Ventilation-perfusion in health & disease

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Where was the beginning ?

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A THEORETICAL STUDY OF THE COMPOSITION OF THE ALVEOLAR AIR AT ALTITUDE[1]

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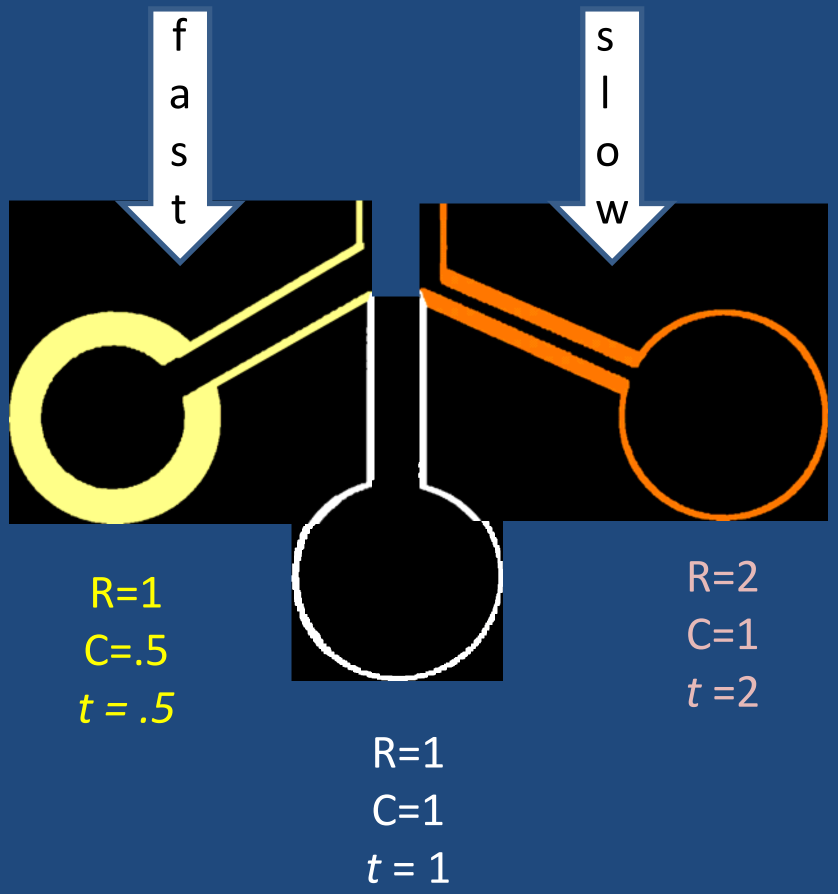
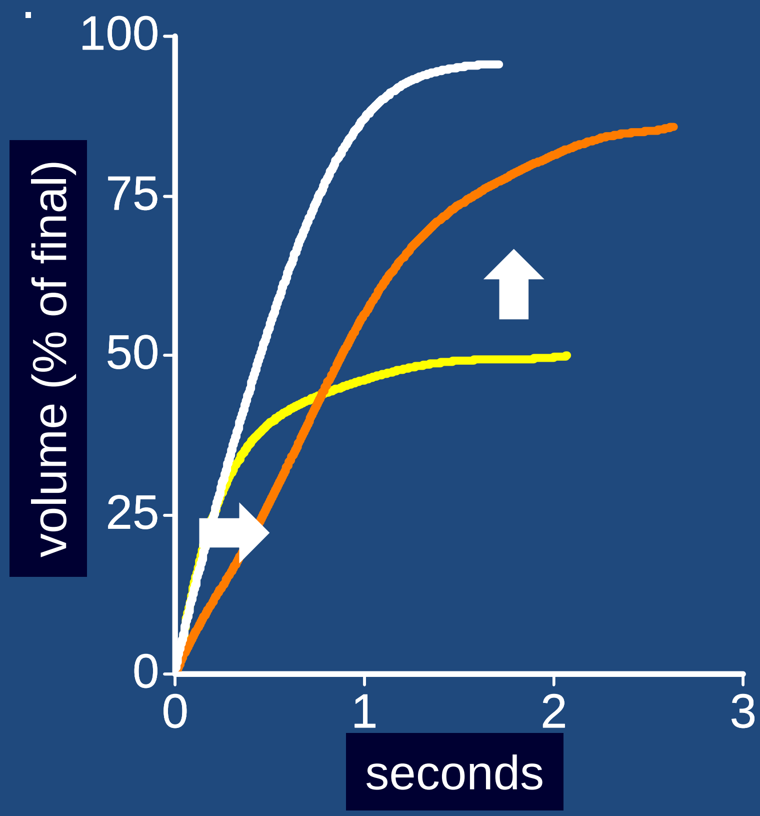
Distribution of ventilation

- Spatial & anatomical variation
- Rate of alveolar filling
- Rate of alveolar emptying

Ventilation distribution- RC *t*





Clinical relevance

- Perfusion is poor & pulsatile at apex
- P_a & P_v proportionately increases from top to bottom
- P_A changes minimally with gravity
- Pressures are max at bottom
  - Pulm edema starts at bottom
  - Redistribution of blood flow to apex – antler's horn

Its not just gravity!

Lung perfusion in zero gravity situations is more uniform but by no means equally distributed

Nunn's Applied respiratory physiology, 6 th edition

Understanding V/Q relationships

- Consider lung as single unit
  - Relationships between $P_A O_2$, $P_A CO_2$, alveolar ventilation & pulmonary blood flow
  - Alveolar gas equation
- Consider lung as multiple units of varying V/Q
  - Clinical consequences in health & disease

Alveolar PO_2 and PCO_2

- Determined by the ratio between ventilation and blood flow: V/Q
- PO_2 and PCO_2 are inversely related through alveolar ventilation
- Increasing V/Q produces higher P_AO_2 and lower P_ACO_2
- Decreasing V/Q produces lower P_AO_2 and higher P_ACO_2

