Introduction

Effective airway management is central to the care of critically ill and injured patients. Competency in assessment and maintenance of the airway using basic airway manoeuvres first, followed by advanced skills such as rapid sequence induction of anaesthesia and tracheal intubation, are core skills for doctors who treat seriously ill or potentially ill patients. In the UK, this typically involves the specialties of:

- anaesthesia;
- emergency medicine;
- intensive care medicine.

The location for emergency airway management is usually outside the relatively controlled environment of an anaesthetic room, most commonly in the resuscitation room of an emergency department, but also in a variety of other in-hospital and pre-hospital settings. Emergency airway management can be difficult and challenging; it requires individuals to work in relatively unfamiliar environments under conditions of stress and uncertainty, and where the principles of elective anaesthesia need modification. Information is often incomplete, normal physiology deranged, and opportunity for delay is infrequent. The problems intrinsic to these patients, such as an unstable cervical spine, poor cardiorespiratory reserve or profound metabolic dysfunction, must be anticipated and surmounted.

Emergency airway management is not simply an extension of elective anaesthesia, and specific training is essential to safely treat this challenging and heterogeneous group of patients. Individuals must practice within the limits of their own competence and work collaboratively with experienced clinicians from several disciplines to ensure patients receive optimal care (Figure 1.1).
Skills and judgement, as well as knowledge, are essential for treating patients who require emergency airway intervention. Careful judgement is required to determine whether an intervention is appropriate, how and when it should be undertaken, and what additional personnel and equipment are needed.

Central to emergency airway management is the recognition of:

1. The fundamental importance of good basic airways skills.
2. The need for close collaboration with those who are already competent to enable effective clinical training. It is essential to work alongside practitioners who have established expertise in emergency airway care in order to build upon and apply theoretical learning. A clinician working alone should not attempt emergency airway interventions that are outside the limits of their own competence.

The second edition of this book addresses the rapidly changing landscape of emergency airway management outside the operating room. Widespread access to technologies such as videolaryngoscopy, updated guidance based on emerging clinical evidence and increasing recognition of the role of human factors are just some of the elements that have shaped changes in clinical practice, and the second edition reflects these.

**Audit and skills maintenance**

Audit and peer review of clinical practice must be undertaken continuously to ensure standards are maintained. Medical simulators are becoming more sophisticated, and have a valuable role in the development, retention and assessment of clinical skills and human factors.

**Summary**

- This manual will not provide competence in emergency airway management, but offers a firm foundation upon which further training and assessment can be based.
- Effective emergency airway management requires commitment to a process of ongoing training, assessment, skill maintenance and audit that will last throughout the practitioner’s professional career.
Chapter 2

Delivery of oxygen
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Objectives
The objectives of this chapter are to:

- Understand the causes of hypoxaemia.
- Understand how much oxygen to give.
- Be familiar with devices that enable an increase in the inspired oxygen concentration.
- Understand the function and use of the self-inflating bag-mask.
- Understand the function and use of the Mapleson C breathing system.
- Understand how to monitor oxygenation.
- Understand the principle of pre-oxygenation.

Causes of hypoxaemia

The strict definition of hypoxaemia is a partial pressure of oxygen in the arterial blood (P_{O2}) below normal. For patients with no respiratory pathology, a value of < 8 kPa or 60 mmHg (equivalent to an arterial oxygen saturation of approximately 90%) is often used to define hypoxaemia requiring treatment; for patients with chronic obstructive pulmonary disease (COPD), a P_{O2} < 8 kPa (S_{O2} < 90%) may be ‘normal’. In nearly all patients hypoxaemia can usually be improved, at least initially, by increasing the inspired oxygen concentration.

