepare the anestesiological equipment to operation.

Short methodical instructions for practical study.

The initial level of students knowledge-abilities performed at the beginning of the study, using the test tasks. Students examine patients which operative intervention coming, see the results of patients laboratory and instrumental investigations, study hospital charts, determine the degree of surgical risk and choose the optimal type of anaesthesia. If it possible they are in an operating-room, if not they decide

situational tasks. All students independent work and their answers controlled by a teacher.

In an educational room students together with a teacher discuss the results of examination, produce the general tactic/pl of conduct of patients in a предоперационном period, optimal type of anaesthesia, general tactic/pl of conduct of patients and послеоперационном period. Students discuss with a teacher by them sufferet errors. After it students pass test control. In the end worked out the totals of work, and students get the estimations of the work on employment.

Mein material

Terminology

Anesthesiology is the science of managing the life functions of the patients organism in connection with surgery or aggressive diagnostic procedure.

Anesthesia, or anaesthesia (from Greek αν-, *an-*, "without"; and αἴσθησις, *aisthēsis*, "sensation".

On October 16, 1846, in Boston, William T.G. Morton conducted the first publicized demonstration of general anesthesia using ether.

The pre-existing word *anesthesia* was suggested by Oliver Wendell Holmes, Sr. in 1846 as a word to use to describe this state.

Anesthesia is reversible, drug-induced condition of:

Amnesia & unconsciousness – inhibition of psychic perception (narcosis, sleep);

Analgesia – blockade of pain impulses;

Neurovegetative blockade;

Immobility (myorelaxation);

Maintenance of adequate gas exchange;

Maintenance of adequate blood circulation;

Maintenance of normal metabolism.

The main aim of anesthesiology is to protect the organism from the operative injury. According to Rene Lerish, "the operation saves the patient by ways which may kill him".

General Anesthesia consist from 3 main parts:

– preoperative evaluation;
– intraoperative management;
– postoperative management

Preoperative Evaluation

Unlike the standard internal medicine H&P, ours is much more focused, with specific attention being paid to the airway and to organ systems at potential risk for anesthetic complications. The type of operation, and the type of anesthetic will also help to focus the evaluation.

The overall goal of the preoperative evaluation is to reduce perioperative morbidity and mortality and alleviate patient anxiety.

The preoperative visit should include the following steps:

I. Problem Identification
II. Risk Assessment
III. Plan of Anaesthetic Technique
IV. Preoperative Preparation.

Problem Identification

Anesthesia preoperative history and physical

A. Note the date and time of the interview, the planned procedure, and a description of any extraordinary circumstances regarding the anesthesia.

B. Current medications and allergies: history of steroids, chemotherapy and herb and dietary supplements .

C. Cigarette, alcohol, and illicit drug history, including most recent use.

D. Anesthetic history, including specific details of any problems.

E. Prior surgical procedures and hospitalizations.

F. Family history, especially anesthetic problems.

Birth and development history (pediatric cases).
G. Obstetrical history: last menstrual period (females).
H. Medical history; evaluation, current treatment, and degree of control.
I. Review of systems, including general, cardiac, pulmonary, neurologic, liver, renal, gastrointestinal, endocrine, hematologic, psychiatric.
J. History of airway problems (difficult intubation or airway disease, symptoms of temporomandibular joint disease, loose teeth, etc).
K. Last oral intake.
L. Physical exam, including airway evaluation (see below), current vital signs, height and body weight, baseline mental status, evaluation of heart and lungs, vascular access.
M.Overall impression of the complexity of the patient's medical condition, with assignment of ASA Physical Status Class (see below).
N. Anesthetic plan (general anesthesia, regional, spinal, MAC). The anesthetic plan is based on the patient's medical status, the planned operation,
and the patient's wishes.
O. Documentation that risks and benefits were explained to the patient.

3. Preoperative laboratory evaluation
A. Hemoglobin**:** menstruating females, children less than 6 months or with suspected sickle cell disease, history of anemia, blood dyscrasia or malignancy, congenital heart disease, chronic disease states, age greater than 50 years (65 years for males), patients likely to experience large blood loss.
B. WBC count**:** suspected infection or immunosuppression.
C. Platelet count: history of abnormal bleeding or bruising, liver disease, blood dyscrasias, chemotherapy, hypersplenism.
D. Coagulation studies: history of abnormal bleeding, anticoagulant drug therapy, liver disease, malabsorption, poor nutrition, vascular procedure.

E. Electrolytes, blood glucose, BUN/creatinine**:** renal disease, adrenal or thyroid disorders, diabetes mellitus, diuretic therapy, chemotherapy.
F. Liver function tests: patients with liver disease, history of or exposure to hepatitis, history of alcohol or drug abuse, drug therapy with agents that may affect liver function.
G. Pregnancy test: patients for whom pregnancy might complicate the surgery, patients of uncertain status by history and/or examination.
H. Electrocardiogram: age 50 or older, hypertension, current or past significant cardiac disease or circulatory disease, diabetes mellitus in a person age 40 or older. An EKG showing normal results that was performed within 6 months of surgery can be used if there has been no intervening clinical event.
I. Chest x–ray: asthma or chronic obstructive pulmonary disease with change of symptoms or acute episode within the past 6 months, cardiothoracic procedures.
J. Urinalysis: genito–urologic procedures; surgeon may request to rule out infection before certain surgical procedures.
K. Cervical spine flexion/extension x–rays: patients with rheumatoid arthritis or Down's syndrome. Routine screening in asymptomatic patients is generally not required.
L. Preoperative pulmonary function tests (PFTs). There is no evidence to suggest that pulmonary function tests are useful for purposes of risk assessment or modification in patients with cigarette smoking or adequately treated brochospastic disease.

Identification of the problems a patient brings to the operating room is one of the most vital, yet easily neglected, components of the perioperative management of the surgical patient. A system–oriented approach to the patient is helpful in completing a thorough preoperative assessment. As is the case elsewhere in medicine, the preoperative evaluation should progress through history (including a review of the

patient's chart), physical examination, and laboratory investigation.

Anaesthetic drugs and techniques have profound effects on human physiology. Hence, a focused review of all major organ systems should be completed prior to surgery. The anaesthetist pays special attention to symptoms and disease related to the cardiovascular, respiratory, and neuromuscular systems as they will directly manipulate these systems during surgery. Because one of the goals of the preoperative evaluation is to ensure that the patient is in the best (or optimal) condition**,** it is important not only to identify symptoms, but also to document their severity and to determine their stability or progress. Patients with unstable symptoms should be postponed for optimization prior to elective surgery.

Cardiovascular system

Symptoms of the following problems must be sought in all patients:

• ischaemic heart disease;
• heart failure;
• hypertension;
• conduction defects, arrhythmias;
• peripheral vascular disease.

Patients with a proven history of myocardial infarction (MI) are at a greater risk of perioperative reinfarction, the incidence of which is related to the time interval between infarct and surgery. This time is variable. In a patient with an uncomplicated MI and a normal exercise test elective surgery may only need to be delayed by 6–8 weeks.

Heart failure is one of the most significant indicators of perioperative complications, associated with increased risk of perioperative cardiac morbidity and mortality. Its severity is best described using a recognized scale, for example the New York Heart Association classification.

New York Heart Association classification

Class I: Cardiac disease without limitation of physical activity.
Class II: Slight limitation of physical activity.
Ordinary physical activity results in angina or fatigue.
Class III: Marked limitation of physical activity.
Class IV: Angina at rest, increased with activity.

Untreated or poorly controlled hypertension may lead to exaggerated cardiovascular responses during anaesthesia. Both hypertension and hypotension can be precipitated, which increase the risk of myocardial and cerebral ischaemia. The severity of hypertension will determine the action required:

• *Mild (SBP 140–159 mmHg, DBP 90–99 mmHg)* No evidence that delaying surgery for treatment affects outcome.
• *Moderate (SBP 160–179 mmHg, DBP 100–109 mmHg)* Consider review of treatment. If unchanged, requires close monitoring to avoid swings during anaesthesia and surgery.
• *Severe (SBP > 180 mmHg, DBP > 109 mmHg)* At this level, elective surgery should be postponed due to the significant risk of myocardial ischaemia, arrhythmias and intracerebral haemorrhage. In an emergency, will require acute control with invasive monitoring.

Respiratory system

Enquire specifically about symptoms of:

• chronic obstructive lung disease;
• emphysema;
• asthma;
• infection;
• restrictive lung disease.

Patients with pre–existing lung disease are more prone to postoperative chest infections, particularly if they are also obese, or undergoing upper abdominal or thoracic surgery. If an acute upper respiratory tract infection is present,

anaesthesia and surgery should be postponed unless it is for a life–threatening condition.

Assessment of exercise tolerance

An indication of cardiac and respiratory reserves can be obtained by asking the patient about their ability to perform everyday physical activities before having to stop because of symptoms of chest pain, shortness of breath, etc.

For example:

• How far can you walk on the flat?
• How far can you walk uphill?
• How many stairs can you climb before stopping?
• Could you run for a bus?
• Are you able to do the shopping?
• Are you able to do housework?
• Are you able to care for yourself?

The problem with such questions is that they are very subjective and patients often tend to overestimate their abilities!

Other conditions which are important if identified in the medical history:

• *Indigestion, heartburn and reflux* Possibility of a hiatus hernia. If exacerbated on bending forward or lying flat, this increases the risk of regurgitation and aspiration.

• *Rheumatoid disease* Limited movement of joints makes positioning for surgery difficult. Cervical spine and tempero–mandibular joint involvement may complicate airway management. There is often a chronic anaemia.

• *Diabetes* An increased incidence of ischaemic heart disease, renal dysfunction, and autonomic and peripheral neuropathy. Increased risk of intra and postoperative complications, particularly hypotension and infections.

• *Neuromuscular disorders* Coexisting heart disease may be worsened by anaesthesia and restrictive pulmonary disease (forced vital capacity predisposes to chest infection

and the possibility of the need for ventilatory support postoperatively. Care when using muscle relaxants.

• *Chronic renal failure* Anaemia and electrolyte abnormalities. Altered drug excretion restricts the choice of anaesthetic drugs. Surgery and dialysis treatments need to be coordinated.

• *Jaundice* Altered drug metabolism, coagulopathy. Care with opioid administration.

• *Epilepsy* Well–controlled epilepsy is not a major problem. Avoid anaesthetic drugs that are potentially epileptogenic.

Previous anaesthetics and operations

These may have occurred in hospitals or, less commonly, dental surgeries. Enquire about any difficulties, for example: nausea, vomiting, dreams, awareness, postoperative jaundice. Check the records of previous anaesthetics to rule out or clarify problems such as difficulties with intubation, allergy to drugs given, or adverse reactions (e.g. malignant hyperpyrexia, see below).

Details of previous surgery may reveal potential anaesthetic problems, for example cardiac, pulmonary or cervical spine surgery.

Family history

All patients should be asked whether there are any known inherited conditions in the family (e.g. sickle–cell disease, porphyria). Have any family members experienced problems with anaesthesia; a history of prolonged apnoea suggests pseudocholinesterase deficiency and an unexplained death malignant hyperpyrexia

Elective surgery should be postponed if any conditions are identified, and the patient investigated appropriately. In the emergency situation, anaesthesia must be adjusted accordingly, for example by avoidance of triggering drugs in a patient with a family history of malignant hyperpyrexia.

Drug history and allergies

Identify all medications, both prescribed and selfadministered, including herbal preparations. Patients will often forget about the oral contraceptive pill (OCP) and hormone replacement therapy (HRT) unless specifically asked. The incidence of use of medications rises with age and many of these drugs have important interactions with anaesthetics.Allergies to drugs, topical preparations (e.g. iodine), adhesive dressings and foodstuffs should be noted.

Social history

• *Smoking* Ascertain the number of cigarettes or the amount of tobacco smoked per day. Oxygen carriage is reduced by carboxyhaemoglobin, and nicotine stimulates the sympathetic nervous system, causing tachycardia, hypertension and coronary artery narrowing. Apart from the risks of chronic lung disease and carcinoma, smokers have a significantly increased risk of postoperative chest infections. Stopping smoking for 8 weeks improves the airways; for 2 weeks reduces their irritability; and for as little as 24 h before anaesthesia decreases carboxyhaemoglobin levels. Help and advice should be available at the preoperative assessment clinic.

• *Alcohol* This is measured as units consumed per week; >50 units/week causes induction of liver enzymes and tolerance to anaesthetic drugs. The risk of alcohol withdrawal syndrome postoperatively must be considered.

• *Drugs* Ask specifically about the use of drugs for recreational purposes, including type, frequency and route of administration. This group of patients is at risk of infection with hepatitis B and human immunodeficiency virus (HIV). There can be difficulty with venous access following IV drug abuse due to widespread thrombosis of veins. Withdrawal syndromes can occur postoperatively.

• *Pregnancy* The date of the last menstrual period should be noted in all women of childbearing age. The

anaesthetist may be the only person in theatre able to give this information if X–rays are required. Anaesthesia increases the risk of inducing a spontaneous abortion in early pregnancy. There is an increased risk of regurgitation and aspiration in late pregnancy. Elective surgery is best postponed until after delivery.

The examination

As with the history, this concentrates on the cardiovascular and respiratory systems; the remaining systems are examined if problems relevant to anaesthesia have been identified in the history. At the end of the examination, the patient's airway is assessed to try and identify any potential problems. If a regional anaesthetic is planned, the appropriateanatomy (e.g. lumbar spine for central neural block) is examined.

Cardiovascular system

Look specifically for signs of:

• arrhythmias;
• heart failure;
• hypertension;
• valvular heart disease;
• peripheral vascular disease.

Don't forget to inspect the peripheral veins to identify any potential problems with IV access.

Respiratory system

Look specifically for signs of:

• respiratory failure;
• impaired ventilation;
• collapse, consolidation, pleural effusion;
• additional or absent breath sounds.

Nervous system

Chronic disease of the peripheral and central nervous systems should be identified and any evidence of motor or sensory impairment recorded. It must be remembered that some disorders will affect the cardiovascular and respiratory systems, for example dystrophia myotonica and multiple sclerosis.

Musculoskeletal system

Patients with connective tissue disorders should have any restriction of movement and deformities noted. Patients suffering from chronic rheumatoid disease frequently have a reduced muscle mass, peripheral neuropathies and pulmonary involvement. Particular attention should be paid to the patient's cervical spine and temperomandibularjoints.

The airway

All patients must have an assessment made of their airway, the aim being to try and predict those patients who may be difficult to intubate.

Observation of the patient's anatomy

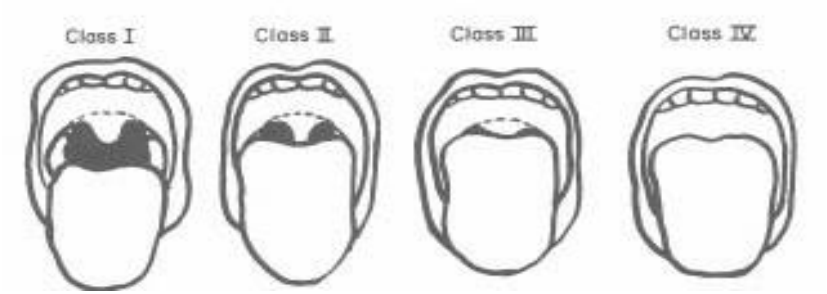
Look for:

• limitation of mouth opening;
• a receding mandible;
• position, number and health of teeth;
• size of the tongue;
• soft tissue swelling at the front of the neck;
• deviation of the larynx or trachea;
• limitations in flexion and extension of the cervical spine.

Finding any of these suggests that intubation may be more difficult. However, it must be remembered that all of these are subjective.

Simple bedside tests

• *Mallampati criteria* The patient, sitting upright, is asked to open their mouth and maximally protrude their tongue. The view of the pharyngeal structures is noted and graded I–IV.



Class 1: Soft palate, fauces, uvula, anterior and posterior faucial pilars can be seen.
Class 2: Soft palate, fauces, uvula can be seen. The tongue masks anterior and posterior faucial pillars.
Class 3: Soft palate and the base of uvula can be seen only.
Class 4: Only hard palate is visible.
Grades III and IV suggest difficult intubation.

• *Thyromental distance* With the head fully extendedon the neck, the distance between the bony point of the chin and the prominence of the thyroid cartilage is measured .A distance of less than 7 cm suggests difficult intubation.

Investigations

There is little evidence to support the performance of 'routine' investigations, and these should only be ordered if the result would affect the patient's management. *In patients with no evidence of concurrent disease* (ASA 1, see below), preoperative investigations will depend on the extent of surgery and the age of the patient. For each age group and grade of surgery, the upper entry, shows 'tests recommended' and the lower entry 'tests to be considered' (depending on

patient characteristics). Dipstick urinalysis need only be performed in symptomatic individuals.

Additional investigations

The following is a guide to those commonly requested. Again these will also be dependent on the grade of surgery and the age of the patient.

• *Urea and electrolytes*: patients taking digoxin, diuretics, steroids, and those with diabetes, renal disease, vomiting, diarrhoea.

• *Liver function test*s: known hepatic disease, a history of a high alcohol intake (>50 units/week), metastatic disease or evidence of malnutrition.

• *Blood sugar*: diabetics, severe peripheral arterial disease or taking long–term steroids.

• *Electrocardiogram (ECG)*: hypertensive, with symptoms or signs of ischaemic heart disease, a cardiac arrhythmia or diabetics >40 years of age.

• *Chest X–ray*: symptoms or signs of cardiac or respiratory disease, or suspected or known malignancy, where thoracic surgery is planned, or in those from areas of endemic tuberculosis who have not had a chest X–ray in the last year.

• *Pulmonary function tests*: dyspnoea on mild exertion, chronic obstructive pulmonary disease COPD) or asthma. Measure peak expiratory flow rate (PEFR), forced expiratory volume in 1 s (FEV1) and FVC. Patients who are dyspnoeic or cyanosed at rest, found to have an FEV1 <60% predicted, or are to have thoracic surgery, should also have arterial blood gas analysed while breathing air.

• *Coagulation screen*: anticoagulation, a history of a bleeding diatheses or a history of liver disease or jaundice.

• *Sickle–cell screen (Sickledex)*: a family history of sickle–cell disease or where ethnicity increases the risk of sickle–cell disease. If positive, electrophoresis for definitive diagnosis.

• *Cervical spine X–ray*: rheumatoid arthritis, a history of major trauma or surgery to the neck or when difficult intubation is predicted.

Risk Assessment

There are three components that must be considered when evaluating perioperative risk: the patient's medical condition preoperatively, the extent of the surgical procedure, and the risk from the anaesthetic. In general, the major contribution to increased risk is that of the patient's health prior to the procedure and the magnitude of the surgery.However, patients presenting for surgery often have more fear about their anaesthetic than the surgery itself. Fortunately, anaesthesia–related morbidity and mortality is rare, but unfortunately, not absent. This does, however, create its own problems. The combination of infrequent but serious events has led one author to state that "Perhaps the most insidious hazard of anaesthesia is its relative safety".

Perioperatlve rlsk assessment:

The wide variety of surgical procedures and anaesthetic techniques, combined with the diversity of a patient's coexisting surgical and medical illnesses, produce a number of risk factors that contribute to overall outcome, and make generalized statements about risk difficult. Specific predictions for a single patient's outcome is virtually impossible, and the complexity of this issue has made research studies addressing outcome very difficult.

The oldest and simplest method for risk assessment is the American Society of Anesthesiology (ASA) physical status.

ASA Physical Status Classification

Class 1: A normal healthy patient.

Class 2: A patient with mild systemic disease that results in no functional limitation.

Class 3: A patient with severe systemic disease that results in functional limitation.
Class 4: A patient with severe systemic disease that is a constant threat to life.
Class 5: A moribund patient that is not expected to survive for 24 hours with or without the operation.
Class 6: A declared brain–dead patient whose organs are being removed for donor purposes.

The modification E is added to the ASA physical status classification to indicate that the case is done emergently.

As this system is simple, easy to use and requires no laboratory investigations, it has now been widely accepted as the standard means of preoperative patient classification.

Plan of Anaesthetic Technique

Classification of anestrsia

I. Local anesthesia

1. Medicamentous:
   terminal;
   infiltration;
   conduction: truncal, plexal, paravertebral;
   regional: epidural, spinal.
2. Nonmedicamentous:
   acupuncture;
   electro– and pharmaco–acupuncture;
   refrigeration.

II. General anesthesia

1. Medicamentous:
   1) mononarcosis:
      a) inhalation:
         – mask (including oro– and nasopharyngeal, with laryngomask);
         – intubation;
      b) noninhalation:

– intravenous;
– peroral;
– rectal;
– intramuscular;
– subcutaneous;
– intraosteal;
– intracavitary;

2) combained:
– two and more inhalation anesthetics;
– two and more noninhalation anesthetics;
– inhalation and noninhalation anesthetics;
–anesthetics and other neurotropic substances (analgetics, ataractics, myorelaxants et al.);
– neurotropic agents without anesthetics.

2. Nonmedicamentous:
1) electroanesthesia;
2) hypnonarcosis.

Classification of operation

Traditionally, surgery was classified as being either elective or emergency. Recognizing that this was too imprecise, the National Confidential Enquiry into Perioperative Deaths (NCEPOD) devised four categories:

• *Elective*: operation at a time to suit both patient and surgeon; for example hip replacement, varicose veins.

• *Scheduled*: an early operation but not immediately life saving; operation usually within 3 weeks; for example surgery for malignancy.

• *Urgent*: operation as soon as possible after resuscitation and within 24 h; for example intestinal obstruction, major fractures.

• *Emergency*: immediate life–saving operation, resuscitation simultaneous with surgical treatment; operation usually within 1h; for example major trauma with uncontrolled haemorrhage, extradural haematoma.

All elective and the majority of scheduled cases can be assessed as described above. However, with urgent cases this will not always be possible; as much information as possible should be obtained about any concurrent medical problems and their treatment, and allergies and previous anaesthetics. The cardiovascular and respiratory systems should be examined and an assessment made of any potential difficulty with intubation. Investigations should only be ordered if they would directly affect the conduct of anaesthesia. With true emergency cases there will be even less or no time for assessment. Where possible an attempt should be madeto establish the patient's medical history, drugs taken regularly and allergies. In the trauma patient enquire about the mechanism of injury. All emergency patients should be assumed to have a full stomach. Details may only be available from relatives and/or the ambulance crew.

A good anesthetic begins with a good plan. There is no rigid format for planning anesthesia. Rather, each plan is adapted to each case. The fundamental goal of anesthetic management is to provide safety, comfort and convenience, first for the patient and second for those caring for the patient. After a good plan, a good preparation is required for a good anesthetic.

Before every anesthetic, every anesthesiologist should go through a checklist of necessary items including, anesthesia machine, ventilator, oxygen and nitrous supply check, suction device, monitors and anesthesia cart.

Before bringing the patient to the operating room, the proper verification of patient's identity, the planned procedure and the site of the procedure should be carried out by the anesthesiologist. All the preparations should be completed before the patient enters the room including the placement of a working peripheral intravenous line.

Preoperative Preparation

Premedication

Nowadays, premedication refers to the administration of any drugs in the period before induction of anaesthesia. Consequently, a wide variety of drugs are used with a variety of aims.

The 6 As of premedication:

- Anxiolysis.
- Amnesia.
- Anti–emetic.
- Antacid.
- Anti–autonomic.
- Analgesia.

Anxiolysis

The most commonly prescribed drugs are the benzodiazepines. They produce a degree of sedation and amnesia, are well absorbed from the gastrointestinal tract and are usually given orally, 45– 90mins preoperatively. Those most commonly used include temazepam 20–30mg, diazepam 10–20mg and lorazepam 2–4mg. In patients who suffer from excessive somatic manifestations of anxiety, for example tachycardia, beta blockers may be given. A preoperative visit and explanation is often as effective as drugs at alleviating anxiety, and sedation does not always mean lack of anxiety.

Amnesia

Some patients specifically request that they not have any recall of the events leading up to anaesthesia and surgery. This may be accomplished bythe administration of lorazepam (as above) to provide anterograde amnesia.

Anti–emetic (reduction of nauseaand vomiting)

Nausea and vomiting may follow the administration of opioids, either pre– or intraoperatively. Certain types of surgery are associated with a higher incidence of postoperative nausea and vomiting (PONV), for example gynaecology. Unfortunately, none of the currently used drugs can be relied on to prevent or treat established PONV.

Drugs with anti–emetic properties

Dopamine antagonists – Metoclopramide – 10mg orally or IV 5–hydroxytryptamine antagonists – Ondansetron – 4–8mg orally or IV Antihistamines – Cyclizine – 50 mg IM or IV. Anticholinergics – Hyoscine –1 mg transdermal patch Antacid (modify pH and volume of gastric contents).

Patients are starved preoperatively to reduce the risk of regurgitation and aspiration of gastric acid at the induction of anaesthesia (see below). This may not be possible or effective in some patients:

• those who require emergency surgery;
• those who have received opiates or are in pain will show a significant delay in gastric emptying;
• those with a hiatus hernia, who are at an increased risk of regurgitation.

A variety of drug combinations are used to try and increase the pH and reduce the volume.

• *Oral sodium citrate (0.3M)*: 30mL orally immediately preinduction, to chemically neutralize residual acid.
• *Ranitidine (H2 antagonist)*: 150mg orally 12 hourly and 2 hourly preoperatively.
• *Metoclopramide*: 10mg orally preoperatively. Increases both gastric emptying and lower oesophageal sphincter tone. Often given in conjunction with ranitidine.
• *Omeprazole (proton pump inhibitor)*: 40mg 3–4 hourly preoperatively.

If a naso– or orogastric tube is in place, this can be used to aspirate gastric contents.

Anti–autonomic effects

Anticholinergic effects.

(a) Reduce salivation (antisialogogue), for example during fibreoptic intubation, surgery or instrumentation of the oral cavity or ketamine anaesthesia.

(b) Reduce the vagolytic effects on the heart, for example before the use of suxamethonium (particularly in children), during surgery on the extra ocular muscles (squint correction), or during elevation of a fractured zygoma.

Atropine and hyoscine have now largely been replaced preoperativelyby glycopyrrolate, 0.2–0.4mg intramuscularly (IM). Many anaesthetists would consider an IV dose given at induction more effective.

Antisympathomimetic effects.

Increased sympathetic activity can be seen at intubation, causing tachycardia and hypertension. This is undesirable in certain patients, for example those with ischaemic heart disease or raised intracranial pressure. These responses can be attenuated by the use of beta blockers given preoperatively (e.g. atenolol, 25–50 mg orally) or intravenously at induction (e.g. esmolol). Perioperative beta blockade may also decrease the incidence of adverse coronary events in high risk patients having major surgery. An alternative is togive a potent analgesic at induction of anaesthesia, for example fentanyl, alfentanil or remifentanil.

Analgesia

Although the oldest form of premedication, analgesic drugs are now generally reserved for patients who are in pain preoperatively. The most commonly used are morphine, pethidine and fentanyl. Morphine was widely used for its sedative effects but is relatively poor as an anxiolytic and has largely been replaced by the benzodiazepines. Opiates have a range of unwanted side–effects, including nausea, vomiting, respiratory depression and delayed gastric emptying.

Preoperative Fasting Guidelines

1. Recommendations (applies to all ages)

Ingested Material Minimum Fasting Period (hrs)

Clear liquids 2

Breast milk 4

Infant formula 6

Non–human milk 6

Light solid foods 6

2. Recommendations apply to healthy patients exclusive of parturients undergoing elective surgery; following these recommendations does not guarantee gastric emptying has occurred.

3. Clear liquids include water, sugar–water, apple juice, non–carbonated soda, pulp–free juices, clear tea,black coffee.

4. Medications can be taken with up to 150 mL of water in the hour preceding induction of anesthesia.

The preparation of equipment and instruments

The delivery of gases to theoperating theatre

Most hospitals use a piped medical gas and vacuum system (PMGV) to distribute oxygen, nitrous oxide, medical air and vacuum. The pipelines' outlets act as self–closing sockets, each specifically configured, coloured and labelled for one gas. Oxygen, nitrous oxide and air are delivered to the anaesthetic room at a pressure of 400 kilopascals (kPa) (4bar, 60 pounds per square inch (psi)). The gases (and vacuum) reach the anaesthetic machine via flexible reinforced hoses, colour coded throughout their length (oxygen white, nitrous oxide blue, vacuum yellow).

These attach to the wall outlet via a gas–specific probe (Figure 1.1) and to the anaesthetic machine via a gas–specific nut and union. Cylinders, the traditional method of supplying gases to the anaesthetic machine, are now mainly used as reserves in case of pipeline failure.