Gas Composition in the Alveolar Space

PiO_2 = (barometric pressure-H_2O vapor pressure) xFiO_2

= (760 – 47) x 0.21 =150 mmHg

Alveolar Gas Equation

$PAO_2 = (PiO_2) – (PaCO_2/R)$

$PaCO_2$ approximates $PACO_2$ due to the rapid diffusion of CO_2

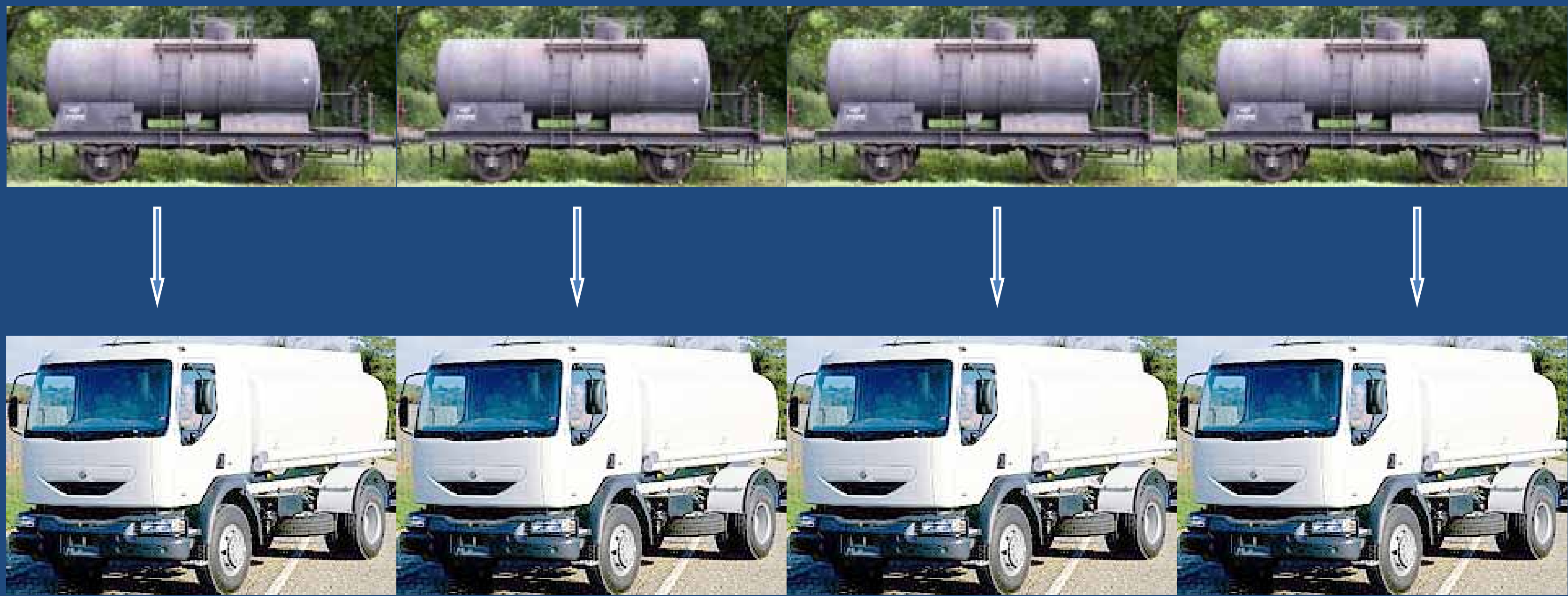
R = Respiratory Quotient ($VCO_2/V0_2$) = 0.8

In a normal individual breathing room air:

$PAO_2 = 150 – 40/0.8 = 100$ mmHg

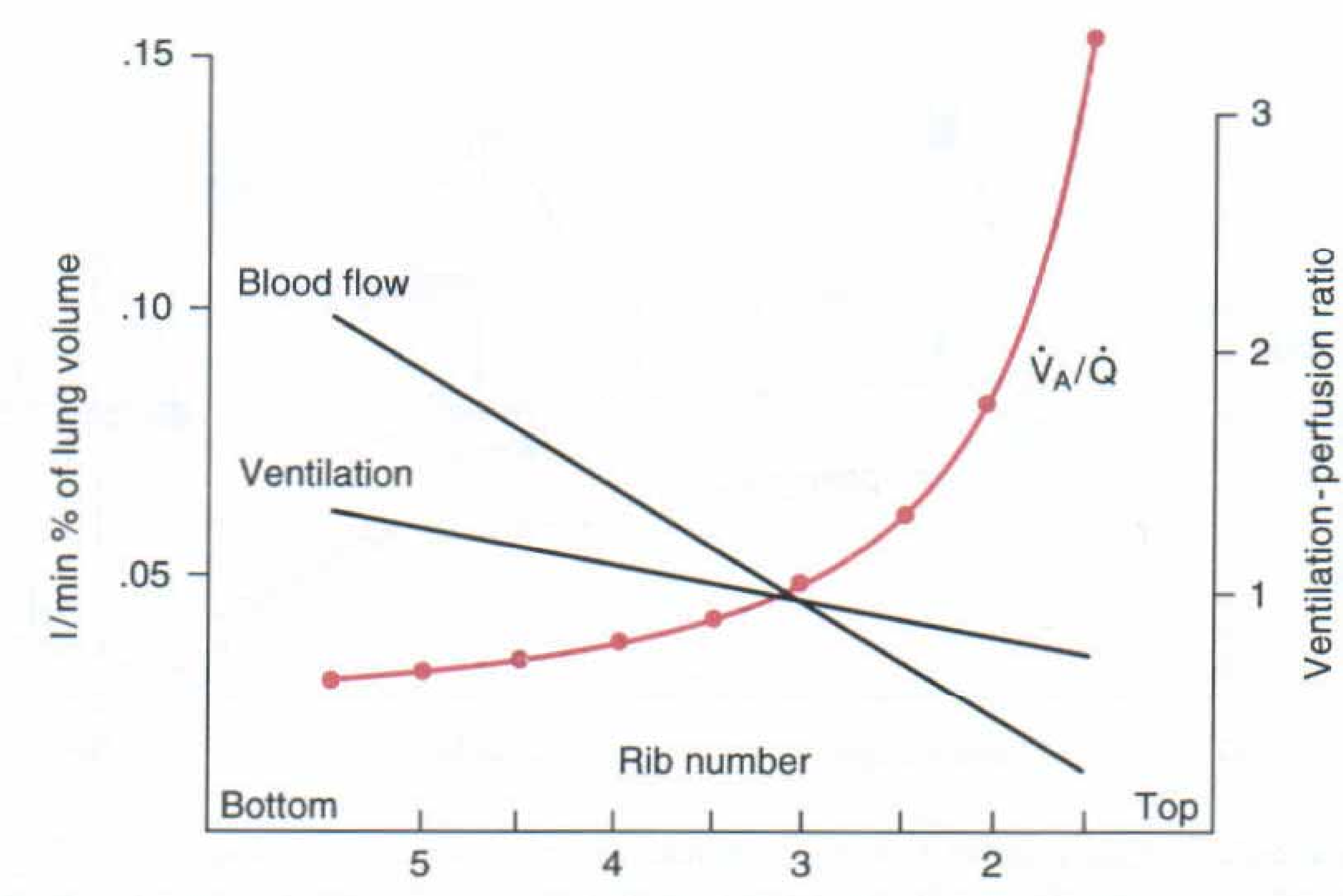
Ventilation/perfusion ratio (V/Q)

