

Intensive care medicine

Intensive care medicine (or 'critical care medicine') is concerned mainly with the management of patients with acute life-threatening conditions ('the critically ill') in a specialized unit. It also encompasses the resuscitation and transport of those who become acutely ill, or are injured, either elsewhere in the hospital or in the community. An intensive care unit (ICU) has the facilities and expertise to provide cardiorespiratory support to these sick patients, some of whom also have kidney or liver failure, and management of this is described in the relevant chapters.

All patients admitted to the ICU require skilled nursing care (patient to nurse ratio of 1:1) and physiotherapy. Many require nutritional support. General medical management includes the prevention of venous thrombosis (p. 217), pressure sores and constipation. A number of scoring systems, such as the APACHE score, are in use to evaluate the severity of the patient's illness.

Acute disturbances of haemodynamic function (shock) (K&C p. 926)

The term 'shock' is used to describe acute circulatory failure with inadequate or inappropriately distributed tissue perfusion resulting in generalized cellular hypoxia. The causes of shock are listed in Table 11.1. Shock is often the result of a combination of these factors.

Pathophysiology

Sympathoadrenal In response to hypotension there is a reflex increase in sympathetic nervous activity and catecholamine release from the adrenal medulla. The resulting vasoconstriction, increased myocardial contractility and heart rate help restore blood pressure and cardiac output. Activation of the renin-angiotensin system leads to

Table 11.1

Causes of shock

Hypovolaemic

Exogenous losses (e.g. haemorrhage, burns)

Endogenous losses (e.g. sepsis, anaphylaxis)

Cardiogenic

Myocardial infarction

Myocarditis

Rupture of a valve cusp

Obstructive

Obstruction to outflow (e.g. pulmonary embolus)

Restricted cardiac filling (e.g. cardiac tamponade)

Distributive

Vascular dilatation (e.g. drugs, sepsis)

Arteriovenous shunting

Maldistribution of flow (e.g. sepsis, anaphylaxis)

vasoconstriction and salt and water retention, which help to restore circulating volume.

Neuroendocrine response There is release of anterior pituitary hormones and glucagon, which are insulin antagonists. They raise blood sugar and may be responsible for some of the cardiovascular changes.

Release of mediators In septic shock components of microorganisms (e.g. endotoxin of Gram-negative bacteria) release cytokines (tumour necrosis factor, interleukin-1 and interferon- γ) from macrophages and white cells, activate the complement system and cause the release of vasoactive mediators (e.g. prostacyclin, endothelin-1 and nitric oxide) from vascular endothelium. The end result of these processes is vasodilatation, increased vascular permeability, endothelial cell damage and platelet aggregation. Vasodilatation and increased vascular permeability are also seen in shock secondary to anaphylaxis.

A similar widespread inflammatory response may occur with non-infectious processes, e.g. trauma and acute pancreatitis, and is referred to as the *systemic inflammatory response syndrome* (SIRS). The clinical features are pyrexia, tachycardia, tachypnoea and a raised white cell count.

Microcirculatory changes In the early stages of septic shock there is vasodilatation, increased capillary permeability with interstitial oedema, and arteriovenous shunting. Vasodilatation and increased capillary permeability also occur in anaphylactic shock. In the initial stages of other forms of shock, and in the later stages of sepsis and anaphylaxis, there is capillary sequestration of blood. Fluid is forced into the extravascular space, causing interstitial oedema, haemoconcentration and an increase in plasma viscosity.

In all forms of shock there may be activation of the coagulation pathway, with the development of disseminated intravascular coagulation (DIC, see p. 213). The disseminated inflammatory response and microcirculatory changes may lead to progressive organ failure (*multiple organ dysfunction syndrome* (MODS), also known as multiple organ failure (MOF)); the lungs are usually affected first, with the development of the *acute respiratory distress syndrome* (ARDS). The mortality in MODS is high and treatment is supportive.

Clinical features

The history will often indicate the cause of shock, e.g. a patient with major injuries (often internal and thus concealed) will often develop hypovolaemic shock. A patient with a history of peptic ulceration may now be bleeding into the gastrointestinal tract, and rectal examination will show melaena. Anaphylactic shock may develop in susceptible individuals after insect stings and eating certain foods, e.g. peanuts.

