People from many different backgrounds, with an enormous variety of problems, present to an emergency department (ED) both by day and by night. Fortunately, certain basic principles are applicable to the care of them all.

**WHAT TO DO IN AN EMERGENCY**

In all cases, swift and accurate assessment must immediately lead to appropriate action:

- **Cardiac arrest** → p. 150.
- **Children** → p. 329.

**A – Airway**

The airway may be:

- patent, partially obstructed or completely obstructed (this results from physical obstruction or loss of muscle tone)
- adequately protected or at risk (this depends on the protective reflexes of the airway).

**Check for responsiveness**

Is the patient alert and responsive to questions? A verbal reply confirms that there is:

- a maintained and protected airway
- temporarily adequate breathing and circulation
- cerebral functioning.

If responsive, then the patient will usually be able to elaborate on the cause of the sudden deterioration that has brought him or her to an ED.

Failure to respond indicates a significantly lowered level of consciousness and therefore an airway that may be obstructed and is definitely at risk. There may be a need for airway-opening manoeuvres and action to protect the airway.

**Look, listen and feel for breathing**

The absence of breath sounds indicates the need to attempt airway-opening manoeuvres (see below) and if unsuccessful to consider the possibility of a foreign body obstruction.

Foreign body obstruction may initially present as a distressed, very agitated, cyanosed patient – ‘choking’.

- **Cardiorespiratory arrest** → p. 150.
- **Choking** → p. 196.
- **Respiratory arrest** → p. 199.

If breathing is present then do the following.

**Look for the signs of partial upper airway obstruction**

- **Snoring**: the familiar sound of obstruction caused by the soft tissues of the mouth and pharynx. Often it accompanies the reduced muscle tone of a lowered level of consciousness.
- **Rattling or gurgling**: the sound of fluids in the upper airway.
- **Stridor**: a harsh, ‘crowing’ noise, which is heard best in inspiration. It is thus different from
Clearance and maintenance of the airway

A patent airway is a prerequisite for life; a blocked airway is a common harbinger of death in emergency situations. There are two main ways in which the airway becomes blocked.

1 **Depressed level of consciousness:** most common cause. The tone of the muscles controlling the patency of the mouth and the pharynx is under neural control, in much the same way as the activity of the other striated muscles of the body. When this control is lost, the soft tissues around the airway prolapse and fail to maintain patency (simplistically, the tongue falls back).

This is overcome by:

- tightening these tissues (chin-lift manoeuvre)
- pushing the jaw and the hyoid bone and their attached soft tissues forward (jaw-thrust manoeuvre)
- putting an artificial airway down the anatomical airway (oro- or nasopharyngeal airways, endotracheal tubes, laryngeal masks, etc. → Figure 1.1).

2 **Physical obstruction:** many things can do this (direct trauma, external or intramural mass, etc.)

Cyanosis and reduced haemoglobin saturation readings on a pulse oximeter are very late signs of airway obstruction.

Drooling: the inability to swallow saliva. It suggests blockage at the back of the throat.

Hoarseness: gross voice change. This suggests obstruction at the level of the larynx.

In cases of suspected supraglottic swelling, examination or instrumentation of the throat should not be carried out for fear of causing complete obstruction.

Assess the need for cervical spine protection before any airway intervention.

**Figure 1.1** Visualisation of the larynx.

Allergic reactions → p. 299.
Laryngotracheal obstruction → p. 196.
Surgical airways → p. 22.
However, in emergency practice, there is usually either something in the airway (vomitus, blood or foreign body) or there is swelling in the wall of the airway (oedema, haematoma, etc.).

This is overcome by:

- removing the cause of the obstruction (suction, manual removal or choking manoeuvres)
- passing an artificial airway (as detailed above) past the obstruction
- reducing the swelling with vasoconstrictor drugs (adrenaline)
- bypassing the obstruction with a surgical airway.

**Protection of the airway**

The airway is normally kept clear of foreign matter by the gag, cough and laryngeal reflexes. These may be attenuated by specific palsies, the effects of drugs or a generalised depression of conscious level. They may also be impaired at the extremes of age and in states of general debilitation. Special vigilance is required in all such situations; the recovery position should be used whenever possible.

Paradoxically, these same reflexes may make advanced airway care extremely difficult in situations where they are not completely absent. At such times, the airway should be managed by a person skilled in both its assessment and the use of sedating and paralysing drugs.

Over 10% of normal individuals have no gag reflex.

Laryngospasm, bleeding, vomiting and consequent hypoxia can result from ill-judged attempts at intubation. It should be noted that the absence of the gag reflex is not a good predictor of the need for (or the ease of) endotracheal intubation.

In a patient with a reduced level of consciousness, the airway must be assumed to be at risk until proved otherwise.

On-going protection of the airway requires continual vigilance. The following are also essential:

- The recovery position uses gravity, both to drain fluid matter away from the airway and to allow the soft tissues to be positioned in such a way that they do not cause obstruction. Once the airway is clear, this position can be used to both maintain and protect the airway.
- A high-flow suction catheter must always be near the patient’s head.
- The patient’s trolley must be capable of tilting ‘head down’ so as to drain vomitus out of the airway.
- If endotracheal intubation is attempted, the airway must be protected by the manoeuvre known as cricoid pressure throughout the period of instrumentation. Pressure is applied to the front of the patient’s cricoid cartilage by an assistant using the thumb and two fingers. This compresses the oesophagus against the cervical spine and thus prevents the passive regurgitation of gastric contents. The airway is vulnerable from the start of induced paralysis until the cuff is inflated on a correctly positioned endotracheal tube.

