VENTILATION AND PERFUSION IN HEALTH AND DISEASE

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Ventilation

- *Total ventilation* - total rate of air flow in and out of the lung during normal tidal breathing.
- *Alveolar ventilation* -represents the amount of fresh inspired air available for gas exchange in alveolar gas compartment

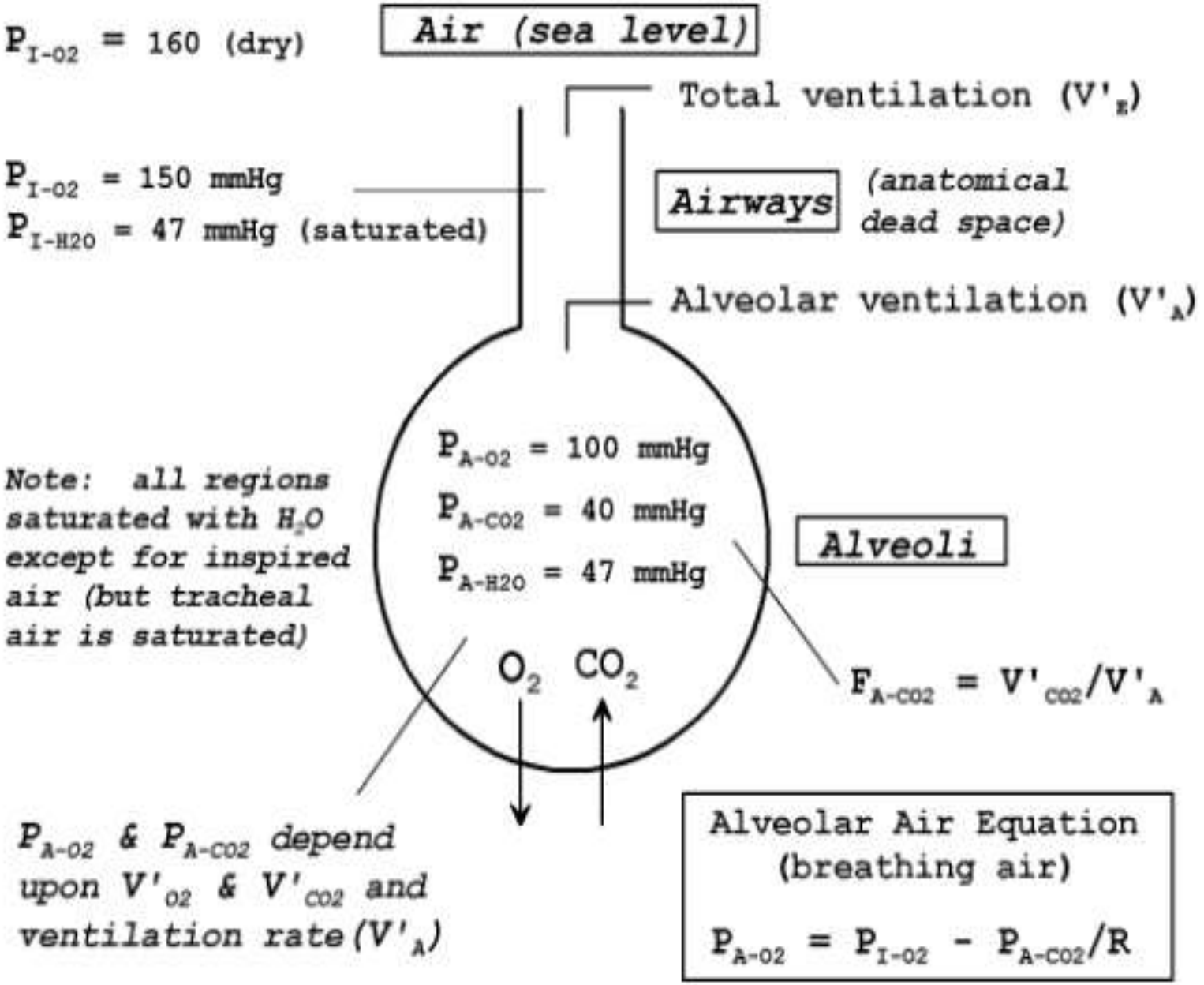
$$V_A = V_E - V_D$$

$$V_A \times n = V_T \times n - V_D \times n$$

Also;

$$V_{CO2} = V_A \times F_{CO2}$$

$$V_A = V_{CO2} / P_{CO2}$$


P_{I-O2} = 160 (dry)
Air (sea level)
Total ventilation (V'_E)
P_{I-O2} = 150 mmHg
P_{I-H2O} = 47 mmHg (saturated)
Airways
(anatomical dead space)
Alveolar ventilation (V'_A)
P_{A-O2} = 100 mmHg
P_{A-CO2} = 40 mmHg
P_{A-H2O} = 47 mmHg
Alveoli
Note: all regions saturated with H_2O except for inspired air (but tracheal air is saturated)
O_2 CO_2
$F_{A-CO2} = V'_{CO2}/V'_A$
P_{A-O2} & P_{A-CO2} depend upon V'_{O2} & V'_{CO2} and ventilation rate (V'_A)
Alveolar Air Equation
(breathing air)
$P_{A-O2} = P_{I-O2} - P_{A-CO2}/R$

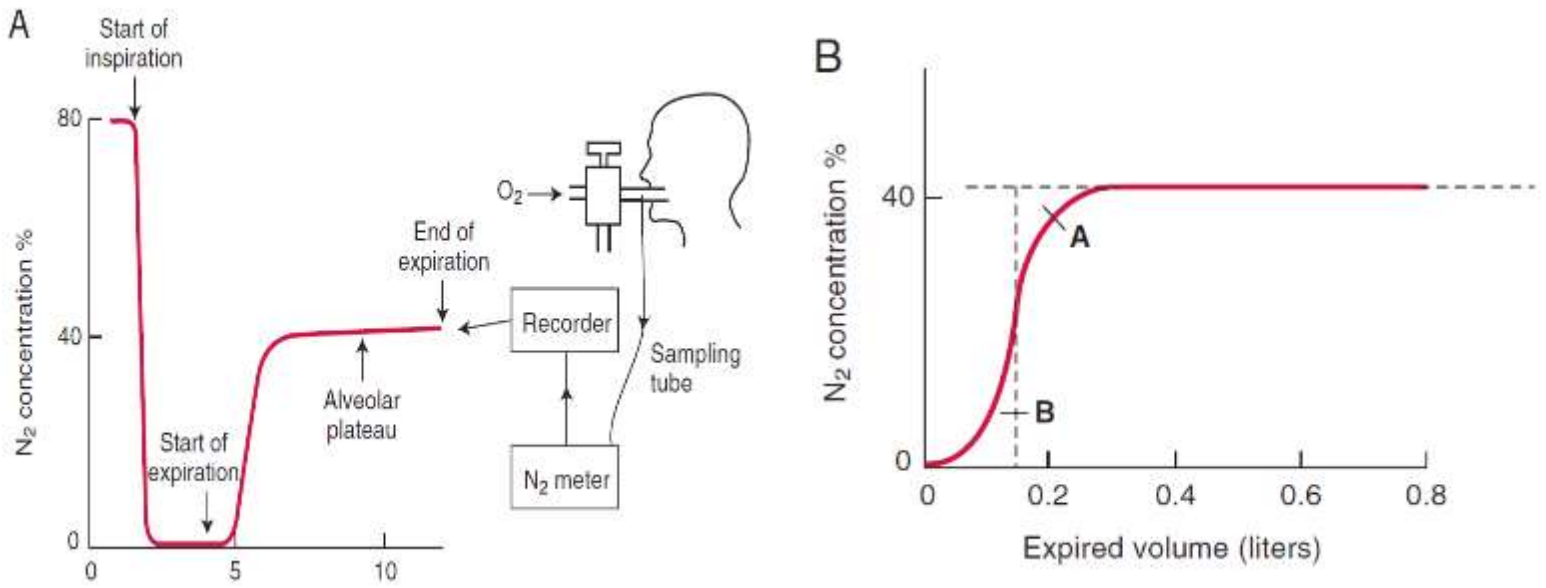
Alveolar gas equation

$$p_AO_2 \approx F_IO_2(P_{ATM} - pH_2O) - \frac{p_aCO_2}{RER}$$ where:

pAO2	The alveolar partial pressure of oxygen	107 mmHg
FIO2	The fraction of inspired gas that is oxygen	.21
PATM	The prevailing atmospheric pressure	760 mmHg
pH2O	The saturated vapour pressure of water at body temperature	47 mmHg
paCO2	The arterial partial pressure of carbon dioxide (pCO2)	40 mmHg
RER	The respiratory exchange ratio	0.8

Anatomical Dead space

- Volume of the conducting airways.
- Usually 150 Ml.
- Increases with large breath.
- Measured by fowler's or N_2 wash out method.



- Subject breaths though a valve box
- N2 concentration at the mouth is analyzed
- Following a single breath of pure O_2,N_2 conc increases as dead space gas is washed out by the alveolar gas, finally reaching a plateau.

Physiologic dead space

Physiologic dead space is the volume of lung that does not eliminate CO2

$$\frac{V_D}{V_T} = \frac{P_{A_{CO_2}} - P_{E_{CO_2}}}{P_{A_{CO_2}}} \quad \text{(Bohr equation)}$$

- In normal subjects,
Anatomical D.S = Physiological DS
- patients with lung disease, the physiologic DS > Anatomical D.S
  - inequality of blood flow and ventilation within the lung

Breathing Pattern	Tidal Volume (ml)	Breathing Frequency (breaths/min)	Minute Ventilation (ml/min)	Dead Space Ventilation (ml/min)	Alveolar Ventilation (ml/min)
normal quiet breathing	500	12	6000	150X12=1800	4200
shallow & fast	150	40	6000	150X40=6000	0
deep & slow	1000	6	6000	150X6=900	5100

Dead space ventilation

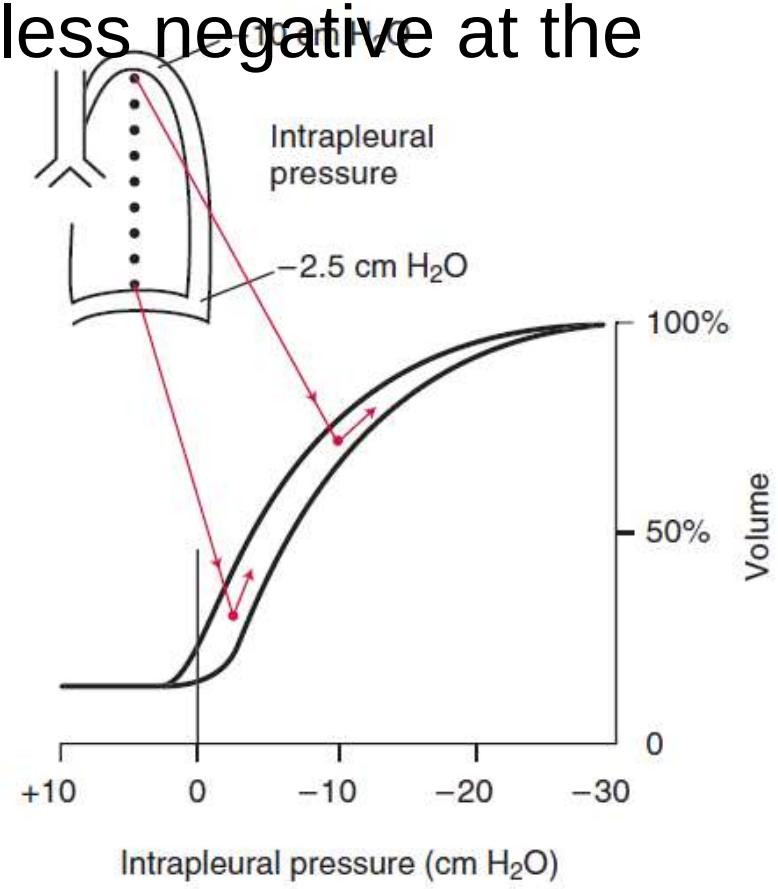
- Pulmonary embolism
- Vascular obliteration ex. PAH
- Emphysema