V



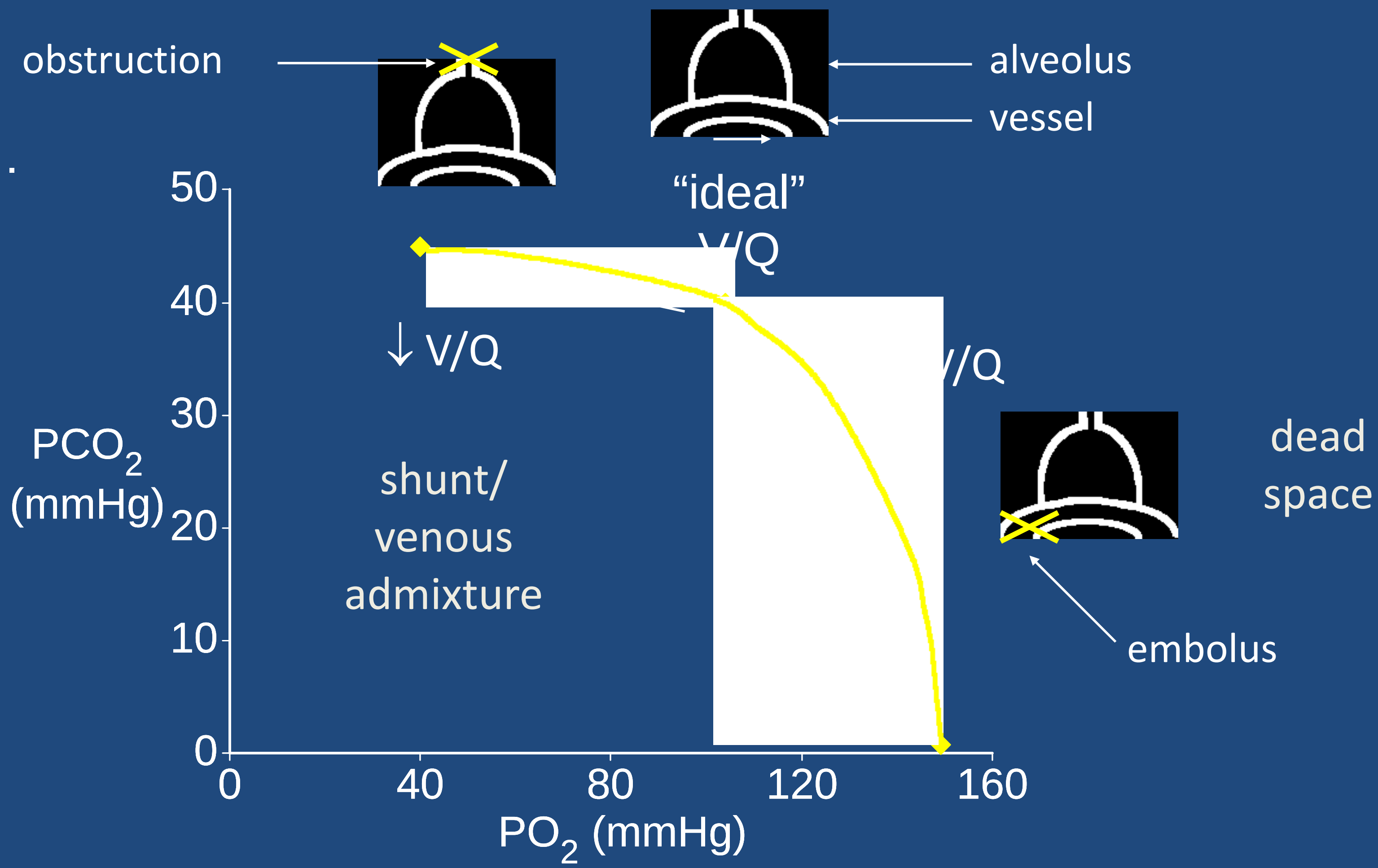
Q

V/Q distribution in health



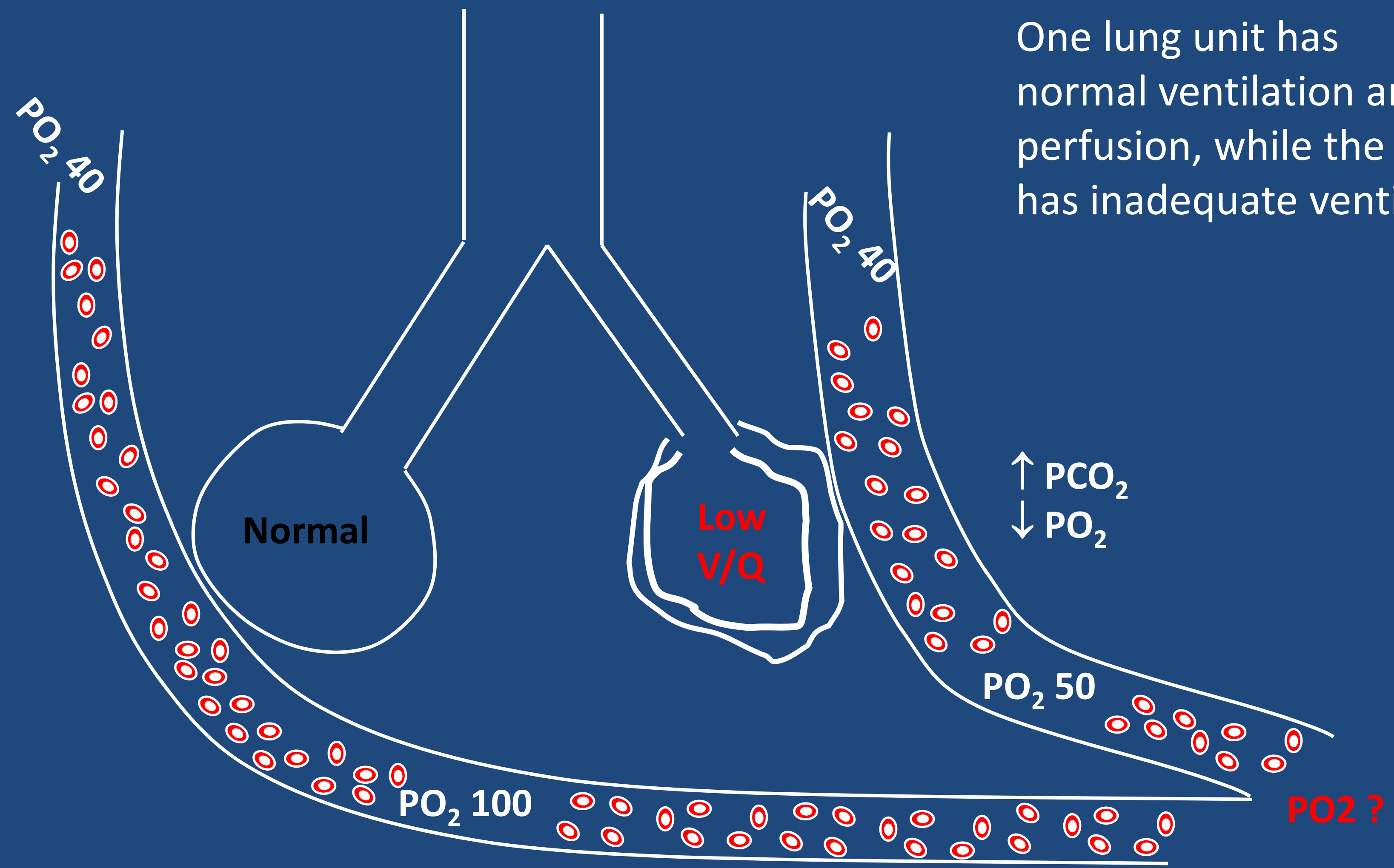
West JB, Respiratory Physiology, The Essentials, 6th ed. 2000, Lippincott, p. 54.