**Protection of the cervical spine**

If the patient has an injury to the cervical spine, there is a risk of damage to the spinal cord during the procedures needed to maintain the airway. Because of the terrible outcome of such damage, it is mandatory to protect the neck immediately in patients who are:

1. unresponsive with a history of trauma or no clear history
2. suffering from multiple trauma
3. difficult to assess
4. showing any symptoms or signs that might be attributable to the cervical spine.

Adequate protection of the potentially unstable cervical spine consists of a rigid collar and either a purpose-made cervical immobiliser or sandbags and tape.

*Exclusion of cervical spine injury* → p. 55.

**B – Breathing**

Breathing is the means by which oxygen is delivered to the alveoli and thus made available to the circulating red cells. At the same time carbon dioxide (CO₂) is eliminated.

**Look for**

- Difficulty in talking
- Abnormal respiratory rate: usually fast, laboured breathing. Very slow respiratory rates may occur just before respiratory arrest or as a consequence of poisoning with narcotic drugs, e.g. methadone
- Nasal flaring and use of shoulder and neck muscles.
- **Paradoxical respiration**: a see-sawing movement of the chest and abdomen, which indicates obstruction of either the upper or lower airways or fatigue of the diaphragm.

- **In children, recession of the chest wall**: indrawing of the elastic tissues caused by increased respiratory effort.

All the above suggest that the patient is struggling to achieve normal respiration. Failure to oxygenate the blood adequately and hence the tissues is shown by:

- **Tachycardia** – the nervous system has detected hypoxia and is stimulating the heart
- **Pallor and sweating** – caused by sympathetic stimulation
- **Cyanosis** – a late sign
- **Irritability, confusion or reduced responsiveness** – the brain is short of oxygen. This is an extremely worrying sign
- **A low SaO₂ (<94%)** – pulse oximetry should be established as soon as possible
- **Unequal, diminished or abnormal breath sounds**
- **Hyperresonance or dullness to percussion**
- **Displacement of the trachea or apex beat**
- **A flail segment**

**Allergic reactions → p. 299.**
**Chest decompression and drainage → p. 74.**
**Chest injuries → p. 72.**
**Respiratory distress → p. 196.**

**Oxygen therapy**

The common denominator of all life-threatening illness, regardless of cause, is a failure to deliver adequate amounts of oxygen to the tissues. In normal circumstances, the oxygen content of atmospheric air (21%) is perfectly adequate but when the mechanisms for breathing are diseased or traumatised supplemental oxygen should be given. The physiological compensatory mechanisms for hypoxia and hypovolaemia all consume oxygen themselves; the immediate administration of supplemental oxygen may maintain these reflexes while more definitive measures are put in place. There are really only two main types of oxygen therapy:

1. **High-concentration oxygen (40–100%)**
2. **Low-concentration oxygen (24–30%).**

The dangers of high-concentration therapy are known to every medical student. The patient with a chronically raised blood CO₂ level may depend on a hypoxic drive to stimulate breathing – give him or her oxygen and the breathing slows, CO₂ levels rise even higher and the patient becomes comatose with CO₂ narcosis. In practice, these patients are a small group in whom the speed of onset of symptoms can be used to determine treatment (→ pp. 199 and 205).

Hypoxia is a swift killer and so patients in the resuscitation room invariably require a high concentration of oxygen. The use of a mask that has a reservoir bag improves the effectiveness of oxygen delivery (to perhaps 60–80%) and should be standard. (The reservoir bag is needed because a patient’s inspiratory flow is always greater than the 15 L/min maximum flow from the oxygen supply.) Blood gases must be obtained at an early stage to monitor the effect of supplemental oxygen. If improvement is not satisfactory, then ventilation may be needed. Continuous positive airway pressure (CPAP) is another method of increasing oxygenation.

**Mechanical ventilation**

This should always be considered when:

- the patient cannot maintain a clear airway
- oxygen enrichment of the inspired gases fails to prevent the signs of cerebral hypoxia
- CO₂ narcosis is present
- there has been a successful but prolonged resuscitation from cardiac arrest
- the patient is multiply injured
- the patient has a severe chest injury (particularly multiple rib fractures and/or flail segments)
- the patient is to be transferred and there is a risk of severe deterioration en route.

The emergency induction of anaesthesia for the purpose of intubation and ventilation in a hypoxic patient is a difficult and demanding task. It requires considerable anaesthetic skills.

A pneumothorax is more likely to tension in a ventilated patient. Chest drains must be inserted before ventilating patients with chest injuries.
Oxygen is a treatment for hypoxaemia, not breathlessness. Oxygen has not been shown to have any effect on the sensation of breathlessness in non-hypoxaemic patients. The essence of this guideline can be summarised simply as a requirement for oxygen to be prescribed according to a target saturation range and for those who administer oxygen therapy to monitor the patient and keep within that target saturation range.

The guideline recommends aiming to achieve normal or near-normal oxygen saturation for all acutely ill patients apart from those at risk of hypercapnic respiratory failure or those receiving terminal palliative care. The recommended target saturation range for acutely ill patients not at risk of hypercapnic respiratory failure is 94–98%. However, some normal individuals, especially people aged over 70 years, may have oxygen saturation measurements below 94% and still do not require oxygen therapy when clinically stable. For most patients with known chronic obstructive pulmonary disease (COPD) or other known risk factors for hypercapnic respiratory failure (e.g. morbid obesity, chest wall deformities or neuromuscular disorders), a target saturation range of 88–92% is recommended pending the availability of blood gas results.

