Lecture 42
Ventilation – Perfusion Matching

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Ventilation-Perfusion Matching

Basic Concept

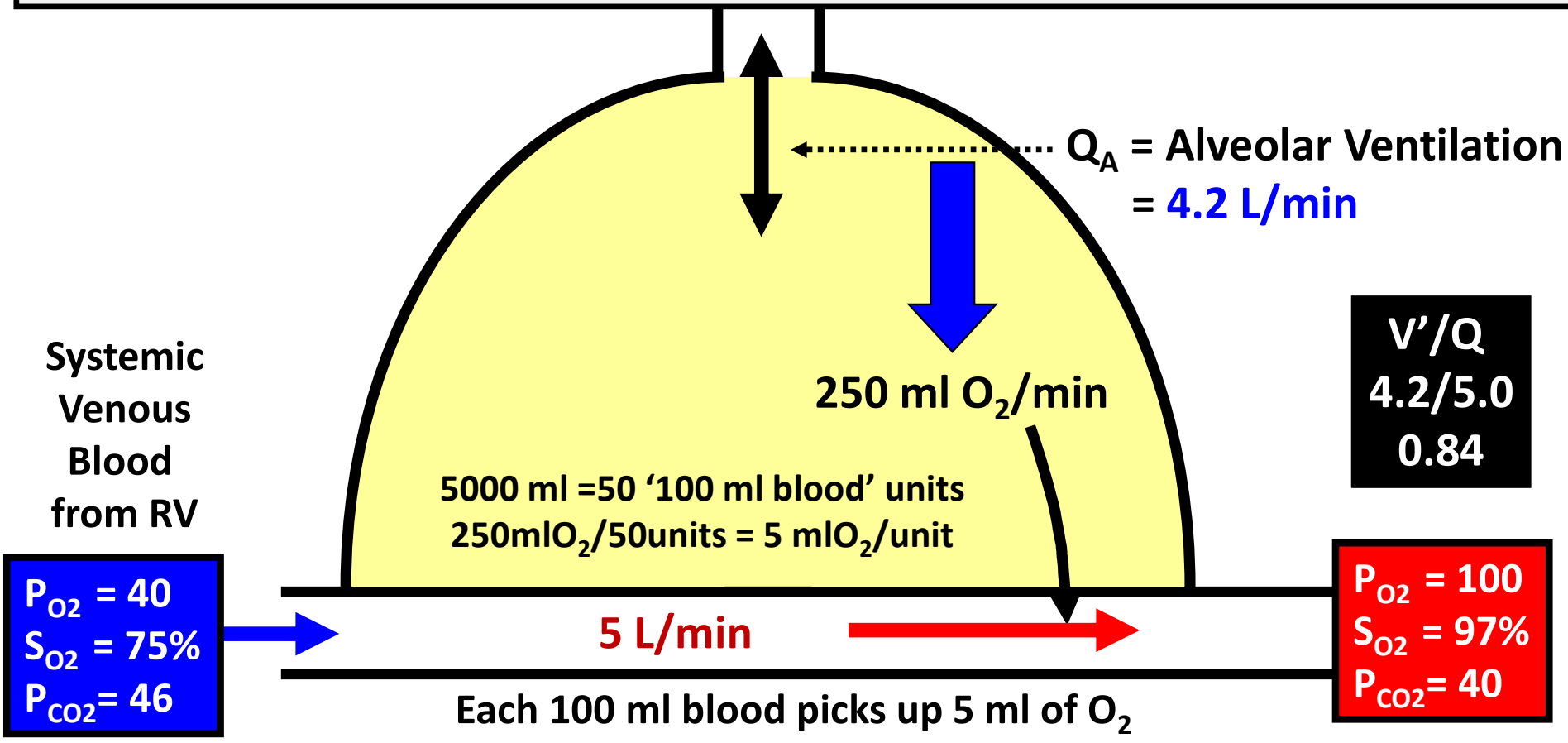
It is neither ventilation nor perfusion alone that determines arterial blood gases. It is the ratio of ventilation to perfusion that is the determinant!

Ventilation/Perfusion = V'/Q ratio

Case A: Ventilation-Perfusion Concept

Assume that an alveolar ventilation of 4.2 L/min will deliver 250 ml O_2/min to capillary blood

- If blood flow is 5 L/min then each of the (50) 100 ml of blood must pick up 5 ml of O_2
- This results in a proper "arterialization" of blood exiting the lung.

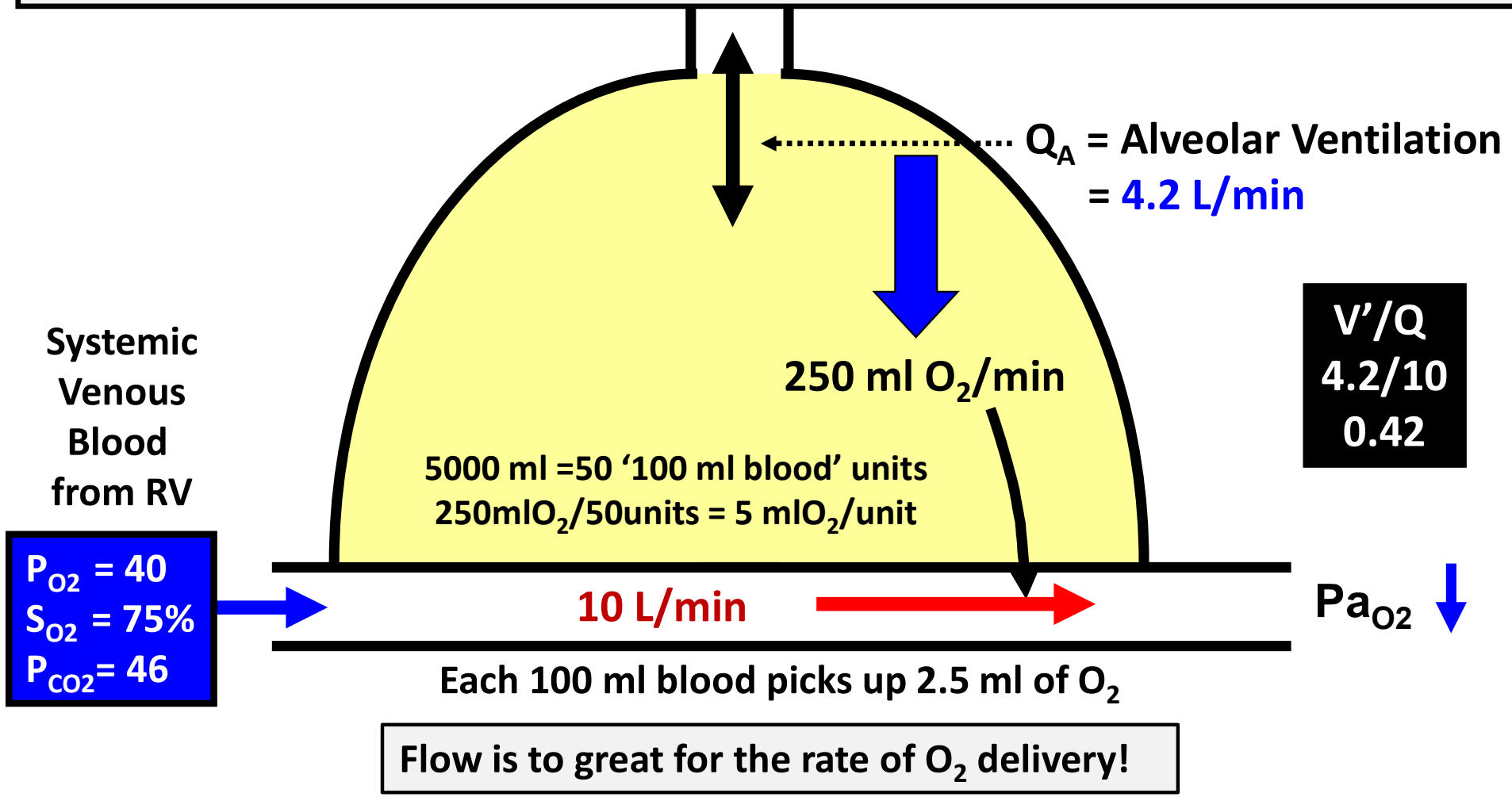


Flow is just right for the rate of O_2 delivery!

Case B: Ventilation-Perfusion Concept

Now assume that an alveolar ventilation remains unchanged at 4.2 L/min BUT that blood flow increases to 10 L/min

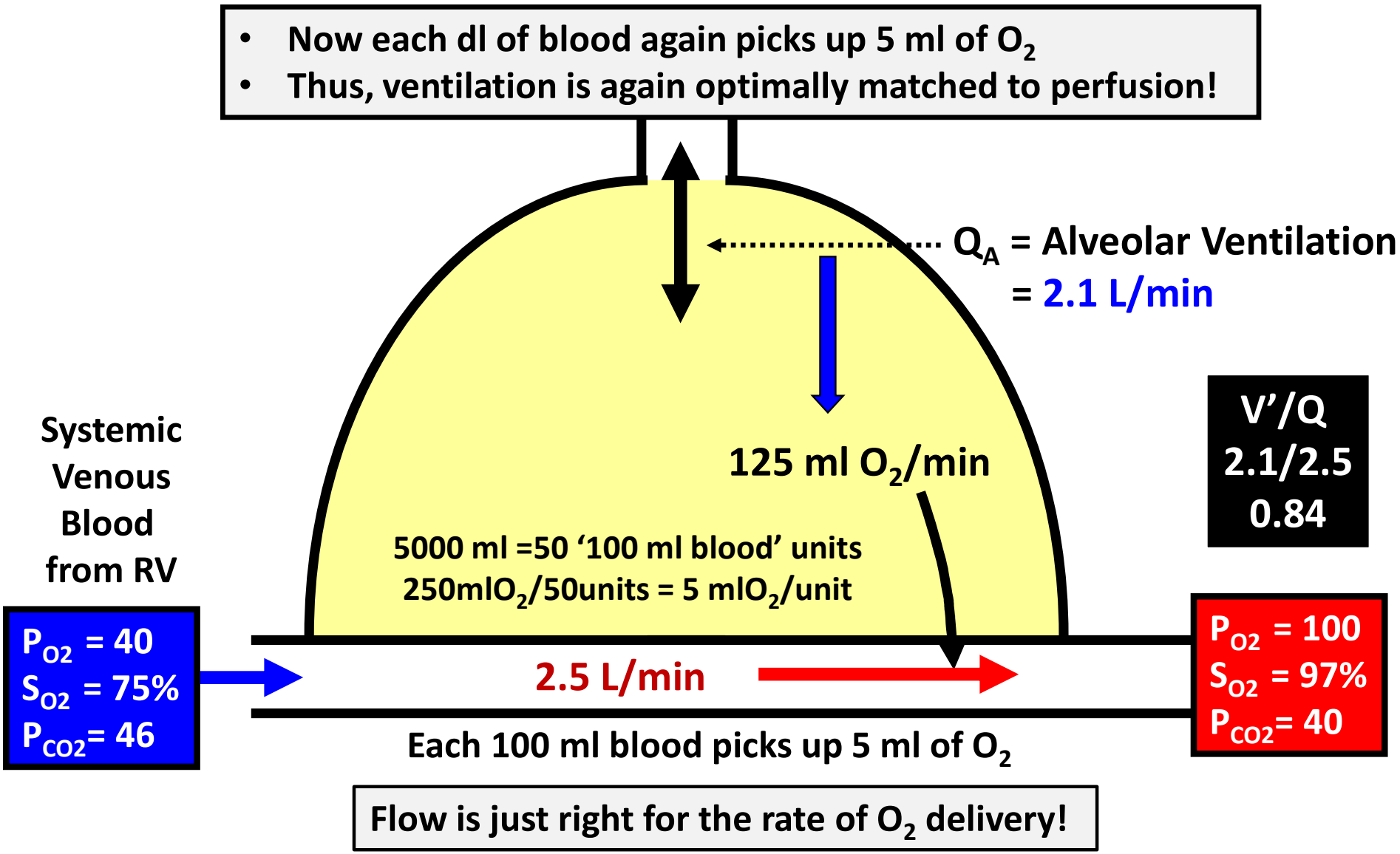
- **Now, 100 "100 ml units" pass by each min - So – each dl picks up only 2.5 ml of O_2**
- **Since this is ½ that needed, blood exiting the lung will have its PO_2 much reduced!**



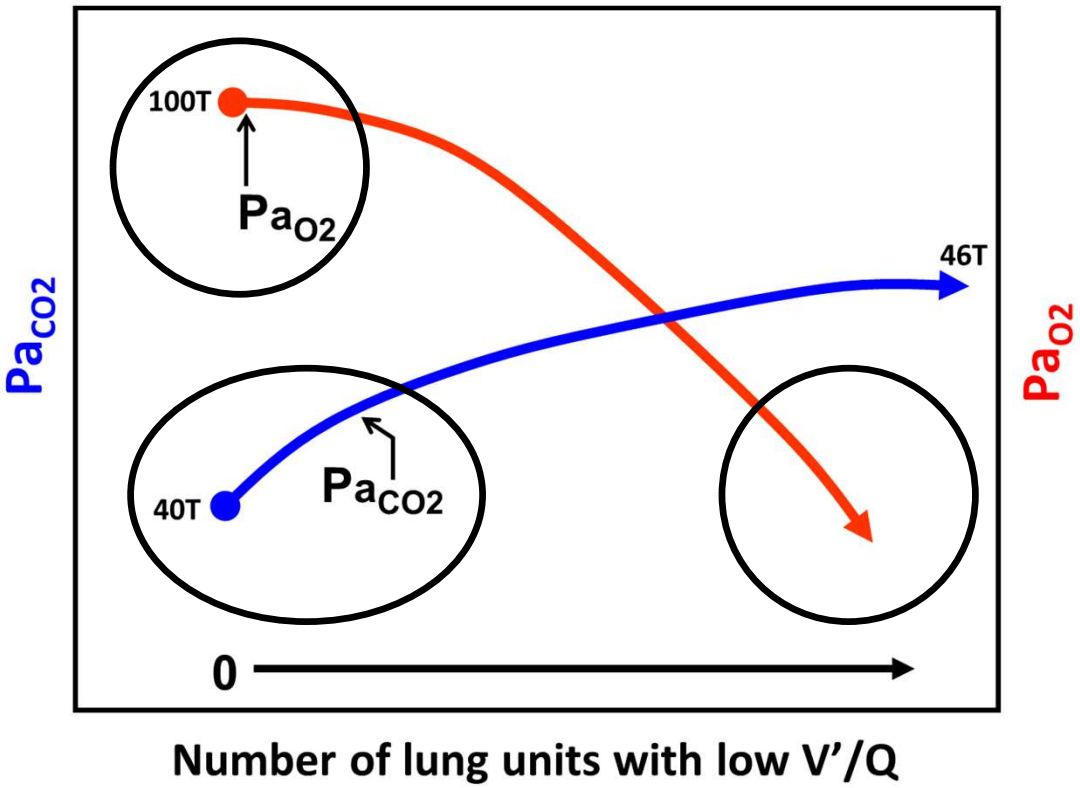
Flow is to great for the rate of O_2 delivery!

Case C: Ventilation-Perfusion Concept

Now suppose ventilation and perfusion become 1/2 of what they originally were

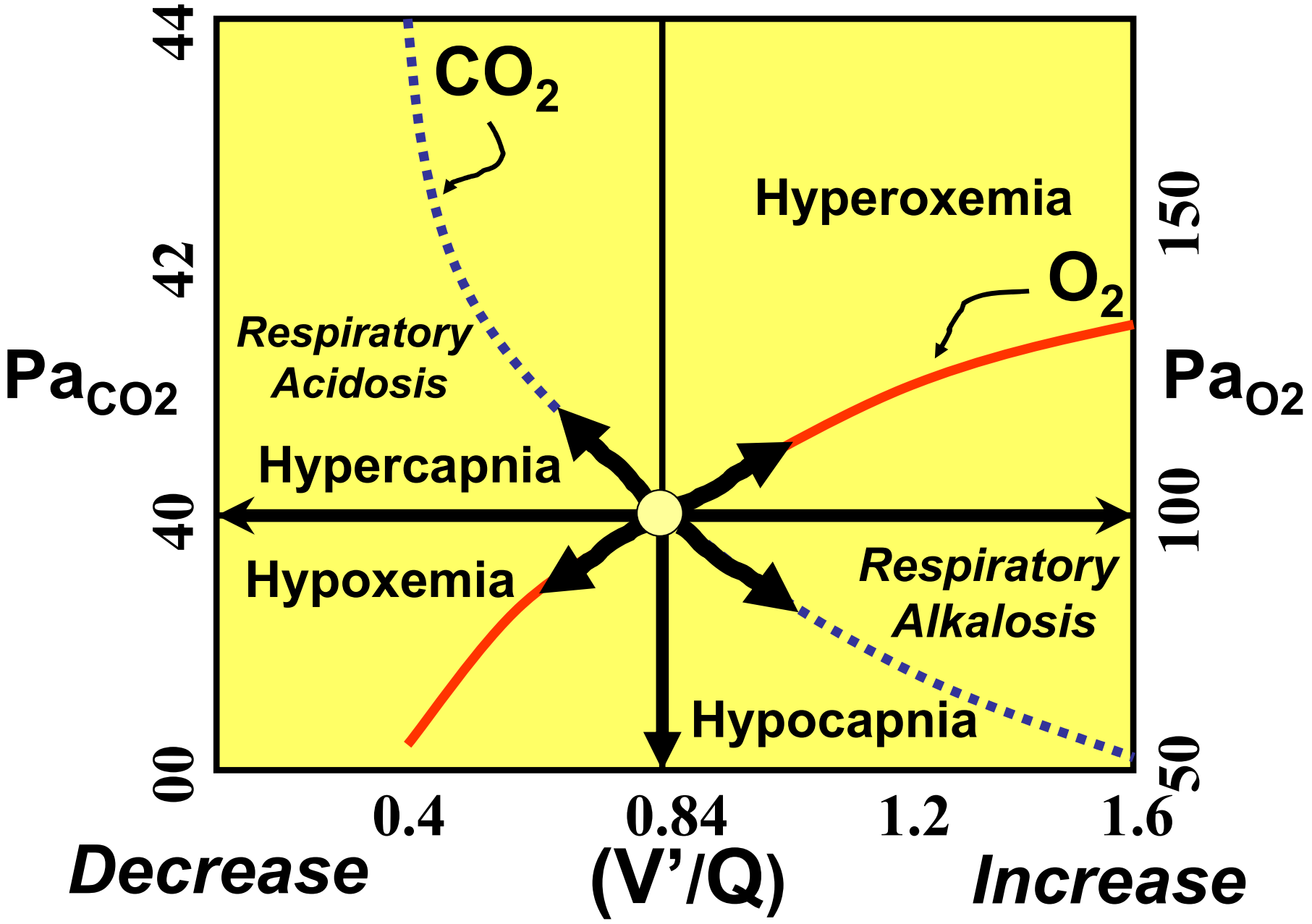


Another View of V'/Q Mismatching

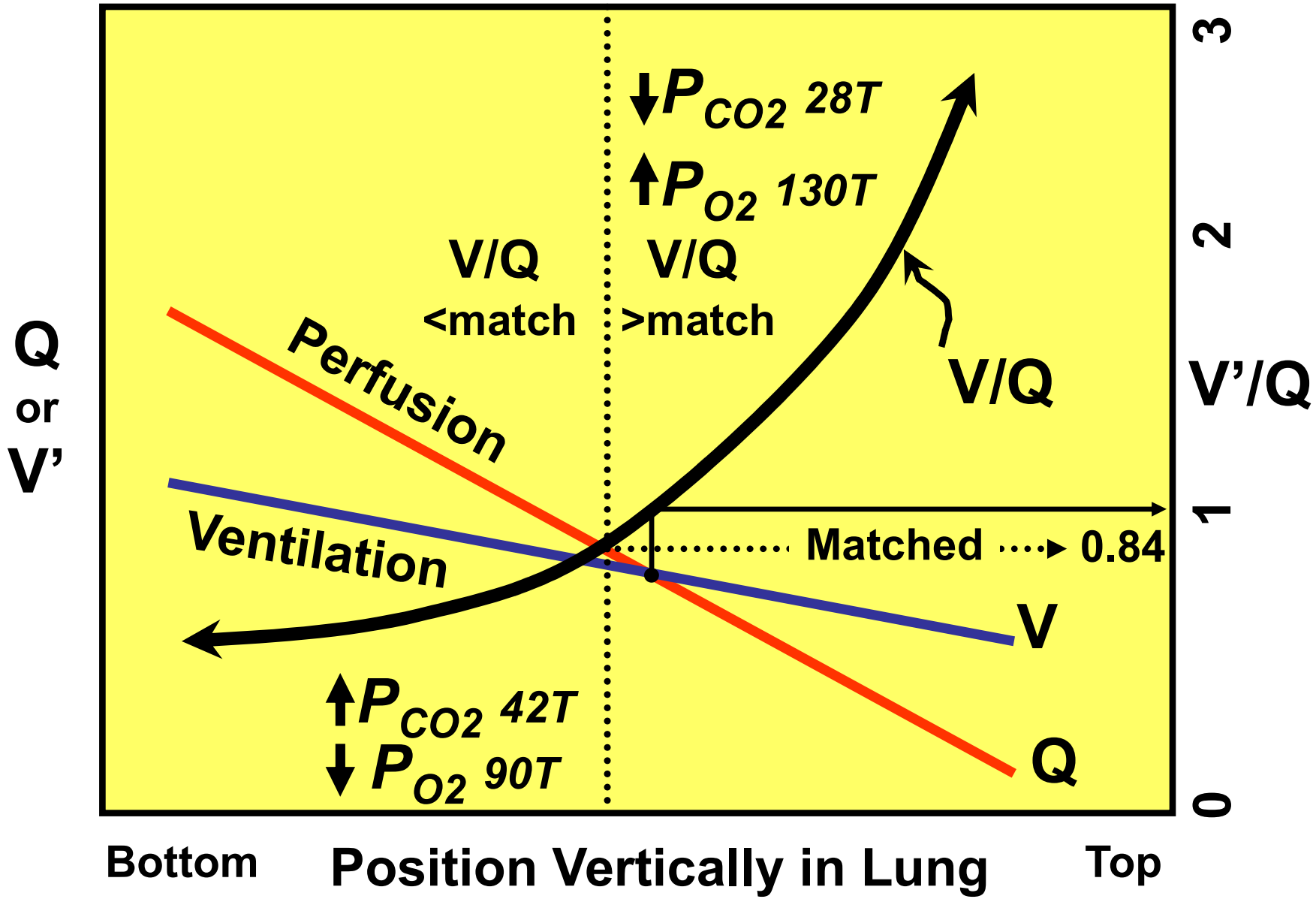


- If all lung units were V'/Q matched, then optimum lung function; but this is not the case
- Some units have V'/Q less than a match and other have V'/Q greater than a match
- The combined blood coming from all units determines the arterial values of O_2 and CO_2
- As the number of low V'/Q lung units increases the blood exiting the lung will have increasing values of CO_2 and decreasing values of O_2