Oxygen

Piped oxygen is supplied from a liquid oxygen reserve, where it is stored under pressure (10–12 bar, 1200kPa) at approximately – 180°C in a vacuuminsulated evaporator (VIE), effectively a thermos flask. Gaseous oxygen is removed from above the liquid, or at times of increased demand, by vaporizing liquid oxygen using heat from the environment. The gas is warmed to ambient air temperature en route from the VIE to the pipeline system. A reserve bank of cylinders of compressed oxygen is kept adjacent in case of failure of the main system. A smaller cylinder is attached directly to the anaesthetic machine as an emergency reserve. The pressure in a full cylinder is 12000kPa (120 bar, 1980 psi) and this falls in direct proportion to the cylinder contents.

Nitrous oxide

Piped nitrous oxide is supplied from large cylinders, several of which are joined together to form a bank, attached to a common manifold. There are usually two banks, one running with all cylinders turned on (duty bank), and a reserve. In addition, there is a small emergency supply. Smaller cylinders are attached directly to the anaesthetic machine. At room temperature, nitrous oxide is a liquid within the cylinder, and while any liquid remains the pressure within the cylinder remains constant (440 kPa, 640 psi). When all the liquid has evaporated, the cylinder contains only gas and as it empties, the pressure falls to zero.

Figure 1.1 – Wall–mounted outlets and gas–specific probes for (left to right) oxygen, nitrous oxide, air

The anaesthetic machine

Its main functions are to allow:

• the accurate delivery of varying flows of gases to an anaesthetic system;

• an accurate concentration of an anaesthetic vapour to be added to the gas stream.

In addition to these functions, many modern anaesthetic machines contain integral monitoring equipment and ventilators.

Measurement of flow

This is achieved on most anaesthetic machines by the use of flowmeters (‘rotameters’; Figure 1.2):

• A specific, calibrated flowmeter is used for each gas.

• A needle valve controls the flow of gas through the flowmeter.

• A rotating bobbin floats in the gas stream, its upper edge indicating the rate of gas flow.

• Several flowmeters are mounted adjacent with oxygen to the left; the control for oxygen has a different knurled finish and is usually more prominent.

• Flowmeters do not regulate pressure.

Figure 1.2 – Flowmeter Figure 1.3 – Vaporizers

The addition of anaesthetic vapours

Vapour–specific devices are used to produce an accurate concentration of each inhalational anaesthetic:

• Vaporizers produce a saturated vapour from a reservoir of liquid anaesthetic (Figure 1.3).

• The final concentration of anaesthetic is controlled by varying the proportion of gas passing into the vapour chamber.

• The vaporizers are temperature compensated (hence –tec suffix, e.g. Sevotec) to account for the loss of latent heat that causes cooling and reduces vaporization of the anaesthetic.

The resultant mixture of gases and vapour is finally delivered to a common outlet on the anaesthetic machine. From this point, specialized breathing systems are used to transfer the gases and vapours to the patient.

Anaesthetic breathing systems

The mixture of anaesthetic gas and vapour travels from the anaesthetic machine to the patient via an anaesthetic circuit, or more correctly an anaesthetic breathing system. Delivery to the patient is via a facemask, laryngeal mask or tracheal tube

As several patients in succession may breathe through the same system, a low–resistance, disposable bacterial filter is placed at the patient end of the system, and changed between each patient to reduce the risk of cross–infection. Alternatively, disposable systems are used, and changed for each patient.

Components of a breathing system

All systems consist of the following:

• *A connection for fresh gas input* Usually the common gas outlet on the anaesthetic machine.

• *A reservoir bag* Usually of 2 L capacity to allow the patient's peak inspiratory demands (30– 40L/min) to be met with a lower constant flow from the anaesthetic machine. Its

excursion gives an indication of ventilation and allows manual ventilation of the patient. It also acts as a further safety device, being easily distended at low pressure if obstruction occurs.

• *An adjustable expiratory valve* To vent expired gas, helping to eliminate carbon dioxide. During spontaneous ventilation, resistance to opening is minimal so as not to impede expiration. Closing the valve allows manual ventilation by squeezing the reservoir bag.

An example of a commonly used system is shownin at Figure 1.5**.**

The circle system

The traditional breathing systems relied on the positioning of the components and the gas flow from the anaesthetic machine to eliminate carbon dioxide in expired gas, thereby preventing rebreathing and hypercapnia. Even the most efficient system is still wasteful; a gas flow of 4–6 L/min is required and the expired gas contains oxygen and anaesthetic vapour in addition to carbon dioxide. The circle system (Figure 1.4) overcomes theseinefficiencies:

• The expired gases, instead of being vented to the atmosphere, are passed through a container of soda lime (the absorber), a mixture of calcium, sodium and potassium hydroxide, to chemically remove carbon dioxide.

• Supplementary oxygen and anaesthetic vapour are added to maintain the desired concentrations, and the mixture rebreathed by the patient. Gas flows from the anaesthetic machine to achieve this can be as low as 0.5L/min. The circle system is therefore the only true ‘anaesthetic circuit’.

• The gases are warmed and humidified as they pass through the absorber (by–products of the reaction removing carbon dioxide). There are several points to note when using a circle system.

• As the inspired gas is a mixture of expired and fresh gas, the concentration of oxygen within the circle is not known

accurately. The inspired oxygen concentration must be monitored to ensure that the patient is not rendered hypoxic

• The inspired anaesthetic concentration must be monitored, particularly when a patient is being ventilated through a circle, to prevent awareness.

• When unable to absorb any more carbon dioxide, a change in the colour of the granules occurs as a result of the incorporation of an indicator. One of the commonly used preparations changes from pink to white.

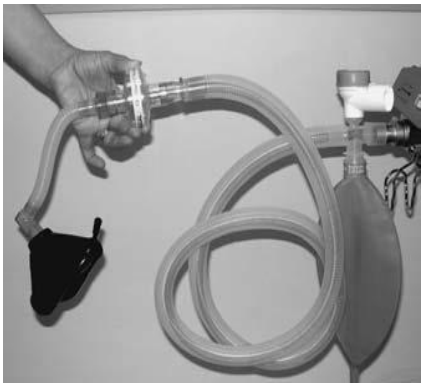


Figure 1.5 – The component parts of a breathing system

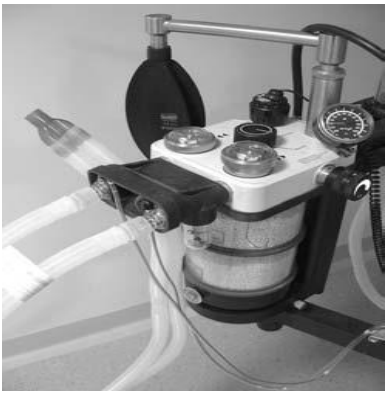


Figure 1.4 – The circle system

Checking the anaesthetic machine

It is the responsibility of each anaesthetist to check that the apparatus used will function in the manner expected at the beginning of each operating session. The main danger is that the anaesthetic machine appears to perform normally, but in fact is delivering a hypoxic mixture to the patient. In order to minimize the risk of this, the Association of Anaesthetists of Great Britain and Ireland (AAGBI) has published a *Checklist for Anaesthetic Machines*. Its main aim is to ensure that oxygen flows through the oxygen delivery system and is unaffected by the use of any additional gas or vapour. Most modern anaesthetic machines now
have built–in oxygen analysers that monitor the inspired oxygen concentration to minimize this risk.

Minimizing theatre pollution

Unless special measures are taken, the atmosphere in the operating theatre will become polluted with anaesthetic gases. The breathing systems described and mechanical ventilators vent varying volumes of excess and expired gas into the atmosphere, the patient expires anaesthetic gas during recovery and there are leaks from anaesthetic apparatus. Although no conclusive evidence exists to link prolonged exposure to low concentrations of inhalational anaesthetics with any risks, it would seem sensible to minimize the degree of pollution within the operating theatre environment. This can be achieved in a number of ways:

• reduce the flow of gases, for example by use of a circle system;
• avoid use of gases, for example by use of TIVA, regional anaesthesia;
• use of air conditioning in the theatre;
• scavenging systems.

Mechanical ventilation

A wide variety of anaesthetic ventilators are available, each of which functions in a slightly different way(Figure 1.6).

During spontaneous ventilation, gas moves into the lungs by a negative intrathoracic pressure. This process is reversed during mechanical ventilation.

A positive pressure is applied to the anaesthetic gases to overcome airway resistance and elastic recoil of the chest, and flow occurs into the lungs. This technique is usually referred to as *intermittent positive pressure ventilation* (IPPV). In both spontaneous and mechanical ventilation, expiration occurs by passive recoil of the lungs and chest wall. In order to generate a positive pressure, the ventilator requires a source of energy: gravity, gas pressure or electricity.

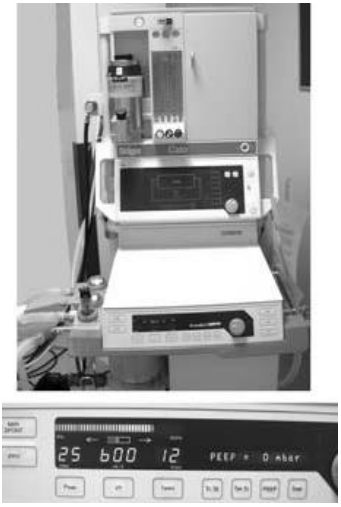


Figure 1.6 – Modern electronic ventilator

Figure 1.7 – Bag–in–bottle ventilator

The effects of positive pressure ventilation:

• There is an increase in both the physiological dead space relative to the tidal volume and ventilation/ perfusion (*V*/*Q*) mismatch, the effect of which is to impair oxygenation. An inspired oxygen concentration of around 30% is used to compensate and prevent hypoxaemia.

• The arterial partial pressure of carbon dioxide (*P*aCO2) is dependent on alveolar ventilation. Overventilation results in hypocapnia, causing a respiratory alkalosis. This 'shifts' the oxyhaemoglobin dissociation curve to the left, increasing the affinity of haemoglobin for oxygen. Hypocapnia will induce vasoconstriction in many organs, including including the brain and heart, reducing blood flow. Underventilation will lead to hypercapnia, causing a respiratory acidosis. The effects on the oxyhaemoglobin dissociation curve are the opposite of above, along with stimulation of the sympathetic nervous system causing vasodilatation, hypertension, tachycardia and arrhythmias.

• Excessive tidal volume may cause overdistension of the alveoli. In patients with pre–existing lung disease this may cause a pneumothorax, and, long term, a condition called ventilator–induced lung injury.

• The positive intrathoracic pressure reduces venous return to the heart and cardiac output.

• Both systemic and pulmonary blood flow are reduced, the latter further increasing *V*/*Q* mismatch.

Measurement and monitoring

Measurement and monitoring are closely linked but are not synonymous. A measuring instrument becomes a monitor when it is capable of deliveri a warning when the variable being measured falls outside preset limits. During anaesthesia, both the patient and the equipment being used are monitored, the complexity of which depends upon a variety of factors including:

• type of operation and operative technique;

• anaesthetic technique used;
• present and previous health of the patient;
• equipment availableng and the anaesthetist's ability to use it;

Monitoring is not without its own potential hazards: faulty equipment may endanger the patient, for example from electrocution secondary to faulty earthing; the anaesthetist may act on faulty data, instituting inappropriate treatment; or the patient may be harmed by the complications of the technique to establish invasive monitoring, for example pneumothorax following central line insertion.

Ultimately, too many monitors may distract the anaesthetist from recognizing problems occurring in other areas.

• preferences of the anaesthetist;
• any research being undertaken.

Clearly, the anaesthetist has a responsibility to check the function of all monitoring equipment before use and ensure that the alarm limits are set appropriately. There is good evidence that monitoring reduces the risks of adverse incidents and accidents. The combination of pulse oximetry, capnography and blood pressure monitoring detects the majority of serious incidents before the patient suffers serious injury. Monitoring should commence before the induction of anaesthesia and continue until the patient has recovered from the effects of anaesthesia, and the information generated should be recorded in the patient's notes. Ultimately, monitors supplement clinical observation; there is no substitute for the presence of a trained and experienced anaesthetist throughout the entire operative procedu.

Monitoring the patient

The AAGBI recommends certain monitoring devices as *essential* for the safe conduct of anaesthesia (Figure 1.8) .

These consist of:

• ECG;

• non–invasive blood pressure;
• pulse oximeter;
• capnography;
• vapour concentration analyser.

In addition, the following monitors should be *immediately available*:
• peripheral nerve stimulator;
• temperature.

Finally, additional equipment *will be required* in certain cases, to monitor, for example:
• invasive blood pressure;
• urine output;
• central venous pressure;
• pulmonary artery pressure;
• cardiac output.

SAM (really ***SAMMM')*** stands for:

S Suctlon checked and functioning.

A Airway equipment checked and prepared. (This includes checking that you have a functioning and backup laryngoscope, an appropriate sized endotracheal tube and stylet, oropharyngeal airways, as well as an oxygen source and manual resuscitation bag).

M Machine checked. (see anaesthesia machine checkout procedure and make sure you know how to check your machine. You can go to the operating room before or after scheduled procedures to explore the machine. Ask your staff anaesthetist to go through this procedure with you.

M Monitors available and functioning.

M Medications prepared and labelled. You should know where the emergency drugs are kept and location of the difficult intubation cart.

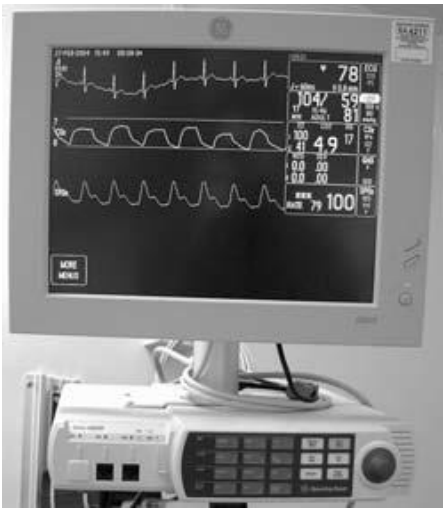


Figure 1.8 – Integrated monitoring system displaying ECG and heart rate (beats/min), non–invasive blood pressure (mmHg), capnograph and end tidal carbon dioxide (kPa), pulse oximeter waveform and saturation (%)

Examples of tascs for determination of knowledge lewel

1. Choose the right combination of components of general anesthesia:

a) Narcotic analgesia and sleep;

b) Cut consciousness, neurovegetative protection, analgesia and muscle relaxation;

c) Cut consciousness and muscle relaxation;

d) Anesthesia, muscle relaxation and neurovegetative protection.

2. Under which of the following diseases relatively contraindicated anesthesia with ketamine (Kalipsol)

a) Thyrotoxicosis;

b) Myasthenia gravis;

c) Pheochromocytoma;

d) Epilepsy.

3. General anesthesia may be:
a) Endotracheal;
b) Mask;
c) Epidural;
d) Intramuscular;

4.Narcosis may be:
a) General;
b) Local;
c) Mask;
d) Endotracheal.

5. The Mallampati test is Class 3 if visible:
a) Soft palate, fauces, uvula, anterior and posterior faucial pillars;
b) Soft palate, fauces, uvula;
c) Soft palate and the base of uvula;
d) Only hard palate.

6. If patient has severe systemic disease that is a constant threat to life, risk of anesthesia according ASA Physical Status Classification is:
a) Class 1;
b) Class 2;
c) Class 3;
d) Class 4.

7. The method of choice for emergency anesthesia for multiple injuries of the lower extremities and pelvis suspected in damage to internal organs is:
a) The mask anesthesia;
b) Epidural anesthesia;
c) Combined endotracheal anesthesia with mechanical ventilation;
d) Spinal anesthesia.

8. A patient with very labile psychics is in the state of drastic emotional exertion before the operation with a predominance of phobia. What preparation will you prefer as the agent for premedication?

a) Droperidol;
b) Morphine;
c) Omnopon;
d) Midasolam.

9. Anesthesia apparatus is necessary for:

a) All forms of anesthesia;
b) All forms of general anesthesia;
c) Inhalation anesthesia;
d) Endotracheal anesthesia.

10. Adsorber to anesthesia machine needs:

a) Oxygen for regeneration;
b) For absorbing moisture;
c) For absorption of carbon dioxide;
d) For the absorption of the anesthetic.

Correct answers:

1–b); 2–a),c),d); 3–a),b),d); 4–a),c),d); 5–c); 6–d); 7–c); 8–d); 9–c),d); 10–c).

Topic 2. ANESTESIA

The main aim: to know main 3 phases of the general anesthesia: induction, maintenance and emergence, metods of regiona anesthesia ,complications of anesthesia

The student must know:

– Pharmacocinetics drugs used for inhalation and non inhalation anaesthesia,opiates and muscle relaxants.
– The metods of maintenance airweys patency.
– Actions sequence during induction to anesthesia, induction methods.
– The technique of oral intubation.
– The control of the anaesthesia depth**:** Guedel's stages of anesthesia, bispectral index (BIS).
– Monitoring during maintenance of anesthesia.
– The blood loss and fluid management during anesthesia.
– Complications of general anesthesia.
– Actions sequence during emergence from anesthesia.
– Pharmacocinetics of localv anaesthetics.
– Regional anaesthetic techniques: brachial plexus block, epidural and spinal anaesthesia.
– Contraindications to epidural and spinal anaesthesia.
– Contraindications to epidural and spinal anaesthesia.

The student must be able:

– Select drugs for general anaesthesia.
– To use simpl devisis for maintenance airweys patency: Saphar manoeuvre, oropharingeal and nasopharingeal airweys,larindeal mask.
– Perform Sellick's manoeuvre.
– To intubate of fantom.
– To use methods of passive and active rewarming.
– To use extubation protocol.
– To select needles for epidural and spinal anaesthesia.

Short methodical instructions for practical study

At the study beginning the students pass control of knowledge–abilities initial level by means of test tasks. Students examine patients with different surgical pathology, which operative intervention coming, got to know of the clinical and laboratory results and additional findings of these patients, study hospital charts, determine the surgical risk level and choose the optimal type of anaesthesia, on possibility are in an operating–room. Also in the intensive care department they are examine postsurgical patients, estimate the adequate consciousness, breathing, hemodynamics and tonus restore, work out the tactic of patients management in an early postsurgical period, got to know of hospital and anesthesia charts, features of management in children's and elderly patients. In default of such patients the students decide situational tasks. Curatio and answers of students controlled by a teacher.

In classroom students together with a teacher discuss the results of patients examination and students mistakes. Then the students pass test control. In the end of the study the totals of work summarize, and students get the marks of the study work.

Mein material

Drugs used during general anaesthesia

Intravenous anesthetics

Mechanism of action: Usually inhibits the activity or activates the inhibitory, signaling pathways in the brain. Facilitatory actions on GABA receptors appear most important, although modulation of many other receptors and channels plays a role as well.

Uses:

- Induction of general anesthesia.
- Supplementation of general anesthetics intraoperatively.
- Maintainance of general anesthesia

- Maintainance of continous IV sedation in OR, ICU and other remote locations.
- Protection of the brain in patients' with increased intracranial pressure.

Propofol (Diprivan)

Propofol is an alkylphenol, formulated as 1–% solution dissolved in 10% intralipid (explaining the milky white color). Site of action: GABA receptors in CNS. GABA is inhibitory neurotransmitter in CNS. Onset of action is within 30 to 60sec after IV injection and duration of action (hypnosis) is between 3 to 10 minutes.

Uses:

- Induction and maintenance of general anesthesia.

Continous intravenous sedation in the ICU and Operating rooms for procedures done under monitored anesthesia care.

Pharmacology:

- Metabolized in liver by conjugation to glucuronide and sulfate.
- Formulation: 10 mg/ml
- Dose: 1–2.5 mg/kg for induction; 50–150 mcg/kg/min. for maintenance of general anesthesia
- 10 to 50 mcg/Kg/min. for sedation in the ICU and remote locations.

Effects and side effects:

- Hypnotic and amnestic properties
- No analgesic properties
- Respiratory depression and bronchodilation
- Cardiovascular depression and hypotension due to peripheral vasodilation
- Antiemetic properties
- Pain on injection (pretreatment with lidocaine will attenuates the pain)
- Myoclonus (rare)

Barbiturates

Barbiturates are derivatives of barbituric acid. Site of action: primarily GABA receptors in CNS. They enhance and mimic the activity of GABAA in CNS. They have hypnotic and amnestic properties but are not analgesics.

Thiopental (Sodium Pentothal)

Uses:

• Intravenous induction agent with rapid onset and offset time.
• Attenuates acute rise in blood pressure (e.g. head pinning during neurosurgical procedures).
• Acute perioperative seizure control.
• Provides brain protection by decreasing cerebral blood flow, cerebral oxygen consumption and intracranial pressure (ICP).

Effects and side effects:

• Thiopental reversibly reduces cerebral electrical activity to the level of EEG silence, with a significant reduction in cerebral metabolism.
• Decrease in blood pressure is mainly due to decreased peripheral vascular resistance, which can be more pronounced in hypovolemic patients or those with cardiovascular disease.
• Temporary depresses ventilation and decreases cerebral responsiveness to increased CO_2.

Contraindications:

• Hypovolemia
• Poor cardiac status

Pharmacology:

• Metabolized in liver
• Formulation: 25 mg/ml
• Induction dose: 3–5 mg/kg
• Onset: 30 seconds
• Duration of action: 5–10 minutes

Etomidate

Etomidate is an imidazole, supplied as a highly hyperosmotic solution (>4500 mOsm/l) in propylene glycol. Site of action: GABAA receptors in CNS.

Uses:

Etomidate is a drug of choice for induction of general anesthesia in hemodynamically unstable patients or in those patients with marginal cardiac reserve.

Effects and side effects:

• Cerebral effects: Decreased cerebral blood flow, decreased cerebral oxygen consumption and decreased ICP.
• In contrast to thiopental, etomidate has minimal effects on the cardiovascular system.
• Respiratory effects are minimal but it reduces the cerebral response to increased CO_2 (hypercarbia).
• Dose–dependent, reversible suppression of adrenal gland by inhibiting 11–b–hydroxylase, a key enzyme in steroid production.
• Myoclonus
• Pain on injection and thrombophlebitis
• Post–op nausea and vomiting.

Pharmacology:

• Metabolized in liver by ester hydrolysis or by N–dealkylation
• Formulation: 2 mg/ml
• Dose: 0.2 to 0.6 mg/kg
• Onset: < 1 min.
• Peak effect 1 min
• Duration of action: 3–10 min.

Ketamine

Ketamine is a phencyclidine derivative (similar to PCP).

Site of action: Inhibition of signaling at the NMDA receptor, although multiple secondary sites (opioid receptors, muscarinic acetylcholine receptors) exist.

Uses:

• Induction of anesthesia in children, by IM or IV route
• Induction of anesthesia in hypovolemic patients (etomidate is preferable).
• Supplementation of sedation during painful procedures due to its analgesic property.
• Ketamine increases cerebral blood flow and ICP; hence, it is contraindicated in patients with increased ICP.

Effects and side effects:

• Potent analgesic
• "Dissociative" anesthesia
• Adrenergic activation
• Bronchodilator and maintains CO_2 responsiveness
• Amnesia
• Nystagmus
• Induces salivation
• Dreaming and emergence reactions (less in children)

Contraindications:

• Increased ICP
• Open globe– eye injury
• Ischemic heart disease
• Psychological disease

Pharmacology:

• Formulation: Two concentrations: 10 mg/ml and 100 mg/ml. Careful!
• Dose: 0.5–2 mg/kg IV, 4–6 mg/kg IM for induction of general anesthesia.
• Onset: 1 min. IV, 5 min. IM
• Duration of action: 15 min.

Benzodiazepines

A large family of drugs, only midazolam currently used in the OR. Potent sedative and amnestic action (anterograde).
Site of action: GABAA receptors.

Midazolam (Versed)

Uses:

• Sedative and hypnotic agent mainly used for sedation perioperatively. Occasionally it is used for induction of anesthesia (cardiac surgery)
• Provides good amnesia in patients who do not tolerate any anesthetic (trauma patients).

Effects and side effects:

• Sedation (especially for therapeutic procedures)
• Amnesia (anterograde)
• Modest respiratory depression (by decreasing tidal volume, not respiratory rate)
• Modest hemodynamic and respiratory effects when used in conjunction with narcotics.

Contraindications:

• Elderly patients can exhibit paradoxical reactions (disinhibition)
• Patients with marginal respiratory function (especially patients who have received narcotics)

Pharmacology:

• Formulation: 1 mg/ml
• Dose: 0.5– 5 mg/hr for sedation
• Onset: 5 min.
• Duration of action: 45 min.

Opiates (Narcotics)

Opiates are derivatives of morphine and act at opiate receptors present at multiple sites. Potent analgesics, some have mild sedative properties.

Uses:

• Supplementation of general anesthesia
• Pain relief (analgesia perioperatively)
• Induction of general anesthesia in cardiac patients (because of cardiac stability).
• Premedication (blunting of hemodynamic response to intubation). Narcotics should not be given long before induction to *pain free* patients because of dysphoric reactions.

Side effects:

• Respiratory depression (they decrease respiratory rate not tidal volume)
• Nausea and vomiting
• Muscle rigidity (especially if given rapidly in large doses such as during induction in cardiac anesthesia).
• Urinary retention, pruritus, dysphoria.

Contraindications:

· Increased ICP, neurological disease and respiratory failure.

Morphine

Use:

Post–operative pain relief; because of its relatively long duration of action, long term ICU pain therapy.

Effects and side effects:

• Strong analgesic
• Histamine release (not seen with most of the other compounds), leading to:
Decrease in blood pressure (hypotension)

Contraindications:

· Morphine allergy

Pharmacology:
• Formulation: 10 mg/ml
• Dose: 0.01–0.1 mg/kg
• Onset: 10 min.
• Duration of action: 2 h

Synthetic narcotic analgesics: fentanyl, alfentanil, sufentanil and Remifentanil

Fentanyl is the "standard" narcotic for perioperative use.

Alfentanil is used primarily for relief of brief, intense pain (e.g. head pinning), or for supplementation of anesthesia close to the end of a case. It is approximately 5 times less potent than fentanyl.

Sufentanil is approximately 10 times as potent as fentanyl, which is *not* reflected in the formulation!

Remifentanil is metabolized by plasma esterase, and therefore, short–acting. It is administered by continuous infusion.

Inhaled Anesthetics

Inhalation anesthetics are substances that are brought into the body via the lungs and are distributed with the blood into the different tissues. The main target of inhalation anesthetics (or so–called volatile anesthetics) is the brain.

Inhalation anesthetics act either by amplifying inhibitory function or decreasing excitatory transmission at the nerve endings in the brain. The role of inhalation agents in general anesthesia is changing. Volatile anesthetics are seldom used alone in our days. A combination of inhalation anesthetics and intravenous drugs is called balanced anesthesia. Currently used inhalation anesthetics include enflurane, halothane, isoflurane, sevoflurane, desflurane, and nitrous oxide. Older volatile anesthetics include ether, chloroform, and methoxyflurane.

Ideally, inhalation agents should provide a quick induction and emergence from anesthesia, good analgesia, muscle relaxation, quick changes and easy maintenance of anesthesia, and no side effects. Unfortunately, the real world of medicine doesn't provide us with such an ideal agent. Relatively long and unpleasant induction times can be overcome by using an intravenous anesthetic. Neuromuscular blockers will provide muscle paralysis and adding opioids can enhance analgesia. This technique, the so–called balanced anesthesia, allows the anesthesiologist to take advantage of different beneficial effects of several drug classes

Inhalational anesthetics are commonly used in anesthesia practice worldwide due to their ease of administration and rapid excretion. With the use of inhalation agents the depth of anesthesia can be altered rapidly and measured readily. **Minimum Alveolar Concentration MAC.**

Is the concentration at which 50% of the patients do not move in response to skin incision at one atmospheric pressure. The value of MAC for each inhalational agent is different.

Mechanism of Action of Inhaled Anesthetics

Inhaled anesthetics act in different ways at the level of the central nervous system. They may disrupt normal synaptic transmission by interfering with the release of neurotransmitters from presynaptic nerve terminal (enhance or depress excitatory or inhibitory transmission), by altering the re–uptake of neurotransmitters, by changing the binding of neurotransmitters to the post–synaptic receptor sites, or by influencing the ionic conductance change that follows activation of the post–synaptic receptor by neurotransmitters. Both, pre– and postsynaptic effects have been found.

Direct interaction with the neuronal plasma membrane is very likely, but indirect action via production of a second messenger also remains possible. The high correlation between lipid solubility and anesthetic potency suggests that

inhalation anesthetics have a hydrophobic site of action. Inhalation agents may bind to both membrane lipids and proteins. It is at this time not clear which of the different theories are most likely to be the main mechanism of action of inhalation anesthetics.