Regional Differences in Ventilation

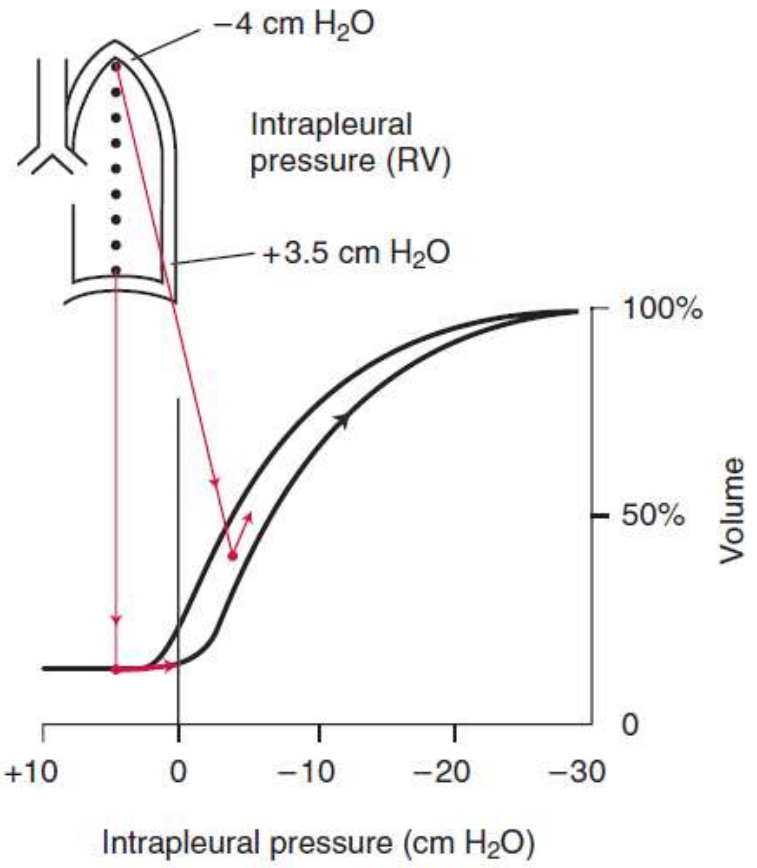
- *“change in volume per unit resting volume” !*
- lower regions of the lung ventilate better than do the upper zones.
- Supine position-ventilation of the lowermost (posterior) lung exceeds that of the uppermost (anterior) lung.
- Lateral position -the dependent lung is best ventilated.

Why ?

- weight of the lung - lower portions of the lung requires a larger pressure below it than above it
- intrapleural pressure is less negative at the bottom.
- Basal region has a small resting volume, expanding pressure & placed on the steep part of the pressure volume curve.
- ‘Paradox’



- At low lung volumes the intrapleural pressu irway pressu
- the lun d, and ve til the intrapl
- apex o ble part of the pressure-volume curve and ventilates well.

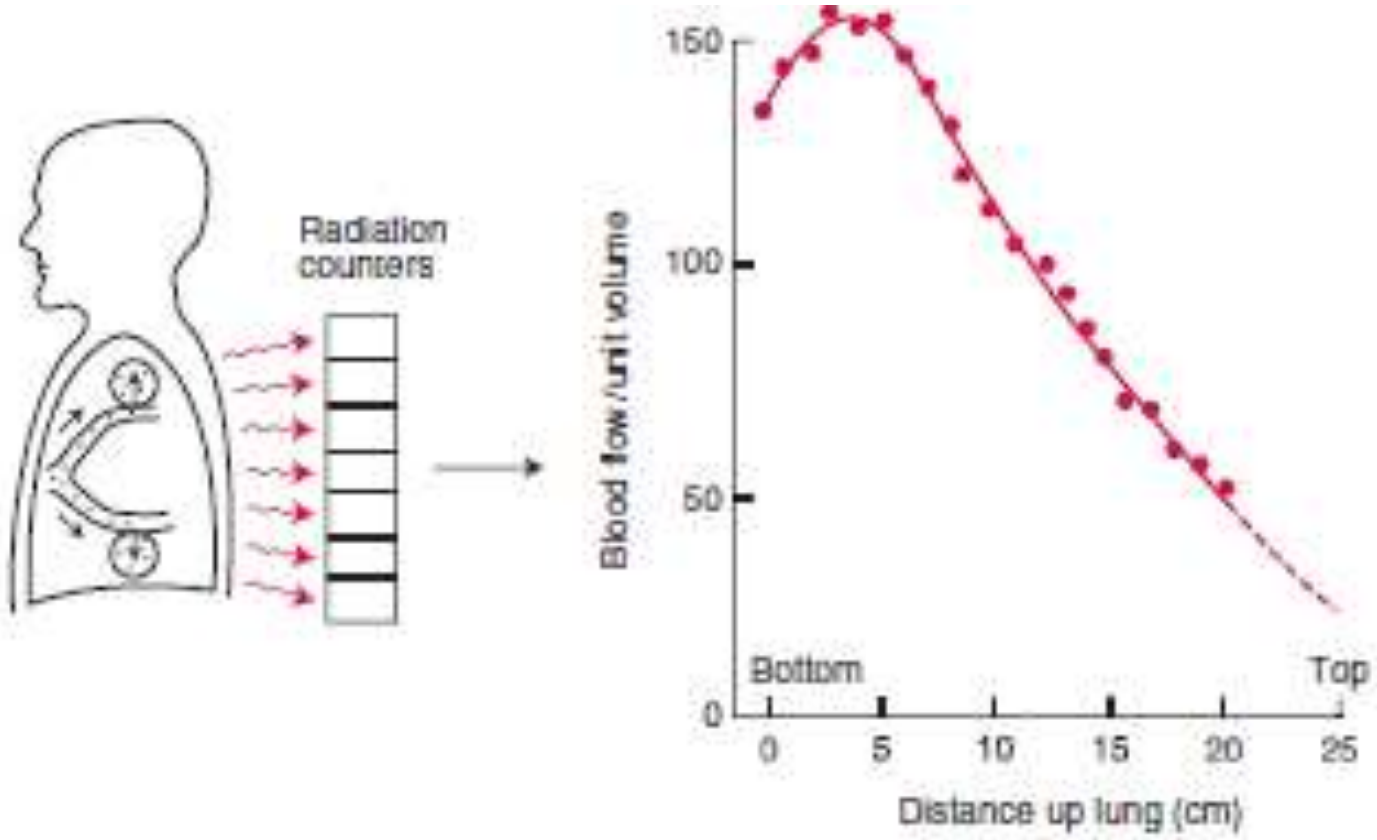


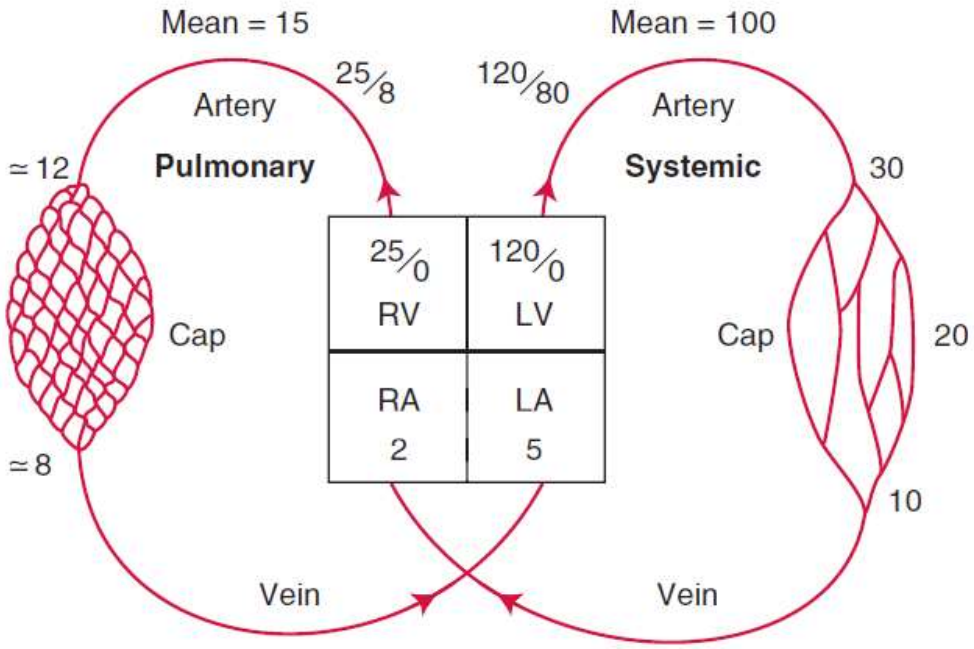
Airway Closure

- The compressed region of lung at the base does not have all its gas exhaled out.
- airways in the region of respiratory bronchioles close first trapping gas in the distal alveoli.
  1. only at very low lung volumes in young normal subjects.
  2. Elderly airway closure in the lowermost regions of the lung occurs at higher volumes
- Dependent regions of the lung may be only intermittently ventilated leading to defective gas exchange

Distribution of Blood Flow

- Blood flow within the lung is unequal.





ɔoth upper and lower
crease, and the regional
e less pronounced.

ɹtion of blood flow is due
ressure differences
within the blood vessels.

