By Adam Hollingworth

6.V:Q Relationships

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Intro

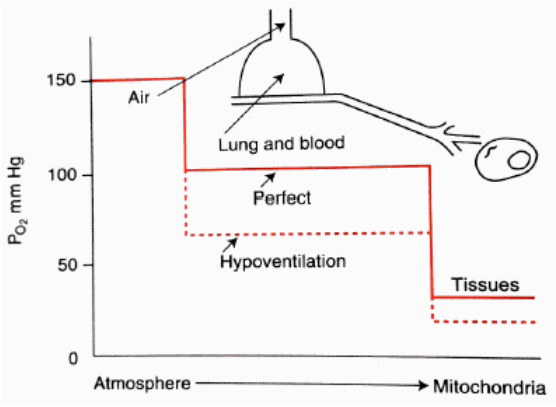
- V (ventilation) – effects alveolar gas:
  - Delivery O2
  - Removal CO2
- Q (perfusion) – delivers venous blood with high PCO2 & low PO2 ready for exchange
- Mismatching of V/Q ⇒ impaired transfer of O2 & CO2
- V/Q ratio determines pp's of gases in alveolar gas & arterial blood

Terminology

- Mixed venous blood
  - = represents mixture of all the systemic venous blood draining from all the tissue capillary beds (incl the myocardium)
  - made from 3 major sources:
    - SVC
    - IVC
    - Coronary sinus – PO2 only 20mmHg but only contributes 5%
  - True mixed venous sample taken from 2-2.5cm into pulmon artery
- Venous admixture:
  - =amount of mixed venous blood which would have to be added to pulmonary end-capillary blood to produce the observed drop in arterial PO2 from the PO2 of end capillary blood ie virtual shunt
  - 2 sources in normal people contribute to venous admixture:
    - blood which is true shunt:
      - bronchial venous blood – via deep bronchial veins into pulmon veins
      - thebesian circulation into L heart = small pt of coronary drainage
    - blood from lung alveoli V/Q <1
      - not true shunt as has passed thru areas of lung that do receive some ventilation
      - blood is not fully oxygenated ∴ wasted perfusion
  - may be calculated from shunt equation
- true shunt = blood entering arterial system without passing through any ventilated part of lung

Hypoxaemia

- 4 causes:
  - (low FiO2 – in effect causes a diffusion problem)
  - hypoventilation
  - diffusion limitation
  - shunt
  - ventilation-perfusion mismatch (alveolar dead space)
    ↳ = most common cause hypoxaemia



Oxygen Transport from Air to Tissues

- by time o2 has reached alveoli Po2 ↓ed by 1/3 (ie to 100mHg)
  ↳because P_AO2 determined by balance of
    - removal of O2 by cap blood
      - governed by o2 consumption by tissues
    - replenishment of o2 by alveolar ventilation
  - in practise removal of o2 constant @ rest ∴ P_Ao2 governed by alveolar vent
    ↳same applies to P_ACO2 ~ 40mmHg
- @tissue capillaries O2 diffuses to mitochondria:
  - tissue PO2 much lower – varies considerably in diff tissues
  - lung essential link in chain of distribution of O2
    ↳ie any ↓in P_aO2 must cause ↓in tissue PO2
    ↳∴ opposite true for tissue PCO2 ie fail of pulmon gas exchange ⇒ ↑tissue PCO2

Hypoventilation

- if alveolar ventilation is low ⇒ ↓P_AO2 & ↑P_ACO2
  ↳= hypoventilation
- ↓in PAO2 can be reversed easily by adding additional O2
- causes:
  - depression of central resp drive eg morphine, barbituates
  - damage to chest wall/mm's of resp
  - high resistance to breathing – eg deep underwater
- alveolar ventilation and P_aCO2 relationship:

$$P_aCO2 = \frac{CO2\ production}{Alveolar\ Ventilation} \times constant$$

↳∴if 1/2 alveolar vent then PCO2 doubled in a steady state

- relationship between fall in PO2 & rise in PCO2 which occurs in hypovent can be calculated
  ↳from **alveolar gas equation**:

$$P_AO2 = P_IO2 - \frac{PACO2}{R} + F$$

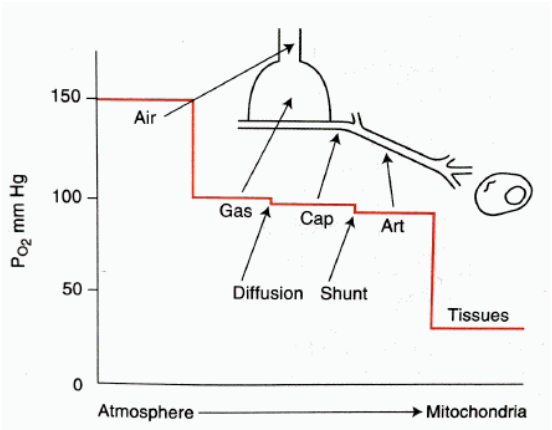
F= small correction factor (~2mmHg)
R = respiratory quotient (~0.8)
↳determined by CO2 prodction/O2 consumption
↳ie metabolism of tissues in steady state
P_IO2 = composition of inspired gas

↳∴ ↓in P_AO2 is slightly greater than ↑P_ACO2 during hypoventilation

- voluntary hyperventilation ⇒ ↑alveolar ventilation
  - take several mins before P_AO2 & P_ACO2 reach new steady states
    ↳due to diff stores of O2 & CO2 in body:
    - large stores of CO2 in form of bicarbonate in blood ∴ longer to steady state