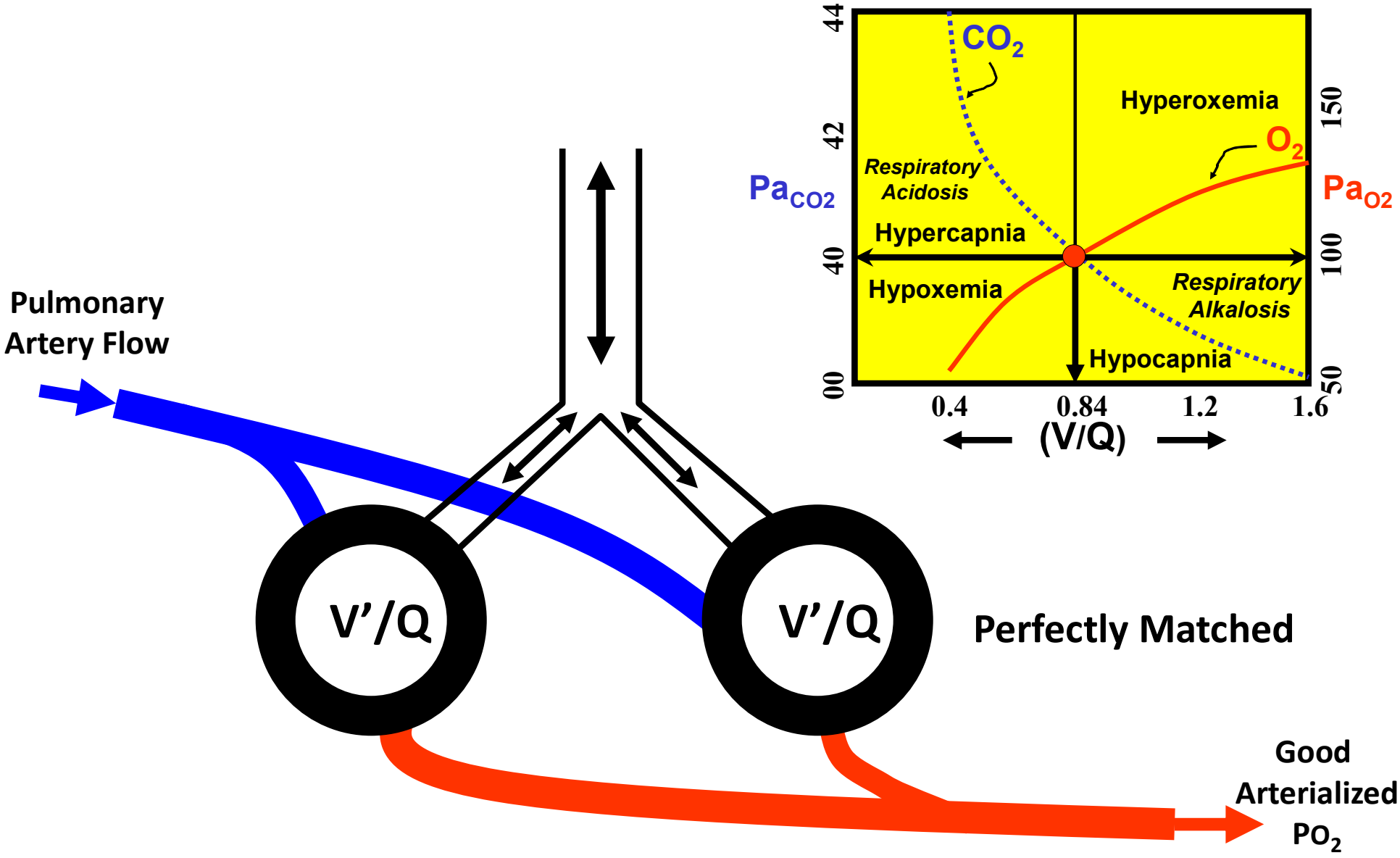
Effects of Changes in V'/Q



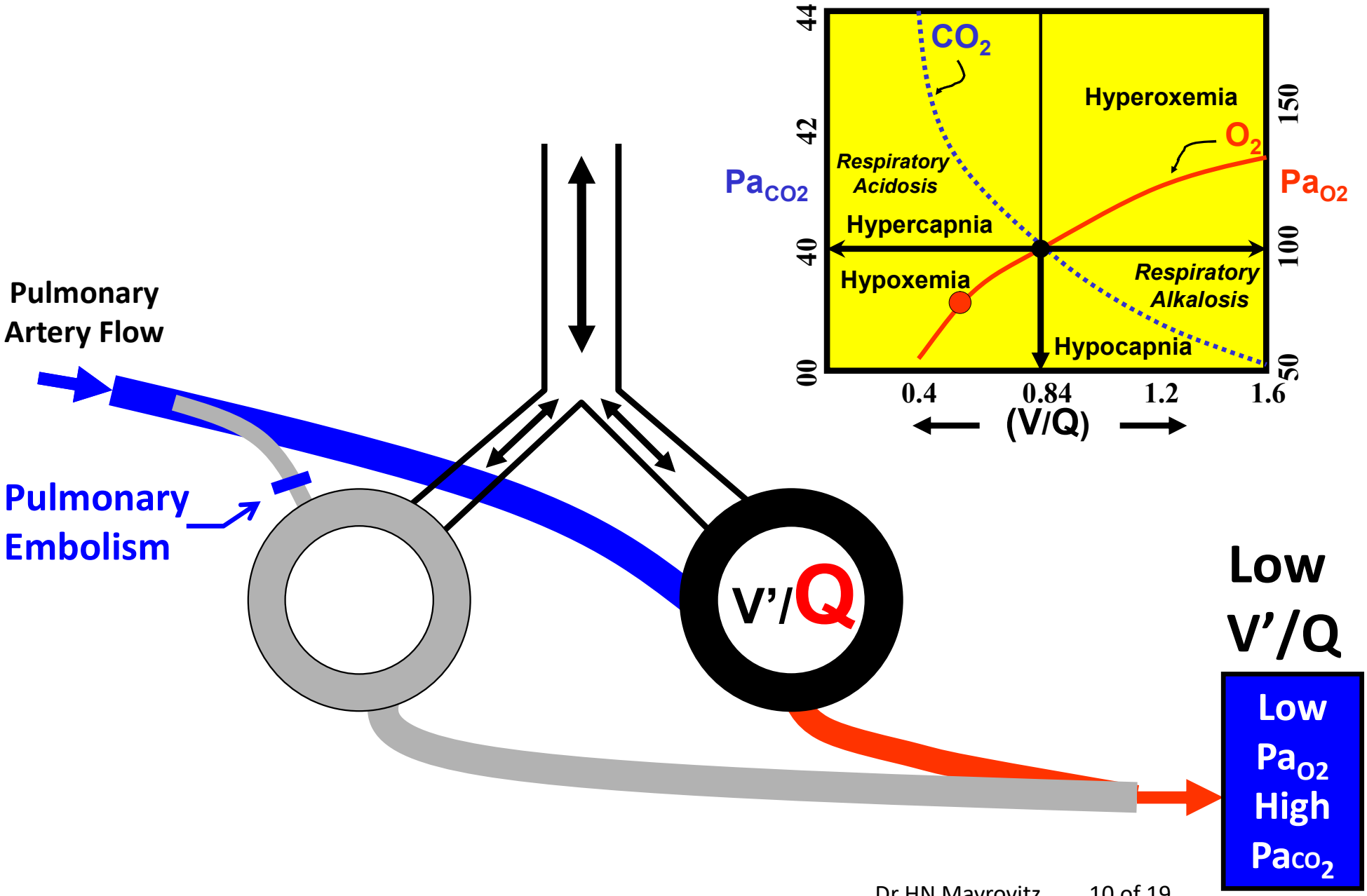
Regional V'/Q Variations



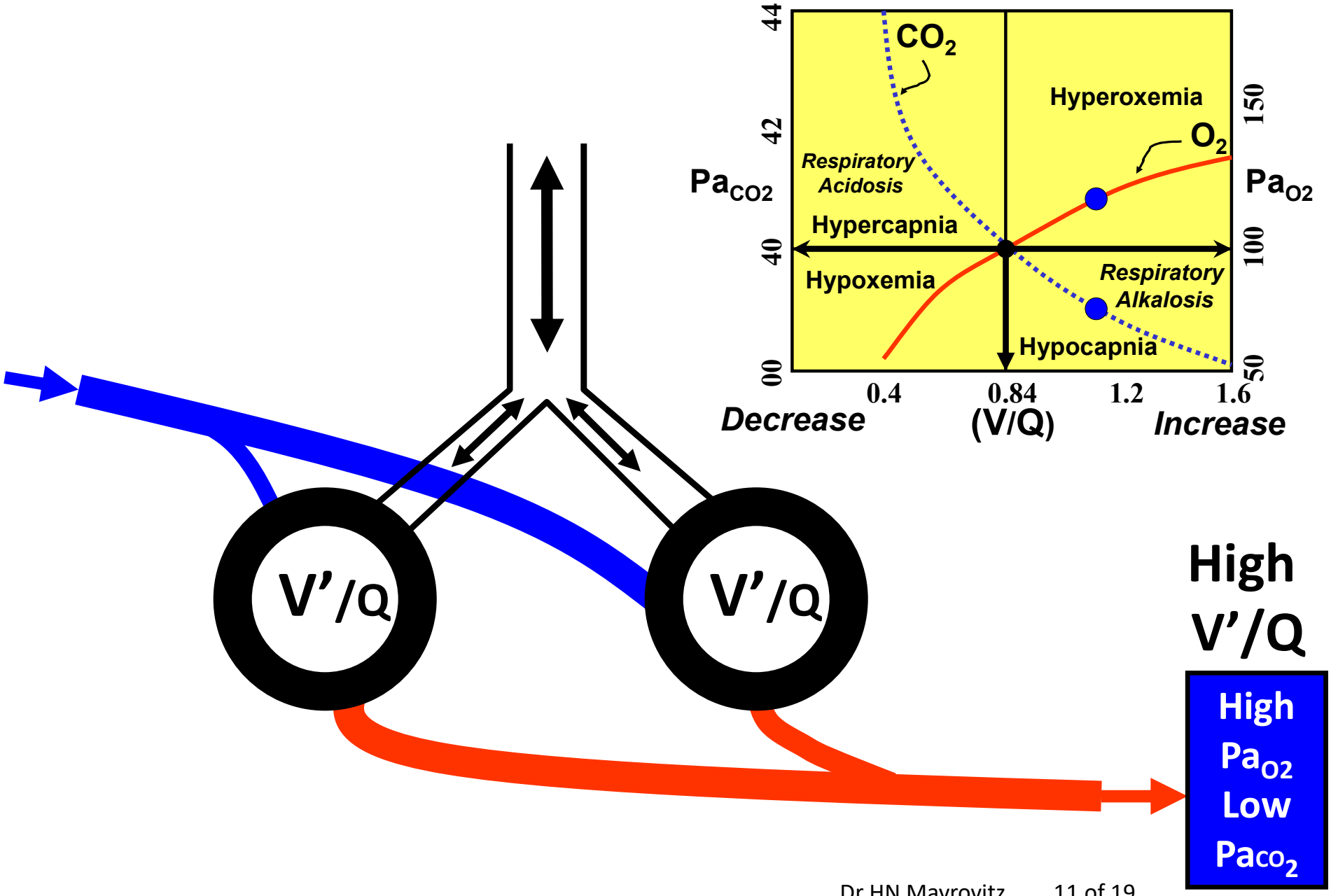
Clinical Correlation: Ventilation matched to Perfusion



Clinical Correlation: Pulmonary Embolism

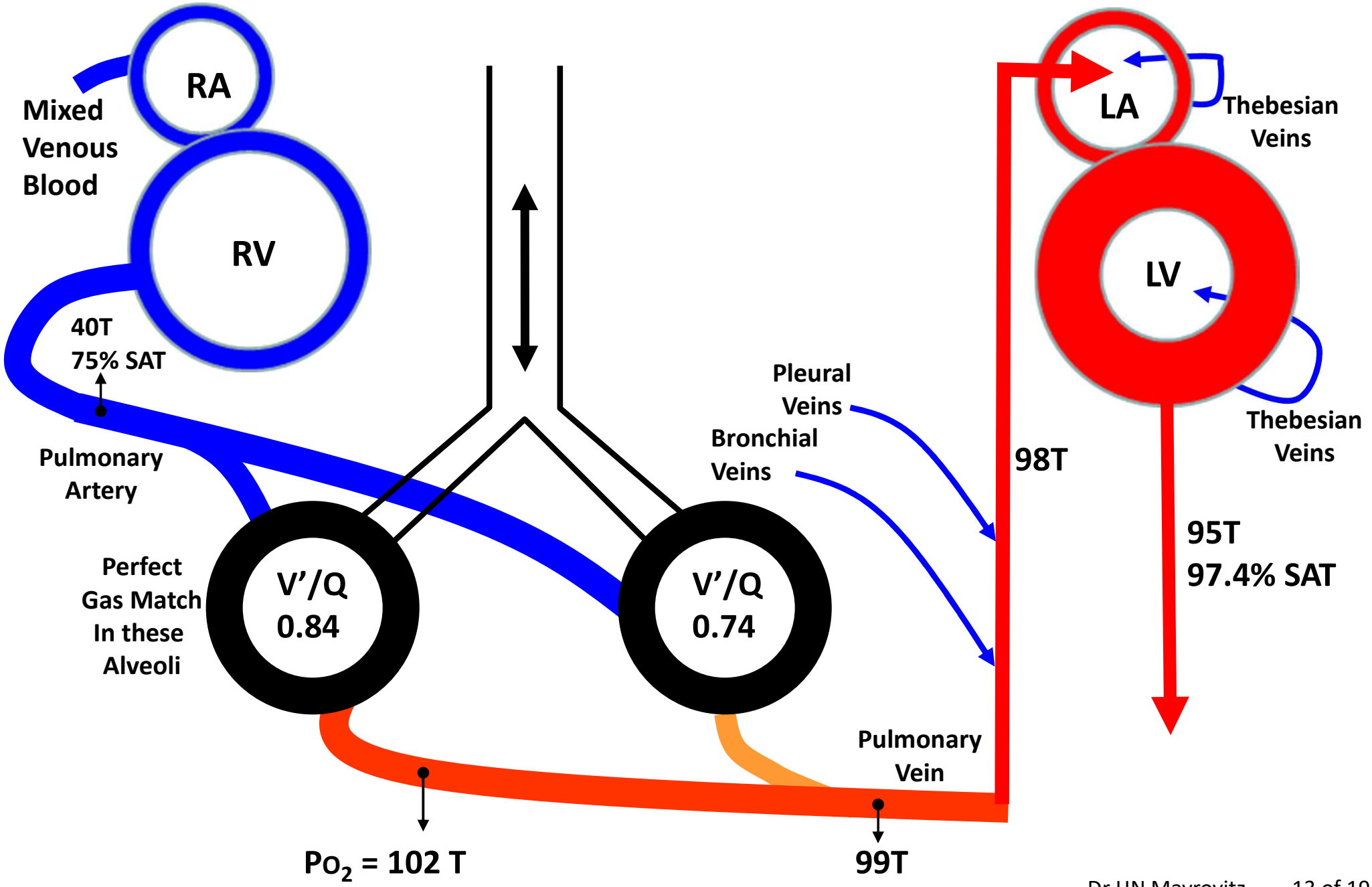


Clinical Correlation: Hyperventilation

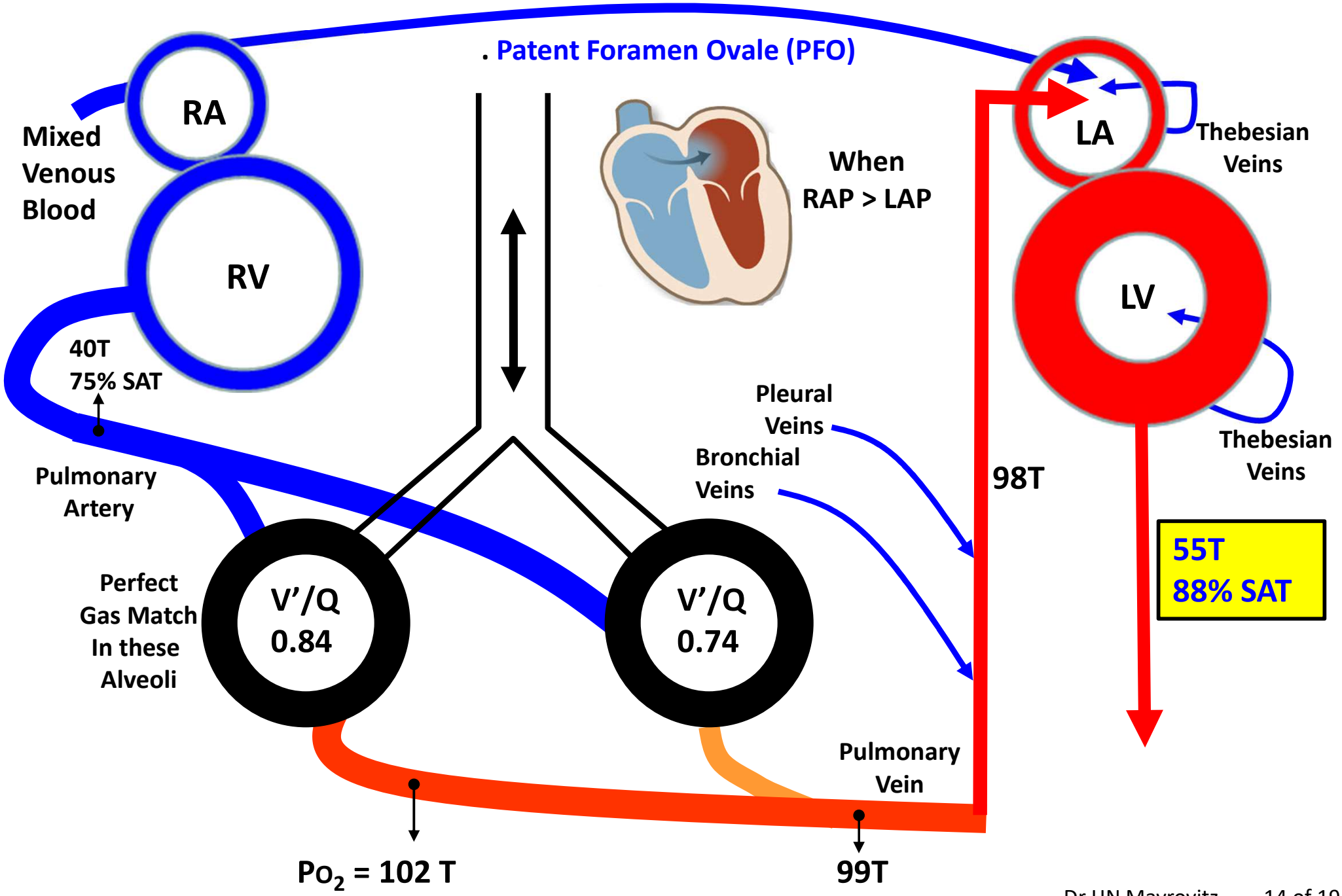


Shunts and Hypoxic States

Shunts: Mixing Low O_2 with Higher O_2: Normal



Shunts: Mixing Low O_2 with Higher O_2: Not Normal



Shunts: Quantitative Aspects – Shunt Equation

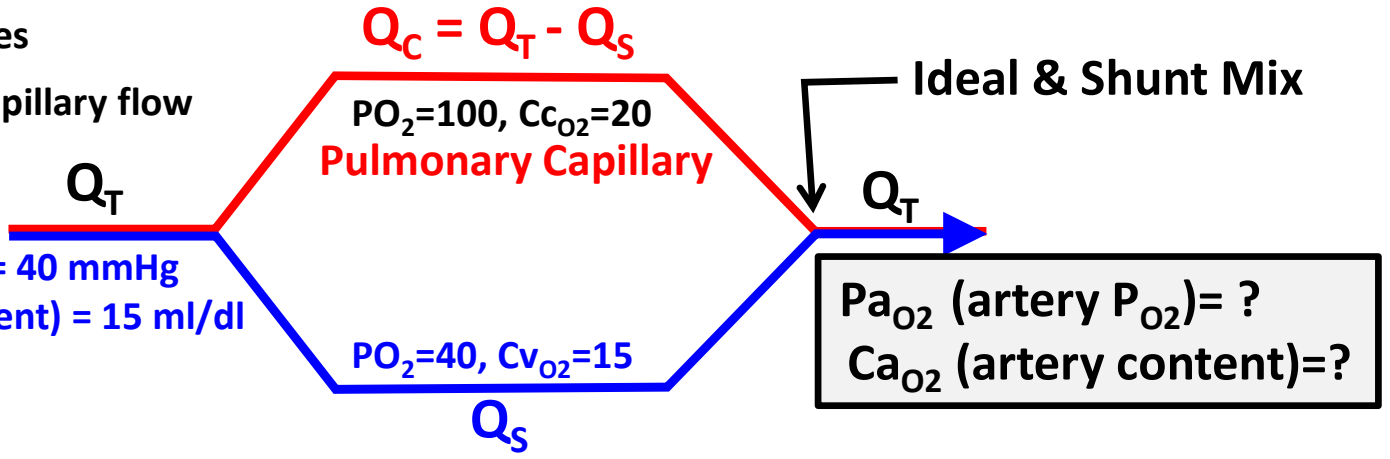
Q_S represents the shunt flow that would cause the measured arterial gas tensions

even though there are causes

Q_C is the ideally matched capillary flow

Q_T is the cardiac output

Pc_{O2} (capillary P_{O2}) = 100 mmHg
Cc_{O2} (capillary content) = 20 ml/dl



$$(1)\ Q_S \times Cv_{O2} + (Q_T - Q_S) \times Cc_{O2} = Q_T \times Ca_{O2}$$

Amt shunted | Amt not shunted | Total Amt

Q_S is shunt flow
Q_T is cardiac output

$$Ca_{O2} = Cc_{O2} - \frac{Q_S}{Q_T}(Cc_{O2} - Cv_{O2})$$

Rearrange (1)

Suppose equal shunt and capillary flows ($Q_S = Q_C \rightarrow Q_S/Q_T = 0.5$)

What is arterial O_2 content in ml/dl?