The Meyer–Overton theory describes the correlation between lipid solubility of inhaled anesthetics and MAC and suggests that anesthesia occurs when a sufficient number of inhalation anesthetic molecules dissolve in the lipid cell membrane. The Meyer–Overton rule postulates that the number of molecules dissolved in the lipid cell membrane and not the type of inhalation agent causes anesthesia. Combinations of different inhaled anesthetics may have additive effects at the level of the cell membrane.

However, the Meyer–Overton theory does not describe why anesthesia occurs. Mullins expanded the Meyer–Overton rule by adding the so–called Critical Volume Hypothesis. He stated that the absorption of anesthetic molecules could expand the volume of a hydrophobic region within the cell membrane and subsequently distort channels necessary for sodium ion flux and the development of action potentials necessary for synaptic transmission. The fact that anesthesia occurs with significant increase in volume of hydrophobic solvents and is reversible by compressing the volume of the expanded hydrophobic region of the cell membrane supports Mullins Critical Volume Hypothesis.

The protein receptor hypothesis postulates that protein receptors in the central nervous system are responsible for the mechanism of action of inhaled anesthetics. This theory is supported by the steep dose response curve for inhaled anesthetics. However, it remains unclear if inhaled agents disrupt ion flow through membrane channels by an indirect action on the lipid membrane, via a second messenger, or by direct and specific binding to channel proteins.

Another theory describes the activation of Gamma–Aminobutyric acid (GABA) receptors by the inhalation

anesthetics. Volatile agents may activate GABA channels and hyperpolarize cell membranes. In addition, they may inhibit certain calcium channels and therefore prevent release of neurotransmitters and inhibit glutamate channels. Volatile anesthetics share therefore common cellular actions with other sedative, hypnotic or analgesic drugs.

Each of the mentioned theories describes a unitary theory of narcosis. They all concentrate more or less on an unique site of action for inhaled anesthetics. The true mechanism of action of volatile anesthetics may be a combination of two or more such theories described as multisite action hypothesis

Effects of inhalational anesthetics

Respiratory System

Airway irritation except Sevoflurane and halothane

Dose related suppression of spontanous ventilation with decreased tidal volume

Progressive decrease in ventilatory response to CO_2 with increasing depth of anesthesia

Bronchodilation and collapse of alveoli in dependent areas of the lungs

Circulatory system

Myocardial depression, Hypotension and decreased sympathoadrenal response with increasing depth of anesthesia

Central Nervous system

Dose dependent increase in cerebral blood flow with cerebral vasodilation

Impaired autoregulation of cerebral blood flow

Dose dependent decrease in cerebral cortex activity (slow waves with greater amplitude) and electrical silence with deeper anesthesia.

Renal system

Dose dependent decrease in renal blood flow and GFR

Gastrointestinal Tract

Nausea and vomiting

Skeletal Muscles

Potent inhalational agents produce modest skeletal muscle relaxation by central depression and enhancement of muscle relaxation produced by non–depolarizing muscle relaxants.

Nitrous oxide (nitrous oxide):

This is an inorganic nonflammable gas that supports combustion. It has a vapor pressure of 39,000 mm Mercury at 20 degree Celsius and boils at minus 88 degree Celsius. The blood/gas coefficient is 0.47 and the MAC in 100 percent oxygen is 104. This means that one MAC nitrous oxide can only be reached in a hyperbaric chamber.

Nitrous oxide is stored in blue cylinders (This is the case in the USA. In some parts of Europe, blue is the color for oxygen and green the color for nitrous oxide). At room temperature, nitrous oxide in the cylinder is in equilibrium between liquid and gaseous form. The pressure within the cylinder is constant as long some of the gas is in liquid form. Therefore, there is only little nitrous oxide left when the pressure in the cylinder decreases. Nitrous oxide is a weak anesthetic. It is used to supplement other inhalation agents. Its low solubility results in rapid induction or awakening. Administration of high concentrations of nitrous oxide will facilitate the increase in alveolar concentration of a simultaneously administered second gas. This is called the second gas effect. Nitrous oxide is resistant to degradation by soda lime and can therefore used in low flow or closed systems anesthesia. Unlike other inhalation anesthetics, nitrous oxide does not inhibit the hypoxic pulmonary vasoconstriction response in the lungs. It might produce an increase in pulmonary vascular resistance, especially in patients with pre–existing pulmonary hypertension. It is therefore contraindicated in patients with intra–cardiac right–to–left shunt. Nitrous oxide is sympathomimetic and increases systemic vascular resistance. It does not cause a decrease in blood pressure. Unlike order inhalation anesthetics, nitrous oxide does not produce skeletal muscle relaxation. It does not

have any significant effect on uterine contractility. It is a weak trigger for malignant hyperthermia.

Nitrous oxide diffuses into air containing cavities 34 times faster than nitrogen can leave that space. This can cause dangerous accumulation of volume and increase in pressure in closed spaces such as bowel, middle year, pneumothorax, pneumocranium, pneumo–peritoneum, or cuffs of endotracheal tubes. In patients with ileus, the volume of air in the bowel can double within 4 hours of nitrous oxide administration. The volume of air within a pneumothorax can double within 10 minutes if 70 percent nitrous oxide is administered. This can lead to a life–threatening tension pneumothorax. Diffusion of nitrous oxide into air bubbles will increase their size. It has therefore to be stopped immediately when air embolism is suspected.

Halothane (Fluothane):

This volatile anesthetic is a nonflammable halogenated alkene. It has a vapor pressure of 244 mm Mercury at 20 degree Celsius and boils at 50.2 degree Celsius. The blood/gas coefficient is 2.3 and the MAC in 100 percent oxygen is 0.74 and in 70 percent nitrous oxide 0.29.

Halothane is susceptible to decomposition. For this reason, it is stored in amber–colored bottles and thymol is added as preservative. It is known to sensitize the myocardium to the action of epinephrine and norepinephrine and to have the potential for serious cardiac dysrhythmias. Halothane lowers airway resistance and might be used in the treatment of asthma if conventional therapy fails. It is not recommended for obstetric anesthesia except when uterine relaxation is required. It crosses the placental barrier and can cause fetal and neonatal depression resulting in hypotension, hypoxemia, and acidosis. Halothane does not cause coronary artery vasodilatation and therefore does not lead to coronary artery steal syndrome. Decrease in blood pressure is due to negative inotropic effects of halothane. Systemic vascular

resistance does not change significantly. Increase in cerebral blood flow due to cerebral vasodilatation produced by halothane is greater than the one produced by isoflurane or enflurane. Halothane is able to trigger malignant hyperthermia, a potential lethal complication of anesthesia.

Fulminant hepatic necrosis and/or jaundice (halothane hepatitis) are other severe complications of halothane anesthesia. Hepatic necrosis occurs in one of 6,000 to 35,000 cases and is often fatal. Anti–trifluoroacetyl protein antibodies probably cause halothane hepatitis. These antibodies may mediate massive hepatic necrosis after re–exposure of the patient with halothane.

Halothane has excellent hypnotic but no analgesic properties. Induction of anesthesia can be achieved by using 1 to 3 percent halothane in air or in oxygen, or by using 0.8 percent halothane in 65 percent nitrous oxide. Induction occurs relatively quickly. This is one of the reasons why halothane was the drug of choice for mask induction of pediatric patients but its popularity changed in the recent years with the availability of sevoflurane. Maintenance of anesthesia can be achieved with 0.5 to 1.5 percent halothane. Emergence might be delayed in obese patients due to storage of the inhalation agent in fatty tissues

Isoflurane (Forane):

This volatile anesthetic is a nonflammable halogenated methyl ethyl ether. It has a vapor pressure of 239 mm Mercury at 20 degree Celsius and boils at 48.5 degree Celsius. The blood/gas coefficient is 1.4 and the MAC in 100 percent oxygen is 1.15 and in 70 percent nitrous oxide 0.50.

Isoflurane is resistant to degradation by the absorber and can therefore be used during low flow or closed system anesthesia. Isoflurane produces a dose–dependent reduction in blood pressure due to peripheral vasodilatation. It does not sensitize the myocardium for arrhythmias. It can cause coronary artery vasodilatation that might lead to coronary artery steal syndrome. During such an event blood is diverted

away from critically perfused areas because of vasodilatation in healthy parts of the heart. This might lead to myocardial ischemia or infarction. However, most clinical studies failed to prove higher incident of myocardial ischemia due to isoflurane. Isoflurane should be avoided in patients with aortic valve stenosis since they poorly tolerate a decrease in systemic vascular resistance. Like halothane, it can trigger malignant hyperthermia.

Induction of anesthesia can be achieved by using 3 to 4 percent isoflurane in air or in oxygen, or by using 1.5 to 3 percent isoflurane in 65 percent nitrous oxide. Induction with isoflurane alone can lead to coughing and apneic periods. Therefore, it should be combined with intravenous anesthetics. Maintenance of anesthesia can be achieved with 1 to 2.5 percent isoflurane. Emergence from anesthesia with isoflurane is faster than with halothane or enflurane.

Enflurane (Ethrane):

This volatile anesthetic is a nonflammable fluorinated ethyl methyl ether. It as a vapor pressure of 172 mm Mercury at 20 degree Celsius and boils at 56.5 degree Celsius. The blood/gas coefficient is 1.8 and the MAC in 100 percent oxygen is 1.68 and in 70 percent nitrous oxide 0.57.

Enflurane is resistant to degradation by soda lime and can be therefore used during low flow or closed system anesthesia. Its biotransformation releases fluoride ions but their concentration does not reach nephrotoxic levels. Enflurane produces a dose–dependent reduction in arterial blood pressure as consequence of negative inotropy. Like isoflurane, enflurane does not sensitize the heart for arrhythmias. In addition, it does not cause a coronary artery steal syndrome. Enflurane has been found to increase intracranial pressure and, especially in combination with hyperventilation, to increase the risk of seizure activity. It is therefore contraindicated in patients with seizure disorders. As halothane and isoflurane, it can trigger malignant

hyperthermia. Enflurane enhances the action of paralyzing agents more than other inhalation anesthetics.

Induction of anesthesia can be achieved by using 3 to 4 percent enflurane in air or in oxygen, or by using 1.5 to 3 percent enflurane in 65 percent nitrous oxide. Maintenance of anesthesia can be achieved with 1 to 3 percent enflurane. Emergence from anesthesia with enflurane is a little slower than with isoflurane.

Desflurane (Suprane):

This volatile anesthetic is a nonflammable fluorinated methyl ethyl ether. It has a vapor pressure of 673 mm Mercury at 20 degree Celsius and boils at 23.5 degree Celsius. The blood/gas coefficient is 0.42 and the MAC in 100 percent oxygen is 6.0 and in 60 percent nitrous oxide 2.8.

Unlike other inhalation anesthetics, desflurane cannot be delivered by standard vaporizers. It requires the use of electrically heated vaporizers. Desflurane is very resistant to degradation by soda lime and can therefore be used during low flow or closed system anesthesia. Desflurane produces a dose–dependent reduction in arterial blood pressure due to peripheral vasodilatation. It might as well cause an increase in heart rate. It should therefore not be used in patients with aortic valve stenosis. It does not sensitize the heart to arrhythmias or cause coronary artery steal syndrome. Like other inhalation anesthetics, it can trigger malignant hyperthermia.

Induction of anesthesia can be achieved by using 6 to 10 percent desflurane in air or in oxygen, or by using 5 to 8 percent desflurane in 65 percent nitrous oxide. Desflurane may cause coughing and excitation during induction and should therefore rather not be used without intravenous anesthetics. Maintenance of anesthesia can be achieved with 5 to 7 percent desflurane. The low tissue solubility of desflurane results in rapid elimination and awakening.

Sevoflurane (Ultane):

This volatile anesthetic is a nonflammable fluorinated isopropyl ether. It has a vapor pressure of 162 mm Mercury at 20 degree Celsius and boils at 58.5 degree Celsius. The blood/gas coefficient is 0.59 and the MAC in 100 percent oxygen is 1.71 and in 63.5 percent nitrous oxide 0.66.

Sevoflurane undergoes temperature dependent degradation by baralyme and soda lime. Therefore, it cannot be used in low flow or closed systems anesthesia. Sevoflurane reacts with CO2 absorbents to form a special haloalkene, the so–called Compound A. Compound A is metabolized to nephrotoxins and can lead to kidney damage. The minimum fresh gas flow has been recommended to be at least two liters per minute. Sevoflurane produces a dose–dependent decrease in arterial blood pressure due to peripheral vasodilatation. It should therefore not be used in patients with aortic valve stenosis. It does not sensitize the heart to arrhythmias or cause coronary artery steal syndrome. Unlike desflurane, sevoflurane does not irritate the airway. Due to its low solubility in blood it can be used for rapid induction of anesthesia without intravenous anesthetics. This is one of the reasons why it is currently replacing halothane for mask induction in pediatric patients. Like all other inhalation anesthetics, sevoflurane can trigger malignant hyperthermia in susceptible patients.

Induction of anesthesia can be achieved by using 1.5 to 3 percent sevoflurane in air or in oxygen, or by using 0.7 to 2 percent sevoflurane in 65 percent nitrous oxide. Sevoflurane does not cause coughing and excitation during induction and can be used without intravenous anesthetics. Maintenance of anesthesia can be achieved with 0.4 to 2 percent sevoflurane. The low tissue solubility of sevoflurane results in rapid elimination and awakening.

4. Muscle relaxants

Muscle relaxants block the nicotinic acetylcholine receptors at the muscle endplate, thereby inhibiting neuromuscular transmission and inducing muscle flaccidity.

Inactivation of the receptor can be attained in two ways: by *depolarizing* the receptor continuously, which leads to a complex form of desensitization (depolarizing muscle relaxants); or by *competitively antagonizing* the receptor (non–depolarizing muscle relaxants).

The degree of relaxation can be assessed using a twitch monitor. The two standard modes of testing are the *train–of–four* (four pulses at 0.5 sec intervals) and *tetanus* (usually at 50 Hz for 5 seconds).

Depolarizing muscle relaxants

The only depolarizer in clinical use is succinylcholine. It is the muscle relaxant with the briefest duration of action, because of its rapid metabolism by butyrylcholinesterase ("pseudocholinesterase") in plasma. The rapid onset (30 to 60 seconds) is dose dependent and minimizes the time for rapid sequence intubation. The rapid degradation allows patients to manage their own airway quickly again after an unsuccessful endotracheal intubation.

Succinylcholine administration results in a parallel decrease in height of all twitches on the train–of–four (no "fade"). After administration of high doses and or repeated administration of succinylcholine, patient can develop phase II block, a pattern similar to that seen with non–depolarizing drugs.

Side effects:

- The initial depolarization of muscles causes *fasciculations due to* . These are associated with muscle pain postoperatively, and can largely be prevented by administration of a small dose of non–depolarizing relaxant (curare 3 mg) one minute prior to succinylcholine. In that case the dose of succinylcholine should be increased by 50% to compensate for the antagonism of succinylcholine by the non–depolarizing drug. Intravenous lidocaine, benzodiazepines, Ca^{+2} channel blockers, etc. also appear to prevent myalgia.

• The muscle depolarization also results in release of K+ from myocyte. In patients with upregulated nicotinic receptors (burns, major trauma, paralyzed limbs, head trauma, neuromuscular disease), the use of succinylcholine can lead to cardiac arrest.

• Succinylcholine can induce malignant hyperthermia in susceptible patients.

• Prolonged paralysis occurs in case of butyrylcholinesterase abnormalities (as succinylcholine is not metabolized).

• Increases in intra–ocular pressure.

• Increases in intracranial pressure (modest).

• Bradycardia, particularly in children, after administration of a second dose (pre–treat with atropine).

Contraindications:

• The difficult airway.

• Documented or suspected susceptibility to malignant hyperthermia.

• Up–regulated nicotinic receptors (burn patients).

• Children <5 yr. (Controversial).

• Patients with open eye injury (controversial)

• Patients with increased ICP (a relative contraindication).

• Hyperkalemia and renal disease

• Patients with history of atrial or ventricular arrhythmias.

Pharmacology:

• Formulation: 20 mg/ml,

• Dose: 1 to 1.5 mg/kg; in children 2 mg/kg,

• Onset: 30 sec,

• Duration of action: 3 to 15 min.

Non–depolarizing muscle relaxants

There are two main types of non–depolarizing muscle relaxants.

1. Steroidal compounds.

Short acting (Rapacurium and mivacurium

Intermediate acting (cis–atracurium, Vecuronium, Rocuronium).

Long Acting (Pancuronium, Pipecuronium, Doxacurium).

They competitively bind to the nicotinic receptors at the neuromuscular junction, preventing depolarization.

Side effects

• Histamine release.

• Vagolytic effect (muscarinic inhibition) (steroidal compounds).

• Sympathomimetic effect (autonomic ganglia stimulation).

Contraindications:

Few. Non–depolarizing muscle relaxants are very safe drugs, as long as the airway is adequately protected.

Caution using pancuronium with Demerol.

Reversal

Inhibitors of plasma cholinesterases, induce increased availability of acetylcholine at the neuromuscular junction, which competitively reverses the neuromuscular blockade.

Side effects:

• Bradycardia from cardiac muscarinic stimulation,

• Bronchoconstriction.

These side effects can be (partially) attenuated by administration of a muscarinic antagonist, which is usually given at the same time as the reversal drug. Two cholinesterase inhibitors are used clinically: neostigmine and edrophonium.

Neostigmine

Neostigmine is slower in onset than edrophonium, but it forms covalent (strong) bond with plasma cholinesterase, thus it can reverse a deeper neuromuscular block. The muscarinic antagonist glycopyrrolate (7–15 mcg/kg), which

has a longer duration of action and a longer time to onset than atropine, is often used with neostigmine to minimize cardiovascular changes and other unwanted nicotinic effects.

Pharmacology:

• Formulation: 1 mg/ml,

• Dose: 0.04–0.07 mg/kg,

• Onset: full reversal is attained in approximately 10 – 15 min.

• Duration of action: 1.5 h

Edrophonium Edrophonium, when given intravenously, has rapid onset of action than neostigmine. At equivalent doses neostigmine and edrophonium has similar duration of action. Atropine (7–10 mcg/Kg) is often used in combination with edrophonium to block muscarinic effects. The degree of block that edrophonium is able to antagonize, however, is much less profound, as it forms ionic (weaker) bond with actelycholinesterase.

Pharmacology:

• Formulation: 10 mg/kg,

• Dose: 0.5–1 mg/kg,

• Onset: full reversal is attained in approximately 5 min.

• Duration of action: 1 to 1.5 hr. (a long–acting relaxants may outlast edrophonium).

Managing the airway

Maintenance of a patent airway is an essential prerequisite for the safe and successful conduct of anaesthesia. However, it is a skill that should be acquired by all doctors, as during resuscitation patients often have an obstructed airway either as the cause or result of their loss of consciousness. The descriptions of airway management techniques that follow are intended to *supplement* practice either on a manikin or, preferably, on an anaesthetized patient under the direction of a skilled anaesthetist.

Basic techniques

Anaesthesia frequently results in loss of the airway, and this is most easily restored by a combination of the head tilt and a jaw thrust (see page 100). When holding a facemask in position with the index finger and thumb, the jaw thrust is achieved by lifting the angle of the mandible with the remaining fingers of one or both hands. The overall effect desired is that the patient's mandible is 'lifted' into the mask rather than that the mask is being pushed into the face (Fig. 2.1).

Facemasks

• A commonly used type in adults is the BOC anatomical facemask (Figure 2.1), designed to fit the contours of the face with the minimum of pressure.

• Leakage of anaesthetic gases is minimized by an air–filled cuff around the edge.

• Masks are made in a variety of sizes, and the smallest one that provides a good seal should be used.

• Some masks have a transparent body allowing identification of vomit, making them popular for resuscitation.

• All masks must be disinfected between Alternatively single use masks are available.

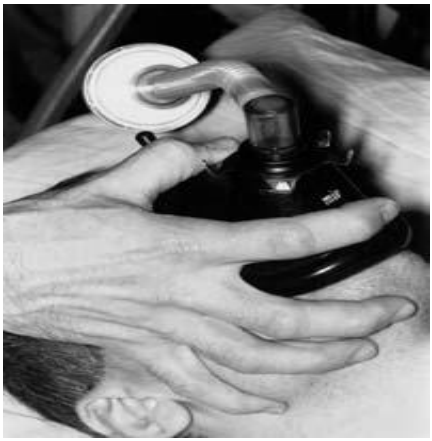


Figure 2.1 – Mask being held on a patient's face

The oropharyngeal airway

The oropharyngeal (Guedel) airway, and to a lesser extent the nasopharyngeal airway, are used in conjunction with the techniques described above to help maintain the airway after the induction of anaesthesia(Figure 2.2).

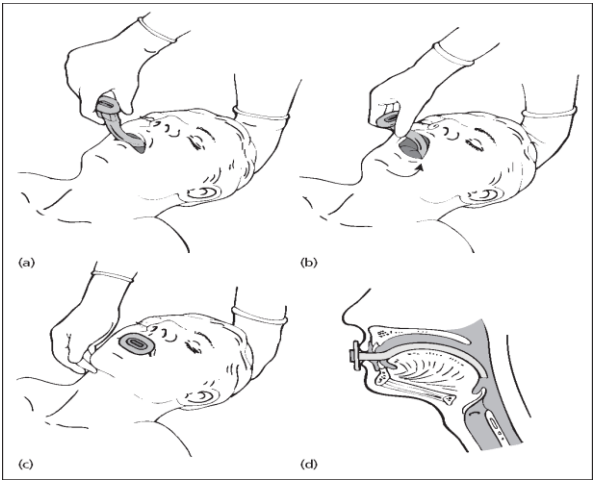


Figure 2.2 – The sequence of inserting an oropharyngeal airway

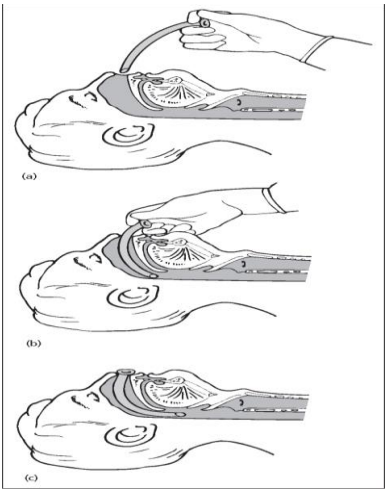


Figure 2.3 – Insertion of a nasopharyngeal airway

Nasopharyngeal airway

• Round, malleable plastic tubes, bevelled at the pharyngeal end and flanged at the nasal end.

• Sized on their internal diameter in millimetres, length increasing with diameter. The common sizes in adults are 6–8 mm, for small to large adults, respectively.

• A guide to the correct size is made by comparing the diameter to the external nares.

• Prior to insertion, the patency of the nostril (usually the right) should be checked and the airway lubricated.

• The airway is inserted along the floor of the nose, with the bevel facing medially to avoid catching the turbinates (Fig. 2.3).

• A safety pin may be inserted through the flange to prevent inhalation of the airway.

• If obstruction is encountered, force should not be used as severe bleeding may be provoked. Instead, the other nostril can be tried (Figure 2.3).

The laryngeal mask airway (LMA)

Originally designed for use in spontaneously breathing patients, it consists of a 'mask' that sits over the laryngeal opening, attached to which is a tube that protrudes from the mouth and connects directly to the anaesthetic breathing system. On the perimeter of the mask is an inflatable cuff that creates a seal and helps to stabilize it (Fig. 2.4a). The LMA is produced in a variety of sizes suitable for all patients, from neonates to adults, with sizes 3, 4 and 5 being the most commonly used in female and male adults. Patients can be ventilated via the LMA provided that high inflation pressures are avoided, otherwise leakage occurs past the cuff. This reduces ventilation and may cause gastric inflation.

The LMA is reusable, provided that it issterilized between each patient. There are now four additional types of LMAs available:

• A version with a reinforced tube to prevent kinking (Fig. 2.4b).

• The Proseal LMA (Fig. 2.4c): this has an additional posterior cuff to improve the seal around the larynx and reduce leak when the patient is ventilated. It also has a secondary tube to allow drainage of gastric contents.

• The intubating LMA (Fig. 2.4d): as the name suggests this device is used as a conduit to perform tracheal intubation without the need for laryngoscopy.

• A disposable version of the original for single use, for example in infected cases.

The use of the laryngeal mask overcomes some of the problems of the previous techniques:

• It is not affected by the shape of the patient's face or the absence of teeth.

• The anaesthetist is not required to hold it in position, avoiding fatigue and allowing any other problems to be dealt with.

• It *significantly reduces* the risk of aspiration of regurgitated gastric contents, but does not eliminate it completely.

Its use is *relatively contraindicated* where there is an increased risk of regurgitation, for example in emergency cases, pregnancy and patients with a hiatus hernia. The LMA has proved to be a valuable aid in those patients who are difficult to intubate, as it can usually be inserted to facilitate oxygenation while additional help or equipment is obtained (see below).

Technique for insertion of the standard LMA

The patient's refle xes must be suppressed to a level similar to that required for the insertion of an oropharyngeal airway to prevent coughing or laryngospasm.

• The cuff is deflated (Fig. 2.4a) and the mask lightly lubricated.

• A head tilt is performed, the patient's mouth opened fully and the tip of the mask inserted along the hard palate with the open side facing but not touching the tongue

• The mask is further inserted, using the index finger to provide support for the tube (Fig. 2.4c). Eventually, resistance will be felt at the point where the tip of the mask lies at the upper oesophageal sphincter (Fig. 2.4d).

• The cuff is now fully inflated using an air–filled syringe attached to the valve at the end of the pilot tube (Figure 2.4).

• The laryngeal mask is secured either by a length of bandage or adhesive strapping attached to the protruding tube.

• A 'bite block' may be inserted to reduce the risk of damage to the LMA

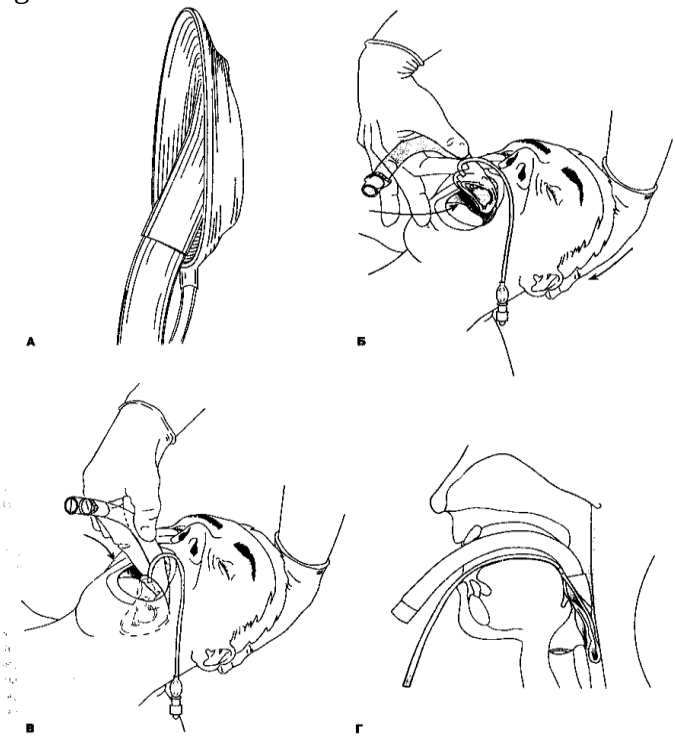


Figure 2.4 – Technique for insertion of the standard LMA

Problems with airways

Snoring, indrawing of the supraclavicular, suprasternal and intercostal spaces, use of the accessory muscles or paradoxical respiratory move– ment (see–saw respiration) suggest that the above methods are failing to maintain a patent airway.

Other problems with these techniques include:

• inability to maintain a good seal between the patient's face and the mask, particularly in those without teeth;

• fatigue, when holding the mask for prolonged periods;

• the risk of aspiration, due to the loss of upper airway reflexes;

• the anaesthetist not being free to deal with any other problems that may arise.

The laryngeal mask airway or tracheal intubation may be used to overcome these problems.

overy.

The technique of tracheal intubation.

The technique of tracheal intubation involves five steps.

I. Positioning the patient.

11. Opening the patients mouth.

111. Performing laryngoscopy.

IV. Insertion of the ETT

V. Confirmation of correct placement and securing the ETT tube.

Equipment for tracheal intubation

The equipment used will be determined by the circumstances and by the preferences of the individual anaesthetist. The following is a list of the basic needs for *adult oral* intubation.

• *Laryngoscope*: with a curved (Macintosh) blade and functioning light(Figure 2.5).

• *Tracheal tubes (cuffed)*: in a variety of sizes. The internal diameter is expressed in millimetres and the length in centimetres. They may be lightly lubricated.