Supplementary oxygen therapy is required for all acutely hypoxaemic patients and for many other patients who are at risk of hypoxaemia, including patients with major trauma and shock. Most acutely breathless patients will require supplementary oxygen therapy, but there are some conditions such as acute hyperventilation or diabetic ketoacidosis in which an apparently breathless patient will not benefit from oxygen therapy. Conversely, there are some other clinical situations such as carbon monoxide poisoning where a patient may benefit from oxygen therapy despite a lack of hypoxaemia or breathlessness because carbon monoxide binds more avidly than oxygen to the haemoglobin molecule. Similarly, high-concentration inhaled oxygen can increase the rate of reabsorption of gas from a pneumothorax by up to fourfold. For this reason, the British Thoracic Society (BTS) guideline on the management of pneumothorax recommends the use of high-concentration oxygen (by reservoir mask) in all non-COPD patients who require hospital admission for observation due to a moderate-sized pneumothorax that does not require drainage.

C – Circulation

Check for a central pulse (over 5 seconds)

The absence of a central pulse (or a rate of <60 beats/ min in infants) indicates the need to follow procedures for cardiorespiratory arrest:

Arrest rhythms:
- Asystole → p. 150.
- Pulseless electrical activity (PEA) or electromechanical dissociation (EMD) → p. 151.
- Ventricular fibrillation (VF) → p. 150.

Look for
- Pallor and coolness of the skin – the body diverts blood away from the skin when there are circulatory problems and these signs are thus very useful indicators of shock.
- Pallor and sweating – signs of gross sympathetic disturbance.
- Active bleeding or melaena
- A fast or slow heart rate – fast heart rates usually mean that either there is a cardiac arrhythmia or more commonly the sympathetic nervous system has detected a problem with the body (such as hypoxia, hypoglycaemia, pain or fear) and is ‘instructing’ the heart to beat faster. A slow heart rate usually means that something is wrong with the heart itself. The worst cause of this is severe hypoxia (or hypovolaemia) and, in this case, it means that terminal bradycardia and asystole are only seconds away.
- Abnormal systolic blood pressure
- A raised capillary refill time – it should be less than 2s if the circulation is satisfactory. However, peripheral shutdown in a cold, wet patient can easily produce a prolonged refill time.
- Absent or quiet heart sounds and raised jugular venous pulse (JVP) – suggestive of tamponade if
accompanied by hypotension and tachycardia; JVP will not be raised if there is also hypovolaemia.

- A precordial wound
- An abnormal electrocardiogram (ECG) trace on the monitor
- Signs of left ventricular failure (dyspnoea, gallop rhythm and crepitations)
- Signs of abdominal, pelvic or occult bleeding (may need per rectum examination and a nasogastric tube or ultrasound scan)
- Signs of dehydration (especially in children)
- Purpura (meningococcal septicaemia)

Inadequate circulation will reduce tissue oxygenation and thus may also cause:

- a raised respiratory rate
- altered mental status.

Bolus fluid therapy should be calculated at 20 mL/kg and repeated as necessary after further assessment. (Reduced to 10 mL/kg for patients with bleeding after trauma in hospital and no more than 5 mL/kg for patients with trauma in the prehospital setting → p. 7.)

**Abdominal bleeding** → pp. 81 and 308.

**Allergic reactions** → p. 299.

**Anaphylaxis** → p. 299.

**Blood transfusion** → p. 25.

**Cardiac arrhythmias** → p. 164.

**Cardiac failure** → pp. 208 and 238.

**Cardiac tamponade** → pp. 78 and 238.

**Emergency thoracotomy** → p. 80.

**Pelvic bleeding** → p. 84.

**Renal effects of shock** → p. 247.

**Shock** → p. 24.

**Cardiac function**

The stroke volume is the amount of blood ejected from the heart with each beat. It is determined by the left ventricular filling pressure, myocardial contractility and systemic vascular resistance. The product of heart rate and stroke volume is the cardiac output – the most important parameter of cardiac function. (Cardiac index is cardiac output divided by body surface area.) An increase in heart rate will directly increase the cardiac output and is the earliest cardiac response to hypoxia. However, the faster the heart beats the less time there is for it to fill and, eventually, a rise in heart rate will no longer be matched by a rise in cardiac output.

Myocardial function is compromised at high pulse rates because coronary blood flow occurs chiefly in diastole. When the heart rate rises above about 130 in an adult, the filling time is so reduced that cardiac output will actually fall.

**Pulse and blood pressure**

The autonomic response to hypovolaemia is complex. Rapid blood loss produces a reflex bradycardia, but, when associated with tissue damage, it produces the more familiar tachycardia. Systolic blood pressure is the product of the cardiac output and the systemic vascular resistance. A high catecholamine response to hypoxia and hypovolaemia will produce a high systemic vascular resistance. This will maintain a ‘normal’ blood pressure in the presence of a falling cardiac output.

Blood pressure when measured by a cuff depends on the production of Korotkoff’s sounds by turbulent flow in the artery. When systemic vascular resistance is high and flow through the artery is relatively low (e.g. in the immediate aftermath of cardiac arrest), these sounds may not be heard easily, even when the mean arterial pressure is good. Great care must be taken when deciding if such patients are beyond salvage or have pulseless electrical activity.