A. 14.5
B. 15.5
C. 16.5
D. 17.5
E. 20.5

$$Ca_{O2} = 0.5\,(Cc_{O2} + Cv_{O2}) = 17.5 \text{ ml/dl}$$

SAT = 17.5/20

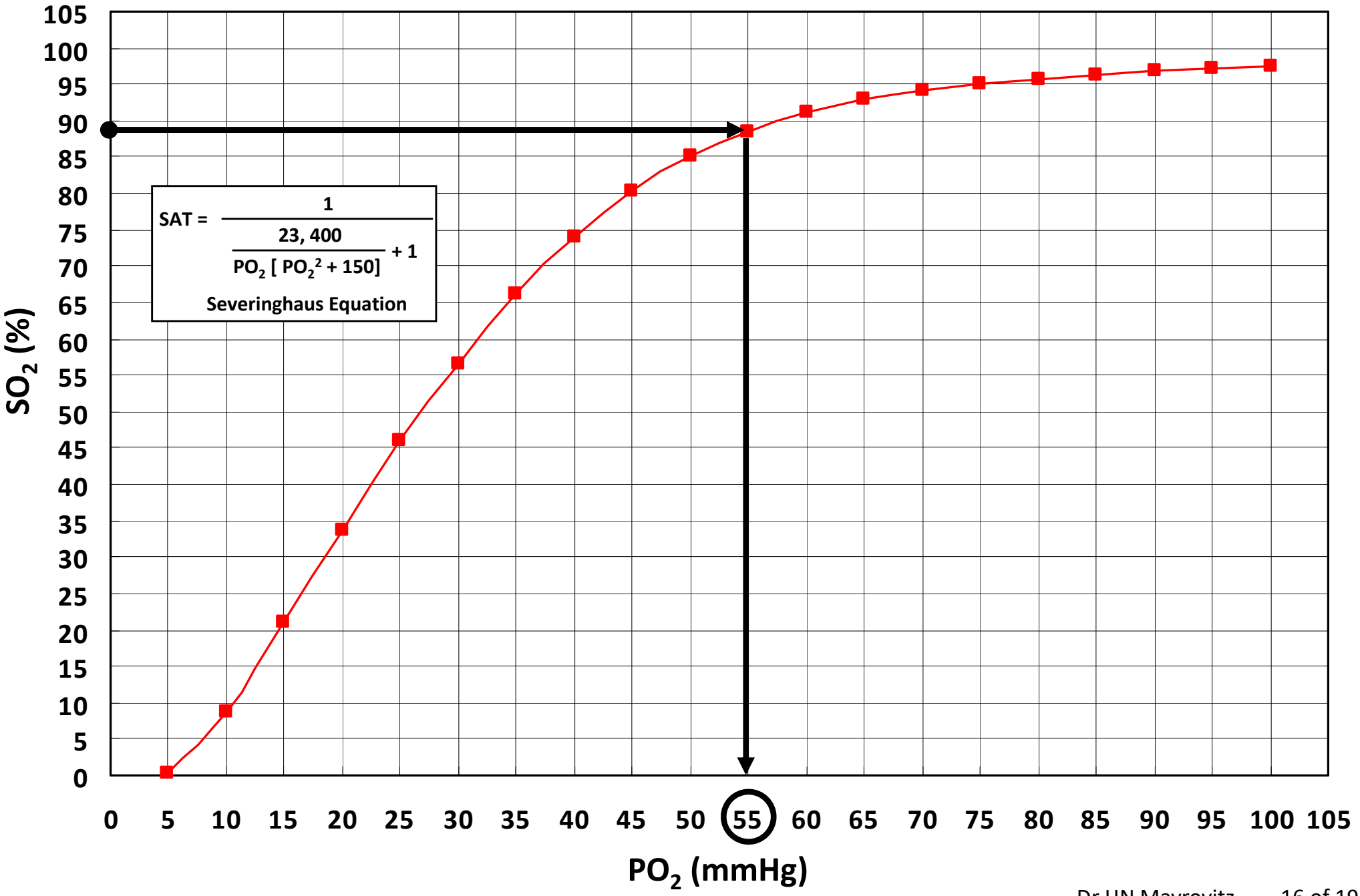
How would you determine the corresponding Pa_{O2}?

Use SO_2 curve or equation and get $Pa_{O2} \approx 55$ mmHg →

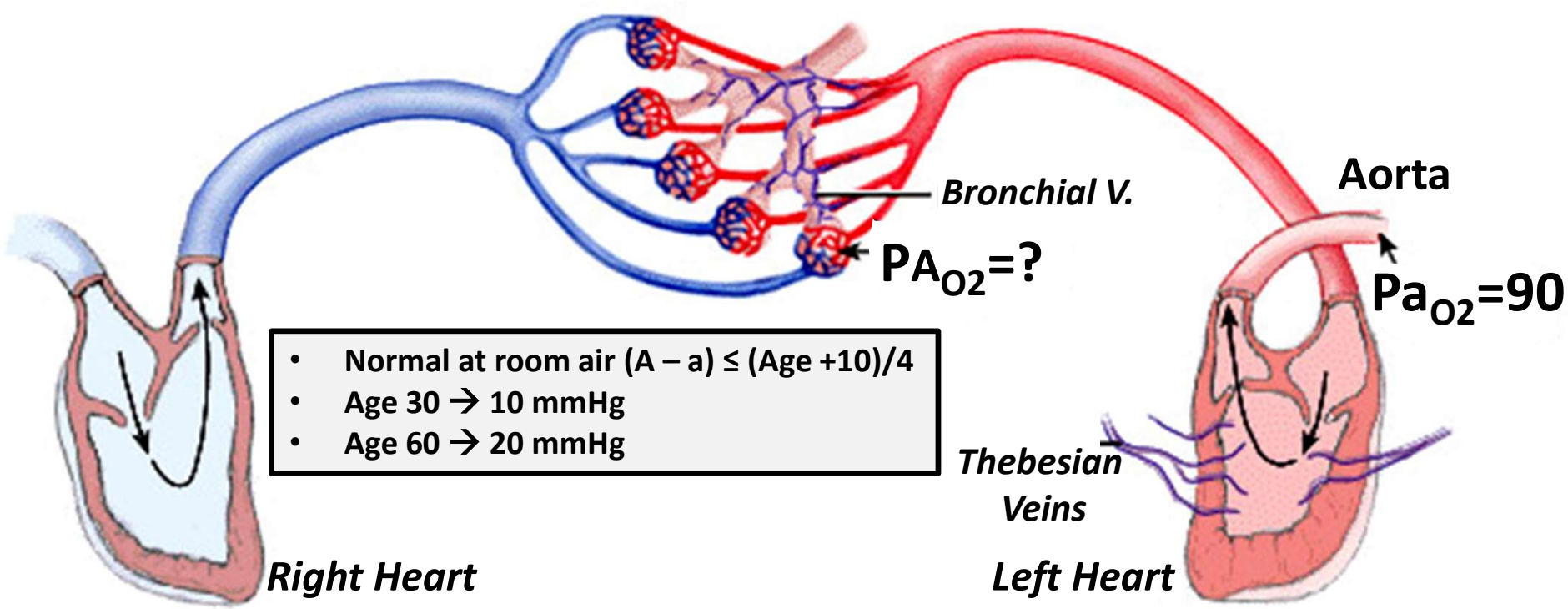
$$SAT = \frac{1}{\frac{23,400}{PO_2\,[\,PO_2^2 + 150]} + 1}$$

Severinghaus Equation

Blood Oxygen SAT vs PaO_2



The (A – a) PO_2 Gradient



Mary is a 60-year-old retired nurse who has just been evaluated for participation in a respiratory-related research study. As part of the study the following initial measurements were made. PA_{CO2} = 40 mmHg, respiratory quotient (R = 0.8) and her arterial oxygen tension (Pa_{O2} = 90 mmHg). Is her (A – a) gradient normal?

$$PA_{O2} \approx FIO_2 (P_{ATM} - 47) - Pa_{CO2} / R$$

$$PA_{O2} \approx 0.21 \times (760 - 47) - 40/.8 = 150 - 50 = 100 \text{ mmHg}$$

$$A - a = 100 - 20 = 10 \text{ mmHg}$$

Dead Space: Definitions and Calculations

- Anatomic (Airway) Dead Space = No gas exchange → Dead Space
- Alveolar Dead Space = Sum of alveolar volumes that receive little or no blood flow
  Ventilated but low or no perfusion (V'/Q → infinity)
  Example is Zone I if low pulmonary artery pressure)
- Physiological Dead Space = Amount of each tidal volume that
  does NOT participate in gas exchange
- Physiological Dead Space = PDS = Anatomic DS + Alveolar DS

Collecting Expired Volume in a Bag

$$PDS = TV \times (1 - P_ECO_2/P_aCO_2)$$

P_aCO_2 = CO_2 tension in arterial blood

P_ECO_2 = CO_2 tension in expired air

- If ratio = 1 → no dead space
- If ratio = 0 → all dead space
- The lower the CO_2 tension in the expired air the greater is the physiological dead space!

Oxygen Deficiency –Terms and Definitions

ANOXIA = No O_2

HYPOXEMIA = Hypoxic Hypoxia

= Low arterial blood PO_2

HYPOXIA = Inadequate O_2 Available for Tissue Needs

Hematological Hypoxia

Low Hb to bind/carry O_2 but normal PO_2

e.g. Anemia or Carbon Monoxide Poisoning

Ischemic Hypoxia

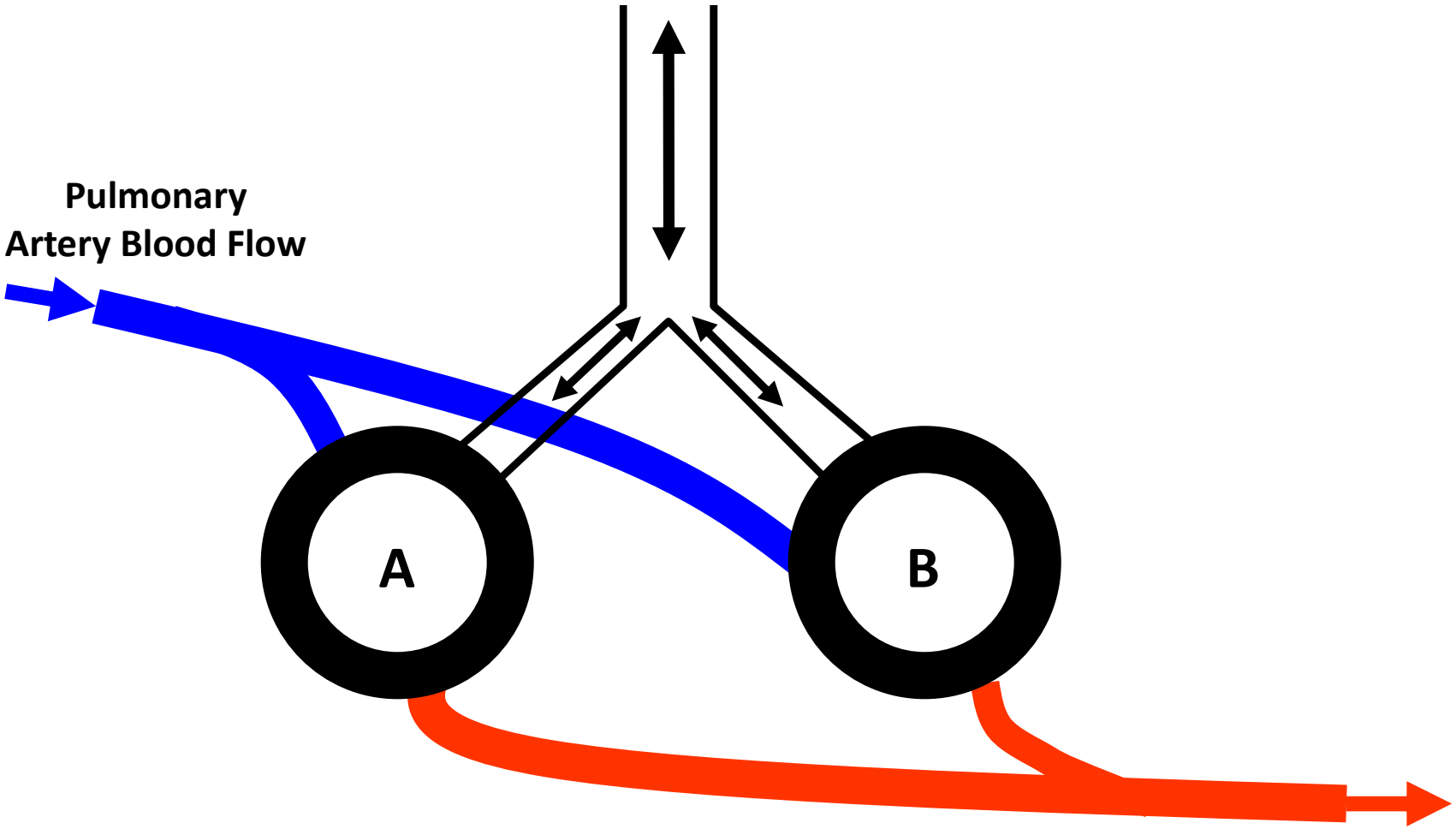
Low tissue O_2 due to low flow (blood PO_2 is normal)

Histotoxic Hypoxia

Normal O_2 supplied but can't be utilized by tissue;

e.g. Cyanide Poisoning

End Respiratory Physiology
Lecture 42


Pulmonary
Artery Blood Flow
A
B

Respiratory Mechanoreceptors

Receptors Located in

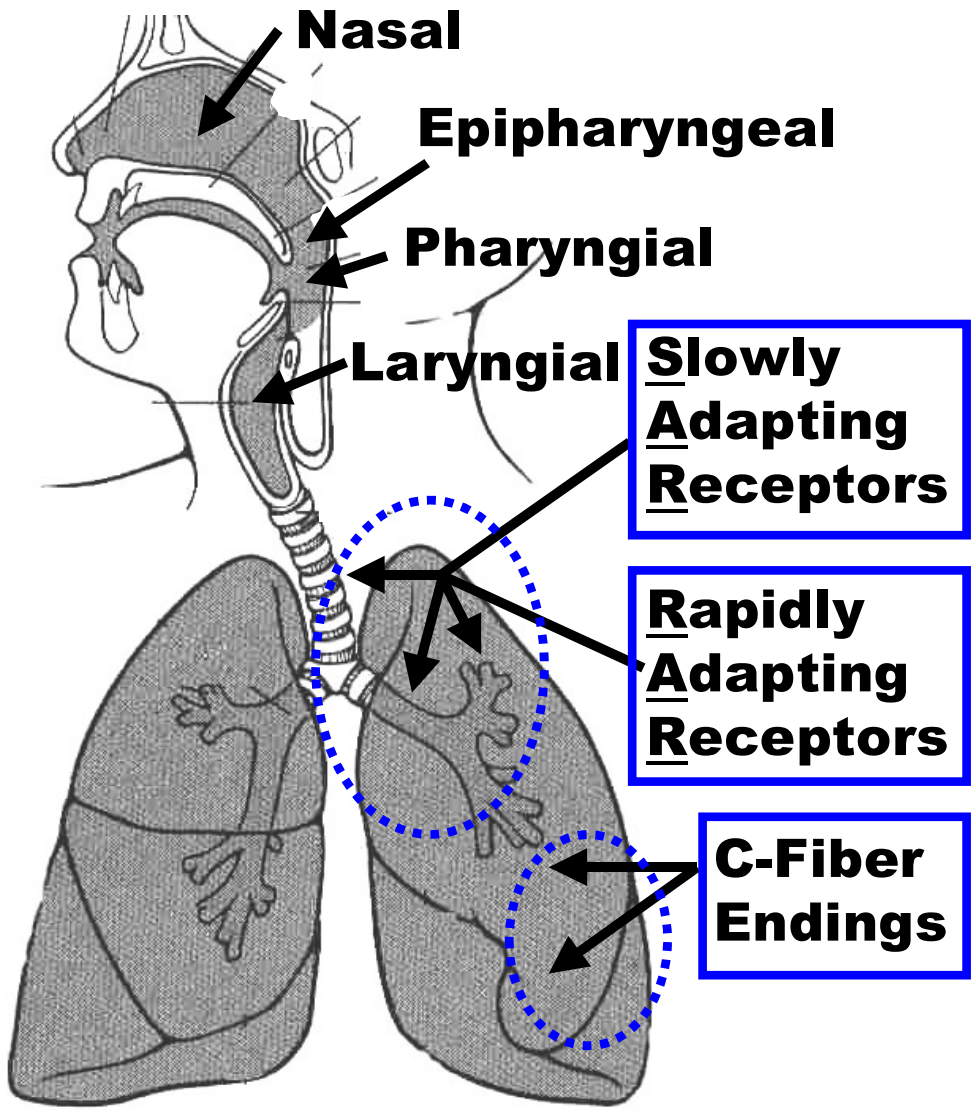
- Upper respiratory
- Tracheo-bronchial tree
- Lung parenchyma

Broadly three types

- Slowly Adapting (SAR)
  Among ASM cells
- Rapidly Adapting (RAR)
  Among airway epithelial cells
- C-fiber endings (J-receptors)
  near blood vessels/capillaries

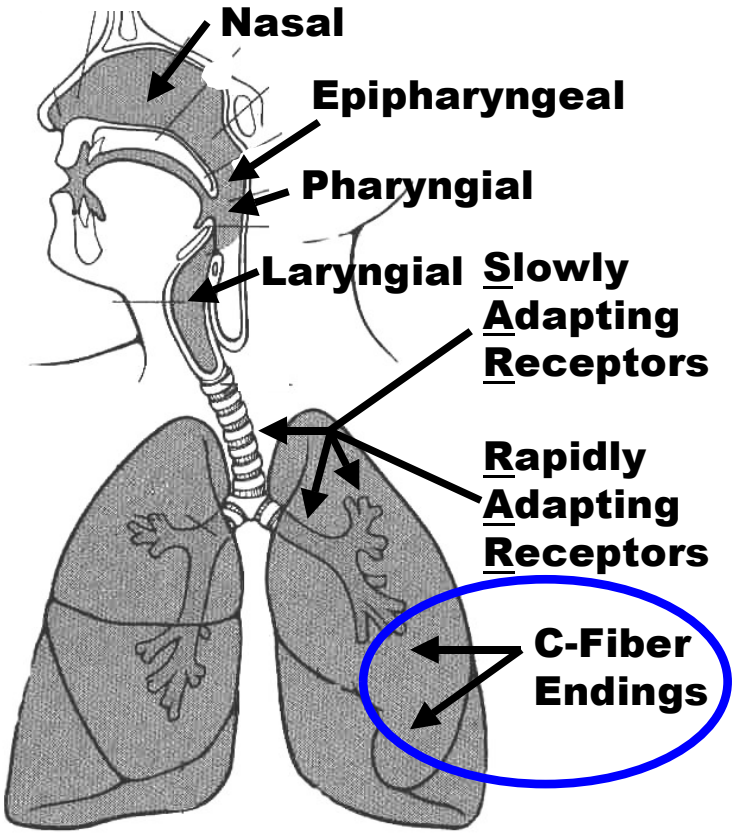
Vagal Afferents

- Connect to respiratory cntr
- Initiate many reflexes

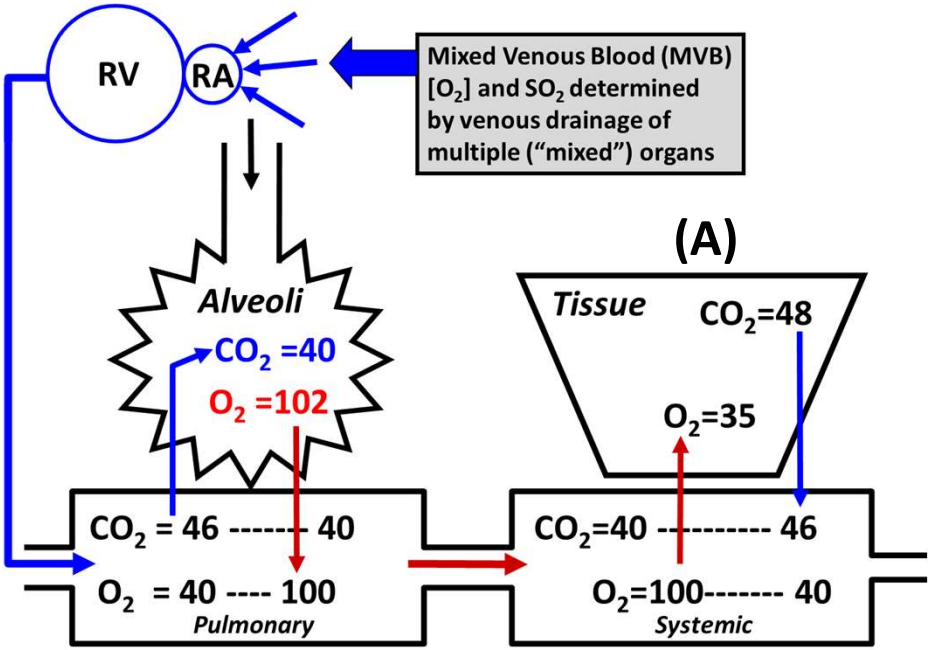


C-Fiber Receptors (Juxtacapillary or J Receptors)

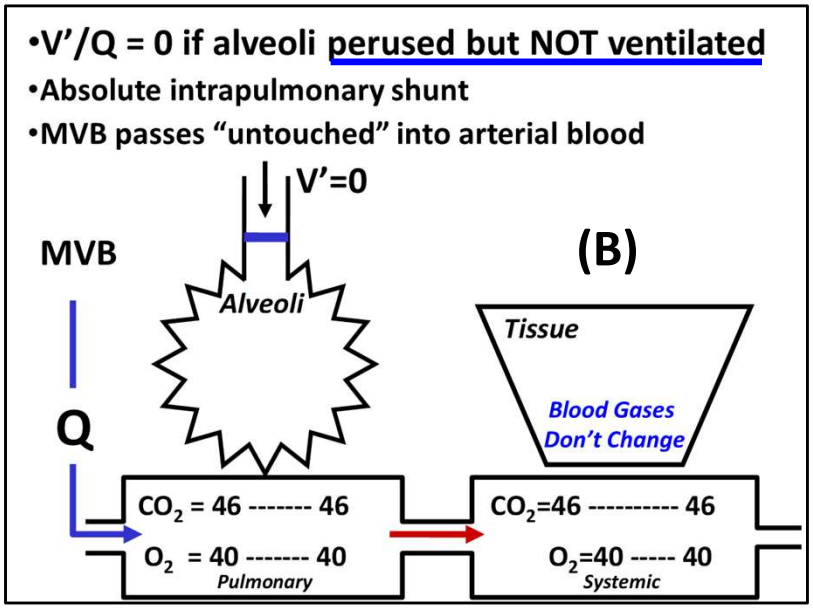
- Network of small unmyelinated axons (C-fibers) innervate receptors in alveoli near or in the walls of pulmonary capillaries
- Sensitive to distension and/or distortion caused by increases in capillary or interstitial volume
- Increased distention leads to increased ventilation (*pulmonary congestion* by LV failure)
- Decreased distention leads to decreased ventilation (e.g. pulmonary *embolism* that obstructs flow proximal to capillaries)