- difference in pressure between the top and bottom- 30 cm of H_2O
- This is a large pressure difference for a low-pressure system as the pulmonary circulation

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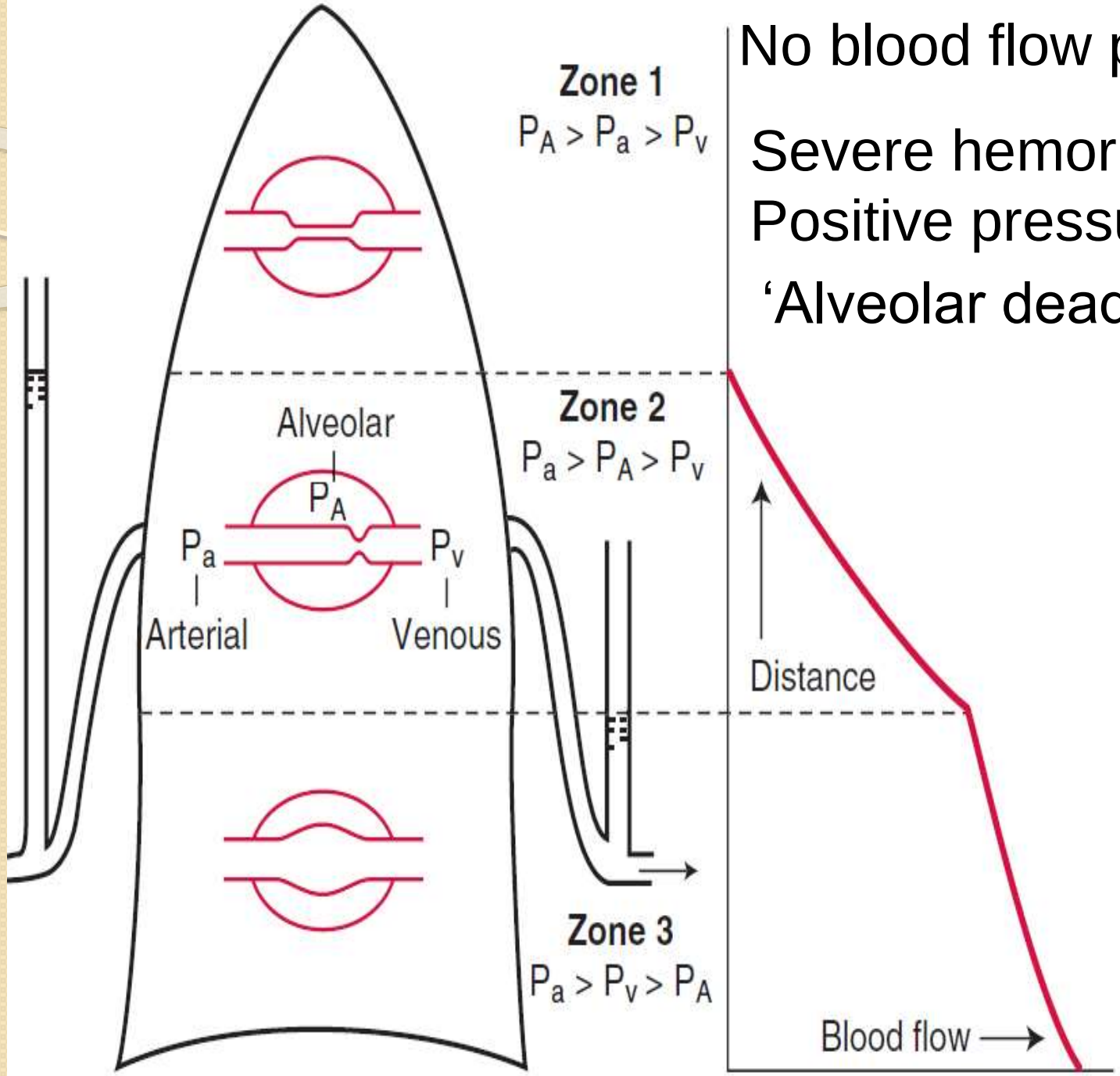


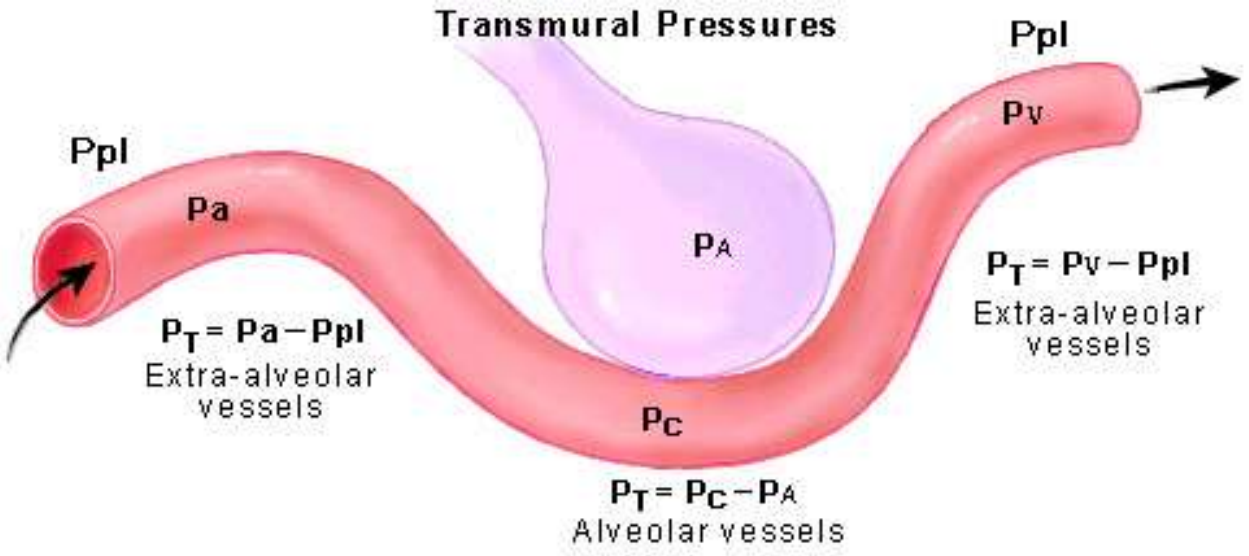
No blood flow possible.

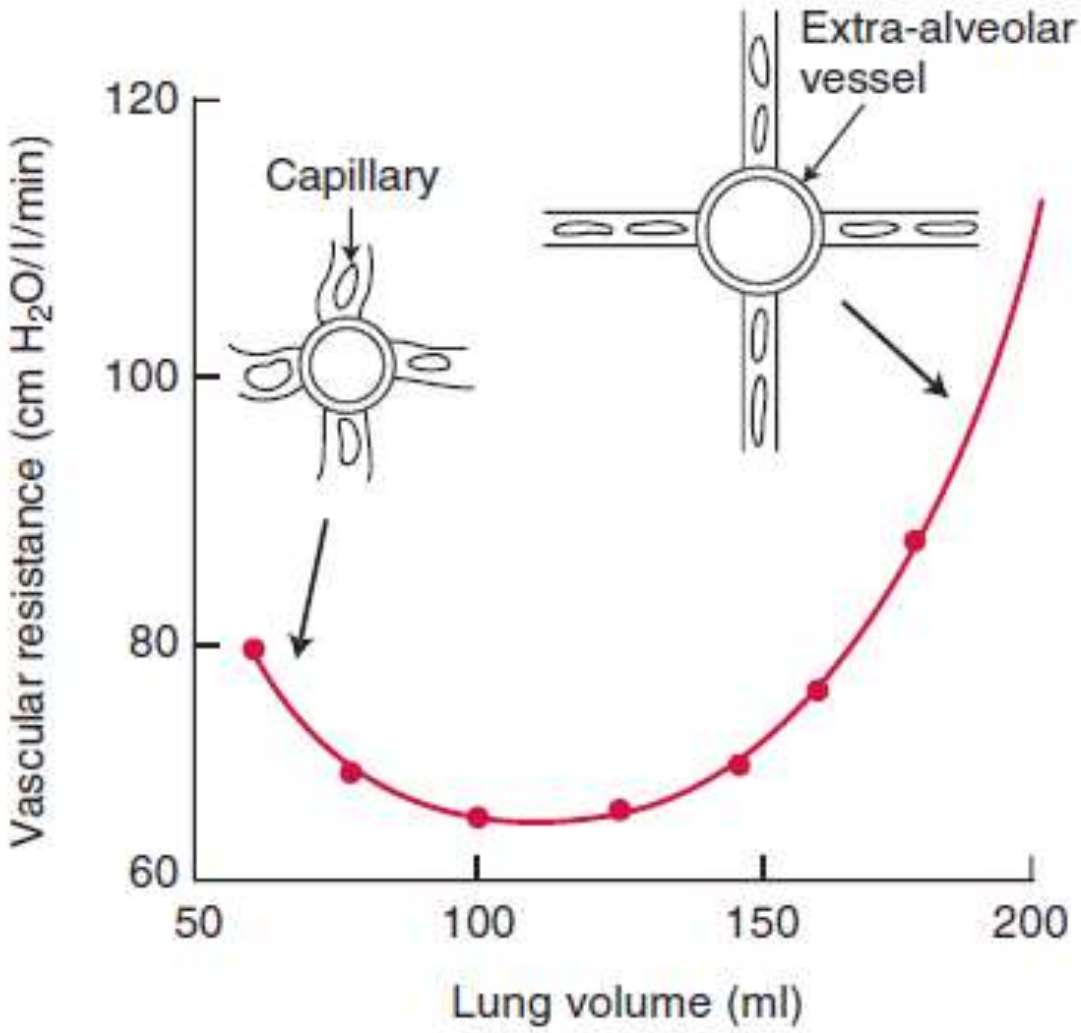
Severe hemorrhage

Positive pressure ventila

'Alveolar dead space.'

- Zone 4 -can be seen at the lung bases at low lung volumes or in Pulmonary oedema.
- Pulmonary interstitial pressure (Pi) rises as lung volume decreases due to reduced radial tethering of the lung Parenchyma
- Pa > Pi > Pv > PA




Extra-alveolar vessel
Capillary
120
100
80
60
Vascular resistance (cm H_2O/l/min)
50
100
150
200
Lung volume (ml)

Shunt

- blood that enters the arterial system without going through ventilated areas of the lung.
- Physiological
  - thebesian veins
  - Bronchial veins
- Pathological
  - Intracardiac
  - A-V malformations

Shunt equation

$$\dot{Q}_T \times Ca_{O_2} = \dot{Q}_S \times C\bar{v}_{O_2} + (\dot{Q}_T - \dot{Q}_S) \times Cc'_{O_2}$$

$$\frac{\dot{Q}_S}{\dot{Q}_T} = \frac{Cc'_{O_2} - Ca_{O_2}}{Cc'_{O_2} - C\bar{v}_{O_2}}$$