V/Q ratio in disease

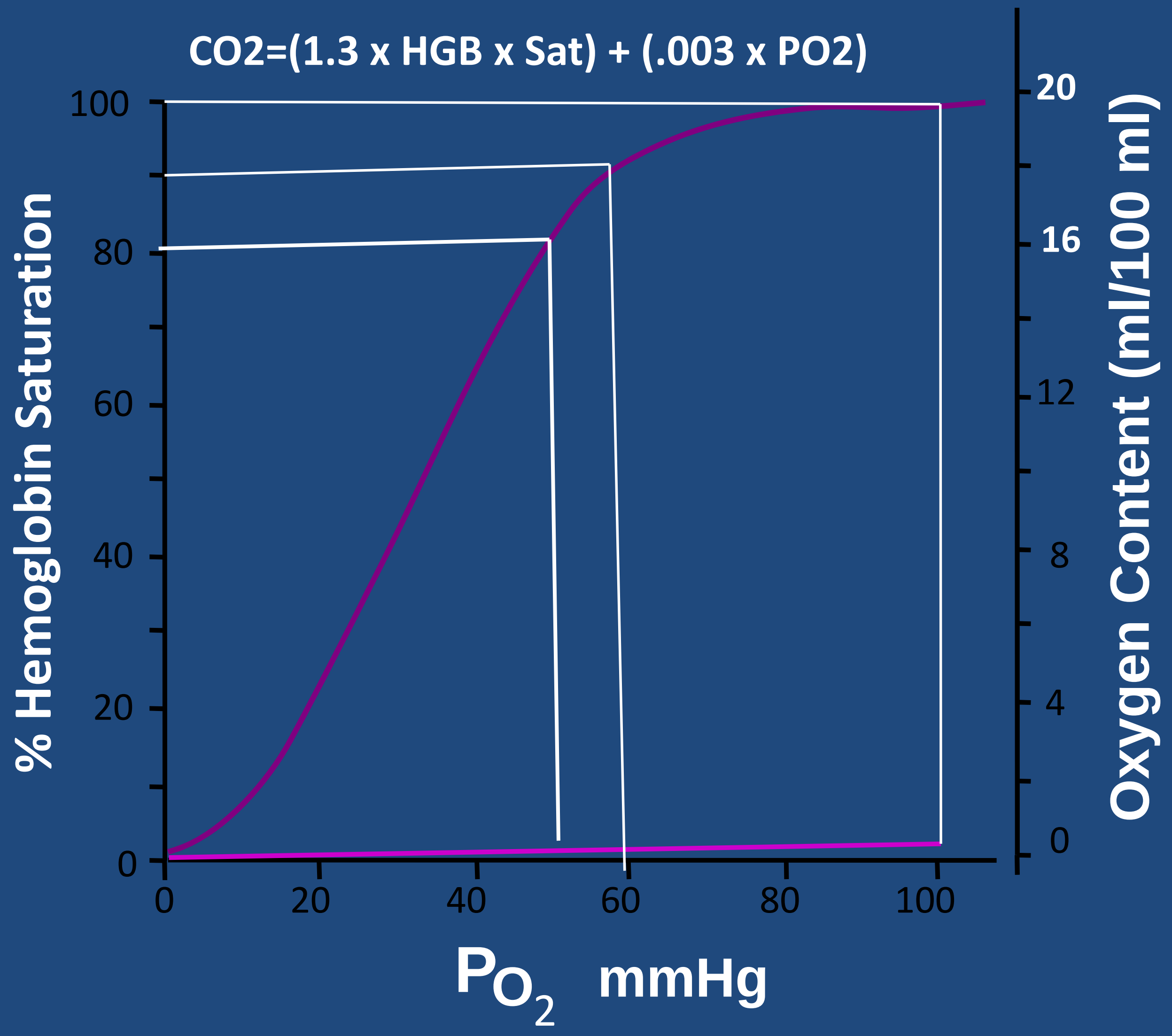


Low V/Q Effect on Oxygenation

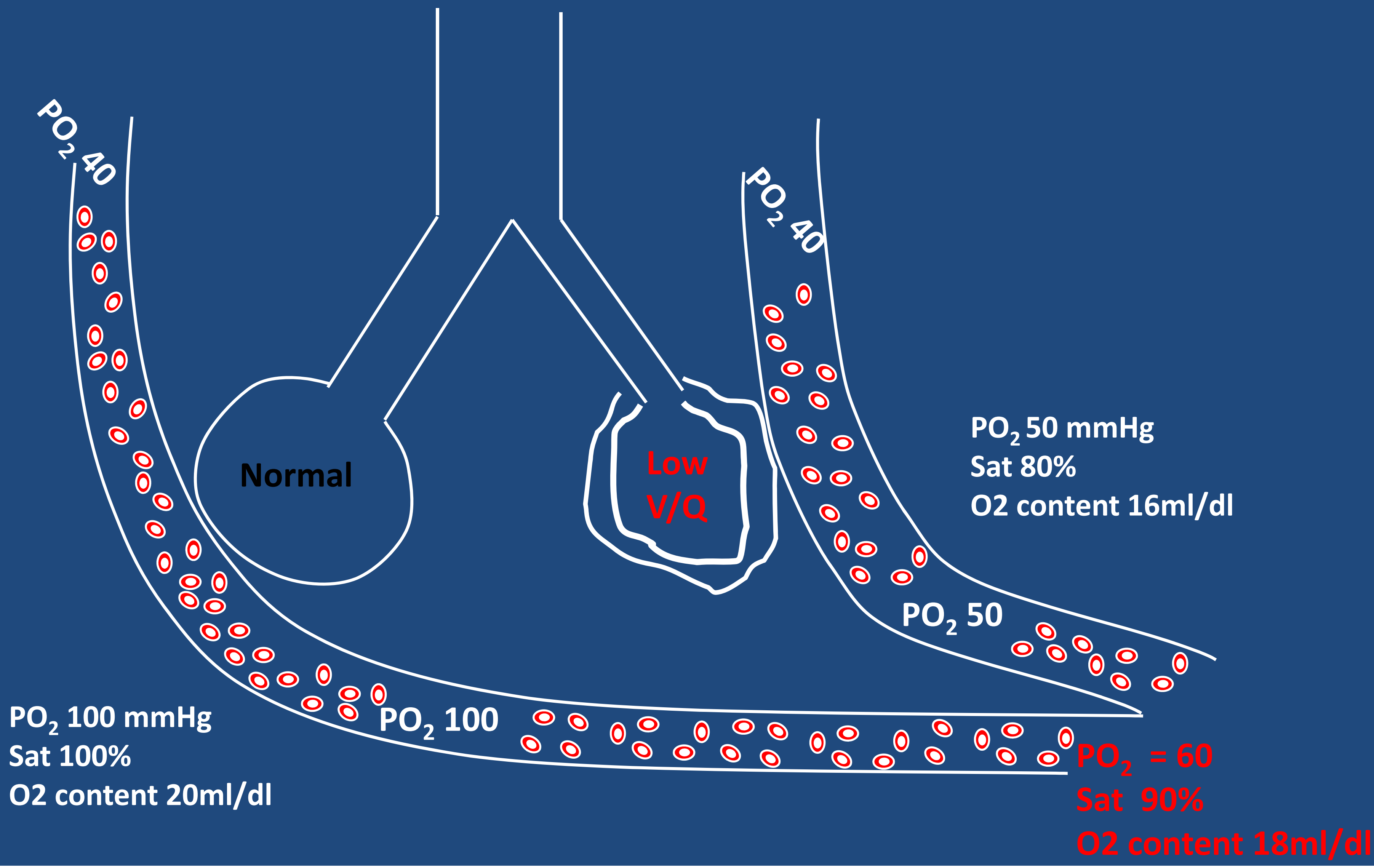
One lung unit has normal ventilation and perfusion, while the has inadequate ventilation