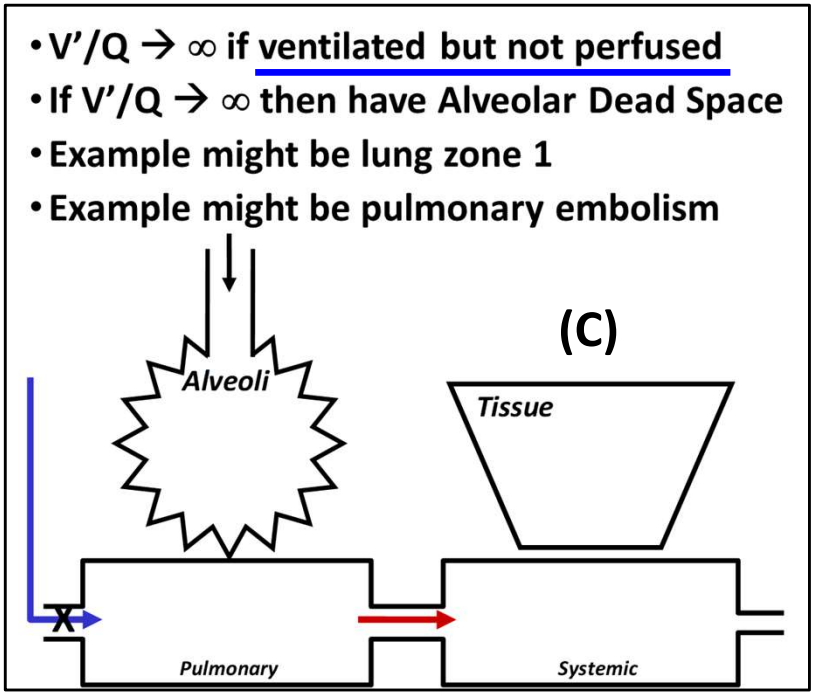


V'/Q: Normal – Low – High Reviewed

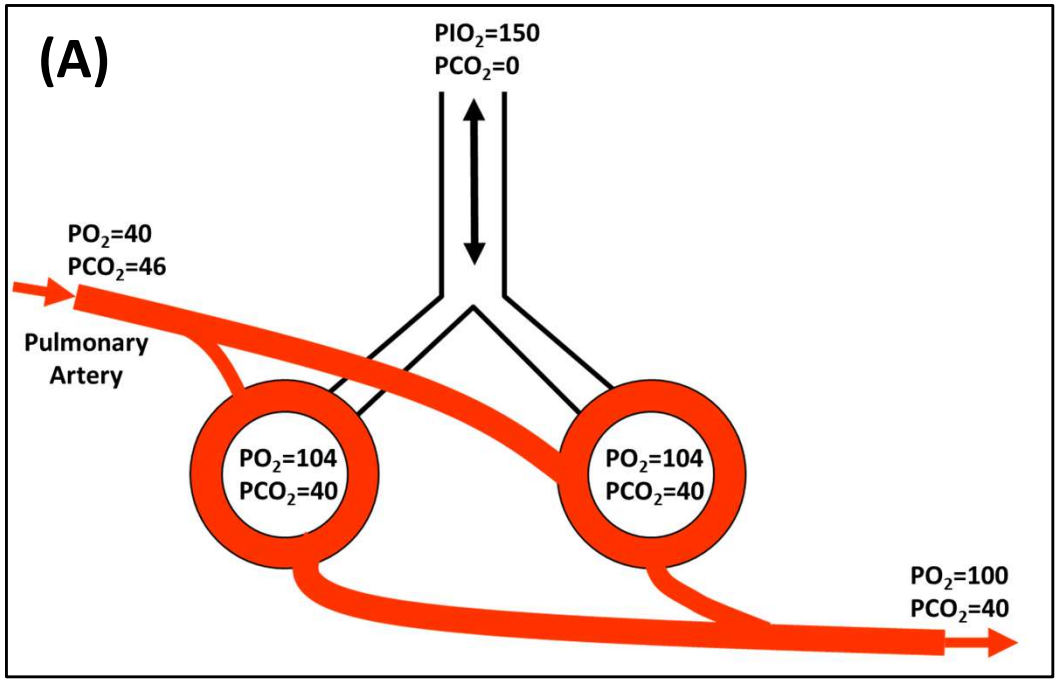


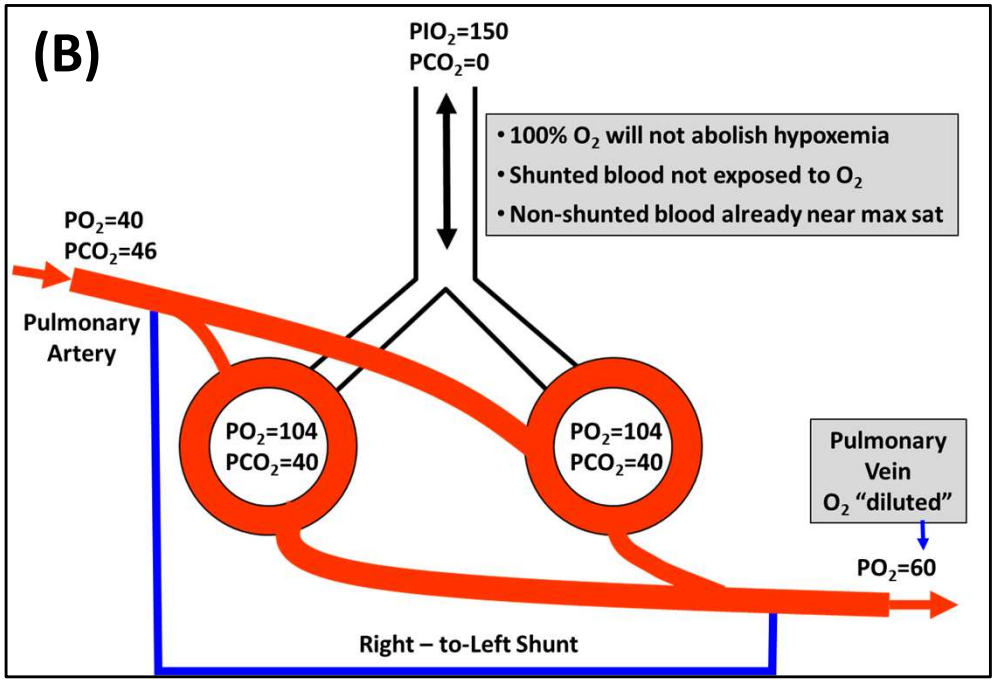
- (A) With normal conditions mixed venous blood enters lung at elevated CO_2 and reduced O_2 with resulting gas tensions indicated in (A)
- (B) <u>Alveoli perfused but not ventilated</u> as with airway blockage then this represents an "absolute" intrapulmonary shunt → blood passes with gas exchange
- (C) <u>Alveoli ventilated but not perfused</u> "wasted" ventilation → alveolar dead space



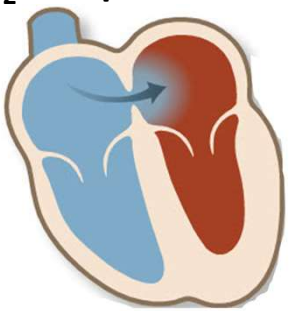


Treating Shunts with Oxygen



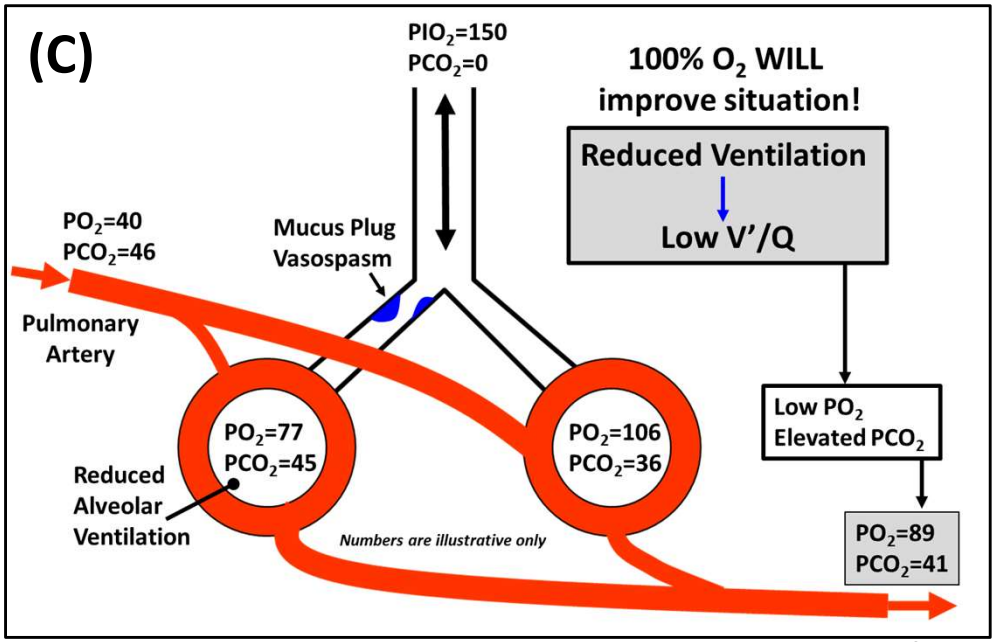


- (A) Normal
- (B) Right-to-left shunt: e.g. Patent Foramen Ovale (PFO). Giving 100% O_2 won't help! Shunted blood won't be exposed to O_2 except minor changes in dissolved O_2

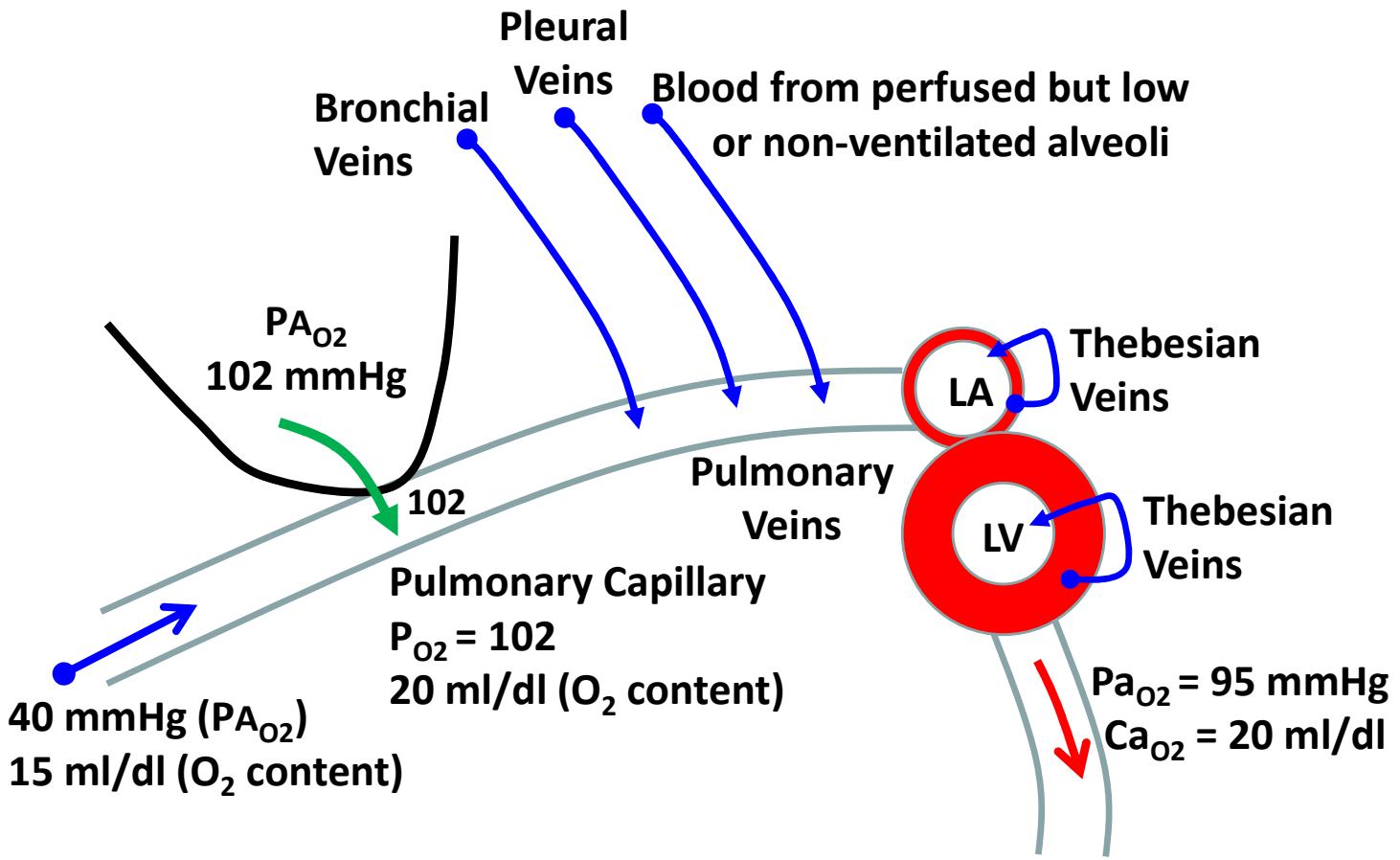


PFO

- (C) Contrastingly, in cases where low V'/Q arises because of inadequate alveolar ventilation, O_2 administered will have an important clinical affect



Shunts: Mixing Low O_2 Blood with Arterial Blood



Shunts: Quantitative Aspects – Shunt Equation

Q_S represents the shunt flow that would cause the measured arterial gas tensions

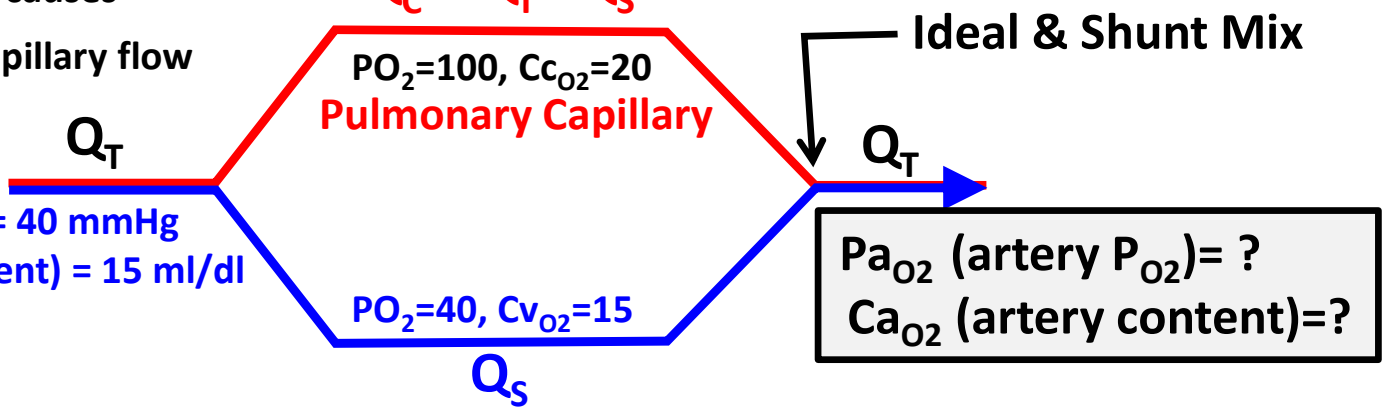
even though there multiple causes

Q_C is the ideally matched capillary flow

Q_T is the cardiac output

Pc_{O2} (capillary P_{O2}) = 100 mmHg
Cc_{O2} (capillary content) = 20 ml/dl

$Q_C = Q_T - Q_S$



$$(1)\ Q_S \times Cv_{O2} + (Q_T - Q_S) \times Cc_{O2} = Q_T \times Ca_{O2}$$

Amt shunted | Amt not shunted | Total Amt

Q_S is shunt flow
Q_T is cardiac output

$$Ca_{O2} = Cc_{O2} - \frac{Q_S}{Q_T}(Cc_{O2} - Cv_{O2})$$

Rearrange (1)

Suppose equal shunt and capillary flows ($Q_S = Q_C \rightarrow Q_S/Q_T = 0.5$)

What is arterial O_2 content in ml/dl?

A. 14.5
B. 15.5
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$$Ca_{O2} = 0.5\,(Cc_{O2} + Cv_{O2}) = 17.5 \text{ ml/dl}$$

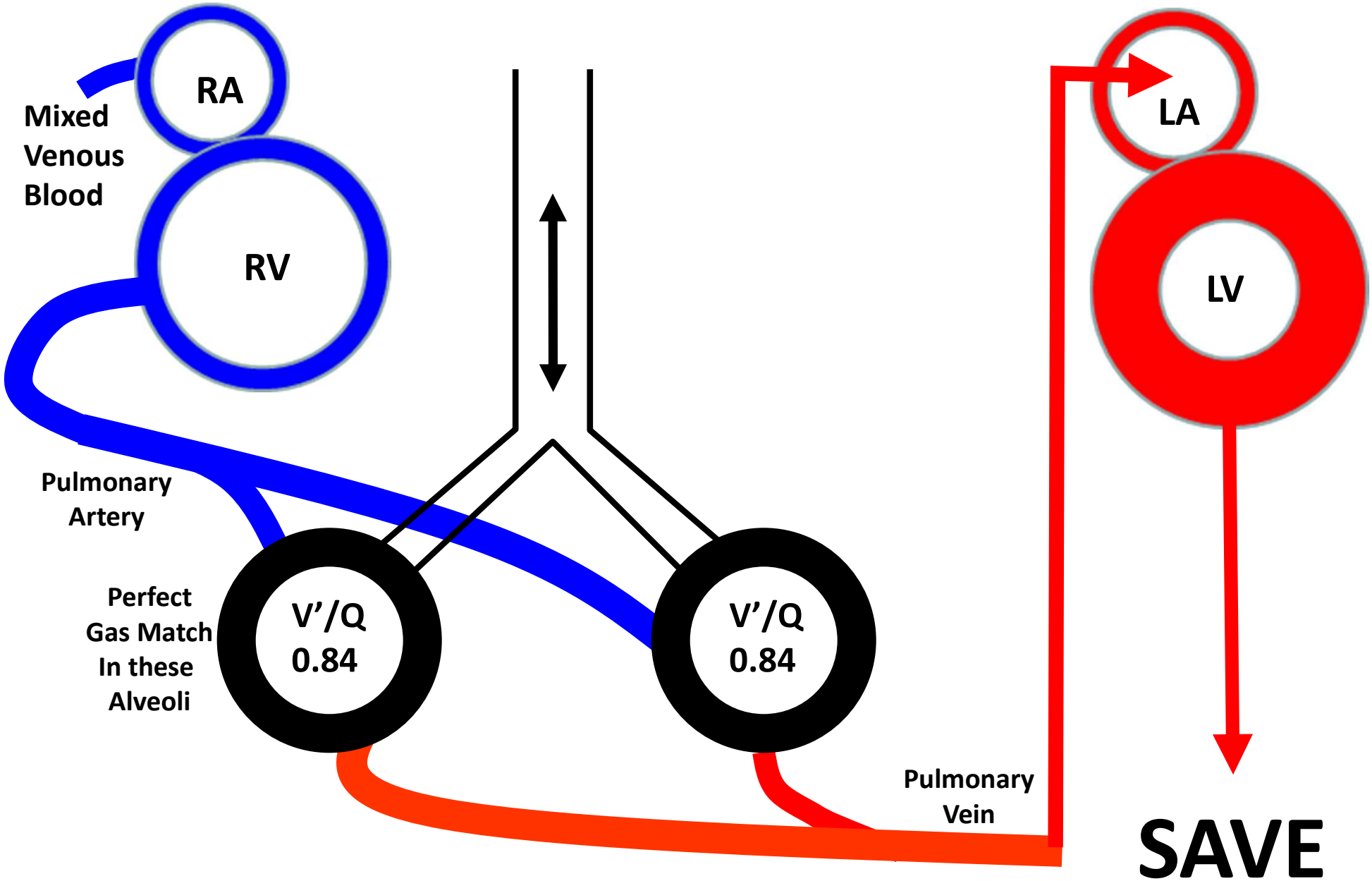
SAT = 17.5/20

How would you determine the corresponding Pa_{O2}?