• For males: 8.0–9.0 mm internal diameter, 22–24 cm length.

• For females: 7.5–8.5 mm internal diameter, 20–22 cm length.

• *Syringe*: to inflate the cuff once the tube is in place.

• *Catheter mount*: or 'elbow' to connect the tube to the anaesthetic system or ventilator tubing.

• *Suction*: switched on and immediately to hand in case the patient vomits or regurgitates.

• *Stethoscope*: to check correct placement of the tube by listening for breath sounds during ventilation.

• *Extras*: a semi–rigid introducer to help mould the tube to a particular shape; Magill's forceps, designed to reach into the pharynx to remove debris or direct the tip of a tube; bandage or tape to secure the tube.

Figure 2.5 – An assortment of laryngoscope blades

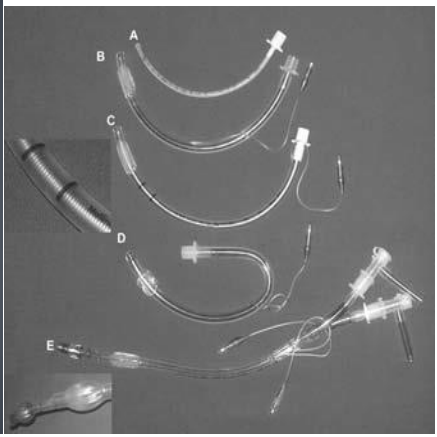


Figure 2.6 – Tracheal tubes

Tracheal tubes

Mostly manufactured from plastic (PVC), and for single use to eliminate cross–infection (Fig. 2.6B). They are

available in 0.5mm diameter intervals, and long enough to be used orally or nasally. A standard 15mm connector is provided to allow connection to the breathing system. In adult anaesthesia, a tracheal tube with an inflatable cuff is used to prevent leakage of anaesthetic gases back past the tube when positive pressure ventilation is used. This also helps prevent aspiration of any foreign material into the lungs (Figure 2.6).

The cuff is inflated by injecting air via a pilot tube, at the distal end of which is a one–way valve to prevent deflation and a small 'balloon' to indicate when the cuff is inflated. A wide variety of specialized tubes have been developed, examples of which are shown in.

• *Reinforced tubes* are used to prevent kinking and subsequent obstruction as a result of the positioning of the patient's head (Fig. 2.6C).

• *Preformed tubes* are used during surgery on the head and neck, and are designed to take the connections away from the surgical field (Fig. 2.6D).

• *Double lumen tubes* are effectively two tubes welded together side–by–side, with one tube extending distally beyond the other. They are used during thoracic surgery, and allow one lung to be deflated whilst ventilation is maintained via the bronchial portion in the opposite lung .

• *Uncuffed tubes* are used in children up to approximately 10 years of age as the narrowing in the subglottic region provides a natural seal.

The technique of oral intubation

Preoxygenation

All patients who are to be intubated are asked to breathe 100% oxygen via a close–fitting facemask for 2–3 mins ('preoxygenation'). This provides a reservoir of oxygen in the patient's lungs, reducing the risk of hypoxia if difficulty is encountered with intubation. Once this has been accomplished, the appropriate drugs will be administered to render the patient unconscious and abolish laryngeal reflexes.

Positioning

The patient's head is placed on a small pillow with the neck flexed and the head extended at the atlanto–occipital joint, the 'sniffing the morning air' position. The patient's mouth is fully opened using the index finger and thumb of the *right* hand in a scissor action (Figure 2.7).

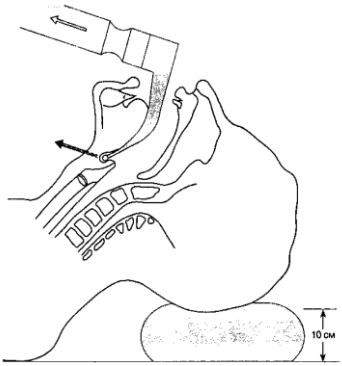


Figure 2.7 – The sniffing position

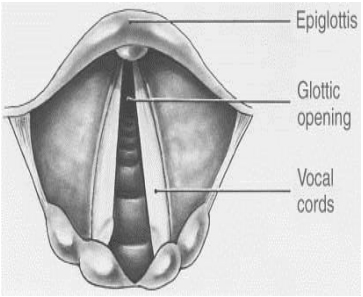


Figure 2.8 – Schematic view of the laryngoscopy and intubation with a acintosh blade

Laryngoscopy

The laryngoscope is held in the *left* hand and the blade introduced into the mouth along the righthand side of the tongue, displacing it to the left. The blade is advanced until the tip lies in the gap between the base of the tongue and the epiglottis, the vallecula. Force is then applied *in the direction in which the handle of the laryngoscope is pointing.* The effort comes from the upper arm not the wrist, to lift the tongue and epiglottis to expose the larynx, seen as a triangular opening with the apex anteriorly and the whitish coloured true cords laterally (Figure 2.8).

The tracheal tube is introduced into the right side of the mouth, advanced and *seen to pass through the cords* until the cuff lies just below the cords. The tube is then held firmly and the laryngoscope is carefully removed, and the cuff is inflated sufficiently to prevent any leak during ventilation.

Finally the position of the tube is confirmed and secured in place. For nasotracheal intubation a well–lubricated tube is introduced, usually via the right nostril along the floor of the nose with the bevel pointing medially to avoid damage to the turbinates. It is advanced into the oropharynx, where it is usually visualized using a laryngoscope in the manner described above. It can then either be advanced directly into the larynx by pushing on the proximal end, or the tip picked up with Magill's forceps (which are designed not to impair the view of the larynx) and directed into the larynx. The procedure then continues as for oral intubation.

Confirming the position of the tracheal tube

This can be achieved using a number of techniques:

• *Measuring the carbon dioxide in expired gas (capnography)*: less than 0.2% indicates oesophageal intubation.

• *Oesophageal detector*: a 50 mL syringe is attached to the tracheal tube and the plunger rapidly withdrawn. If the tracheal tube is in the oesophagus, resistance is felt and air cannot be aspirated; if it is in the trachea, air is easily aspirated.

• *Direct visualization*: of the tracheal tube passing between the vocal cords.

• *Fogging*: on clear plastic tube connectors during expiration.

• *Less reliable signs are*:

• diminished breath sounds on auscultation;

• decreased chest movement on ventilation;

• gurgling sounds over the epigastrium and 'burping' sounds as gas escapes;

• a decrease in oxygen saturation detected by pulse oximetry. This occurs late, particularly if the patient has been preoxygenated.

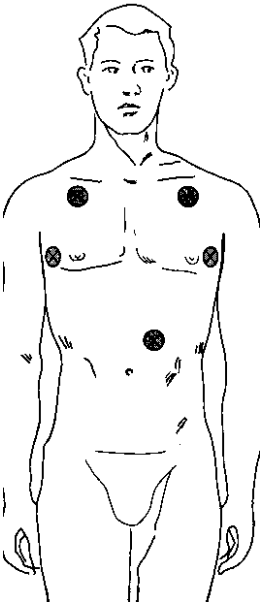


Figure 2.9 – Sites for auscultation of breath sounds at the apices and over the stomach

Complications of tracheal intubation

The following complications are the more common ones, not an attempt to cover all occurrences.

Hypoxia due to:

• *Unrecognized oesophageal intubation*

If there is any doubt about the position of the tube it should be removed and the patient ventilated via a facemask.

• *Failed intubation and inability to ventilate the patient*

This is usually a result of abnormal anatomy or airway pathology. Many cases are predictable at the preoperative assessment.

• *Failed ventilation after intubation*

Possible causes include the tube becoming kinked, disconnected, or inserted too far and passing into one main bronchus; severe bronchospasm and tension pneumothorax.

• *Aspiration*

Regurgitated gastric contents can cause blockage of the airways directly, or secondary to laryngeal spasm and bronchospasm. Cricoid pressure can be used to reduce the risk of regurgitation prior to intubation (see below).

Trauma

• *Direct*

During laryngoscopy and insertion of the tube, damage to lips, teeth, tongue, pharynx, larynx, trachea, and nose and nasopharynx durin nasal intubation; causing soft tissue swelling or bleeding.

• *Indirect*

To the recurrent laryngeal nerves, and the cervical spine and cord, particularly where there is pre–existing degenerative disease or trauma.

Reflex activity

• *Hypertension and arrhythmias*

Occurs in response to laryngoscopy and intubation. May jeopardize patients with coronary artery disease. In patients at risk, specific action is taken to attenuate the response; for example pretreatment with beta blockers or potent analgesics (fentanyl, remifentanil).

• *Vomiting*

This may be stimulated when laryngoscopy is attempted in patients who are inadequately anaesthetized. It is more frequent when there is material in the stomach; for example in emergencies when the patient is not starved, inpatients with intestinal obstruction, or when gastric emptying is delayed, as after opiate analgesics or following trauma.

• *Laryngeal spasm*

Reflex adduction of the vocal cords as a result of stimulation of the epiglottis or larynx.

Difficult intubation

Occasionally, intubation of the trachea is made difficult because of an inability to visualize the larynx. This may have been predicted at the preoperative assessment or

may be unexpected. A variety of techniques have been described to help solve this problem and include the following:

• Manipulation of the thyroid cartilage by backwards and upwards pressure by an assistant to try and bring the larynx or its posterior aspect into view.

• At laryngoscopy, a gum elastic bougie, 60cm long, is inserted blindly into the trachea, over which the tracheal tube is 'railroaded' into place.

• A fibreoptic bronchoscope is introduced into the trachea via the mouth or nose, and is used as a guide over which a tube can be passed into the trachea. This technique has the advantage that it can be used in either anaesthetized or awake patients.

• An LMA or ILM can be inserted and used as a conduit to pass a tracheal tube directly or via a fibreoptic scope.

Induction of general anesthesia

1. ASA standard monitors (Pulse oximeter, NIBP cuff, ECG and temperature).

2. Preoxygenation with 100% Oxygen or Denitrogenation with proper fitting mask.

3. Inhalation or Intravenous induction of general anesthesia.

4. Endotracheal intubation and securing the ET tube.

5. Protection of the pressure points.

Induction is the process that produces a state of surgical anaesthesia in a patient. The term is used only in the context of general anaesthesia and not with local anaesthesia. It is the first step in the process of anaesthesia whereby the patient is rendered unconscious, preventing both awareness of, and response to, surgical stimuli. Anaesthesia and physiological sleep are different because sleep has structured, specific EEG patterns and endocrine changes, whereas

anaesthesia is associated with a diffuse damping–down of EEG function and a stress–type endocrine response.

The process of induction should ensure patient safety, produce a state of unconsciousness, ensure optimal conditions for the surgeon and prepare the patient for waking and recovery.

Before anaesthesia is induced, the anaesthetist must:

• assess the patient as completely as circumstances allow, and institute preoperative preparations (e.g. intravenous fluids, sedative drugs, analgesics),

• discuss the surgery with the surgeon, particularly in complex or unusual cases,

• plan the anaesthetic technique,

• ensure all necessary equipment and drugs are available, and that the equipment is working,

On arrival in the anaesthetic suite, the anaesthetist must ensure:

• the correct patient has arrived,

• the correct operation is planned on the correct side,

• consent has been given,

• jewellery or prostheses have been removed or declared,

• blood for transfusion is available if required.

Induction methods

Most anaesthetic inductions are performed using intravenous or inhalational (‘gas’) induction; each has advantages and disadvantages. Less commonly, induction may be carried out by the intramuscular route in uncooperative patients, children or those with difficult venous access. Ketamine, 10 mg/kg, provides up to 30 minutes of surgical anaesthesia, but induction times are unpredictable (15–45 minutes) and recovery is slow. Rectal induction with thiopentone, methohexitone, chloral hydrate or benzodiazepines was popular for children at one time, but is seldom used now. Oral induction is effectively a heavy

sedative or ‘premedicant’, rather than induction, and recovery time is prolonged.

There are several tasks to be accomplished with any form of induction:

- last–minute preliminary checks (as specified above),
- establish monitoring,
- establish intravenous access,
- produce unconsciousness,
- secure the airway,
- establish ventilation,
- commence analgesia (systemic or local),
- position the patient,
- establish maintenance of anaesthesia.

Intravenous and inhalational induction

Intravenous	Inhalation
Advantages	*Advantages*
• Rapid onset	• Does not require IV access
• Patient comfort	• Useful for children
• Airway protection in rapid–sequence induction	• Useful for adults with needle–phobia
Disadvantages	*Disadvantages*
• Contraindicated in patients with a ‘difficult airway’	• Slow induction • Excitement’ phase • Risk of vomiting • Risk of arrhythmias

Intravenous induction

Pulse oximetry and ECG monitoring should be established before intravenous access because occasionally a cardiovascular event (e.g. vasovagal syncope) occurs during cannulation. Intravascular access commonly consists of a simple venous cannula; however, complex cases may require

an arterial line, a central venous line and/or a pulmonary artery catheter. These may be sited with local anaesthesia before induction, to provide additional monitoring if haemodynamic instability is expected.

Following intravenous access, preliminary drugs (e.g. analgesics, antibiotics, anti–emetics) may be given. These vary according to the clinical circumstances and a detailed discussion is beyond the scope of this article. A regional analgesic block, if required, may also be given at this stage.

Pre–oxygenation: some anaesthetists routinely pre–oxygenate their patients before induction. The correct technique is for the patient to breathe 100% oxygen via an anaesthetic circuit and close–fitting mask for about 3 minutes of tidal volume breathing. Alternatively, pre–oxygenation by three vital capacity breaths has been demonstrated to be effective. The aim is to replace nitrogen–containing air in the resting volume of the lungs (the functional residual capacity, FRC) with a high oxygen concentration. The gas within the FRC acts as an important oxygen store, and therefore pre–oxygenation lengthens the time before hypoxaemia occurs following the onset of apnoea. This may provide valuable time in which the airway can be secured if an unexpectedly difficult airway is encountered. Mask phobia and the difficulties in achieving a mask seal in non–compliant patients and children are the only significant contraindications to pre–oxygenation. Pre–oxygenation is mandatory in rapid–sequence induction.

Intravenous drugs: slow, smooth injection of an intravenous anaesthetic agent usually results in loss of consciousness in less than 1 minute. Thiopentone, for example, starts to work in a period of one ‘arm–brain circulation time’. This is the period of time taken for the drug to travel from the site of injection (the arm) to the site of action (the brain) and is about 15 seconds in a healthy patient. The dose is carefully titrated according to patient response.

Typical induction doses for healthy individuals are shown in Figure 3, but dose reduction may be required in:

- patients who are less fit,
- the elderly or frai,
- neonates,
- patients with hypotension or poor cardiac reserve,
- patients with chronic renal or liver disease,
- patients with raised intracranial pressure,
- patients who have been premedicated.

It can be difficult to judge when enough induction agent has been given. Care and experience are needed to titrate dose to effect but some indicators include:

- loss of response to verbal command,
- loss of eyelash reflex (in which brushing the eyelash produces a blink response),
- relaxation of a motor posture (e.g. a raised arm or a grip on an object),
- cooperation with bag/mask ventilation.

The eyelash reflex has conventionally been regarded as a good end–point for thiopentone induction, but it is less reliable in propofol induction, for which loss of verbal response or motor relaxation is a more useful end–point.

Induction doses of intravenous drugs

• Propofol – 1.5–2.5 mg/kg. Popular and widely used drug associated with rapid and ‘clear– headed' recovery. Rapid metabolism and lack of cumulative effects has made it popular for total intravenous anaesthesia

• Thiopentone – 3–5 mg/kg (2.5% solution).

The ‘gold–standard’ against which all other drugs are judged. Smooth induction in one arm–brain circulation time.

• Etomidate – 0.2–0.3 mg/kg Marked cardiovascular stability makes this drug popular for use in unstable patients.

• Ketamine – 0.5–2 mg/kg Useful for sedation with profound analgesia. Increases pulse rate and blood pressure and useful for the induction of patients suffering from acute trauma.

• Midazolam 0.15–0.5 mg/kg A benzodiazepine that may provide stable induction for the elderly and frail, in combination with an opioid.

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Inhalational induction

Gas induction, controversially, is often used as a means of inducing anaesthesia (particularly in a child) without having to site an intravenous cannula first. However, in the event of difficulties (e.g. laryngospasm, arrhythmias) instant intravenous access should be available, because otherwise the anaesthetist controlling the airway will be unable to attempt rapid cannulation. For this reason, some anaesthetists seldom perform gas induction; others permit gas induction if a second anaesthetist is present to assist with intravenous cannulation. Gas induction of children and adults has taken place without intravenous access for over 150 years, in most cases safely and without incident.

Indications: gas induction (with intravenous access) is indicated for patients in whom airway difficulties are expected. In these cases, the patient continues to breathe spontaneously throughout and apnoea is avoided, since it may then be impossible to manually ventilate the lungs with bag and mask.

Upper airway obstruction is an important indication for inhalation induction, and in these circumstances fibre–optic techniques for intubating the trachea are contraindicated for fear of producing complete airway obstruction. However, in patients with an unobstructed ‘difficult’ airway, the increasing availability of fibre–optic intubation equipment and the growing skill of anaesthetists in awake intubation techniques may reduce the need for gas induction. The difficult airway may best be secured even before anaesthesia is induced.

Technique: there is controversy over whether to induce in 100% oxygen or to use nitrous oxide as well.

• Concurrent use of volatile and nitrous oxide exploits the second–gas effect for a cumulatively more rapid induction. The rapid absorption of the second gas (nitrous oxide) has the effect of increasing the alveolar concentration of the first agent. The partial pressure of anaesthetic gas in the alveolus reflects the partial pressure of anaesthetic in the brain and hence the anaesthetic effect.

• 'Pre–induction', with 33% oxygen and 66% nitrous oxide only, may render a child sleepy enough not to resist when the odour of the volatile agent is added. Clearly the more nitrous oxide is used the more anaesthetic effect is achieved; but likewise the less reserve there is against desaturation. A minimum of 30% oxygen should be given.

• Induction in 100% oxygen is least smooth, but should laryngospasm occur, it is an advantage to have as much of the lung FRC filled with oxygen as possible. This maximizes oxygen stores and thus delays the onset of hypoxaemia.

Conventional practice for inhalational induction with halothane is to start with a low inspired concentration of 0.5%, and to increase it by 0.5% every four breaths up to 4%.

Sevoflurane has greatly enhanced gas induction, because it is faster, better tolerated by patients, and is less arrhythmogenic than halothane. It has been suggested that the lower incidence of arrhythmias has contributed to a decrease in dental anaesthetic deaths in recent years. The high blood–gas solubility of sevoflurane accounts for its rapid onset and offset. Because sevoflurane is less pungent it is often used in high concentrations (maximum 8% on most vaporizers) for faster induction.

Enflurane is seldom used for gas inductions because it is slow; isoflurane and desflurane are almost never used because they are pungent and irritating to the airway.

The last–minute checks before inhalational induction are the same as those for the intravenous route. Monitoring should be established; some children make this difficult, but

ECG should be the minimum monitoring instituted. Induction should ideally take place via a tight–fitting face mask (even small leaks may significantly delay induction). However, the use of a cupped hand may be less threatening to a small child in the first instance.

'Single–breath induction' has been described with halothane and sevoflurane. A Mapleson A breathing system containing a 4–litre reservoir bag is filled with a maximum concentration of volatile anaesthetic (4% halothane or 8% sevoflurane) in 66% nitrous oxide and 33% oxygen. The patient is asked to exhale to residual volume, then, via a tight–fitting mask, to inhale a full vital–capacity breath of gas, and then to hold their breath for as long as possible. This technique produces a faster induction than conventional tidal volume inhalational induction in cooperative adults. In the case of single–breath 8% sevoflurane, the speed of induction is comparable with induction with intravenous propofol. It may be a useful technique to use in cooperative needle–phobic adults, but it offers few other advantages.

Four main variables determine the speed of inhalational anaesthetic induction.

• The inspired partial pressure of the anaesthetic agent relative to its minimum alveolar concentration (MAC) alters the speed of induction. MAC is the partial pressure of the agent, expressed in volumes %, which at equilibrium prevents gross muscle movement in response to a skin incision in 50% of patients. It is thus the effective dose in 50% of patients and is a measure of anaesthetic potency.

• The faster the patient breathes, or the greater the alveolar ventilation, the faster the alveolar partial pressure of the agent approaches the inspired partial pressure. In a child, crying speeds up gas induction by increasing minute ventilation.

• The higher the cardiac output the more anaesthetic agent is removed from the alveoli and hence the slower the partial pressure rises in the alveoli. Thus, an anxious,

hyperdynamic patient is slow to induce, whereas a shocked patient with a low cardiac output is quicker.

• The higher the solubility of an agent (i.e. a high blood–gas solubility coefficient), the more the agent will dissolve in blood and thus a lower partial pressure will be generated. Agents with a low solubility (e.g. sevoflurane) result in more rapid induction.

During a gas induction, most patients pass briefly through a phase of excitability during which they may be agitated and at increased risk of laryngospasm or, more rarely, arrhythmias. If a child is being induced, it is useful to warn the parents of this disinhibition in advance. The disinhibition is not remembered by the patient.

Once the patient is unconscious, anaesthesia should be deepened, assisting the ventilation by hand, using bag and mask if necessary. If not already obtained, intravenous access should be secured, which requires the help of an assistant. Muscle relaxants may then be given intravenously to assist in securing the airway. If the patient is sufficiently deeply anaesthetized, as evidenced by a regular respiratory pattern and a forward gaze in eyes with small pupils, the airway may be secured (even by intubation) purely under inhalational anaesthesia. Nevertheless, it is valuable to have intravenous access before attempting to manipulate the airway, in case any untoward airway reflexes are produced (e.g. arrhythmias, laryngospasm). With the airway secured, the remainder of the induction sequence proceeds as above.

Further procedures: the induction agent may be followed by a muscle relaxant, particularly if tracheal intubation is planned. It is important to confirm that the patient’s lungs can be ventilated via a bag and mask before paralysing. A muscle relaxant should not be given until it has been confirmed that ventilation is possible. Once anaesthesia is adequate, a clear airway is established using a simple face mask (with or without an oral or nasal airway), laryngeal mask airway or tracheal intubation. If the level of anaesthesia

proves to be inadequate to allow an airway or tracheal tube to be tolerated, it may be deepened either with supplementary doses of intravenous agent, or by ventilating with volatile anaesthetic. With the airway secure, ventilation can continue by the patient's own effort, by manual 'hand' ventilation, or by mechanical ventilator.

At this stage, further invasive procedures may take place, such as additional vascular access, regional blocks, bladder catheterization, passing of a nasogastric tube or insertion of a temperature probe.

Transfer to theatre: if anaesthesia has been induced in the anaesthetic room, the patient is now transferred to theatre. This is potentially hazardous because for a short time the patient is separated from monitoring equipment and the mechanical ventilator. The patient is also potentially unstable because of the effects of induction drugs and is at risk of injury during the physical transfer. It is the anaesthetist's responsibility to guarantee the patient's safety. It should be ensured that ventilation and anaesthetic maintenance are re–established in good time, that tubes and lines are not dislodged, and that changes in clinical condition are detected promptly, in particular, cardiovascular instability, desaturation or signs of waking from anaesthesia.

Once in theatre, the priorities are:

• prompt transfer on to the operating table

• prompt re–establishment of ventilation

• prompt re–establishment of anaesthetic maintenance if using volatile anaesthetic drugs

• check correct drug delivery if using intravenous maintenance technique

• prompt re–establishment of monitoring equipment

• safe positioning of the patient

• commencement of maintenance fluids and temperature control.

Drug injection: the chosen drug is rapidly adminstered. Thiopentone is the drug of choice because of its rapid onset of action in one arm–brain circulation time, but propofol or etomidate are alternatives, albeit slightly slower–acting. The use of rapid–acting opioids such as alfentanil, 10 μg/kg, or fentanyl, 1 μg/kg, helps to reduce the pressor response to laryngoscopy.

Cricoid pressure: or **Sellick's manoeuvre**, is traditionally practised in rapid–sequence induction and is applied by the anaesthetic assistant as the patient starts to lose consciousness. Pressure is applied to the cricoid cartilage to compress it against the oesophagus, preventing passive regurgitation of stomach contents.

If active vomiting occurs, it is recommended that cricoid pressure be removed to prevent oesophageal rupture – suction, head–down tilt and turning the patient's head to one side is then used instead. Otherwise, cricoid pressure is removed only on the instruction of the anaesthetist, once the airway has been secured with a cuffed tube. Occasionally, it may be removed to facilitate an intubation that is being made more difficult by its continued application.

Muscle relaxation: suxamethonium, 1.5 mg/kg, is the drug of choice. Its rapid onset produces ideal intubating conditions, with a peak effect of muscle relaxation within 50 seconds of injection. It is important to allow the drug time to work, and not to begin the intubation sequence before muscle fasciculations have subsided.

The duration of apnoea is usually about 5 minutes in healthy individuals, and thus spontaneous respiration may be re–established early in the event of a failed intubation. Suxamethonium is contraindicated in patients with:

- previous allergy,
- susceptibility to malignant hyperpyrexia,
- myotonia,

• severe burns, muscle damage or paraplegia (of over 1 week's duration,,)

• known raised serum potassium.

In these patients, rocuronium, 0.6–0.9 mg/kg, may provide relaxation as rapidly as suxamethonium, but with longer duration. Rocuronium is also the drug of choice in patients with reduced or absent plasma cholinesterase activity, in whom suxamethonium has a long and unpredictable duration of action.

Intubation: the trachea is intubated with a cuffed tube following unconsciousness and muscle relaxation. Uncuffed tubes are used in children to avoid local pressure on the tracheal wall and to maximize the internal diameter of tube available. If difficulty is encountered with direct laryngoscopy, then simple steps may be taken to facilitate intubation:

• manipulate the larynx (the assistant providing the cricoid pressure may be distorting the view of the larynx)

• change to a larger blade laryngoscope,

• change to a different type of blade (e.g. a McCoy levering blade),

• use the gum–elastic bougie (this thin, flexible stylet may be used to pass through the cords providing a 'track' over which the tracheal tube can be railroaded).

Before the cricoid pressure is released, the correct position of the tube must be checked carefully by auscultation and capnometry. Once the cuff has been inflated and the airway has been secured, a nasogastric tube should be passed and aspirated, if not done previously. The rest of the anaesthetic proceeds as usual; but at the end of the procedure extubation should take place with the patient awake, with their protective airway reflexes re–established, positioned on their side in a head–down tilt, and with suction available

Maintenance of anaesthesia

This contribution is an overview of what is meant, and required, by maintenance of anaesthesia. Many of the topics are covered in greater detail in separate contributions; the aim here is to put these topics in context.

Maintenance of general anaesthesia involves four priorities:

- keeping the patient safe,
- keeping the patient comfortable,
- presenting the best possible operating conditions for the surgeon,
- preparing the patient for the postoperative period.

Keeping the patient safe

There are several aspects involved in keeping the patient safe:

- airway, breathing and circulation,
- temperature control,
- monitoring,
- positioning.

Airway: this will usually have been established at the time of induction. However, it should not be assumed that this remains secure. Airway disconnection or obstruction may occur and the anaesthetist must be able to detect and correct these incidents promptly, assisted by monitors such as the capnograph, pulse oximeter, disconnect alarm and airway pressure monitor. Movement of the tracheal tube (either towards the right main bronchus or withdrawal from the trachea) or dislodgement of the laryngeal mask airway (LMA) are particularly likely to occur when patients are transferred to the operating table or when changes are made to patient position. These periods require increased vigilance by the anaesthetist. The integrity of the airway should be rechecked

both clinically and by monitoring after each patient movement.

Tracheostomy, rigid bronchoscopy or ‘one–lung’ thoracic surgery may require intraoperative changes to the airway. The anaesthetist must be prepared for such changes and, if necessary, should formulate a plan with the surgeon in advance as to how the airway will be managed.

Breathing will often depend on whether muscle relaxants are being employed as part of the anaesthetic technique. Although the use of muscle relaxants is not essential for controlled ventilation, they are usually employed for ventilated patients. Surgical factors often influence the decision to use muscle relaxants. For example, abdominal surgery is greatly facilitated by muscle relaxation. In specialized surgery, such as ophthalmic surgery or neurosurgery, where the slightest move or cough may have disastrous consequences and carbon dioxide must be controlled, paralysis is ideal. Otherwise, anaesthetists have widely differing views as to whether breathing should be controlled. Some anaesthetists use muscle relaxants almost routinely for any operation lasting longer than about 30 minutes, on the basis that carbon dioxide retention is avoided, potent short–acting opioids can be used without fear of respiratory depression, and good lung aeration with avoidance of atelectasis may be easier to achieve. Others control ventilation less often unless indicated for reasons of airway management or the requirements of surgery. Some advantages and disadvantages of paralysis and ventilation are listed .