$Q_{T\,=}$ total blood flow

Qs= shunt blood flow

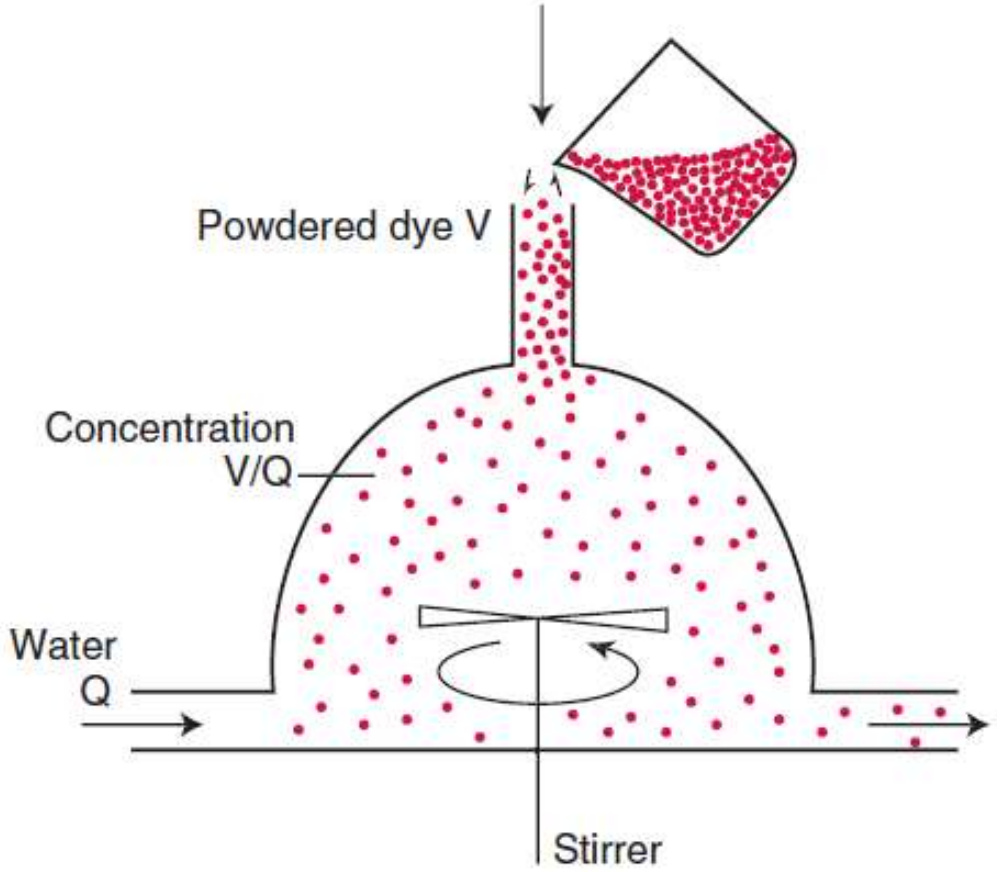
CvO2= O2 concentration in shunted blood

Cc'O2= O2 concentration in end-capillary blood

$CaO_{2\,=}O_2$ concentration in the arterial blood

- a 5% shunt for every 100 mm Hg decrease in Pao2 below 700 mm Hg while the patient is breathing 100%.
- If Pa_{O2} on Fi_{O2} 1.0 is 200, the shunt is approximately (700-200)* 5 ie 25 %.
- Pure shunt does not respond to increase in $Fi_{O2.}$
- ARDS – 25-50% shunt, responds to PEEP.

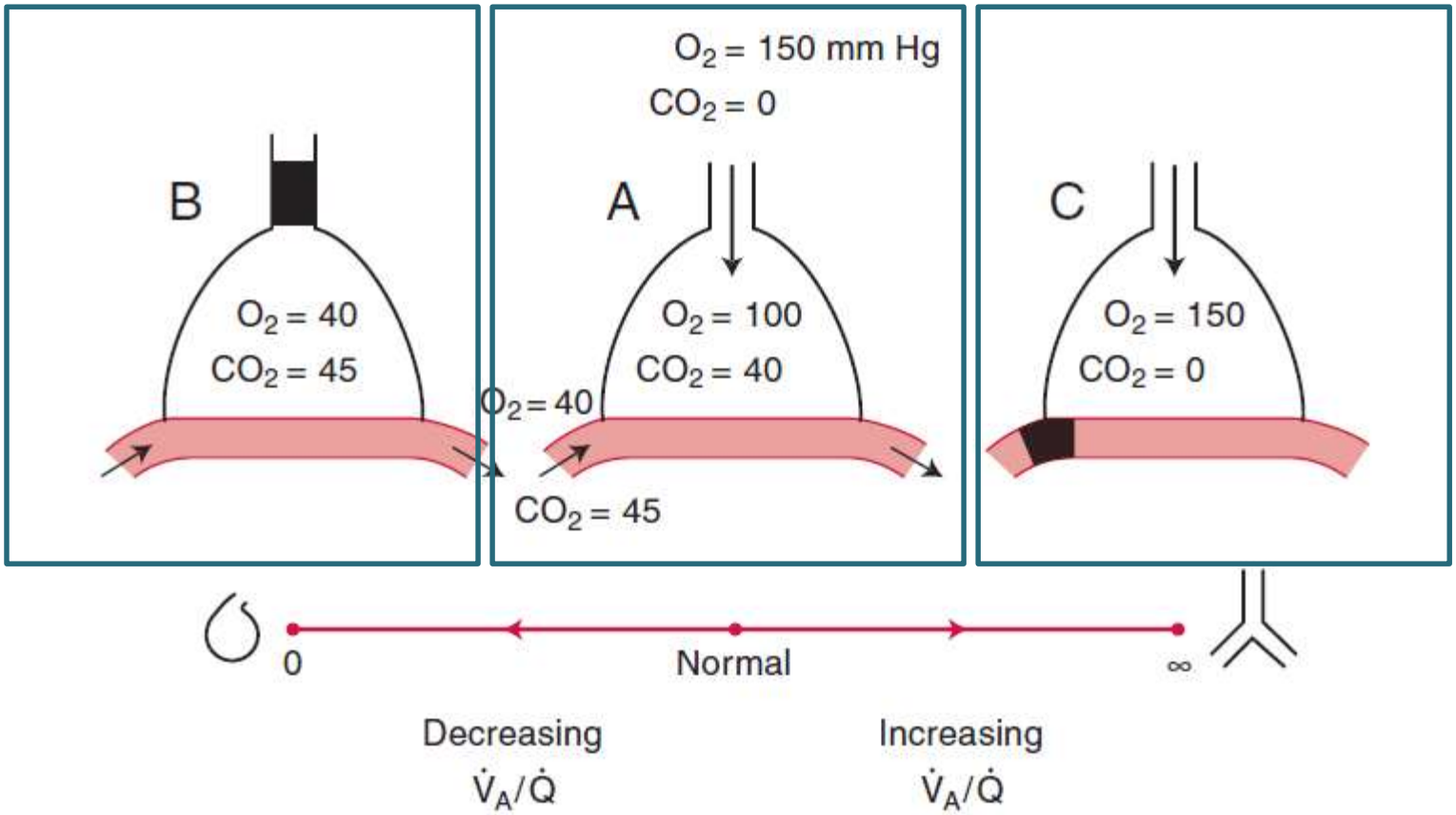
Ventilation-Perfusion Ratio



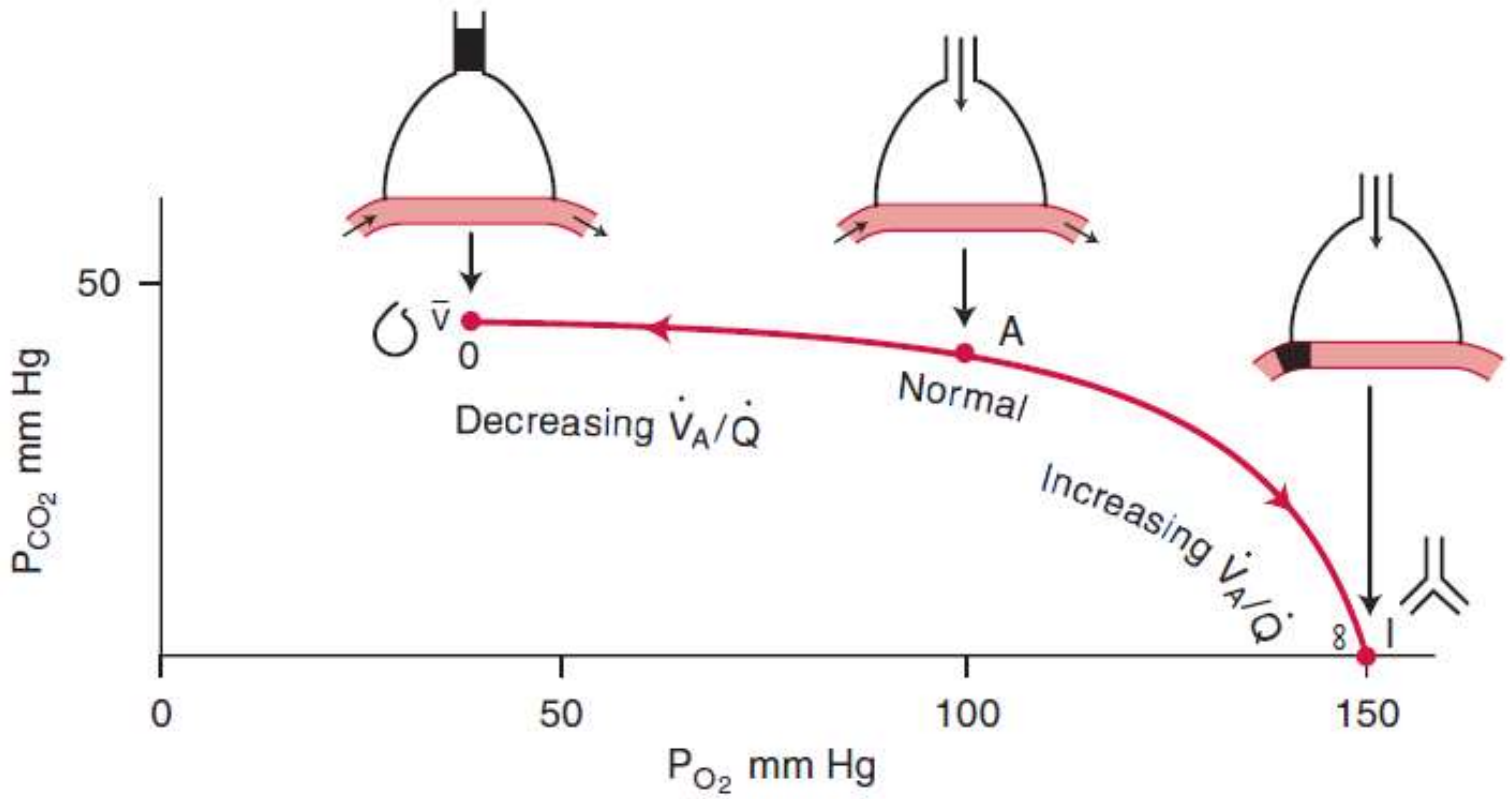
What determines the concentration of dye ?

- rate at which the dye is added (ventilation)
- rate at which water is pumped (blood flow)

