Chapter 3

Postanaesthesia care

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.

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.

> 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 30 mins, and agreed discharge criteria (Table 3.1).

Table 3.1 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

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 Po_2 will be <8 kPa (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 Po_2 (PAo_2) and arterial Po_2 (Pao_2) fall. In most patients, increasing their inspired oxygen concentration will restore alveolar and arterial Po_2. 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 ($Paco_2$) 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 (see page 100). An oropharyngeal or nasopharyngeal airway may be required to help maintain the airway (see page 18).

> 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 (see page 39).
- *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 patient unable 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 see page 35. 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 splinting* 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 within the lungs

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

Table 3.2 Effect of alveolar oxygen concentration on oxygen content of blood

	Alveolar oxygen concentration (%)	Haemoglobin saturation (%)	Oxygen content (mL/100 mL blood)
Alveoli containing air	21	97	20
Alveoli containing oxygen	100	100	21
Non-ventilated alveoli	Very low	75	15

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, Table 3.2), 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.

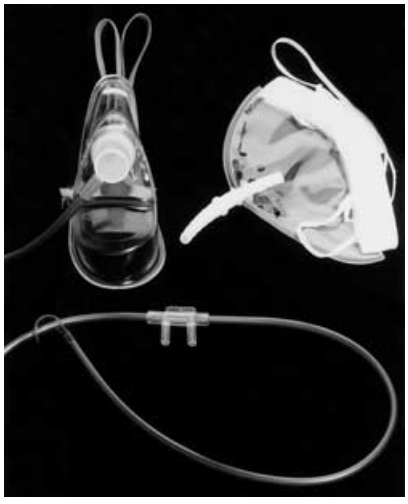

Fig. 3.1 Hudson mask (top left), MC mask (top right) and nasal catheters (bottom).

Examples of this type of device are Hudson and MC masks (Fig. 3.1). 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 (see Fig. 3.1). 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 (see Fig. 3.2). 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 (12–15 L/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.

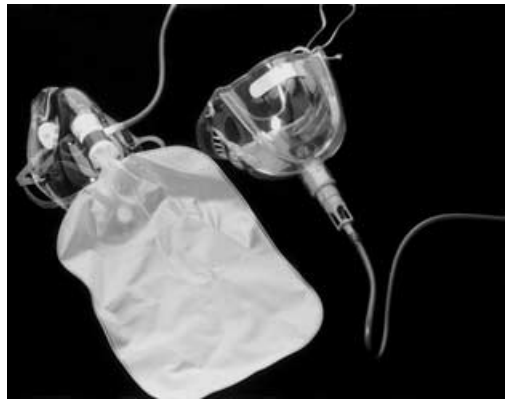

Fig. 3.2 Hudson mask with reservoir and high airflow oxygen enrichment (HAFOE; Venturi) mask.

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 45 L/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 (Fig. 3.2).

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 (see also page 96):

- 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 (>2 s) 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 (<0.5 mL/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. during aortic aneurysm surgery).

> The commonest cause of oliguria is hypovolaemia; anuria is usually due to a blocked catheter.

The extent to which these changes occur will depend primarily upon the degree of hypovolaemia. A tachycardia may not be seen in the patient taking beta blockers and up to 15% of the blood volume may be lost without detectable signs in a fit, young patient. An arterial blood sample should be analysed; a metabolic acidosis is usually found after a period of poor tissue perfusion.

Management **(see also page 97)**

- 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. The diagnosis 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 for hypovolaemia 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 5 mL/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 20–40 mg) 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) (see page 121). Unfortunately thrombolysis is contraindicated after surgery.

Vasodilatation

This is common during spinal or epidural anaesthesia (see page 67). 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 Gram-negative 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 (see page 98).
- *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 cardiac output:

- heart rate 100–150/min with critical perfusion will require cardioversion followed by IV amiodarone 300 mg over 1 h;
- 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 epidural anaesthesia;
- 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 0.5 mg 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 spinal anaesthesia.
- 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 50 mg intramuscularly, up to 6 hourly. Also has anticholinergic actions; may cause a tachycardia when given IV.

• *5-HT3 (hydroxytryptamine) antagonists* Ondansetron (Zofran). Adults 4–8 mg 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 10 mg 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 (see page 59), 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 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:

1.5 mL/kg/h to make up the deficit from 0800 until 1800
$= 1.5 \times 70\,(\text{kg}) \times 10\,(\text{h}) \approx 1000\,\text{mL}$;
1.5 mL/kg/h from 1800 until 0800 the next morning:
$= 1.5 \times 70\,(\text{kg}) \times 14\,(\text{h}) \approx 1400\,\text{mL}$.
The total IV fluid requirement = 2400 ml in the next 14 h.