Diffusion Limitation

- lung is not perfect ∴ P_aO2 not same as that in alveolar gas. Due to:
  - usually immeasurably small unless:
    - heavy exercise
    - thick blood-gas barrier
    - low O2 mixture inhaled



Shunt

- = blood which enters arterial system without going through ventilated area of lung
- venous admixture includes blood from alveoli with V/Q ratios <1 BUT is not 'true shunt'
  ↳ has passed through lung units with at least some vent
  ↳ ∴ blood to V/Q = 0 could be called true shunt
- sources of true shunt:
  - bronchial artery blood which outflows deoxygenated blood into pulmon veins
  - coronary venous blood which drains directly into LV through thebesian veins
  - abnormal pulmonary a-v fistula
  - cardiac septal defects ⇒ R to L cardiac shunt
- shunt equation – allows amount of venous admixture or shunt to be calculated
- value from equation = virtual shunt or 'as if' shunt'
- virtual shunt = amount of shunt that would be present if the shunt was entirely of mixed venous blood
  ↳ this amount of shunt would fully account for drop in PaO2

- hypoxaemia due to shunt would not respond to ↑ in inspired O2:
  - because shunted blood never exposed to high O2 level
  - Look at OHDC curve below:
    - PaO2 is actually markedly dropped by shunt being added to end capillary blood
    - CaO2 drops a little but PaO2 drops markedly
    - As desaturated shunted blood soaking up PaO2

  ↳ ∴ diagnostic shunt test: FiO2 100% ⇒ PaO2 does not rise to expected level

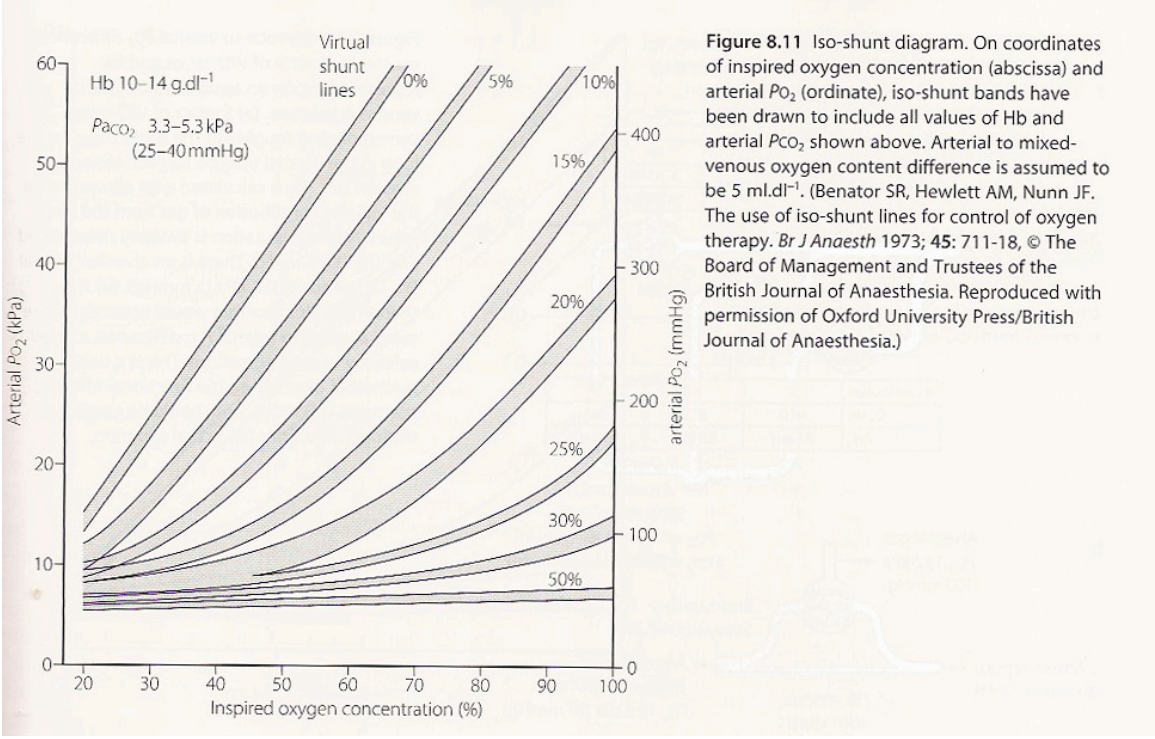
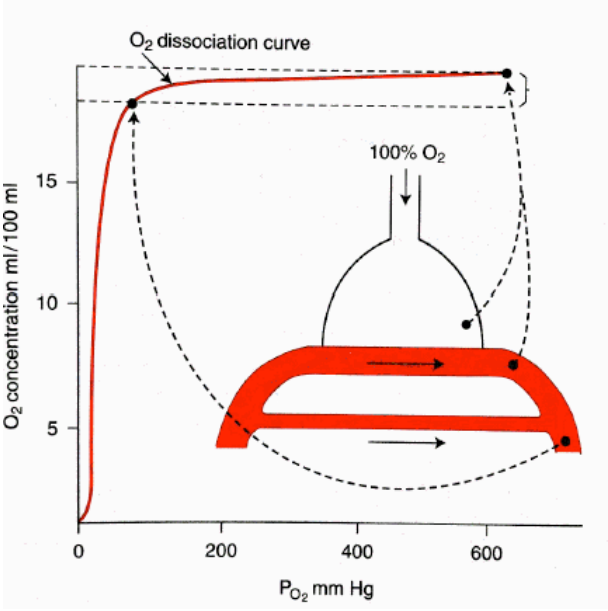


Figure 8.11 Iso-shunt diagram. On coordinates of inspired oxygen concentration (abscissa) and arterial Po_2 (ordinate), iso-shunt bands have been drawn to include all values of Hb and arterial Pco_2 shown above. Arterial to mixed-venous oxygen content difference is assumed to be 5 ml.dl^{-1}. (Benator SR, Hewlett AM, Nunn JF. The use of iso-shunt lines for control of oxygen therapy. *Br J Anaesth* 1973; **45**: 711-18, © The Board of Management and Trustees of the British Journal of Anaesthesia. Reproduced with permission of Oxford University Press/British Journal of Anaesthesia.)