Maintenance of systemic vascular resistance is a vital response to hypovolaemia and hypoxia. (Skin pallor reflects this early on but is an imprecise clinical sign.) Similar to other compensatory mechanisms to hypoxia, this vasomotor response consumes oxygen and will eventually fail.

Measurements of pulse and blood pressure are very poor indicators of haemodynamic function in critically ill patients. Central venous pressure may not reflect the functioning of the left side of the heart and is thus of limited use in the assessment of overall cardiac performance. Indwelling pulmonary artery flotation and systemic arterial catheters will provide much more useful information and allow measurement of the cardiac output. Critically ill patients should be moved to an intensive care unit where such monitoring facilities are available as soon as possible.
Direct ultrasound measurement of cardiac output may soon become routine in EDs, but this facility must not delay consultation with intensivists.

**Fluid replacement**

Left ventricular filling pressure (and hence cardiac output) is a function of the circulating blood volume. Increases in heart rate, systemic vascular resistance and myocardial contractility can maintain cardiac output and blood pressure in the early stages of hypovolaemia. However, this will be at the expense of increased oxygen demands by the cardiovascular system and reduced tissue perfusion in many other areas. The rapid restoration of circulating volume will prevent a sudden failure of these mechanisms and an often irreversible fall in cardiac output.

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**Early restoration of blood pressure by transfusion does not necessarily indicate correction of the circulatory deficit.**

The delivery of oxygen to the tissues depends not only on the pumping mechanism of the heart but also on the red cells in the circulating blood. A modest fall in haematocrit can reduce viscosity and increase blood flow while maintaining oxygen delivery, but will still require an increase in cardiac output to be effective. Maintenance of haemoglobin levels by blood transfusion reduces the impact of hypoxia by increasing the effectiveness of each cardiac cycle and reducing the need for an increase in cardiac output. Transfused blood will also maintain the oncotic pressure of the circulating fluid, thereby increasing the filling pressure. However, adequate levels of 2,3-diphosphoglycerate (2,3-DPG) are also necessary for satisfactory oxygen delivery → p. 26.

Maintenance of adequate tissue perfusion is not synonymous with the return of a normal blood pressure. Indeed, the latter may be contraindicated in the ED in an actively bleeding patient (e.g. with an aortic aneurysm). Resuscitation can be achieved while keeping the blood pressure relatively low. This has been shown to improve survival until definitive surgery can be undertaken. A similar approach is recommended in the prehospital management of injured adults and older children with presumed blood loss. Intravenous (IV) fluids should not be administered if a radial pulse can be felt (or, in the case of penetrating torso injuries, if a central pulse can be felt). In the absence of these pulses, IV crystalloids should be administered, en route to hospital, in boluses of no more than 250 mL until the relevant pulse becomes palpable. The same advice is probably applicable for young children and infants also, in which case boluses of 5 mL/kg should be used.

When rapid fluid replacement is required, warmed IV fluids (40°C) should be used. A ratio of crystalloids to colloids of at least 50:50 has been shown to be safe. Many doctors believe that Hartmann’s solution is preferable to 0.9% saline if large volumes are to be given. The role of hypertonic saline in resuscitation looks promising but has yet to be fully established.

The management of massive blood loss is summarised in Figures 1.2 (adults) and 1.3 (children).

**D – Disability**

After A, B and C (airway, breathing and circulation) have been attended to, it is necessary to look at the state of the brain. In this context, the term **disability** is now widely used to describe a brief assessment of neurological functioning.

**Look for**

1 A reduced level of consciousness – this is the most important sign of any problem affecting the brain. AVPU scoring is useful initially:
   - **A** Alert
   - **V** Voice elicits a response
   - **P** Pain elicits a response. (Attending relatives are usually in a highly distressed state so be careful how you elicit this sign. Pressure on a fingernail [with a pencil] is probably the most subtle way.)
   - **U** Unresponsive

   Later, the Glasgow Coma Scale (GCS) should be used (→ p. 34 for adults and → p. 332 for children).

   **Always consider hypoglycaemia as a cause for a reduced level of consciousness.**

2 Abnormal pupils – look for size, equalness and reactivity. These features can be affected by both drugs and brain disease.

3 Abnormal posture and limb movements.

Severe intracerebral problems may also cause:
- airway obstruction
- respiratory depression (respiration, unlike the heart beat, requires an intact brain stem)
- bradycardia and hypertension (Cushing’s response)
- neurogenic pulmonary oedema (caused by massive sympathetic vasoconstriction).
What every emergency physician must know

Figure 1.2 The management of major haemorrhage in adults. Reproduced with the kind permission of Dr Kate Pendry, Consultant Haematologist, on behalf of the North West Regional Transfusion Committee.
Ensure a consultant is aware of the massive haemorrhage and a senior member of staff is available to take charge of resuscitation if not already present.

