Chapter 4

Complications

Respiratory Complications

The function of the respiratory system is the delivery of oxygen to, and the elimination of carbon dioxide from the blood. Any respiratory complication will, if uncorrected, lead to inadequate oxygenation (hypoxaemia) and/or retention of CO_2 (hypercarbia), and these conditions must be readily recognised by recovery staff. The incidence of such problems is greater after lengthy anaesthesia and surgery.

Signs of hypoxaemia

1. Cyanosis. This may be difficult to detect in the presence of anaemia or poor peripheral perfusion
2. Restlessness and confusion. This indicates impaired cerebral oxygenation
3. Tachycardia followed by bradycardia

Signs of hypercarbia

1. Tachycardia
2. Hypertension
3. Sweating
4. Irregular pulse, especially pulsus bigeminus
5. Flushed skin due to capillary vasodilatation. (This may give a mistaken impression of well-being)
6. Clouding of consciousness

The increased use of pulse oximeters allows hypoxaemia to be recognised early before cyanosis develops. The diagnosis may be confirmed by taking a sample of arterial blood for blood gas analysis.

R. Eltringham et al., *Post-anaesthetic Recovery*
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Upper Airway Obstruction

Partial obstruction of the airway may be indicated by:

1. Stertorous breathing i.e. snoring
2. Inspiratory stridor i.e. a crowing noise on inspiration
3. Laboured breathing. Use of the accessory muscles of respiration (sternomastoids, scalenes) with retraction of the head on inspiration and flaring of the nostrils
4. Rocking movements of the abdomen and chest. Instead of the abdomen and chest rising and falling in phase together, downward descent of the diaphragm with abdominal distension is accompanied by retraction or indrawing of the thorax, creating a see-saw or rocking motion of the chest and abdomen (external paradoxical respiration). This becomes more marked as the degree of obstruction increases

If respiratory obstruction is complete:

1. No movement of air is detectable at the airway
2. There are no breath sounds
3. Signs of hypoxia rapidly develop
4. Dysrhythmias and bradycardia occur

It is important to note that movements of the chest are not synonymous with a clear airway; indeed, excessive chest movements may occur in the presence of complete airway obstruction. Remember also that although partial obstruction is accompanied by noisy respiration, total obstruction is silent.

Causes

1. *Tongue.* In the unconscious patient with the jaw relaxed the tongue may fall back and obstruct the airway.
2. *Foreign material in the pharynx*
   a) Excess mucous or saliva
   b) Gastric contents from vomiting or regurgitation
   c) Blood following oral or nasal surgery
   d) Broken or dislodged teeth
   e) Dental packs not removed before extubation.
3. *Laryngospasm.* Resulting from stimulation of the larynx during emergence from anaesthesia. This may be caused by foreign material (as above) or by clumsy extubation or suction.

The following are less common but nevertheless potentially lethal:

4. *Laryngeal oedema* following trauma, intubation or infection. This is especially dangerous in the young, when the airway is of narrow diameter.

5. *External pressure on the trachea.*
   a) Haematoma following thyroid surgery or following attempts at internal jugular cannulation
   b) Use of constrictive Elastoplast bandages.
6. *Abductor paralysis of vocal cords.* This may occur following damage to the recurrent laryngeal nerve during thyroid surgery (see p. 121).
7. *Tracheal collapse* following thyroidectomy.

Management

1. Extend the neck
2. Lift the jaw forward
3. Insert an oral airway. If the teeth are tightly clenched due to spasm of the masseters, firm pressure on the mandible may be necessary to enable the airway to be inserted. If this fails a nasopharyngeal airway may be inserted to bypass the obstruction. If the obstruction remains unrelieved, foreign material in the pharynx must be suspected. If the patient is not already on his side then:
4. Turn the patient on to his side (preferably his left in case subsequent laryngoscopy becomes necessary)
5. Tilt head down (Trendelenburg position) to help clear any foreign material
6. Apply suction to pharynx with rigid Yankauer sucker or large suction catheter

If these measures are unsuccessful proceed to:

7. Laryngoscopy – so that foreign material can be sucked out under direct vision or removed using Magill forceps. If the larynx is clear but the vocal cords are in spasm:
8. Give oxygen by anaesthetic face mask and Mapleson C circuit (Fig. 2.14). Apply gentle pressure to the reservoir bag to try and overcome the spasm. If this is unsuccessful, give intravenous suxamethonium (succinylcholine) to relax the cords and ventilate the lungs. Endotracheal intubation may be necessary.

Special consideration will be required for the less common causes of stridor (nos. 4–7 above):

Laryngeal oedema. Although this usually resolves spontaneously, preparations for rapid intubation should be made. The following may aid spontaneous resolution:

a) Head up position to improve venous drainage
b) Humidification
c) Steroids
d) Diuretics
e) Inhalation of nebulised adrenaline (racemic epinephrine)

The inhalation of a mixture of helium 80% and oxygen 20% will reduce the resistance to air flow and make breathing easier.

External pressure on the trachea
Abductor paralysis of vocal cords
Tracheal collapse

See complications of thyroid surgery (p. 121)

Inadequate Ventilation (Hypoventilation)

If correction of upper airway obstruction does not lead to the resumption of a normal breathing pattern or if signs of hypoxaemia or hypercarbia develop, then inadequate alveolar ventilation must be suspected. If there is doubt, confirmation can be obtained by:

1. *Attaching a pulse oximeter* to the patient's finger or ear lobe. Usually, the haemoglobin saturation is greater than 95%.
2. *Measuring the respiratory minute volume*, using a Wright's spirometer (normal values should exceed 5 litres/minute). Because of the difficulty of obtaining an airtight fit with a mask, this method may be unsatisfactory in patients who are not intubated.
3. *Analysing blood gases* on an arterial sample. Inadequate alveolar ventilation is characterised by a respiratory acidosis (pH < 7.35, $PaCO_2 > 6kPa$).

Except in an emergency, when intubation and controlled ventilation must be instituted without delay, an attempt should be made to determine the cause of the inadequate ventilation so that specific treatment aimed at correcting this may be undertaken.

Causes

Causes of inadequate ventilation in the immediate post-operative period are:

1. Depression of the respiratory centre/Cheyne-Stokes respiration
2. Residual muscle paralysis
3. Interference with the mechanics of respiration

Depression of Respiratory Centre

Depression of the respiratory centre may be due to:

1. Drugs
   a) Opiates given before or during anaesthesia
   b) Barbiturates
   c) Inhalation agents

2. Lack of respiratory drive
   a) Low $PaCO_2$ following hyperventilation during anaesthesia
   b) Loss of hypoxic drive due to administrations of high concentrations of oxygen to patients suffering from chronic pulmonary disease

Central depression may be suspected by:

1. Delayed return of consciousness. Patients should normally show signs of returning consciousness within 15 min of arrival in the recovery room
2. Respiratory rate below 10 breaths/minute with a low tidal volume
3. Constricted pupils following opiate administration
4. History of chronic pulmonary disease

Management. If central depression due to opiates is suspected, this may be corrected by either of the following:

1. Intravenous naloxone, 0.1–0.4 mg. This drug is a specific opiate antagonist and is therefore ineffective in other forms of respiratory depression. It should be given slowly in increments of 0.1 mg every 2–3 min and its effect titrated against the patient's respiratory response. Excessive doses will reverse not only the respiratory depression but also the analgesic effect of the opiates, causing the patient unnecessary pain. To extend the duration of action, subsequent injections can be given by the intramuscular route.
2. Intravenous doxapram, 1 mg/kg. This drug is a direct stimulant of the respiratory centre and has advantages over naloxone as it is effective for other causes of central respiratory depression and does not reverse analgesia.

Since the depressant effects of the opiates may outlast either of these antidotes, they may have to be repeated.

If there is a history of pulmonary disease, graded concentrations of oxygen should be given using a Venturi mask and progress monitored by repeated blood gas analysis.

If the situation does not improve, it is safer to intubate the trachea and electively ventilate the lungs until the effects of anaesthesia and surgery have worn off.

Cheyne-Stokes Respiration

This is an irregular pattern of respiration characterised by periods of hyperventilation alternating with hypoventilation. It is more commonly seen in elderly patients. Causes of Cheyne-Stokes respiration are:

1. Left ventricular failure
2. Depression of respiratory centre
3. Raised intracranial pressure
4. Uraemia

Management. This condition may be aggravated by the administration of sedative drugs which should be used with caution in the recovery period.

An underlying condition should be sought and treated if possible. If respiration becomes inadequate, intubation and controlled ventilation will be required.

Residual Paralysis

Residual paralysis may be due to the continued action of muscle relaxants given during anaesthesia causing neuromuscular block of either the depolarising or non-depolarising type. Residual paralysis should be suspected if there is:

1. Laboured breathing – use of accessory muscles of respiration (extension of neck on inspiration)
2. Rapid shallow breathing with minimal chest movement
3. Flaring of the nostrils and/or raising of the eyebrows
4. Unexplained restlessness
5. The patient whispers that he cannot breathe properly
6. Tracheal tug (downward movement of trachea and thyroid cartilage on inspiration)

Simple bedside tests to indicate residual paralysis

1. Ask the patient to grip your hand, raise his head from the pillow or protrude his tongue for several seconds.
2. Measure his vital capacity: normal value should exceed 10 ml/kg

The type of treatment will depend on whether the residual paralysis is due to a depolarising (phase I) or non-depolarising (phase II) block. A knowledge of the type, quantity and timing of the muscle relaxants given during anaesthesia will usually clarify this but if doubt remains additional information may be obtained from:

1. *Peripheral nerve stimulation.* If a train of four supramaximal stimuli at a frequency of 2 Hz is applied to the ulnar nerve, contraction of the hand muscles will result. This is painful for the conscious patient and should not be applied more frequently than is essential. Fade with successive stimuli confirms non-depolarising block (Fig. 4. 1). Significant paralysis is indicated if the ratio of the fourth to the first response is less than 50%.
2. *Edrophonium test.* An intravenous injection of the short-acting anticholinesterase drug edrophonium will increase muscle strength if the patient has a non-depolarising block. It is unwise to use the longer-acting neostigmine as it will exacerbate a depolarising block.