Shunt Equation

Total O2 delivery = O2 delivery from ventilated lung + O2 delivery from shunt

(shunted blood assumed to be mixed venous blood)

Remember:

Delivery (DO2) = CaO2 x Q, here Q = CO

$Qt \times CaO_2 = [(Qt-Qs) \times Cc'O_2] + [Qs \times CvO_2]$

Qs = shunt flow

Qt = CO

Cc'O2 = O2 content of end capillary blood,

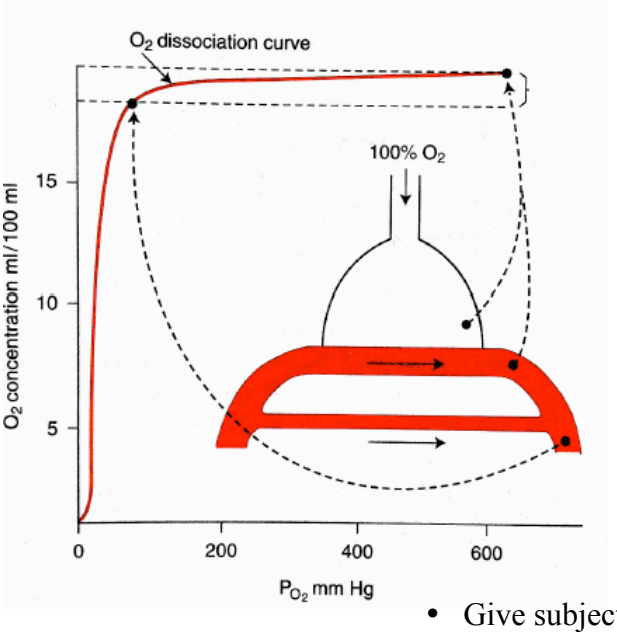
CaO2 = arterial blood O2 content

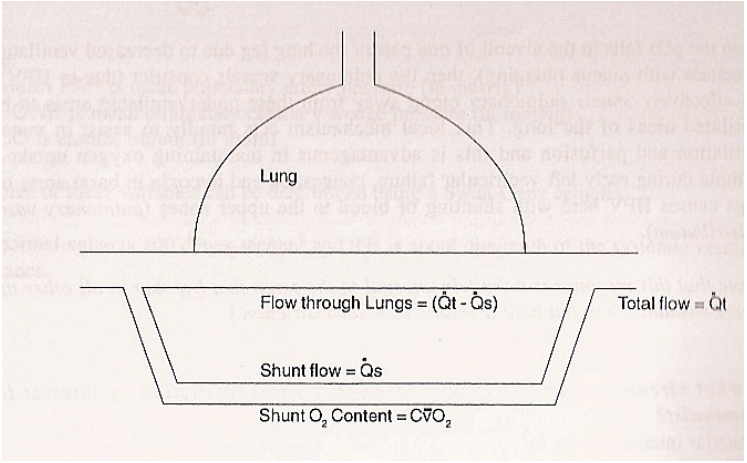
C vO2 = mixed venous blood content.

Qt-Qs = flow through the lungs

Can rearrange to give shunt equation:

$$\frac{Qs}{Qt} = \frac{(C_cO2 - C_aO2)}{(C_cO2 - C_vO2)}$$





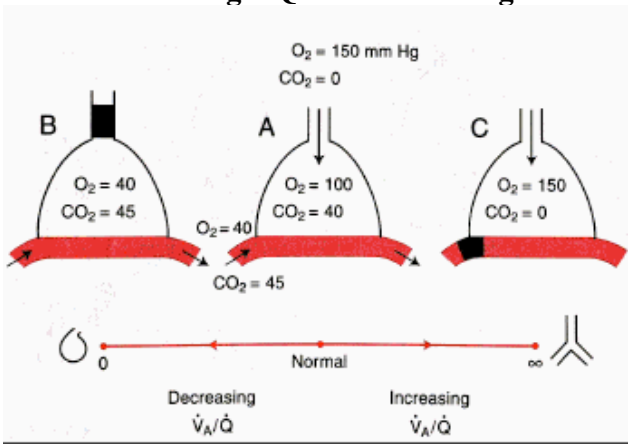
- Give subject 100% O2 measures shunt well
- When PO2 is high, small depression of arterial O2 concentration causes large fall in PO2
  ↳due to flat slope of O2 curve here

- shunt does not result in ↑PaCO2 (even though blood high in CO2)
  ↳cos chemoreceptors sense ↑PaCO2 ⇒ ↑ventilation
  - in fact in some pts with shunt PaCO2 is low because hypoxaemia ↑resp drive

Ventilation Perfusion Ratio

- ideal situation is V/Q = 1
- does not occur in normal healthy humans due to:
  - flow variation (lungs as a whole):
    - alveolar ventilation ~4l/min
    - blood flow ~5l/min
    - ↳ ∴ V/Q for whole lung = 0.8
  - effect of gravity – see later 'regional gas exchange in lung)
- if vent & blood flow are mismatched in various regions of lung; ↓transfer of both O2 and CO2 result
- concentration of O2 in any lung unit is determined by ration of ventilation to blood flow
- ventilation / blood flow (V/Q ratio)