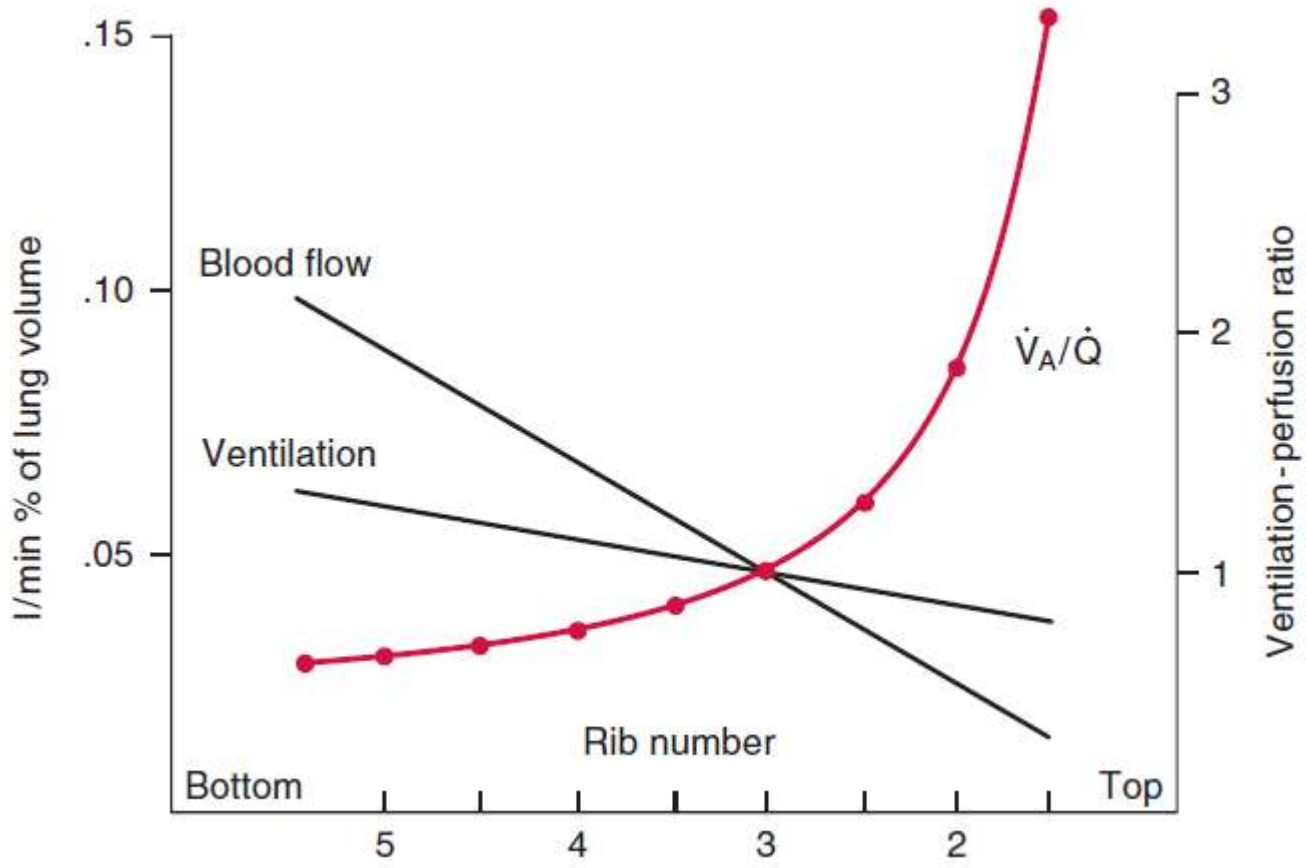
Alterations in the Ventilation-Perfusion Ratio