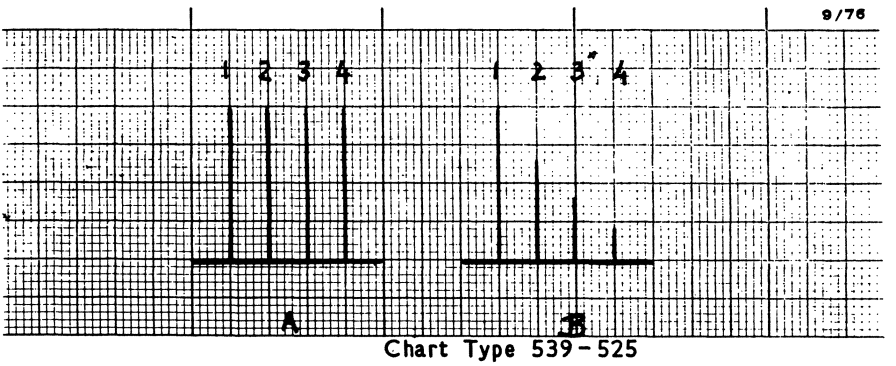

Fig. 4.1. Results of train of four stimulation. A, normal response; B, fade of successive stimuli

Depolarising Block (Phase I Block)

Suxamethonium (succinylcholine) is normally metabolised by cholinesterase in the blood within 5–10 min of administration with the resumption of spontaneous respiration. Paralysis is prolonged in the presence of:

1. *Abnormal cholinesterase.* A rare inherited condition in which there is an impaired ability to metabolise suxamethonium
2. *Reduced amounts of cholinesterase.* Cholinesterase is synthesised in the liver and may be deficient in liver disease or malnutrition
3. *Concurrent administration of anticholinesterases,* e.g. ecothiopate (phospholine iodide) used in the treatment of glaucoma

Management of Phase I Block. If, following the administration of suxamethonium (succinylcholine), spontaneous respiration has not returned by the time the patient arrives in the recovery unit, assisted ventilation must be continued. Spontaneous respiration is normally resumed within 2 hours but can be expedited by the administration of cholinesterase in the form of fresh frozen plasma.

Before the patient leaves hospital, blood should be taken for estimation of cholinesterase level and dibucaine number. If abnormal cholinesterase is demonstrated by a low dibucaine number (normal 80%), the family practitioner must be notified and other members of the family investigated as they may also be affected.

Non-depolarising Block (Phase II Block)

Non-depolarising block may be due to:

1. *Excessive administration of non-depolarising relaxants* in relation to the patient's size and the duration of surgery. During hypothermia there is

resistance to the non-depolarising relaxants and large quantities are required to produce a block. On re-warming, signs of overdose may become apparent when normal sensitivity is restored.

2. *Sensitivity to relaxants,* e.g. in patients with myasthenia gravis
3. *Potentiation of relaxants* due to:
   a) Hypokalaemia (diuretic therapy, prolonged pre-operative bowel wash-out)
   b) Acidosis (vomiting, blood transfusion, hypotension)
   c) Administration of large quantities of antibiotics, especially streptomycin and related aminoglycosides, e.g. polymyxins, tetracycline, lincomycin
   d) Hypocalcaemia
4. *Impaired excretion or metabolism of relaxants* (kidney or liver disease)
5. *Excessive administration of depolarising relaxants.* When the amount of suxamethonium (succinylcholine) administered exceeds 300 mg the depolarising block (phase I) may develop into a non-depolarising (phase II) block

Management of Phase II Block

1. Administer intravenous neostigmine (preceded by atropine or glycopyrolate) to a total dose, including that given at the end of surgery, of 0.08 mg/kg. If this does not reverse the neuromuscular block, controlled ventilation is continued while further attempts are made to determine the cause.
2. Take blood for blood gas and electrolyte estimation.
3. Correct metabolic acidosis with intravenous sodium bicarbonate using the formula: base deficit × body weight in kg × 1/3 = mmol of sodium bicarbonate required. This is normally given in several increments and the effect measured by serial blood gas estimations.
4. Correct hypokalaemia by intravenous potassium chloride. Up to 20 mmol of a dilute solution may be given per hour with continuous ECG monitoring.
5. If hypocalcaemia is suspected following massive transfusion of stored blood, or if large quantities of antibiotics have been given, 10 ml of 10% calcium chloride intravenously may correct the situation.

Conditions Affecting the Mechanics of Respiration

Inadequate ventilation may occur post-operatively if respiratory movements are impaired by:

1. Pain from a high abdominal or thoracic incision
2. Obesity (p. 158)
3. Tight abdominal or thoracic strapping
4. Pneumothorax or haemothorax

Management

1. Give oxygen by face mask to increase the inspired oxygen concentration (F_1O_2)
2. Sit patient up to lessen pressure on diaphragm
3. Ensure adequate analgesia
4. Encourage deep breathing by means of physiotherapy

If the history or clinical findings suggest pneumothorax or haemothorax, an X-ray of the chest should be taken and appropriate management instituted (p. 78).

Progress can be monitored by serial blood gas estimation. If there is no improvement with the above measures, intubation and controlled ventilation will be required.

Hypoxaemia

Causes

In addition to the hypoxaemia resulting from generalised underventilation of the lungs and a reduced respiratory minute volume (p. 70), it may also be caused post-operatively by the following:

1. *Diffusion hypoxaemia* (Fink effect). In the first few minutes after nitrous oxide is discontinued, it comes out of solution in the blood and diffuses into the alveoli. The concentration of oxygen is, therefore, reduced below normal if the patient is breathing room air. Oxygen should always be administered after nitrous oxide is discontinued.
2. *Increased oxygen utilisation* accompanying shivering, convulsions, pyrexia, thyroid crisis
3. *Ventilation perfusion (V:Q) imbalance* caused by regional underventilation. Some alveoli continue to receive a normal blood supply but are not adequately ventilated. This occurs if there is atelectasis due to:
   a) Absorption collapse distal to an obstruction caused by plugs of mucus or inhalation of foreign material
   b) Surgical compression of the lung during thoracotomy
   c) Airway closure at the bases due to pain causing restricted movements
   d) Pneumonia
   e) Pulmonary oedema

Management

1. Administer oxygen by face mask to increase the proportion of inspired oxygen (F_1O_2). This is particularly important in the elderly and in those with a reduced cardiopulmonary reserve

If atelectasis is the likely cause:

2. Encourage deep breathing and arrange regular physiotherapy
3. Ensure there is adequate analgesia
4. Monitor progress by serial blood gas estimations

Bronchospasm

In spontaneously breathing patients, bronchospasm is characterised by dyspnoea and wheezing, especially during expiration. In patients being ventilated, an increased airway pressure is required to inflate the lungs (decreased compliance).

Causes

1. *Predominance of parasympathetic tone*, e.g. following neostigmine or non-selective β-blockers such as propranolol
2. *Irritation of larynx* during emergence from anaesthesia, e.g. by secretions, gastric contents, endotracheal tube, suction catheter. Chronic bronchitics and smokers are especially prone to this problem
3. *Anaphylactoid reactions* due to histamine release following administration of drugs such as *d*-tubocurarine and Haemaccel or following blood transfusion
4. *Asthma*

Management

1. Administration of oxygen
2. Bronchodilators, e.g. aminophylline 250–500 mg intravenously given slowly to minimise tachycardia *or* salbutamol (albuterol) either intravenously or via nebuliser (see Fig. 4.2)
3. Hydrocortisone 100 mg i.v. to reduce mucosal swelling

If bronchospasm and dyspnoea persist despite these measures:

4. Intubate and ventilate the lungs

Aspiration of Gastric Contents

During recovery from general anaesthesia the laryngeal reflex may be depressed. Therefore, if vomiting or regurgitation occurs, gastric contents may be aspirated into the trachea and lungs. To reduce this risk patients recovering from

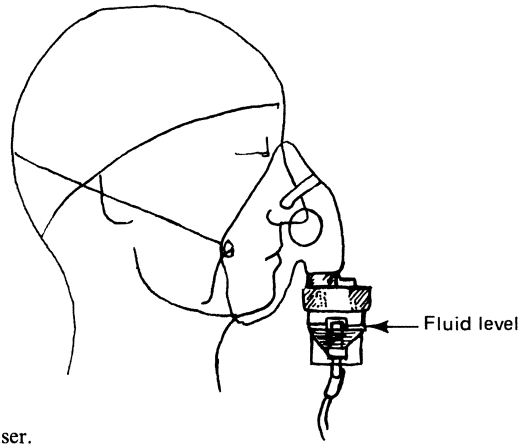

Fig. 4.2. Drug administration via nebuliser.

anaesthesia are normally nursed on the side so that regurgitated gastric contents do not pool in the posterior pharynx but are cleared with the aid of gravity.

