PULMONARY AND ALVEOLAR VENTILATION ($\dot{V}_A$)

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Lecture objectives:

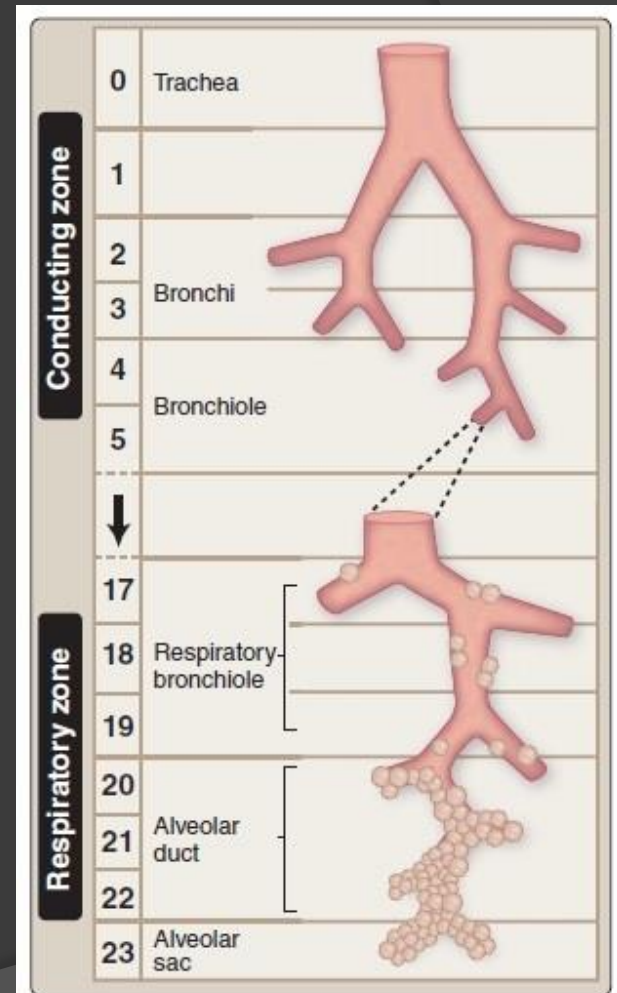By the end of this lecture the student should be able to:

1. Describe pulmonary and alveolar ventilation.

2. Define and contrast the following terms: anatomic dead space, physiologic dead space, total minute ventilation and alveolar minute ventilation.

3. Define and contrast the relationships between alveolar ventilation and the arterial PCO_2 and PO_2.

4. Predict the effects of alterations of alveolar ventilation on alveolar carbon dioxide and oxygen levels.

5. Describe the effect of dead space on alveolar ventilation.

6. Understand Fowler's method of measuring the anatomic dead space. And calculate the volume of dead space in a lung using the Bohr equation.

7. Describe and explain the mechanism of regional differences in alveolar ventilation in the normal lung.

Alveolar Ventilation ($\dot{V}_A$)

The rate at which new air reaches gas exchange areas (with **pulmonary blood**). These areas include the alveoli, alveolar sacs, alveolar ducts, and respiratory bronchioles.

The total area of the alveolar walls in contact with capillaries in both lungs is about 70 m^2

0	Trachea
1	
2	Bronchi
3	
4	Bronchiole
5	
17	
18	Respiratory bronchiole
19	
20	
21	Alveolar duct
22	
23	Alveolar sac

Conducting zone
Respiratory zone

The **Respiratory zone** houses the blood–gas interface

Dead space (V_D): The space in the respiratory passages where gas exchange <u>**does not**</u> occur.

1. The **anatomic** dead space: is the volume of gas that occupies the airways, which are called the conducting zone. The conducting zone <u>**does not**</u> participate in gas exchange because of the thickness of the airway walls (nose, pharynx, and trachea).

The functions of the anatomic dead space is to

- warm the inspired gas to the body temperature
- saturate the gas with H_2O vapor that will slightly dilute the O_2 and N_2 concentrations and prevent drying of the alveolar surface
- assist in the removal of foreign materials

2. The **total (physiologic)** dead space: includes the anatomic dead space and alveolar dead space (alveoli that <u>**do not**</u> receive any blood flow and therefore <u>**do not**</u> participate in gas exchange). It is approximately 2ml/kg or 150 ml in an young adult, roughly a third of the tidal volume. This value increases slightly with age.

Note:

- Air in the dead space is the first part of air to be removed on expiration.

- Normally, the anatomic and physiologic dead spaces are nearly equal, significant difference only occurs in patients with lung diseases because of the increase in alveolar dead space.