Although the cause of hypoxaemia is usually multifactorial, there are several distinct mechanisms:

- alveolar hypoventilation;
- mismatch between ventilation and perfusion within the lungs;
- pulmonary diffusion abnormalities;
- reduced inspired oxygen concentration.
Alveolar hypoventilation

If insufficient oxygen enters the alveoli to replace that taken up by the blood, both the alveolar (P_{A}O_2) and arterial (P_{a}O_2) partial pressure of oxygen decrease. In most patients, increasing the inspired oxygen concentration will restore both. When an adult’s tidal volume decreases below approximately 150 mL there is no ventilation of the alveoli, only the ‘dead space’, which is the volume of the airways that plays no part in gas exchange. No oxygen reaches the alveoli, irrespective of the inspired concentration, and profound hypoxaemia will follow. At this point ventilatory support and supplementary oxygen will be required. Hypoventilation is always accompanied by hypercapnia, as there is an inverse relationship between arterial partial pressure of carbon dioxide (P_{a}CO_2) and alveolar ventilation.

Common causes of hypoventilation are as follows:

Airway obstruction:
- tongue;
- blood;
- vomit;
- bronchospasm;
- oedema (infection, burns, allergy).

Central respiratory depression:
- drugs;
- alcohol;
- central nervous system injury (cerebrovascular event, trauma, etc.);
- hypothermia.

Impaired mechanics of ventilation:
- pain;
- pneumothorax or flail chest;
- haemothorax;
- pulmonary oedema;
- diaphragmatic splinting;
- pre-existing lung disease.

Mismatch between ventilation and perfusion within the lungs

Normally, ventilation of the alveoli (\dot{V}) and perfusion with blood (\dot{Q}) are well matched (\dot{V}/\dot{Q} = 1), ensuring that haemoglobin in blood leaving the lungs is saturated with oxygen (Figure 2.1). If this process is disturbed (\dot{V}/\dot{Q} mismatch) regions develop where:

1. Ventilation is less than perfusion (\dot{V}/\dot{Q}<1), resulting in haemoglobin with reduced oxygen content, e.g. pneumothorax, pneumonia.
2. Perfusion is less than ventilation ($V/Q > 1$). This can be considered wasted ventilation as very little additional oxygen is taken up when haemoglobin is already almost fully saturated (98%), e.g. hypotension, pulmonary embolus. At its most extreme, some regions of the lung may be perfused but not ventilated ($V/Q = 0$); blood leaving these areas remains ‘venous’, and is often referred to as shunted blood. This is then mixed with oxygenated blood leaving ventilated regions of the lungs. The final oxygen content of blood leaving the lungs is dependent on the relative proportions of blood from these two regions:

- Blood perfusing ventilated alveoli leaves with an oxygen content of approximately 20 mL/100 mL blood (assuming a haemoglobin concentration of 150 gL$^{-1}$).
- Blood perfusing unventilated alveoli remains ‘venous’, leaving with an oxygen content of 15 mL/100 mL blood.
The effect of small regions of V/Q mismatch can be corrected by increasing the inspired oxygen concentration; however, once more than 30% of the pulmonary blood flow passes through regions where $V/Q < 1$, hypoxaemia is inevitable, even when breathing 100% oxygen. This is because the oxygen content of the pulmonary blood flowing through regions ventilated with 100% oxygen will increase by only 1 mL/100 mL blood (to produce 21 mL of oxygen per 100 mL blood), and this is insufficient to offset regions of low V/Q, where the oxygen content will be only 15 mL/100 mL blood.

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

Pulmonary diffusion defects

Any condition that causes thickening of the alveolar membrane (e.g. pulmonary oedema, fibrosing alveolitis) impairs transfer of oxygen into the blood. This is treated first by giving supplementary oxygen to increase the $P_AO_2$ (partial pressure of oxygen in the alveoli) and then treating the underlying problem.

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. At ambient pressure there are no circumstances where it is appropriate to administer less than 21% oxygen.

How much oxygen?