Effect of Altering VQ Ration of Lung Unit



Picture A:

- normal VQ ration ~ 1
- P_AO2 (100) determined by balance of addition of o2 by ventilation & removal by blood flow

↳P_ACO2 (40) set by similar

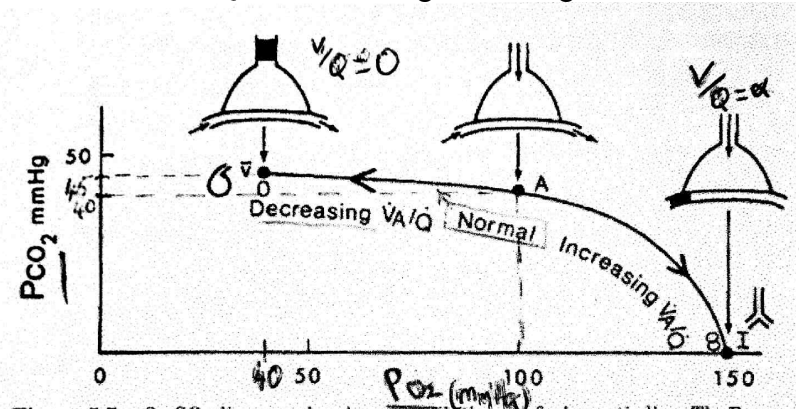
Picture B:

- VQ ration = zero
- Loss of ventilation by obstruction with no change in flow ie V = 0: Q = normal
- With complete vent obstruction P_A values will equalise with blood values
- = true shunt (or wasted perfusion)

Picture C:

- VQ ratio = infinity
- Obstruction of blood flow with normal ventilation; ie ↑norm V: Q = 0
- With total flow obstruction P_A values equalise with inspired air
- = alveolar dead space (or wasted ventilation)
- can be assessed by measuring physiological dead space (Bohr Equation)

↳ ∴ effects of V/Q mismatch on gas exchange are those of shunt & dead space



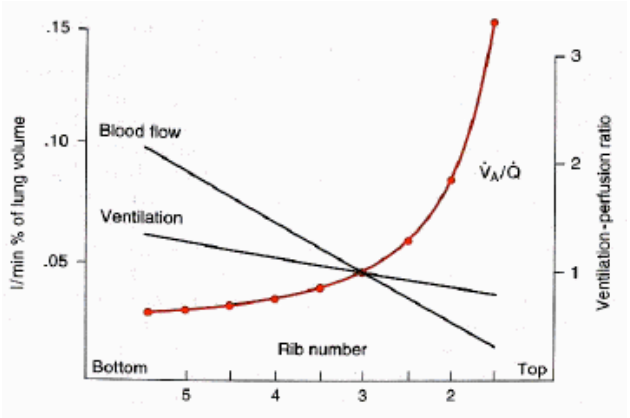
- VQ = zero (no ventilation):
  - ↓O2
  - ↑CO2
- VQ = high (no flow)
  - ↑O2
  - ↓Co2

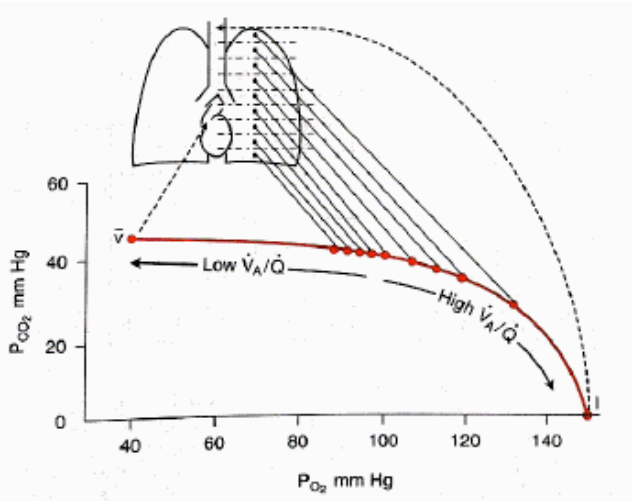
O2-CO2 diagram – PO2 & PCO2 of lung unit move along line from $\bar{v}$ to inspired gas point 1 as ↑ing V/Q

Regional Gas Exchange in Lung

- in upright lung:
  - ventilation ↓s slowly from bottom to top of lung (vent > flow at top)
  - perfusion ↓s much more rapidly from bottom to top (perfusion >vent at bottom)

    ↳∴ top lung – high VQ ratio
    bottom lung – lower VQ ratio





∴ from pictures –

- basal V/Q = 0.63
- apex V/Q = 3.3
- gradients:

- ventilation: V_{apex}:V_{Base} = 1:4
- Q_{Apex}:Q_{Base} = 1:18

- apex lung = v high VQ & high Po2
- base lung = low VQ & high CO2
- gravity explains most of scatter of V/Q but study in zero gravity also shown sig V/Q mismatch
  ↳see review below ↳ cause of this unknown
- P_aO2 change much greater than P_aCO2 change from apex to base lung:
  - Diff in CO2 less because closely related to ventilation
  - Bigger diff in O2 as more related to blood flow (bigger diff) & very poor flow to apex @rest
- Respiratory exchange ratio = CO2 output/O2 uptake
  ↳higher at apex than base as poor O2 uptake as no blood flow
  ↳during exercise much improved flow to apex lung ∴ ratio decreases
- Note large change in pH apex to base – high to low
  ↳due to ↑PCO2 in base