VOLUMES

FLOWS

Tidal volume — Total ventilation
500 ml 7,500 ml/min

Anatomic dead space — Frequency
150 ml 15/min

Alveolar ventilation
5,250 ml/min

Alveolar gas —
3,000 ml

$\div \simeq 1$

Pulmonary — Pulmonary
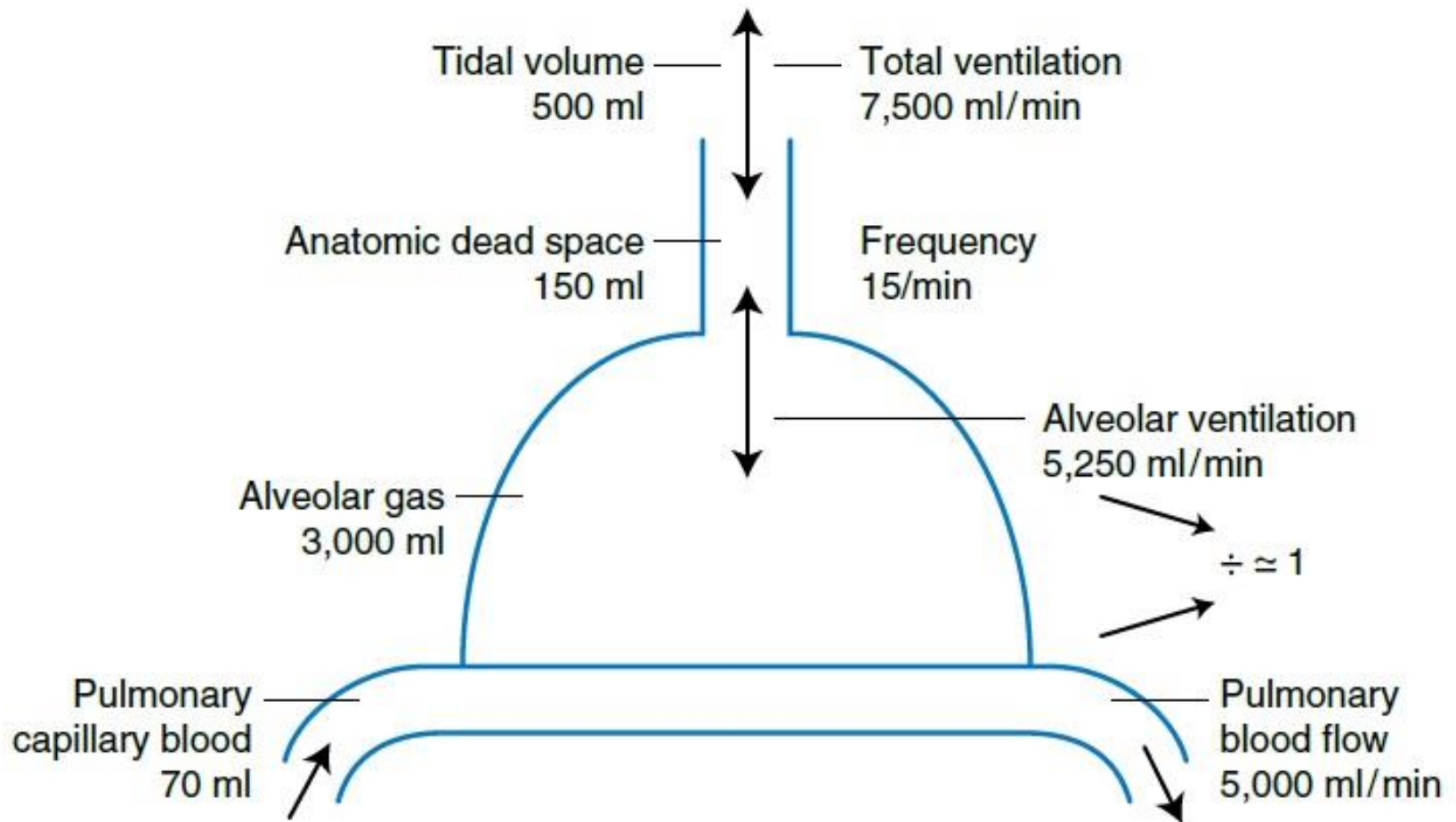capillary blood blood flow
70 ml 5,000 ml/min

Diagram of a lung showing typical volumes and flows. There is considerable variation around these values depending on the size and gender of the patient.

Methods of measuring the total dead space:

1. Bohr's method: depends on determining CO_2 tensions in the mixed expired and alveolar gases.

$$V_D = V_T \cdot (1 - \frac{P_{ECO_2}}{P_{ACO_2}})$$

V_D = Volume of dead space

V_T = Tidal Volume

P_{ECO_2} = CO_2 tension in mixed expired gas

P_{ACO_2} = CO_2 tension in alveolar gas = P_{aCO_2} in healthy individuals

Note: Bohr's method measures the volume of the lung that does not eliminate CO_2 (i.e. physiologic dead space) if P_{aCO_2} is used.