If vomiting occurs during the recovery period:

1. *Turn patient on to side* if not already in this position. The left side is preferable since if the patient is supine, inhaled material may normally enter the right lung and drainage will be facilitated with this lung uppermost. Also subsequent laryngoscopy is easiest with the patient on the left side
2. *Tilt bed head down* (Trendelenburg position)
3. *Apply suction to pharynx.* Laryngoscopy and Magill forceps may be required to remove solid material
4. *Give oxygen by face mask*

If aspiration has occurred:

5. *Intubate* and give 100% oxygen
6. *Apply suction* to the trachea and main bronchi using a fine catheter via the endotracheal tube
7. *Consider bronchoscopy* if hypoxia persists or if solid material has been inhaled
8. *Give bronchodilators* as required to relieve bronchospasm (see p. 76)
9. *Administer hydrocortisone* 100–500 mg i.v. to reduce mucosal swelling
10. *Encourage coughing* and arrange vigorous physiotherapy in an attempt to clear the lungs

Antibiotics are not usually recommended at this stage, as gastric contents are usually sterile, but may be added if pyrexia develops or when the results of sputum culture become known. An early X-ray of the chest may provide a baseline for subsequent comparison.

Chemical Pneumonitis (Mendelson's Syndrome)

If the gastric contents are highly acid (pH < 2.5), as may occur during labour, aspiration into the lung may also cause chemical pneumonitis.

Aspiration of small volumes of acidic gastric contents may be silent and the signs of chemical pneumonitis may not develop for several hours. It must be suspected if a patient develops tachypnoea, cyanosis, tachycardia and wheezing in the recovery period. The chest X-ray may show diffuse opacities over the affected area, often the right base, thus confirming the diagnosis.

These patients should be transferred to an intensive care unit for management as severe respiratory difficulties may subsequently occur. The recovery room nurse must always record suspected regurgitation and vomiting.

Pneumothorax and Haemothorax

Pneumothorax (the presence of air in the pleural cavity) may become evident during recovery from anaesthesia. It is suggested by:

1. Chest pain
2. Dyspnoea
3. Cyanosis
4. Diminished air entry on affected side
5. Pulsus alternans

A chest X-ray will confirm the diagnosis.

Causes

1. Damage to pleura following surgery or trauma
2. Accidental pleural puncture following intercostal or supraclavicular brachial plexus block or during attempts at internal jugular or subclavian vein cannulation
3. Alveolar rupture during intermittent positive pressure ventilation or the spontaneous rupture of an emphysematous bulla

Management

A small pneumothorax unaccompanied by clinical features may resolve spontaneously and not require treatment.

A larger pneumothorax requires the insertion of a chest drain with underwater seal. A suitable site is the second interspace in the mid-clavicular line.

N.B. The administration of nitrous oxide in the presence of a pneumothorax will increase its size and should be avoided.

If the chest X-ray demonstrates the presence of fluid in the pleural cavity (haemothorax, pleural effusion), this should be released by a chest drain placed in the eighth space in the posterior axillary line.

Tension Pneumothorax

If the pneumothorax is under tension, the signs will be more acute. In addition there will be:

1. Cardiovascular collapse due to diminished venous return
2. Deviation of the trachea and displacement of the apex beat away from the affected side
3. Increasing difficulty in inflating the lungs in the ventilated patient

Management

Urgent insertion of a chest drain (see p. 128). In an emergency, the increased intrapleural pressure can be reduced rapidly by inserting a large-bore intravenous cannula into the second intercostal space while equipment is prepared for a more formal procedure.

Cardiovascular Complications

Hypotension

The post-operative blood pressure should not be considered in isolation but as part of a trend and interpreted in relation to the pulse rate and to other findings such as the pre-operative value and the state of the peripheral perfusion. For example, a reduction of 30 mmHg in the systolic pressure of a hypersensitive patient may be a significant fall yet still be within the normal range. On the other hand, in the presence of good peripheral perfusion, a blood pressure below the normal range may be satisfactory following certain anaesthetic techniques (p. 143).

A reduced blood pressure is frequently due to the continuing action of drugs used during anaesthesia (Table 4.1) and will usually revert to normal as the agents are eliminated. While patients are recovering from the effects of these agents, vasomotor tone may be impaired and with it the patient's ability to compensate for sudden changes in posture. A temporary fall in blood pressure may follow transfer of the patient from the operating table or the lowering of

Table 4.1 Drugs used during anaesthesia causing hypotension

1. *Myocardial depression*
   Inhalational anaesthetic agents, e.g. halothane, enflurane
   β-blocking agents, e.g. propranolol, labetolol
   Intravenous induction agents, e.g. thiopentone, propofol
2. *Diminished peripheral vascular resistance*
   Inhalational anaesthetic agents, e.g. isoflurane
   Sympathetic blockade:
   a) Ganglion-blocking agents, e.g. trimetaphan
   b) Local anaesthetic agents used for spinal or epidural anaesthesia
3. *Vasodilating drugs*: nitroglycerine, nitroprusside, chlorpromazine, droperidol, opiates

the legs from the lithotomy position and patients should not sit up until the effects of these agents have worn off.

If, on admission to the recovery unit, the systolic blood pressure is below 100 mmHg systolic, the patient's progress should be monitored carefully until normal values are restored. If peripheral perfusion is impaired, hypovolaemia and diminished cardiac output must be excluded.

Hypovolaemia

Hypotension and poor peripheral perfusion are accompanied by increasing heart rate, pallor, collapsed veins (a low central venous pressure reading will confirm this) and oliguria (urine output $< 0.5\,ml/(kg \cdot h)$).

Causes

1. Inadequate fluid replacement following pre-operative dehydration or prolonged surgery with bowel exposure
2. Inadequate replacement of blood

Management

1. Give oxygen by face mask to increase the percentage of oxygen inspired (F_1O_2)
2. Elevate foot of bed
3. Increase rate of intravenous infusion

N.B. Vasoconstricting agents are not recommended in the presence of hypovolaemia since they will further decrease tissue perfusion.

If there has been haemorrhage, a blood transfusion may be necessary. Until cross-matched blood is ready, the following substitutes may be used:

1. Haemaccel (a modified gelatin)
2. Hespan (a modified starch)
3. Human plasma protein fraction (HPPF)
4. Dextran 70. If cross-matching has not already been undertaken, a sample of blood should be taken first as dextran may interfere with subsequent cross-matching techniques

If hypotension, pallor and collapsed veins persist despite these measures then continued bleeding must be suspected. This may be either:

1. *Revealed*, e.g. blood in drainage bottles, bladder irrigation or on dressings and packs
2. *Concealed*, e.g. intra-abdominal

In either case the transfusion must be continued and the surgeon and anaesthetist notified as further surgery may be required. Alternatively, persistent bleeding may be due to a failure of coagulation and this may be suspected if a sample of blood in a plain tube does not clot within 10 min (see p. 104). Blood should then be taken for a formal coagulation screen.

Diminished Cardiac Output

If hypotension and poor peripheral perfusion are not due to hypovolaemia then a diminished cardiac output must be considered. This may be indicated by distended veins, a raised CVP and a falling pulse pressure. The last is the difference between the systolic and diastolic pressure and is normally about 40 mmHg.

Causes and Management

1. *Cardiac failure*. A review of the patient's previous medical history and the intra-operative fluid balance may suggest this possibility. Breathlessness, tachycardia and pulmonary and peripheral oedema may confirm it. Management will include:
   a) Oxygen therapy
   b) Posture (reverse Trendelenburg position)
   c) Diuretics
   d) Fluid restriction
   e) Inotropic support (dopamine, dobutamine, digoxin)
   f) Intubation and intermittent positive pressure ventilation (in extreme cases only)
2. *Myocardial infarction*. There may be a history of myocardial ischaemia or the classical description of chest pain may be present. However, infarction

can occur silently in patients with no previous history of cardiac disease. A 12-lead ECG should be performed and blood taken for enzyme studies. Although these may not contribute to the immediate management of the patient, they may provide a valuable baseline for future reference.

3. *Pulmonary embolus.* Although this is uncommon in the immediate post-operative period, pleuritic chest pain and haemoptysis may suggest this diagnosis. A chest X-ray and full ECG should be obtained. Treatment includes oxygen therapy, pain relief and heparinisation.
4. *Cardiac tamponade.* This may follow surgery or trauma in the region of the mediastinum or, rarely, perforation of the myocardium by a central venous or pulmonary artery catheter. Faint heart sounds and a rising CVP accompanied by a falling blood pressure may suggest this possibility. Widening of the mediastinum on chest X-ray will provide confirmation. Emergency treatment is by needle aspiration from below the xiphisternum but a formal thoracotomy may be necessary.
5. *Tension pneumothorax* (see p. 79).

Other Causes of Hypotension

If hypovolaemia and diminished cardiac output have been excluded and the cause of hypotension remains obscure, the following should be considered:

1. *Septicaemia.* Especially following bowel or urological surgery. This may be suspected by pyrexia, tachycardia, flushing, sweating or delirium. Management includes vigorous intravenous therapy to achieve and maintain normal venous pressure, and antibiotic and steroid therapy.
2. *Inadequate steroid cover.* For patients on long-term steroid therapy or with undiagnosed Addison's disease. Initial management will consist of fluid replacement and steroid therapy (p. 145).
3. *Mismatched blood transfusion.* Hypotension may accompany other signs of mismatched transfusion, such as pyrexia, urticaria, flushing, shivering, haematuria and persistent bleeding. The transfusion should be stopped immediately and samples of the transfused blood and patient's blood sent to the laboratory for further investigations (see p. 102).
4. *Pain.* Usually causes hypertension but hypotension is sometimes seen and may respond to the administration of analgesics.

Hypertension

Like pre-operative hypertension, this is not uncommon in the immediate post-operative period.