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:

2 × 1000 mL 5% glucose and 500 mL 0.9% (normal) saline; or

2 × 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)); or
  • 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 1 mmol/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 1L 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 (see page 52) 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, fol-

lowed 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 surgical procedures.
- Economic costs:
  - prolonged hospital stay, increased medical complications;
  - increased time away from normal occupations.
- 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).

> 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 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

Table 3.3 A simple practical scoring system for acute pain

Pain score	Staff view	Patient's view	Action
0	None	Insignificant or no pain	Consider reducing dose or changing to weaker analgesic, e.g. morphine to NSAID plus paracetamol
1	Mild	In pain, but expected and tolerable; no reason to seek (additional) treatment	Continue current therapy, review regularly
2	Moderate	Unpleasant situation; treatment desirable but not necessarily at the expense of severe treatment side-effects	Continue current therapy, consider additional regular simple analgesia, e.g. paracetamol and/or NSAID
3	Severe	Intolerable situation—will consider even unpleasant treatments to reduce pain	Increase dose of opioid, or start opioid; consider alternative technique, e.g. epidural

analgesia should allow mobilization with only mild to insignificant pain.

Analgesic drugs used postoperatively

(Fig. 3.3)

The most commonly used drugs are opioids and NSAIDs.

Opioids

The pharmacology of opioid drugs and their side-effects are covered on page 37. In the UK, 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 (Table 3.4). 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. 0.4 mg 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.

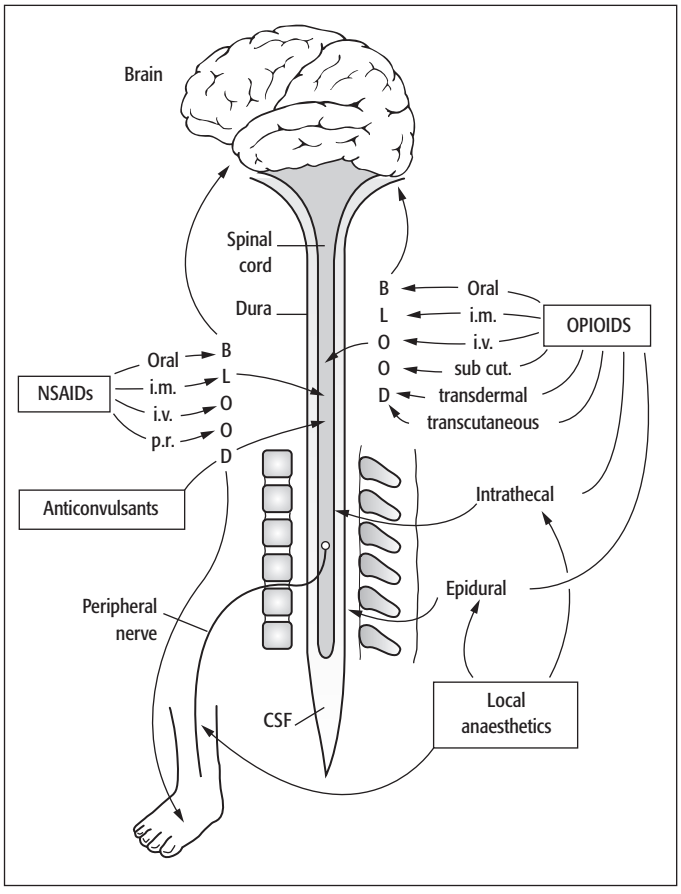

Fig. 3.3 Sites of action of analgesic drugs.

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 (8 mg 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 (see page 37).

Neither is a controlled drug and so are more easily accessible.

Non-steroidal anti-inflammatory drugs (NSAIDs)

The pharmacology of these drugs has been covered on page 40.

• *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 1 g 4–6 hourly, maximum 4 g/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 15 mins and is effective in 5–10 mins. The dose is the same as for the oral preparation. It is the safest of all

Table 3.4 Administration of morphine

Oral	Immediate release (IR) tablets or liquid • Absorption and effect within minutes • Usual adult dose 20 mg hourly prn • Less in elderly, more if opioid tolerant • Providing the gut is working, useful even after major surgery • Usually used for acute pain where the opioid requirement is unknown or changing rapidly Modified release (MR) tablets, capsules or granules • Dose released over either 12 or 24 h • Avoids frequent dosing with immediate release preparations • Useful when opioid requirement is prolonged and also for gradually weaning down the dose at the end of treatment The two formulations are usually used together to provide a steady background level of analgesia (MR) with additional breakthrough doses (IR) as required *It is important that everybody understands the difference between MR and IR forms of morphine*
Intravenous	Morphine 10 or 20 mg diluted to 1 mg/mL with 0.9% sodium chloride can be given: • In increments initially of 1–3 mg at 3 min intervals; effective dose may range from 1 to 50 mg or more (the latter in opioid-tolerant patients) • Via patient controlled analgesia device (see below) • As a continuous infusion. Useful where patient cooperation is limited, e.g. in elderly patients or intensive care units. Problems occur in predicting the correct infusion rate, given the variability of dose requirement between patients Very close supervision is required to avoid underinfusion (pain) or overinfusion (toxicity) This method can be used to replace high doses of oral opioids during the perioperative period *The intravenous dose of morphine is about one-third of the oral dose*
Intramuscular	• A predetermined dose (e.g. morphine 10 mg) at fixed minimum intervals, e.g. hourly • Delayed and variable rate of effect • Precise titration is difficult with repeated cycles of pain and relief • Does not require complex equipment or a co-operative patient • Widely available • Intermittent injections through an indwelling cannula may be more acceptable to staff and patients