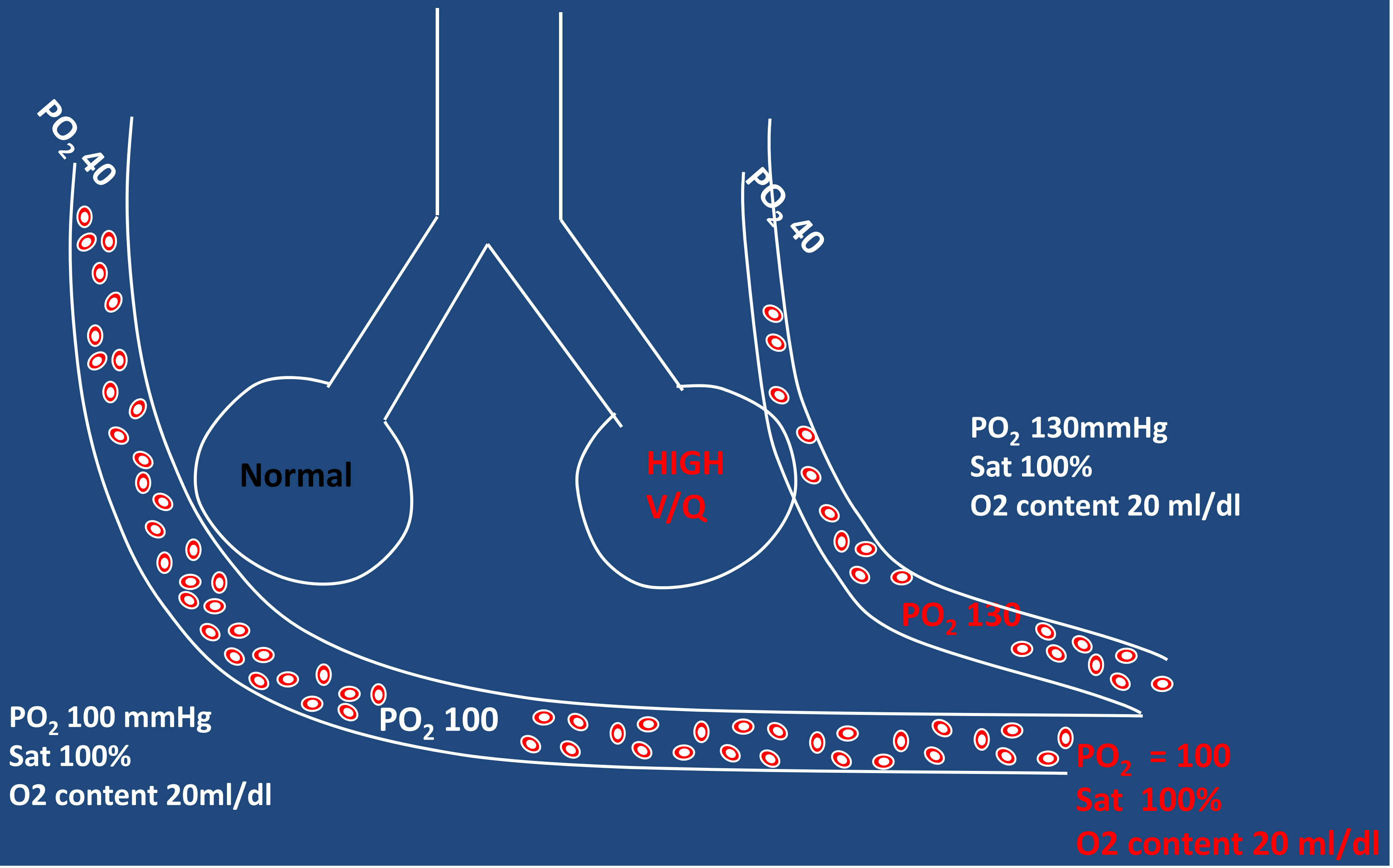
Oxyhemoglobin Dissociation Curve



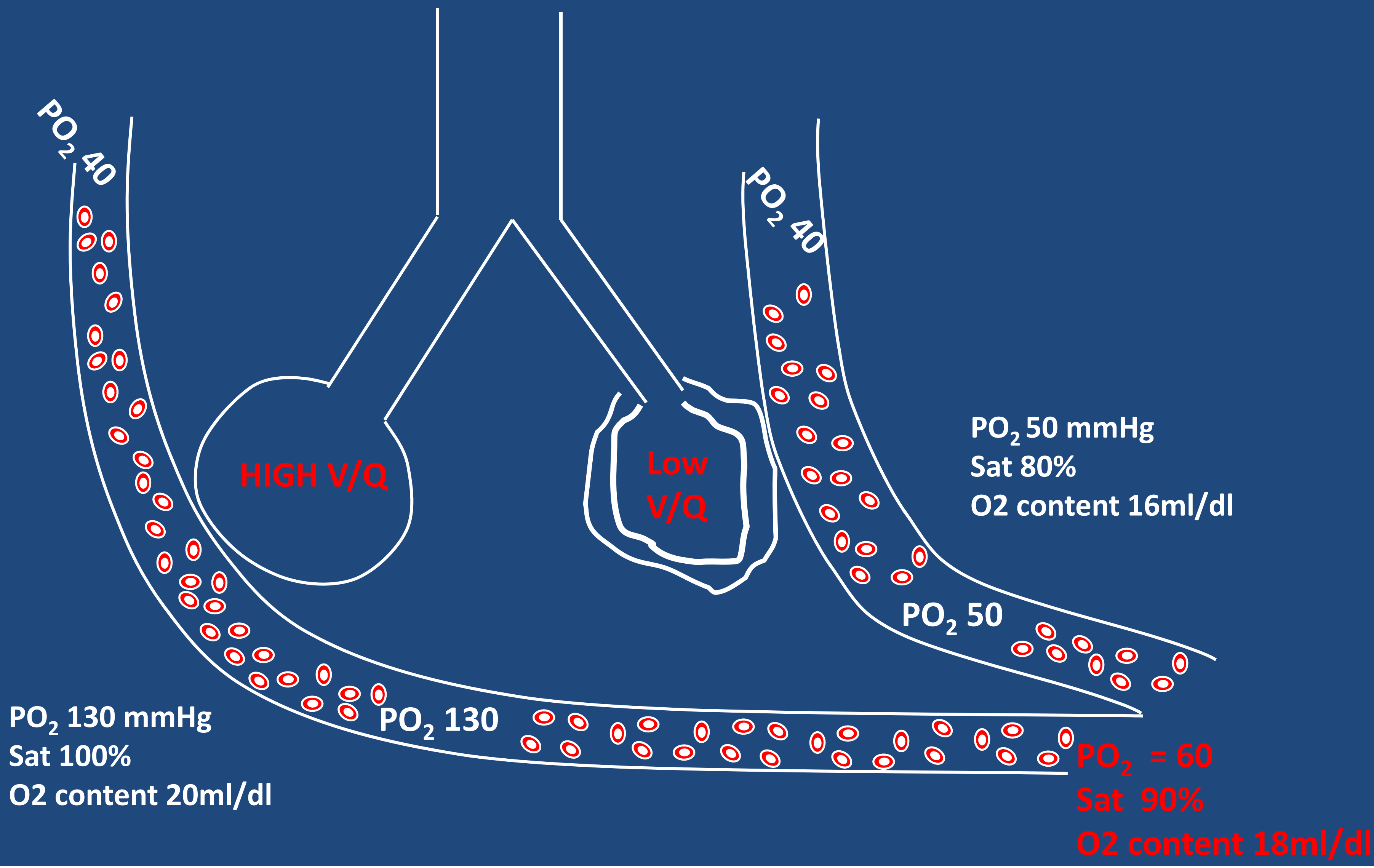