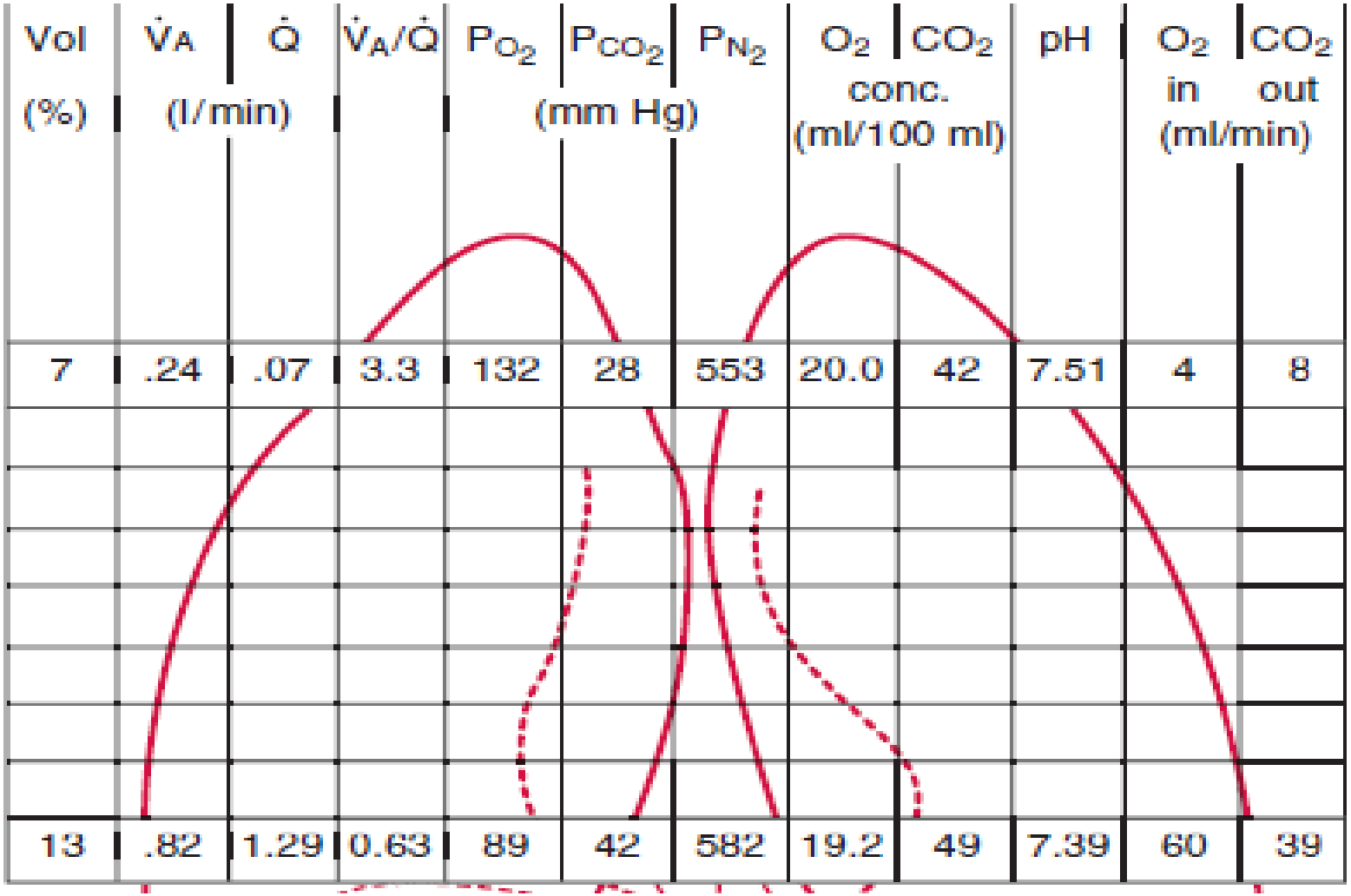
O2-CO2 diagram



Regional changes in V-Q

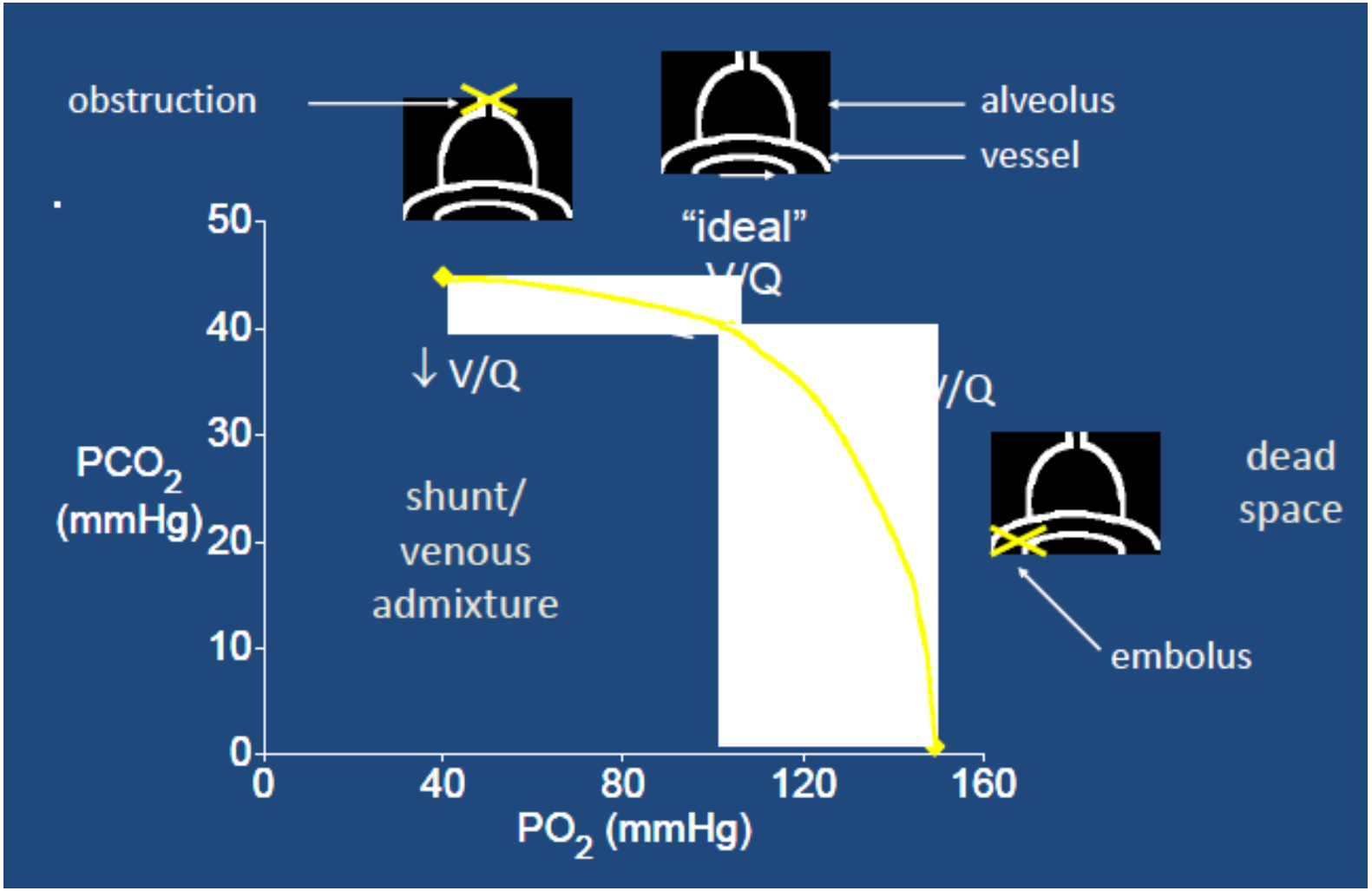


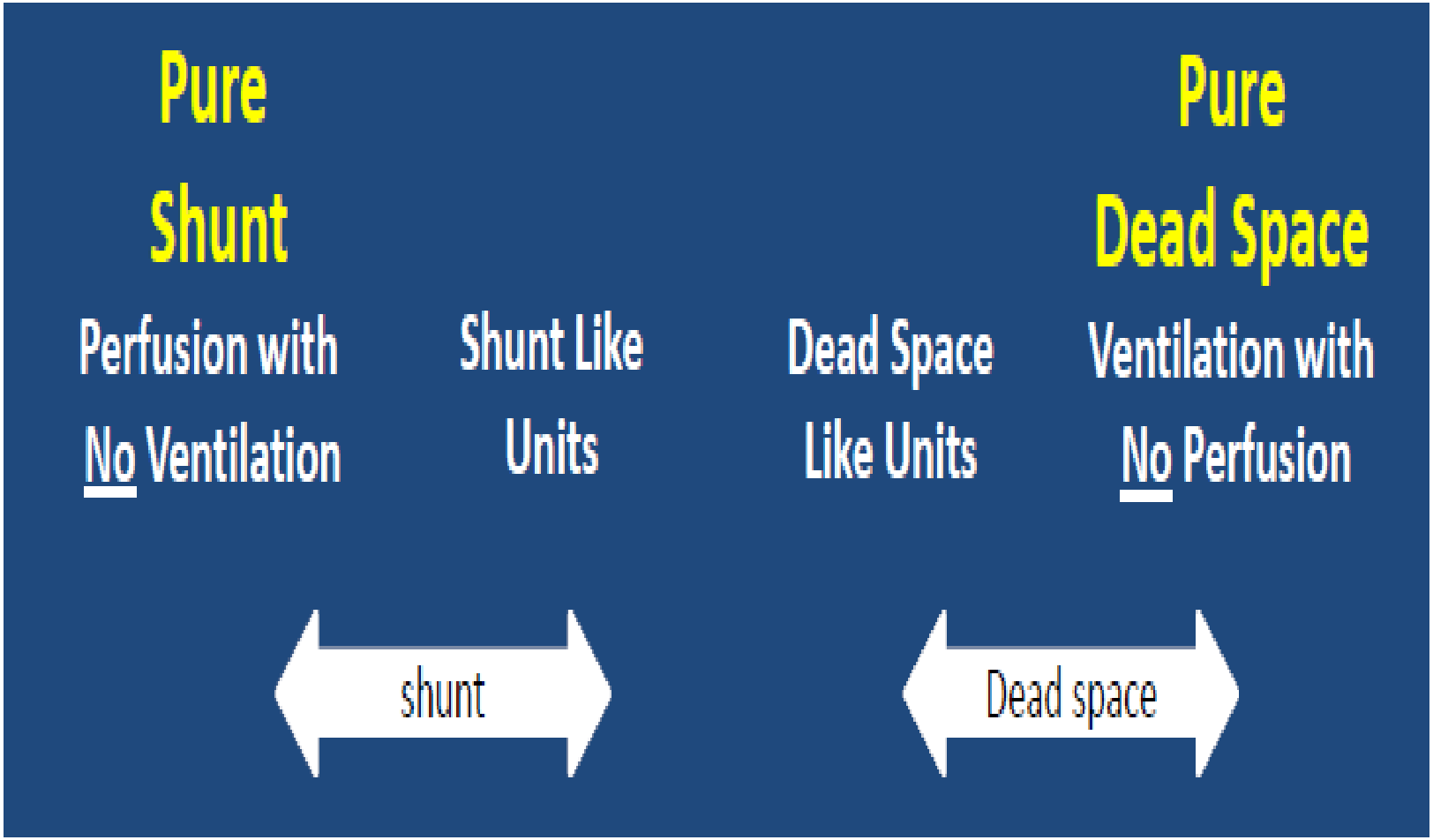
Regional differences in gas exchange down the normal lung



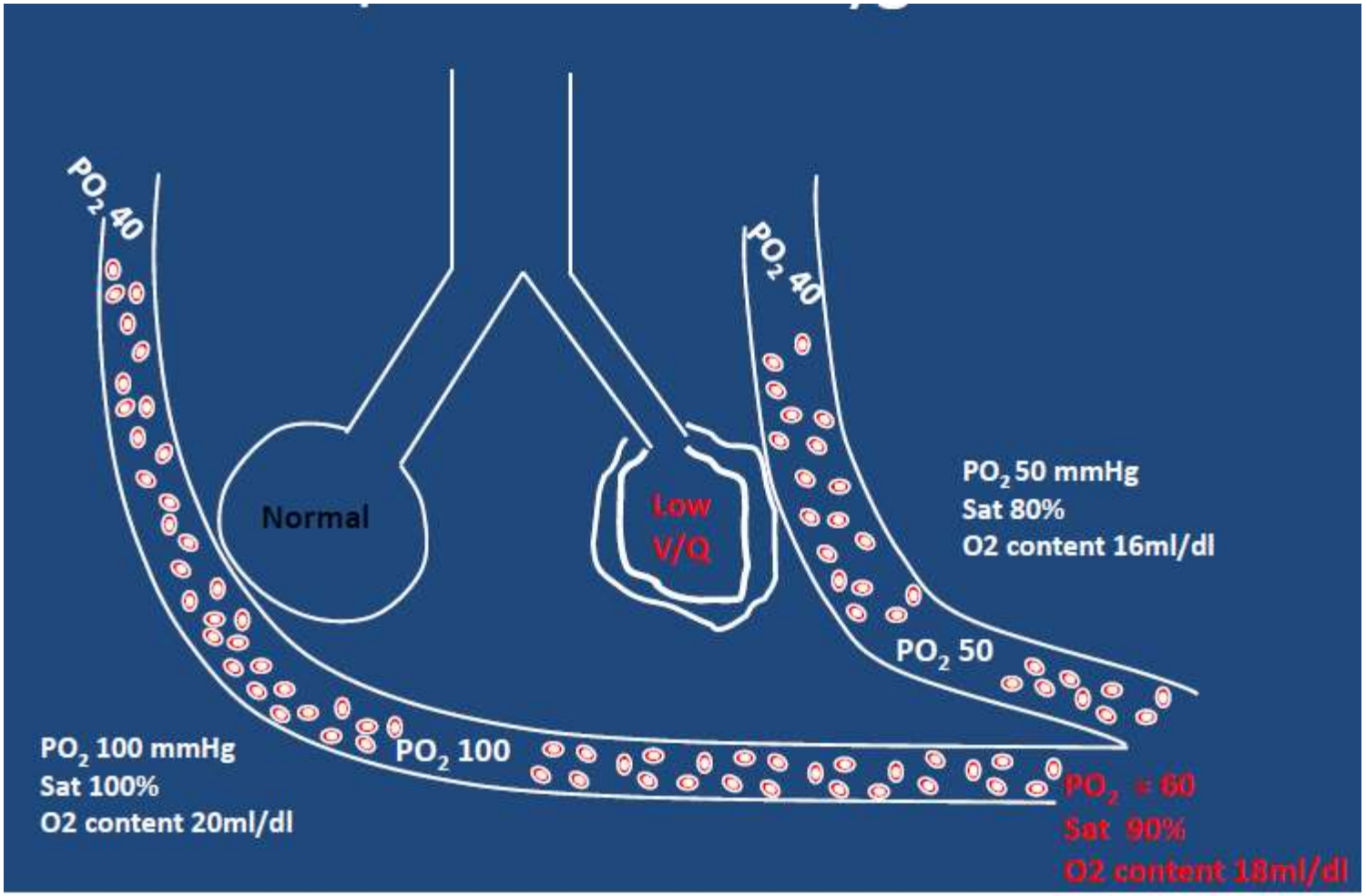
- Po2 at the apex is higher than at the base of the lung.
- However, the major share of the blood leaving the lung comes from the lower zones, where the Po2 is low.
- This depresses the arterial oxygen saturation.

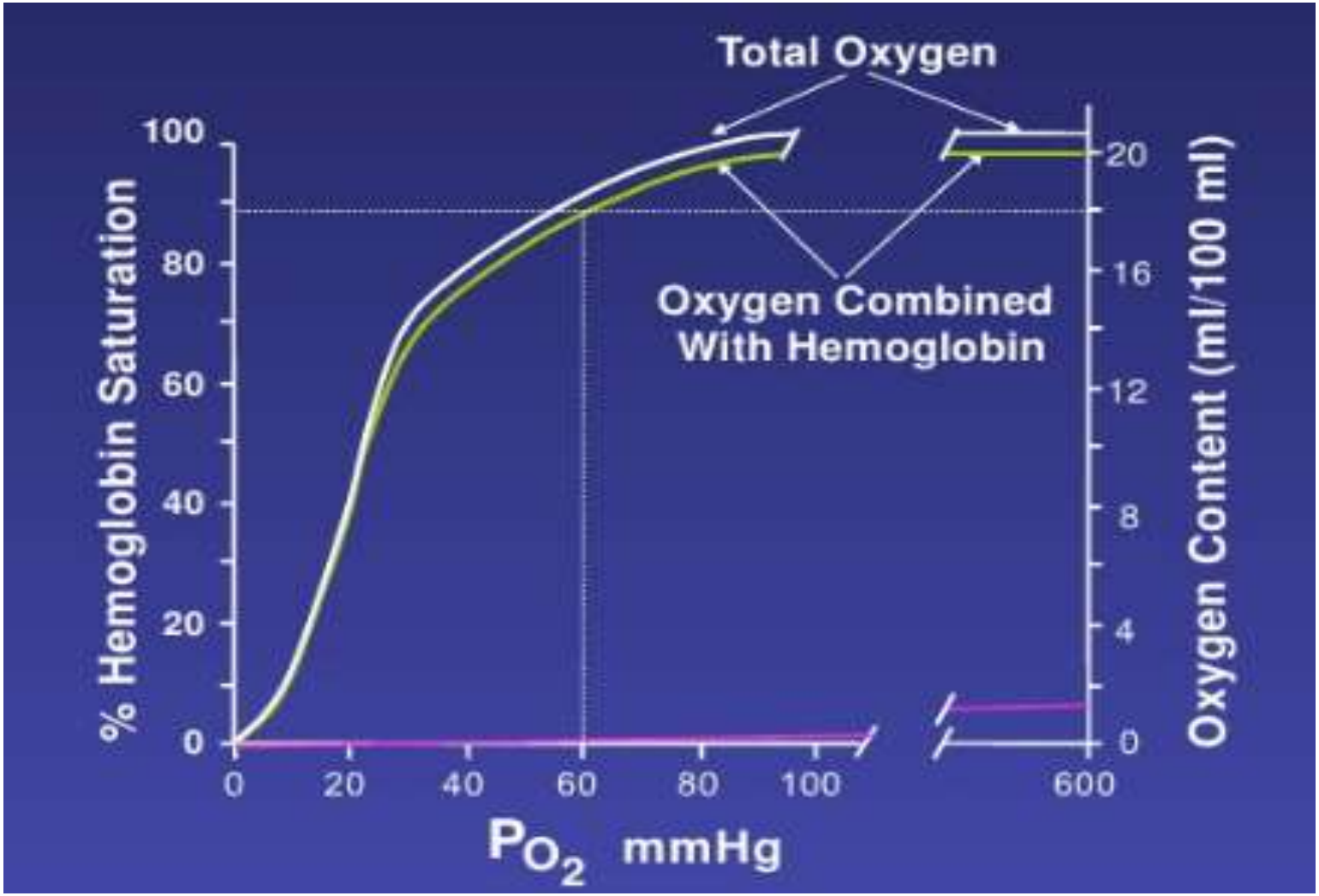
V-Q mismatch in disease state.




Pure
Shunt
Perfusion with
No Ventilation
Shunt Like
Units
Dead Space
Like Units
Pure
Dead Space
Ventilation with
No Perfusion
shunt
Dead space

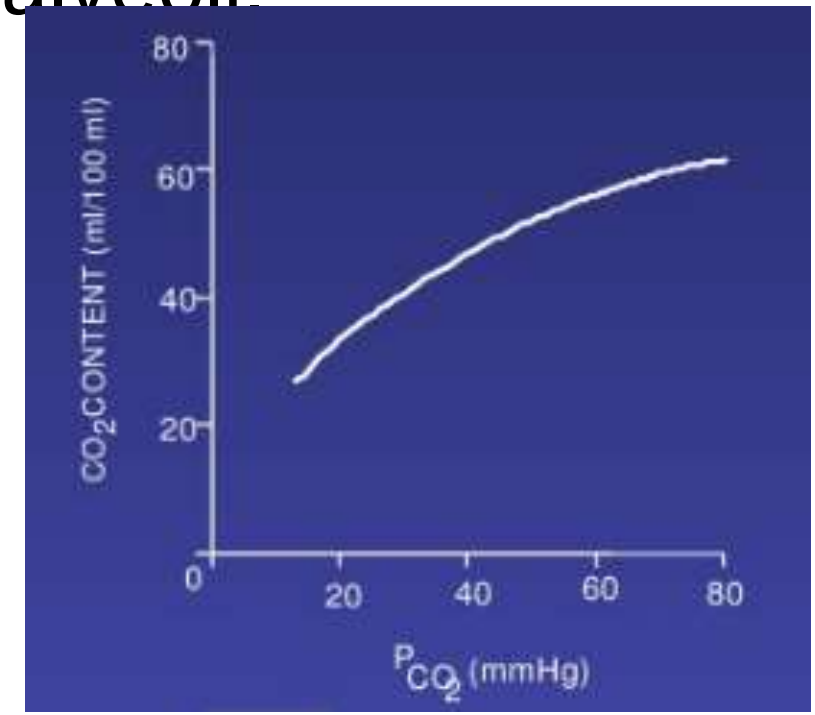
Low V-Q ratio.


