Ventilation-Perfusion Relationship ($\dot{V}A/\dot{Q}$)

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Objectives:

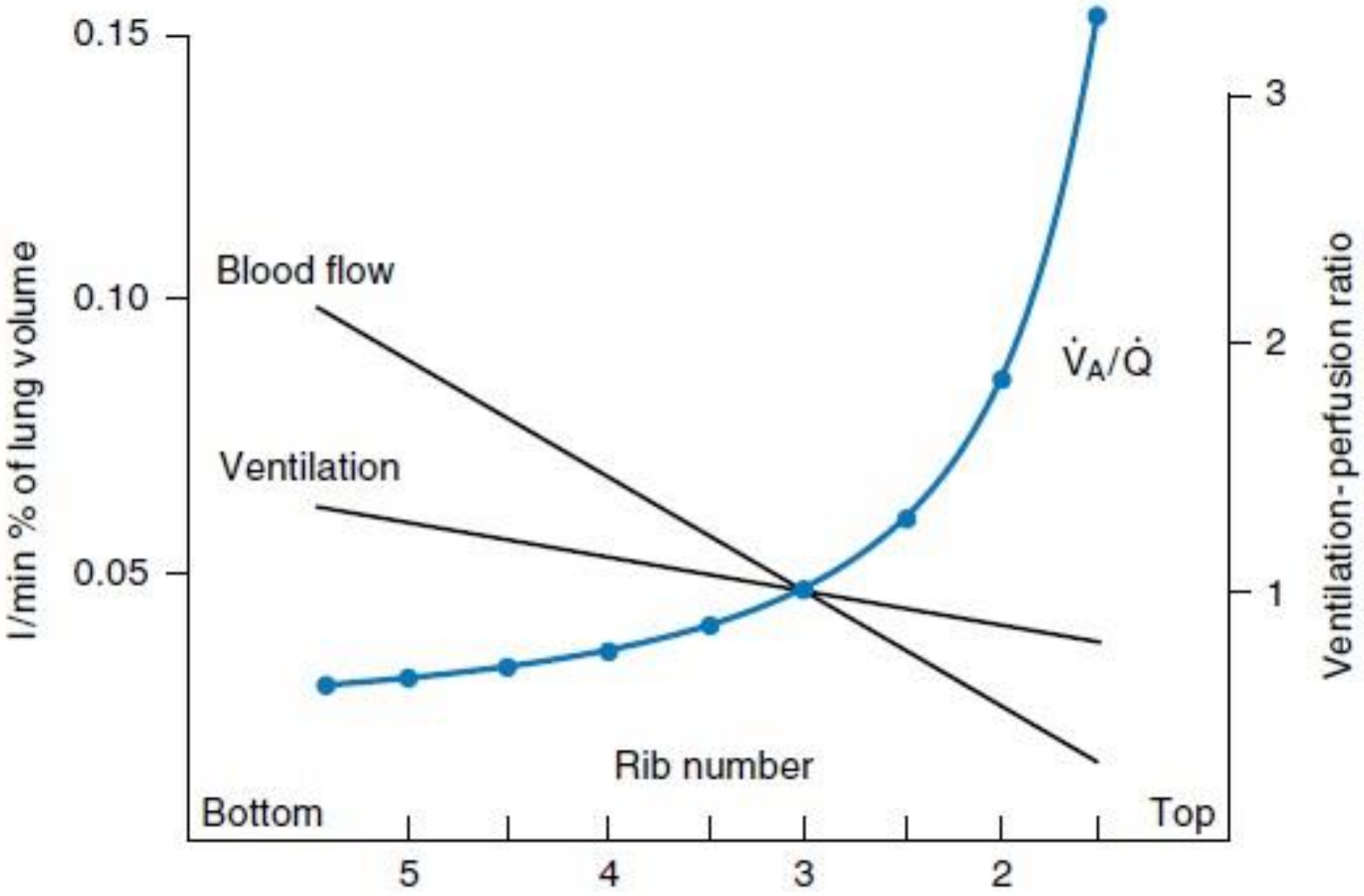
1. Describe how the ventilation/perfusion (V/Q) ratio of an alveolar-capillary lung unit determines the PO_2 and PCO_2 of the blood emerging from that lung unit.
2. Identify the average V/Q ratio in a normal lung and explain how V/Q is affected by the vertical distribution of ventilation and perfusion in the healthy lung.
3. Describe the normal relative differences from the apex to the base of the lung in alveolar and arterial PO_2, PCO_2, pH, and oxygen and carbon dioxide exchange.
4. Predict how the presence of abnormally low and high V/Q ratios in a person's lungs will affect arterial PO_2 and CO_2.
5. Define right-to-left shunts, anatomic and physiological shunts, and physiologic dead space (wasted ventilation).
6. Describe the airway and vascular control mechanisms that help maintain a normal ventilation/perfusion ratio.
7. Characterize the pathophysiology of abnormal ventilation perfusion inequality.