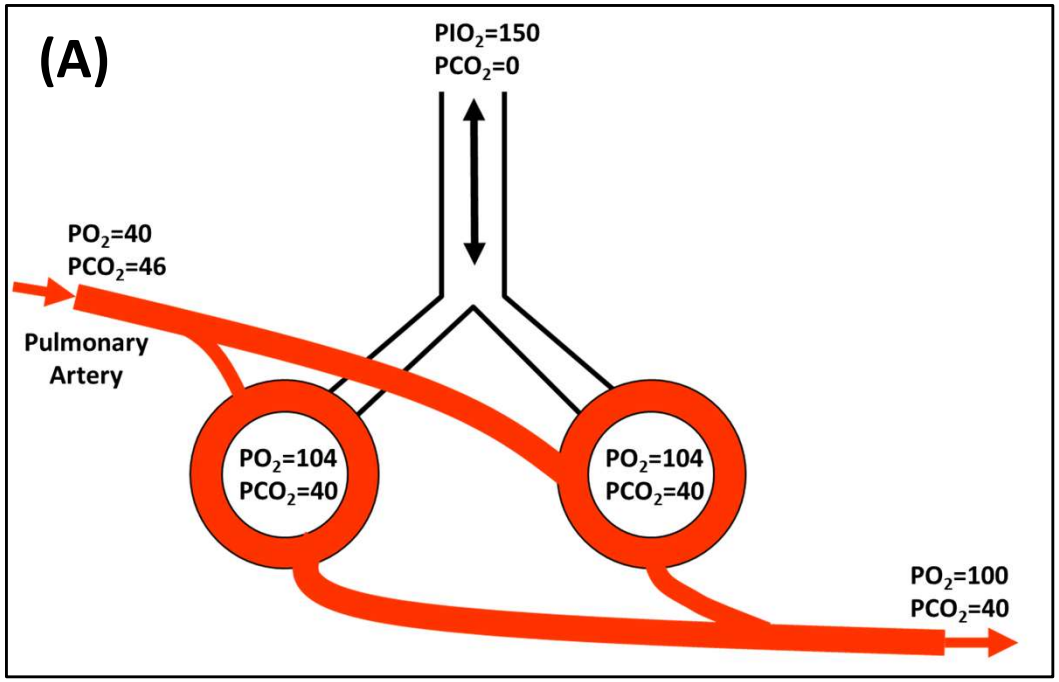
Use SO_2 curve or equation and get $Pa_{O2} \approx 55$ mmHg →

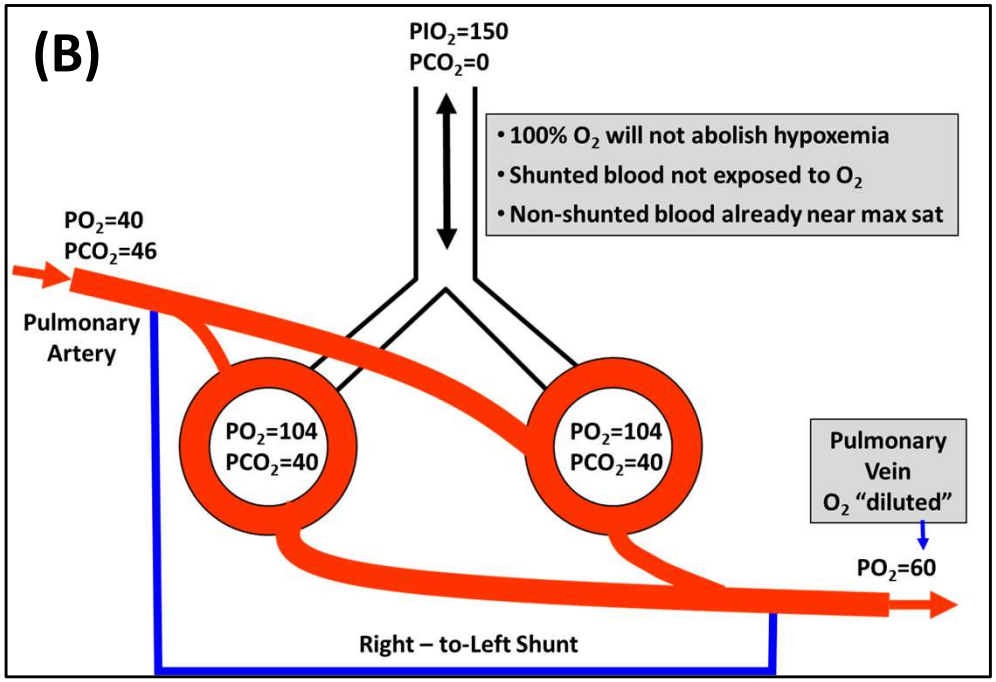
$$SAT = \frac{1}{\frac{23,400}{PO_2\,[\,PO_2^2 + 150]} + 1}$$

Severinghaus Equation


RA
Mixed
Venous
Blood
RV
LA
LV
Pulmonary
Artery
Perfect
Gas Match
In these
Alveoli
V'/Q
0.84
V'/Q
0.84
Pulmonary
Vein
SAVE

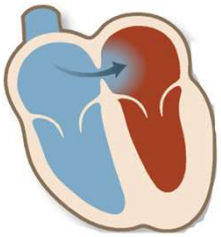
Treating Shunts with Oxygen



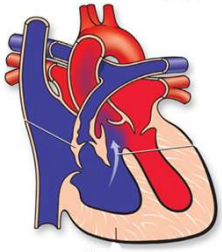


- (A) Normal
- (B) Right-to-left shunt: e.g. patent foramen ovale (PFO) or Tetralogy of Fallot (TOF). Giving 100% O_2 won't help since shunted blood not be exposed to the O_2 except the very minor effect on changes in dissolved oxygen

PFO



TOF



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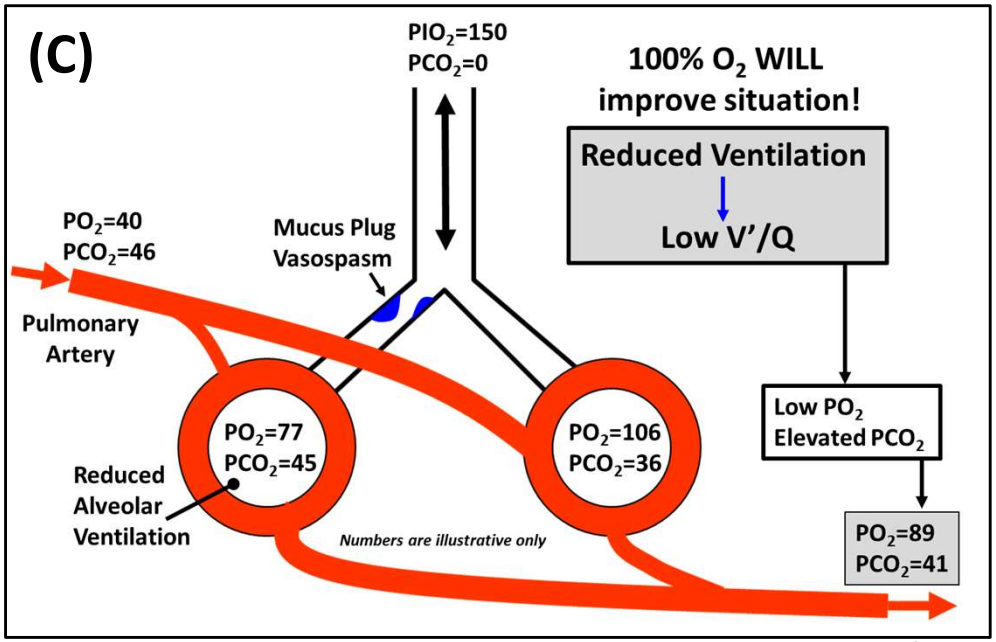
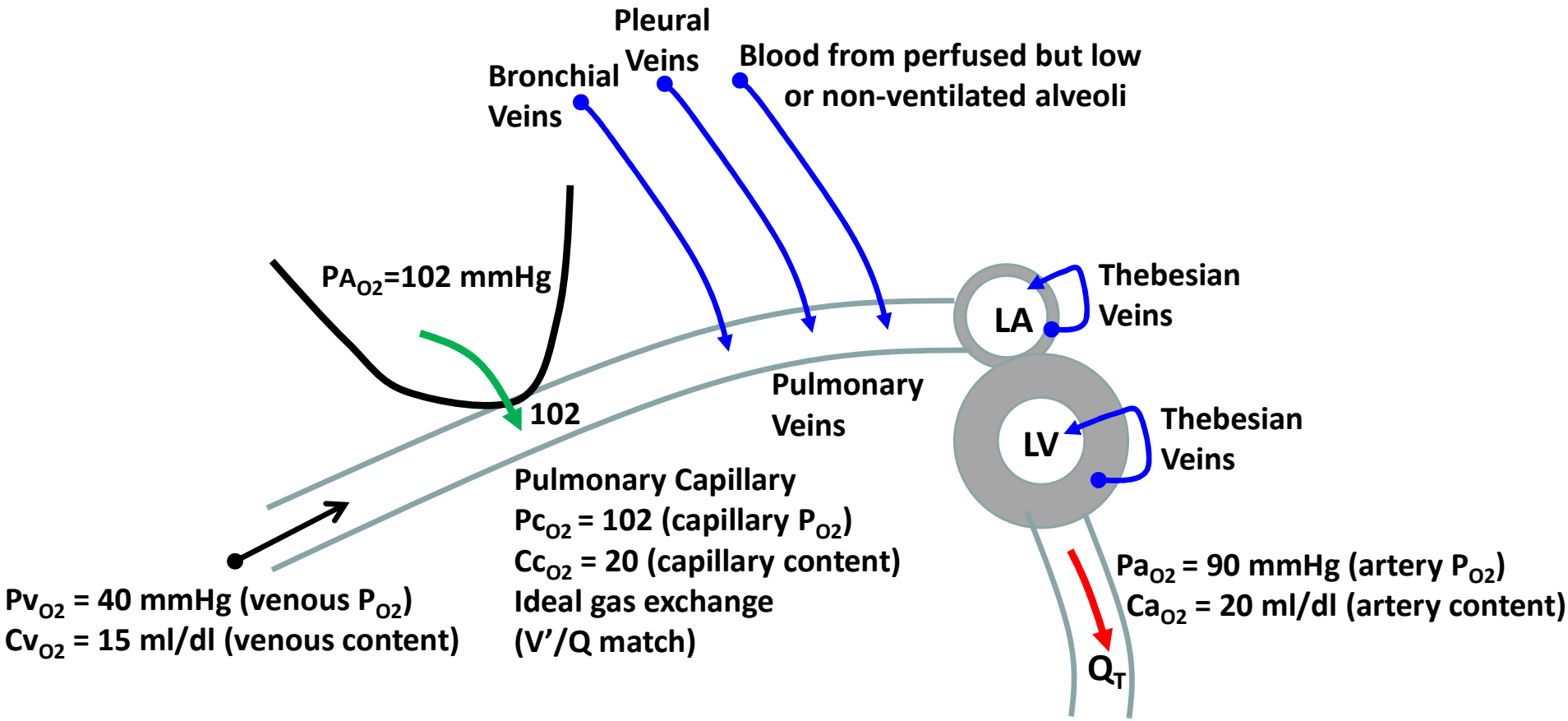


Shunts: Mixing Low O_2 Blood with Arterial Blood



$$Q_S \times Cv_{O2} + (Q_T - Q_S) \times Cc_{O2} = Q_T \times Ca_{O2}$$

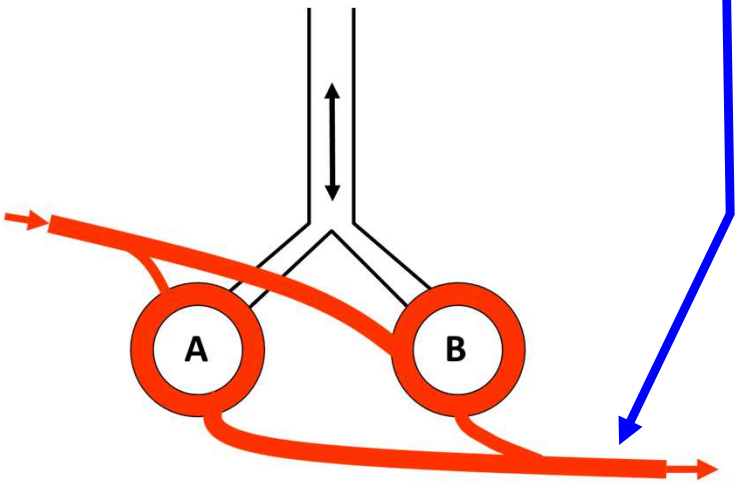
Amt shunted | Amt not shunted | Total Amt

Q_S is shunt flow
Q_T is cardiac output

$$\frac{Q_S}{Q_T} = \frac{Cc_{O2} - Ca_{O2}}{Cc_{O2} - Cv_{O2}}$$

Mechanisms – Hypoxemia – Low O_2 in Blood

PROCESS	EXAMPLE
Reduced O_2 in inspired air ------>	Altitude Fires
Inadequate Ventilation ---------->	CNS depression (anesthesia) Neural deficit Muscle deficit
Diffusion Abnormality ---------->	Edema, Fibrosis



Shunts ----------> Anatomical → Bronchial artery drainage
Functional → Perfusion with no ventilation

V/Q mismatching ----------> Airway block → low V' → low v'/Q
Embolus → increased Q to remaining area
→ Low v'/Q

Alveolar DS → V' with no perfusion → Zone I

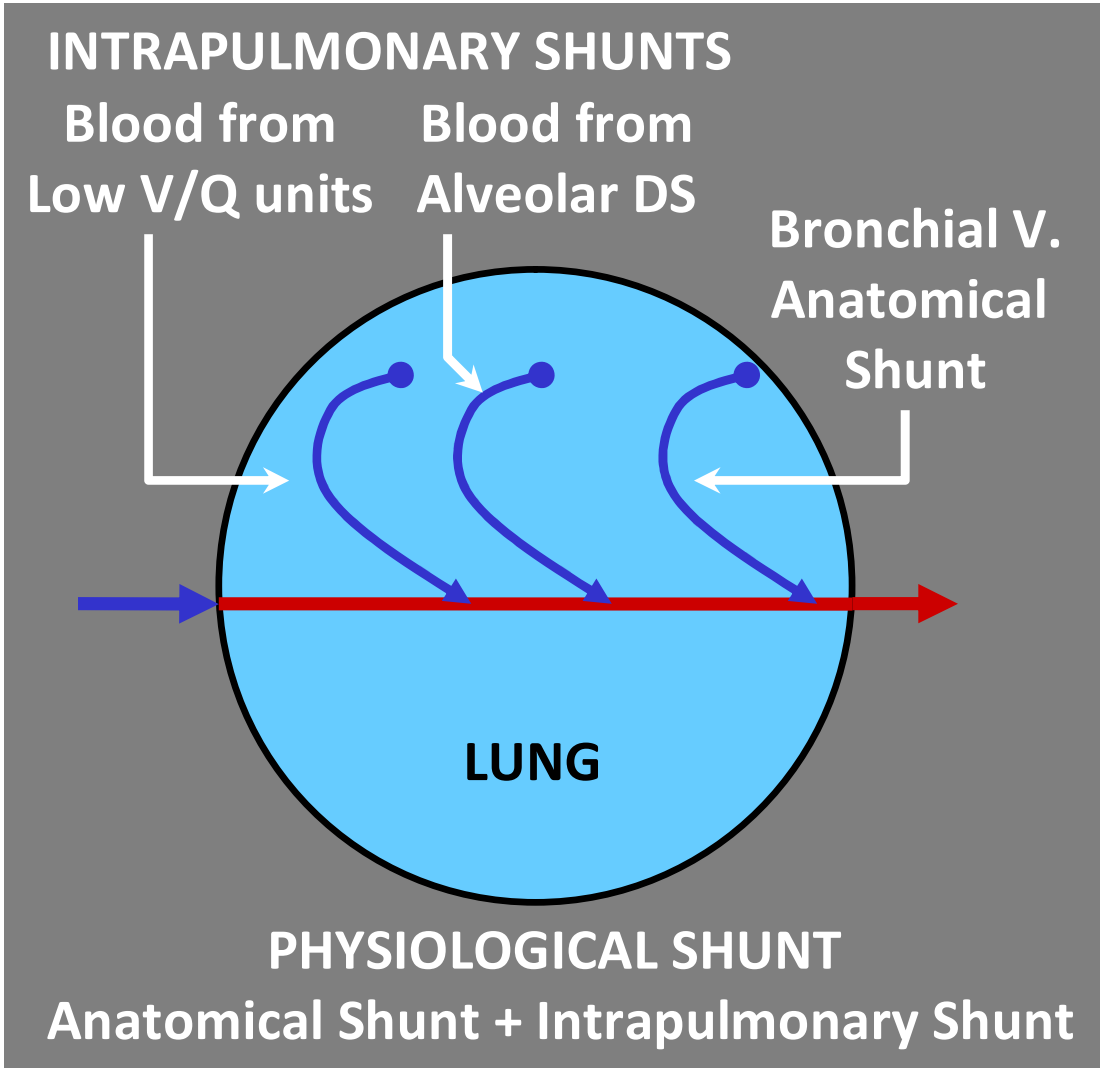
Shunts: Mixing Low O_2 Blood with Arterial Blood

Anatomical Shunts:
systemic venous blood

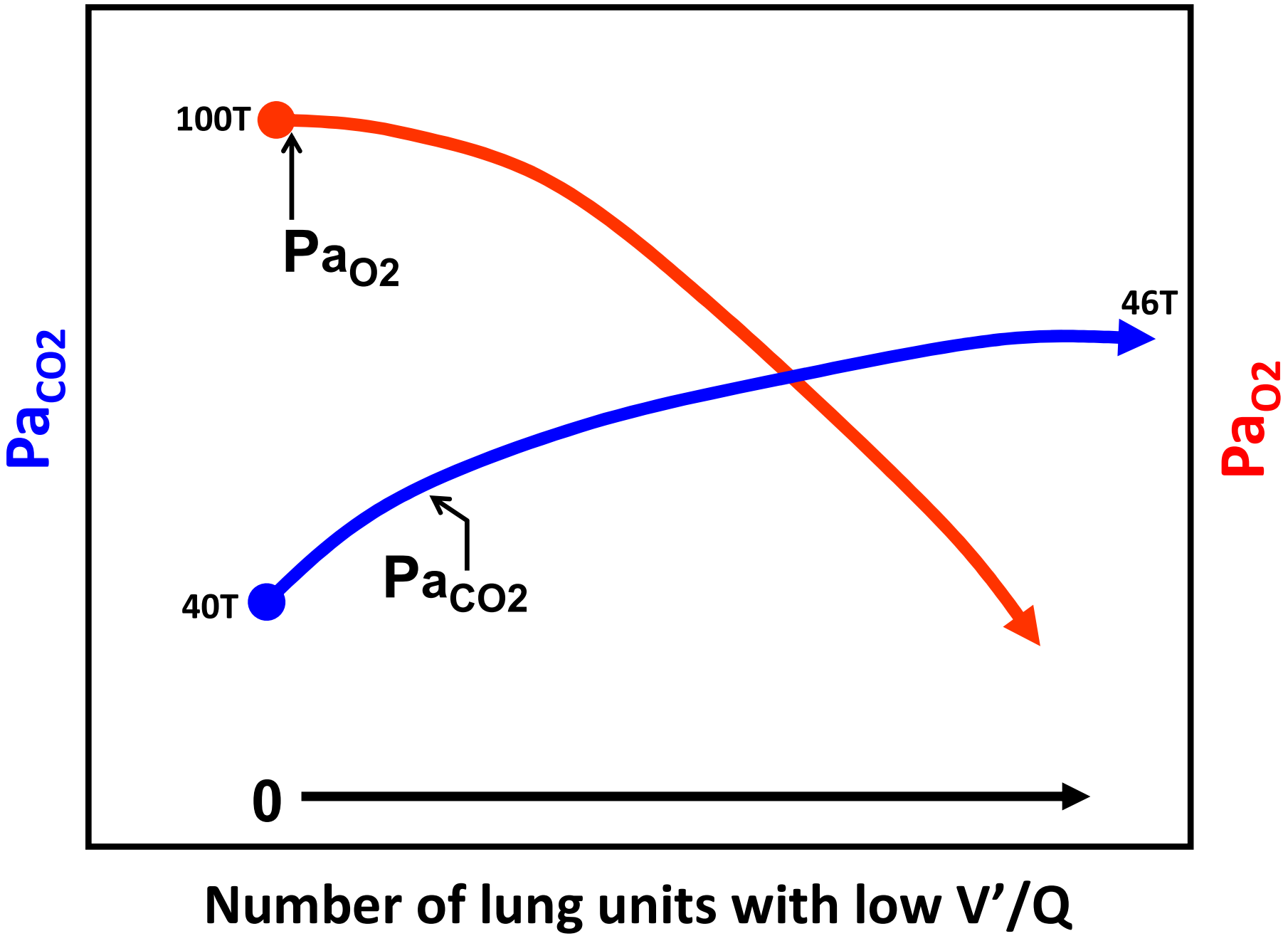
- Bronchial veins
- Thebesian veins
- Pleural veins

Intrapulmonary Shunts:

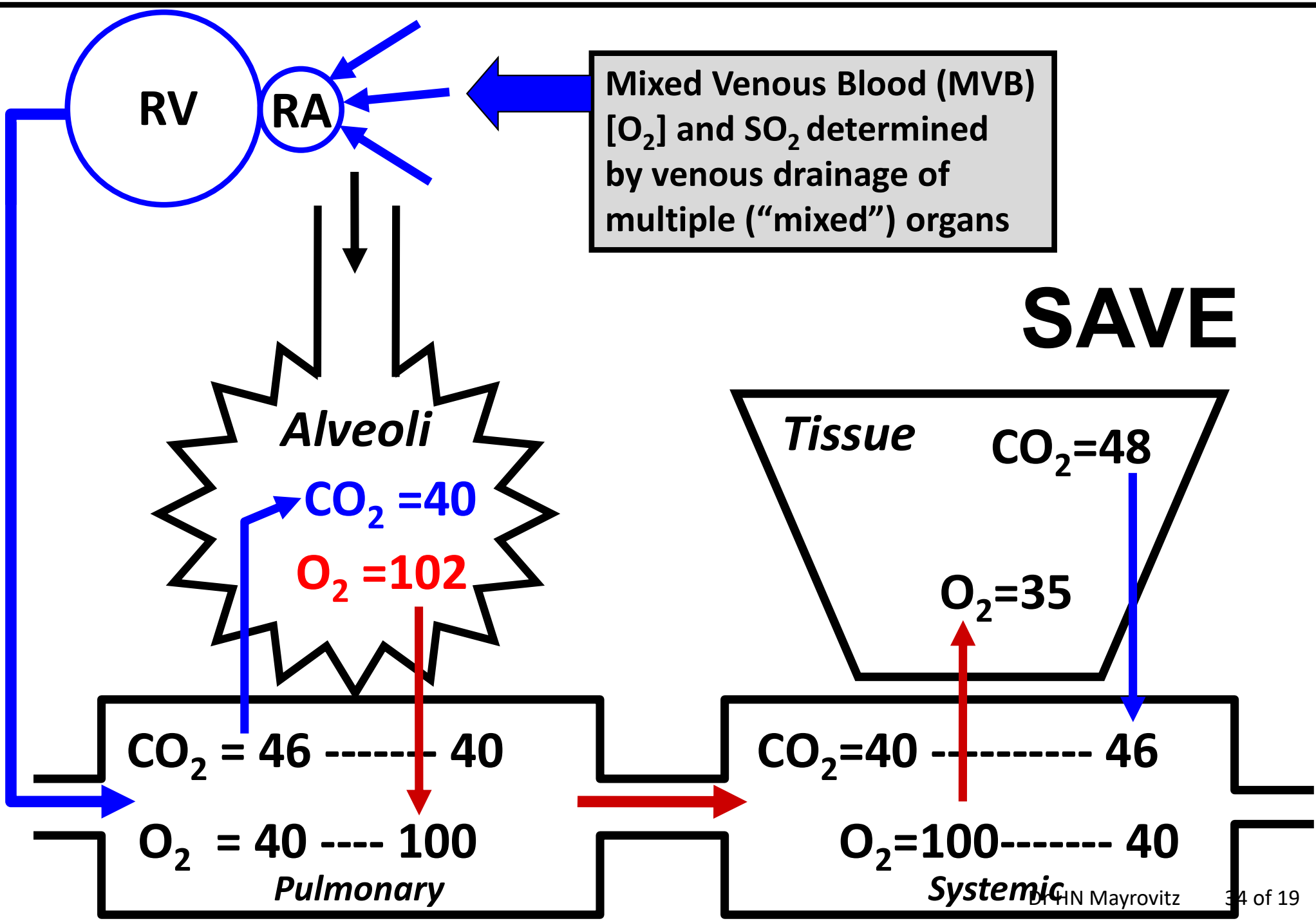
- Mixed venous blood has zero alveolar gas exchange (e.g. airway obstruction)
- Low V/Q → low O_2 mixes with all oxygenated blood