PO2 40
PO2 40
Normal
Low V/Q
PO2 50 mmHg
Sat 80%
O2 content 16ml/dl
PO2 50
PO2 100 mmHg
Sat 100%
O2 content 20ml/dl
PO2 100
PO2 = 60
Sat 90%
O2 content 18ml/dl

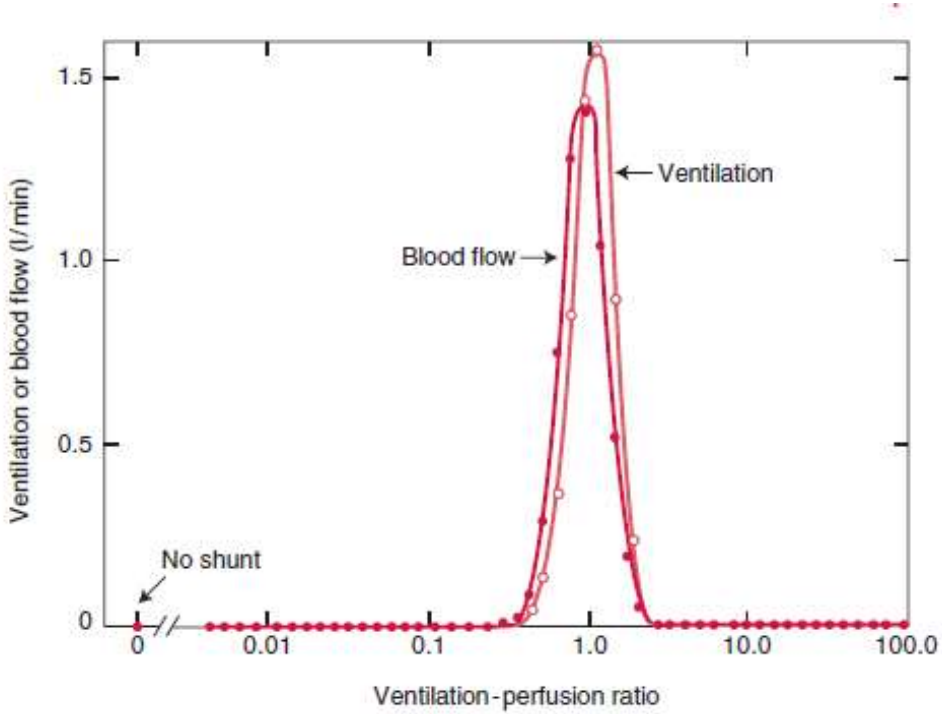

Total Oxygen
Oxygen Combined With Hemoglobin
% Hemoglobin Saturation
Oxygen Content (ml/100 ml)
100
80
60
40
20
0
20
16
12
8
4
0
0
20
40
60
80
100
600
P_{O_2} mmHg

- Units with high V-Q ratio add little oxygen to the blood, compared the decrement caused by the alveoli with the low ratio.
- The net result of these mechanisms is a depression of the arterial Po2 below that of the mixed alveolar Po2—alveolar-arterial O2 difference.
- Normal-4

- CO2 dissociation curve is linear
- Although the elimination of CO2 is impaired by V/Q inequality, this can be corrected by increasing the ventilation to the alveoli



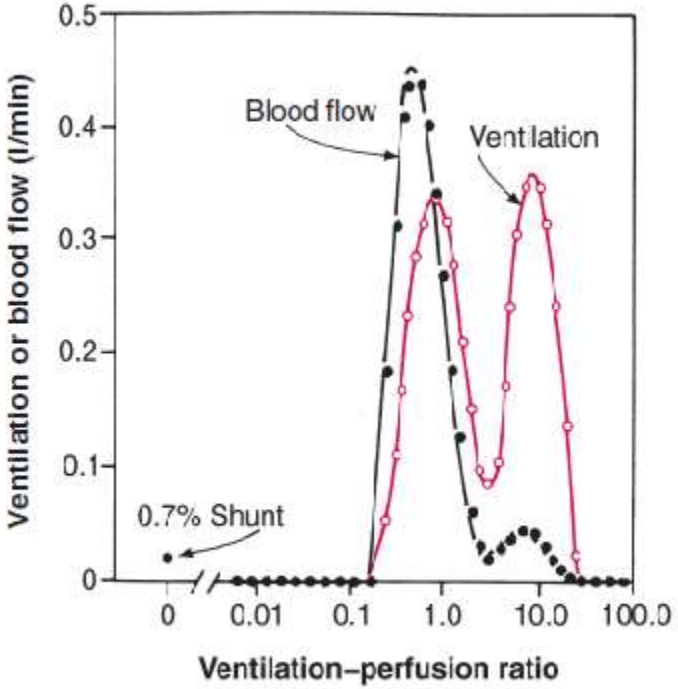
Distributions of Ventilation-Perfusion Ratios



- All the ventilation and blood flow goes to compartments with normal V-Q ratio of 1.0.
- no blood flow to the unventilated compartment (shunt).

V-Q distribution in COPD

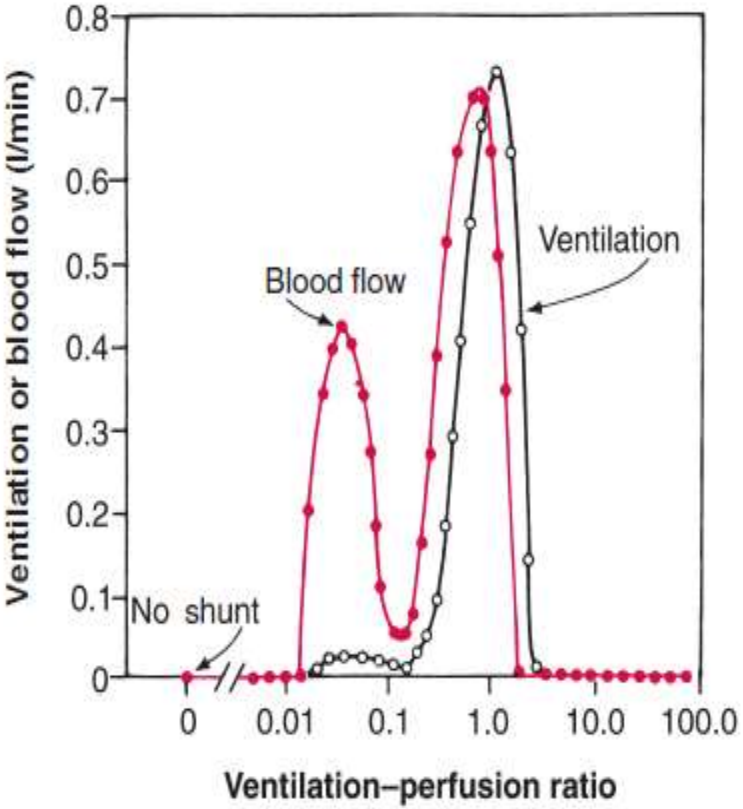
- large amount of ventilation to lung units with high V-Q ratios.
- ‘Physiologic dead space’



• excessive ventilation to high V-Q units constitute ‘wasted ventilation’.

• There is little blood flow to units with an abnormally low VA /Q, causing hypoxemia.

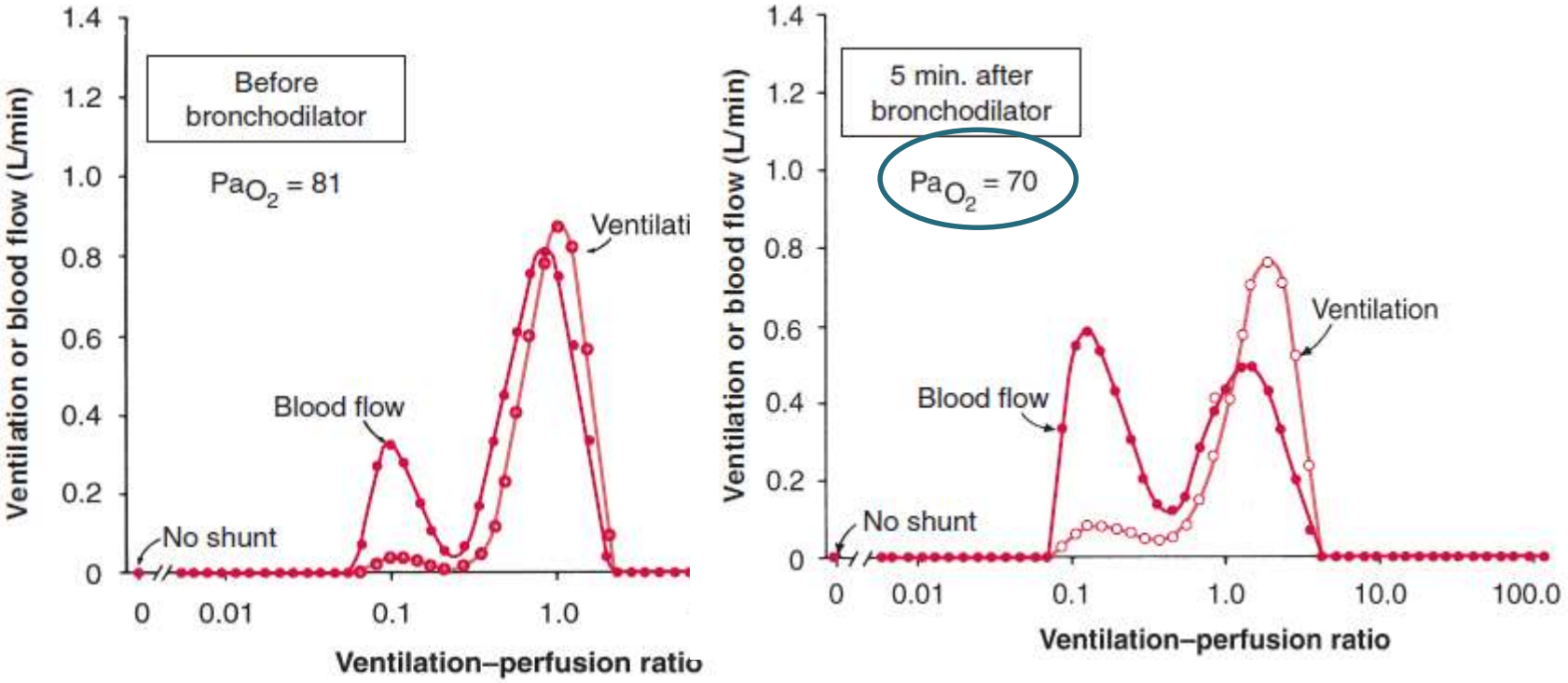
- Increase in ventilation to high VA /Q units →physiologic dead space.
- large amounts of blood flow to low VA /Q units →physiologic shunt → severe hypoxemia
- type B disease.



