

Decreased muscle strength
 Myopathy
 Neuropathy
 Spinal cord injury
Increased lung elastance
 Pulmonary oedema
 Atelectasis
 Pulmonary fibrosis
 Loss of lung tissue
Increased chest wall elastance
 Pleural effusion
 Haemothorax
 Pneumothorax
 Kyphoscoliosis
 Obesity
 Ascites
Reduced FRC
 Atelectasis
 Premature airway closure (e.g. COPD)

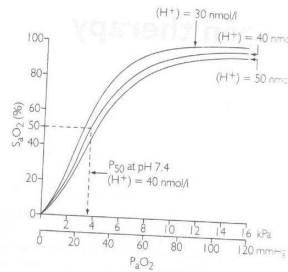


Fig. 22.1 Hb oxygen dissociation curve. Normal curve at 40 nmol/l (H⁺) and shifts to left and right. P₅₀ = tension at 50% saturation.

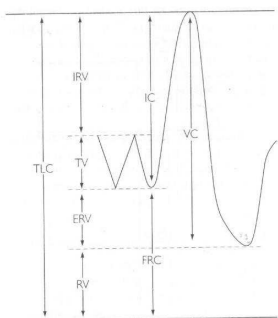


Fig. 28.1 Lung volumes and capacities. TLC, total lung capacity; IRV, inspiratory reserve volume; TV, tidal volume; ERV, expiratory reserve volume; RV, residual volume; IC, inspiratory capacity; FRC, functional residual capacity; VC, vital capacity.

Oxygen flow rate (l/min)	Approximate FiO ₂
4	0.35
6	0.50
8	0.55
10	0.60
12	0.65
15	0.70

Apparatus/device	Oxygen flow (l/min)	Concentrations (%)
Nasal catheters	2-6	25-40
Semi-rigid mask (e.g. MC, Edinburgh, Hudson, Harris)	4-15	35-70
Venturi-type mask		
individual concentration masks	24, 28, 35	
interchangeable entrainment discs	6-12	40, 50, 60
Soft plastic masks (e.g. Pneumask, Polymask, Oxyaire)	4-15	40-80
ventilators	varying	21-100
Anaesthesia circuits	varying	21-100
CPAP circuits	varying	21-100
Plastic head hood	4-8	30-50
Oxygen tent/cot	7-10	60-80
incubator	3-8	up to 40

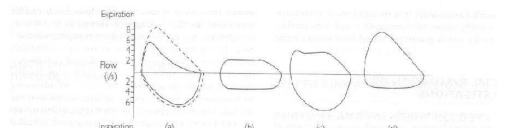


Fig. 23.3 Flow-volume loops. Patterns resulting from different pathological lesions: (a) lower airway obstruction (e.g. chronic obstructive pulmonary disease or asthma). (b) fixed, non-variable upper airway obstruction (e.g. fibrous ring in trachea). (c) variable upper airway obstruction, intrathoracic (e.g. tumour in the lower trachea); (d) variable upper airway obstruction, extrathoracic (e.g. vocal cord tumour or paralysis).

$$PAO_2 = FIO_2 \times (760 \text{ mmHg} - 47) - PaCO_2 / 0.8$$

alveolar gas equation

pathological flow-volume loops

O₂ flow through masks

O₂ from various devices

lung volumes & capacities

Hb dissociation curve

factors that decrease vital capacity

factors shifting O₂ dissociation curve left

- Factors decreasing P₅₀ (curve shifts to left)**
- Hypothermia
 - Increased pH (alkalaemia)
 - Decreased PCO₂
 - Decreased 2, 3 DPG
 - Fetal haemoglobin
 - Carboxyhaemoglobin
 - Methaemoglobin

factors shifting O₂ dissociation curve right

- Factors increasing P₅₀ (curve shifts to right)**
- Hyperthermia
 - Decreased pH (acidaemia)
 - Increased PCO₂ (Bohr effect)
 - Increased 2, 3 DPG

oxygen cascade

	mmHg
Inspired air	150
Alveolar	103
Arterial	100
Capillary	51
Tissue	20
Mitochondrial	1-20

pulmonary oxygen transfer

- the best measure of pulmonary gas transfer is by the multiple inert gas elimination technique, although this is not normally available at the bedside
 - the method has identified V/Q mismatch and intrapulmonary shunt as the two main causes of reduced pulmonary oxygen transfer in critical illness
 - intrapulmonary shunt predominates in the acute respiratory distress syndrome, in lobar pneumonia & after cardiopulmonary bypass, whereas V/Q mismatch is more prominent in chronic lung disease