Definition: Is the ratio of the alveolar ventilation to the pulmonary blood flow. The $\dot{V}_A/\dot{Q}$ for the whole lung at rest is about 0.8-0.9.

$\dot{V}_A/\dot{Q}$ = alveolar ventilation/cardiac output

$\dot{V}_A/\dot{Q}$ = (4.5-5 L/min) / (5-5.5 L/min)

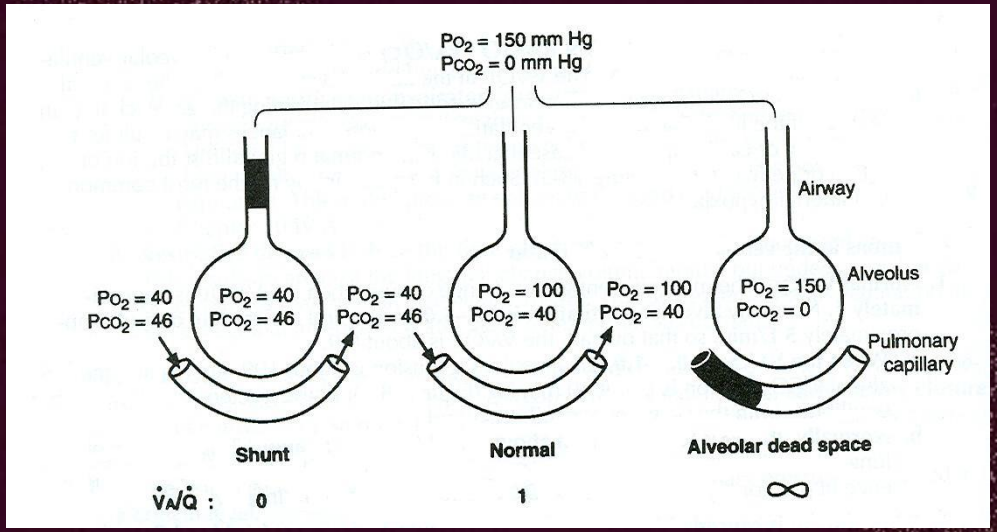
In upright position the ratio is less than 1 (about 0.6) at the base of the lungs, and greater than 1 (about 2.5) at the apex. The most efficient gas exchange occurs when the V_A/Q is approximately 1.



Distribution of ventilation and blood flow down the upright lung
Note that the ventilation-perfusion ratio decreases down the lung