The technique of ‘paralysing and ventilating’ may be preferred when a tracheal tube is present. It is likely that a relaxant will already have been given to enable intubation. Also, the presence of a tube within the trachea is a potent stimulus, and unless local anaesthesia has been applied to the respiratory tract, a deeper level of anaesthesia may otherwise be needed for the patient to breathe spontaneously without coughing. Lighter planes can be tolerated when a laryngeal

mask or oropharyngeal airway are employed, because these devices are less stimulating to the patient. Paralysing and ventilating may also be used for longer operations, where carbon dioxide control and prevention of atelectasis is important.

However, avoiding relaxants wherever possible can reduce two important complications of general anaesthesia:

• unrecognized awareness – patient movement usually warns of light anaesthesia before the patient becomes aware

• accidental hypoxia – the spontaneously breathing patient may maintain oxygenation even in the event of circuit disconnection.

Ventilator settings – most anaesthesia ventilators are time–cycled and volume– or flow–driven. A rate, usually with a ratio of inspiratory time to expiratory time (I:E), and a tidal volume or flow need to be set. Most adults have a minute volume of 80–100 ml/kg, therefore a tidal volume of 8 ml/kg (e.g. 500–600 ml) and a rate of 10 breaths/minute is a good starting point; this can be adjusted according to the observed end–tidal partial pressure of carbon dioxide ($PE'CO_2$). For routine surgery, a $PE'CO_2$ of about 4.5 kPa should be aimed for. If metabolic rate and cardiac output (and hence pulmonary perfusion) are assumed to be constant, then PE'CO2 varies inversely with the alveolar ventilation.

In general, an I:E ratio of 1:2 is often the best compromise between maintaining low inflation pressures (e.g. 15–20 cm H_2O), satisfactory oxygenation (SpO_2 > 95%) and adequate carbon dioxide removal ($PE'CO_2$ 4.5 kPa). However, this can be decreased to 1:1.5 or even 1:1 to prolong inspiratory time. This may be useful in some patients to help decrease high airway pressures (e.g. > 25–30 cm H_2O) or improve oxygenation (e.g. in the obese). Some patients with asthma or chronic airway disease may benefit from a longer expiratory time to allow the lungs to empty, and an I:E ratio of 1:3 or 1:4 may be preferable.

Children have a proportionately higher minute ventilation than adults – up to 200 ml/kg in infancy, approaching adult values weight–for–weight by about age 10 years. Given that their tidal volume is slightly less weight–for–weight than adults (7 ml/kg), this increased minute volume must be provided for by an increase in rate. For example, in a 5 kg infant, a minute volume of 1000 ml might be provided by 30 breaths each of about 33 ml/minute.

However, paediatric ventilation is complicated by the obligatory leak around the uncuffed tube. Volume control is unlikely to inflate the lungs to the set volume (especially if a Newton valve is used with T–piece ventilation). This is usually overcome by increasing the tidal volume by titrating against inflation pressure and/or $PE'CO_2$. Some modern anaesthesia ventilators have pressure control capabilities (e.g. the *Draeger Julian*). Specific compliance varies little with age, therefore pressure control mode can be used to deliver an inflation pressure of 15–20 cm H_2O. This provides an adequate tidal volume for any age, despite small to moderate leaks, as long as lung compliance is normal.

Regardless of the mode of ventilation, a fresh gas flow must be selected. This depends on the breathing system in use as well as the size of the patient. A high flow should be used initially (e.g. 6 litres/minute), regardless of the circuit being used. This allows time for denitrogenation and equilibration of inhaled anaesthetic agent during this early period of rapid anaesthetic uptake. After about 10 minutes, flows can be reduced considerably if the system permits. The circle system with carbon dioxide absorber is widely used, and modern lightweight valves and low–resistance tubing make it suitable for paediatric use well below the traditional lower patient weight limit of 20 kg. With appropriate monitoring of gas concentrations within the circle, this permits total fresh gas flows of 1 litre/minute or less. Higher flows are required for T–pieces such as the Bain and Jackson Rees systems. Formulae exist for each of these circuits to predict the flow of

fresh gas required per kilogram patient weight to eliminate rebreathing for controlled and spontaneous respiration. However, a more practical approach is to reduce the fresh gas flow gradually, stopping when rebreathing of carbon dioxide begins to appear on the capnograph.

General anaesthesia, particularly when using volatile anaesthetic agents, causes the development of atelectasis in the dependent parts of the lung and impairs the pulmonary vascular response to hypoxia (hypoxic pulmonary vasoconstriction). These two factors produce a small but noticeable degree of shunt, usually about 10%. This can be corrected by giving anaesthetized patients higher inspired oxygen than the 21% present in air; 30% is usually given, though this can be titrated against observed oxygen saturation.

The fluid management

Circulation: appropriate fluid management is an important component of anaesthetic maintenance. Intravenous fluid requirements may range from none in short and relatively non–invasive procedures, to many times the circulating volume in long and traumatic procedures.

Fluid requirement consists of:

• replacement of existing deficit – crystalloid or blood

• metabolic maintenance requirements – crystalloid

• replacing additional ongoing losses – crystalloid, colloid or blood.

Existing deficit – in the adult elective surgical patient, who has been starved for at least 4 hours preoperatively (and sometimes much longer), a deficit of 500 ml of crystalloid can be assumed. This deficit is generally well tolerated and does not necessarily need replacing, but it decreases the patient's margin of reserve against any further losses, or against ongoing postoperative dehydration as a result of nausea or vomiting. Many anaesthetists routinely administer 500–1000 ml of crystalloid in all patients except those at risk from fluid overload (e.g. those with renal or cardiac failure). There is

evidence to suggest that this may improve the quality of early recovery and help to decrease postoperative nausea.

In the emergency patient, fluid deficit may be considerable, as a result of either trauma–related blood loss, or anorexia, vomiting and/or interstitial (third–space) fluid loss from surgical pathology. In either case, the deficit should be assessed and replaced with the appropriate fluid. The larger the deficit, the more important that it be replaced before induction, whenever circumstances permit. The exception to this rule is during major ongoing blood loss, such as a ruptured aortic aneurysm or major trauma, where the priority is stopping the bleeding rather than prolonging attempts to normalize the circulation preoperatively.

Maintenance – for many patients this is the least important component of intraoperative fluid management – the average adult requirement of about 100 ml/hour is negligible in the context of a short procedure and other fluid losses. However, in small children, maintenance requirements are proportionately larger and more significant. Paediatric maintenance fluid is usually calculated according to: 4 ml/kg/hour for the first 10 kg, plus 2 ml/kg/hour for the next 10 kg, plus 1 ml/kg/hour for the remainder. Thus, a 25 kg child would have a maintenance requirement of 40 + 20 + 5 = 65 ml/hour. The smaller the child, the less reserve there is for managing without ongoing maintenance fluids, and the greater the impact of preoperative starvation.

Infants have small glycogen reserves, and therefore need intravenous dextrose (e.g. as 5% dextrose in 0.18% saline) as maintenance to sustain their blood sugar levels. For longer procedures, careful attention must be paid to electrolyte balance, because infants have less ability to correct excessive salt loads or water loads.

Ongoing losses – the most significant aspect of intraoperative fluid management can be the most difficult to estimate.

Losses from extravascular spaces (e.g. gastrointestinal, evaporative, third–space fluid losses) are usually crystalloid losses. Evaporative losses can be large – an adult may lose in excess of 1 litre/hour from a laparotomy or thoracotomy wound, and even larger amounts from extensive open skin wounds such as burns or large graft sites. There is no way of measuring these losses directly, and measures such as blood pressure, pulse rate or central venous pressure (CVP) provide only an indirect measure of total body water. Urine output may be the most useful clinical measure. Blood tests such as plasma urea, sodium and haematocrit may provide information regarding the hydration state, and plasma electrolytes and haematocrit are now commonly available from blood gas machines.

Losses also occur from the intravascular space (i.e. blood). Losses in suction bottles may be complicated by wash, faeces, urine or amniotic fluid. Visual estimates of blood loss on swabs are often inaccurate – weighing of swabs is more precise, but still neglects losses on drapes, gloves and instruments. A dilutional technique relies on washing all swabs, instruments and gloves in a fixed volume of water, which can then be measured colorimetrically to give an accurate measure of blood loss. Such instruments are not widely available. Intravascular loss may be estimated indirectly from clinical measures such as blood pressure, pulse rate or CVP, though other anaesthetic factors such as the balance between surgical stimulation and depth of anaesthesia complicate these measures. Estimation of haemoglobin is useful only when adequate intravascular volume has been replaced. Blood losses may initially be replaced by colloid solution (e.g. a gelatin solution or hydroxyethyl starch), but larger losses (> 20% blood volume) may require replacement by packed–cell blood. Very large losses (> 1 blood volume) may require supplementation with fresh frozen plasma and/or platelets to maintain clotting function.

In practice, it is likely that fluid losses are under–replaced in many patients. This is offset by the hormonal 'stress response' to surgery: aldosterone, cortisol and antidiuretic hormone levels rise and atrial natriuretic peptide falls; all contribute to postoperative water retention.

Intraoperative fluid administration

The type and volume of fluid administered during surgery varies for each and every patient, but must take into account:

- any deficit the patient has accrued;
- maintenance requirements during the procedure;
- losses due to surgery;
- any vasodilatation secondary to the use of a regional anaesthetic technique

Intravenous fluids

During anaesthesia fluids are given intravenously to replace losses due to surgery and provide the patient's normal daily requirements. Three types are used: crystalloids, colloids, and blood and its components.

Crystalloids

These are solutions of crystalline solids in water.

Those containing sodium in similar concentrations to plasma are rapidly distributed throughout the extracellular fluid space (i.e. intravascular and interstitial volumes). Ultimately, only 25–30% of the volume administered remains intravascular. If such fluids are used to restore the circulating volume, three to four times the deficit will need to be given. If crystalloids containing a lower concentration of sodium than plasma (e.g. 4% glucose plus 0.18% saline) are given, then once the glucose is metabolized the remaining fluid is distributed throughout the entire body water (i.e. extracellular and intracellular volumes), and as little as 10% will remain intravascular. Crystalloids are used primarily either as an

emergency resuscitation fluid or to provide a patient's daily requirements of water and sodium.

Colloids

These are suspensions of high molecular weight particles. The most commonly used are derived from gelatin (Haemaccel, Gelofusine), protein (albumin) or starch (Hespan, HAES–steril).

Colloids primarily expand the intravascular volume and can initially be given in a volume similar to the deficit to maintain the circulating volume. However, they have a finite life in the plasma and will eventually be either metabolized or excreted and therefore need replacing.

Blood and blood components

There are several forms of blood and its components available. In the intraoperative period the most commonly used are red cell products, platelet concentrates and clotting factors.

• *Whole blood* Despite its name, this is basically red cells, plasma proteins and clotting factors (levels of V and VIII are low). There are no platelets. Each unit contains approximately 510mL with a haematocrit of 35–45%. Not widely available.

• *Red cell concentrate* This is the by–product of the removal of plasma from whole blood. Each unit contains 250mL with a haematocrit of 60–75%, and is hence very viscous with a poor flow rate.

• *Red cells in optimal additive solution (SAG–M)* A red cell concentrate to which a mixture of saline, adenine and glucose and mannitol has been added. This improves both red cell survival and flow characteristics. Each unit contains _300mL with a haematocrit of 50–70%. White cells are routinely removed to prevent the risk of prion transmission.

• *Platelet concentrates* Supplied either as 'units' containing 50–60mL (55 \ 109 platelets) or as bags equivalent

to four units. Four units or one bag will raise the platelet count by 30–40000mm3. Given via a standard giving set *without* the use of a microaggregate filter, as this will result in the loss of significant numbers of platelets.

• *Fresh frozen plasma (FFP)* This consists of the plasma separated from a single donation and frozen within 6 h. Each pack contains 200–250mL, with normal levels of clotting factors (except factor VIII, 70% normal). It should be infused as soon as it has thawed.

• *Cryoprecipitate* This is produced as a precipitate formed on the controlled thawing of FFP, which is collected and suspended in plasma. It contains large amounts of factor VIII and fibrinogen. It is supplied as a pooled donation from six packs of FFPin one unit and must be used as soon as possible after thawing.

Risks of intravenous blood and blood products

All blood donations are routinely tested for hepatitis B surface antigen, hepatitis C, syphilis and antibodies to the HIV. However, a period exists between exposure and the development of antibodies. The resultant infected red cells would not be detected by current screening techniques. The risk is very small, and has been estimated for hepatitis B at 1 :105 and for HIV at 1 :106 units transfused.

In order to try and eliminate these risks, techniques now exist for using the patient's own blood in the perioperative period.

• *Predepositing blood* Over a period of 4 weeks prior to surgery, the patient builds up a bank of two to four units of blood for retransfusion perioperatively.

• *Preoperative haemodilution* Following induction of anaesthesia 0.5–1.5 L of blood is removed and replaced with colloid. This can then be transfused at the end of surgery.

• *Cell savers* These devices collect blood lost during surgery via a suction system; the red cells are separated,

washed and resuspended, ready for retransfusion to the patient.

Intraoperative fluid administration

The type and volume of fluid administered during surgery varies for each and every patient, but must take into account:

- any deficit the patient has accrued;
- maintenance requirements during the procedure;
- losses due to surgery;
- any vasodilatation secondary to the use of a regional anaesthetic technique.

Temperature control: there is now good evidence that the maintenance of a physiological body temperature reduces postoperative morbidity. Patients suffering from head injury or undergoing certain neurosurgical procedures are possible exceptions. Shivering increases oxygen consumption, and predisposes to myocardial ischaemia, dysrhythmias, hypotension and acidosis. Hypothermia also increases susceptibility to infection and tends to lengthen hospital stay.

Temperature control is of particular importance in patients at the extremes of age (who have less intrinsic control over body temperature and a larger surface area–to–volume ratio through which to lose heat), in operations involving open body cavities, and in all patients undergoing lengthy procedures. About 20% of trauma patients who arrive at hospital are hypothermic (core temperature < 35°C).

A change in core temperature as a result of general anaesthesia is a three–phase response.

- The first phase is a brisk fall in core temperature as a result of vasodilatation, caused by redistribution of heat to the peripheries. Core temperature may be as low as 35.5°C in some patients after transfer to the operating theatre from the anaesthetic room.

• The second phase is a slower but more sustained fall as a result of accelerated loss of heat to the environment from the warmer peripheries.

• The third phase is a stable equilibrium at a lower core temperature.

It is important to note that the anaesthetic itself brings about these changes, which are independent of the nature of the surgery, though surgery may modify the pattern. The use of spinal or epidural anaesthesia instead of general anaesthesia reduces, but does not abolish, the three–phase response described above.

Passive rewarming is the prevention of heat loss, typically by raising the temperature of the environment. Passive rewarming can serve to modify only the second phase of the heat loss response. Covering with blankets or the use of foil 'space' blankets decreases heat loss by reduced transfer of heat by convection, conduction, evaporation and radiation.

Dry inspired anaesthetic gases need to be humidified, and a heat and moisture exchange filter in the breathing system reduces heat loss by evaporation from the respiratory tract (latent heat). Low flows in a circle system (e.g. 1 litre/minute) further reduce the quantity of dry gas needing to be humidified and help retain heat and moisture within the breathing system.

A 'normal' operating room temperature of 21°C is a compromise between that which is warm enough for the patient, but cool enough for the comfort of theatre personnel. An increase in theatre temperature (e.g. to 25°C) helps to reduce the gradient for heat loss in patients at high risk (e.g. young children, patients with extensive burns).

Active rewarming is the addition of heat into the patient. It may be used to prevent or reverse the first phase of the temperature drop. The local environmental temperature can be raised so much that the transfer of heat is reversed. Warm air convection blankets (e.g. the *Bair Hugger*) are considered as active rewarming.

Intravenous fluid warmers play an increasingly important role the greater the volume of intravenous fluid given. A fluid–warming device should be used in all at–risk patients and especially when stored blood is administered. Ventilatory gases may be warmed using a heated humidifier. Overhead radiant heaters may be used when a large area of the body needs to be exposed, and neonates may undergo surgery on an open incubator such as the *Resuscitaire*.

Cardiopulmonary bypass represents the ultimate in active rewarming, but is practical only in cardiothoracic surgery or in uncommon resuscitation situations.

Care is needed not to overheat the patient and temperature monitoring is necessary when active rewarming methods are used. Monitoring probes are available for a variety of body cavities, including nasopharynx, oesophagus, rectum, bladder and tympanic membrane. In routine clinical practice, core temperature is usually measured by nasopharyngeal or rectal temperature probes. Surface temperature monitors do not reflect core temperature, but the core–peripheral gradient may provide a useful measure of peripheral vasodilatation. A gradient of less than 2°C implies good peripheral perfusion.

Monitoring. The recommendations on standards of monitoring:

• An anaesthetist must be present throughout the conduct of general anaesthesia.

• Monitoring should be commenced before induction and continued until the patient has recovered from the effects of anaesthesia.

• These recommendations also apply to the administration of local anaesthesia, regional analgesia or sedation where there is a risk of unconsciousness or cardiovascular or respiratory complications.

• The anaesthetist should check all equipment before use. Monitoring of anaesthetic machine function during the administration of anaesthesia should include an oxygen

analyser with alarms. During spontaneous ventilation, clinical observation and a capnometer should be used to detect leaks, disconnection, and rebreathing and high pressure in the breathing system. Measurement of airway pressure, expired volume and carbon dioxide concentration is strongly recommended when mechanical ventilation is employed.

• A pulse oximeter and capnometer must be available for every patient.

• It is strongly recommended that clinical observation of the patient should be supplemented by continuous monitoring devices displaying heart rate, pulse volume or arterial pressure, oxygen saturation, the electrocardiogram and expired carbon dioxide concentration. Devices for measuring intravascular pressures, body temperature and other parameters should be used when appropriate. It is useful to have both waveform and numerical displays.

• Intermittent non–invasive arterial pressure measurement must be recorded regularly if invasive monitoring is not indicated. If neuromuscular blocking drugs are used, a means of assessing neuromuscular function should be available.

• Additional monitoring may be required in certain situations. These recommendations may be extended at any time on the judgement of the anaesthetist.

Blood sugar should be monitored in infants and diabetic patients. The blood sugar of diabetic patients should be known before.45

Positioning: an unconscious patient cannot move to relieve an uncomfortable position, and it is the anaesthetist's responsibility to prevent discomfort from becoming damage. The anaesthetist is also responsible for protecting the patient during movement on and off the operating table, and during changes of position. Traditionally, the anaesthetist has particular responsibility for the head and airway. It must be ensured that all members of the team are working to a

common agenda and with coordinated timing. It is usually easiest and safest to disconnect as much equipment as possible before moving the patient.

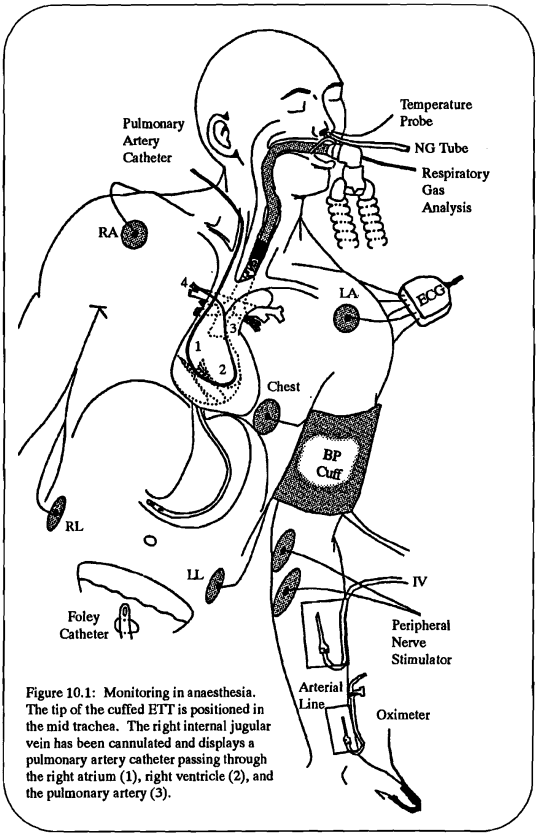

Figure 10.1: Monitoring in anaesthesia. The tip of the cuffed ETT is positioned in the mid trachea. The right internal jugular vein has been cannulated and displays a pulmonary artery catheter passing through the right atrium (1), right ventricle (2), and the pulmonary artery (3).

Figura 2.9 – Standards of monitoring

Prophylaxis against venous thromboembolism – the thrombotic process often starts intraoperatively. It is difficult to identify patients at high risk, though coexisting medical illness, major surgery, malignancy, trauma (especially hip and pelvis), obesity, high–dose oestrogen therapy and age greater than 40 years are well–known risk factors. Thromboprophylaxis should commence before anaesthesia;

gradated compression stockings and low–dose unfractionated heparin, 5000 IU s.c. twice daily, continued until full mobilization, is popular and effective. When positioning for surgery, raising the heels on foam pads prevents venous stasis in the calves. Intermittent pneumatic compression pumps assist venous return, but it is not known whether this reduces the incidence of postoperative pulmonary embolism.

Maintenance of unconsciousness is usually achieved by anaesthetic drug delivery via the inhalational or intravenous route, or both. The intramuscular route is seldom used in hospital practice owing to the relatively slow onset of drug action, unpredictable duration and delayed recovery. Ketamine, 10 mg/kg, is the only useful intramuscular agent, with an onset of 5–10 minutes producing up to 30 minutes of anaesthesia. It has a role in the provision of emergency anaesthesia in difficult locations.

Inhalational route – this is the most widely used technique, using a volatile anaesthetic agent with or without nitrous oxide. It therefore requires a supply of compressed gas, a vaporizer and a breathing system for drug delivery. Compressed gas may not be required if a 'drawover' type vaporizer is used.

The potency of an inhaled anaesthetic agent may be described in terms of its minimum alveolar concentration (MAC). MAC is defined as the alveolar concentration of the anaesthetic agent which at equilibrium is required to prevent gross reflex muscular movement in response to a standardized skin incision in 50% of healthy, unpremedicated patients. It is therefore a measure of anaesthetic potency, and is the effective dose in 50% of the population (ED50). It should be borne in mind that not all operations are 'a standardized skin incision'. The amount of anaesthetic needed to remove a foreign body from the nose is very different from that needed for an anal stretch. It is important to know the MAC of individual inhalational agents and the factors on which they depend (Figures 3 and 4).

Of any given inhalational anaesthetic, 0.7–1.3 MAC will anaesthetize 95% of the population. MACs are also additive: 0.5 MAC of nitrous oxide (52%) plus 0.5 MAC of isoflurane (0.6%) is equivalent to 1 MAC of any other inhalational agent given alone.

The principle of MAC acts as a useful guide. It allows the anaesthetist to select a vapour concentration that is likely to maintain unconsciousness. The state of anaesthesia is related to the partial pressure of anaesthetic within the brain, which is taken to be equivalent to the alveolar partial pressure. This can be measured by analysis of the end–tidal partial pressure of the anaesthetic agent. What is dialled on the vaporizer or the nitrous flowmeter is not necessarily what is in the patient's alveoli – the fresh gas flow takes time to equilibrate both with the dead space of the circuit and with the uptake by the patient. Observing how the ratio of end–tidal to inspired partial pressure varies with time can assess this rate of uptake (or 'wash–in').

MAC is useful to estimate the amount of anaesthetic required. In clinical practice this must be adjusted against indicators such as pulse rate, blood pressure, respiratory rate, patient movement, pupillary size, lacrimation and sweating. Many of these variables may be abolished by factors other than anaesthetic depth. Tachycardia may be prevented by co–administered β–blockers, hypertension masked by hypovolaemia, respiratory rate and patient movement abolished by paralysing drugs, and pupillary size altered by use of opioids or anti–muscarinic drugs. Thus, lacrimation and sweating, though crude indicators of inadequate anaesthesia, reflect the need for clinical observation in addition to monitoring.

Intravenous route – a popular alternative to inhalational anaesthesia is total intravenous anaesthesia (TIVA). Many intravenous anaesthetics have been used for TIVA, including barbiturates, ketamine, etomidate and propofol. The pharmaco–kinetic profile of propofol makes

this drug the most commonly used for TIVA. It has a high clearance (1300–1900 ml/minute), short metabolic half–life (60–100 minutes) and inactive metabolites. For short procedures, propofol may be administered following initial intravenous induction by intermittent bolus with no special infusion equipment (e.g. 50 mg as required every 3–5 minutes).

For longer procedures, the advent of reliable electronic syringe pumps, and in particular the development of target–controlled infusion (TCI) software, have contributed to the widespread use of TIVA techniques. Some advantages and disadvantages of inhalational or TIVA maintenance are shown in Figure 5.

Consider a three–compartment model: vascular space, richly perfused organs, and poorly perfused organs plus clearance. A large initial bolus of propofol is needed to fill the vascular compartment, namely the induction dose. Thereafter an initially high rate of infusion is needed to keep up with losses to the richly perfused compartment until it approaches saturation. Then a slower rate is required to keep up with losses to the poorly perfused but difficult to saturate compartment, and with metabolic clearance.

The ‘Bristol regimen’ reflects these kinetics. This regimen aims to maintain a plasma propofol concentration of about 3 μg/ml by giving patients receiving 67% nitrous oxide an initial bolus of 1 mg/kg, followed immediately by infusion at 10 mg/kg/hour for 10 minutes, then 8 mg/kg/hour for 10 minutes, then 6 mg/kg/hour thereafter. A dose of 10 mg/kg/hour is equal to the patient's weight (in kg) as ml/ hour of 1% propofol – hence a 60 kg patient will initially receive 60 ml/hour of 1% propofol. At the end of the operation, switching off the propofol allows rapid redistribution from the vascular compartment (and therefore from the richly perfused compartment also) to the still unsaturated third compartment. It is this rapid redistribution that allows a prompt wake–up even after a long period of TIVA.

TCI microprocessor–controlled technology (e.g. as incorporated in the Graseby 3500 ‘Diprifusor’ syringe pump) requires manual input of patient age, weight and desired plasma concentration. The pump then administers propofol according to the three–compartment pharmacokinetic model incorporated into its software. Change to a higher propofol concentration is achieved by a rapid zero–order infusion, and the plasma concentration is calculated until the new predicted value is reached. Change to a lower concentration is achieved by temporary cessation of drug infusion until the predicted plasma level falls to the required level, followed by continuation of infusion at a lower rate. The system is used in a similar fashion to adjusting the vaporizer setting during inhalational anaesthesia; the predicted plasma concentration of drug is analogous to the end–tidal concentration of the inhalational agent. Maintenance of satisfactory anaesthesia requires a plasma concentration of propofol of 2–6 mg/ml, depending on patient fitness, coexisting drug therapy and degree of surgical stimulation.

Analgesia: modern inhalational or intravenous anaesthetic drugs possess little analgesic activity, with the exception of ketamine. For all but the simplest procedures, analgesia must be provided by systemic analgesics (usually opioids) or by local anaesthetics. Analgesia has several effects.

- It reduces the required MAC (or plasma concentration) of co–administered anaesthetic drugs. Analgesia is an important component of the balanced anaesthetic technique.
- It reduces the immediate autonomic activity in response to pain. Sympathetic stimulation otherwise results in cardiovascular and respiratory responses that may lead to myocardial ischaemia and dysrhythmias.
- It reduces the neuroendocrine ‘stress response’ caused by surgery.

Opioid analgesics such as fentanyl, 15 μg/kg, reduce circulating concentrations of the stress hormones that increase after moderate and major surgery (e.g. noradrenaline, adrenaline, cortisol, growth hormone, glucagon, antidiuretic hormone). The stress response is largely detrimental and leads to increased catabolism, metabolic rate and oxygen consumption. This response is not significant in minor surgery.

The short–acting synthetic opioid drugs such as fentanyl and alfentanil are widely used to provide intraoperative analgesia. Fentanyl, a synthetic opioid structurally related to pethidine, is the most popular (1–2 μg/kg for minor procedures, onset 1–2 minutes, duration 30 minutes). Its potency and minimal effect on pulse and blood pressure make it commonly used for the provision of intense analgesia during surgery. These drugs are unsuitable for routine use in postoperative analgesia because of their short duration of action and their tendency to produce marked respiratory depression. They are commonly substituted by longer–acting analgesics (e.g. morphine 0.1–0.2 mg/kg i.v.) towards the end of the procedure to provide pain relief following surgery.

The control of the anaesthesia depth

Monitoring and controlling the depth of anesthesia is really important, since over dosing and under dosing can be dangerous for the patients**.**

Advanced monitoring of drug effect might help to optimizequality of drug delivery, possibly reduce costs and improve patient outcomes.