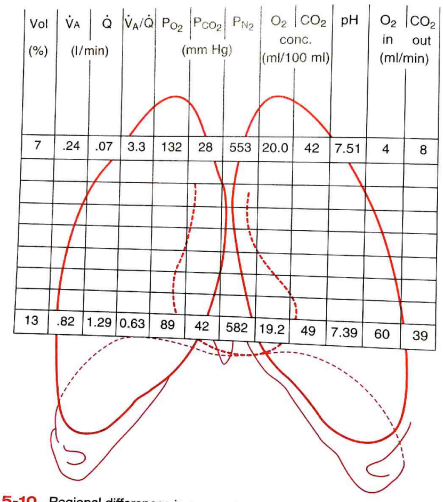


Figure 5-10. Regional differences in gas exchange down the normal lung. Only the apical and basal values are shown for clarity.

Regional Differences in Ventilation (Review)

- Resting volume of dependant airways is smaller than non-dep areas
  ↳ this due to relative ↑+ve intrapleural pressure (IPP)
- During inspiration: change in volume/unit resting volume ∴ ↑ed in dep lung
- @ low volumes surfactant plays a role in compliance curve:
  - low volume live on steep part of compliance curve ⇒ easier to inflate basal areas
- 4:1 ratio seen as move up lung

Regional Differences in Perfusion (review)

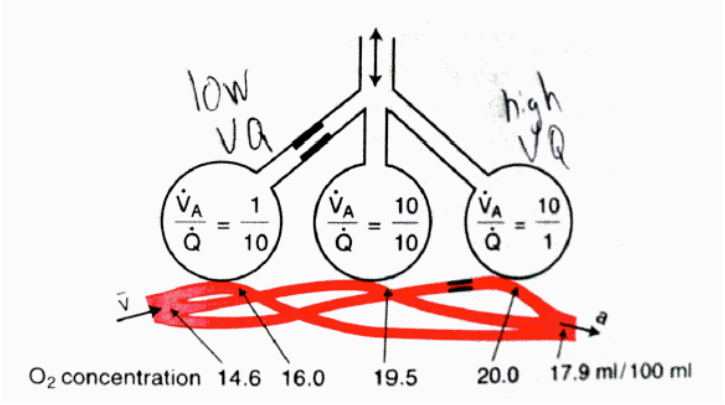
- linear ↓blood moving up lung
- gravity causes hydrostatic pressure differences in vessels
- West zones & starling resistor in action (see 4. Blood flow doc)

Effect Of VQ Mismatch on Overall Gas Exchange

- If VQ mismatch in effect:
  - Same amounts of gas must be transferred – as set by metabolic demands of tissues
  - ∴ cannot maintain as high PaO2 or as low PaCO2 compared to perfect lung
- Reasons:
  - Amount of blood drained from diff V/Q ratio's differ:
    - in upright lung:
      - apex units (non dep units):
        - have high P_AO2 & Low P_ACO2s
        - But they drain less blood ∴ contribute less to total amount of blood leaving lung
      - Basal units (dep units):
        - Have lower P_AO2s & higher P_ACO2
        - More blood leaves these areas by definition as have a lower V/Q ratio

      ↳ ∴ ↓ed arterial PO2 and ↑ed arterial PCO2 seen
  - Shape of OHDC:
    - Units with high VQ ratio add relatively little extra O2 to blood
      ↳ flat upper part of curve
      ↳ eg high V/Q CaO2 ~ 20ml O2/100ml blood
      perfect V/Q of 1: CaO2 ~ 19.5ml O2/100ml blood
    - Units with low VQ ⇒ much lower PO2 ⇒ corresponding low CaO2
      ↳due to steep part of OHDC
      - Eg CaO2 = 16ml O2/100ml blood
        ↳ close to mixed venous blood
    - Summary:
      - high VQ units add relatively little O2 to blood as opposed to decrement caused by units with low V/Q

    ↳ does not apply in converse to PCO2 as CO2 dissociation curve = linear in working range



- NET result of these mechanisms (in normal healthy) is a depression of P_aO2 below that of mixed P_AO2

↳ = alveolar-arterial o2 difference (aka D(A-a)O2 (D=difference in partial pressure))

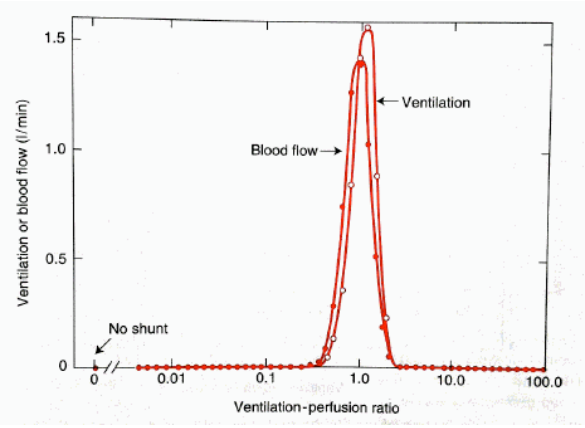
- A-a difference:
  - In normal upright lung trivial
    - ~4mmHg, for Spo2 98% & PAO@ of 104mmHg
  - In disease ~ extreme lowering P_aO2