Causes

1. Pain
2. Distension of bladder
3. Respiratory depression with hypercarbia
4. Overtransfusion
5. Cardiovascular surgery
6. Drugs used during anaesthesia, e.g. ketamine, methoxamine, ephedrine
7. Underlying condition, e.g. phaeochromocytoma, hyperthyroidism, raised intracranial pressure, pre-eclamptic toxaemia

Management

Once pain, bladder distension and respiratory complications have been treated, the hypertension usually reverts to normal within 2 hours without the need for specific treatment. However, in those with coronary artery or cerebrovascular disease, excessive hypertension may cause myocardial ischaemia or cerebral haemorrhage and active management is required. Similarly, following vascular surgery hypertension will cause an unnecessary strain on the graft and should be avoided.

Numerous drugs are available to reduce blood pressure, for example:

1. Alpha-blocking agents, e.g. phentolamine, chlorpromazine
2. Ganglion-blocking agents, e.g. trimetaphan
3. Drugs acting directly on peripheral vessels, e.g. sodium nitroprusside nitroglycerine, hydralazine

In the presence of an accompanying tachycardia a combined α- and β- blocking agent such as labetalol may be useful.

It is important to exclude any of the underlying conditions which will require specific treatment, although in 30% of the patients no obvious cause can be found.

Bradycardia

Sinus Bradycardia

A heart rate of less than 60 per minute is normal in fit athletes but may signify an underlying condition requiring correction.

Causes

1. Continuing action of drugs used before or during anaesthesia, e.g. opiates, neostigmine, β-blocking agents (even when used as eye-drops), digoxin

2. High sympathetic blockade following spinal or epidural anaesthesia
3. Parasympathetic stimulation due to pain or pharyngeal suction.
4. Hypoxia
5. Raised intracranial pressure
6. Decreased metabolic rate due to hypothermia, hypothyroidism
7. Acute gastric dilatation.

Management

Intravenous atropine (0.5–2 mg) will usually correct any excessive parasympathetic tone or, alternatively, ephedrine may be used if there is an accompanying sympathetic blockade with hypotension.

If there is no response and hypoxia and raised intracranial pressure can be excluded, an ECG is required to differentiate between sinus bradycardia and heart block. In sinus bradycardia the PR interval is normal, i.e. less than 0.2 s (Fig. 4.3).

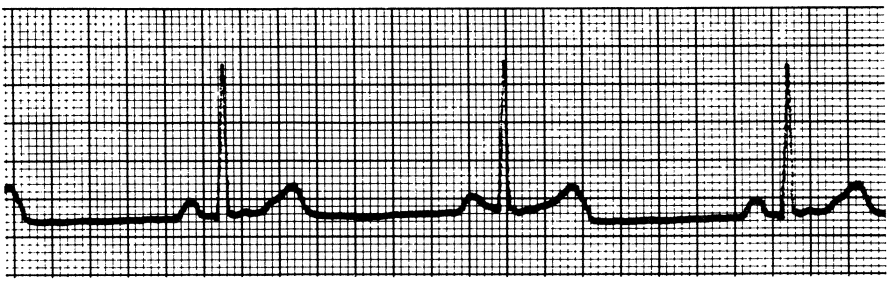

Fig. 4.3. Sinus bradycardia

Heart Block

Three types of heart block are identified on ECG.

1st degree heart block: PR interval greater than 0.2 s

2nd degree heart block: Mobitz type I (Wenckebach phenomenon). Gradual lengthening of PR interval until a dropped beat occurs. Mobitz type II. There is a failure of conduction so that only every 2nd or 3rd atrial impulse is conducted to the ventricles (2:1 or 3:1 block)

3rd degree heart block: Complete AV dissociation. No atrial impulses are conducted to the ventricles, which beat independently at a slow rate (Fig. 4.4)

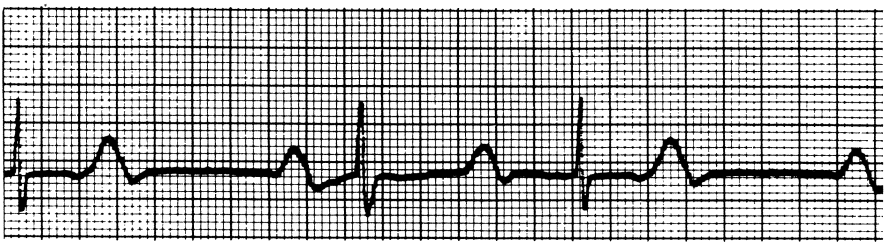

Fig. 4.4. Third degree heart block

Causes

1. Myocardial infarction
2. Drugs depressing myocardial conduction, e.g. digitalis, disopyramide, β-blocking agents

Management

Symptoms due to heart block, e.g. dizziness, fainting and poor peripheral perfusion, can frequently be treated by the infusion of a dilute solution of isoprenaline (isoproterenol) 4 mg in 500 ml 5% dextrose given via a paediatric infusion set. If this fails a pacemaker must be inserted.

Tachycardia

Sinus Tachycardia

There is a normal sinus rhythm but with a rate of over 100 per min.

A tachycardia is normal in infants and small children. Rates of up to 150 per min are well tolerated in patients with normal cardiac function and seldom require treatment but problems may arise in those with underlying heart disease because of the increased myocardial oxygen consumption and diminished stroke volume.

Causes

1. Pain
2. Respiratory problems causing hypercarbia or hypoxia
3. Circulatory disturbance, e.g. hypovolaemia, hypervolaemia
4. Infection
5. Drugs, e.g. atropine, ephedrine, adrenaline (epinephrine), ketamine

6. Anxiety
7. Underlying condition, e.g. hyperthyroidism, phaeochromocytoma

Management

Consists of treatment of the underlying cause.

1. Administer analgesics as required to relieve pain
2. Assess respiratory function using blood gas analysis if necessary. For treatment of inadequate ventilation, see p. 70
3. Assess circulation for signs of hypovolaemia or hypervolaemia:
   a) Hypovolaemia: cold clammy skin, thready pulse, collapsed veins, hypotension. Treat by fluid replacement
   b) Hypervolaemia: strong pulse, distended veins, normal or high blood pressure. Treat by fluid restriction, diuretics.
4. Give reassurance and add anxiolytics as required

Supraventricular and Ventricular Tachycardia (SVT and VT)

A heart rate in the region of 150–250 per min suggests either supraventricular or ventricular tachycardia. These may be accompanied by dizziness, palpitations, angina or chest pain and, if untreated, may lead to circulatory failure.

Causes

1. Hypoxia
2. Hypercarbia
3. Electrolyte disturbance, especially hypokalaemia
4. Acidosis
5. Coronary artery disease
6. Thyrotoxicosis

Management

1. Correct underlying cause if possible
2. Connect ECG monitor in order to distinguish supraventricular from ventricular tachycardia

Supraventricular tachycardia is characterised by normal QRS complexes. P waves may be abnormal or obscured by the T wave of the preceding complex (Fig. 4.5). Its management consists of:

1. Increasing vagal tone by carotid sinus massage, Valsalva manoeuvre or pressure on the eyeball

2. Verapamil 5-10 mg i.v.
3. Cardioversion using a synchronised DC shock. Further anaesthesia is necessary if consciousness has returned

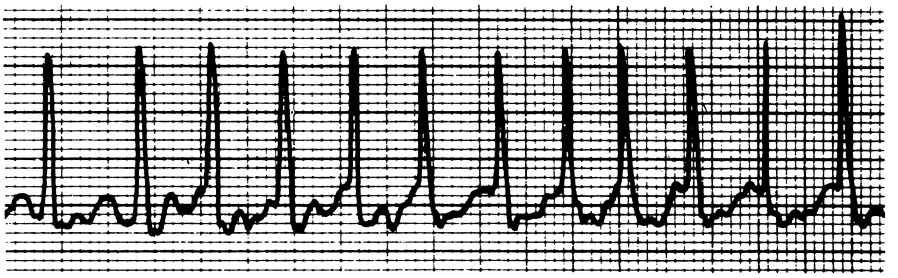

Fig. 4.5. Supraventricular tachycardia

Ventricular tachycardia is characterised by wide and abnormal QRS complexes and absence of P waves (Fig. 4.6). Treatment as listed below should be instituted without delay as ventricular fibrillation may follow.

1. Intravenous lignocaine (lidocaine) 1 mg/kg. If a ventricular tachycardia returns or there are frequent ventricular ectopic beats, a lignocaine infusion may be needed.
2. Flecainide 2 mg/kg over 20 min.
3. Cardioversion

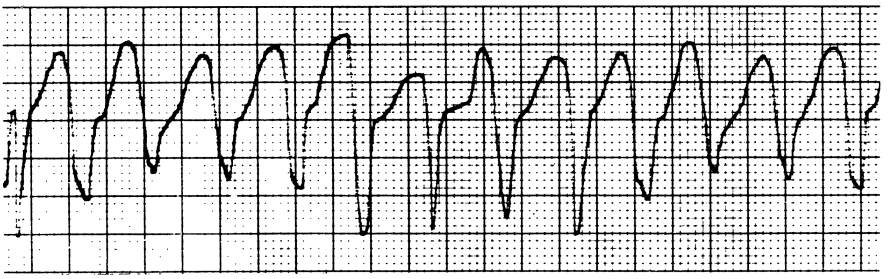

Fig. 4.6. Ventricular tachycardia

Dysrhythmias

An irregular pulse in the immediate post-operative period is not an uncommon finding, especially in children.

If the colour is good with the peripheral circulation satisfactory and the blood pressure maintained, no immediate treatment is required. The irregularity is

probably due to residual effects of inhalational agents sensitising the myocardium to catecholamines and will pass off as the anaesthetic is eliminated.

If the irregularity persists or is accompanied by hypotension or poor peripheral perfusion, its nature should be established by ECG monitoring.

Although any type of dysrhythmia can occur post-operatively, premature atrial and ventricular contractions and atrial fibrillation are most commonly seen.