analgesics but patients may need reassurance that regular dosing of 1 g every 6 h is not associated with hepatic toxicity. A summary of the use of these drugs is given in Table 3.5.

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, continuous infusion.

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.

Table 3.5 Non-steroidal anti-inflammatory drugs (NSAIDs)

Route given	Non-specific (COX-1 + 2)	COX-2 specific	CNS effect only
Oral	Ibuprofen 200–400 mg 8-hourly	Rofecoxib 50 mg once daily	Paracetamol 1 g 6-hourly
Intravenous	Ketorolac 10–30 mg 6-hourly, max 90 mg/24 h	Paracoxib 40 mg 12-hourly	Paracetamol 1 g equivalent
Effects			
Anti-inflammatory	Yes	Yes	No
Analgesic (central)	Yes	Yes	Yes
Side-effects			
Reduced renal blood flow	Yes	Probably	No
Gastric ulceration	Yes	Unlikely	No
Anti-platelet	Yes	Unlikely	No
Delay bone healing	Possible	Unlikely	No

• Typical settings for an adult using morphine delivered by a PCA device might be:
  • bolus dose: 1 mg;
  • lockout interval: 5 mins.

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. Any patient whose respiratory rate is less than 8 breaths/min and sedation score is 2 or 3 should be treated as described in Table 3.6.

Table 3.6 Management of overdose with patient-controlled analgesia (PCA)

- Stop the PCA
- Give oxygen via a mask
- Call for assistance
- Consider giving naloxone (as described on page 82)
- If the patient is apnoeic, commence ventilation using a self-inflating bag-valve-mask device

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.

• Intravenous administration with greater certainty of adequate plasma levels.

Disadvantages

• Equipment is expensive to purchase and maintain.

• 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 1 h prior to discontinuing PCA, to ensure continuity of analgesia.

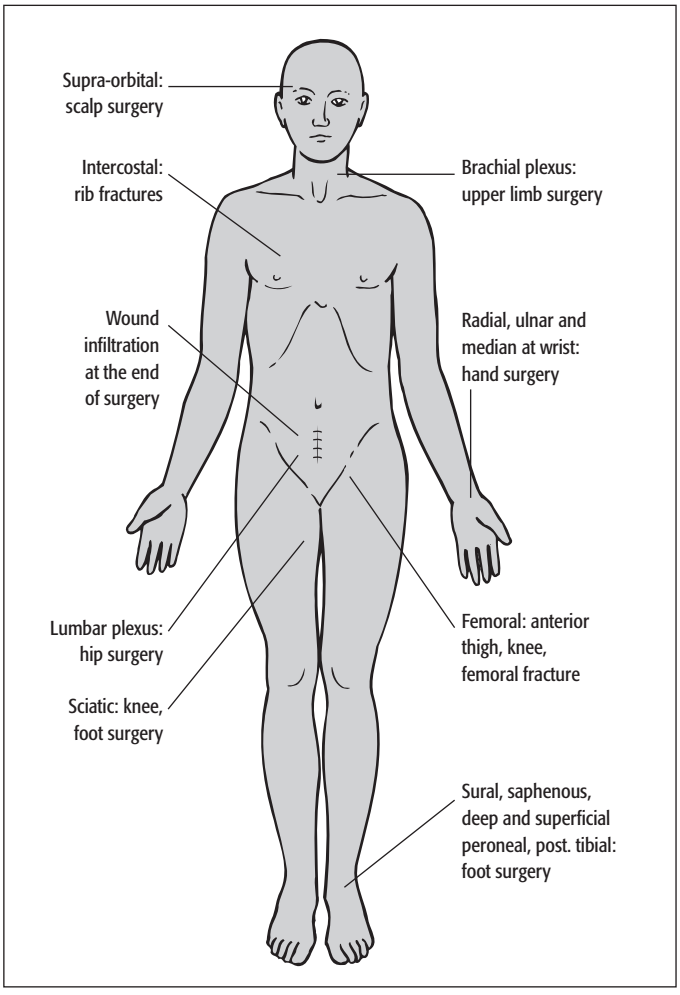

Fig. 3.4 Some commonly used nerve blocks.