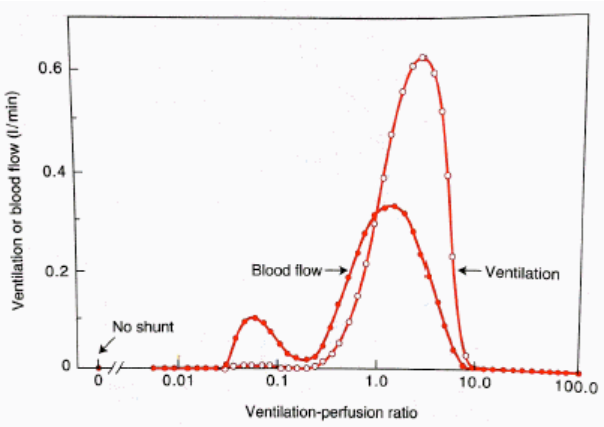
Causes of Increased D(A-a)O2

- Causes include:
  - V/Q Mismatch
  - Shunting
  - diffusion problems - can also contribute but in practise neglibly small & can be ignored
  - 100% Fio2 – see O2 cascade

[arterial end expiratory PCO2 difference = index of alveolar dead space

Distributions of VQ Ratios In Normal/Diseased Lung





- Left picture =
  - normal lung
  - all vent & blood flow goes to compartments close to VQ 1
    ↳esp little/no shunting to unmatched compartments
- Right picture =
  - Lung with COPD
  - ↑blood flow to compartments low VQ ⇒ deoxygenated blood from that unit ⇒ ↓PaO2
  - ↑vent to compartments with high VQ ⇒ these units poor blood flow ∴ poor elimination CO2
- units with
  - high V/Qs: can be assessed by measuring physiological dead space with Bohr Equation
  - low V/Q's: measuring venous admixture with the shunt equation

VQ Mismatch Causing CO2 Retention

- In VQ mismatch in a lung all other things equal would expect equal ↓P_aO2 & ↑P_aCO2
- BUT in pts with VQ mismatch often see normal P_aCO2
  - due to chemoreceptors sensing ↑P_aCO2 ⇒ ↑vent drive ⇒ ↓P_aCO2
  - ↑in ventilation = wasted ventilation (for high VQ units) (but beneficial for low V/Q units)
    ↳is necessary as lung unit with high VQ ratio are inefficient at elim CO2
    ↳= alveolar (physiological) dead space
- ↑ventilation works for CO2 elimination but not for ↑P_aO2
  ↳due to O2 dissociation curve:
    - CO2 curve linear in physiological range ∴ potentially low PCO2 values from high V/Q units is equally offset by high PCO2 from units with low V/Q ratio
    - O2 curve plateau at top ∴ only unit with low VQ ratio will benefit from ↑ed ventilation
      ↳ practically we overcome this by ↑ing FiO2

Measurement of VQ Mismatch

- can be measured using radio-isotope scanning of ventilation and perfusion separately (eg xenon & technecium)
- In practise use alveolar-arterial PO2 difference
- Need to calculate predicted P_AO2 & use alveolar gas equation:

$$P_AO2 = P_I02 - \left(\frac{P_ACO2}{R} \right) + F$$

- Measured arterial PCO2 used for P_ACO2
- P_IO2 = inspired alveolar PO2

- Then A-a difference = P_Ao2 (calculated) – P_aO2 (measured)
- Should be <10

- Eg pt breathing air at sea level has
  - Inspired Po2 149mmHg
  - a measured PaO2 of 50mHg
  - measured PaCO2 of 60mmHg
  - R = 0.8
  - F is ignored as so small

$$149 - \frac{60}{0.8} = 74 \text{ mmHg predicted PaO2}$$

$\therefore$ 74 – 50 = 24mmHg A-a difference
↳high $\therefore$ VQ difference

Summary V/Q Mismatch

- VQ mismatch can
  - markedly ↓PaO2 ⇒ ↑↑ed D(A-a)O2
  - less effect on:
    - CaO2, PaCO2, CaCO2
- CaO2 is mostly preserved because amount of blood added to end-capillary blood due to V/Q mismatch (or shunt) is small compared to overall blood flow
- Marked drop in PaO2 = PO2 of end capillary blood lies at flat upper part of OHDC & addition of even small amounts of blood with a low CaO2 (venous admixture) ⇒ ↓↓PaO2 markedly
- CaCO2 & PaCO2 are less effected by V/Q mismatch (& shunt) due to steep linear CO-Hb curve & ↑ed ventilatory response to any ↑in PaCO2

Effects of FiO2 100%

- Calculate PAO2 with FiO2 of 100%:

  PAO2 = (760-47) – (40/0.8) = 663mmHg

- PaO2 will be lower than PAO2 due to venous admixture
  ↳ it will still be over 600mmHg
- D(A-a)O2 will be larger than when on RA:
  - Due to depressing effect on PO2 on the flat part of OHDC by blood with low O2 content (ie low Spo2)
- CaO2 is only slightly increased with FiO2 100%:
  - PO2 of blood adds relatively little to content equation
  - Eg depending on Hb level 20 ⇒ 21.5mlO2/100ml blood
- PVO2 is only slightly elevated to ~48-50mmHg
- See O2 cascade examples next page:

By Adam Hollingworth

Different O2 Cascades –

Partial Pressure cascade:

FiO2 21%

Dry room air = **159** mmHg

↓ ← humidification of dry gas → ↓

Saturated room air = **149**

↓ ← gas exchange in alveoli (alv gas equation)→ ↓

Alveolar gas = **100**

↓← Incomplete **diffusion** (·immeasurably small)→↓

End-capillary blood ~ **100**

↓ ← venous admixture (shunt, V/Q mismatch) → ↓

Arterial blood = **97**

↓ ← diffusion of O2 to cells → ↓

End-tissue capillary blood = **40**

↓ ← consumption in cells(mainly mitochondria)→ ↓

Mitochondria = **4 – 22**

FiO2 100%

Dry room Air = **760**

↓ ← humidification of dry gas → ↓

Saturated room air = **711**

↓ ← gas exchange in alveoli (alv gas equation)→ ↓

Alveolar Gas **661**

↓← Incomplete **diffusion** (·immeasurably small)→↓

End capillary blood ~ 661

↓ ← venous admixture (shunt, V/Q mismatch) → ↓

Arterial blood ~ **600**

↓ ← diffusion of O2 to cells → ↓

End-tissue capillary blood ~48-50

↓ ← consumption in cells(mainly mitochondria)→ ↓

Mitochondria **~4-22**

O2 content (concentration) cascade for Hb 14g/dl:

FiO2 21%	**FiO2 100%**
Dry room air = **159** mmHg	Dry room Air = **760**
↓ ← humidification of dry gas → ↓	
Saturated room air = **149**	Saturated room air = **711**
↓ ← gas exchange in alveoli (alv gas equation)→ ↓	
Alveolar gas = **100**	Alveolar Gas **661**
↓← Incomplete **diffusion** (·immeasurably small)→↓	
End-capillary blood ~ **20** (**mlO2/dl for Hb14g/dl**)	End capillary blood ~ **21,5**
↓ ← venous admixture (shunt, V/Q mismatch) → ↓	
Arterial blood = **20**	Arterial blood ~ **21,3**
↓ ← diffusion of O2 to cells → ↓	
End-tissue capillary blood = **15**	End-tissue capillary blood **~16,3**
↓ ← consumption in cells(mainly mitochondria)→ ↓	
Mitochondria = 4 – 22mmHg	Mitochondria ~4-22mmHg

- Nb:
  - A-a difference is different on RA compared to 100% O2:
    - 21% = ~4mmHg
    - 100% = ~61mmHg

    ↳ this Big difference is due addition of small amount of physiological venous admixture OHDC at very high PaO2s is very flat; **MORE** flat than PaO2s at RA
- **NB the arterial/venous content differences for the two sets are the same**

Carbon Dioxide

- PaCO2 (ie tension) = most important stimulus to breath via central chemoreceptors
- CO2 content via tension:
  - Vent response based on tension
  - Easier to measure PCO2 than CO2 content
  - Co2 always moves down tension gradients even when it is opposite to the concentration gradients
  - Concept of tension can be applied to both gas & liquid phases with same significance
- PACO2 usually be taken as same as PaCO2 because:
  - Diffusion
  - V/Q mismatch
  - Shunt

  ↳ have less effect on CO2 than O2

  ↳ 10% shunt only ⇒ ↑PaCO2 by 0.7mmHg

  ↳ due to CO2 dissociation curve shape & rapid vent response to any ↑PaCO2
- ∴ factors effecting PACO2 will closely effect PaCO2 – see diagram

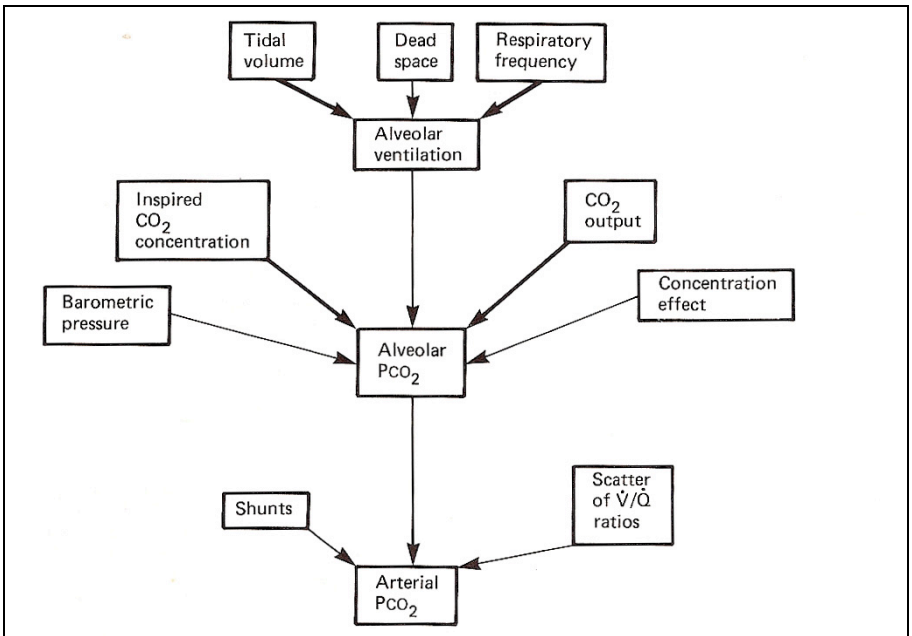


Figure 10.8 Summary of factors which influence P_{CO_2}; the more important ones are indicated with the thicker arrows. In the steady state, the CO_2 output of a resting subject usually lies within the range 150–200 ml/min and the alveolar P_{CO_2} is largely governed by the alveolar ventilation, provided that the inspired CO_2 concentration is zero. The barometric pressure is the only limit to the elevation of P_{CO_2} which may be brought about by the inhalation of gas mixtures containing CO_2. See text for explanation of the concentration effect.