Another View of Mismatching

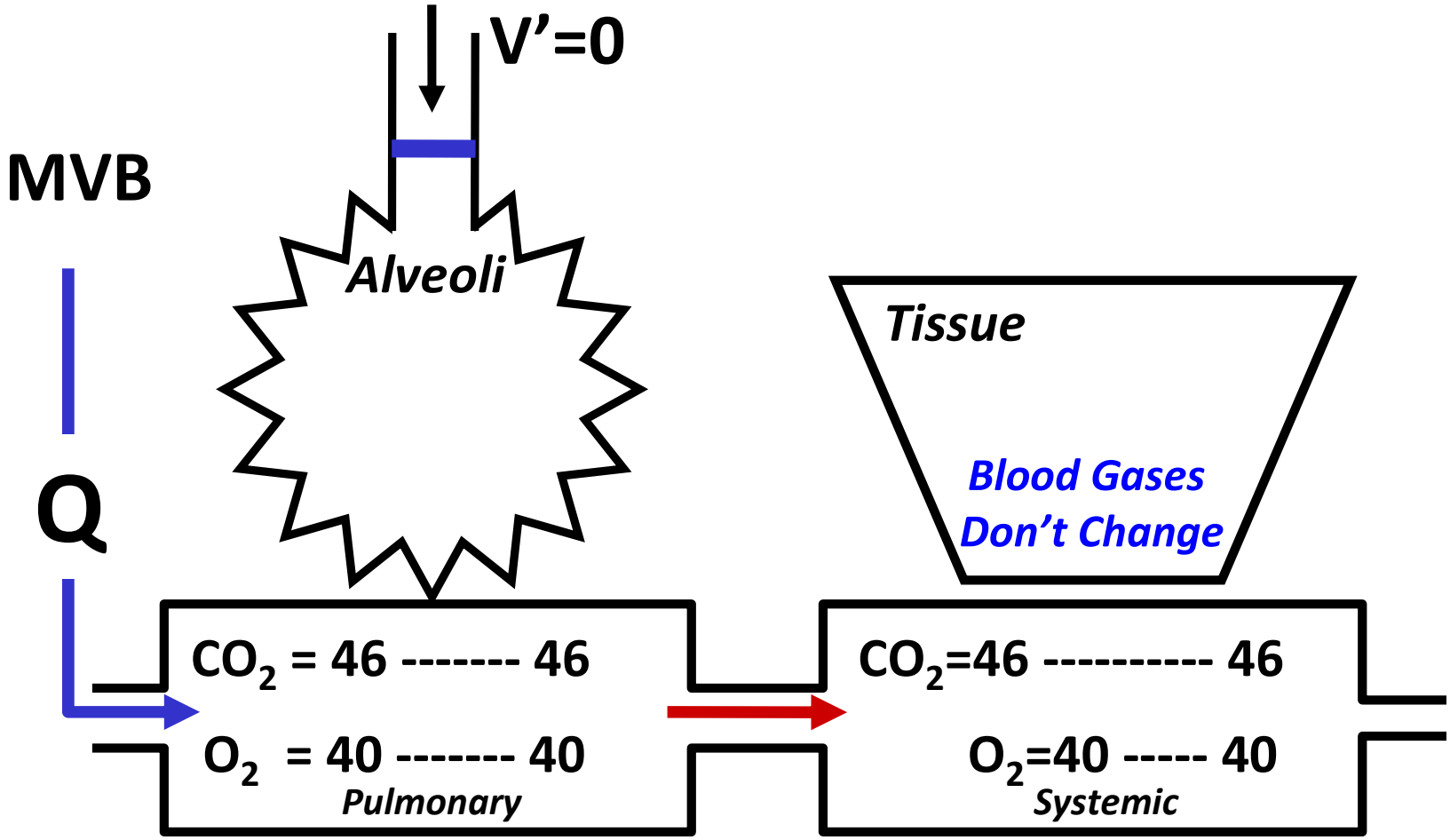


Review of Normal O_2 –CO_2 Process

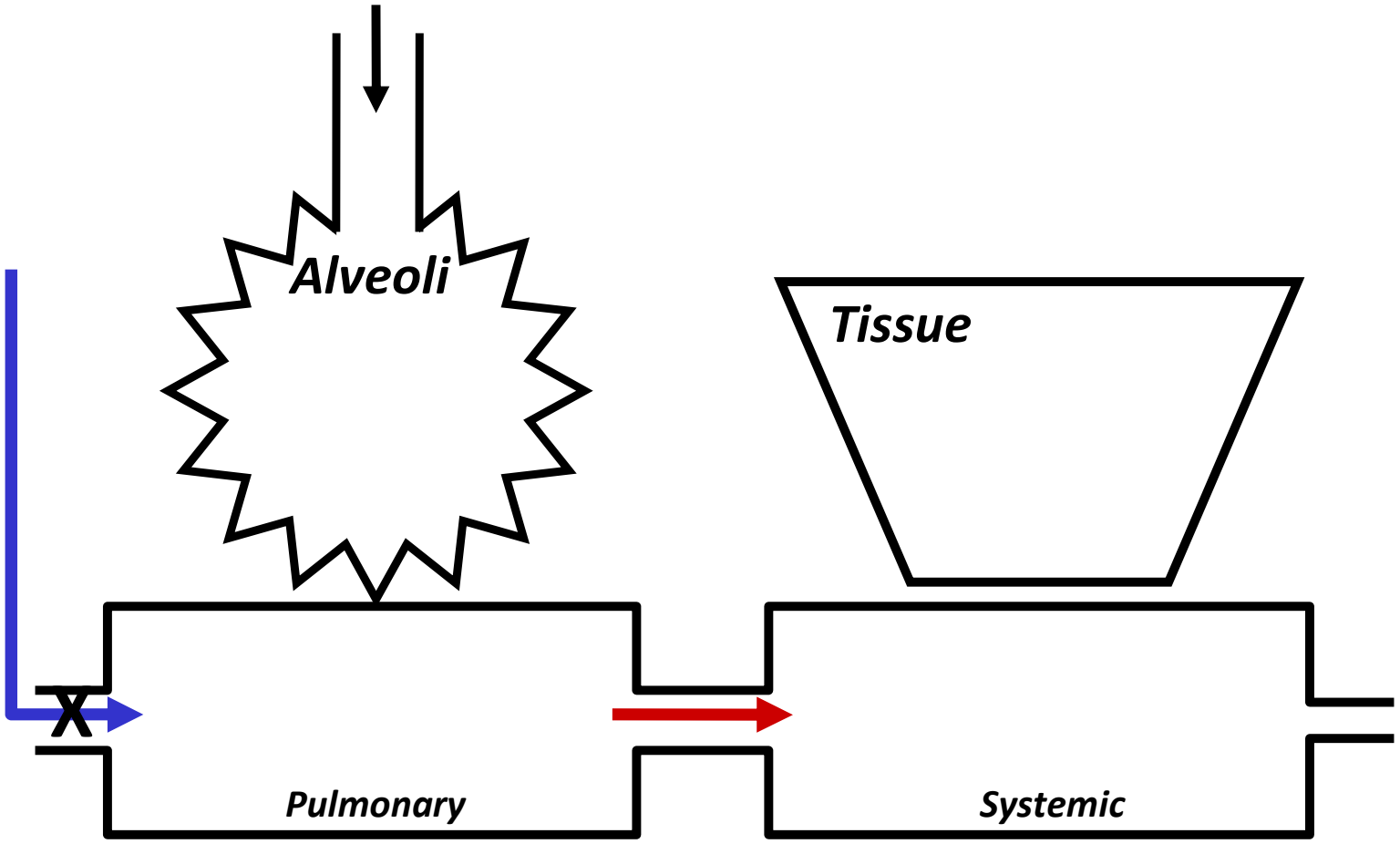


Review: V'/Q Low Limit

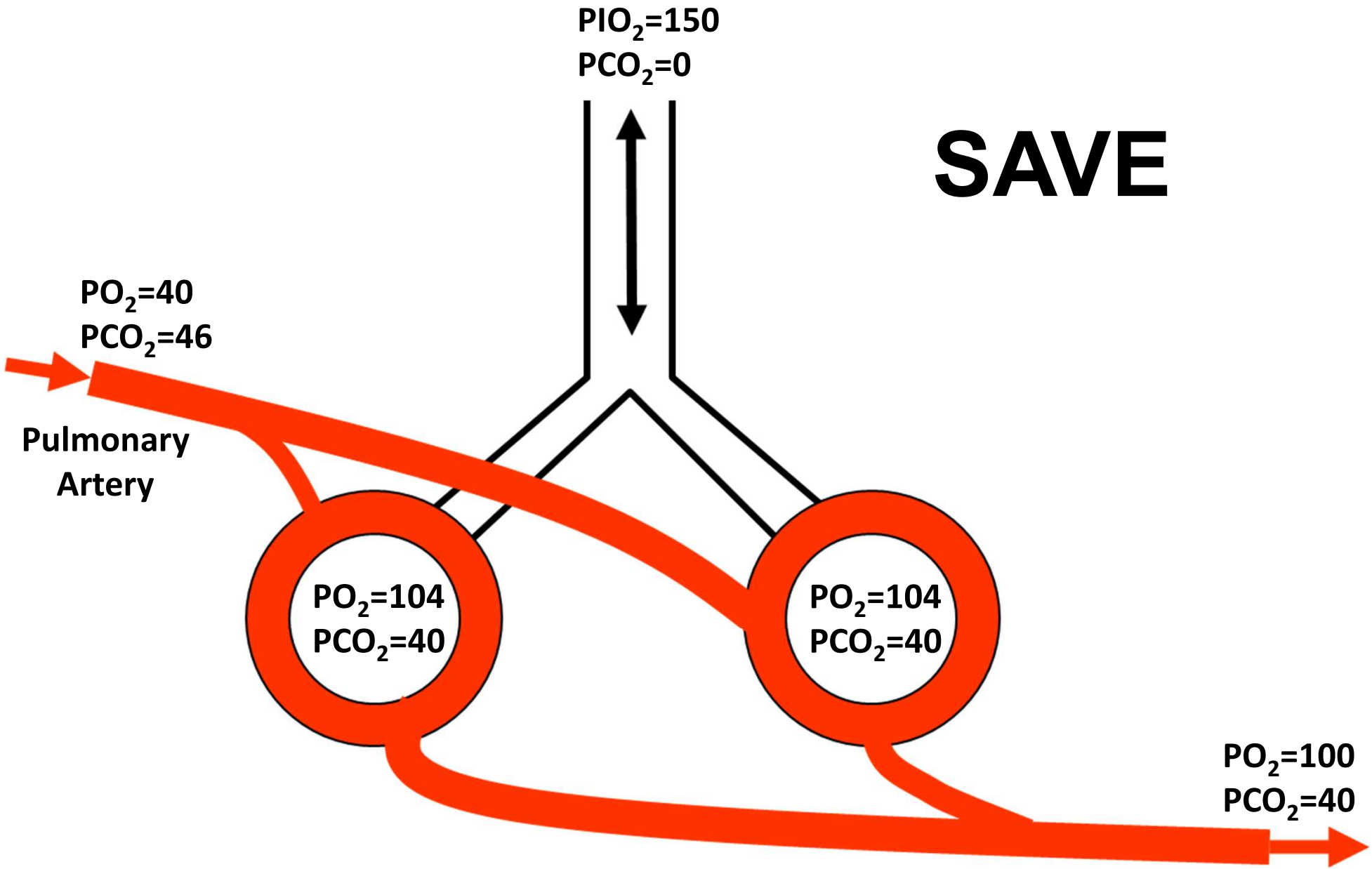
- V'/Q = 0 if alveoli perused but NOT ventilated
- Absolute intrapulmonary shunt
- MVB passes "untouched" into arterial blood



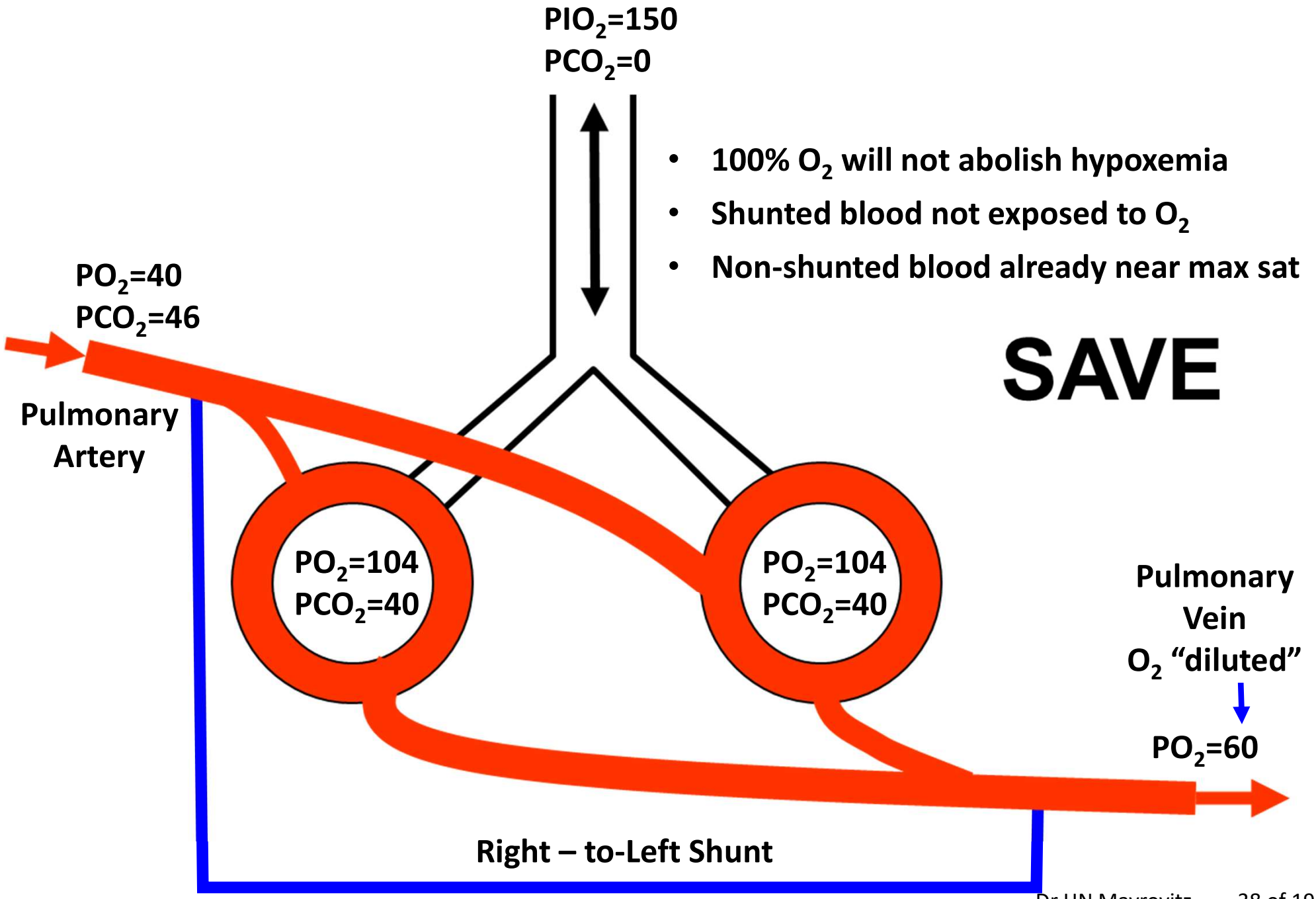
- V'/Q → ∞ if ventilated but not perfused
- If V'/Q → ∞ then have Alveolar Dead Space
- Example might be lung zone 1
- Example might be pulmonary embolism



Normal: No Significant Shunting



Anatomical Shunt (Right-to-Left)



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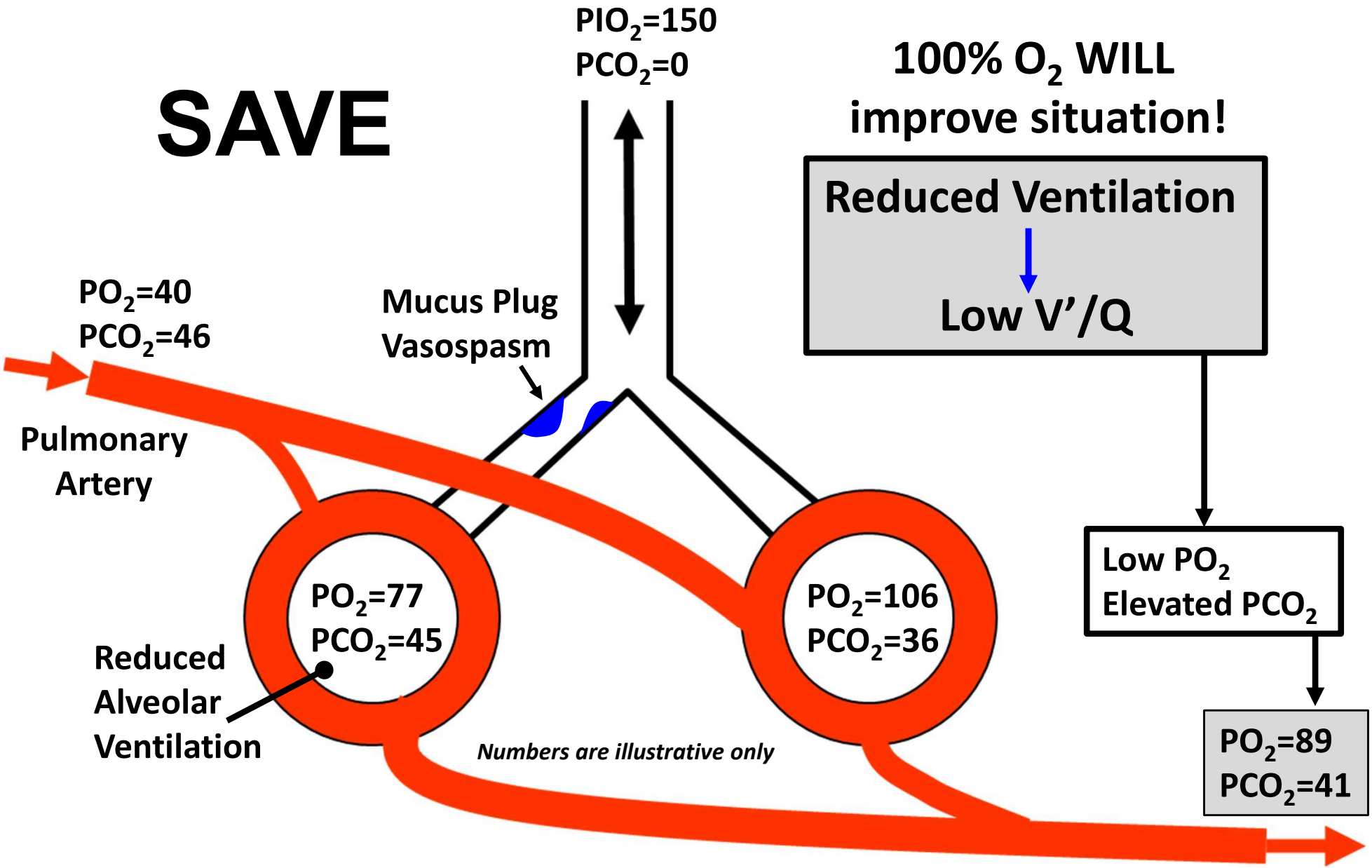
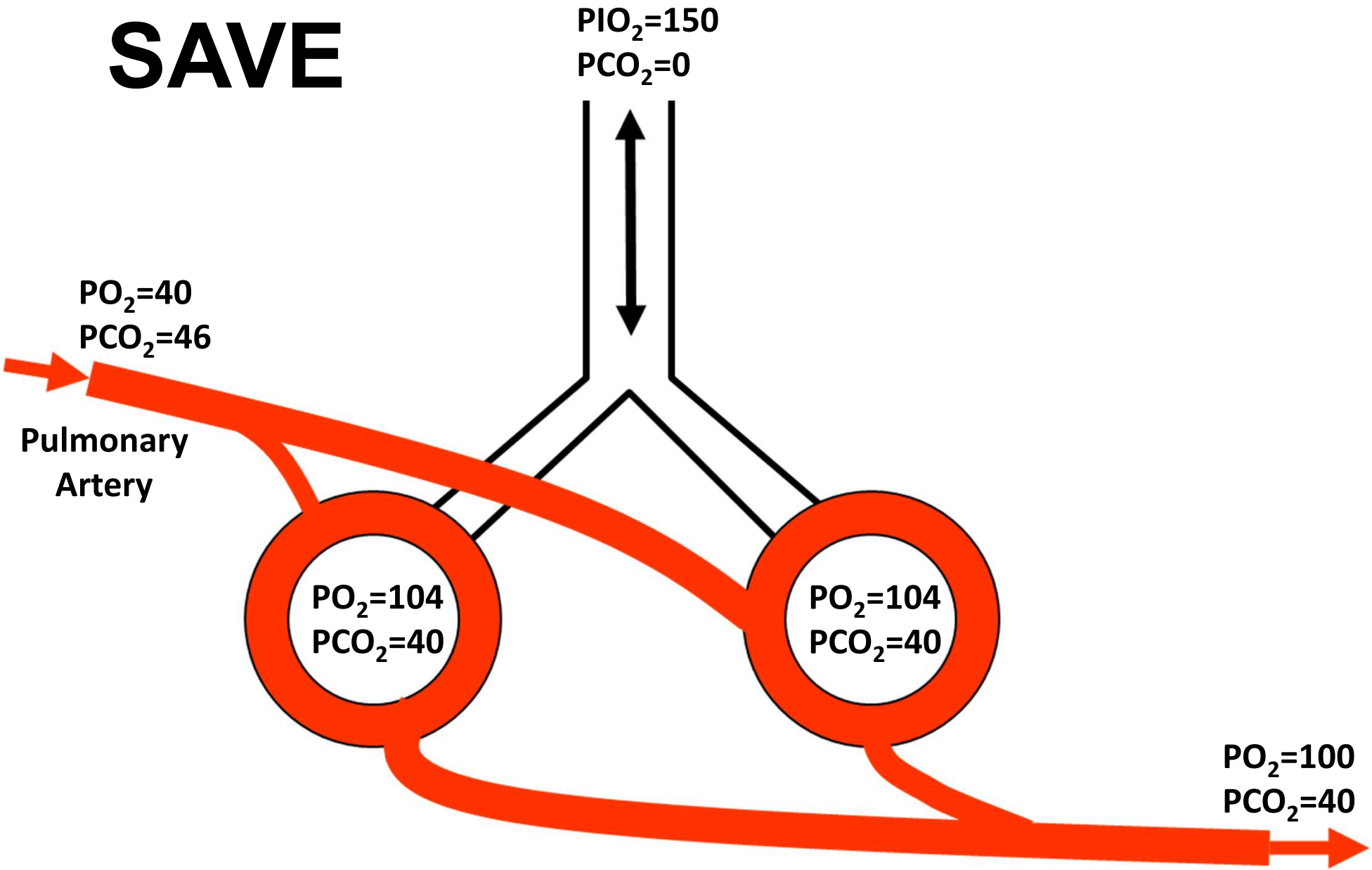
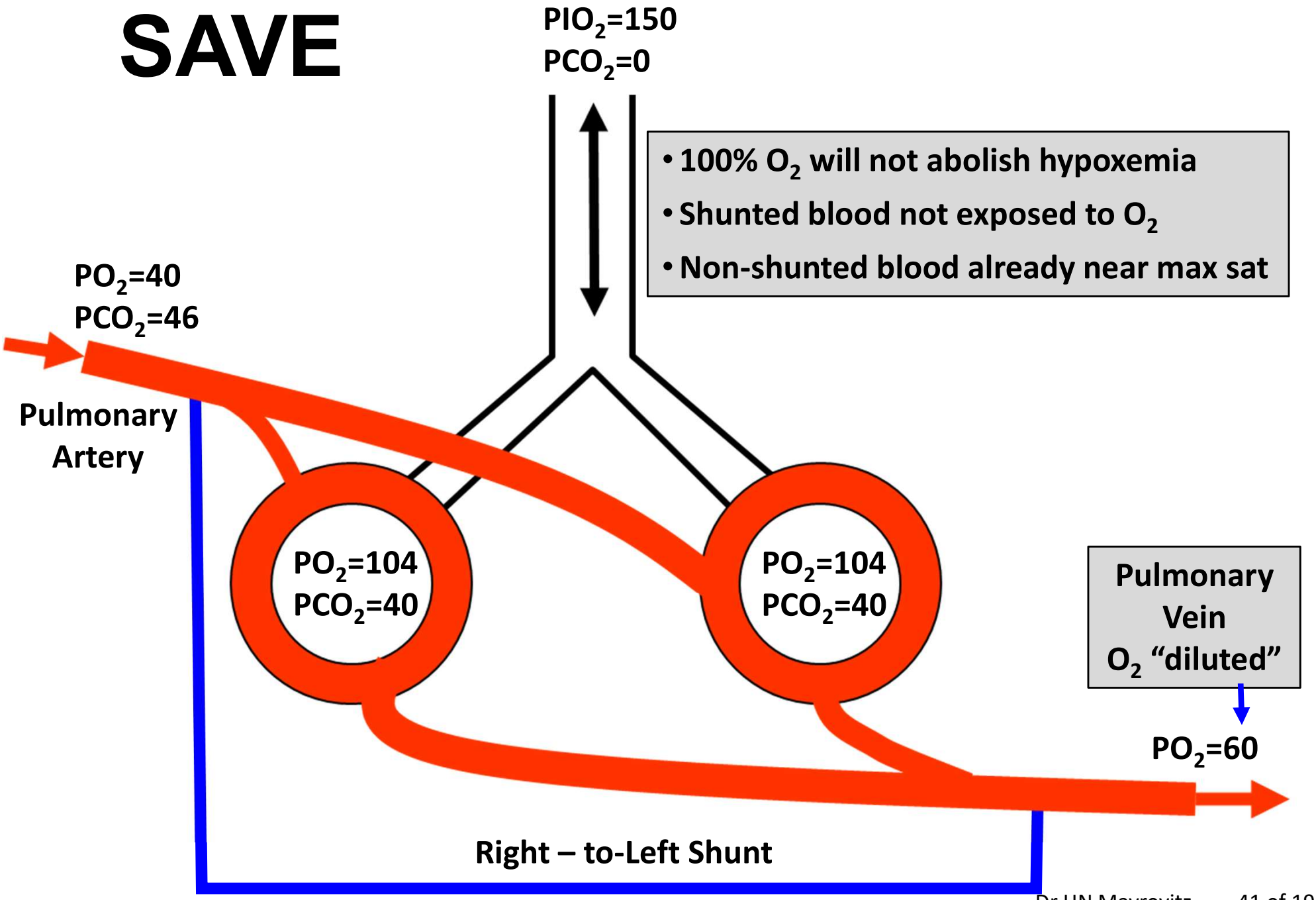
SAVE