Guedel's stages of anaesthesia

Stage I (stage of analgesia or disorientation): from beginning of induction of general anesthesia to loss of consciousness.

Stage II (stage of excitement or delirium): from loss of consciousness to onset of automatic breathing. Eyelash reflex disappear but other reflexes remain intact and coughing,

vomiting and struggling may occur; respiration can be irregular with breath–holding.

Stage III (stage of surgical anesthesia): from onset of automatic respiration to respiratory paralysis.

It is divided into four planes:

Plane I – from onset of automatic respiration to cessation of eyeball movements. Eyelid reflex is lost, swallowing reflex disappears, marked eyeball movement may occur but conjunctival reflex is lost at the bottom of the plane

Plane II – from cessation of eyeball movements to beginning of paralysis of intercostal muscles. Laryngeal reflex is lost although inflammation of the upper respiratory tract increases reflex irritability, corneal reflex disappears, secretion of tears increases (a useful sign of light anesthesia), respiration is automatic and regular, movement and deep breathing as a response to skin stimulation disappears.

Plane III – from beginning to completion of intercostal muscle paralysis. Diaphragmatic respiration persists but there is progressive intercostal paralysis, pupils dilated and light reflex is abolished. The laryngeal reflex lost in plane II can still be initiated by painful stimuli arising from the dilatation of anus or cervix. This was the desired plane for surgery when muscle relaxants were not used.

Plane IV – from complete intercostal para ysis to diaphragmatic paralysis (apnea).

Stage IV: from stoppage of respiration till death. Anesthetic overdose cause medullary paralysis with respiratory arrest and vasomotor collapse. Pupils are widely dilated and muscles are relaxed.

Bispectral index (BIS) is one of several technologies used to monitor depth of anesthesia. BIS monitors are intended to replace or supplement Guedel's classification system for determining depth of anesthesia. Titrating anesthetic agents to a specific bispectral index during general anesthesia in adults (and children over 1 year old) allows the anesthetist to adjust the amount of anesthetic agent to the

needs of the patient, possibly resulting in a more rapid emergence from anesthesia. Use of the BIS monitor could reduce the incidence of intraoperative awareness during anaesthesia.

The bispectral index is a statistically based, empirically derived complex parameter. It is a weighted sum of several electroencephalographic subparameters, including a time domain, frequency domain, and high order spectral subparameters. The BIS monitor provides a single dimensionless number, which ranges from 0 (equivalent to EEG silence) to 100. A BIS value between 40 and 60 indicates an appropriate level for general anesthesia, as recommended by the manufacturer. The BIS monitor thus gives the anesthetist an indication of how "deep" under anesthesia the patient is.[The essence of BIS is to take a complex signal (the EEG), analyse it, and process the result into a single number. Several other systems claim to be able to perform the same thing. This calculation is very computer–intensive. The recent availability of cheap, fast computer processors has enabled great advances in this field. When a subject is awake, the cerebral cortex is very active, and the EEG reflects vigorous activity. When asleep or under general anesthesia, the pattern of activity changes. Overall, there is a change from higher–frequency signals to lower–frequency signals (which can be shown by Fourier analysis), and there is a tendency for signal correlation from different parts of the cortex to become more random.

Emergence from general anesthesia

Emergence is the process of return to baseline physiologic function of all organ systems after the cessation of administration of general anaesthetic agent(s).

Emergence from general anesthesia includes

1. Reversal of muscle relaxation.
2. Turning off the inhalation agents and nitrous oxide.

3. Meeting the extubation criteria.
4. Extubation of trachea.
5. Transfer of the patient to post anesthesia care unit.

First, the patient's neuromuscular blockade must be re–assessed, and if necessary reversed and then rechecked with a twitch monitor. Next, the patient has to be able to breathe on his own, and ideally follow commands, demonstrating purposeful movement and the ability to protect his airway following extubation. Suction must always be close at hand, since many patients can become nauseous after extubation, or simply have copious oropharyngeal secretions. Once the patient is reversed, awake, suctioned, and extubated, care must be taken in transferring him to the gurney and oxygen must be readily available for transportation to the recovery room/Post–Anesthesia Care Unit (PACU). Finally, remember that whenever extubating a patient, you must be fully prepared to reintubate if necessary, which means having drugs and equipment handy.

Extubation management

Extubation refers to removal of the endotracheal tube (ETT). It is the final step in liberating a patient from mechanical ventilation. Issues that need to be considered prior to extubation, the extubation procedure itself, and management after extubation are described here. Outcomes following extubation are also discussed. Predictors of weaning success and methods of weaning from mechanical ventilation are reviewed separately.

Prior to extubation

At the end of the weaning process, it may be apparent that a patient no longer requires mechanical ventilation to maintain sufficient ventilation and oxygenation. However, extubation should not be ordered until it has been determined that the patient is able to protect the airway and the airway is patent.

Airway protection

Airway protection is the ability to guard against aspiration during spontaneous breathing. It requires sufficient cough strength and an adequate level of consciousness, each of which should be assessed prior to extubation. The amount of secretions should also be considered prior to extubation because airway protection is significantly more difficult when secretions are increased.

Extubation failure is highest when a combination of risk factors is present. As an example, when reduced cough peak expiratory flow rate (≤60 L/min), increased sputum volume (>2.5 mL/hr), and impaired neurologic function (inability to follow commands) are present, the incidence of extubation failure was 100 percent, compared to 3 percent when none of the risk factors were present .

Universally accepted threshold levels of cough strength, level of consciousness, and suctioning frequency that prohibit extubation have not been established. For many patients, it seems reasonable to delay extubation if the cough strength is weak, the GCS is <8, or suctioning is required more frequently than every two to three hours. However, the final decision to delay or proceed with extubation should be made on a case–by–case basis since delayed extubation is associated with adverse outcomes, such as ventilator–associated pneumonia and increased length of stay .

Weaning from mechanical ventilation: Readiness testing.

Discontinuing mechanical ventilation is a two–step process:

Readiness testing – During readiness testing, objective clinical criteria are evaluated to determine whether a patient is ready to begin weaning. Some clinicians also consider physiological tests, known as weaning predictors, because they are hesitant to begin weaning on the basis of clinical criteria alone. The clinical criteria and weaning predictors are defined and described below.

Weaning is the process of decreasing ventilator support and allowing patients to assume a greater proportion of their ventilation. It may involve either an immediate shift from full ventilatory support to a period of breathing without assistance from the ventilator (ie, a spontaneous breathing trial [SBT]) or a gradual reduction in the amount of ventilator support. Regardless of which approach is chosen, extubation is considered once the patient demonstrates the ability to breathe without the ventilator and both airway patency and airway protection have been assessed.

Extubation protocol

Inclusion

Resolution of clinical issue requiring intubation:

Sat > 95% on $Fi0_2$ < 40%, PEEP < 5 cm H_20,

RR < 30, SBP >100, HR < 130.

Patient not known to be a difficult intubation.

Preparation

Turn off sedatives.

Leave opioids on at a low dose (e.g., fentanyl 50 pg/h).

Allow patient to regain full mental status.

If patient shows signs of discomfort, consider administering more pain medication.

Patient should be able to understand and respond to commands.

Testing for Readiness

Ask patient to raise arm and leave in air for 15 seconds.

Ask patient to raise their head off the bed.

Ask patient to cough, they should be able to generate a strong cough.

Place Patient on Pressure Support at a setting of 5 cm H_20. Sit patient up to at least 45*. Observe for 15*30 minutes. If Sat < 90%, HR > 140, SBP > 200, severe anxiety, or decreased LOC–discontinue extubation attempt.

Procedure

Have a nebulizer filled with normal saline attached to a mask.

Sit pt up to at least 45°.

Suction ET tube with bronchial suction catheter.

Suction oropharynx with Yankeur suction.

Deflate the ET tube cuff.

Have the patient cough, pull the tube during the cough

Suction the oropharynx again.

Encourage the patient to keep coughing up any secretions.

Place the nebulizer mask on the patient at 4–6 LPM.

After Extubation

Patient should receive close monitoring for at least 60 minutes.

If patient develops respiratory distress, NIV will often be sufficient to avoid reintubation.

Complications of general anesthesia

Complications related to the delivery of anesthesia care are inevitable. Even the most experienced, diligent and careful practitioner will have to manage complication despite acting well within the standard of care. These complication will range from minor (e.g. – infiltrated intravenous line) to catastrophic (hypoxic brain injury or death). These complications will trigger institutional review, peer review and potential legal action. Litigation may occur despite the best effort to communicate with the patient family about the intraoperative events, management decisions and avoidance of catastrophic complications. It is essential to document the preoperative airway examination, to record maneuvers such as preoxygenetion and cricoid pressure and details of laryngoscopy and write a complete post anesthesia note so that the action of anesthesiologist can be defended should litigation occur.

Perioperative mortality is usually define as death within 48hr of surgery. It is clear that most perioperative fatalities are due to patient's – Preoperative disease . – or the surgical procedure. Recent perioperative mortality rate is 1:20,000.

Anesthetics mishaps can be categorized as:– 1.Unpreventable: sudden death syndrome, fatal idiosyncratic drug reactions.

2. Preventable incidents: human error, equipment malfunctions and misuse. Common human error: unrecognized breathing circuit disconnection, mistaken drug administration, airway mismanagement, anesthesia machine misuse, fluid mismanagement, intravenous line disconnection.

Complications

Cardiovascular

– Myocardial ischemia and infarction.
– Hypotension.
– Hypertension.
– Cardiac arrhythmias.
– Stroke.
– Air or gas embolism.
– Thromboembolism.

Respiratory

– Due to ET.
– Upper airway obst.
– Lower airway obst.
– Atelctasis and infection.
– Sleep apnea.
– Pulmonary barotraumas.
– Pneumothorax.
– Inadequate ventilation.
– Decrease compliance.
– Increased CO2 production.

Neurological

– Abnormal muscle movt , convulsion.
– Delayed recovery.
– Acute dystonic reactions.
– Awareness .

Complication due to posture.

– Regurgitation, Aspiration.
– Malignant hyperthermia.
– Masseter m. spasm.
– Stress response.
– Hypothermia.
– Hypoxia.
– Hypercarbia.
– Anaphylactic reaction.
– Electrical.
– Fire explogions.
– Ophthalmic.
– Pollution.
– Radiation.
– Infection.

The respiratory system complications

(A), vomiting, reflux and choking and vomiting is a reflex action by forcing the discharge of gastric contents. Reflux of gastric contents by gravity or because of the effects of intra–abdominal pressure reflux into the throat cavity. Vomiting or regurgitation was easily result in aspiration, which leads to airway obstruction, choking or aspiration pneumonia, one of the main risk for the whole Ma. Vomiting and reflux often occurs in three square meals, the increase of intraabdominal pressure (such as intestinal obstruction, maternal), trauma, blood loss, shock, high intracranial pressure and coma patients. Certain drugs such as ether, the role of thiopental, abdominal viscera and throat operation of mechanical stimulation, oxygen and carbon dioxide accumulation and so influential. To prevent vomiting

and reflux caused by aspiration of the accident, should not eat or drink before anesthesia, the use of sedation, antiemetic or anti–acid drugs, needed for decompression. Of anesthesia in patients with full stomach should first awake intubation or rapid intubation, esophageal blocker can also be used, and strive to smooth induction of anesthesia. Under general anesthesia vomiting and reflux, they should immediately take head–down, so that the glottis above the esophageal entrance, head to one side to facilitate the timely removal of respiratory secretions. As a result of aspiration of acid gastric juice, especially occur when the acid aspiration syndrome, in addition to endotracheal suctioning, the use of dexamethasone, aminophylline, antibiotics and other medication, and the acid is diluted and used in 10 ml of saline were washing and cleaning endotracheal suction, and artificial respiration.

(B) is divided into parts of respiratory tract obstruction by airway obstruction and lower respiratory tract obstruction or both. Into mechanical obstruction by nature, such as tongue fall, secretions or foreign body obstruction and functional obstruction such as laryngeal or bronchial spasm.

Intraoperative bronchospasm :

Respiratory inflammation

Increased excitability of the vagus nerve

Increased the release of histamine bronchospasm –>

Propranolol and other non–selective B–blocker

Endotracheal tube inserted too deep

Existing asthma and other diseases

Performance: shortness of breath, difficulty breathing, three concave disease, cyanosis, heart rate, arrhythmia, SO2 decreased respiratory resistance increased, wheezing and other lung.

Treatment: find out the reasons, eliminate the original stimulus, applied the expansion of bronchus drugs (aminophylline, selective Î²2 agonist), and with the application of hormones.

1. Glossocoma jaw relaxation under general anesthesia, so that the tongue blocking the throat after the fall channel, resulting in partial or complete upper airway obstruction, can be heard snoring sounds (snoring), normal sleep may also appear. Treatment includes: 1) hold jaw; 2) into the oropharyngeal or nasopharyngeal airway; 3) partial side or shoulder padded head tilt the head back position .Unawakened under anesthesia should not be cushions the head, in order to avoid falling behind the tongue.

(1) The base of the tongue and blocking the respiratory tract after the fall.

(2) airway can take the lead to the back,

(3) oropharyngeal airway Law,

(4) nasopharyngeal airway Law,

Zebian: lili correction.

First, the vagus nerve reflex including:

1) OCR: oppression can lead to heart rate, cardiac arrest; nausea and vomiting.

2) The carotid sinus reflex: stimulation can cause changes in blood pressure reflex, heart rate, reflex syncope, cardiac arrhythmias.

3) The vagus–vagal reflex: the most common, vagus nerve stimulation of oppression by the regional if, through the inhibition of nerve fibers inhibit the heart of the heart, can cause arrhythmias and even cardiac arrest.

Parts: ear, nose throat, heart, lung, trachea, esophagus, liver, stomach, gallbladder, pancreas, spleen, small intestine, large intestine, rectum, bladder, reproductive system. More common following surgery: Department of Otolaryngology – biliary tract – the heart – the neck – the esophagus. The rest are mostly incidental. These parts of the surgical compression or stimulation, vagus – vagal reflex, resulting in unexpected changes in the cardiovascular.

2. Diagnosis:

Can occur at any age, children with more performance for the reflex laryngospasm, tonsils, palate repair, throat direct

examination when the possibility of large, adult to the high incidence of gallbladder surgery.

In addition to mechanical stimulation surgery, the following factors increase the incidence of:

Students with high sensitivity idiosyncratic unstable autonomic function

Hypoxemia and hypercapnia

Hypotension, acidosis

Too shallow or incomplete narcotic analgesia sick sinus surgery, a few in pre–block and pre–excitation syndrome

Sudden unexpected death syndrome: reflex laryngospasm, hypoxia asphyxia, respiratory center depression. Cardiac depression, cardiac arrest.

3. Processing:

1) mental preparation: surgical site and the operation is easy to cause, whether the original vagal nervous.

2) anesthesia: depth appropriate, to an additional local anesthesia block.

3) drugs: atropine, may give (different view), can be combined with ephedrine.

4) cardiac arrest: immediate chest compressions, controlled breathing. Emphasis: prevention, close observation, early detection, timely treatment, the recovery success rate.

Intraoperative hypoxaemia: decreased mainly as SPO2 Pathophysiology:

Hypotonic hypoxia: allergies can cause bronchospasm, ventilation ventilation dysfunction;

Hypoxia the blood: methemoglobinemia after poisoning, such as procaine;

Cyclic hypoxia: Hemorrhagic shock, protein loss decreased colloid osmotic pressure caused by pulmonary interstitial edema.

More common in:

Pulmonary disease (pneumothorax, pulmonary edema, atelectasis, chronic bronchitis and emphysema the original);

Heart disease (previous myocardial infarction, heart function insufficiency, cardiac ischemia, arrhythmia, etc.);

The pulmonary embolism:

1. Reason: emboli include blood clots (the most common, and the calf and femoral vein deep vein thrombosis the most common, such as indwelling catheters), air embolism, fat bolt, bolt of amniotic fluid, thrombosis and so on. The incidence of fatal pulmonary embolism: General elective surgery 0.1–0.8%, elective 0.3% –1.7% of hip surgery, emergency hip surgery 4% –7%.

2. Pathophysiology: Pulmonary embolism and damage lung tissue, pulmonary circulation, right ventricular and left ventricular function.

The main changes are as follows:

(1) Respiratory:

Increase in physiological dead space, ventilation / perfusion imbalance

Alveolar collapse and atelectasis, right–left shunt

Embolism atelectasis, pulmonary hypertension Gas exchange, hypoxemia.

(2) pulmonary infarction: some patients will occur (because of the bronchial circulation.)

(3) blood flow dynamics: decreased pulmonary blood flow increased pulmonary resistance load of pulmonary hypertension increases right heart right heart failure.

3. Diagnosis:

Symptoms: The most common are chest pain, breathing difficulties (there is no such two can not be diagnosed), while there is irritability, cough, fear, hemoptysis, syncope.

Signs: shortness of breath, rapid heartbeat, jugular vein distention, cyanosis, fever, pleural crepitus, pulmonary second sound hyperthyroidism, pulmonary valve area systolic murmur, hepatomegaly, hypotension, shock.

Other: ECG, approximately 40% of the patients had left axis deviation or right side, pulmonary P wave,

ventricular premature beats, atrial fibrillation. X ray, ultrasound, scans can help diagnose.

4. Treatment and Care:

(1). General support: oxygen, transfusion, boost, inotropic agents, anti–arrhythmia.

(2). Heparin therapy: venous thrombosis in heparin are required to quickly build, continuous intravenous injection once or q4h until the prolonged prothrombin time 1.5–2 times the normal time of 5–10 days. Serious bleeding disabled.

(3). Long–term anticoagulation: China Flynn can be oral (warfarin), the basic method as above.

(4). Thrombolytic therapy: streptokinase, urokinase, tissue plasminogen activator f.

(5). Other methods: vena cava occlusion, embolectomy and so on.

Severe intraoperative hemodynamic changes:

Abnormal heart rate too deep anesthesia

Intraoperative bleeding VF –> –> drop in blood pressure

Heart attack cardiac arrest

Arrhythmias during anesthesia:

A variety of arrhythmias can occur, such as tachycardia, bradycardia, atrial premature, ventricular premature premature junction, atrial fibrillation, atrioventricular block and so on. Including the elderly, the original heart disease–prone.

VIII, endotracheal tube blockage: to occur in infants and young children Cause: The catheter tip sharp, young tracheal mucosa, bleeding, secretions mixed easily in front of the formation of scabs.

Performance: CO2 accumulation induced heart rate, facial flushing, the misconception that "good", the further development of the induced fall in blood pressure, pulse small speed, cardiac arrest.

Treatment: urgent change management.

Severe hypotension (surgery, anesthesia, etc.);
Oxygen deficiency (including mechanical barriers, etc.).

The Malignant Hyperthermia:

Malignant Hyperthermia is an acute, fatal inherited metabolic disease. By the anesthesia (inhalation) and succinylcholine in susceptible individuals inspired, the performance characteristics of skeletal muscle metabolism in hyperthyroidism anesthesia for the crisis. 70 –90% early mortality rate, now reduced to 5 –10%, although a small but attention should be paid.

1. Diagnosis:

Sudden muscle hyperactivity syndrome. Sharp increase in skeletal muscle metabolic, showing oxygen consumption, CO_2, lactic acid and heat production were significantly increased. Induced respiratory acidosis, metabolic acidosis, muscle rigidity, high fever, muscle fiber damage hyperkalemia, myoglobinuria, CK increased, arrhythmia, and even cardiac arrest, brain damage, pulmonary edema, coagulation barriers, tube failure.

2. Incidence: rare, occurring in various reports vary, it is reported incidence of 1:15000 anesthetics – 1: 200000, children <15 years old accounted for 52.1%, 56.8% men. Can cause all of the volatile anesthetic, and when combined with the high incidence of forest Sikao.

3. Treatment:

(1). Integrated treatment: withdrawal, oxygen inhalation, hyperventilation and support.

(2). Note Dantrolene: 2.5 mg / kg, i vein, can be used repeatedly until symptom control.

(3). To correct respiratory acidosis: arterial blood gas guide, or directly to the baking soda 1–2mmol / kg i vein.

(4). Lower the body temperature: core temperature measurement, gastric lavage with cold saline irrigation, bladder, rectum, or other cooling measures.

(5). Antiarrhythmic: but not with calcium channel blockers.

(6). Against hyperkalemia: hyperventilation, $NaHCO_3$, intravenous glucose and insulin, can be fatal hyperkalemia when calcium chloride or calcium gluconate.

(7). Diuretic: mannitol, furosemide and so on.

(8). Cardiac arrest: note that the treatment with anti–potassium. Prevention: ready to dantrolene.

Low body temperature: with the following factors:

1) peripheral serious environmental temperature;

2) Enter the large number of short–term cold liquids (including laparoscopic lavage, abdominal cavity fluid);

3) central cooling effect of certain anesthetics;

4) respiratory loss of moisture and heat (open or semi–closed inhalation anesthesia).

Performance: heart rate, blood pressure, SPO_2 decreased, confusion, no spontaneous breathing, body temperature range.

Treatment: Rapid rewarming, closely monitored, controlled breathing, CVP, until recovery. *Acute pulmonary edema:*

Reason:

1) cardiac overload: Enter the excess liquid, such as application of vasoconstrictor drugs, particularly more common in children and heart function insufficiency;

2) heart failure;

3) hypoalbuminemia: the original or input over the blood–thinning liquid crystals;

4) respiratory tract obstruction: severe hypoxia and CO2 accumulation, aspiration, allergies and so on.

Performance: R rapid, TV down, coarse breath sounds lungs, blisters sound, SPO2 drop and so on;

Treatment: control infusion, given cedilanid, furosemide, aminophylline, dexamethasone, and so on.

Acute cerebral edema: Postoperative mortality

Possible reasons:

1) the expansion of acute cerebral palsy, cerebral blood flow increased;

2) surgical operation on the brain tissue extrusion or gross;

3) position;

4) hypoxia, CO_2 storage;

5) The volume of transfusion over;

6) The anesthetic itself;

7) anesthesia too shallow.

Performance: a sudden increase in intracranial pressure and brain swelling bone window, blood pressure and heart first and then decreased

Treatment: fully oxygen supply, adequate hyperventilation, mannitol diuretic hormone.

Prevention: For the reasons to avoid inhalation of anesthetic drugs and with intravenous anesthetics such as fentanyl, SP, etomidate, muscle relaxants used non–depolarizing muscle relaxants appropriate.

Local and regional anaesthesia

When referring to local and regional techniques and the drugs used, the terms ‘analgesia’ and ‘anaesthesia’ are used loosely and interchangeably. For clarity and consistency the following terms will be us.

Analgesia The state when only relief of pain is provided. This may allow some minor surgical procedures to be performed, for example infiltration analgesia for suturing.

Anaesthesia The state when analgesia is accompanied by muscle relaxation, usually to allow major surgery to be undertaken. Regional anaesthesia may be used alone or in combination with general anaesthesia.

All drugs will be referred to as local anaesthetics irrespective of the technique for which they are being useded:

Local anaesthetic drugs

EMLA

This is a *e*utectic mixture of local anaesthetics lignocaine and prilocaine in equal proportions (25mg of each per gram). It is applied as a cream to the skin and produces surface analgesia in approximately 60 mins. It is used to reduce the pain associated with venepuncture in children.

Ametop

This is a topical preparation of 4% amethocaine. It is used like EMLA to produce surface analgesia, but in a slightly shorter time. A synopsis of the drugs used for local and regional anaesthesia is given in Table 2.15.

Epinephrine (adrenaline)

This is added to local anaesthetics to reduce the rate of absorption, reduce toxicity and extend their duration of action. This is most effective during infiltration anaesthesia and nerve blocks, and less effective in epidurals or spinals. Some authorities recommend that solutions containing epinephrine should never be used intrathecally. Only very small concentrations of epinephrine are required to obtain intense vasoconstriction (a–adrenergic effect). The concentration is expressed as the weight of epinephrine (g) per volume of solution (mL). Commonly used concentrations range from 1:80000 to 1 : 200000.

Local anaesthetics containing vasoconstrictors must never be used around extremities (e.g. fingers, toes, penis), as the vasoconstriction can cause fatal tissue ischaemia. The maximum safe dose in an adult is 250 mg, that is, 20 mL of 1:80000 or 50 mL of 1:200000. This should be reduced by 50% in patients with ischaemic heart disease.

Calculation of doses

For any drug it is essential that the correct dose is given and the maximum safe dose never exceeded. This can be confusing with local anaesthetic drugs as the volume containing the required dose will vary depending upon the concentration (expressed in per cent), and a range of

concentrations exists for each drug. The relationship between concentration, volume and dose is given by the formula:

Local anaesthetic toxicity

This is usually the result of one of the following:

– *Rapid absorption of a normally safe dose* Use of an excessively concentrated solution or injection into a vascular area results in rapid absorption. It can also occur during intravenous regional anaesthesia (IVRA–see below) if the tourniquet is released too soon or accidentally.

– *Inadvertent IV injection* Failure to aspirate prior to injection via virtually any route.

– *Administration of an overdose* Failure or error in either calculating the maximum safe dose or taking into account any pre–existing cardiac or hepatic disease.

Signs and symptoms of toxicity are due to effects on the central nervous system and the cardiovascular system. These are dependent on the plasma concentration and initially may represent either a mild toxicity or, more significantly, the early stages of a more severe reaction.

– *Mild or early*: circumoral paraesthesia, numbness of the tongue, visual disturbances, lightheaded ness, slurred speech, twitching, restlessness, mild

hypotension and bradycardia.

– *Severe or late*: grand mal convulsions followed by coma, respiratory depression and, eventually, apnoea, cardiovascular collapse with profound hypotension and bradycardia, and ultimately, cardiac arrest.

Management of toxicity

If a patient complains of any of the above symptoms or exhibits signs, stop giving the local anaesthetic immediately! The next steps consist of:

– *Airway* Maintain using basic techniques. Tracheal intubation will be needed if the protective reflexes are absent to protect against aspiration.

– *Breathing* Give oxygen (100%) with support of ventilation if inadequate.

– *Circulation* Raise the patient's legs to encourage venous return and start an IV infusion of crystalloid or colloid. Treat a bradycardia with IV atropine.

If no major pulse is palpable, start external cardiac compression. If inotropes and vasopressors are required, invasive monitoring will be needed and this should be performed on the intensive care unit.

– *Convulsions* These must be treated early. Diazepam 5–10mg intravenously can be used initially but this may cause significant respiratory depression. If the convulsions do not respond or they recur, then seek assistance.

Because of the risk of an inadvertent overdose of a local anaesthetic drug, they should only be given where full facilities for monitoring and resuscitation are immediately available. In this way the patient will recover without any permanent sequelae.

The role of local and regional anaesthesia

Regional anaesthesia is not just an answer to the problem of anaesthesia in patients regarded as not well enough for general anaesthesia. The decision to use any of these techniques should be based on the advantages offered to both the patient and surgeon.

The following are some of the considerations taken into account.

– Analgesia or anaesthesia is provided predominantly in the area required, thereby avoiding the systemic effects of drugs.

– In patients with chronic respiratory disease, spontaneous ventilation can be preserved and respiratory depressant drugs avoided.

– There is generally less disturbance of the control of coexisting systemic disease requiring medical therapy, for example diabetes mellitus.

– The airway reflexes are preserved and in a patient with a full stomach, particularly due to delayed gastric emptying (e.g. pregnancy), the risk of aspiration is reduced.

– Central neural blockade may improve access and facilitate surgery, for example by causing contraction of the bowel or by providing profound muscle relaxation.

– Blood loss can be reduced with controlled hypotension.

– There is a considerable reduction in the equipment required and the cost of anaesthesia. This may be important in underdeveloped areas.

– When used in conjunction with general anaesthesia, only sufficient anaesthetic (inhalational or IV) is required to maintain unconsciousness, with analgesia and muscle relaxation provided by the regional technique.

– Some techniques can be continued postoperatively to provide pain relief, for example an epidural.

– Complications after major surgery, particularly orthopaedic surgery, are significantly reduced.

A patient should never be forced to accept a local or regional technique. Initial objections and fears are best alleviated, and usually overcome, by explanation of the advantages and reassurance.

Whenever a local or regional anaesthetic technique is used, facilities for resuscitation must always be immediately available in order that allergic reactions and toxicity can be dealt with effectively. At a minimum this will include the following:

– Equipment to maintain and secure the airway, give oxygen and provide ventilation.

– Intravenous cannulae and a range of fluids.

– Drugs, including epinephrine, atropine, vasopressors and anticonvulsants.

– Suction.

– A surface for the patient that is capable of being tipped head–down.

