

OXFORD DESK REFERENCE CRITICAL CARE

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OXFORD UNIVERSITY DRESS



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UNIVERSITY PRESS

Great Clarendon Street, Oxford OX2 6DP

Oxford University Press is a department of the University of Oxford. It furthers the University's objective of excellence in research, scholarship, and education by publishing worldwide in

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Published in the United States by Oxford University Press Inc., New York

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First published 2008

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British Library Cataloguing in Publication Data Data available

Library of Congress Cataloguing in Publication Data Data available

Typeset by Cepha Imaging Private Ltd., Bangalore, India Printed in Great Britain on acid-free paper by CPI Antony Rowe, Chippenham, Wiltshire

ISBN 978-0-19-922958-1

10 9 8 7 6 5 4 3 2 1



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 produce agenosine tripposphate (ALF). The absence or 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 (DO₂) and uptake (VO₂) of oxygen are often abnormal. Currently there are few therapeutic strategies for improvement of VO₂. 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 Inis chain of events is DO2, Faiture of DO2 to Matchi VO2 leads to shock. This occurs when DO2 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 DO2 can result from several pathological mechanisms which can occur as a single problem or in combination (Table 1.11). 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 The impact or tow DO₂ can be made worse by an increase in VO₂. 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 DO₂ and VO₂ 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 SvO₂ or ScvO₂. 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 DO₂ by increasing the environmental oxygen fraction (FiO₂) 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 DO2

By assessing the type of hypoxia and its likely cause, the correct choice of DO₂-improving strategy can be chosen. correct cnoice or Lung-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

Princibles

In the hypoxic self-ventilating patient, delivery of oxygen to the alveoli is usually achieved by increasing the FiO₂. 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 Anaemic hypoxia	Reduced supply of oxygen to the body leading to a low arterial oxygen tension	1. Low environmental oxygen (e.g aititude) 2. Ventilatory failure (respiratory arrest, drug overdose, neuromuscular disease) 3. Pulmonary shunt a. Anatomical—ventricular septal defect with right to left flow b. Physiological—pneumonia, pneumothorax, pulmonary oedema, asthma
	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 FiO₂.

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 FiO2. The actual FiO2 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 FiO2. This is particularly true of the variable performance masks, but is also seen in Venturi-type masks, particularly when higher FiO2 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 FiO₂ delivered to a patient by oxygen delivery devices⁵

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

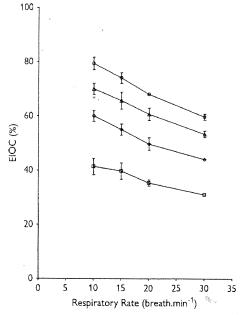


Fig. 1.1.1 The performance of a Hudson non-rebreather mask on a model of human ventilation. Tidal volume of 500ml and four oxygen flow rates ($2l/\min$ (\square), $6l/\min$ (\triangle), $10l/\min$ (\triangle) and $15l/\min$ (\bigcirc). As the respiratory rate increases, so the effective inspired oxygen 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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