PIO_2=150
PCO_2=0
100% O_2 WILL improve situation!
Reduced Ventilation
Low V'/Q
PO_2=40
PCO_2=46
Mucus Plug
Vasospasm
Pulmonary Artery
PO_2=77
PCO_2=45
PO_2=106
PCO_2=36
Reduced Alveolar Ventilation
Numbers are illustrative only
Low PO_2
Elevated PCO_2
PO_2=89
PCO_2=41

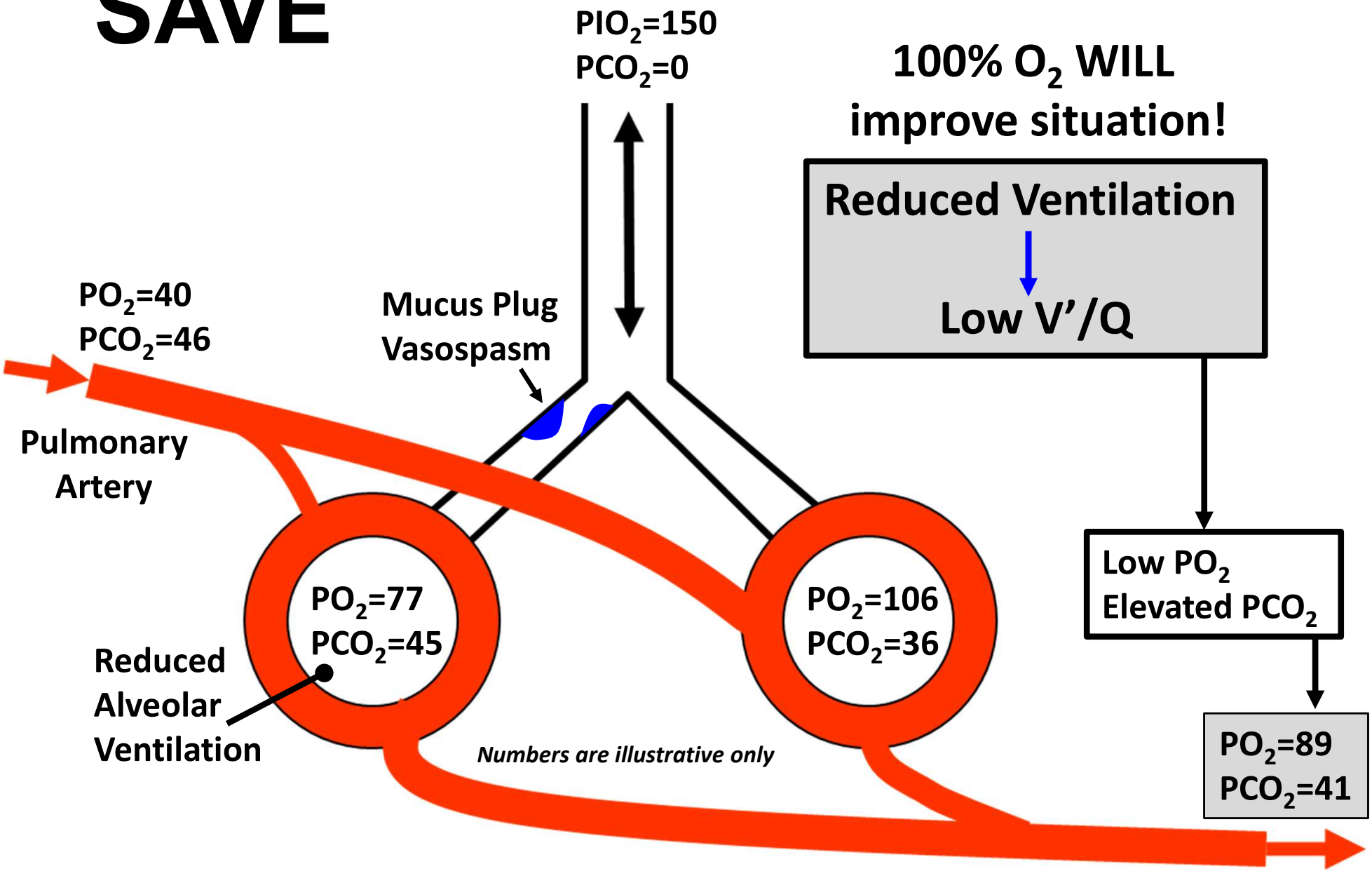
SAVE


PIO_2=150
PCO_2=0
PO_2=40
PCO_2=46
Pulmonary Artery
PO_2=104
PCO_2=40
PO_2=104
PCO_2=40
PO_2=100
PCO_2=40

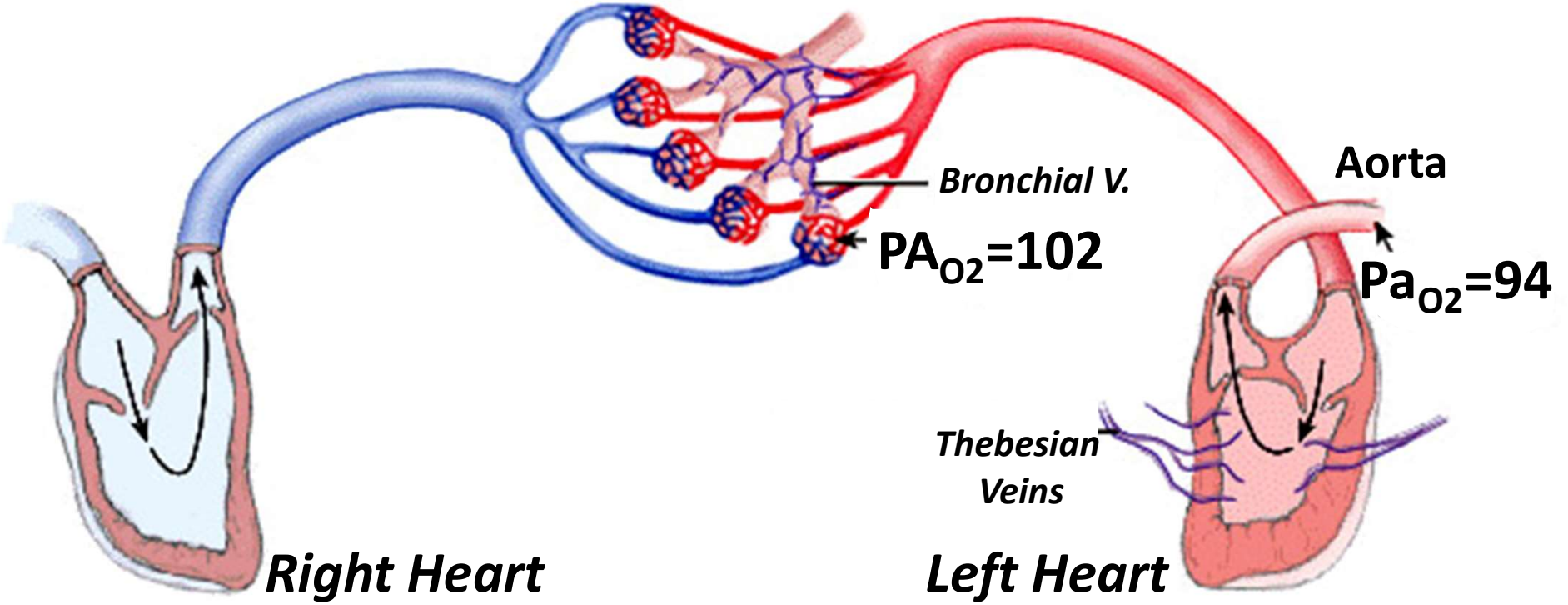
SAVE


PIO_2=150
PCO_2=0
• 100% O_2 will not abolish hypoxemia
• Shunted blood not exposed to O_2
• Non-shunted blood already near max sat
PO_2=40
PCO_2=46
Pulmonary Artery
PO_2=104
PCO_2=40
PO_2=104
PCO_2=40
Pulmonary Vein
O_2 "diluted"
PO_2=60
Right – to-Left Shunt

SAVE


PIO_2=150
PCO_2=0
100% O_2 WILL improve situation!
Reduced Ventilation
Low V'/Q
PO_2=40
PCO_2=46
Mucus Plug
Vasospasm
Pulmonary Artery
PO_2=77
PCO_2=45
PO_2=106
PCO_2=36
Reduced Alveolar Ventilation
Numbers are illustrative only
Low PO_2
Elevated PCO_2
PO_2=89
PCO_2=41

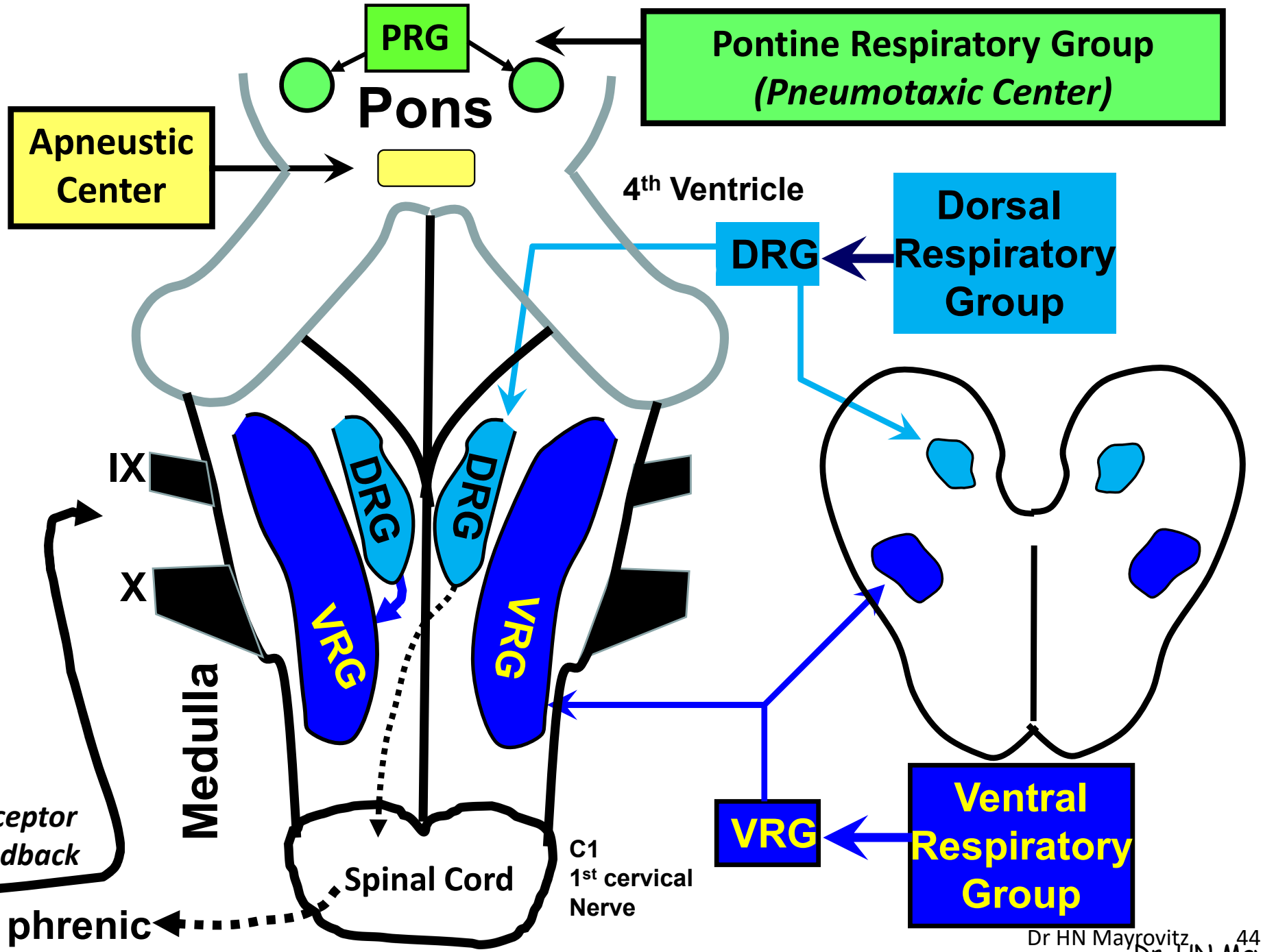
A-a Gradient Question to Ponder



Bill is 24 years old

A) What is his A-a gradient
B) is it within the normal range for his age?