Hypovolaemic shock Inadequate tissue perfusion causes blue cold skin with slow capillary refill. The blood pressure (particularly when supine) may be maintained initially, but later hypotension supervenes (systolic BP < 100 mmHg) with oliguria (< 30 mL of urine/h), confusion and restlessness. Increased sympathetic tone causes tachycardia (pulse > 100/min) and sweating.

Cardiogenic shock Additional clinical features are those of myocardial failure, e.g. raised jugular venous pressure

Acute disturbances of haemodynamic function (shock)

(JVP), pulsus alternans (alternating strong and weak pulses) and/or a 'gallop' rhythm (p. 389).

Mechanical shock Muffled heart sounds, pulsus paradoxus (pulse fades on inspiration), elevated JVP and Kussmaul's sign (JVP increases on inspiration) occur in cardiac tamponade. In pulmonary embolism there are signs of right heart strain, with a raised JVP with prominent 'a' waves, right ventricular heave and a loud pulmonary second sound.

Anaphylactic shock Profound vasodilatation leads to warm peripheries and low blood pressure. Erythema, urticaria, angio-oedema, bronchospasm, and oedema of the face and larynx may all be present.

Septic shock In the early stages there is vasodilatation, pyrexia and rigors. At a later stage there are features of hypovolaemic shock. Sepsis in elderly people or in the immunosuppressed is common without the classic clinical features.

Management (K&C p. 937)

This is summarized in Emergency Box 11.1. The underlying cause must be identified and treated appropriately. Whatever the aetiology of shock, tissue blood flow and blood pressure must be restored as quickly as possible to avoid the development of MOF.

Expansion of the circulating volume Volume replacement is obviously important in hypovolaemic shock, but also in anaphylactic and septic shock, where there is vasodilatation, sequestration of blood and loss of circulating volume secondary to capillary leakage. High filling pressures may also be needed in mechanical shock. Care must be taken to prevent volume overload, which leads to a reduction in stroke volume and a rise in left atrial pressure with a risk of pulmonary oedema. The choice of fluid depends on the clinical situation:

- Whole blood is the fluid of choice for haemorrhage. Crossmatched blood must be used if possible, but in

**Emergency Box 11.1****Management of shock****Ensure adequate oxygenation and ventilation**

- Maintain patent airway: use oropharyngeal airway or endotracheal tube if necessary.
- Administer 100% oxygen via tight-fitting face mask.
- Monitor respiratory rate, blood gases and chest X-ray.

Restore cardiac output and BP

- Lay patient flat or head-down.
- Expand circulating volume with appropriate fluids given quickly via large-bore cannulae.
- Monitor skin colour, pulse and blood pressure, peripheral temperature, urine output, ECG.

CVP monitoring is required in most cases, Swan–Ganz catheter in selected cases.

Investigations

- FBC, U+E, glucose, liver biochemistry, and blood gases in all cases.
- Infection screen, lactate levels, fibrinogen degradation products and crossmatch blood in selected cases. Echocardiogram in post-MI patients to identify patients with intra- or extramyocardial rupture.

Treat underlying cause

- Haemorrhage
- Sepsis
- Anaphylaxis.

Treat complications

- e.g. Coagulopathy, renal failure.

extreme emergencies the ‘universal donor’ group O rhesus-negative blood is used. Complications of massive blood transfusion are hypothermia, thrombocytopenia, hypocalcaemia and depletion of clotting factors.

- Colloidal solutions increase colloid osmotic pressure and produce a greater and more sustained increase in plasma volume than crystalloid solutions. They are used to replace fluid in hypovolaemic patients and are useful for the maintenance of blood volume, but have

Acute disturbances of haemodynamic function (shock)

no oxygen-carrying capacity. Polygelatin solutions (e.g. Gelofusin and Haemacel) are the most widely used. Human albumin solution and dextrans are less commonly used because of the expense (albumin) and higher complication rate (dextrans). Colloid solutions are often used for acute blood loss before whole blood becomes available, and for volume replacement in anaphylactic and septic shock.