Premature Atrial Contractions (PACs)

A premature P wave is followed by a normal QRS complex (Fig. 4.7). This dysrhythmia usually causes no problems and no treatment is required.

Premature Ventricular Contractions (PVCs)

There is no P wave before a premature QRS complex. The complex is abnormally wide, notched or large and followed by a compensatory pause (Fig 4.8). If premature contractions follow each normal contraction, the term pulsus bigeminus is used.

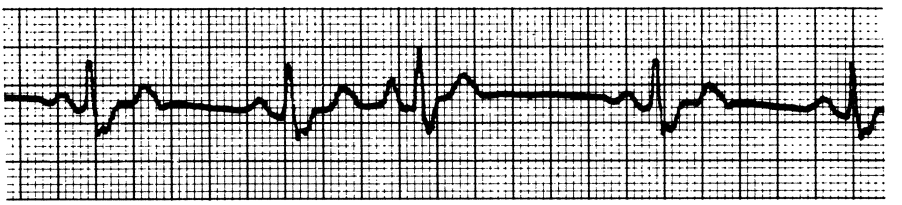

Fig. 4.7. Premature atrial contractions

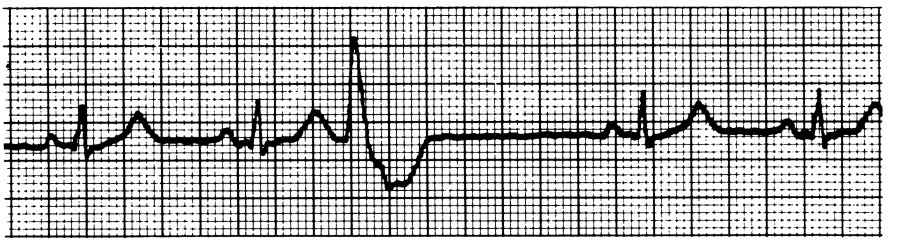

Fig. 4.8. Premature ventricular contractions

Causes

1. Hypoxia
2. Hypercarbia
3. Acidosis
4. Hypokalaemia
5. Digitalis overdose
6. Excess circulating catecholamines
7. Hyperthyroidism

Management

If the premature beats are infrequent and there is no accompanying hypotension, no treatment is required. If, however, they occur frequently (i.e. more than 5 per min), the underlying cause should be sought and corrected, since it may lead to ventricular tachycardia or ventricular fibrillation.

Treatment is by intravenous lignocaine (lidocaine) in a bolus of 1 mg/kg followed by an intravenous infusion at a rate of 1–4 mg/minute. Alternatively a β-blocker such as propranolol may be given in 1 mg increments.

Atrial Fibrillation

Absent P waves. Irregularly occuring QRS complexes (Fig. 4.9). Usually occurs as a result of a long-standing condition, e.g. mitral stenosis or coronary artery disease.

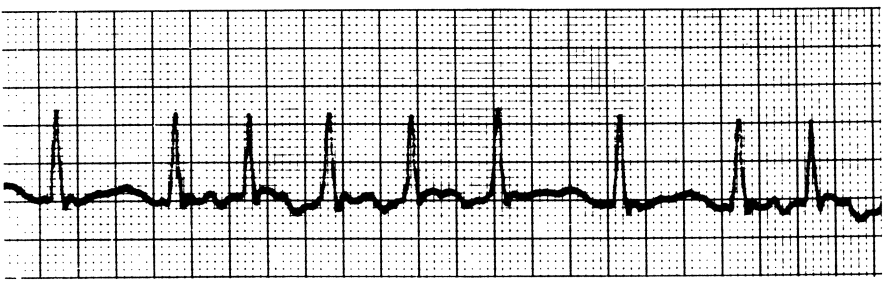

Fig. 4.9. Atrial fibrillation

Management

No treatment is required unless the ventricular response is rapid and the pulse rate exceeds 120 per minute or there are accompanying signs of heart failure or hypotension. The following should then be considered:

1. Cardioversion, using a synchronised D.C. shock
2. Digitalisation. This should not precede cardioversion, as the latter may precipitate a cardiac arrest in the digitalised patient

Many patients with atrial fibrillation are already digitalised prior to surgery so that neither cardioversion nor further digitalisation are appropriate. In this case a slow intravenous injection of disopyramide over 5 min to a total of 2 mg/kg or until the ventricular rate drops may be successful.

Cardiac Arrest

Cardiac arrest occurring in the recovery room should be treated vigorously and has every prospect of success because:

1. The precipitating cause is usually reversible, e.g. hypoxia, electrolyte imbalance, hypovolaemia
2. The patient is under continuous observation and there may be some advance warning, e.g. hypotension, cyanosis, bradycardia
3. Resuscitation equipment and trained staff are instantly available

The diagnosis is made when there is:

1. Loss of consciousness
2. No breathing
3. No pulse palpable in a major artery e.g. carotid or femoral artery

Examining the pupils, listening for heart sounds or connecting an ECG monitor are unnecessary at this stage. If no major pulse is palpable, cardiopulmonary resuscitation must be commenced without delay.

Initial Management

1. Establish a clear airway.
2. Commence artificial ventilation using (a) mouth-to-mouth respiration if no equipment available, (b) a bag and mask or Ambu bag with 100% oxygen or (c) intubation with cuffed tube as soon as possible. Give two large breaths ensuring that the lungs are seen to inflate.
3. Commence external cardiac massage by giving vigorous downwards thrusts on the lower third of the sternum with the palms of the hands at a rate of 80 per minute. After every 15 compressions of the heart, pause and give two inflations of the lungs. At this rate, there will be about 60 compressions of the heart each minute at a rate of 15 compressions to 2 inflations. This cycle must be continued until a spontaneous heartbeat is established.
4. Elevate foot of bed to improve venous return.

Subsequent Management

5. Establish an intravenous infusion if this is not already present so that all drugs can be given intravenously.
6. Montior ECG to establish the electrical activity of the heart. If ventricular fibrillation is shown (Fig. 4.10):

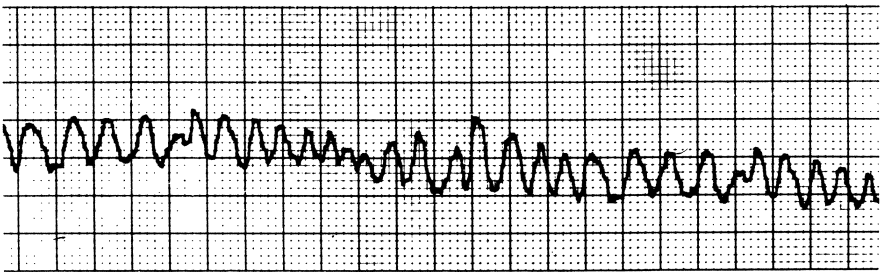

Fig. 4.10. Ventricular fibrillation

a) Defibrillate heart using D.C. defibrillator with paddle placed across heart (Fig 4.11). Start with 200 joules. If no pulse is palpable immediately after the first shock, continue cardiac massage whilst the defibrillator is re-charged to 200 joules. Give a second shock.
b) If defibrillation fails again, repeat cardiac massage (and ventilation) and then give a third shock of 400 joules.

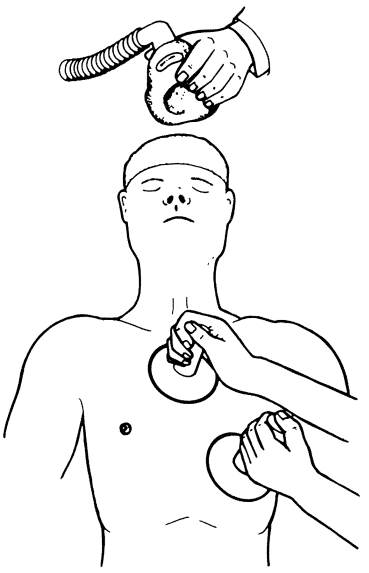

Fig. 4.11. Application of defibrillator paddles. Assistants must stand well clear.

c) If the third shock fails to establish a normal rhythm, give lignocaine 100 mg (10 ml 1% solution or 5 ml 2% solution) followed by a fourth shock of 400 joules.
d) If unsuccessful, give adrenaline (epinephrine) 1 mg (10 ml 1:10 000 solution) followed by a fifth shock of 400 joules.
e) If unsuccessful, give sodium bicarbonate 50 ml 8.4% solution and a sixth shock.

Ventilation and external cardiac massage should only be interrupted when a D.C. shock is about to be administered.

In refractory ventricular fibrillation, bretylium 400 mg may be tried. It has a slow onset, so resuscitation should be continued even if it initially appears to be ineffective.

7. If, instead of ventricular fibrillation, the monitor shows asystole, ventilation and external cardiac massage should be performed as above and the following drugs administered:

a) Atropine 1 mg
b) Adrenaline (epinephrine) 1 mg (10 ml of 1 : 10 000 solution)
c) Isoprenaline (isoproterenol) 100 μg

If they fail to produce ventricular fibrillation or a rhythm with a cardiac output, transvenous or oesophageal pacing should be considered.

8. There is a third mechanism of cardiac arrest: electro-mechanical dissociation. There may be normal or near-normal electrical activity shown on the monitor but it is not accompanied by any useful cardiac output. It has a poor prognosis.

a) Consider and exclude mechanical causes such as cardiac tamponade or tension pneumothorax
b) Adrenaline (epinephrine) 1 mg (10 ml of 1: 10 000 solution) may be helpful *or*
c) Calcium chloride 10 ml of 10% solution

If there is difficulty in inserting an intravenous cannula in a collapsed patient, atropine, adrenaline and lignocaine can be given via the endotracheal tube. Absorption is rapid from the bronchial mucosa. Twice the intravenous dose is usually given. Intracardiac injections are hazardous, offer few advantages and are best avoided.