Local and regional anaesthetic techniques

Local anaesthetics can be used:

– topically to a mucous membrane, for example the eye or urethra;

– for subcutaneous infiltration;

– intravenously after the application of a tourniquet (IVRA);

– directly around nerves, for example the brachial plexus;

– in the extradural space (‘epidural anaesthesia’);

– in the subarachnoid space (‘spinal anaesthesia’).

The latter two techniques are more correctly called ‘central neural blockade’; however, the term ‘spinal anaesthesia’ is commonly used when local anaesthetic is injected into the subarachnoid space and it is in this context that it will be used.

Infiltration analgesia

Lignocaine 0.5% is used for short procedures, for example suturing a wound, and 0.5% bupivacaine for pain relief from a surgical incision. A solution containing epinephrine can be used if a large dose or a prolonged effect is required, providing that tissues around end arteries are avoided. Infiltration analgesia is not instantaneous and lack of patience is the commonest reason for failure. The technique used is as follows:

– Calculate the maximum volume of drug that can be used.

– Clean the skin surrounding the wound with an appropriate solution and allow to dry.

– Insert the needle subcutaneously, avoiding any obvious blood vessels.

–Aspirate to ensure that the tip of the needle does not lie in a blood vessel.

– Inject the local anaesthetic in a constant flow as the needle is withdrawn. Too–rapid injection will cause pain.

– Second and subsequent punctures should be made through an area of skin already anaesthetized.

When suturing, the needle is inserted into an area of intact skin at one end of the wound and advanced parallel to the wound, and local anaesthetic is injected as described. In a clean wound, local anaesthetic can be injected directly into the exposed wound edge. This technique can be also used at the end of surgery to help reduce wound pain postoperatively.

IVRA, bier's block

Local anaesthetic is injected into the veins of an exsanguinated limb and retained by using an arterial tourniquet. Anaesthesia is produced in 10– 15mins and the duration is limited by discomfort caused by the tourniquet. Sensation returns soon after release of the tourniquet. This is a useful technique for surgery of the distal upper limb. Correct functioning of the tourniquet is essential otherwise there is the risk of the patient being given the equivalent of a massive intravenous injection.

Contraindications are relatively few but include patients with impaired peripheral circulation or sickle–cell disease.

Brachial plexus block. The nerves of the brachial plexus can be anaesthetized by injecting the local anaesthetic drug either above the level of the clavicle (supraclavicular approach) or where they enter the arm through the axilla along with the axillary artery and vein (axillary approach). A nerve stimulator is frequently used to locate the nerves more precisely.

These techniques can be used for a wide range of surgical procedures below the elbow and will frequently provide good analgesia in the immediate postoperative period. As the block may last several hours, it is important to warn both the surgeon and patient of this.

Epidural anaesthesia

Epidural (extradural) anaesthesia involves the deposition of a local anaesthetic drug into the potential space *outside* the dura .This space extends from the craniocervical junction at C1 to the sacrococcygeal membrane, and anaesthesia can theoretically be safely instituted at any level in between. In practice, an epidural is sited adjacent to the nerve roots that supply the surgical site; that is, the lumbar region is used for pelvic and lower limb surgery and the thoracic region for abdominal surgery. A single injection of local anaesthetic can be given, but more commonly a catheter is inserted into the epidural space and either repeated injections or a constant infusion of a local anaesthetic drug is used.

Figure 2–10. Set for epidural anaesthesia

To aid identification of the epidural space, a technique termed ‘loss of resistance’ is used. The (Tuohy) needle is advanced until its tip is embedded within the ligamentum flavum (yellow ligament).

This blocks the tip and causes marked resistance to attempted injection of either air or saline from a syringe attached to the needle. As the needle is advanced further, the

ligament is pierced, resistance disappears dramatically and air or saline is injected easily.

A plastic catheter is then inserted into the epidural space via the needle. The catheter is marked at 5cm intervals to 20cm and at 1cm intervals between 5 and 15 cm. If the depth of the epidural space is noted, this allows the length of catheter in the space to be determined.

Varying concentrations of local anaesthetics are used depending on what effect is required. For example, bupivacaine 0.5–0.75% will be needed for surgical anaesthesia with muscle relaxation, but only 0.1–0.2% for postoperative analgesia. Local anaesthetic will spread from the level of injection both up and down the epidural space. The extent of anaesthesia is determined by:

–The spinal level of insertion of the epidural. For a given volume, spread is greater in the thoracic region than in the lumbar region.

– The volume of local anaesthetic injected.

– Gravity: tipping the patient head–down encourages spread cranially, while head–up tends to limit spread.

The spread of anaesthesia is described with reference to the limits of the dermatomes affected; for example: the inguinal ligament, T12; the umbilicus, T10; and the nipples, T4. An opioid is often given with the local anaesthetic to improve the quality and duration of analgesia, for example fentanyl 50mg. For details of infusions of local anaesthetics and opioids for postoperative analgesia.

Spinal anaesthesia

Spinal (intrathecal) anaesthesia results from the injection of a local anaesthetic drug directly into the cerebrospinal fluid (CSF), within the subarachnoid space . The spinal can only be inserted below the second lumbar and above the first sacral needle vertebrae; the upper limit is determined by the termination of the spinal cord, and the lower limit by the fact that the sacral vertebrae are fused and

access becomes virtually impossible. A single injection of local anaesthetic is usually used, thereby limiting the duration of the technique.

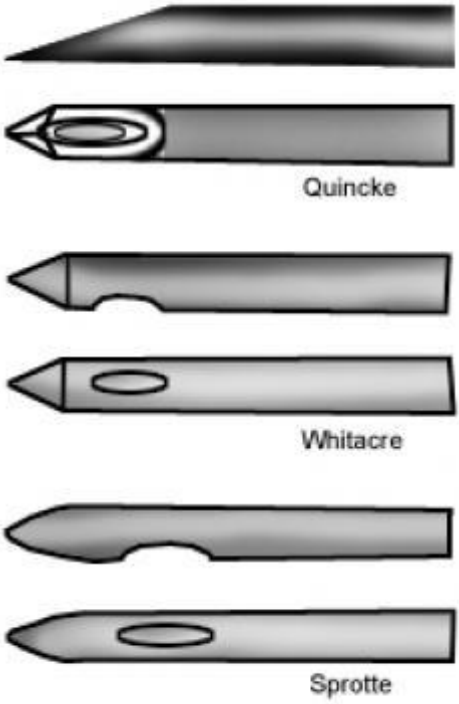

Figure 2–11. Spinal needles

A fine, 22–29 gauge needle with a ‘pencil point’ or tapered point (for example Whitacre or Sprotte needle) is used (Figure 2–11). The small diameter and shape are an attempt to reduce the incidence of postdural puncture headache (see below). To aid passage of this needle through the skin and interspinous ligament, a short, wide–bore needle is introduced initially and the spinal needle passed through its lume.

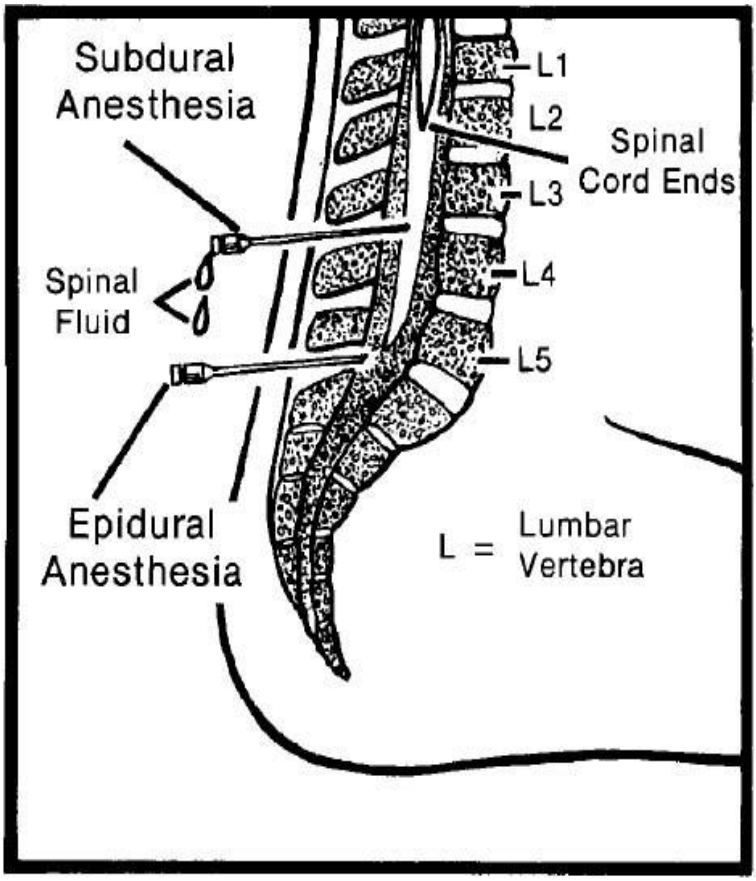

Figure 2–12. Sites for spinal anesthetics.

Factors influencing the spread of the local anaesthetic drug within the CSF, and hence the extent of anaesthesia, include:

– Use of hyperbaric solutions (i.e. its specific gravity is greater than that of CSF), for example ‘heavy’ bupivacaine (0.5%). This is achieved by the addition of 8% dextrose. Posture is then used to control spread.

– Positioning of the patient either during or after the injection. Maintenance of the sitting position after injection results in a block of the low lumbar and sacral nerves. In the supine position, the block will extend to the thoracic nerves around T5–6, the point of maximum backwards curve (kyphosis) of the thoracic spine. Further extension can be obtained with a head–down tilt.

– Increasing the dose (volume and/or concentration) of local anaesthetic drug.

– The higher the placement of the spinal anaesthetic in the lumbar region, the higher the level of block obtained.

Small doses of an opioid, for example morphine 0.1–0.25 mg, may be injected with the local anaesthetic. This extends the duration of analgesia for up to 24 h postoperatively.

Monitoring during local and regional anaesthesia

During epidural and spinal anaesthesia, the guidelines on monitoring (see page 49) should be followed. A conscious patient is not an excuse for inadequate monitoring! Particular attention must be paid to the cardiovascular system as a result of the profound effects these techniques can have. Maintenance of verbal contact with the patient is useful as it gives an indication of cerebral perfusion.

Incidence of common complications with spinal anaesthesia.

– Hypotension 33%.

– Nausea 18%.

– Bradycardia 13%.

– Vomiting 7%.

– Dysrhythmias 2%.

Early signs of inadequate cardiac output are complaints of nausea and faintness, and subsequent vomiting. The first indication of extensive spread of anaesthesia may be a complaint of difficulty with breathing or numbness in the fingers.

Clearly, these valuable signs and symptoms will be lost if the patient is heavily sedated.

Complications of central neural blockade

These are usually mild and rarely cause any lasting morbidity. Those commonly seen intraoperatively are due predominantly to the effects of the local anaesthetic. Their management is covered below.

Hypotension and bradycardia

Anaesthesia of the lumbar and thoracic nerves causes progressive sympathetic block, a reduction in the peripheral resistance and venous return to the heart and fall in cardiac output. If the block extends cranially beyond T5, the cardioaccelerator nerves are also blocked, and the unopposed vagal tone results in a bradycardia. Small falls in blood pressure are tolerated and may be helpful in reducing blood loss. If the blood pressure falls >25% of resting value, or the patient becomes symptomatic (see below), treatment consists of:

– oxygen via a facemask;

– IV fluids (crystalloids or colloids) to increasevenous return;

– vasopressors to counteract the vasodilatation, either ephedrine, an a– and b–agonist (3mg IV) or metaraminol, an a–agonist (0.25 mg IV);

– atropine 0.5 mg IV for a bradycardia.

Nausea and vomiting

These are most often the first indications of hypotension and cerebral hypoxia, but can also result from vagal stimulation during upper abdominal surgery. Any hypotension or hypoxia is corrected as described above. If due to surgery, try to reduce the degree of manipulation. If this is not possible then it may be necessary to convert to general anaesthesia. Atropine 0.3–0.6 mg is frequently effective, particularly if there is a bradycardia. Antiemetics can be tried (e.g. metoclopramide 10mg intravenously), but this must not be at the expense of the above.

Postdural puncture headache

Caused by a persistent leak of CSF from the needle hole in the lumbar dura. The incidence is greatest with large holes, that is, when a hole is made accidentally with a Tuohy needle, and least after spinal anaesthesia using fine needles (e.g. 26 gauge) with a pencil or tapered point (<1%). Patients usually complain of a headache that is frontal or occipital, postural, worse when standing and exacerbated by straining. The majority will resolve spontaneously. Persistent headaches can be relieved (>90%) by injecting 20–30 mL of the patient's own venous blood into the epidural space (epidural blood patch) under strict aseptic conditions.

Contraindications to epidural and spinal anaesthesia

Hypovolaemia Either as a result of blood loss or dehydration.

Such patients are likely to experience severe falls in cardiac output as compensatory vasoconstriction is lost.

*A low, fixed cardiac output A*s seen with severe aortic or mitral stenosis. The reduced venous return further reduces cardiac output, jeopardizing perfusion of vital organs.

Local skin sepsis. Risk of introducing infection.

Coagulopathy. Either as a result of a bleeding diathesis (e.g. haemophilia) or therapeutic anticoagulation. This risks causing an epidural haematoma. There may also be a very small risk in patients taking aspirin and associated drugs which reduce platelet activity. Where heparins are used perioperatively to reduce the risk of deep venous thrombosis, these may be started after the insertion of the epidural or spinal.

Raised intracranial pressure. Risk of precipitating coning.

Known allergy to amide local anaesthetic drugs.

A patient who is totally uncooperative.

Concurrent disease of the CNS Some would caution against the use of these techniques for fear of being blamed for any subsequent deterioration.

Previous spinal surgery or has abnormal spinal anatomy Although not an absolute contraindication, epidural or spinal anaesthesia may be technically difficult.

Examples of tascs for determination of knowledge lewel

1. What anesthetics, from those given below, has the least toxic properties in relation to vitally important human organs?

a) Nitrous oxide.
b) Fluothane.
c) Cyclopropane.
d) Halothane.

2. A patient, 53 years of age, with disturbed hepatic function, is admitted for the operation on the abdominal cavity. Which of the given below anesthetics has the greatest hepatotoxicity and undesirable in carrying out general anesthesia?

a) Cyclopropane.
b) Nitrous oxide.
c) Fluothane.
d) Ketamine.

3. In the process of anesthesia and controlled respiration by semiclosed circuit because of the technical error, committed in the operation of narcosis apparatus, the patient developed progressively increasing hypertension and tachycardia, replaced by bradycardia and catastrophic drop of blood pressure. The attention was paid to the increased skin moisture. What error, in all probability, was committed by anesthesiologist?

a) Oxygen dosimeter is not open.

b) Absorber is not switched on.
c) Depressurization of the apparatus has occurred.
d) Intubation tube came out from the trachea.

4. In a 20–year old female patient being on the operating table after an extubation of trachea, bradypnea, tachycardia and acrocyanosis were noted. A muscular tension, in hand shaking, is diminished. She cannot lift her head independently. What is the doctor's tactics?

a) To administer cardiac glycosides.
b) APV with a mask method.
c) Administration of anticoagulants.
d) To transfer to the ward.

5. The patient, aged 41, with phlegmon of the oral cavity and disturbance of patency of the air conducting ways owing to this phlegmon, is indicated narcosis with:

a) Calypsol i/v without APV.
b) Thiopental i/v without APV.
c) I/v with APV through tracheostoma.
d) With a mask.

6. In order to intubate trachea they apply:

a) Fogg's straight blade.
b) Mackintosh's curved blade.
c) Air duct.
d) Gegars retractor.

7. The minimum concentration of anesthetic in the alveolar air, causing the absence of motor response to pain in 50% of patients, is called:

a) a minimum pulmonary concentration;
b) minimum alveolar concentration;
c) the maximum analgesic concentration;
g) The minimum analgesic concentration.

8. Upon reaching what level of surgical anesthesia is possible to safely perform abdominal surgery?

a) 1–1;

b) III–1;

c) III–2;

d) III–3.

9. What complications can be caused by epidural anesthesia:

a) meningitis;

b) hypotension;

c) paralytic ileus;

d) spinal anesthesia.

10. The cause of regurgitation in anesthesia can be:

a) increasing intragastric and intra–abdominal pressure;

b) functional deficiency of the cardiac sphincter;

c) hypoxia from airway obstruction;

g) the availability of content in the stomach;

d) all answers are correct.

Correct answers:

1.–a); 2.–c); 3.–b); 4.–b); 5.–c); 6.–b); 7.–b); 8.–b); 9.–a),b),d); 10.–d).

Topic 3. POSTANESTESIA CARE

The main aim: to know foundations of observation and treatment patients in postoperative intensive care unit.

The student must know:

–Arrangement and equipment of postoperative intensive care unit.

– Postoperativ complications and their management.

– Postoperative intravenous fluid therapy.

– Ttreatment of postoperative pain.

– Analgesic techniques used postoperatively.

– Criterias for Discharging of the patient.

The student must be able:

– To use pulse oximeter.

– To use ECG monitoring device.

– To appoint treatment of hypoxaemia.

– To use defibrillator.

– To calculate Postoperative intravenous fluid requirements.

Mein material

The recovery area

The vast majority of patients recover from anaesthesia and surgery uneventfully, but a small and unpredictable number suffer complications. It is now accepted that all patients recovering from anaesthesia should be nursed in an area with appropriate facilities to deal with any of the problems that may arise, and by trained staff. Most patients will recover on a trolley capable of being tipped head–down. Patients who have undergone prolonged surgery, or where a prolonged stay is expected, may be recovered on their beds to minimize the number of transfers.

Each patient should be cared for in a dedicated area equipped with:

– oxygen supply plus appropriate circuits for administration;

– suction;

– ECG monitoring device;

– pulse oximeter;

–non–invasive blood pressure monitor.

In addition the following must be available immediately:

– *Airway equipment* Oral and nasal airways, a range of endotracheal tubes, laryngoscopes, a bronchoscope and the instruments to perform a cricothyroidotomy and tracheostomy.

– *Breathing and ventilation equipment* Self–inflating bag–valve–masks, a mechanical ventilator and a chest drain set.

– *Circulation equipment* A defibrillator, drugs for cardiopulmonary resuscitation, a range of IV solutions, pressure infusers and devices for IV access.

– *Drugs* For resuscitation and anaesthesia.

– *Monitoring equipment* Transducers and a monitor capable of displaying two or three pressure waveforms, end–tidal carbon dioxide monitor and thermometer.

Postanaesthesia care

A patient who cannot maintain his/her own airway should never be left alone.

The length of time any patient spends in recovery will depend upon a variety of factors, including length and type of surgery, anaesthetic technique and the occurrence of any complications. Most units have a policy determining the minimum length of stay, which is usually around 30mins, and agreed discharge criteria.

Postoperativ complications and their management

Hypoxaemia

This is the most important respiratory complication after anaesthesia and surgery. It may start at recovery and in some patients persist for 3 days or more after surgery. The presence of cyanosis is very insensitive and when detectable the arterial *P*O2 will be <8kPa (55 mmHg), a saturation of 85%. The advent of pulse oximetry has had a major impact on the prevention of hypoxaemia and should be used routinely in all patients. If hypoxaemia is severe, persistent or when there is any doubt, arterial blood gas analysis should be performed. Hypoxaemia can be caused by a number of factors, either alone or in combination:

– alveolar hypoventilation;
– ventilation and perfusion mismatch within the lungs;
– diffusion hypoxia;
– pulmonary diffusion defects;
– a reduced inspired oxygen concentration.

Alveolar hypoventilation

This is the commonest cause of hypoxaemia and results in insufficient influx of oxygen into the alveoli to replace that taken up by the blood. As a result, alveolar *P*O2 (*P*AO2) and arterial *P*O2 (*P*aO2) fall. In most patients, increasing their inspired oxygen concentration will restore alveolar and arterial *P*O2. Eventually a point is reached where there is only ventilation of ‘dead space’, that is, the volume of the airways that plays no part in gas exchange. No oxygen reaches the alveoli irrespective of the inspired oxygen concentration and profound hypoxaemia will follow. Hypoventilation is always accompanied by hypercapnia, as there is an inverse relationship between arterial carbon dioxide (*P*acO2) and alveolar ventilation.

Common causes of hypoventilation include:

– *Obstruction of the airway*. Most often due to the tongue. Consider vomit, blood or swelling (e.g. post–thyroid surgery). Partial obstruction causes noisy breathing; in

complete obstruction there is little noise despite vigorous efforts. There may be a characteristic 'see–saw' or paradoxical pattern of ventilation. A tracheal tug may be seen. It is prevented by recovering patients in the lateral position, particularly those recovering from surgery where there is a risk of bleeding into the airway (e.g. ear, nose and throat (ENT) surgery), or regurgitation (bowel obstruction or a history of reflux). If it is not possible to turn the patient (e.g. after a hip replacement), perform a chin lift or jaw thrust.

An oropharyngeal or nasopharyngeal airway may be required to help maintain the airway. No patient should be handed to the care of the recovery nurse with noisy respiration of unknown cause.

– *Central respiratory depression.* The residual effects of anaesthetic drugs decrease the ventilatory response to hypoxia and hypercarbia and also reduce the level of consciousness. Support ventilation until effects have worn off or reversed. Opioid analgesics (in excess) cause respiratory depression and reduce the level of consciousness. If severe, the administration of the specific antagonist naloxone may be required .

– *Hypothermia.* Reduces ventilation but, in the absence of any contributing factors, it is usually adequate for the body's needs.

– *Cerebral haemorrhage or ischaemia.* May cause direct damage to the respiratory centre or, more commonly, a deeply unconscious to maintain a patent airway.

– *Impaired mechanics of ventilation.* Pain, particularly after upper abdominal or thoracic surgery, prevents coughing, leading to sputum retention and atelectasis. Provide adequate analgesia (consider central neural block). Residual neuromuscular blockade is suggested by unsustained, jerky movements with rapid, shallow breathing in a hypertensive, tachycardic patient. For test to confirm the diagnosis. The patient should be given oxygen, reassured, sat upright to

improve the efficiency of ventilation, and a (further) dose of neostigmine and an anticholinergic given.

– *Pneumothorax or haemothorax.* Prevents ventilation of the underlying lung. Will require insertion of chest drain.

– *Diaphragmatic splintin.* Abdominal distension and obesity push the diaphragm into the thorax and increase the work of breathing. Such patients are greatly helped by being sat up.

– *Ventilation and perfusion mismatch.* Normally, ventilation of the alveoli (V) and perfusion with blood (Q) are well matched (V/Q = 1) to ensure that the haemoglobin in blood leaving the lungs is saturated with oxygen. During anaesthesia and the recovery period, this process is disturbed (ventilation/perfusion (V/Q) mismatch). Areas develop where:

– *Perfusion exceeds ventilation (V/Q < 1)*: this results in haemoglobin with a reduced oxygen content.

–*Ventilation exceeds perfusion (V/Q > 1)*: this can be considered wasted ventilation. Only a small additional volume of oxygen is taken up as the haemoglobin is already almost fully saturated (98%). In the most extreme situation, there is perfusion of areas of the lung but no ventilation (V/Q = 0). Blood leaving these areas remains 'venous' and is often referred to as 'shunted blood'. This is then mixed with oxygenated blood leaving ventilated areas of the lungs. The net result is:

– Blood perfusing alveoli ventilated with air has an oxygen content of approximately 20 mL/100 mL of blood.

– Blood perfusing unventilated alveoli remains venous, with an oxygen content of 15 mL/100 mL of blood.

– The final oxygen content of blood leaving the lungs will be dependent on the relative proportions of shunted blood and non–shunted blood.

For an equivalent blood flow, areas of V/Q < 1 decrease oxygen content more than increasing the oxygen concentration in areas of V/Q > 1 increases content.

The aetiology of V/Q mismatch is multifactorial but the following are recognized as being of importance:

– Mechanical ventilation reduces cardiac output. This reduces perfusion of non–dependent areas of the lungs, whilst ventilation is maintained. This is worst in the lateral position, when the upper lung is better ventilated and the lower lung better perfused.

– A reduced functional residual capacity (FRC). In supine, anaesthetized patients, particularly those over 50 years of age, the FRC falls below their closing capacity – the lung volume below which some airways close and distal alveoli are no longer ventilated. Eventually, areas of atelectasis develop, mainly in dependent areas of the lung that are perfused but not ventilated.

– Pain restricts breathing and coughing, leading to poor ventilation of the lung bases, sputum retention, basal atelectasis and, ultimately, infection.

This is more prevalent in the following circumstances:

– smokers;

– obesity;

– pre–existing lung disease;

– elderly;

– after upper gastrointestinal or thoracic surgery;

– 3 days after surgery.

The effects of small areas of V/Q mismatch can be corrected by increasing the inspired oxygen concentration. However, because of the disproportionate effect of areas V/Q < 1, once more than 30% of the pulmonary blood flow is passing through such areas, even breathing 100% oxygen will not eliminate hypoxaemia. The oxygen content of the pulmonary blood flow through areas ventilated with 100% oxygen will only increase by 1 mL/100 mL of blood (21 mL/100 mL of blood, insufficient to offset the lack from the areas of low V/Q.

Diffusion hypoxia

Nitrous oxide absorbed during anaesthesia has to be excreted during recovery. As it is very insoluble in blood, it rapidly diffuses down a concentration gradient into the alveoli, where it reduces the partial pressure of oxygen in the alveoli, making the patient hypoxaemic. This can be treated by giving oxygen via a facemask to increase the inspired oxygen concentration (see below).

Pulmonary diffusion defects Any chronic condition causing thickening of the alveolar membrane, for example fibrosing alveolitis, impairs transfer of oxygen into the blood. In the recovery period it may occur secondary to the development of pulmonary oedema following fluid overload or impaired left ventricular function. It should be treated by first administering oxygen to increase the partial pressure of oxygen in the alveoli and then by management of any underlying cause.

A reduced inspired oxygen concentration

As the inspired oxygen concentration is a prime determinant of the amount of oxygen in the alveoli, reducing this will lead to hypoxaemia. There are no circumstances where it is appropriate to administer less than 21% oxygen.

Management of hypoxaemia

All patients should be given oxygen in the immediate postoperative period to:

– counter the effects of diffusion hypoxia when nitrous oxide has been used;

– compensate for any hypoventilation;

– compensate for V/Q mismatch;

– meet the increased oxygen demand when shivering.

Patients who continue to hypoventilate, have persistent V/Q mismatch, are obese, anaemic or have ischaemic heart disease, will require additional oxygen for an extended period of time. This is best determined either by arterial blood gas analysis or by using a pulse oximeter.

Devices used for delivery of oxygen

Variable–performance devices: masks or nasal cannulae.

These are adequate for the majority of patients recovering from anaesthesia and surgery. The precise concentration of oxygen inspired by the patient is unknown as it is dependent upon the patient's respiratory pattern and the flow of oxygen used (usually 2–12 L/min). The inspired gas consists of a mixture of:

– oxygen flowing into the mask;

– oxygen that has accumulated under the mask during the expiratory pause;

– alveolar gas from the previous breath which has collected under the mask;

– air entrained during peak inspiratory flow from the holes in the side of the mask and from leaks between the mask and face.

Examples of this type of device are Hudson and MC masks. As a guide, they increase the inspired oxygen concentration to 25–60% with oxygen flows of 2–12 L/min. Patients unable to tolerate a facemask who can nose breathe may find either a single foam–tipped catheter or double catheters, placed just inside the vestibule of the nose, more comfortable. Lower flows of oxygen are used, 2–4 L/min increasing the inspired oxygen concentration to 25–40%.

If higher inspired oxygen concentrations are needed in a spontaneously breathing patient, a Hudson mask with a reservoir can be used .A one–way valve diverts the oxygen flow into the reservoir during expiration. During inspiration, the contents of the reservoir, along with the high flow of oxygen (12–15L/min), result in minimal entrainment of air, raising the inspired concentration to _85%. An inspired oxygen concentration of 100% can only be achieved by using either an anaesthetic system with a close–fitting facemask or a self–inflating bag with reservoir and non–rebreathing valve and an oxygen flow of 12–15 L/min.

Fixed–performance devices

These are used when it is important to deliver a precise concentration of oxygen, unaffected by the patient's ventilatory pattern. These masks work on the principle of high airflow oxygen enrichment (HAFOE). Oxygen is fed into a Venturi that entrains a much greater but constant flow of air. The total flow into the mask may be as high as 45L/min. The high gas flow has two effects: it meets the patient's peak inspiratory flow, reducing entrainment of air, and flushes expiratory gas, reducing rebreathing. Masks deliver either a fixed concentration or have interchangeable Venturis to vary the oxygen concentration .