- Crystalloids, e.g. 5% dextrose, 0.9% saline, are readily available and cheap. Once in the circulation they quickly redistribute into the interstitial fluid; therefore large volumes are needed to restore circulating volume and the excess fluid in the interstitial space may contribute to pulmonary oedema. Large volumes of crystalloid (> 2 litres) as a treatment for shock are best avoided. However, crystalloids are frequently used for volume replacement with diarrhoea and vomiting, and sometimes with burns.

Myocardial contractility and inotropic agents Myocardial contractility is impaired in cardiogenic shock and at a later stage in other forms of shock as a result of hypoxaemia, acidosis and the release of mediators. It is recommended that the treatment of acidosis should concentrate on correcting the cause; intravenous bicarbonate should only be administered to correct extreme (pH < 7.0) persistent metabolic acidosis. Drugs that impair cardiac performance, e.g. β -blockers, should be stopped. When a patient remains hypotensive despite adequate volume replacement inotropic agents are administered. This must be via a large central vein and the effects carefully monitored. The inotropic agents used and their clinical effects are shown in Table 11.2. Many consider dopamine to be the inotrope of choice in critically ill patients, but dobutamine is a better choice when vasoconstriction caused by dopamine could be dangerous. Norepinephrine (noradrenaline) in combination with dobutamine (depending on the cardiac output) is used for shocked patients with a low peripheral resistance, e.g. septic patients.

Additional treatment Vasodilators, e.g. sodium nitropruside and isosorbide dinitrate, may be useful in selected

Table 11.2

Inotropic agents used in the management of shock: the effect of each inotrope on the adrenergic and dopaminergic receptors is shown

(Dose, $\mu\text{g}/\text{kg}/\text{min}$)	β_1	β_2	α_1	α_2	DA_1	DA_2	Comments
Epinephrine (adrenaline) Low dose (0.06–0.1) Moderate dose (0.1–0.18) High dose (>0.18)	++ ++ ++(+)	+ + +	+ ++ +++	+ + +++	0 0 0	0 0 0	A potent inotrope used in patients not responding to dobutamine or dopamine. At high doses vasoconstriction may increase renal perfusion pressure and urine output, but as dose is further increased marked vasoconstriction leads to decreased cardiac output, oliguria and peripheral gangrene. Agent of choice in septic shock when haemodynamic monitoring not available
Norepinephrine (noradrenaline)	++	0	+++	+++	0	0	Particularly useful in septic shock as administration leads to increased inotropy and an increase in peripheral vascular resistance. Requires full haemodynamic monitoring
Isoprenaline	+++	+++	0	0	0	0	Rarely used

**Table 11.2**
(continued)

(Dose, $\mu\text{g}/\text{kg}/\text{min}$)	β_1	β_2	α_1	α_2	DA ₁	DA ₂	Comments
Dopamine							
Low dose (1–3)	+	0	0	+	++	+	At low dose general vasodilatory action which may increase urine output and preserve function of vital organs. Increases cardiac output at all doses, but at high doses this beneficial effect may be offset by vasoconstriction, thus increasing afterload and ventricular filling pressure
Moderate (3–10)	++	+	++	+	++(+)	+	
High dose (> 10)	+++	++	+++	+	++(+)	+	
Dopexamine	+	+++	0	0	++		Dopamine analogue. Most useful in patients with a low cardiac output and peripheral vasoconstriction
Dobutamine	++	+	+	0	0		Similar actions to dopexamine, useful in patients with cardiogenic shock
Enoximone							Phosphodiesterase inhibitor with inotropic and vasodilator actions
							Occasionally useful in acute heart failure

0, no agonism; + mild agonism; ++, moderate agonism; +++, profound agonism; α_1 , α -adrenergic receptors; β , β -adrenergic receptors; DA, dopamine receptors

patients who remain vasoconstricted and oliguric despite adequate volume replacement and a satisfactory blood pressure. Finally, in patients with a potentially reversible depression of left ventricular function (e.g. cardiogenic shock secondary to a ruptured interventricular septum), intra-aortic balloon counterpulsation (IABCP) may be used as a temporary measure to maintain life until definitive surgical treatment can be carried out.