Once a spontaneous heartbeat has been re-established and any precipitating factors corrected, the patient should be transferred to an intensive care unit so that intensive monitoring and further treatment can be given:

1. Ensure breathing is adequate. Consider a period of artificial ventilation. If a pneumothorax is suspected, insert a chest drain.
2. Estimate arterial blood gases: further bicarbonate may be necessary.
3. Estimate serum potassium.
4. Obtain a chest X-ray.
5. Measure the arterial blood pressure.

6. Insert a urinary catheter and monitor the urine output.
7. Insert a nasogastric tube and aspirate the stomach contents.
8. Insert a central venous catheter if indicated.
9. Obtain a 12-lead ECG.
10. Consider high-dose steroids to protect an ischaemic brain.

Miscellaneous Complications

Delayed Return of Consciousness

Most patients will have regained consciousness within 15 min of arrival in the recovery room. If unconsciousness persists for longer than 30 min a cause should be sought.

Causes

1. Drugs
   a) Relative overdose of drugs
      i) Excess administration, e.g. premedication, opiate supplements.
      ii) Increased sensitivity, e.g. in the elderly, cachectic or hypothyroid patient
      iii) Diminished metabolism, e.g. in liver dysfunction, hypothermia, hypothyroidism
   b) Drugs with prolonged action, e.g. ketamine, droperidol, lorazepam, repeat doses of barbiturates
2. Hypoglycaemia
3. Hypercarbia
4. Metabolic acidosis
5. Cerebral damage, which may result from a period of cerebral hypoxia or from a cerebrovascular accident during anaesthesia
6. Uraemia

Management

1. Ensure that the airway is clear and that respiration is adequate.
2. Reverse depression due to opiates with naloxone.
3. Check blood sugar. Treat hypoglycaemia with 50 ml of 50% glucose intravenously.

4. Check blood gases to exclude hypercarbia or metabolic acidosis. Treat with assisted ventilation or intravenous sodium bicarbonate as appropriate.
5. Consider flumazenil if benzodiazepines have been given.
6. Make a thorough examination of the central nervous system, taking a special note of localising signs, and record the findings as a baseline for future reference.
7. If there is still no response after 2–3 hours and the obvious causes have been treated, the patient should be transferred to an intensive care unit for subsequent management.

Restlessness, Excitement and Delirium

The immediate post-operative period may be accompanied by various degrees of excitement ranging from mild restlessness to violent uncontrolled movement.

Causes

1. Airway obstruction, especially caused by nasal packs (see p. 120)
2. Anxiety
3. Pain
4. Full bladder
5. Inadequate reversal of muscle relaxants
6. Cerebral hypoxia
7. Drugs. Elderly patients in particular can be extremely sensitive to drugs used in premedication, e.g. hyoscine, phenothiazines and barbiturates
8. Middle ear surgery. Restlessness is frequent following this type of surgery, possibly due to a temporary disturbance of the labyrinthine mechanism
9. Raised intracranial pressure
10. Hyperthyroidism
11. Psychological distress, e.g. following termination of pregnancy

Management

1. *Reassurance.* Anxious patients finding themselves in unfamiliar surroundings may become restless. They will frequently settle with gentle handling and sympathetic reassurance.
2. *Restraint.* Care must be taken to prevent the more vigorous patients injuring themselves. Cot sides should be raised. If it is necessary for patients to be restrained, minimal force should be used.

3. *Analgesics.* Administration should be intravenous, as required to relieve pain. Many patients will be unable to complain of pain in the immediate post-operative period. However, in the absence of other obvious causes, restlessness, especially if accompanied by tachycardia and hypertension, will usually respond to analgesic therapy.
4. *Give oxygen by face mask.*
5. *Ensure adequate reversal of relaxants,* giving further atropine and neostigmine if required. If ventilation remains inadequate, intubation and assisted ventilation may be needed.
6. *Reverse excessive premedication* due to above mentioned drugs by intravenous physostigmine 1–3 mg.
7. *Catheterise the bladder* if it is distended.
8. Following trauma or neurosurgery, raised intracranial pressure must always be considered, especially if the restlessness is accompanied by hypertension and bradycardia. If this is the case, a *neurological observation chart* should be begun at once and further advice sought.

Nausea and Vomiting

With improvements in anaesthetic drugs and techniques over the years, the incidence of post-operative vomiting has undoubtedly decreased. However, as one can never be absolutely certain that the stomach is empty even in fasted patients, recovery staff must be alert to the possibility of vomiting.

Causes

Various individual factors contribute to the incidence of post-operative vomiting. The results of numerous studies have shown it to be more frequent:

1. In those prone to motion sickness
2. In females rather than males
3. Following the use of ether or cyclopropane
4. With increasing duration of anaesthesia
5. Following the use of opiate medication. The effect is exacerbated with early mobilisation and reduced with the concurrent use of atropine, hyoscine or antihistamines
6. Following episodes of hypoxia or hypotension
7. Following intra-abdominal surgery
8. In the presence of severe pain
9. After middle ear surgery
10. Following the use of nitrous oxide

Management

Because of the danger of respiratory obstruction or aspiration resulting from vomiting, the recovery patient should be nursed on a tipping trolley and with suction apparatus to hand. Unless contraindicated (e.g. because of orthopaedic traction), the patient should normally be on his side to enable vomited material to be cleared from the pharynx by gravity. In the event of vomiting:

1. Tip head down
2. Suck out pharynx
3. Turn patient on to side if not already in this position (normally on left side in case subsequent laryngoscopy is required). This requires the help of an assistant.
4. If respiratory obstruction persists, clear the pharynx under direct vision using a laryngoscope
5. Give oxygen by mask

For treatment of aspiration of gastric contents, see p. 76

When vomiting or nausea persists for more than a brief period and pain can be excluded as a cause, anti-emetics such as prochlorperazine, metoclopramide or droperidol may be given intravenously or intramuscularly. The routine use of anti-emetics, however, is not recommended since persistent vomiting is not a problem in the majority of patients. Prophylactic anti-emetics are given:

1. Where vomiting is particularly undesirable, such as following a perforating eye injury, where it may cause a rise in intra-ocular pressure, or when the jaws have been wired together and suction is difficult
2. If there is a history of post-operative vomiting

   Following anti-emetic therapy, extra-pyramidal effects (e.g. muscle rigidity, restlessness, oculogyric crisis) have sometimes occurred, particularly after repeated doses of long-acting agents. These are very frightening to the patient and can be treated with anti-Parkinson-type drugs such as benzhexol or procyclidine.

Shivering

Shivering is frequently seen during recovery from general anaesthesia and may be so severe as to resemble grand mal epilepsy.

Causes

1. *Inhalational anaesthetic agents.* Shivering is commonest after the use of halothane but may follow other agents. The exact mechanism is unclear, although it has been shown to be unrelated to temperature change

2. *Blood transfusion reactions* (p. 101)
3. *Hypothermia* (p. 98).

Management

Although shivering is usually transient and does not often present a problem the following measures can be taken:

1. *Administration of oxygen* to increase F_1O_2. Shivering increases metabolic rate and so causes excess oxygen demand. Demand may exceed supply unless additional oxygen is administered.
2. *Extra blankets* if the patient is cold. Aluminium foil space blankets can be used if there is significant hypothermia.

Convulsions

Causes

1. *Cerebral irritation* due to:
   a) Trauma
   b) Neurosurgery
   c) A hypoxic episode
   d) Presence of intracranial mass
2. *Febrile convulsions*
   The combination of:
   a) Pre-operative pyrexia
   b) Dehydration
   c) Atropine medication, which reduces sweating
   d) Inhalational anaesthetics, which may interfere with the heat-regulating centre (especially diethyl ether)
   e) Excessive coverings, which may result in convulsions, especially in children
3. *Epilepsy*, particularly if anticonvulsant drugs have been omitted prior to surgery
4. *Drugs*
   a) Local anaesthetics: convulsions may result if the total quantity injected is excessive or if there has been an inadvertent intravascular injection. Special care is required when releasing a tourniquet following a Bier's block and when topping up an epidural in the recovery room in case the tip of the catheter has migrated into a vein

b) Diethyl ether, especially in large doses accompanied by dehydration or fever
c) Enflurane, especially in high concentrations with hyperventilation
d) Methohexitone (methohexital)

5. *Eclampsia*
   Preventative measures against convulsions must be continued into the post-operative period.
6. *Hypoglycaemia*
7. *Dilutional hyponatraemia*
8. *Uraemia*

Management

Regardless of the cause, oxygen is administered and convulsions terminated as rapidly as possible because of the dangers posed by an uncontrolled airway and excessive oxygen demand. Thiopentone (thiopental), diazepam, phenobarbitone (phenobarbital) and sodium valproate (valproic acid) are suitable drugs for this purpose. In extreme cases muscle relaxants, such as suxamethonium, (succinylcholine), intubation and artificial respiration may be needed. Once the convulsions have been controlled, attempts should be made to determine the underlying cause and to correct it.

Hypothermia

Agents used during general anaesthesia may depress the heat-regulating centre in the hypothalamus, causing vasodilatation or impaired shivering. Consequently some fall in temperature is not uncommon post-operatively, but with the use of thermostatically controlled operating theatres this is seldom severe except at the extremes of age.