Hypoxic vasoconstriction

- Alveolar hypoxia constricts small pulmonary arteries
- direct effect of the low PO2 on vascular smooth muscle
- Directs blood flow away from poorly ventilated areas →minimizing the arterial hypoxemia.
- Bronchodilators can abolish this mechanism and cause mild hypoxia by increasing the blood flow to poorly ventilated areas.

V-Q mismatch in Asthma

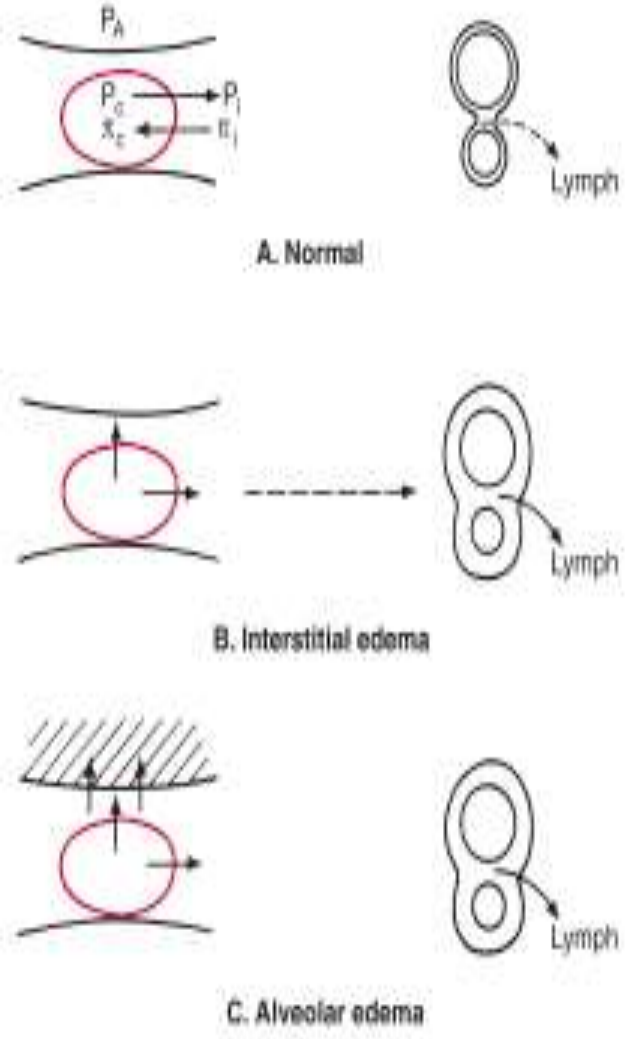


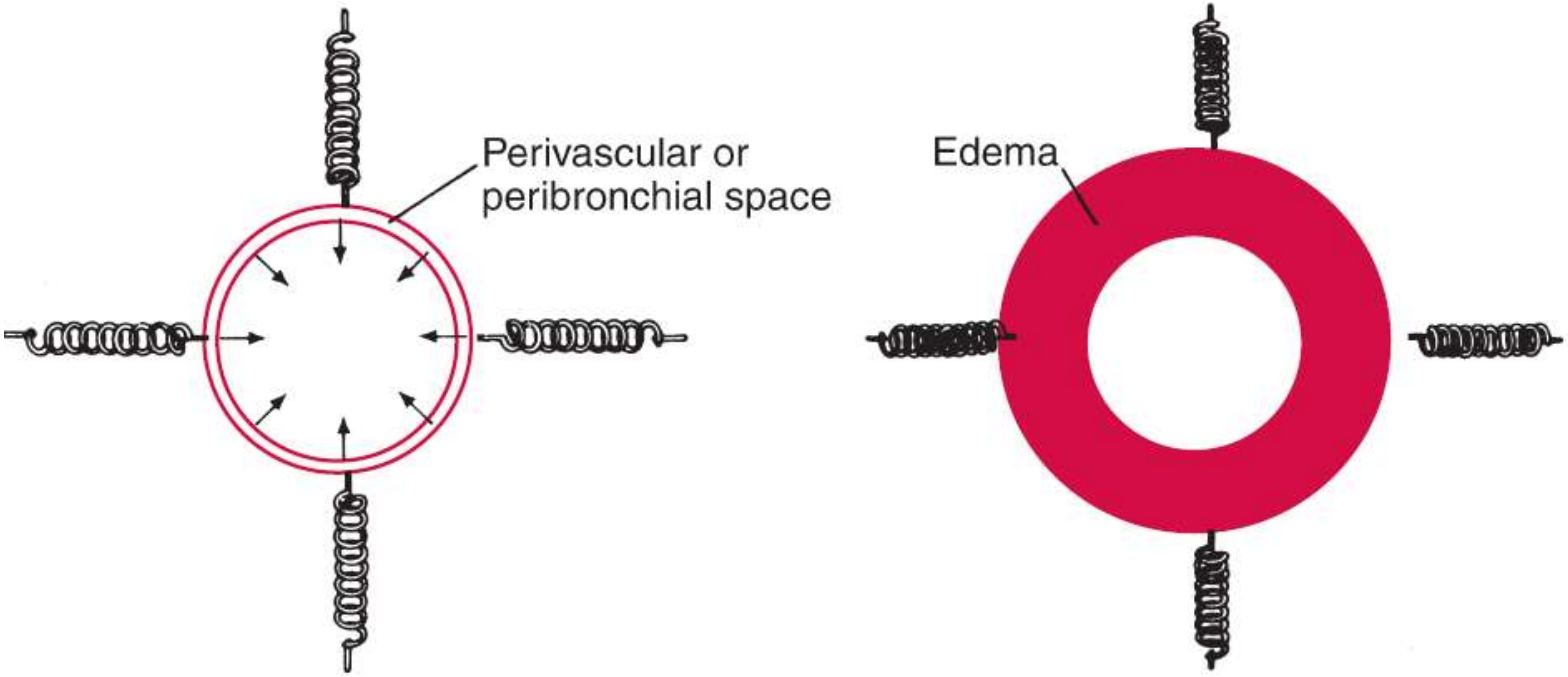
- considerable amount of the total blood flow to units with a low VA /Q →mild hypoxemia

- Bronchodilators increase the hypoxemia, by abolishing the HPV and , increasing the blood flow to lung units with low V/Q ratio’s

V-Q mismatch in Pulmonary Edema

- *abnormal accumulation of fluid in the extravascular spaces and tissues.*
- severe hypoxemia results from;
  - Shunt - > 50 %
  - Blood flow to low V-Q areas




Perivascular or
peribronchial space
Edema

Pulmonary Embolism

- Moderate hypoxemia without carbon dioxide retention
  - diffusion impairment
  - opening up of latent pulmonary a-v anastomoses
  - V-Q inequality-areas with High V-Q ratios
  - dead space ventilation
  - Shunting

- blood flow is greatly reduced but not completely absent, leading to areas with high V-Q ratio’s.
- Shunt occurs due to blood flow through the areas with hemorrhagic atelectasis, in which the alveoli are not functional.

- The large physiological dead space in pulmonary embolism can cause hypercapnia, however the substantial increase in ventilation maintains the $Paco_2$ at a normal level.
- In the setting of acute PE, hypercapnia reflects massive embolism as the ventilatory muscles are unable to sustain the marked increase of minute ventilation needed to maintain normal arterial Paco2

Interstitial Pulmonary Fibrosis

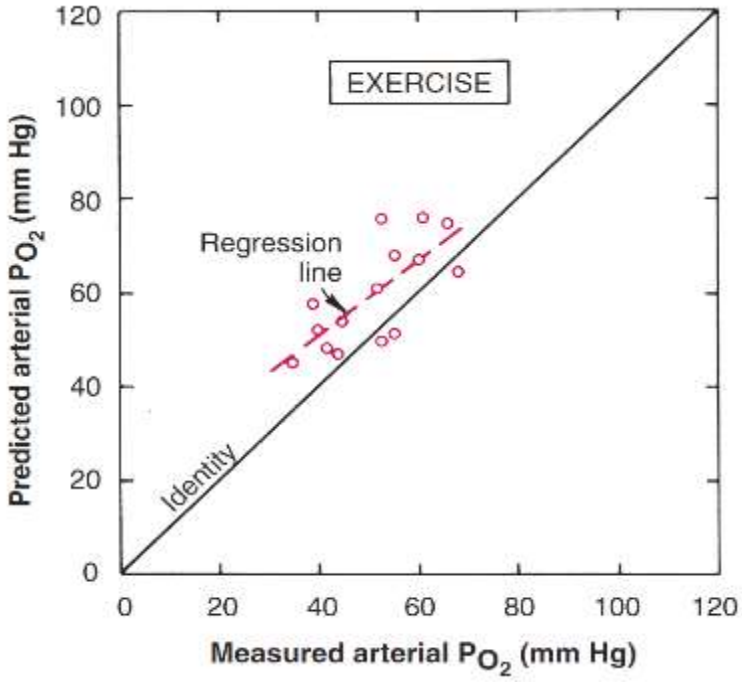
- Basic pathology -thickening of the interstitium of the alveolar wall
- Spirometry -restrictive pattern
- Characterized by hypoxia and hypocapnia at rest.
- mild hypoxia at rest, worsened by exercise.

Cause of hypoxia ?

Rest	Exercise
V-Q Inequality Major factor during rest D.I minor factor, as lung as enormous reserves of diffusion at rest.	Diffusion impairment Major factor during exercise Exercise -↓ time for RBC'S in pulmonary circulation Further worsens hypoxia
	V-Q Inequality
	Inadequate ↑ in Cardiac output (↓PaO2 in mixed blood, ↑PVR)
	Inappropriately high Respiratory rate→↑ dead

- hypoxemia can be explained by the degree of VA /Q inequality at rest.
- Measured PaO2 below predicted in exercise !

  Additional hypoxemia is due to D.I



DL_{Co} as a diagnostic test ?

- Dlco is strikingly reduced in ILD
- Remains *low in exercise*, normal- ↑ 2-3 fold.
- Etiology-
  - thickening of the blood–gas barrier
  - ↓blood volume because of obliterated of blood vessels by the fibrotic process
- →if the Dlco is *not low*, the diagnosis of ILDshould be regarded with suspicion.

Diseases of the Chest Wall

- **Scoliosis -**lateral curvature of the spine
  - More serious
- kyphosis - posterior curvature.
- Restrictive lung disease !
- Hypoxemia due to :
  - V-Q inequality
    - Atelectasis & compression of dependent areas
    - Airway closure

- Hypercapnia
  - Due to ↑ W.O.B caused by
    - Stiff chest wall
    - respiratory muscles operating at mechanical disadvantage
  - reduced ventilatory response to CO_2

Take home points !

- V-Q matching is required for maintaining normal PO_2 and PCO_2 levels in blood.
- V-Q mismatching is one of the most common causes of hypoxia and rarely hypercapnia in most of the lung diseases.
- Shunt and dead space ventilation are the two ends of the spectrum of V-Q mismatch.