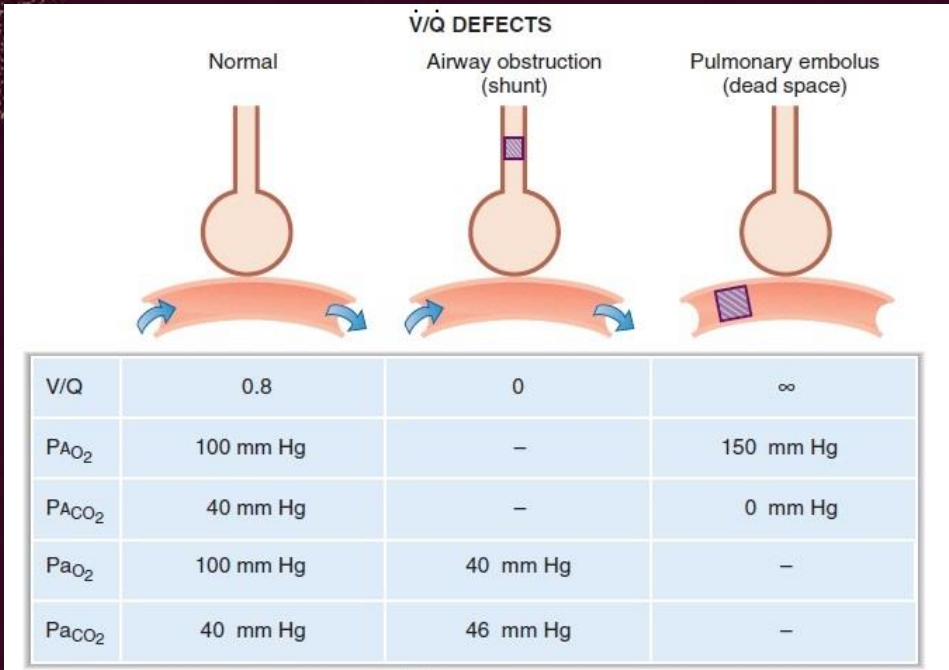
Effect of V_A/Q on alveolar gas concentration:

1. When V_A/Q equals zero, this means that there is no alveolar ventilation (=shunt). The air in the alveolus equilibrates with blood oxygen and CO_2. Since the perfusing blood is the venous blood returning to the lungs from the systemic circulation, the alveolar PO_2 sets at 40 mmHg and PCO_2 at 45 mmHg.



Effect of V_A/Q on alveolar gas concentration:

2. When V_A/Q equals infinity, this means that there is no capillary blood flow (= alveolar dead space). The alveolar air becomes equal to humidified inspired air (PO_2 of 149 mmHg and a PCO_2 of 0 mmHg).
3. When V_A/Q is normal; alveolar PO_2 is normal (104 mmHg) and PCO_2 is 40 mmHg.



V/Q	0.8	0	∞
$P_{A_{O_2}}$	100 mm Hg	–	150 mm Hg
$P_{A_{CO_2}}$	40 mm Hg	–	0 mm Hg
$P_{a_{O_2}}$	100 mm Hg	40 mm Hg	–
$P_{a_{CO_2}}$	40 mm Hg	46 mm Hg	–

FIGURE Effect of ventilation/perfusion ($\dot{V}/\dot{Q}$) defects on gas exchange. With airway obstruction, the composition of systemic arterial blood approaches that of mixed venous blood. With pulmonary embolus, the composition of alveolar gas approaches that of inspired air. $P_{A_{O_2}}$ = alveolar P_{O_2}; $P_{A_{CO_2}}$ = alveolar P_{CO_2}; $P_{a_{O_2}}$ = arterial P_{O_2}, $P_{a_{CO_2}}$ = arterial P_{CO_2}.

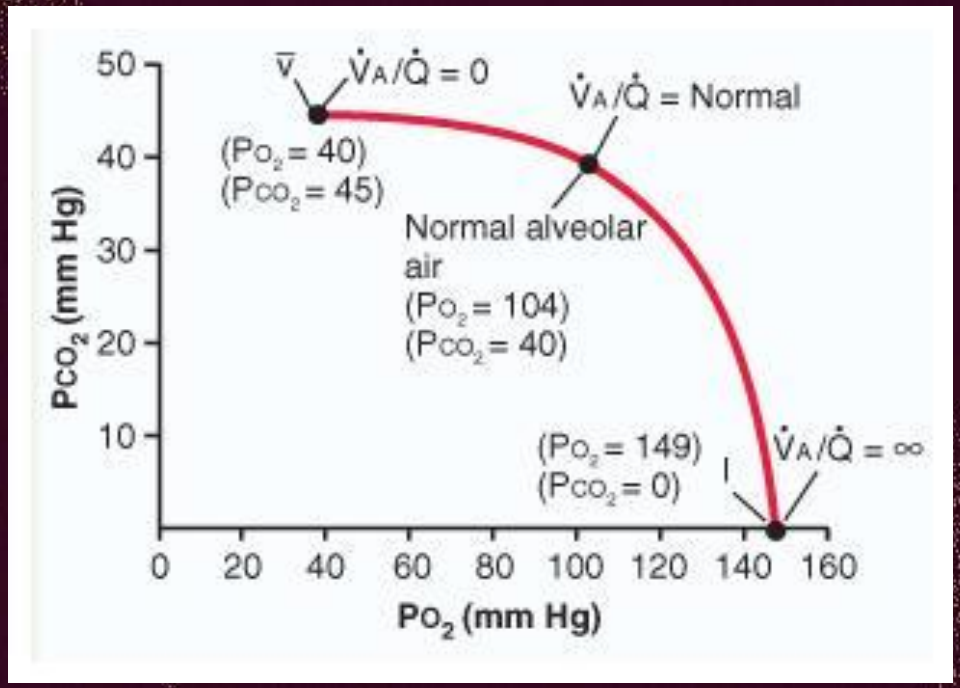
Note:

