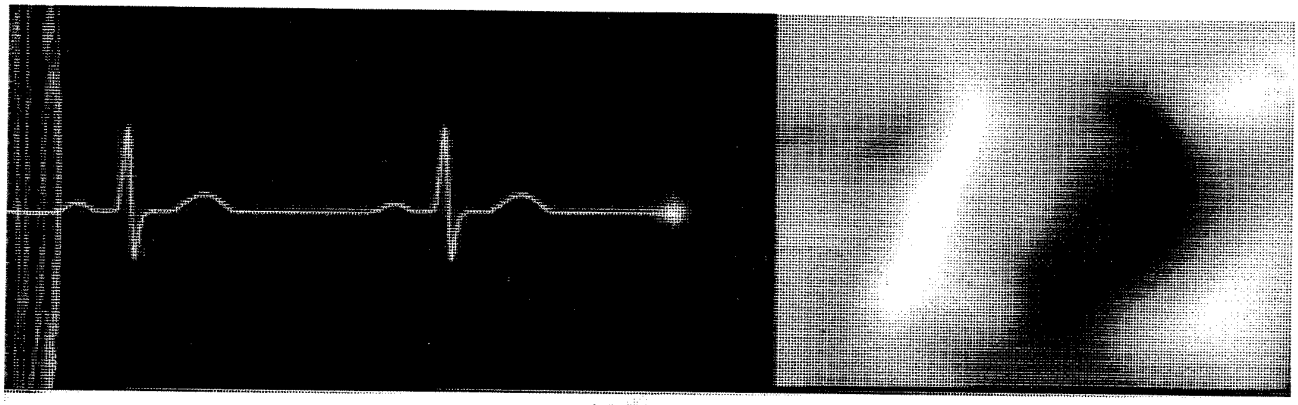

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# CRITICAL CARE

Carl Waldmann | Neil Soni | Andrew Rhodes

# Oxford Desk Reference
# Critical Care

**Carl Waldmann**
Consultant in Anaesthesia and Intensive Care
Royal Berkshire Hospital
Reading

**Neil Soni**
Honorary Clinical Senior Lecturer
Division of Surgery, Oncology,
Reproductive Biology and Anaesthetics
Imperial College
London