Causes

1. Prolonged bowel exposure
2. Prolonged surgery in infants (because of their greater tendency to lose heat, see p. 137)
3. Intravenous infusions of large quantities of cold solutions or blood
4. Following deliberate hypothermia employed during cardiac or neurosurgery
5. Hypothyroidism
6. Bladder irrigation with cold fluids

Management

Hypothermia may lead to:

1. Myocardial depression or irritability
2. Metabolic acidosis
3. Altered response to neuromuscular blocking agents
4. Poor respiratory effort and hypoxia, particularly in children

If the temperature is low:

1. Prevent further heat loss by giving extra warmed blankets or wrapping the patient in an aluminium foil space blanket
2. Give intravenous fluids through a warming coil
3. In the case of infants the use of a warmed incubator or overhead heater is recommended
4. Monitor temperature. This should be done continuously if possible or at frequent intervals. The core temperature is a more valuable guide than skin temperature and this can be measured either with a nasopharyngeal, oesophageal or rectal probe. A low reading thermometer may be necessary in extreme cases
5. Keep patient in recovery unit until temperature has risen above 35 °C
6. In neonates hypothermia can interfere with respiratory effort. If this occurs, intubation and assisted ventilation may be required until normothermia has been achieved

Hyperthermia

A moderate rise in temperature (e.g. up to 39 °C) unaccompanied by other signs or symptoms does not in itself constitute a problem. However, the origin shoud be sought since it may require treatment in the recovery room.

Causes

1. *Infection.* This may have been present before surgery or may first become apparent immediately afterwards, especially following bowel or urological surgery
2. *Impaired heat loss.* The combination of pre-operative pyrexia, atropine (which reduces sweating), a high ambient temperature and the use of agents which interfere with the heat-regulating centre (especially diethyl ether) can cause hyperthermia, particularly in children
3. *Pyrogens* introduced during blood transfusion
4. *Malignant hyperpyrexia* (see below)

Management

1. Where infection is considered likely, appropriate antibiotic therapy may be commenced intravenously. If blood culture is contemplated, it should precede the administration of antibiotics.
2. If hyperthermia follows blood transfusion this should be stopped immediately and a sample of the transfused blood saved for analysis (p. 107).
3. Severe hyperpyrexia (>39 °C), especially in children, will result in increased oxygen consumption and carbon dioxide production with metabolic and respiratory acidosis, causing increased demands on cardiac and respiratory function. This may result in cerebral hypoxia and convulsions and should be treated vigorously by:

   a) Active cooling with tepid sponging, ice and fans
   b) Oxygen therapy
   c) Intravenous diazepam as required to control convulsions.

Malignant Hyperthermia

This is a rare condition of unknown aetiology characterised by a rise in temperature of up to 1 °C every 15 min which can be rapidly fatal unless treated immediately. It is often triggered off by anaesthesia and especially by the use of suxamethonium (succinylcholine) or halothane, although other anaesthetic agents and relaxants have been implicated. Evidence suggests that there is a hereditary defect in the calcium-storing membrane of the skeletal and cardiac muscle cells so that calcium is released into the cytoplasm with the production of heat.

The temperature rise may immediately follow after the triggering mechanism or there may be an interval of 30–45 min, so that it may not become obvious until the patient has reached the recovery room.

The family practitioner should be notified so that other members of the family may be screened for this abnormality.

Clinical features

1. Extreme pyrexia
2. Cyanosis
3. Tachycardia and tachypnoea
4. Metabolic and respiratory acidosis
5. Hyperkalaemia
6. Muscle rigidity (in 60% of patients)
7. Coagulopathy

Management

1. Hyperventilation with 100% oxygen
2. Vigorous cooling with ice and fans

3. Sodium bicarbonate to correct acidosis. (Monitoring of blood gases will be required)
4. Intravenous dextrose 20% and 10 units of soluble insulin to reduce hyperkalaemia
5. Intravenous dantrolene 1 mg/kg every 5 min (up to 10 mg/kg may be required). Dantrolene inhibits the release of calcium into the muscle cell
6. Intravenous frusemide (furosemide) or mannitol to increase urinary output and prevent casts of myoglobin from blocking the renal tubules

Because of the rapidity of onset and the urgency of treatment, a malignant hyperthermia pack with all the necessary drugs should be readily available in the recovery room.

Blood Transfusion Reactions

Blood transfusion may be in progress when patients are admitted to the recovery room or alternatively it may become necessary during the immediate post-operative period. In either event, recovery staff must be alert for transfusion reactions which may be febrile, allergic or haemolytic. As patients are covered with drapes during surgery, skin reactions may only become obvious when these are removed at the end of the procedure.

Febrile Reactions

Febrile reactions occur in 1%–2% of transfusions and are generally caused by anti-leucocyte antibodies in the transfused blood. They are relatively slow in onset, the usual time from the start of transfusion being 2–4 h. Reactions may be more severe when blood is being transfused rapidly.

1. *Mild febrile reactions.* Temperature below 39 °C, no other symptoms or signs
   a) Slow the transfusion rate
   b) Administer antipyretics, e.g. paracetamol (acetaminophen)
2. *Severe febrile reactions.* Temperature above 39 °C, accompanied by rigors
   a) Stop transfusion
   b) Actively cool patient with tepid sponging and fans

   Bacterial contamination of transfused blood may initially present in this way. If this is suspected or there are accompanying signs of cardiovascular collapse:

   c) Give broad spectrum antibiotic
   d) Support the circulation by intravenous infusion
   e) Take appropriate blood samples and return the unit of blood and any previous units to the blood bank for bacteriological examination and serological testing

Allergic Reactions

1. *Mild.* Itching, rash
   a) Continue transfusion
   b) Administer antihistamines, e.g. chlorpheniramine 10 mg i.m.
2. *Severe.* Oedema, bronchospasm, hypotension
   a) Stop transfusion
   b) Support circulation by intravenous infusion
   c) Administer hydrocortisone 100 mg i.v.
   d) Consider adrenaline (epinephrine) 1 mg (1 ml 1:1000 solution) i.v.

Haemolytic Reactions

Haemolytic reactions are caused by incompatible blood transfusion. Haemolysed red cells release free haemoglobin which can cause renal damage. Reactions are characterised by:

1. Localised pain
2. Loin or retrosternal pain

} These signs are masked by unconsciousness

3. Flushing
4. Pyrexia
5. Dyspnoea
6. Cardiovascular collapse
7. Oliguria
8. Haematuria

The picture may be complicated in 50% of patients by the development of disseminated intravascular coagulation. If this occurs:

1. Stop transfusion and take down giving set
2. Support circulation by intravenous infusion
3. Give intravenous hydrocortisone 1 g
4. Send the unit of blood and any previously used packs together with a clotted sample of blood and anticoagulated samples for platelet count, clotting studies and examination for free Hb (p. 104)
5. Catheterise the bladder and monitor urine output. Send sample of urine for examination for Hb and urobilinogen
6. Stimulate urine production with frusemide (furosemide) or mannitol
7. Alkalinise urine with i.v. sodium bicarbonate to increase solubility of free Hb

Problems Associated with Massive Blood Transfusion

In addition to the normal hazards of any blood transfusion, whenever large volumes of blood have to be transfused rapidly (e.g. 500 ml every 5 min for 30 min), further problems may be anticipated as a result of changes in the stored blood.

Hypothermia

Since blood is normally stored at 4 °C the rapid infusion of cold blood will reduce body temperature and lead to:

1. Dysrhythmias and, in extreme cases, cardiac arrest
2. A shift of the oxygen dissociation curve to the left with impairment of oxygen release in the tissues

To eliminate these complications, large transfusions of blood should first pass through a blood warmer so that when it is transfused it is at body temperature.

Acidosis

Stored blood becomes progressively more acidostic and may have a pH of below 7.0. This is partly due to the presence of citrate in the anticoagulant and partly due to continuing anaerobic metabolism with lactic acid production. Since the shocked patient may already be acidotic, the resulting pH may become so low as to interfere with myocardial function and the reversal of muscle relaxants. This can be corrected by giving sodium bicarbonate intravenously, titrating the amount according to serial acid–base determinations. Bicarbonate should not, however, be given routinely as a metabolic alkalosis may result and further impair myocardial function.

Citrate Intoxication

Ionised calcium in stored blood is reduced by binding to citrate which is used as an anticoagulant. Under normal circumstances, the body has sufficient stores of calcium in the skeleton to compensate for this. However, following a massive transfusion or when citrate metabolism is impaired by liver disease, hypocalcaemia may result. This causes myocardial depression and hypotension. On the ECG, the ST segment is prolonged.

The treatment is to give 10 ml of 10% calcium gluconate slowly until the hypotension and ECG abnormalities are corrected.

Hyperkalaemia

Potassium diffuses from red cells during storage at a rate approaching 1 mmol per day so that at the end of 28 days' storage, blood may contain up to 30 mmol per litre. This may lead to significant hyperkalaemia (especially if renal function is impaired) with high peaked T waves on ECG and cardiac irritability. These effects can be countered by giving calcium gluconate, as described above. Alternatively, as insulin causes potassium to move intracellularly, 10 units of soluble insulin and 20 ml 50% dextrose can be given.

Micro-emboli

Stored blood contains micro-aggregates consisting of cell remnants and threads of fibrin ranging in diameter from 20 to 200 μm. Following transfusion these are filtered by the microcirculation in the lungs and lead to the development of adult respiratory distress syndrome (ARDS). This effect can be minimised by passing blood through a microfilter of pore size 20–40 μm and this is recommended for transfusions exceeding 1 litre. The resulting increased resistance can be overcome by using a pressure bag to maintain flow.

Failure of Coagulation (see below)

Stored blood rapidly becomes deficient in clotting factors (especially V, VII, and VIII) and platelets so that following a massive transfusion, their levels may become significantly reduced. These deficiencies can normally be made good with fresh frozen plasma, one unit for every five units of blood, and platelet concentrate, one unit for every ten units of blood. Coagulation studies will be required if bleeding persists despite this regime.