Low V/Q Effect on Oxygenation



HIGH V/Q Effect on Oxygenation

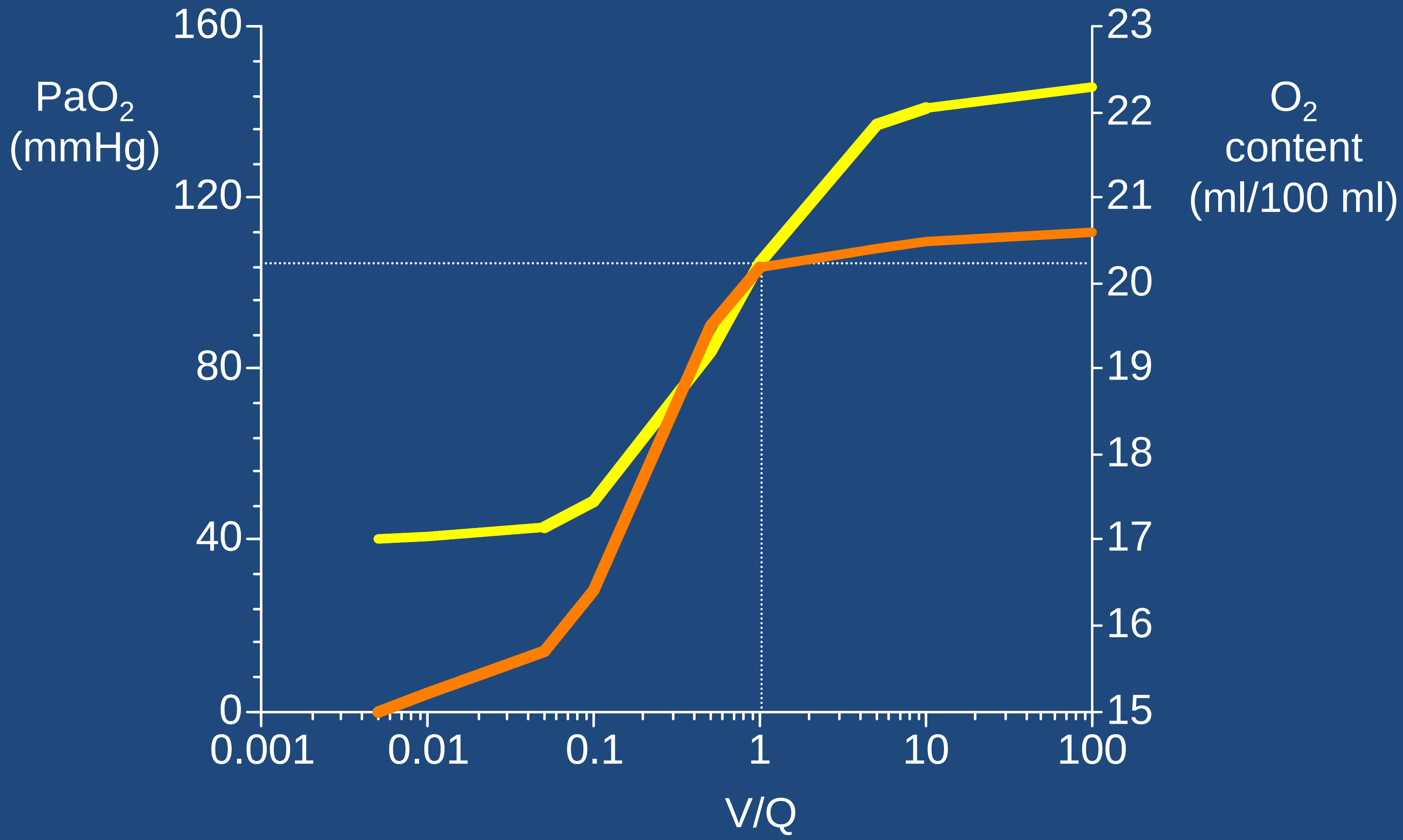


High + low V/Q = ?