(from hypoventilation section at start of this doc section:)

- alveolar ventilation and P_aCO2 relationship:

$$P_aCO2 = \frac{\text{CO2 production}}{\text{Alveolar Ventilation}} \times \text{constant}$$

↳ ∴ if 1/2 alveolar vent then PCO2 doubled in a steady state

CO2 production vs Output

- Co2 produced in mitochondria as product of metabolism (citric acid cycle)
- Amount produced depends on metabolic substrate ∴ effects RQ value:

- Carbohydrate (CHO) = 1
- Fats = 0.7
- Protein = 0.82

(RQ = volume of CO2 produced / volume of O2 consumed at steady state)

- Total CO2 in mixed venous blood – 52mlCO2/100ml blood
  ↳10% carried in dissolved form = PvCO2 45mmHg
- States of incr CO2 production:
  - Fever
  - Thyrotoxicosis
  - MH etc
- It is CO2 output & NOT production which effects PACO2
- In steady state: production = output
- In acute vent changes ie acute hypoventilation:
  - Temporary movement of CO2 into body stores ⇒
  - ↓output fall to very low levels
  - CO2 stores will fill ⇒ inevitable PACO2 rise (just temporarily delayed)

CO2 elimination

- Alveolar ventilation is vital to CO2 elimination
- Alveolar ventilation = (Vt – physiological dead space) x RR
  ↳ NB rarely does an ↑physiological dead space limit effective elimination

Factors influencing the carbon dioxide tension in the steady state 235

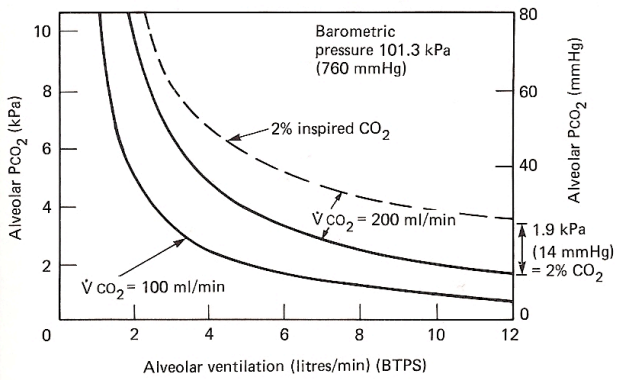


Figure 10.9 The effect of CO_2 output, alveolar ventilation and inspired CO_2 concentration on alveolar PCO_2. The lowest continuous curve shows the relationship between ventilation and alveolar PCO_2 for a carbon dioxide output of 100 ml/min (STPD). The upper continuous curve shows the relationship when the carbon dioxide output is 200 ml/min (STPD). The broken curve represents the relationship when the carbon dioxide output is 200 ml/min and there is an inspired CO_2 concentration of 2%. Two per cent CO_2 is equivalent to about 1.9 kPa (14 mmHg) and each point on the broken curve is 1.9 kPa above the upper of the two continuous curves. The continuous curves are rectangular hyperbolas with identical asymptotes (zero alveolar PCO_2 and zero alveolar ventilation). The broken curve is also a rectangular hyperbola but the horizontal asymptote is PCO_2 1.9 kPa (14 mmHg) which is the tension in the inspired gas.

Curve:

- Double alveolar vent from 4-8L/min = >1/2 PACO2 (40 ⇒ ~18mmHg)
- Half alveolar vent 4 – 2L /min = double PACO2 (40 ⇒ 80mmHg)

Inspired CO2

- Effect any inspired CO2 = additive to PACO2
  ↳ Eg rebreathing in Mapleson-type anaesthetic circuits with failing absorbers or insufficient fresh gas flow

Concentration Effect

- = where there is net transfer of inert soluble gasses across alveolar-capillary interface
  ↳ eg N2O at beginning of anaesthetic taken up in rapid & large quantities ⇒ ↑conc of PACO2 (& PAO2)

NB

The arterial-end **tidal** difference ($\mathbf{PaCO_2 - P_E{,}CO_2}$) = an index of ALVEOLAR dead space (eg ↓ CO, pulmonary embolism)

Causes of Hypercapnea

- in norm person rare to have PaCO2 >45mmHg
- breath holding can only achieve level ~50mmHg
- 4 possible mechanisms:
  - alveolar hypoventilation
    - most common by far
    - if spont breath on RA not possible to have PaCO2>100mmHg
      ↳ because accompanying hypoxaemia is critical (alveolar gas eq)
  - ↑dead space (V/Q mismatch)
    - rare
    - need excessively large alveolar dead space is large PE, v low cardiac output
  - ↑FiCO2
    - rare
    - rebreathing
  - ↑ed CO2 production:
    - eg MH
    - only common when ventilation is fixed ie on vent

Summary

- 4 causes of hypoxaemia: hypovent, diffusion limitation, shunt, VQ mismatch
- 4 causes of hypercapnia: hypovent, VQ mismatch, ↑FiCO2, ↑CO2 production
- shunt only cause of hypoxaemia where P_aO2 does not rise to expected level when given 100% O2
- VQ ration determines PO2 & PCO2 in any lung unit
- At apex of lung VQ ratio is high ∴ PO2 high, PCO2 low
- VQ mismatch ↓s gas exchange efficiency of lung for all gases
  ↳but P_aCO2 changes masked as chemoreceptors compensate by ↑ing ventilation
  ↳though P_aO2 always low
  ↳attributable to dissociation curves
- A-a Po2 difference = useful measure of VQ mismatch. Expected PaO2 calculated using alveolar gas equation
- Hypoxaemia due to hypoventilation can easily be corrected by ↑ing Fio2
- Hypercapnia due to hypoventilation can only be remedied by correcting the ventilation