and

**Andrew Rhodes**
Consultant in Intensive Care
St George's Hospital
London

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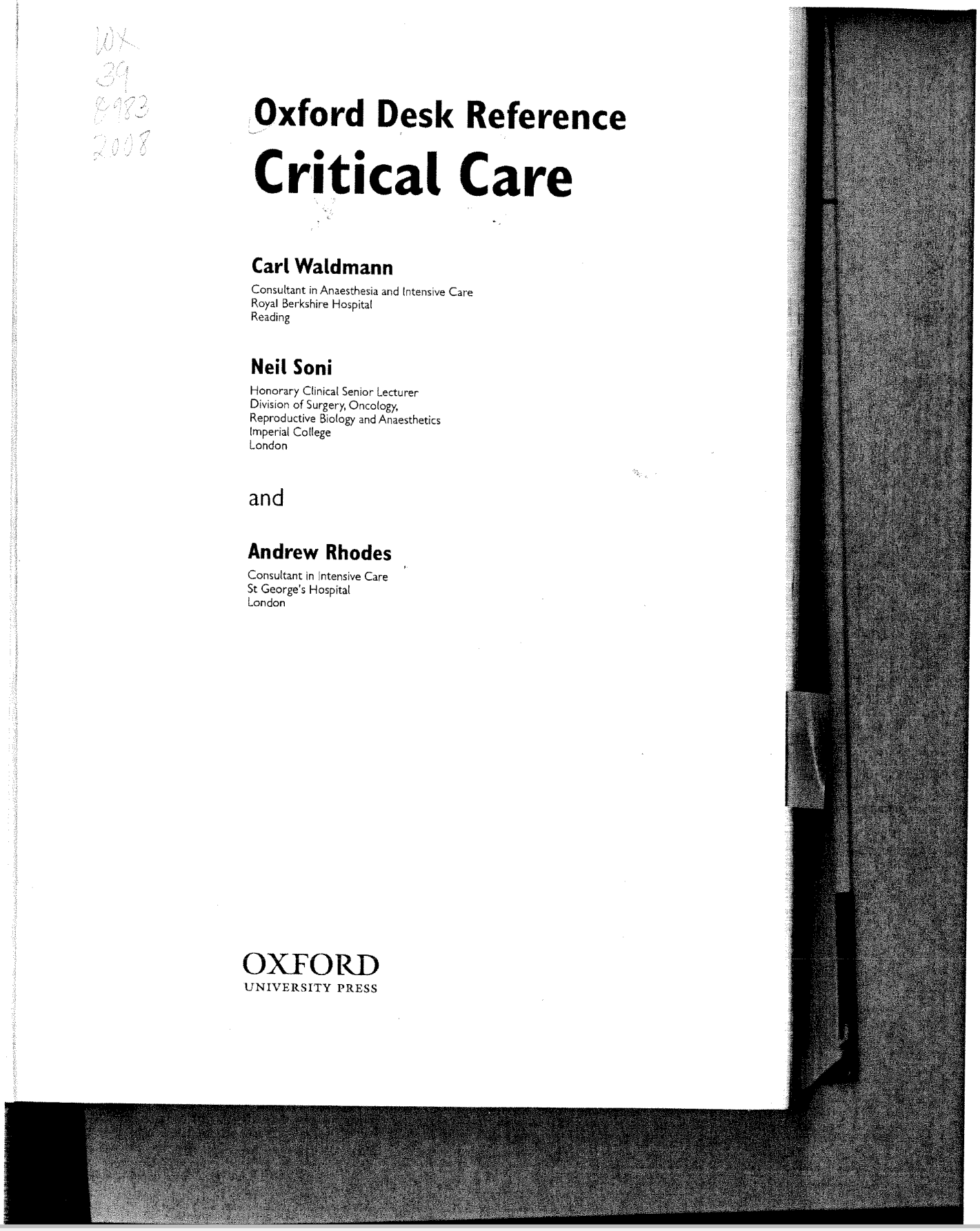

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# Oxygen therapy

Aerobic respiration is the most efficient method of energy production in the mammalian cell. It utilizes oxygen to produce adenosine triphosphate (ATP). The absence of oxygen or low oxygen levels result in more inefficient anaerobic respiration. Cellular energy levels become inadequate, and this can lead to loss of cellular homeostasis, which in turn can lead to cellular death and very possibly organism death. A substantial part of critical care is targeted at treating and/or preventing hypoxia.

## Pathophysiology of oxygen delivery

In critical illness the delivery ($DO_2$) and uptake ($VO_2$) of oxygen are often abnormal. Currently there are few therapeutic strategies for improvement of $VO_2$. Most methods of oxygen therapy target improvement in $DO_2$.

Delivery of oxygen from the environment is necessary to provide for cellular metabolism. In single-celled organisms (e.g. amoeba), simple diffusion suffices. However, in the multi-cellular, multi-organ human, more sophisticated mechanisms have evolved, each with their problems in illness.

Transport of oxygen to the cells follows six stages reliant only on the laws of physics.

1 Convection from the environment (ventilation).
2 Diffusion into the blood.
3 Reversible chemical bonding with haemoglobin.
4 Convective transport to the tissues (cardiac output).
5 Diffusion into the cells and organelles.
6 The redox state of the cell.

This chain of events is $DO_2$. Failure of $DO_2$ to match $VO_2$ leads to shock. This occurs when $DO_2$ declines to below approximately 300ml/min. Shock is defined loosely as failure of delivery of oxygen to match tissue demand. Commonly this refers to circulatory failure, but low $DO_2$ can result from several pathological mechanisms which can occur as a single problem or in combination (Table 1.1.1).

The impact of low $DO_2$ can be made worse by an increase in $VO_2$. Metabolic rate increases with exercise, inflammation, sepsis, pyrexia, thryotoxicosis, shivering, seizures, agitation, anxiety and pain. This mismatch leads to the need for early detection of shock and prompt treatment. This has been shown to be beneficial in surviving sepsis.