PO2 40
HIGH V/Q
Low
V/Q
PO2 40
PO2 50 mmHg
Sat 80%
O2 content 16ml/dl
PO2 50
PO2 130 mmHg
Sat 100%
O2 content 20ml/dl
PO2 130
PO2 = 60
Sat 90%
O2 content 18ml/dl

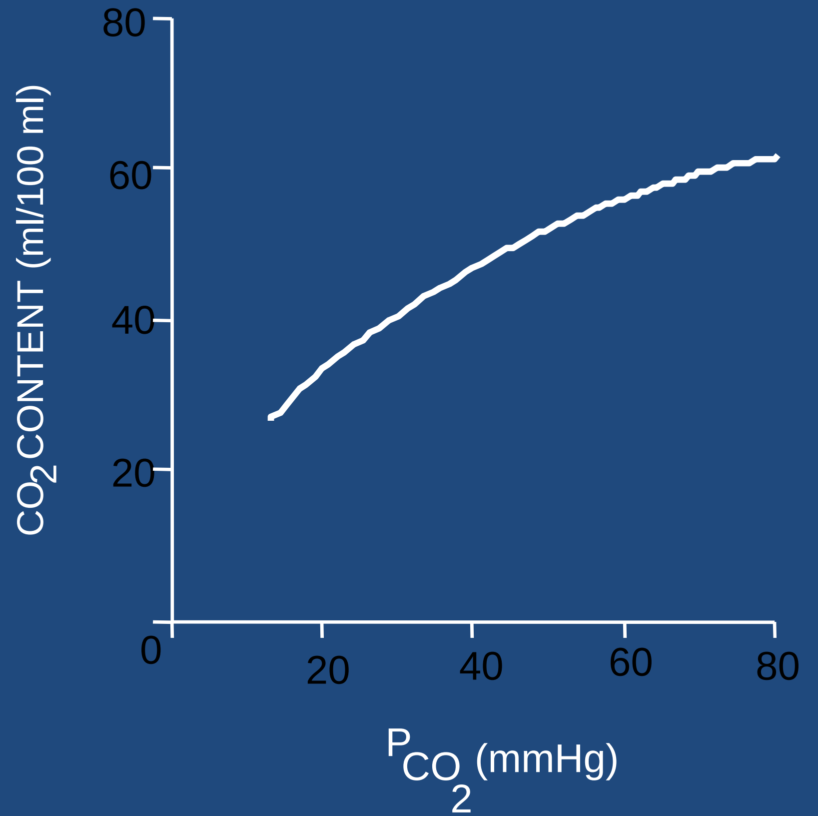
Local ↑ V/Q above ~1 has minimal effect on $[O_2]$



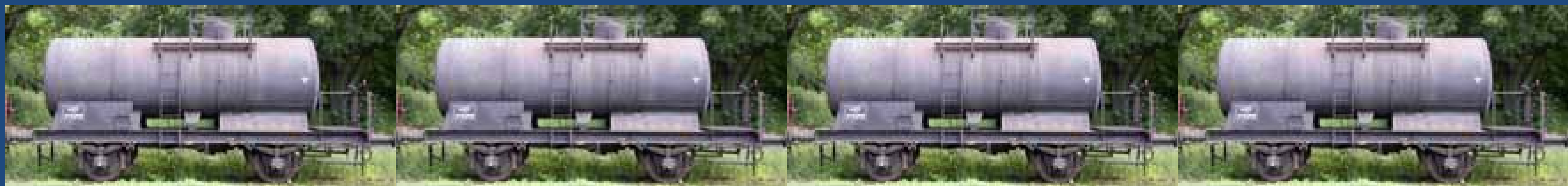
Thus ↑ V/Q in one part of lung can't compensate for ↓ elsewhere

PCO_2 in V/Q Mismatch

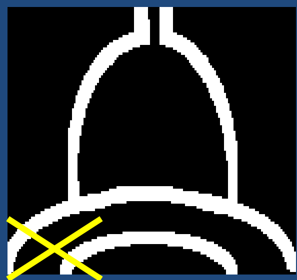
- Increased ventilation can compensate for low V/Q units
  - Shape of CO2 curve
- Total ventilation (VE) must increase for this compensation