The term V/Q mismatch is more appropriate to be used to describe conditions fall in between these two extremes.

Effect of V_A/Q on alveolar gas concentration:

Note:

1. There can be marked decrease in oxygen tension with only a minimal rise in the CO_2 tension. (See the graph)
2. Many of the conditions that cause mismatching of ventilation and perfusion involve both dead air space and shunt. In chronic obstructive lung disease, for example, there may be impaired ventilation in one area of the lung and impaired perfusion in another area



Physiologic shunt and physiologic dead space:

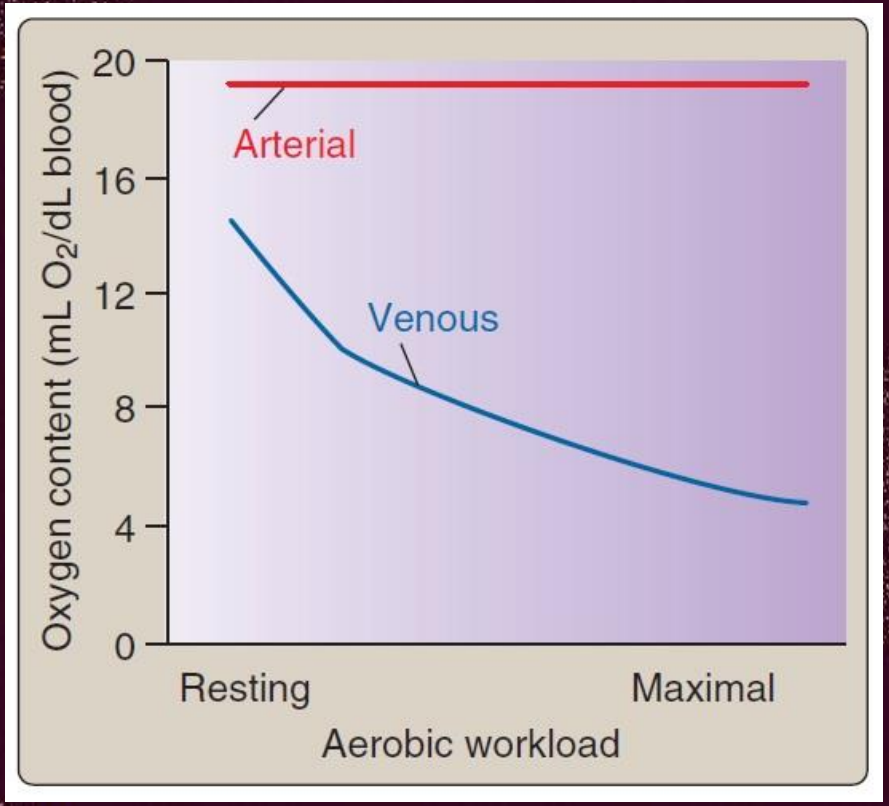
- When V_A/Q is *below* normal this means that there is **inadequate ventilation** needed for full oxygenation, or **excessive blood flow** → some of the venous blood remains unchanged (= shunted blood).
- The total amount of shunted blood/minute is called **Physiologic shunt**. Physiologic shunt → decrease in the overall arterial PO_2 and oxygen content.
- When V_A/Q is *greater* than normal this means that some of the ventilated air is wasted (alveolar dead space) → ↑ **physiologic dead space** → CO_2 retention and hypoxia → compensated hyperventilation → correction of hypercapnea only.