Insert local arrangements:
- activation tel number(s)
  - Emergency O red cells – location of supply
  - Time to receive at this clinical area:
    - Group-specific red cells
    - XM red cells
  - Insert local arrangements:
    - activation tel number(s)

STOP THE BLEEDING

Haemorrhage control
- Direct pressure/ tourniquet if appropriate
- Stabilise fractures
- Surgical intervention (consider damage limitation surgery)
- Interventional radiology
- Endoscopic techniques

Haemostatic drugs
- Tranexamic acid 20 mg/kg bolus over 10 min (max 1.5 g) and 10 mg/kg per h infusion
- Vitamin K and prothrombin complex concentrate for warfarinised patients

Other haemostatic agents: discuss with consultant haematologist

Thromboprophylaxis should be considered when patient stable

(A)BG – (arterial) blood gas
APTT – activated partial thromboplastin time
FFP – fresh frozen plasma
MHP – massive haemorrhage pack
NPT – near patient testing
PT – prothrombin time
XM – cross-match

What every emergency physician must know

Figure 1.3 The management of major haemorrhage in children (also Tables 1.1 and 1.2). Reproduced with the kind permission of Dr Kate Pendry, Consultant Haematologist, on behalf of the North West Regional Transfusion Committee.
Table 1.1 Major haemorrhage pack 1 (MHP 1) order these volumes, which are also the maximum volumes to be administered from each pack in each weight category

<table>
<thead>
<tr>
<th>Weight (kg)</th>
<th>Red cells: group O, group specific, cross-matched depending on availability</th>
<th>FFP</th>
<th>Platelets</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;5</td>
<td>2 paediatric units (80–100mL)</td>
<td>2 ‘neonatal’ units of MB-treated FFP (100mL)</td>
<td>1 paediatric pack of platelets (50mL)</td>
</tr>
<tr>
<td>5–10</td>
<td>1 adult unit (250mL)</td>
<td>1 paediatric unit MB-treated FFP (225mL)</td>
<td>2 paediatric packs of platelets (100mL)</td>
</tr>
<tr>
<td>10–20</td>
<td>2 adult units (500mL)</td>
<td>2 paediatric units MB-treated FFP (450mL)</td>
<td>1 adult apheresis pack (200mL)</td>
</tr>
<tr>
<td>&gt;20</td>
<td>4 adult units (1000mL)</td>
<td>4 paediatric units MB-treated FFP (900mL)</td>
<td>1 adult apheresis pack (200mL)</td>
</tr>
</tbody>
</table>

FFP, fresh frozen plasma; MB, methylene blue.

Calculate volumes to be administered as detailed in Figure 1.3, but do not exceed these maxima (see example below Table 1.2).

Table 1.2 Major haemorrhage pack 2 (MHP 2) order these volumes, which are also the maximum volumes to be administered from this pack in each weight category

<table>
<thead>
<tr>
<th>Weight (kg)</th>
<th>Red cells</th>
<th>FFP</th>
<th>Cryoprecipitate – request if fibrinogen &lt;1g/L or according to TEG/ROTEM</th>
<th>Platelets</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;5</td>
<td>2 paediatric units (80–100mL)</td>
<td>2 ‘neonatal’ units of MB-treated FFP (100mL)</td>
<td>1 single donor unit MB treated (40mL)</td>
<td>1 paediatric pack of platelets (50mL)</td>
</tr>
<tr>
<td>5–10</td>
<td>1 adult unit (250mL)</td>
<td>1 paediatric unit MB-treated FFP (225mL)</td>
<td>2 single donor units (80mL)</td>
<td>2 paediatric packs of platelets (100mL)</td>
</tr>
<tr>
<td>10–20</td>
<td>2 adult units (500mL)</td>
<td>2 paediatric units MB-treated FFP (450mL)</td>
<td>1 pool (5 units) (200mL)</td>
<td>1 adult apheresis pack (200mL)</td>
</tr>
<tr>
<td>&gt;20</td>
<td>4 adult units (1000mL)</td>
<td>4 paediatric units MB-treated FFP (900mL)</td>
<td>2 pools (10 units) (400mL)</td>
<td>1 adult apheresis pack (200mL)</td>
</tr>
</tbody>
</table>

FFP, fresh frozen plasma; MB, methylene blue; TEG/ROTEM, thromboelastography.

Calculate volumes to be administered as detailed in Figure 1.3, but do not exceed these maxima (see example). An example: in a 5-kg child, you may administer up to 200 mL red blood cells (RBCs: 40 mL/kg) and 50 mL platelets (10 mL/kg); however, in a 30-kg child do not administer more than 4 adult units of RBCs (33 mL/kg) or 1 adult therapeutic dose of platelets (6 mL/kg).

Brain-stem death → p. 386.
Head injury → p. 33.
Hypoglycaemia → p. 240.
Intracranial pathology → p. 223.
Poisoning → p. 268.

**Depression of consciousness**

A decreased level of consciousness indicates that something is wrong with the brain or its fuel supply → Box 1.2. There is a continuum of consciousness that ranges from an alert and oriented patient to one with brain-stem death.

Unconsciousness is an imprecise term usually describing a condition of an unaware patient with whom verbal communication is not possible; unresponsive is thus a better description. Such patients will usually be amnesic for the duration of the unresponsiveness.

The ability to maintain the airway decreases as the coma score falls and finally the ability to protect the airway is also lost. Breathing indicates a functioning...
What every emergency physician must know

Loss of the protective reflexes of the eyes
Areas where pressure sores might form (→ p. 222)
The proximity of the next of kin.

Attention to these details early on can radically change the wellbeing (and demeanour) of a patient.

Box 1.2 Causes of impaired consciousness

- Hypoxia, hypovolaemia or cerebral ischaemia
- Hypoglycaemia
- Hypothermia
- Poisoning or gross metabolic disturbance (including CO₂ narcosis)
- Injury to the brain
- Intracranial pathology (bleeding, thrombosis, embolism, infection, swelling, tumour, fits, etc.)