Respiratory Cell Groups



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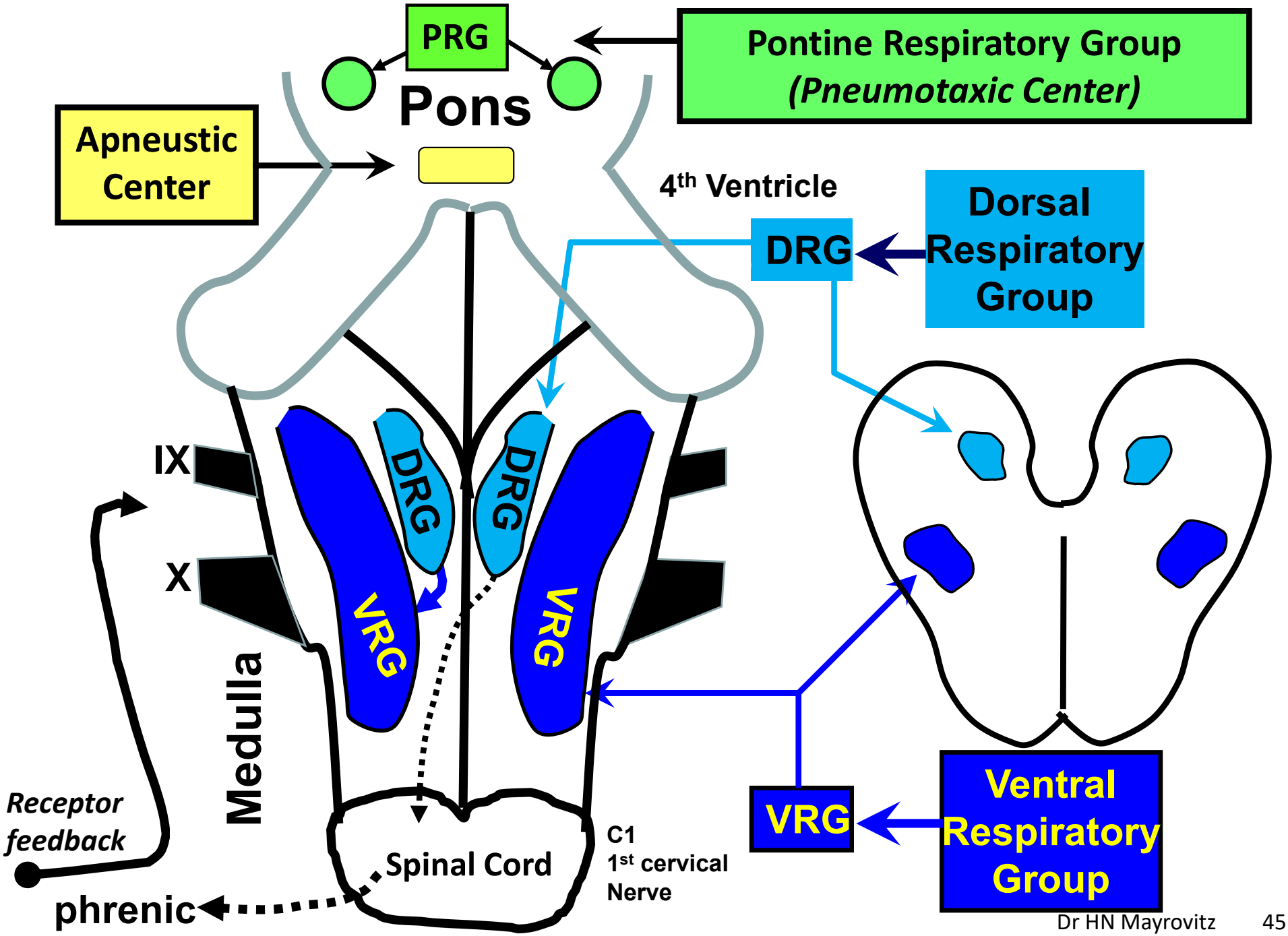
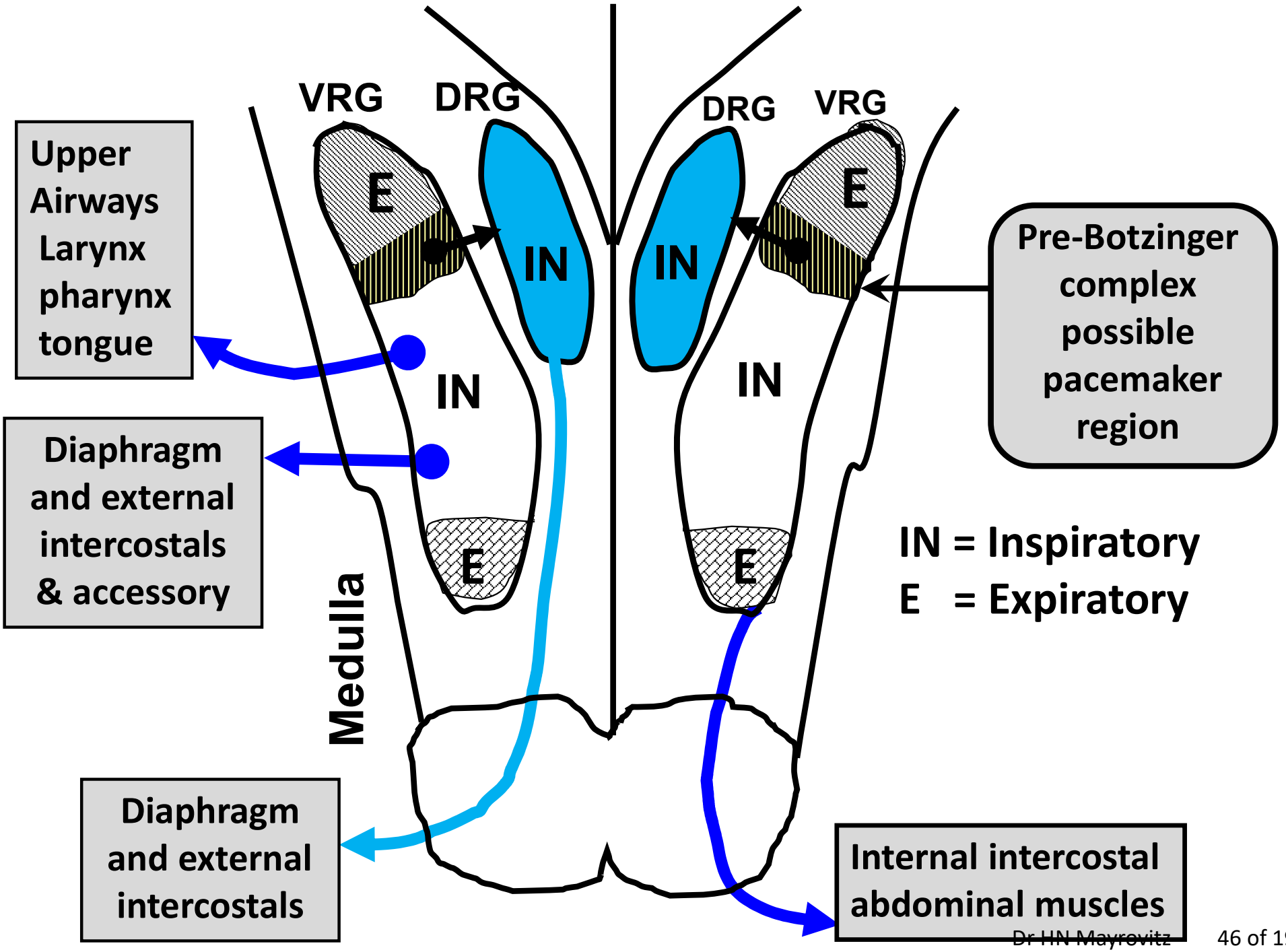
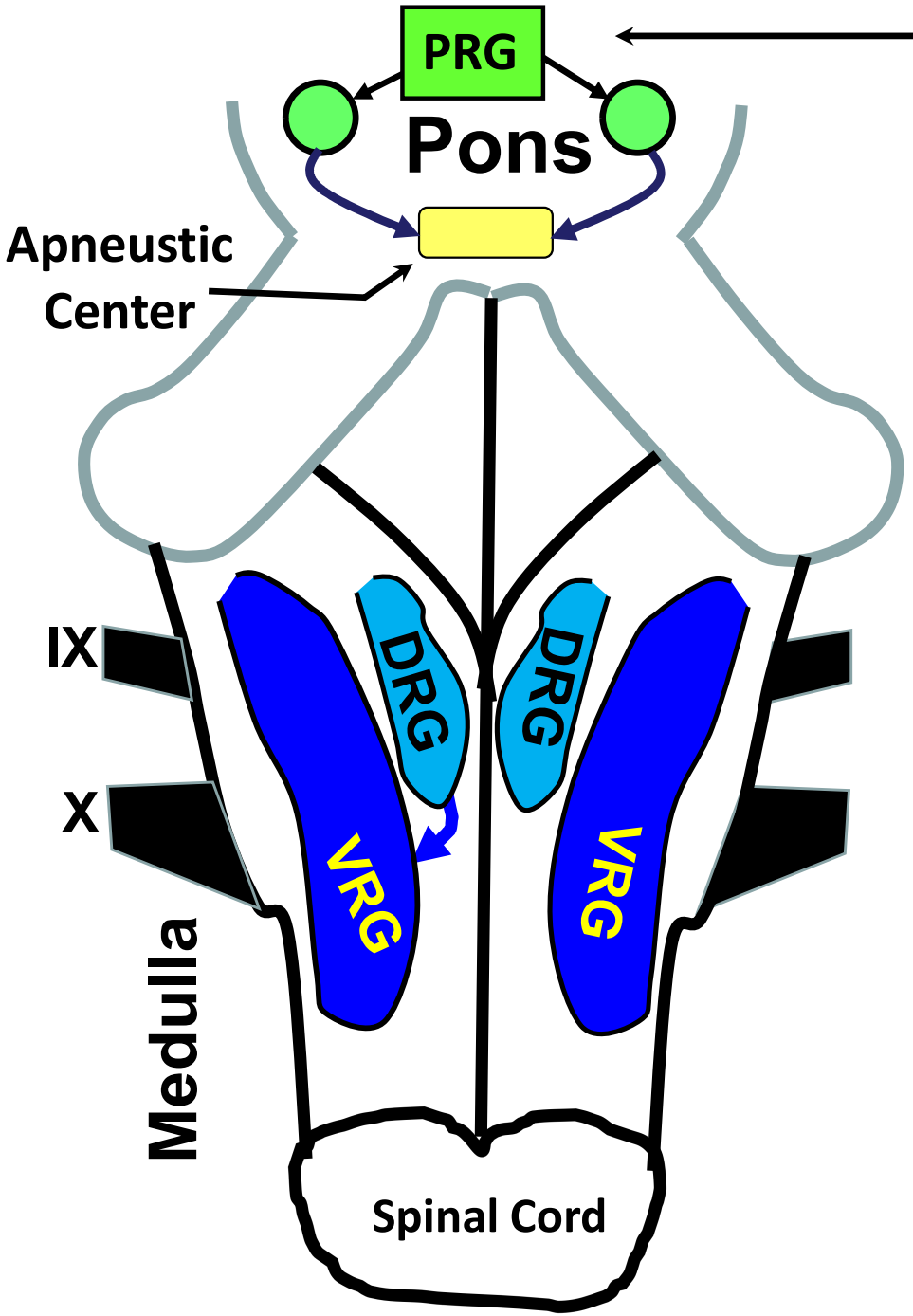
Respiratory Cell Groups



Medullary Respiratory Center



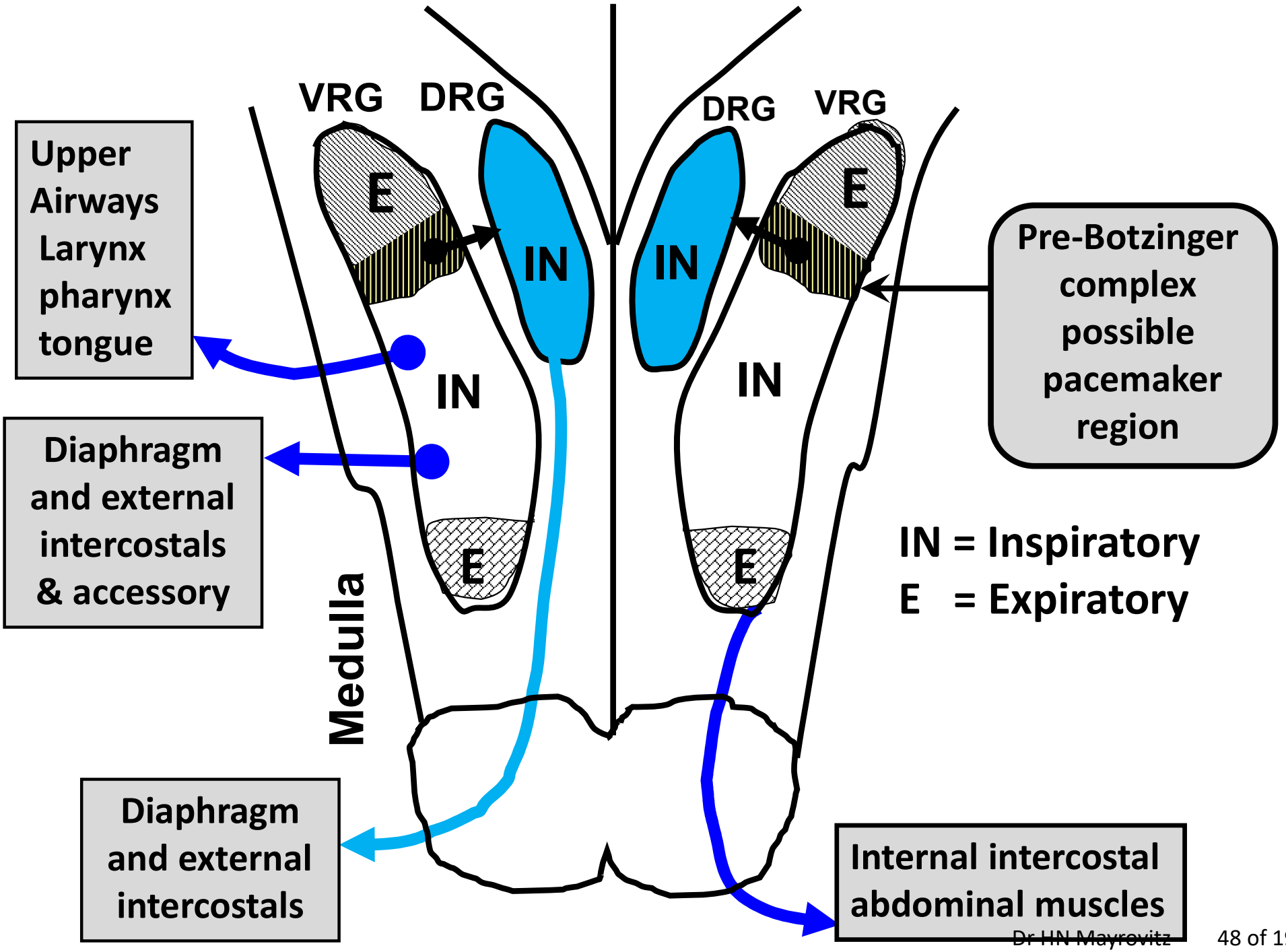
Pontine Respiratory Group (PRG)



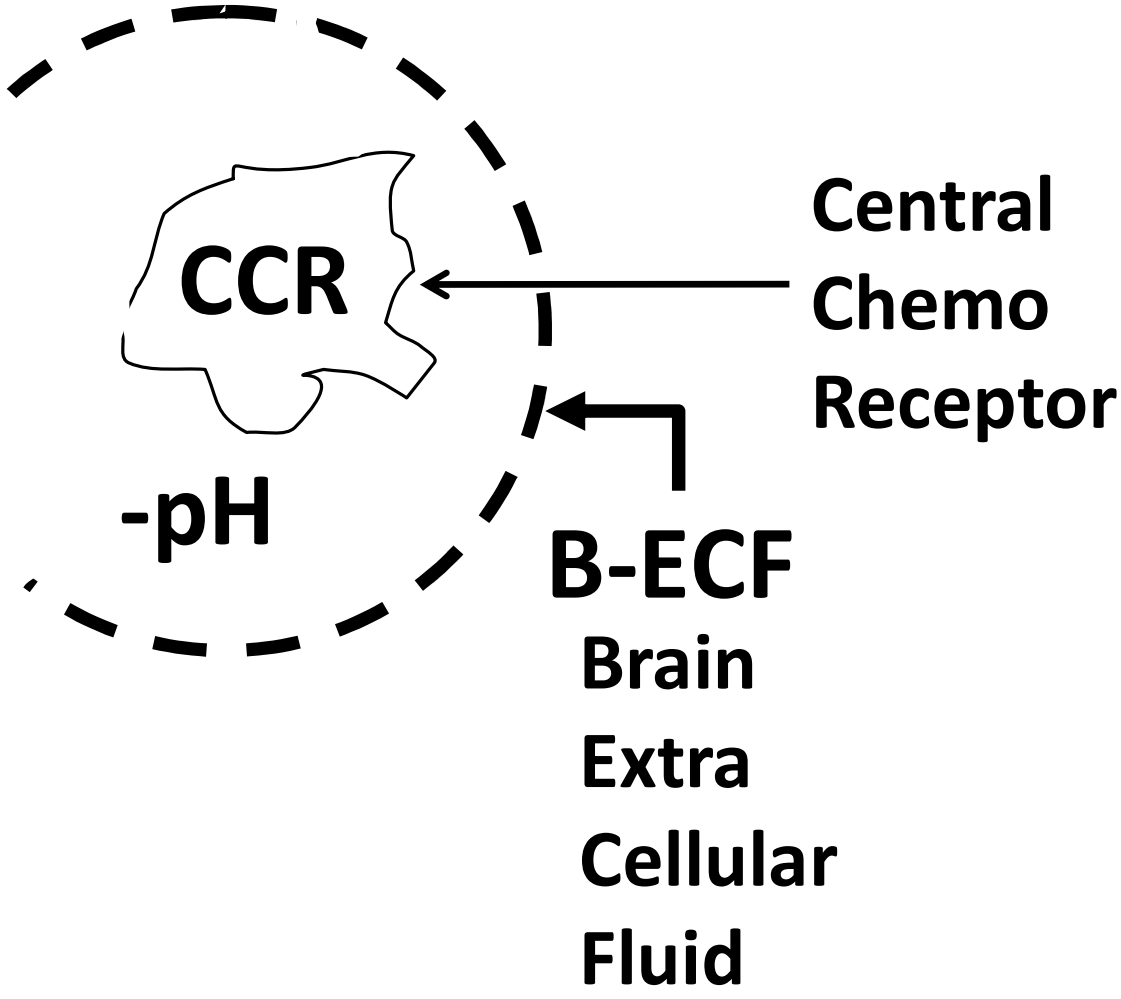
Pneumotaxic Center

- In upper pons
- Some neurons active during inspiration & some in expiration
- Important role in *switching* off/limiting inspiration
- If damaged leads to apneusis: prolonged inspiratory spasms with short intervals of expiration
- Also fine-tunes breathing based on receptor feedback

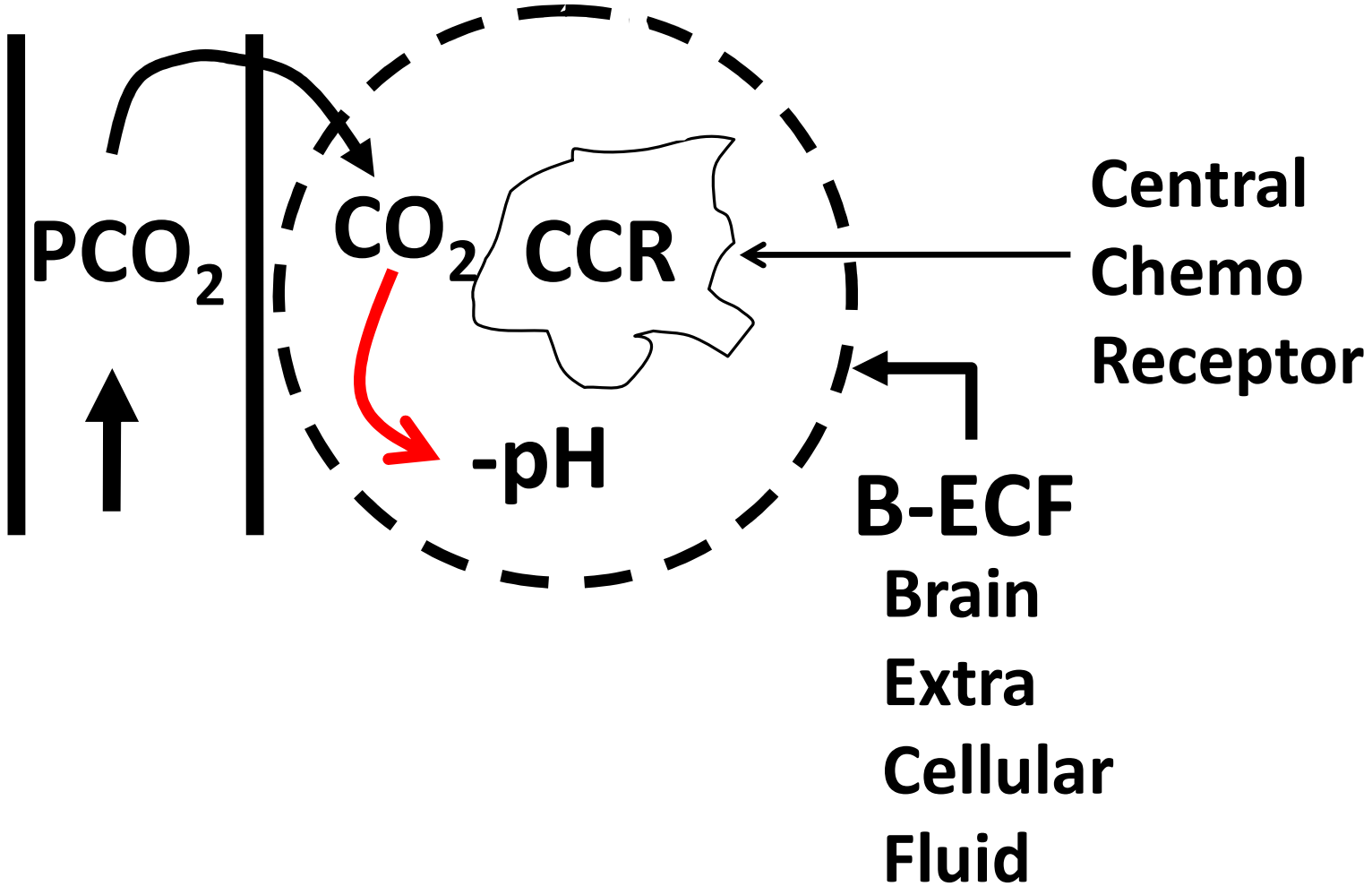
Medullary Respiratory Center



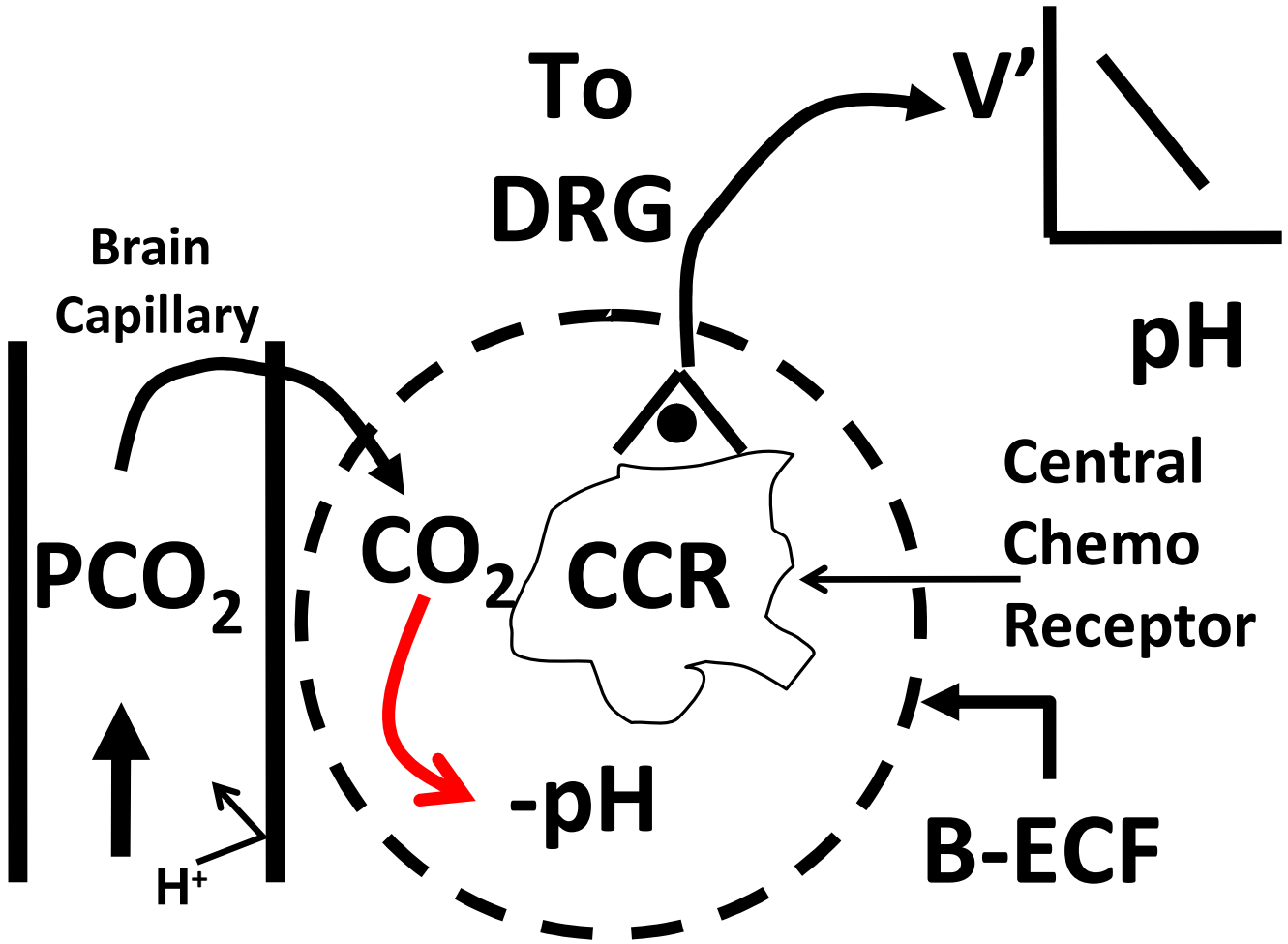
Central Chemoreceptor Function Overview



Central Chemoreceptor Function Overview



Central Chemoreceptor Function Overview



B-ECF
Brain Extracellular Fluid

SUMMARY: Central Chemoreceptors (CCR)

- CCR in brain parenchyma bathed in brain extracellular fluid/CSF
- If blood gases and pH near normal CCR are main control of ventilation
- CCR are sensitive to arterial hypercapnia (and associated fall in pH)
- CCR actually sense pH (H^+) around receptor neurons bathed in CSF
- pH changes may occur due to:
  1) increased cerebral blood CO_2 diffusing across the blood brain barrier resulting in a rapid (60 sec) decrease in the pH of CSF
  2) decreased pH of brain or CSF not due to changes in Pa_{CO2} (delayed)
- CCR do not respond to hypoxia
- CCR and PCR both affect ventilation response to increased CO_2 levels