Respiratory responses to exercise:

- Cardiac output increases approximately linearly with work level.
- The change in cardiac output (in liter/min) is only about a quarter of the increase in ventilation.
- Ventilation increases linearly with O_2 uptake until the ventilatory (or anaerobic) threshold is reached after which ventilation increases more rapidly.
- Despite the increase in ventilation, the difference between inspired and expired O_2 concentrations does not change.
- In normal subjects, the amount of ventilation-perfusion inequality decreases during moderate exercise because of the more uniform topographical distribution of blood flow.

PaO_2 remains stable during exercise because of:

- ↑ Ventilation : maintains a steep PO_2 gradient across the blood-gas barrier
- ↑ Tidal volume : improves V_A/Q ratio at the lung base
- ↑ Pulmonary flow : recruits pulmonary capillaries and increases blood-gas interface surface area



Summary of Respiratory Responses to Exercise

Parameter	Response
O_2 consumption	↑
CO_2 production	↑
Ventilation rate	↑ (Matches O_2 consumption/CO_2 production)
Arterial P_{O_2} and P_{CO_2}	No change
Arterial pH	No change in moderate exercise
	↓ In strenuous exercise (lactic acidosis)
Venous P_{CO_2}	↑
Pulmonary blood flow (cardiac output)	↑
$\dot{V}/\dot{Q}$ ratios	More evenly distributed in lung

$\dot{V}/\dot{Q}$ = ventilation/perfusion ratio.

Test Question 1:

Q. The apex of the upright human lung compared with the base has?

A. A higher alveolar PO_2 .

B. A higher ventilation.

C. A lower pH in end-capillary blood.

D. A higher blood flow.

E. Smaller alveoli.

Pathophysiology:

A mismatch in ventilation and perfusion can arise due to either reduced ventilation of part of the lung or reduced perfusion.

Reduced Perfusion of the Lungs

- This finding is typically associated with **pulmonary embolism**. Ventilation is wasted, as it fails to oxygenate any blood.
- The V_A/Q ratio is high with decreased $PACO_2$ and increased PAO_2, because of the increased dead space ventilation.
- The PaO_2 is reduced and thus also the peripheral oxygen saturation is lower than normal, leading to tachypnea and dyspnea.

Pathophysiology:

Reduced Ventilation of the Lungs

- Reduced ventilation can occur for a number of reasons.
- This includes **pneumonia**, whereby the alveoli are filled with exudate, limiting the ability to maintain ventilation.
- **Asthma** and **COPD** may also result in a reduction in ventilation, as well as **respiratory distress syndrome** of the newborn, whereby **reduced surfactant** production results in multiple collapsed alveoli, limiting its ventilating capability.
- The effect of reduced ventilation is hypoxemia. However, as the rest of the lung can still remove CO_2, hypercapnia does not occur.
- In severe asthmatics, **aerosolized bronchodilator therapy**, by increasing blood flow to potentially underventilated lung units, increases shunt and arterial desaturation. This may lead to worsening hypoxemia following bronchodilator therapy.
- Drugs, such as anesthetics can lower V_A/Q ratios, as there is impaired matching of ventilation and perfusion during **anesthesia**.

Pathophysiology:

- The pathophysiology in some pulmonary diseases is complex, and can be associated with multiple pathologies.
- Prolonged smocking can end in chronic obstructive lung diseases (e.g. **emphysema**)

bronchial obstruction →↓ ventilation → V_A/Q ≈ zero
(i.e. shunt)

Emphysema

enlarged alveolar sacs → wasting of ventilated air
(↑physiologic dead space)

- The effectiveness of gas exchange can be reduced in emphysema to one-tenth the normal value.

Test Question 2:

Q. If the ventilation-perfusion ratio of a lung unit is decreased by partial bronchial obstruction while the rest of the lung is unaltered, the affected lung unit will show?

A. Increased alveolar PO_2.

B. Decreased alveolar PCO_2.

C. No change in alveolar PN_2.

D. A rise in pH of end-capillary blood.

E. A fall in oxygen uptake.