Specific treatment of the cause In all cases the cause of shock must be identified if possible and specific treatment given when indicated.

- *Septic shock.* Antibiotic therapy should be directed towards the probable cause. In the absence of helpful clinical guidelines, 'blind' intravenous antibiotic therapy (e.g. cefuroxime and gentamicin) should be started after performing an infection screen: chest X-ray and culture of blood, urine and sputum. Lumbar puncture, ultrasonography and CT of the chest and abdomen are useful in selected cases. Abscesses require drainage. Steroids have no role in the treatment of septic shock.
- *Anaphylactic shock* must be identified and treated immediately (Emergency Box 11.2).

Monitoring (K&C p. 932)

This is by both clinical and invasive means.

Clinical An assessment of skin perfusion, measurement of pulse, BP, JVP and urinary flow rate will guide treatment in a straightforward case. Additional invasive monitoring will be required in seriously ill patients who do not respond to initial treatment.

Invasive

- *Blood pressure.* A continuous recording may be made with an intra-arterial cannula, usually in the radial artery.
- *Central venous pressure (CVP)* is related to right ventricular end-diastolic pressure, which depends on circulating blood volume, venous tone, intrathoracic



Emergency Box 11.2

Management of anaphylactic shock

Remove the precipitating cause, e.g. stop administration of the offending drug.

Administer:

- 0.5 mg epinephrine (adrenaline) intramuscularly, * i.e. 5 mL of a 1 in 10 000 solution
- Colloid, e.g. Haemaccel, 1 L rapid i.v. infusion and continue depending on response
- High concentration inhaled oxygen
- Antihistamine, e.g. chlorphenamine (chlorpheniramine) 10 mg i.v. over 1–2 min
- Hydrocortisone 200 mg i.v.
- Repeat epinephrine every 10 minutes until improvement occurs.

* Give intravenous epinephrine (0.5 mg over 5 minutes) with full ECG monitoring if patient is extremely unwell with hypotension and severe dyspnoea. There is a risk of relapse even after full recovery. Admit patient to hospital for 24 hours for monitoring and treatment with hydrocortisone and chlorphenamine.

Patients who have had an attack of anaphylaxis and who are at risk of developing another should carry a preloaded syringe of epinephrine for subcutaneous self-administration (e.g. EpiPen device) and wear an appropriate information bracelet (e.g. MedicAlert).

pressure and right ventricular function. CVP is measured by inserting a catheter percutaneously into the superior vena cava and connecting it to a manometer system (p. 740). The normal range is 0–4 cmH₂O above the manubriosternal angle in a supine patient. In shock CVP may be normal, because in spite of hypovolaemia there is increased venous tone. A better guide to circulating volume is the response to a fluid challenge (Fig. 11.1).

- *Left atrial pressure.* In uncomplicated cases the CVP is an adequate guide to the filling pressures of both sides of the heart. However, if there is disparity in function between the two ventricles (e.g. infarction of the left ventricle), left atrial pressure must be measured. A Swan–Ganz catheter is introduced percutaneously into a central vein and then guided through the chambers

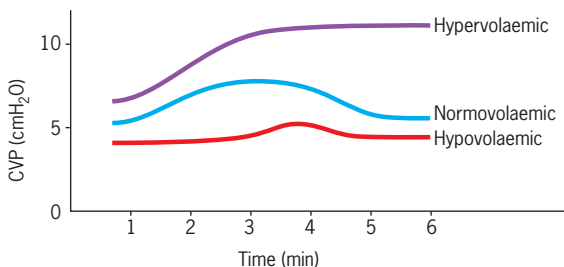


Fig. 11.1 The effect of rapid administration (200 mL of 0.9% saline over 1–3 minutes) of a fluid challenge to patients with a CVP within the normal range. (From Sykes MK (1963) Venous pressure as a clinical indication of adequacy of transfusion. *Annals of the Royal College of Surgeons of England* 33: 185–197.)

of the heart into the pulmonary artery. By inflating a balloon at the tip of the catheter, pulmonary artery wedge pressure (PAWP) is measured, which is a reflection of left atrial pressure.