If prolonged, many of the above problems (including hypoxia, ischaemia, hypoglycaemia and status epilepticus) will lead to a remarkably similar outcome – selective neuronal necrosis and permanent brain damage.

brain stem; in an arrested patient it often returns quickly after cerebral circulation is restored. Sudden cerebral trauma may cause transitory apnoea and all of the causes of impaired consciousness listed in Box 1.2 may lead to terminal apnoea. The heart beat is less immediately dependent on an intact brain but asystole is inevitable within hours of brain-stem death.

Hyperpyrexia → p. 245.
Hyperthermia → pp. 245 and 271.
Hypothermia → p. 243.

F – Fits

Fits deprive the brain of oxygen and make assessment almost impossible. Stopping them is as important as the ABCs.

Look for

- Frank tonic or clonic activity
- Spasmodic twitching
- Post-ictal drowsiness
- Gurgling, rattling or other signs of post-ictal airway obstruction
- Cyanosis: there is increased demand for oxygen and also respiratory distress
- Signs of head injury
- Signs of other injury caused by a convulsion (e.g. a bitten tongue and intraoral bleeding)
- Reasons to consider hypoglycaemia
- Pyrexia or other signs of infection (especially in children).

Convulsions must be terminated before any further action can be effective. Meanwhile, the patient must be prevented from harming him- or herself.

Convulsions in adults → p. 231 and in children → p. 343
Hypoglycaemia → pp. 240 and 333.

Fitting indicates that something is wrong with the brain or its fuel supply. The list of possibilities is almost the same as that for causes of reduced consciousness → Box 1.3. Convulsive activity causes a dramatic increase in cerebral and muscle oxygen demand; a post-ictal acidosis is inevitable. The uncoordinated muscle action that occurs during the tonic or clonic stages of a fit makes control of the airway extremely difficult; some regurgitation may also occur. Ventilation of the lungs is usually reduced for the same reason. Alveolar oxygenation is thus poor.

E – Environment and exposure

In cases of trauma, collapse and depressed conscious level, the whole body must be exposed – including the back – so that nothing important is missed. However, control of body temperature is important to successful resuscitation. So remove wet clothes and, if high volumes of IV fluids are to be given, they should be warmed. Even at this early stage avoid extrinsic factors that may harm the patient.

Look for

- Cold extremities
- Shivering
- Wet clothing
- Pyrexia and clamminess
- The position in which the patient is most comfortable
- Uncomfortable splints (including collars and spinal boards)
at a time of high oxygen demand. This combination explains why prolonged fitting is associated with permanent neurological damage.

**G – Glucose**

The human body can be compared to an engine that needs an oxygen supply (airway and breathing) delivered in the bloodstream (circulation). However, we should not forget that the oxygen is required to burn fuel (glucose). Fat and protein are, of course, also important but the brain uses glucose almost exclusively.

**Look for**

- Restlessness, agitation or other mental change ('jitteriness' in a neonate)
- Inappropriate lack of cooperation or aggression
- A reduced level of consciousness
- Convulsions
- Signs of insulin usage
- A low blood sugar level on testing with a reagent strip.

A reagent-strip measurement of blood glucose should be performed in all patients who have depression of consciousness. If hypoglycaemia is found it should be immediately treated with IV glucose solution (50 mL of 50% glucose for a normal adult, 0.2 g/kg for a child). If no venous access can be found, then glucagon 1 mg by intramuscular injection is a useful standby.

**Hypoglycaemia → pp. 240 and 333.**

**H – History**

At this juncture a brief history becomes a necessity and brief is AMPLE:

- **A** Allergies
- **M** Medication
- **P** Past and present illnesses of significance
- **L** Last food and drink
- **E** Events leading up to the patient’s presentation

The people who accompany the patient to the department are a vital source of this information, hence the need to collect facts before the paramedical staff leave the ED.

**Adrenocortical suppression**

Patients who are undergoing prolonged treatment with steroids (i.e. for more than 3 weeks) may develop adrenocortical suppression. This can also occur for up to a year after stopping long-term steroid therapy. During a medical crisis, such patients should be given supplementary corticosteroids (e.g. IV hydrocortisone 200 mg) in the ED.

**I – Immediate analgesia and investigations**

This is the point (if you have not already done it) to call for help. There should be no hesitation in seeking another pair of hands or a more experienced opinion.

In many patients who are not in extremis, the above will take only a matter of seconds. Once life-threatening problems have been identified and treated, it is necessary to perform the tasks that are at the very heart of emergency medicine – to ensure the immediate relief of suffering. This will include the following:

Hypoglycaemia is always waiting to catch you out. A comatose patient with profuse sweating should always make you think of a low blood sugar.
The needs of relatives and friends

The needs of relatives and friends cannot be ignored. These may vary from simple reassurance to medical treatment. As soon as practicable, the relatives must be informed of the patient’s current situation and what is going to happen next.

For a summary of this section on immediate assessment and management of emergency patients → Box 1.5.
FURTHER MANAGEMENT IN THE ED

How much history, how much examination?

History-taking in emergencies should be guided by the presenting complaint. The familial medical history will rarely be relevant in a patient with a dog bite; it might be vital in someone with haemophilia and a swollen joint. The art of adjusting the acquisition of information is a difficult one.

Most new ED staff take an over-long history for the first few days; they then record less than a bare minimum for the next few months. Mechanism of injury is particularly important in trauma; events that lead to the patient’s presentation, the timing and speed of onset are essential in medical cases.