Clinical signs such as heart rate, blood pressure and urine output can be misleading, especially in the young. This therefore requires the concept of an effective cardiac output (ECO). This couples the clinical signs with evidence of normal $DO_2$ and $VO_2$ balance. The assessment includes peripheral temperature, oxygen haemoglobin saturation and arterial partial pressure, the presence of acidosis with a base excess greater than −2, lactataemia and abnormal $SvO_2$ or $ScvO_2$. These more technical measures of adequacy of oxygen delivery and uptake must always be taken in the clinical context. For example, in cyanide poisoning, both circulatory and ventilatory indices appear normal, yet the severe acidosis and lactataemia seen in this condition demonstrates tissue hypoxia. Manipulating $DO_2$ by increasing the environmental oxygen fraction ($FiO_2$) or cardiac output in this setting is unlikely to be helpful, and, even in sepsis and other more common types of shock, achieving supranormal values for $DO_2$ is not thought to be beneficial.

### *Strategies for increasing $DO_2$*

By assessing the type of hypoxia and its likely cause, the correct choice of $DO_2$-improving strategy can be chosen. In the critically ill, the commonly seen combination of mechanisms leading to hypoxia may require several techniques to be instigated in parallel. The methods for improving oxygen delivery to the tissues are based on reversing problems seen at each of the six stages of oxygen delivery. Improving the transport of oxygen once in the body will be covered later in this book. This chapter is concerned with improving oxygen delivery from the environment to the bloodstream. Oxygen delivery at this stage should be considered a support mechanism, and treatment of the underlying cause is most important to reverse hypoxia.

## Oxygen therapy apparatus

### *Principles*

In the hypoxic self-ventilating patient, delivery of oxygen to the alveoli is usually achieved by increasing the $FiO_2$. Commonly this involves the application of one of the many varieties of oxygen masks to the face, such that it covers the mouth and/or nose. Each type of delivery system consists of broadly the same six components:

1 **Oxygen supply**. Delivery of oxygen can be from pressurized cylinders, hospital supply from cylinder banks or

**Table 1.1.1** Types of hypoxia

| Type of hypoxia | Pathophysiology | Examples |
|---|---|---|
| Hypoxic hypoxia | Reduced supply of oxygen to the body leading to a low arterial oxygen tension | 1. Low environmental oxygen (e.g altitude)<br>2. Ventilatory failure (respiratory arrest, drug overdose, neuromuscular disease)<br>3. Pulmonary shunt<br>a. Anatomical—ventricular septal defect with right to left flow<br>b. Physiological—pneumonia, pneumothorax, pulmonary oedema, asthma |
| Anaemic hypoxia | Normal arterial oxygen tension, but circulating haemoglobin is reduced or functionally impaired | Massive haemorrhage, severe anaemia, carbon monoxide poisoning, methaemoglobinaemia |
| Stagnant hypoxia | Failure of oxygen transport due to inadequate circulation. | Left ventricular failure, pulmonary embolism, hypovolaemia, hypothermia |
| Histotoxic hypoxia | Impaired cellular metabolism of oxygen despite adequate delivery. | Cyanide poisoning, arsenic poisoning, alcohol intoxication |

a vacuum-insulated evaporator (VIE), or an oxygen concentrator.

2 **Oxygen flow control.** For example an OHE ball valve flow meter.

3 **Connecting tubing.** Both from supply to control, and from control to patient. The bore of the tubing is important as it has effects on the oxygen flow rate. In some systems it can also act as a reservoir.

4 **Reservoir.** All have reservoirs. In the simple oxygen mask it is the mask itself. Nasal cannulae use the nasopharynx as the reservoir. An oxygen tent is a large-volume reservoir. The reservoir serves to store oxygen, but must not allow significant storage of exhaled gases leading to rebreathing of carbon dioxide.

5 **Patient attachment.** This permits delivery of oxygen to the airway. This is achieved either by directly covering the upper airway, e.g. plastic mask/head box, or by increasing the oxygen concentration in the wider environment, e.g. oxygen tent.

6 **Expired gas facility.** Expired gas needs to dissipate to the environment. This can be achieved by having a small reservoir with holes, one-way valves as in the non-rebreather masks, or high oxygen flows as seen in some of the continuous positive airway pressure (CPAP) systems.