- *Cardiac output* is measured, using a modified Swan–Ganz catheter, by recording temperature changes in the pulmonary artery after injecting a bolus of cold dextrose into the right atrium.

Respiratory failure (K&C p. 944)

Respiratory failure occurs when pulmonary gas exchange is sufficiently impaired to cause hypoxaemia with or without hypercapnia.

It can be divided into two types (Table 11.3):

- Type 1 respiratory failure is caused by a diffusion defect in the gas exchange area of the lung, ventilation/perfusion mismatch or right-to-left shunts (e.g. with cyanotic congenital heart disease). The P_{aO_2} is low (< 8 kPa) and the P_{aCO_2} is normal or low.
- Type 2 respiratory failure is caused by hypoventilation. The P_{aO_2} is low and the P_{aCO_2} is high (> 7 kPa).

Monitoring

Clinical Assessment should be made on the following criteria: tachypnoea, tachycardia, sweating, pulsus paradoxus,

Table 11.3

Causes of respiratory failure

Type 1	Type 2
Pulmonary oedema	COPD
Pneumonia	Severe asthma
Asthma	Muscle weakness, e.g. Guillain–Barré syndrome
COPD	Respiratory centre depression, e.g. with sedatives
Pulmonary embolism	Chest wall deformities
Acute respiratory distress syndrome	
Fibrosing alveolitis	
Right-to-left cardiac shunts	

use of accessory muscles of respiration, and inability to speak. Signs of carbon dioxide retention may be present, such as asterixis (coarse tremor), bounding pulse, warm peripheries and papilloedema.

Pulse oximetry Lightweight oximeters placed on an earlobe or finger can give a continuous reading of oxygen saturation by measuring the changing amount of light transmitted through arterial blood. In general, if the saturation is greater than 90% oxygenation can be considered to be adequate. Although simple and reliable, these instruments are not very sensitive to changes in oxygenation. They also give no indication of carbon dioxide retention.

Arterial blood gas analysis Analysis of arterial blood gives definitive measurements of P_aO_2 , P_aCO_2 , oxygen saturation, pH and bicarbonate (p. 738). In type 2 respiratory failure, retention of carbon dioxide causes P_aCO_2 and $[H^+]$ to rise, resulting in respiratory acidosis. The kidney compensates by retaining bicarbonate, reducing the $[H^+]$ towards normal. In type 1 respiratory failure or in hyperventilation there may be a fall in P_aCO_2 and $[H^+]$, resulting in respiratory alkalosis. Other abnormalities of acid–base balance are discussed on page 302.

Capnography This allows the continuous breath-by-breath analysis of expired carbon dioxide concentrations,

and is mandatory in patients having tracheal intubation outside the ITU.

Management

This includes the administration of supplemental oxygen, control of secretions, treatment of pulmonary infection, control of airway obstruction and limiting pulmonary oedema. In most patients oxygen is given by a face mask or nasal cannulae. With these devices, inspired oxygen concentration varies from 35% to 55%, with flow rates between 6 and 10 litres. However, in patients with chronically elevated carbon dioxide (e.g. COPD), hypoxia rather than hypercapnia maintains the respiratory drive and thus fixed-performance masks (e.g. Venturi masks) should be used, in which the concentration of oxygen can be accurately controlled. Respiratory stimulants such as doxapram have a very limited role in treatment.

Respiratory support Respiratory support should be considered when the above measures are not sufficient. The type depends on the underlying disorder and its clinical severity. Careful consideration should be given to ventilating patients with severe chronic lung disease, as those who are severely incapacitated may be difficult to wean from the ventilator.

- *Continuous positive airway pressure (CPAP)*. Oxygen is delivered to the spontaneously breathing patient under pressure via a tightly fitting face mask (non-invasive positive-pressure ventilation, NIPPV) or endotracheal tube. Oxygenation and vital capacity improve and the lungs become less stiff.
- *NIPPV* has been shown to be of use in patients with hypercapnic respiratory failure secondary to acute exacerbations of COPD who do not require immediate intubation and ventilation. NIPPV should be instituted at an early stage in the hospital admission when the pH falls below 7.35 and the respiratory rate exceeds 30 breaths per minute. NIPPV is usually given for at least 6 hours a day, and oxygen is administered to maintain arterial oxygen saturation above 90%. NIPPV

Respiratory failure

reduces the need for intubation, complications, mortality and hospital stay.