The examination must also be tailored to the patient. At times it can be limited, but often it must be thorough. Experience teaches the relative uselessness of some physical signs and the enormous value of others. The examination in trauma patients is usually performed from top to toe rather than in systems and is called the secondary survey. The back of the patient and the perineum are the parts that are often missed.

Investigations

Investigations should be requested only if the results could have an impact on immediate care or disposal. Major radiographs are initially requested. In patients who have suffered multiple trauma, these are views of the chest, lateral cervical spine and pelvis. In other patients a chest radiograph (CXR) is usually sufficient. Twelve-lead ECG and blood gas analysis are also helpful early on.

Other tests should be performed for precise indications rather than as a general screen. The exception to this is in elderly patients who present with non-specific events such as collapse. They can be very difficult to evaluate clinically. Consequently, before considering sending them home, it is best to carry out a brief screen including CXR, ECG, haemoglobin, white cell count and blood urea level – and a pelvic radiograph if concerned about mobility.

Definitive care

This may involve:

- safe transport to another facility
- careful follow-up arrangements
- rehabilitation

Homelessness

The standardised mortality rate for homeless people is three times higher than that of the UK population as a whole.

Many patients who come to an ED have nowhere to sleep or to shelter. People with psychiatric disease often become homeless, and there are many other illnesses (such as tuberculosis and alcohol abuse) that are associated with homelessness.

The average life expectancy for someone on the streets is just 47 years.

Efforts should always be made to find temporary accommodation for these patients. The most likely sources of help are:

- the Social Services
- the Salvation Army
- Shelter Nightline (in London).

Prehospital care

Outside the hospital situation, there is a clear organisational hierarchy:

- The police are in overall control of the scene.
- The fire service is in charge of rescue and extrication, and of scene safety for the other personnel.
- The ambulance service is responsible for the evacuation of casualties.
- The medical team is present at the request of the ambulance service.

Entrapment of casualties is now the most common reason for paramedics to request the assistance of a hospital team – either prolonged entrapment (more
What every emergency physician must know

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(Note that the last is a legal obligation in the UK under the Civil Contingencies Act 2004. The Act is divided into two parts – Civil Protection (Part I) and Emergency Powers (Part II). For the purposes of Civil Protection, local responders are divided into two categories. Category 1 responders (which include hospitals, primary care trusts and ambulance services) have a duty to make and execute effective plans for major incidents. Category 2 organisations (such as utility and transport companies) are obliged to cooperate with other responders.)

Contamination and irradiation → p. 145.

SEDATION AND GENERAL ANAESTHESIA

Most ED patients are self-referred and many will return to their homes or workplaces within a matter of hours. Some patients arrive in a coma and others will require sedation. All are, by definition, unexpected. This combination of circumstances leads to special problems with the safe administration of sedative drugs:

• Most patients are not fasted.
• Painful conditions delay gastric emptying.
• Many patients will have ingested alcohol.
• There is no opportunity for preplanned assessment of fitness or review of case notes.
• The need for sedation is invariably immediate.
• The fitness of patients for discharge and the circumstances to which they depart require very careful consideration.

The techniques used for assessment and monitoring of pathological coma are applicable to the depression of consciousness that occurs during sedation. The safety of the patient is paramount; emergency situations do not obviate the need for standard precautions. Consideration must be given to the medical preparedness and fasting state of all patients. If in doubt, senior staff should be consulted.

Preparation for sedation and selection of patients

The immediacy of the situation does not obviate the need for a concise but adequate work-up of the patient. Most patients will not require investigations to assess their fitness for the procedure; those who do are generally not suitable for sedation in the ED. Pulse oximetry (while breathing air) is a useful screening test – a fit patient will invariably have an $\text{SaO}_2$ of $>94%$. 

than 20 min) or situations where release requires analgesia.

An on-site medical team must be formed from experienced, regularly trained staff who have:

• high-visibility protective clothing
• adequate equipment
• insurance cover for this type of work.

Furthermore, the parent department must be left adequately staffed. The only measures that have been shown conclusively to save lives in the prehospital situation are ABC:

Airway Clearance, maintenance and protection
Breathing Oxygen and ventilation
Circulation Chest compression and defibrillation

In cardiac arrest, the time taken to initiate the above interventions is crucial to survival → p. 151.

Extensive clinical examination and the establishment of IV infusions are of no proven benefit. Nevertheless, other prehospital treatments may contribute greatly to the relief of pain and suffering.

Time at the scene must not be extended by anything other than essential treatment. The priority is to get the patient to a major ED as soon as possible.

The basic principles of prehospital care are the same as those for in-hospital care. Specific resuscitation courses are now available where the applied skills may be mastered.

Major incidents

The management of major incidents is outside the scope of this book, but in essence the following should be implemented:

• All departments should have a written policy for dealing with events that have the potential to overwhelm the standard facilities of the hospital.
• Action cards for all personnel should be available together with a special supply of equipment and drugs.
• Regular practice sessions are essential.
Patients with the following problems are unsuitable for sedation in the ED:

- Severely intoxicated
- Full stomach
- Previous problems with sedation
- Chronic illnesses – may complicate sedation or aftercare
- Other severe injuries
- Coexistent significant head injury
- At the extremes of age
- Inadequate circumstances for discharge.

Patients who are deemed unsuitable for sedation but whose condition does not allow delay (e.g. vascular compromise distal to a dislocation) must be discussed with a senior colleague immediately.