Additional features of oxygen breathing systems are the presence of humidification such as a water bath, to prevent drying of the mucosal membranes. Some devices have an oxygen monitor incorporated into the apparatus to permit more accurate defining of the $FiO_2$.

### *Factors that affect the performance of oxygen delivery systems*

Most of the simpler oxygen delivery devices, e.g. plastic masks, nasal cannulae, etc., deliver oxygen at relatively low oxygen flow rates. The patient inspiratory flow rate varies throughout inspiration (25–100+L.min$^{-1}$) and exceeds the oxygen flow rate. This drains the small reservoir and causes entrainment of environmental air. The effect is to dilute the oxygen concentration to the final $FiO_2$. The actual $FiO_2$ that reaches the alveolus is therefore unpredictable and is dependent on the interaction of patent factors and device factors (Table 1.1.2). In the hypoxic patient it is common to find significant increases in inspiratory flow rates as well as the loss of the respiratory pause. This causes significant entrainment of air, lowering the alveolar $FiO_2$. This is particularly true of the variable performance masks, but is also seen in Venturi-type masks, particularly when higher $FiO_2$ inserts are used. The presence of a valve-controlled reservoir bag on a non-rebreather mask should compensate for high inspiratory flows, hence the belief that such devices can deliver an $FiO_2$ of 1.0 which does not actually happen. This is not seen in models of human ventilation (Fig. 1.1.1)

**Table 1.1.2** Factors that influence the $FiO_2$ delivered to a patient by oxygen delivery devices[5]

| Patient factors | Device factors |
|---|---|
| Inspiratory flow rate | Oxygen flow rate |
| Presence of a respiratory pause | Volume of mask |
| Tidal volume | Air vent size |
| | Tightness of fit |

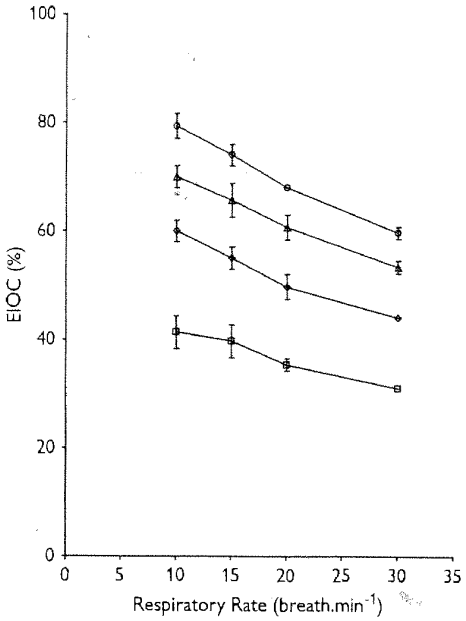


**Fig. 1.1.1** The performance of a Hudson non-rebreather mask on a model of human ventilation. Tidal volume of 500ml and four oxygen flow rates (2l/min (□), 6l/min (◇), 10l/min (△) and 15l/min (○)). As the respiratory rate increases, so the effective inspired oxygen concentration (EIOC) deteriorates.

### *Classification of oxygen delivery devices*

Methods of delivering oxygen to the conscious patient with no airway instrumentation can be broadly divided into the following categories.

- Variable performance systems
- Fixed performance systems
- High flow systems
- Others

Variable performance systems are so called because their $FiO_2$ can vary as described above. Fixed performance systems cannot. High flow systems use high oxygen flows to maintain a fixed performance. The common types and their properties are summarized in Table 1.1.3.

## Hazards of oxygen therapy

Oxygen is a drug and, like most drugs, its use is not without risk. It is also a gas and commonly delivered from compressed sources.

### *Supply*

Medical oxygen is supplied at 137bar from a cylinder, and 4bar from hospital pipelines. Direct administration at delivery pressures is highly dangerous and requires properly functioning pressure-limiting valves. Oxygen supports combustion. Patients must not smoke cigarettes when receiving oxygen therapy, and oxygen should be removed from the environment when sparking may occur, e.g. during defibrillation.

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