Failure of Coagulation

Persistent bleeding in the immediate post-operative period may be due to defective coagulation. Damage to blood vessels results in an accumulation of platelets around which fibrin clot is formed. A variety of clotting factors must be present in the blood to allow the conversion of fibrogen into the fibrin filaments:

I Fibrinogen
II Prothrombin
III Thromboplastin
IV Calcium
V Pro-accelerin
VI Not allocated
VII Pro-convertin

VIII Anti-haemophilic factor
IX Christmas factor
X Stuart-Power factor
XI Plasma thromboplastin antecedent
XII Hageman factor
XIII Fibrin stabilising factor

The fibrin clot is eventually lysed by plasmin, which is formed by conversion of the inactive plasminogen.

Causes

1. *Congenital deficiency of clotting factors.* Normally a single factor is deficient, e.g. haemophilia A (factor VIII) or Christmas disease (factor IX). This is usually recognised prior to surgery and the deficient factor is replaced pre- and post-operatively. This requires frequent assays of the appropriate factor. Formerly, some preparations of these factors were contaminated with the AIDS virus and many haemophiliacs were infected. The preparations currently used carry no risk.
2. *Massive blood transfusion.* Stored blood rapidly becomes deficient in clotting factors (especially V, VII and VIII) and subsequently in platelets. In addition, calcium is bound by the citrate anticoagulant and the resulting hypocalcaemia may contribute to deficient coagulation.
3. *Anticoagulants*
   a) *Heparin* acts at several sites in the coagulation process. It is frequently given intravenously during cardiovascular surgery and may be reversed by protamine sulphate. Care must be taken with protamine treatment since excess amounts may also act as an anticoagulant.
   b) *Coumarin-type drugs*, e.g. warfarin sodium. These inhibit the vitamin K-dependent carboxylation of clotting factors II, VII, IX and X in the liver. Rapid reversal cannot, therefore, be achieved by simply giving intravenous vitamin K_1 preparations such as phytomenadione (phytonadione) as fresh clotting factors have to be synthesised in the liver. If rapid reversal of anti-coagulation is required, fresh frozen plasma should be given.
4. *Liver disease.* Most clotting factors are synthesised in the liver and may be deficient in severe liver dysfunction.
5. *Vitamin K deficiency.* Vitamin K absorption is impaired in obstructive jaundice and following pre-operative bowel sterilisation with anitbiotics.
6. *Disseminated intravascular coagulation (DIC).* In the following clinical states there is massive deposition of fibrin throughout the microcirculation, resulting in the consumption of clotting factors and platelets with consequent bleeding:
   a) Prolonged shock with tissue hypoxia
   b) Extensive tissue damage, e.g. trauma, burns, prolonged surgery

c) Infection, e.g. acute viral infection, septicaemia
d) Obstetric emergencies, e.g. amiotic fluid embolism, intrauterine death, abruptio placentae, toxaemia
e) Acute haemolysis, e.g. mismatched transfusion
f) Following prostatectomy
g) Extracorporeal circulation

Extensive fibrinolysis follows, with the liberation of fibrin degradation products and thence further bleeding. The condition is therefore characterised by clotting and bleeding occurring simultaneously.

7. *Thrombocytopenia* (platelet deficiency). Platelets are not only required to initiate haemostasis by plugging damaged blood vessels but are also essential in the coagulation process. If the platelet count falls below 50×10^9/litre, a failure of coagulation may occur.

   Causes of thrombocytopenia seen post-operatively:

   a) *Dilution of platelets.* Transfusion of stored blood deficient in platelets leads to a reduction in circulating platelet count. This reduction is more than can be accounted for by dilution alone. Significant reduction in platelet count is often observed after the rapid transfusion of six or more units of stored blood.
   b) *Depressed platelet formation*, e.g. as seen in patients with leukaemia, cancer, uraemia and during chemotherapy.
   c) *Excessive utilisation of platelets*, e.g. in disseminated intravascular coagulation.
   d) *Idiopathic thrombocytopenia.* A rare condition of unknown aetiology seen mainly in young adults and characterised by purpura and petechiae.
   e) *Excessive destruction of platelets*, e.g. in hypersplenism.

Management

To determine the cause of a failure of coagulation will require a series of laboratory tests:

1. *Platelet count* (EDTA bottle). Normal values $150–400 \times 10^9$/litre. Bleeding is unusual if the value is above 50×10^9/litre.
2. *Prothrombin time* (sodium citrate bottle). Normal value, approximately 12 s. Therapeutic ratio is 2–4 times the normal value. Prolonged in deficiency of factors II, V, VIII and X, e.g. as seen in liver disease, vitamin K deficiency, anticoagulant therapy and DIC.
3. *Activated partial thromboplastin time* (APTT) (sodium citrate bottle); also kaolin cephalin clotting time (KCCT). Normal values, 25–40 s. Prolonged in deficiency of factors II, V, VIII, IX, X, XI, and XII e.g. as seen in anticoagulant therapy, haemophilia A, Christmas disease.
4. *Fibrin degradation products* (FDPs) (bottle with soya bean trypsin inhibitor). Normal values $< 10\mu g$/ml. Raised in DIC. (Smaller rises are found following major surgery, trauma, deep vein thrombosis and pulmonary embolism.

5. *Fibrinogen level* (sodium citrate bottle). In this test the plasma is diluted to give the titre, i.e. the greater the fibrinogen content the more the dilution. Normal values > 1 : 64; Low values, < 1 : 64. The test may be modified to detect the presence of a circulating inhibitor or fibrinolysins. Fibrinogen is deficient in liver disease, in DIC and after massive blood transfusion.
6. *Thrombin clotting time* (TCT) (sodium citrate bottle). Normal values, 20–30 s. Prolonged when fibrinogen is deficient or abnormal and in the presence of inhibitory substances, e.g. FDPs, heparin.

The above tests are not, however, necessary for rational therapy to be given; the immediate history will usually supply sufficient information for this purpose.

1. *Following major transfusion of stored blood* it can be predicted that clotting factors, platelets and available calcium will be reduced. This can be corrected by the administration of:
   a) Fresh frozen plasma. This contains all the clotting factors and can normally be made available within 20 min. Adequate levels can be maintained by giving one unit of fresh frozen plasma for every five units of blood.
   b) Platelet concentrate. This has to be specially prepared at a transfusion centre from freshly donated blood. As it deteriorates rapidly it should be used without delay. A blood filter should not be used for the infusion of platelets.
   c) Intravenous calcium gluconate 10%. Increments of 10 ml may be needed for every litre of blood if it is being transfused rapidly. This is mainly to counter myocardial depression due to hypocalcaemia, which only rarely interferes with coagulation.
2. *Following cardiovascular surgery* persistent heparinisation may require reversal by intravenous protamine sulphate. In calculating dosage, allowance should be made for the metabolism of heparin, and protamine given in minimal amounts since excess protamine itself interferes with coagulation.

If these simple measures do not correct the situation and the clinical picture suggests the possibility of disseminated intravascular coagulation, further action is urgently required:

1. *Consult a haematologist.* It is wise to enlist the help of an experienced haematologist at an early stage since inappropriate therapy will not only waste valuable time but make subsequent management more difficult.
2. *Draw blood for coagulation tests.* As the coagulation profile may be constantly changing it is important that all the blood required for the various tests is taken at the same time.
3. *Continue to replace blood* with the addition of fresh frozen plasma, platelets and calcium as required to prevent further deficiencies.

In the event of DIC being diagnosed, subsequent treatment will be aimed at:

1. Removal of precipitating stimulus is possible, e.g. evacuation of uterine contents,

2. Replacement of coagulation factors and, possibly,
3. Heparinisation — despite persistent bleeding this may be necessary to prevent continued intravascular coagulation with consumption of clotting factors.

Oliguria

Causes

Inadequate urine output (less than 0.5 ml/(kg·h)) may be due to the following causes:

1. *Pre-renal.* Inadequate renal perfusion due to hypovolaemia or hypotension, e.g. systolic readings of below 60 mmHg.
2. *Renal damage* due to:
   a) Sepsis
   b) Haemolysis
   c) Hypoxaemia
   d) Hypotension
   e) Antibiotic therapy, e.g. gentamicin
   f) Release of myoglobin, e.g. following crush injury, malignant hyperthermia
3. *Post-renal outflow obstruction*

The urine output is monitored post-operatively in patients at risk of developing acute renal failure. Indications include:

1. Impaired renal or cardiac function pre-operatively
2. Obstructive jaundice (see p. 150)
3. Episodes of hypoxia or hypotension
4. Cardiac or aortic surgery
5. Major trauma or severe blood loss
6. Septicaemia
7. Extensive burns
8. Crush injury
9. Mismatched transfusion
10. Pancreatitis
11. Malignant hyperthermia

Management

1. Exclude mechanical obstruction of urinary catheter caused by clots of blood or kinking. Bladder distension will suggest this. Change catheter if obstruction cannot be cleared.

2. Consider hypotension or hypovolaemia as the most likely causes. Measure the CVP, if necessary.
3. Infuse 250–500 ml of sodium chloride 0.9% intravenously. A subsequent increase in urine output confirms hypovolaemia, which must be corrected.

If these measures are unsuccessful and the urine production remains below 0.5 ml/(kg·h), acute renal failure may be imminent.

4. Urinary production may be stimulated by:
   a) mannitol 100 ml of 20% over 15 min
   b) frusemide (furosemide) 20–40 mg
   c) low dose dopamine infusion, i.e. up to 5 μg/(kg·min).

If an adequate renal output is not established following these measures, acute renal failure must be assumed to have occurred and the advice of a nephrologist should be sought as soon as possible. While the patient remains in the recovery unit it is important not to overload the circulation.

Further Reading

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