- (1) A-a gradient
 - part of APACHE-II score & is calculated using the alveolar gas equation
 - hypoxaemia with raised A-a gradient can be due to:
 (i) alveolar hypoventilation (elevated PACO₂)
 (ii) low FIO₂ or altitude
 - hypoxaemia with a raised A-a gradient can be due to:
 (i) diffusion defect (rare)
 (ii) V/Q mismatch
 (iii) shunt (intracardiac or intrapulmonary)
 (iv) increased arteriovenous oxygen extraction
 - drawbacks of A-a gradient include:
 (i) it is both FIO₂ & age dependent
 (ii) for lungs with an unchanging intrapulmonary shunt the A-a gradient alters with FIO₂
 (iii) in constant V/Q mismatch the relationship between the A-a gradient and FIO₂ is even more complex

- (2) PaO₂/FIO₂ ratio
 - PF ratio forms part of the definition of acute lung injury and ARDS & it is also an input in SAPS 2 & lung injury scoring systems
 - at sea level normal value is >500mmHg; unlike the A-a gradient it cannot distinguish between alveolar hypoventilation and other causes of hypoxaemia
 - drawbacks are:
 (i) in ARDs PF ratio is unreliable unless estimated when FIO₂>0.5, PaO₂>100mmHg, and CaO₂-CvO₂ is constant
 (ii) alters with altitude
 (iii) alters when FIO₂ is varied in lungs with V/Q mismatch
 (iv) alters when there are fluctuations in CaO₂-CvO₂ (eg in sepsis)

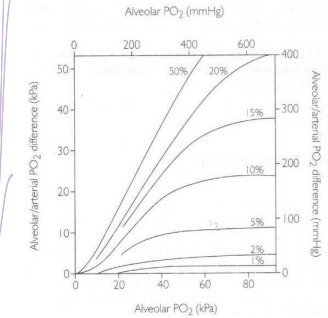


Fig. 11.1 Effect of varying FIO₂ (via alveolar PO₂) on A-a gradient with different degrees of intrapulmonary shunt.

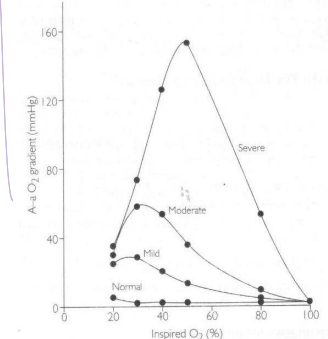


Fig. 11.2 Effect of varying FIO₂ on A-a gradient with mild, moderate and severe V/Q mismatch. No allowance has been made for absorption atelectasis or alterations in hypoxic pulmonary vasoconstriction. (Figures 11.2 and 11.3 reproduced)

pulmonary oxygen transfer - content based indices

- (1) Venous admixture (Qs/Qt)
 - a calculation derived from a three compartment model of the lung, the three compartments consisting of the ideal compartment (V/Q = 1), the perfused but unventilated alveoli (V/Q = 0) & the alveolar dead space (V/Q is infinite)
 - venous admixture is the proportional of mixed venous blood flowing through the theoretical shunt compartment where V/Q = 0
 - calculation is: Qs/Qt = [(CcO₂ - CaO₂) / (CcO₂ - CvO₂)]
 where CcO₂, CaO₂ & CvO₂ represent pulmonary end-capillary, arterial & mixed venous oxygen respectively (CcO₂ is estimated from the PAO₂ by the alveolar gas equation & saturation is calculated from HbO₂ dissociation curve
 - advantages of this technique are:
 (i) it is unaffected by barometric pressure
 (ii) it is unaffected by alveolar hypoventilation
 (iii) provided that intrapulmonary shunt is the dominant pathology it is stable across the entire range of FIO₂ despite variations in CaO₂-CvO₂
 (iv) when determined at FIO₂ = 1, venous admixture is a measure of true shunt
 - disadvantages of this technique are:
 (i) need for a PA catheter
 (ii) highly variable with FIO₂ in V/Q mismatch
 (2) VQI: the dual oximetry method:
 - by assuming pulmonary capillary saturation is 1, the equation for venous admixture can be simplified. Qs/Qt calculated in this way is termed VQI
 - the advantage of this technique is that VQI can be continuously monitored by pulse oximetry with mixed venous oximetry
 (3) Estimated shunt fraction:
 - estimated shunt fraction relies on CaO₂-CvO₂ being assumed to be constant (which it is not in critical illness)