The above systems all deliver dry gas to the patient that may cause crusting or thickening of secretions and difficulty with clearance. For prolonged use, a HAFOE system should be used with a humidifier.

Hypotension

This can be due to a variety of factors, alone or in combination, that reduce the cardiac output, the systemic vascular resistance or both:

– hypovolaemia;

– reduced myocardial contractility;

– vasodilatation;

– cardiac arrhythmias.

Hypovolaemia.

This is the commonest cause of hypotension after anaesthesia and surgery. Although intraoperative blood loss is usually obvious, continued bleeding, especially in the absence of surgical drains, may not be. Fluid loss may also occur as a result of tissue damage leading to oedema, or from evaporation during prolonged surgery on body cavities, for example the abdomen or thorax (see below). The diagnosis can be confirmed by finding:

– Reduced peripheral perfusion; cold clammy skin or delayed capillary refill (>2s) in the absence of fear, pain and hypothermia.

– Tachycardia; a pulse rate >100 beats/min of poor volume.

– Hypotension. Initially, systolic blood pressure may be reduced minimally but the diastolic elevated as a result of compensatory vasoconstriction (narrow pulse pressure). The blood pressure must always be interpreted in conjunction with the other assessments.

– Inadequate urine output (<0.5mL/kg/h), best measured hourly via a catheter and urometer.

Consider also the following as causes of reduced urine output:

– a blocked catheter (blood clot or lubricant);

– hypotension;

– hypoxia;

– renal damage intraoperatively (e.g. duringaortic aneurysm surgery).

Management

– Ensure adequate oxygenation and ventilation.

– Intravenous fluid, either crystalloid or colloid, should be given, using a pressure infusor to speed administration.

– Consider cross–matching blood if not already done.

– Stop any external haemorrhage with direct pressure.

– Get surgical assistance if internal haemorrhage suspected.

Monitoring of the patient's central venous pressure (CVP) may be indicated if cardiac function is in question. In the presence of significant hypovolaemia do not waste time inserting a CVP line for venous access alone. The trend of the patient's acid–base status is a useful indicator of therapeutic success.

Reduced myocardial contractility

The commonest cause is ischaemic heart disease, causing any degree of left ventricular failure.

Thediagnosis should be considered on finding:

– poor peripheral circulation;
– tachycardia;
– tachypnoea;
– distended neck veins;
– basal crepitations on auscultation of the lungs;
– wheeze with a productive cough;
– a triple rhythm on auscultation of the heart.

It is not uncommon to mistake this condition forhypovolaemia based on the first three findings. A chest X–ray is usually diagnostic.

Management

– Sit the patient upright.
– Give 100% oxygen.
– Monitor the ECG, blood pressure and peripheral oxygen saturation.

If the diagnosis is unclear, a fluid challenge (maximum 5mL/kg) can be given and the response observed; an improvement in the circulatory status suggests hypovolaemia. Where there is no doubt about the diagnosis, fluids can be restricted initially and a diuretic (e.g. frusemide 20–40mg) given intravenously. Trends in the CVP can be monitored as a guide to therapy. Patients with ventricular failure are best cared for in a critical care area. If there is acute myocardial infarction, contractility may only improve with the use of inotropes in conjunction with vasodilators, and this is best undertaken on the intensive care unit (ICU).

Unfortunately thrombolysis is contraindicated after surgery.

Vasodilatation

This is common during spinal or epidural anaesthesia.

Another example is following prostate surgery under spinal anaesthesia. As the legs are taken down from the lithotomy position, vasodilatation in the lower limbs is unmasked, and as the patient is moved to the recovery area he becomes profoundly hypotensive.

The development of septic shock may present initially as peripheral vasodilatation, hypotension and tachycardia in the absence of blood loss. The patient may be pyrexial and if the cardiac output is measured, it is usually elevated. Gradually, vasoconstriction ensues along with a fall in cardiac output. The diagnosis should be suspected in any patient who has had surgery associated with a septic focus, for example free infection in the peritoneal cavity or where there is infection in the genitourinary tract. This usually presents several hours after the patient has left the recovery area, often during the night following daytime surgery. The causative micro–organism is often a Gramnegative bacterium.

Management

Hypotension secondary to regional anaesthesia is corrected by the administration of fluids (crystalloid, colloid), the use of vasopressors (e.g. ephedrine), or a combination of both. Oxygen should always be given. The combination of hypovolaemia and vasodilatation will cause profound hypotension. Patients developing septic shock require early diagnosis, invasive monitoring and circulatory support in a critical care area. Antibiotic therapy should be guided by a microbiologist.

Cardiac arrhythmias

Occur more frequently in the presence of:

– hypoxaemia;
– hypovolaemia;
– hypercarbia;
– hypothermia;
– sepsis;
– pre–existing ischaemic heart disease;
– electrolyte abnormalities;
– hypo/hyperkalaemia, hypocalcaemia, hypomagnesaemia;
– acid–base disturbances;
– inotropes, antiarrhythmics, bronchodilators;

– antidepressants in overdose.

Tachycardias result in insufficient time for ventricular filling, thereby reducing cardiac output, while bradycardias reduce the heart rate below the point where no further increase in ventricular filling can occur to maintain cardiac output.

Coronary artery flow is dependent on diastolic pressure and time. Hypotension and tachycardia are therefore particularly dangerous.

Management

Correction of the underlying problem will result in spontaneous resolution of most arrhythmias. Specific intervention is required if there is a significant reduction in cardiac output and hypotension. The Resuscitation Council (UK) publishes guidelines that are regularly updated.

Sinus tachycardia (>100 beats/min). The commonest arrhythmia after anaesthesia and surgery, usually as a result of pain or hypovolaemia. If there is associated pyrexia, it may be an early indication of sepsis. Treatment consists of oxygen, analgesia and adequate fluid replacement. If the tachycardia persists, then providing there is no contraindication a small dose of a beta blocker may be given intravenously whilst monitoring the ECG. Rarely, the development of an unexplained tachycardia after anaesthesia may be the first sign of malignant hyperpyrexia.

– *Supraventricular tachycardia.* The most common is atrial fibrillation usually secondary to ischaemic heart disease or the presence of sepsis. Treatment will depend on the rate and reduction in cardiacoutput:

– heart rate 100–150 /min with critical perfusion will require cardioversion followed by IV amiodarone 300mg over 1h;

– heart rate <100 /min with good perfusion, consider amiodarone 300 mg IV over 1 h.

– *Sinus bradycardia (<60 beats/min).* Usually the result of:

– an inadequate dose of an anticholinergic (e.g. glycopyrrolate) given with neostigmine to reverse neuromuscular block;

– excessive suction to clear pharyngeal or tracheal secretions;

– traction on the viscera during surgery;

– excessive high spread of spinal or epiduralanaesthesia;

– the development of acute inferior myocardial infarction;

– excessive beta–blockade preoperatively or intraoperatively.

Treatment should consist of removing any provoking stimuli and administering oxygen. If symptomatic, atropine 0.5mg intravenously may be required.

Hypertension

This is most common in patients with pre–existing hypertension. It may be exacerbated or caused by:

– Pain.

– Hypoxaemia.

– Hypercarbia.

– Confusion or delirium.

– Hypothermia.

A coexisting tachycardia is particularly dangerous in the presence of ischaemic heart disease as this may cause an acute myocardial infarction. If the blood pressure remains elevated after correcting the above, a vasodilator or beta blocker may be necessary. Senior help should be sought.

Postoperative nausea and vomiting (PONV)

This occurs in up to 80% of patients following anaesthesia and surgery. A variety of factors have been identified which increase the incidence:

– Age and sex: more common in young women and children.

– Site of surgery: abdominal, middle ear or the posterior cranial fossa.

– Giving opioid analgesics pre–, intra– and postoperatively.

– Anaesthetic drugs: etomidate, nitrous oxide.

– Gastric dilatation, caused by manual ventilation with a bag and mask without a clear airway.

– Hypotension associated with epidural or spinalanaesthesia.

– Patients prone to travel sickness.

Patients identified as being at risk of PONV should be given an anti–emetic before emergence from anaesthesia. Failure of treatment may be addressed in the recovery area by giving a second or third drug from different classes of compound.

Drugs used to treat nausea and vomiting.

Before resorting to the administration of drugs to treat nausea and vomiting, it is essential to make sure that the patient is not hypoxaemic or hypotensive.

– *Antihistamines* Cyclizine. Adults 50mg intramuscularly, up to 6 hourly. Also has anticholinergic actions; may cause a tachycardia when given IV.

• *5–HT3 (hydroxytryptamine) antagonists* Ondansetron (Zofran). Adults 4–8mg intravenously or orally, 8 hourly. Has both central and peripheral actions; in the gut it blocks 5–HT3 receptors in the mucosal vagal afferents. It does not cause dystonic movements.

• *Dopamine antagonists* Metoclopramide (Maxolon). Adults 10mg intravenously, intramuscularly or orally, 6 hourly. Although a specific anti–emetic, minimal effect against PONV. Not related to the major tranquillizers and has no sedative or antihistamine effects. Has an effect at the chemoreceptor trigger zone and increases gastric motility. An alternative is domperidone (Motilium) 10 mg orally.

– *Phenothiazine derivatives.* Prochlorperazine (Stemetil). Adults 12.5 mg intramuscularly 6 hourly or 15–30 mg orally, daily in divided doses. May cause hypotension due to alpha–blockade. Some have antihistamine activity and may cause dystonic muscle movements.

– *Anticholinergic drugs* Atropine and hyoscine; the latter is available as a transdermal patch. Severe side–effects, particularly dry mouth and blurred vision.

– *Steroids* Dexamethasone 8 mg IV may be useful in resistant cases.

Postoperative intravenous fluid therapy

Oral intake should be encouraged as not all patients require routine IV fluids after anaesthesia and surgery. For those that do, the volume and type of fluid will be determined by a variety of factors, including:

– the site of surgery;
– the extent of tissue damage;
– blood loss during and after surgery;
– any delay in starting to drink;
– continuing losses from the gastrointestinal tract.

A wide range of fluids are available, and for each patient the type and volume will be dependent upon the calculated maintenance requirements of water and electrolytes plus the replacement of any abnormal losses. This is complemented by clinical evaluation of the patient to ensure that they are adequately hydrated, as assessed by degree of thirst, moisture of mucous membranes, blood pressure, pulse, peripheral circulation and an adequate urine output. In complex cases, monitoring the trend of the CVP may also prove useful.

Minor surgery

Following minor surgical procedures (i.e. taking less than 30 mins, with minimal blood loss and tissue tissue trauma), most patients start drinking within 1–2 h of surgery

and IV fluid is not required. If a patient has failed to drink within 4–6 h (usually as a result of nausea and vomiting), consideration should be given to commencing IV fluids. Providing that the volume of vomit is not excessive, only maintenance fluids are required. These are calculated at 1.5 mL/kg/h, but must take into account the accrued deficit.

For example, a 70 kg patient starved from 0800 to 1400, who is still unable to take fluids by mouth at 1800 will require:

An appropriate rate for the IV fluid would be:

– 1000 mL over the first 4 h;

– 1000 mL over the following 6 h;

– 500 mL over the last 4 h.

This should contain the daily requirement of Na++ 1–1.5 mmol/kg and could be given either as: 1000 mL 5% glucose and 500mL 0.9% normal saline; or 1000 mL 4% glucose/0.18% saline, and 500 mL 4% glucose/0.18% saline.

The patient should be reviewed at 0800 with regard to further management.

Major surgery

Following major surgery, postoperative fluid balance is more complex. Assuming that appropriate volumes of water, electrolytes and blood have been given during the operation, then postoperatively the fluid and electrolyte requirements will depend upon:

– the volume needed for ongoing maintenance, which will be increased if the patient is pyrexial;

– replacement of continuing losses from the gastrointestinal tract, for example via a nasogastric tube;

– any continued bleeding;

– rewarming of cold peripheries causing vasodilatation.

The patient who has undergone major surgery will require close monitoring to ensure that sufficient volumes of the correct fluid are administered. A standard postoperative

regimen for the first 24 h postoperatively might therefore consist of:

– 1.5 mL/kg/h water, increased by 10% for each °C if the patient is pyrexial;

– sodium, 1 mmol/kg;

– replacement of measured gastrointestinal losses with an equal volume of Hartmann's solution;

– replacement of blood loss of <500 mL with either:

– Hartmann's solution (three times the volume of blood lost will be needed as it is distributed throughout the extracellular fluid (ECF));

– colloid, the same volume as the blood loss;

– blood loss >1000 mL will require transfusion with stored blood.

It is essential that the patient is reviewed at the end of the day as described above to ensure that the volumes and type of fluid prescribed are adequate for the patient's needs. On the second and subsequent days, the same basic principles are used. In addition:

– The fluid balance of the previous 24 h must be checked.

– Ensure that all sources of fluid loss are recorded.

The patient's serum electrolytes must be checked to ensure adequate replacement.

– The urine output for the previous 6 and 24 h should be noted; if decreasing, consider other causes of fluid loss, for example increasing pyrexia, development of an ileus.

– Potassium will be required (in addition to sodium) at the rate of 1mmol/kg per 24 h. If surgery is associated with significant tissue trauma (e.g. total hip replacement, major gastrointestinal surgery), then there will be continued losses into the tissues, which have the same effect as any other form of fluid loss and are often referred to as 'third space losses'. Such volumes are difficult to measure and usually become evident as a result of the above regimen failing to keep the patient adequately hydrated. This is usually seen as thirst, a

dry mouth, cool peripheries with empty superficial veins, hypotension, tachycardia and a decrease in the urine output to less than 0.5 mL/kg/h. An additional 1 L of Hartmann's solution per 24 h may need to be added to the above regimen to account for such losses and adjusted according to the patient's response. These losses may continue for up to 48 h after surgery and sufficient extra volumes of fluid should be administered to maintain hydration and an adequate circulating volume. Where large volumes of fluid are required and/or there is underlying heart disease, then the CVP should be measured and the trend noted and serum electrolytes monitored twice daily.

The stress response

Following major surgery and trauma, various neuroendocrine responses result in an increased secretion of a variety of hormones. Antidiuretic hormone (ADH) secretion is maximal during surgery and may remain elevated for several days. The effect of this is to increase water absorption by the kidneys and reduce urine output. Aldosterone secretion is raised secondary to increased cortisol levels and activation of the renin–angiotensin system. This results in sodium retention and increased urinary excretion of potassium. Despite this retention of water and sodium, it is important that fluid input is not restricted in these patients, as the continued losses identified above more than offset the volume retained.

After 2–3 days, hormone levels return to normal and this is followed by an increase in the volume of urine passed, which may be augmented by loss of fluid as tissue oedema resolves.

Postoperative analgesia

After injury, acute pain limits activity until healing has taken place. Modern surgical treatment restores function more rapidly, a process facilitated by the elimination of postoperative pain. A good example is the internal fixation of fractures, followed by potent analgesia allowing early mobilization.

Ineffective treatment of postoperative pain not only delays this process, but also has other important consequences:

–Physical immobility:

–reduced cough, sputum retention and pneumonia;

– muscle wasting, skin breakdown and cardiovascular deconditioning;

– thromboembolic disease–deep venous thrombosis and pulmonary embolus;

– delayed bone and soft tissue healing.

– Psychological reaction:

–reluctance to undergo further, necessary surgicalprocedures.

– Economic costs:

– prolonged hospital stay, increased medical complications;

– increased time away from norma loccupations.

– Development of chronic pain syndromes.

Sometimes pain is a useful aid to diagnosis and must be recognized and acted upon, for example:

– pain due to ischaemia from tissue swelling, haematoma formation restricting the circulation causing a compartment syndrome or by dressings becoming too tight;

– pain of infection from cellulitis, peritonitis or pneumonia;

– referred visceral pain in myocardial infarction (arm or neck) or pancreatitis (to the back). What to expect postoperatively, what types of analgesia are available and also by allowing patients to explore their concerns.

– Patients who have a pre–existing chronic pain problem are vulnerable to suffering with additional acute pain Their nervous systems can be considered to be sensitized to pain and will react more strongly to noxious stimuli. Bad previous pain experiences in hospital or anticipation of severe pain for another reason suggest that extra effort will be required to control the pain.

– Older patients tend to require lower doses of analgesics as a result of changes in drug distribution, metabolism, excretion and coexisting disease.

Prescribing should take these factors into account rather than using them as an excuse for inadequate analgesia. There is no difference between the pains suffered by the different sexes having the same operation.

– Upper abdominal and thoracic surgery cause the most severe pain of the longest duration, control of which is important because of the detrimental effects on ventilation. Pain following surgery on the body wall or periphery of limbs is less severe and for a shorter duration.

Management of postoperative pain

This can be divided into a number of steps:

– assessment of pain;

– analgesic drugs used;

– techniques of administration;

– difficult pain problems.

Assessment of acute pain

Regular measurement of pain means that it is more difficult to ignore and the efficacy of interventions can be assessed. There are a variety of methods of assessing pain; Table 3.3 shows a simple, practical system that is easily administered and understood by patients. The numeric score is to facilitate recording and allows trends to be identified. Pain must be assessed with appropriate activity for the stage of recovery; for example, 5 days after a hip joint replacement a patient would not be expected to have pain while lying in bed, but adequate Any patient who complains of pain that unexpectedly increases in severity, changes in nature or site, or is of new onset should be examined to identify the cause rather than simply be prescribed analgesia.

Factors affecting the experience of pain

Pain and the patient's response to it are very variable and should be understood against the background of the

individual's previous personal experiences and expectations rather than compared with the norm.

– Anxiety heightens the experience of pain. The preoperative visit by the anaesthetist plays a significant role in allaying anxiety by explaining analgesia should allow mobilization with only mild to insignificant pain.

The most commonly used drugs are opioids and NSAIDs.

Opioids

Morphine is most commonly used to control severe postoperative pain on surgical units, and diamorphine (heroin) on medical wards, for example coronary care units, mainly for historical reasons. There are few pharmacological differences between these two drugs. Morphine can be given by several routes. One of the principal metabolites, morphine–6–glucuronide (M6G), has potent opioid effects and may accumulate and cause toxicity in patients with renal failure, particularly the elderly. Fentanyl and oxycodone have less active metabolites than morphine and so may be more suitable for these patients.

For most painful clinical conditions there will be a blood level of opioid that provides useful analgesia, that is, a reduction in pain level. The dose required to achieve this may vary enormously between patients as a result of differences in:

– pharmacodynamics: the effect of the drug on the body (via the receptors); pharmacokinetics: how the body distributes, metabolizes and eliminates the drug;

– the nature of the stimulus;

– the psychological reaction to the situation.

The biggest step forward in the treatment of acute pain with opioids has been the recognition that individual requirements are very variable and the dose needs to be titrated for each patient:

– There is no minimum or maximum dose.

–Even with best practice some pain will remain.

–Minimum levels of monitoring and intervention are necessary for safe, effective use.

–Additional methods of analgesia should be considered if opioid requirements are high.

Overdose

Profound respiratory depression and coma due to opioids must be treated using the ABC principles described elsewhere (page 99). Having created a patent airway and supported ventilation using a bag–valve–mask with supplementary oxygen, the effects of the opioid can be pharmacologically reversed (antagonized) using naloxone. 0.4mg is diluted to 5 mL with 0.9% saline and given in incremental doses of 1 mL IV (adult dosing). Analgesia will also be reversed, and careful thought must be given to continuing analgesia. HDU care is usually advisable in this situation.

Long–term complications of opioids

Adequate treatment of acute pain with opioids is not associated with dependency.

Less potent opioid agonists

– *Codeine (3–methyl morphine)* Well absorbed orally, dose 30–60 mg 6 hourly (can be given intramuscularly but never intravenously). Available in a range of tablets, often combined with paracetamol, for example co–codamol (8mg codeine, 500 mg paracetamol). Exerts its effect by a small amount (10%) being metabolized to morphine in the liver. Some patients lack the necessary enzyme and therefore get no effect from codeine.

– *Tramadol* Similar potency to codeine and used for mild to moderate pain.

Neither is a controlled drug and so are more easily accessible.

Non–steroidal anti–inflammatory drugs (NSAIDs)

– *Paracetamol* An analgesic and antipyretic with little anti–inflammatory action, but usually classified with NSAIDs. Inhibits prostaglandin synthesis, mainly in the CNS.

It is used to treat mild to moderate pain. Well absorbed orally, causing little irritation of the gastrointestinal tract. Widely used orally in a dose of 1g 4–6 hourly, maximum 4g/day. Often incorporated into compound preparations with aspirin or codeine. An intravenous preparation is available containing 10 mg/mL, in 100 mL vials (1 g). This can be infused over 15mins and is effective in 5–10 mins. The dose is the same as for the oral preparation. It is the safest of all analgesics but patients may need reassurance that regular dosing of 1 g every 6 h is not associated with hepatic toxicity.

Analgesic techniques used postoperatively

Patient–controlled analgesia (PCA)

– A microprocessor–controlled syringe pump capable of being programmed is used to deliver a predetermined dose of a drug intravenously.

– Activation is by the patient depressing a switch that is designed to prevent accidental triggering (hence 'patient–controlled').

– There may be a background, low–dose, continuousinfusion.

To prevent the administration of an overdose:

– The dose and any background infusion is preset (usually by a doctor).

– After successful administration of a dose, a subsequent dose cannot be administered for a preset period, the 'lockout period'.

– The total quantity of drug given over a predetermined period can be limited. Typical settings for an adult using morphine delivered by a PCA device might be:

– bolus dose: 1mg;

– lockout interval: 5mins.

Effective PCA requires:

– That the patient be briefed by the anaesthetist and/or nursing staff preoperatively and, if possible, be shown the device to be used.

–A loading dose of analgesic, usually intravenously before starting. Failure to do this will result in the patient being unable to get sufficient analgesia from the PCA device and the system will fail.

– A dedicated IV cannula or non–return valve on an IV infusion to prevent accumulation of the drug and failure of analgesia. Observation and recording of the patient's pain score, sedation score and respiratory rate to ensure success.

Advantages of PCA

– Greater flexibility; analgesia matched to the patient's perception of the pain.

– Reduced workload for the nursing staff.

– Elimination of painful IM injections.

Management of overdose with patientcontrolled analgesia (PCA)

– Stop the PCA.

– Give oxygen via a mask.

– Call for assistance.

– Consider giving naloxone.

– If the patient is apnoeic, commence ventilation using a self–inflating bag–valve–mask device.

– Intravenous administration with greater certainty of adequate plasma levels.

Disadvantages

– Equipment is expensive to purchase andmaintain.

– Requires patient comprehension of the system.

– Patient must be physically able to trigger the device.

– The elderly are often reluctant to use a PCA device.

– The potential for overdose if the device is incorrectly programmed.

As pain subsides the PCA can be discontinued, and oral analgesics can be used. The first dose should be given 1h prior to discontinuing PCA, to ensure continuity of analgesia.

Regional analgesic techniques

• *Peripheral nerve blocks* Used mainly for pain relief after upper or lower limb surgery. A single injection of local anaesthetic, usually bupivacaine, results in 6–12 h of pain relief. An infusion of local anaesthetic via a catheter inserted close to the nerve may enable the block to be continued for several days. An alternative effective form of analgesia must be prescribed for when the local anaesthetic is discontinued to prevent the patient being in severe pain.

– *Epidural analgesia.* Infusions of a local anaesthetic into the epidural space, either alone or in combination with opioids, act on the transiting nerve roots and the dorsal horn of the spinal cord, respectively, to provide dramatic relief of postoperative pain. It is essential that patients who are offered an epidural receive an explanation by the anaesthetist at the preoperative visit of what to expect postoperatively, in particular altered sensation, weakness of the lower limbs and the potential need for a urinary catheter. The epidural is often sited preoperatively and used as part of the anaesthetic technique. For upper abdominal surgery an epidural in the mid–thoracic region (T6/7) is used, while a hip operation would need a lumbar epidural (L1/2).

Different combinations of local anaesthetic and opioid infusion have been used successfully.

Ideally, the concentration of local anaesthetic should block sensory nerves, leaving motor nerves relatively spared. The choice and dose of opioid should be such that the drug passes through the dura into the CSF in sufficient quantities to block the opioid receptors in the spinal cord but not spread cranially to cause respiratory depression. For example:

– bupivacaine 0.167% plus diamorphine 0.1 mg/mL;

– bupivacaine 0.125% plus fentanyl 4 mg/mL.

Epidural infusions can be used to maintain analgesia for several days. Opioid side–effects are less common and less severe than when given systemically as the dose is much less.

Points to note

–The infusion rate and the site of the catheter determine the spread of the solution. In the thoracic epidural space a starting infusion rate might be 4 mL/h; in the lumbar space commence at 8 mL/h.

–The efficacy of the infusion must be monitored in a similar manner as for PCA.

– If analgesia is inadequate, a 'top–up' of 3–4 mL of solution may be necessary.

– Observations of the patient's vital signs should then be made on a regular basis according to local protocol.

• In patients over the age of 60 years, the concentration of opioid is often halved.

Difficult pain problems

Patients in whom there is evidence of regular opioid use preoperatively, for example drug addicts, cancer and chronic pain patients and those patients with a previous bad pain experience, will pose a particular problem postoperatively. They are best managed using a team approach that will include:

– Liaison with the Acute Pain Team to inform it of the patient's admission.

– Discussion with the anaesthetist, and surgical and nursing staff to plan perioperative care, to:

– ensure any current opioid medication is continued on admission to prevent withdrawal;

– understand that much larger doses of opioids than normal may be required;

– explain that toxicity from high doses of opioid is very unlikely;

– reassure that addiction is not a concern.

– Discussion with the patient to explain:

– types and effectiveness of analgesic regimes available postoperatively;

– that analgesia may not be 100% effective;

– that long–term continuation may be necessary;

– potential side–effects, especially if regional analgesia planned.

– Plan regular reviews during postoperative period.

– Coordination of care.

Discharge of the patient

The anaesthetist's responsibility to the patient does not end with termination of the anaesthetic. Although care is handed over to the recovery staff (nurse or equivalent), the ultimate responsibility remains with the anaesthetist until discharge from the recovery area. If there are inadequate numbers of recovery staff to care for a newly admitted patient, the anaesthetist should adopt this role.

Minimum criteria for discharge from recovery area

– Fully conscious and able to maintain own airway (although patient may still be 'sleepy')

– Adequate breathing

–Stable cardiovascular system, with minimal bleeding from the surgical site

–Adequate pain relief

–Warm

Examples of tascs for determination of knowledge lewel

1. After carrying out anesthesia in the patient with the use of arduan as a myorelaxant, a respiratory depression has occurred as a result of residual curarization. What preparation should be applied?

a) Adrenalin.
b) Euphylline.
c) Proserin.
d) Unithiolum.

2. In the immediate postanesthetic period the patient developed a complication, named as "Mendelson's syndrome". What is the essence of this complication?

a) Reflex cardiac arrest.

b) Ventricular fibrillation.
c) Sharp depression of respiration.
d) Acute exudative pneumonitis.

3. Signs of threatening disturbance of vital functions are:
a) Systolic AP 60 mm Hg.
b) Temperature 39° C.
c) Dyspnea at rest.
d) Coma.

4. The following substances are anticonvulsants:
a) midazolam;
b) suksametonium;
a) nifedipine;
g) atracurium;
d) trifluoperazine.

5. Malignant hyperthermia:
a) develops intraoperatively in operations for more than 3 hours;
b) triggered by succinylcholine;
c) requires the monitoring of body temperature for the diagnosis;
d) more frequent in patients with renal impairment.

6. Acute heart failure at high central venous pressure is not typical for one of the following states:
Possible answers:
a) tension pneumothorax;
b) venous air embolism;
c) heart failure;
d) bleeding.

7. For the state of hypovolemia is not typical:
a) A decrease in blood volume (CBV);
b) reduction of blood pressure, tachycardia;

c) a decrease in stroke volume and cardiac output (SV and CO);

d) increasing the CVP;

8. Insulin resistance in the postoperative period in patients with diabetes may develop under the influence:

a) inadequate local anesthesia;

b) metabolic acidosis;

c) use of glucocorticoids;

d) all of the above.

9. Mechanical hyperventilation in the normal patient during anesthesia will lead to:

a) marked decrease in postoperative analgesia requirements;

b) shifting oxyhemoglobin dissociation curve to the right;

c) reduction of PaO_2;

d) postoperative hypoventilation.

10. At early postoperative period hypotension may be more related to:

a) hemorrhage, hypovolemia, or ongoing bleeding;

b) with pain;

c) with an overdose of anesthetic;

d) endocrine insufficiency.

Correct answers:

1.–c; 2.–d); 3.–a),c),d); 4.–a); 5.–b); 6.–d) ;7.–d); 8.–d); 9.–d); 10.–a).

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