In the past, oxygen has usually been given on the basis that if some is good, more must be better. It is now recognized that in most circumstances, there is a range of optimal oxygenation and in some conditions (e.g. post-acute myocardial infarction, ischaemic stroke), excess oxygen may be detrimental. In 2008, the British Thoracic Society (BTS) published ‘Guidelines for Emergency Oxygen Use in Adult Patients’. These guidelines recommend that for most acutely ill patients, oxygen should be given to achieve a target saturation of 94–98%, or 88–92% for those at risk of hypercapnic respiratory failure.

Give all critically ill patients (Table 2.1) high-flow oxygen (15 L min$^{-1}$) until they are stable; then reduce the inspired oxygen concentration to achieve a target saturation of 94–98%. Patients with COPD and other risk factors for hypercapnic respiratory failure who are critically ill are treated similarly, but
aim for a saturation of 88–92% once they are stable. When pulse oximetry is unavailable, give high-flow oxygen until definitive treatment is available.

Early assessment of gas exchange based on the analysis of an arterial blood sample is essential in all critically ill patients to guide the need for subsequent oxygen therapy or ventilatory support.

Whenever oxygen is given to a patient, it must be prescribed and the target oxygen saturation to be maintained written on the patient’s drug chart.

### Devices used for delivery of oxygen

### Spontaneous ventilation

#### Variable-performance devices: masks or nasal cannulae

With these devices, the precise concentration of oxygen inspired by the patient is unknown, because it depends on the patient’s respiratory pattern and the oxygen flow (usually 2–15 L min$^{-1}$). When breathing through a mask 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 exhaled during the previous breath that has collected under the mask;
- air entrained during inspiration from the holes in the side of the mask and from leaks between the mask and face.

An example of this type of device is the Hudson mask (Figure 2.2). As a guide, the inspired oxygen concentration will be 25–60% with oxygen flows of 2–15 L min$^{-1}$. Patients unable to tolerate a facemask, but 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 (Figure 2.3). Lower flows of oxygen are used; 2–4 L min$^{-1}$ increases the inspired oxygen concentration to 25–40%.
In a critically ill patient breathing spontaneously who requires a higher concentration of oxygen, a Hudson mask with a reservoir (non-rebreathing bag) can be used (Figure 2.4). 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⁻¹), ensure minimal entrainment of air, raising the inspired concentration to approximately 80%, providing that the reservoir bag inflates and deflates with each breath. This requires a well-fitting, functioning mask and reservoir, and is often overlooked in clinical practice. An inspired oxygen concentration of 100% can be achieved only by using a close-fitting facemask with an anaesthetic breathing system that includes a reservoir, combined with an oxygen flow of 12–15 L min⁻¹ (see below). Once stable, these patients should have oxygen therapy adjusted as described above.

**Fixed-performance devices**

These are used to deliver a precise concentration of oxygen, unaffected by the patient’s ventilatory pattern. These devices work on the principle of high-airflow oxygen enrichment (HAFOE). Oxygen is delivered to a Venturi that entrains a much greater, but constant, flow of air (Figure 2.5). The total flow into the mask needs to be as high as 45 L min⁻¹. The high gas flow has two effects: it exceeds the patient’s peak inspiratory flow, reducing entrainment of air, and flushes expiratory gas, reducing rebreathing. These are the devices of choice for patients with known hypercapnic respiratory failure, or who are at risk of this condition.
These devices deliver a fixed concentration for a given flow, and there are several interchangeable Venturis to vary the oxygen concentration (Table 2.2). The above systems all deliver dry gas to the patient, which may cause crusting or thickening of secretions, difficulty with clearance, and patient discomfort. For prolonged use, a HAFOE system should be used with a humidifier.

**Assisted ventilation**

Patients whose ventilation is inadequate to maintain oxygenation despite an increase in the inspired oxygen concentration using one of the devices described above, or who are apnoeic, will require oxygenation using a mechanical device. The simplest and most widely used device is the bag-mask (Figure 2.6). An alternative is an anaesthetic breathing system (Figure 2.9).