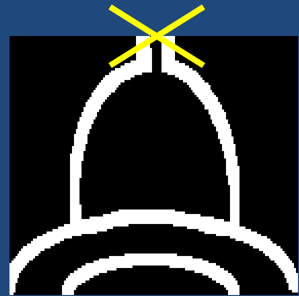
Infinity



↑V/Q
Dead space



Zero



↓V/Q
Shunt
Venous admixture

Ventilation to Perfusion Mismatch

Pure
Shunt
Perfusion with
<u>No</u> Ventilation

Shunt Like
Units

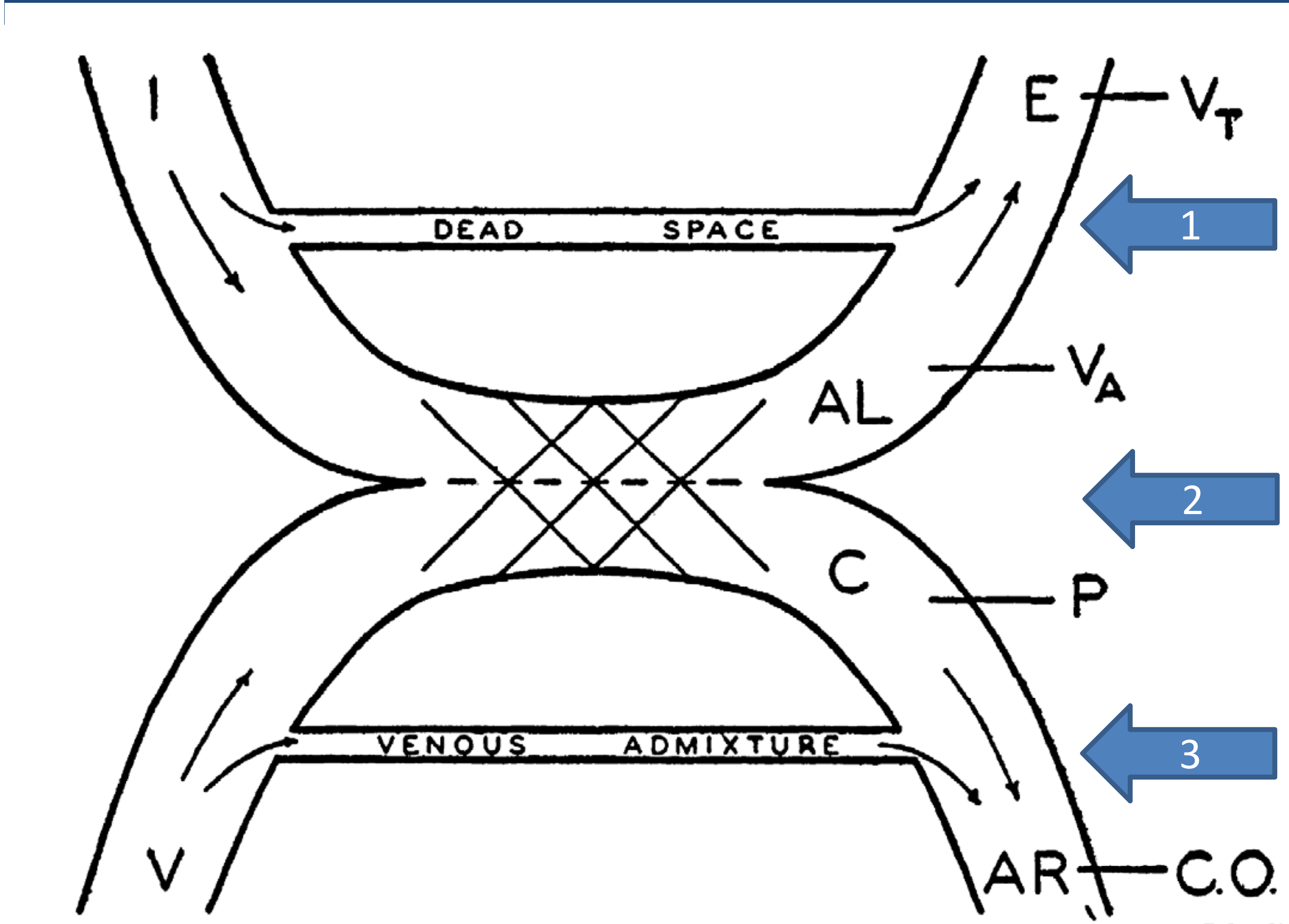
Dead Space
Like Units

Pure
Dead Space
Ventilation with
<u>No</u> Perfusion

shunt

Dead space

3- compartment model



'Ideal' Alveolar Air and the Analysis of Ventilation–Perfusion Relationships in the Lung R. L. RILEY 1949

Dead space

- Defined as wasted ventilation

- Classification
  - Anatomical (1 ml / pound of ideal body wt)
  - Alveolar
  - Physiological
    - Anatomical + alveolar

Physiological Dead Space by Bohr's Method (for CO2)

$V_T \times F_E = V_A \times F_A$

$V_T = V_A + V_D$

$V_A = V_T - V_D$

$V_T \times F_E = (V_T - V_D) \times F_A$

$$\frac{V_D}{V_T} = \frac{F_A - F_E}{F_A}$$

$$\frac{V_D}{V_T} = \frac{P_{ACO2} - P_{ECO2}}{P_{ACO2}}$$ (Bohr Equation), so…

$$\mathbf{\frac{V_D}{V_T} = \frac{P_{aCO2} - P_{ECO2}}{P_{aCO2}}}$$

Physiologic Dead Space & V/Q Mismatch

Hyperinflation

- Airway Obstruction
- Dynamic Hyperinflation
- Tidal Volume
- PEEP

Low Perfusion

- Low Cardiac Output
- Pulmonary Vascular Injury
- Extravascular Compression

No Perfusion

- Pulmonary Embolus
- Vascular Obliteration
- Emphysema

Shunt equation

$$Qt \times CaO_2 = [(Qt - Qs) \times Cc'O_2] + [Qs \times C\bar{v}O_2]$$

$$\frac{Qs}{Qt} = \frac{Cc'O_2 - CaO_2}{Cc'O_2 - C\bar{v}O_2}$$

Causes of Shunt

- Physiologic shunts
  - Bronchial veins, pleural veins
- Pathologic shunts
  - Intracardiac
  - Intrapulmonary
    - Vascular malformations
    - Unventilated or collapsed alveoli

Normal shunt fraction

- ~ 5 %

- Due to
  - Physiological shunt
  - Gravity related V/Q mismatch

- Contributes to A-a D O_2
  - 10 – 15 mm hg

A a DO_2

- PAO2 (alveolar gas equation)- arterial PaO2
- In a totally efficient lung unit with matched V/Q, alveolar and capillary PO2 would be equal
- Admixture of venous blood or V/Q scatter from low V/Q lung units will increase A a DO_2
- Increases ~ 3 mm hg every decade after 30 yrs of age

Shunt VS. V/Q scatter

- Calculated shunt fraction or A a DO_2
  - Does not differentiate

- Response of pa O_2 to increasing Fi O_2

- V/Q scatter
  - Pa O_2 increase with small increase in FiO_2(.21-.35)

- Pure shunt
  - Not much response

MIGET

- 50 parallel gas exchange units characterized by V_A-Q ratio.
- Fit predicted P_E & P_a to experimental data.
- Output: Ventilation and perfusion distributions.

Compensation of V/Q inequality ?

- ↑V/Q → local hypocapnia → ↑pH → local bronchoconstriction (↓V/Q)

- ↓V/Q → ↑CO_2 → ↑ ventilation
  - improves CO_2 > O_2 (dissociation curves)

- ↓V/Q → hypoxic pulmonary vasoconstriction

Points to remember

- Ventilation and Perfusion must be matched at the alveolar capillary level
- Most important cause of hypoxemia in most respiratory diseases
- V/Q ratios close to 1.0 result in alveolar PO2 close to 100 mmHg and PCO2 close to 40 mmHg
- V/Q greater than 1.0 increase PO2 and Decrease PCO2. V/Q less than 1.0 decrease PO2 and Increase PCO2
- Shunt and Dead Space are Extremes of V/Q mismatching.
- A-a Gradient of 10-15 Results from gravitational effects on V/Q and Physiologic Shunt