Regional analgesic techniques (Fig. 3.4)

• *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 (see also page 65)* 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 µg/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.

Management of complications during postoperative epidural analgesia

This will depend upon whether local anaesthetics alone or in combination with opioids have been used. The complications arising as a result of the use of local anaesthetics intraoperatively are covered on page 63.

- *Hypotension* Sympathetic block causes vasodilatation and increased venous pooling. Treat acutely with IV fluid and vasopressors (e.g. ephedrine according to local policy). Prevented by ensuring the patient has an adequate fluid regimen prescribed. Patients with additional fluid losses, for example haemorrhage, are particularly vulnerable to severe hypotension. Check the extent of the block; if extensive, reduce the rate of infusion.
- *Respiratory depression* Caused by opioid reaching the respiratory centre in the medulla. Highly lipid soluble opioids (diamorphine, fentanyl) are rapidly taken up by the spinal cord, limiting their spread and systemic absorption, and respiratory depression tends to occur early; less soluble opioids (morphine) are taken up slowly, and respiratory depression tends to occur later. A high infusion rate of either drug may also lead to respiratory depression. Prevented by regular assessment and recording of vital signs. Treat by supporting ventilation if necessary; stop the epidural infusion; give naloxone according to the severity; seek expert help.
- *Sedation* Due to opioid reaching the brain either directly via the CSF or after absorption into the systemic circulation via the epidural veins. May be secondary to hypotension and cerebral hypoxaemia. Prevent by regular assessment and recording of vital signs. Treat by stopping the infusion; if unresponsive or the level of sedation progresses, give naloxone in 0.1 mg increments intravenously; seek expert help.
- *Pruritus* Can be severe and frequently localized to the nose; may respond to antihistamines, atropine or naloxone.
- *Retention of urine* May be due to the effect of the opioid on bladder sphincter control or the local anaesthetic removing the sensation of a full bladder. More common in males, particularly if there are already symptoms of prostatism. Prevented by routine monitoring of urine output in all postoperative patients. May require short-term catheterization.
- *Numbness and weakness of the legs* Usually due to excessive rates of infusion or a too-high concentration of local anaesthetic. May lead to pressure ulcers on the patient's heels or sacrum due to lack of movement, or falls whilst mobilizing. Prevented by regular observation of effects of epidural and correct adjustment of infusion rate.
- *Vertebral canal haematoma* Can occur as a result of trauma by the needle or catheter insertion. Greater risk in patients on warfarin, heparin, NSAIDs and other antiplatelet drugs, those with a

coagulopathy or when severely haemodiluted. Rare, but consider if profound motor and sensory block far greater than anticipated.

• *Infection* Introduced via the catheter. May cause the formation of an epidural abscess and compromise of the spinal cord. Patients complain typically of increasing back pain but this may be delayed for several weeks so that the connection to the surgery and epidural may be missed.

If there is any doubt about spinal cord function the epidural infusion should be stopped and a magnetic resonance imaging (MRI) scan considered. Damage to nerves or the spinal cord during insertion of the needle and systemic toxicity of the local anaesthetic are both unusual complications.

Intrathecal (spinal) analgesia

(see page 66)

Spinal anaesthesia is of insufficient duration to provide postoperative pain relief. However, if a small dose of opioid, for example morphine 0.1–0.25 mg, is injected along with the local anaesthetic, this may provide up to 24 h of analgesia. Complications are the same as those due to opioids given epidurally, and managed in the same way.

Other techniques

Entonox is a mixture of nitrous oxide (50%) and oxygen (50%). It is a weak analgesic with sedative properties. Useful for short-term analgesia for painful procedures, for example change of dressings. It should be avoided in patients with a pneumothorax because the nitrous oxide may diffuse into the gas-filled space, increasing the volume.

Combining analgesic techniques

Examples of good practice are:

• bupivacaine and fentanyl in an epidural infusion;

• intravenous PCA morphine and intravenous paracoxib in the early postoperative period when nil by mouth;

• oral morphine immediate release (IR) tablets and paracetamol prescribed to be given as a spinal wears off.

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.

Further reading

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Useful websites

http://www.aagbi.org/pdf/Postanaes2002.pdf
[Immediate postanaesthetic recovery. The Association of Anaesthetists of Great Britain & Ireland. September 2002.]
http://oac.med.jhmi.edu/res_phys/
[The Johns Hopkins School of Medicine interactive respiratory physiology website.]
http://www.resus.org.uk/pages/periarst.htm
[Current Resuscitation Council UK guidelines on peri-arrest arrhythmias.]
http://www.jr2.ox.ac.uk/bandolier/booth/painpag/
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