The urgency of most of the conditions that present to an ED does not allow for the conventional period of preoperative fasting. However, in all but the most pressing circumstances, a period of 2 h should separate sedation from the last ingestion of food or drink to allow for gastric emptying. No further food or drink must be allowed from the moment of entering the department.

Painful conditions, such as dislocations, must be alleviated with parenteral analgesia (before the radiograph) and the subsequent doses of sedative drugs then adjusted accordingly.

**Conditions during the period of sedation**

During sedation, and until recovery is complete, all patients should:

- be accompanied by a responsible member of staff
- be on a trolley with side rails, which can be tipped head-down
- have an IV cannula in situ
- be given a high concentration of oxygen by mask
- be monitored by pulse oximetry as a minimum standard – respiratory rate, ECG and BP recording are also highly desirable
- have a high-volume suction catheter in place under their pillow.

The protective reflexes of the airway are depressed by all sedative and opioid/narcotic drugs to an unpredictable degree. Even at a high GCS there may be enough impairment of airway protection to allow aspiration of gastric contents. Therefore, all sedated patients must be placed in the recovery position as soon as is practically possible.

Effective sedation is greatly facilitated by pleasant, quiet surroundings and the presence of attentive, reassuring and obviously competent staff.

**Assessment of the level of sedation**

The scores for measuring coma (AVPU and GCS) must be understood and practised by all ED staff. They are ideal for use in assessing the level of sedation.

General anaesthesia and sedation are both induced states of depression of consciousness. Sedation is characterised by an ability to maintain verbal contact at all times. This does not mean that the patient either speaks sensibly or remembers the conversation afterwards. It is equivalent to ‘V’ on the AVPU scale and 10 or above on the GCS. It is certainly not the same as a GCS of 8 (coma). Full general anaesthesia is characterised by a GCS of 3. Gentle stroking of the upper eyelashes usually causes blinking (the eyelash reflex) and the loss of this reflex (at a GCS of around 8) is a good guide as to the imminent onset of general anaesthesia.

**Facilities for the administration of sedation or general anaesthesia**

The minimum requirements include the following:

- Medical and nursing staff trained in the management of patients with a depressed level of consciousness
- Areas suitable for high-dependency observation
- A full range of resuscitation and monitoring equipment
- All necessary drugs for resuscitation, including the specific benzodiazepine antagonist flumazenil and the opioid antagonist naloxone.

When a doctor is performing a procedure on a sedated patient, a separate member of staff must be responsible for the overall care of that patient. The operator should never try to monitor the patient at the same time.

**Drugs for sedation in the ED**

All drugs that are used for sedation are capable of inducing general anaesthesia and vice versa. The
Proper sedation with nitrous oxide requires a constant flow of gas and this is best achieved with a purpose-built system such as the Quantiflex machine. Using this apparatus, nitrous oxide may be administered in subanaesthetic concentrations of 30–70% via a non-re-breathing circuit with a guaranteed minimum of 30% oxygen.

Ketamine

Ketamine is a derivative of phencyclidine and is often recommended as an anaesthetic for prehospital use. This is because it has both sedative and analgesic effects and does not compromise the airway or the circulation as much as other comparable drugs. In a lower range of doses, it is also an excellent and safe sedative for use in the ED with a success rate of more than 90%. Contraindications to sedation with ketamine include:

- Age <12 months due to an increased risk of laryngospasm and airway complications (children aged between 12 and 24 months should receive ketamine sedation only from expert staff)
- High risk of laryngospasm (active respiratory infection, active asthma)
- Unstable or abnormal airway
- Active upper or lower respiratory tract infection
- Proposed procedure within the mouth or pharynx
- Significant cardiac disease
- Recent significant head injury or reduced level of consciousness
- Intracranial hypertension with cerebrospinal fluid (CSF) obstruction
- Intraocular pathology (glaucoma, penetrating injury)
- Uncontrolled epilepsy
- Hyperthyroidism or thyroid medication
- Porphyria
- Previous psychotic illness
- Severe psychological problems such as cognitive or motor delay or severe behavioural problems
- Prior adverse reaction to ketamine.

The recommended doses are as follows:

- IV dose of ketamine for sedation = 1 mg/kg. The sedation starts within 60s and lasts for 5–10min.
- Intramuscular (IM) dose of ketamine for sedation = 2.5 mg/kg. The onset of sedation is around 5min with an effective duration of up to 35min. A top-up dose of a further 1mg/kg can be given if either the first dose proves to be inadequate or there is a requirement to prolong the duration of the sedation. The effects of ketamine are surprisingly predictable and reliable when it is administered by the IM route.
In these low doses, laryngospasm and dysphoria are uncommon and excess salivation is not a problem. Consequently, emergence from sedation is usually uneventful and prior administration of atropine is unnecessary. However, around 5% of children will have transient clonic movements and the same number will vomit during the recovery phase.

**Discharge of patients who have received sedative drugs in the ED**

The patient who is fit for discharge after sedation must fulfil all of the following criteria:

- Alert and oriented
- Able to walk steadily and unaided
- Able to drink liquids
- Not suffering from any disabling condition such as
  - vomiting
  - dizziness
  - shortness of breath
  - severe pain
- Accompanied by a responsible adult
- Suitable for the available transport
- Returning to adequate home circumstances.

*Aftercare advice for patients who have received sedative or opioid (narcotic) drugs in the ED → p. 390.*