- *Intermittent positive-pressure ventilation (IPPV)*. IPPV requires tracheal intubation and therefore anaesthesia if the patient is conscious. The beneficial effects of IPPV (Table 11.4) include improved carbon dioxide elimination, improved oxygenation, and relief from exhaustion as the work of ventilation is removed. High concentrations of oxygen (up to 100%) may be administered accurately. If adequate oxygenation cannot be achieved, a positive airway pressure can be maintained at a chosen level throughout expiration by attaching a threshold resistor valve to the expiratory limb of the circuit. This is known as positive end-expiratory pressure (PEEP), and its primary effect is to re-expand underventilated lung areas, thereby reducing shunts and increasing P_{aO_2} .
- *Intermittent mandatory ventilation (IMV)*. This technique allows the ventilated patient to breathe spontaneously between mandatory tidal volumes delivered by the ventilator. These coincide with the patient's own respiratory effort. It is used as a method of weaning patients from artificial ventilation, or as an alternative to IPPV.

The major complications of intubation and assisted ventilation are:

- Trauma to the upper respiratory tract from the endotracheal tube
- Secondary pulmonary infection
- Barotrauma – overdistension of the lungs and alveolar rupture may present with pneumothorax (p. 501) and surgical emphysema
- Reduction in cardiac output – the increase in intrathoracic pressures during controlled ventilation impedes cardiac filling and lowers cardiac output.

Table 11.4
Indications for IPPV

Indication	Comment
Acute respiratory failure	Particularly when exhaustion, confusion, agitation or decreased consciousness are present
Acute ventilatory failure, e.g. myasthenia gravis, Guillain-Barré syndrome	Institute when vital capacity fallen to 10–15 mL/kg
Prophylactic postoperative ventilation	In poor-risk patients
Head injury	With acute brain oedema. Intracranial pressure is decreased by elective hyperventilation as this reduces cerebral blood flow
Trauma	e.g. Chest injury and lung contusion
Severe left ventricular failure	
Coma with breathing difficulties	e.g. Following drug overdose



Acute lung injury/acute respiratory distress syndrome (K&C p. 951)

Acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) are defined as respiratory distress occurring with stiff lungs, diffuse bilateral pulmonary infiltrates, refractory hypoxaemia, in the presence of a recognized precipitating cause and in the absence of cardiogenic pulmonary oedema (i.e. the pulmonary capillary wedge pressure is less than 16 mmHg).

Aetiology

The commonest precipitating factor is sepsis. Other causes include trauma, burns, pancreatitis, fat or amniotic fluid embolism, aspiration pneumonia or cardiopulmonary bypass.

Pathophysiology

The cardinal feature is pulmonary oedema as a result of increased vascular permeability caused by the release of inflammatory mediators. Oedema may induce vascular compression resulting in pulmonary hypertension, which is later exacerbated by vasoconstriction in response to increased autonomic nervous activity. A haemorrhagic intra-alveolar exudate forms which is rich in platelets, fibrin and clotting factors. This inactivates surfactant, stimulates inflammation and promotes hyaline membrane formation. These changes may result in progressive pulmonary fibrosis.

Clinical features

Tachypnoea, increasing hypoxia and laboured breathing are the initial features. The chest X-ray shows diffuse bilateral shadowing, which may progress to a complete 'white-out'.

Management

This is based on the treatment of the underlying condition. Pulmonary oedema should be limited with fluid restriction, diuretics, and haemofiltration if these measures fail.

Steroids currently have no role in the prophylaxis of this condition, but may be beneficial when administered during the late fibroproliferative phase. Aerosolized surfactant, inhaled nitric oxide and aerosolized prostacyclin are experimental treatments whose exact role in the management of ARDS is unclear.

Prognosis

Although the mortality has fallen over the last decade, it remains at 30–40%, most patients dying from sepsis. The prognosis is very dependent on the underlying cause, and rises steeply with age and with the development of multi-organ failure.

WESMOSIS ©

WESMOSIS@YAHOO. DK