# Methods of measuring the total dead space:

2. Fowler's method: uses expired N_2 as an indicator. The subject inspires a single deep breath of 100% oxygen, and then the expired N_2 is measured during the subsequent full expiration through a rapidly recording nitrogen meter.

$$V_D = \frac{\text{Gray area} \times V_E}{\text{Pink area} + \text{Gray area}}$$

V_D = The volume of dead space gas

V_E = The total volume of expired air

*Note*: Fowler's method reflects the morphology of the lung (i.e. anatomic dead space).
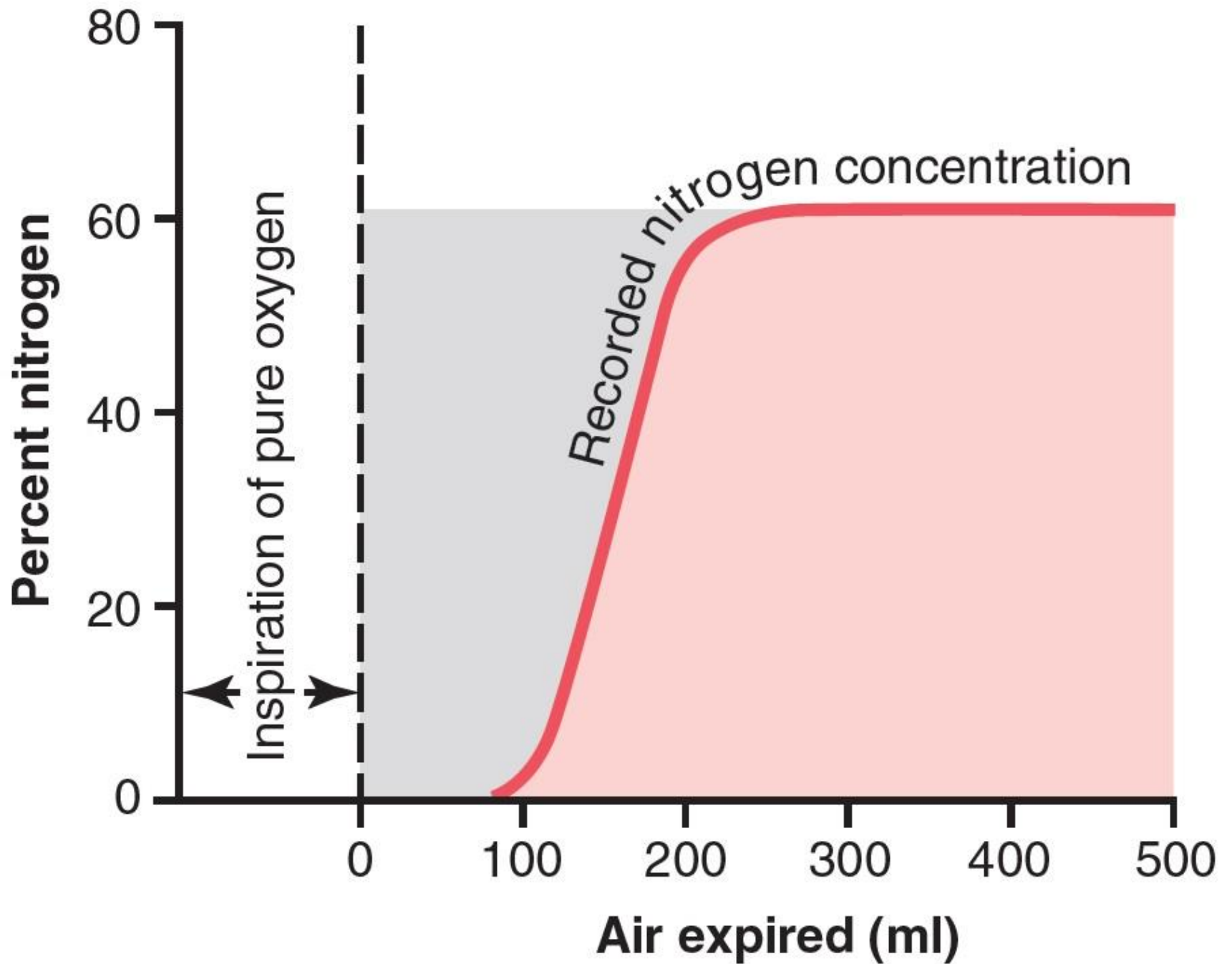
Measurement of Alveolar Ventilation ($\dot{V}_A$)

To measure the volume of new air that participates in the exchange of O_2 and CO_2 per minute the following equation is used;

$$\dot{V}_A = f \cdot (V_T - V_D)$$

- With a respiratory rate of 12-15 /min, $\dot{V}_A$ is about 4200-5000 ml/min.

- Adequate alveolar ventilation is critical because it determines O_2 and CO_2 tensions in the lungs.

Regional differences in ventilation
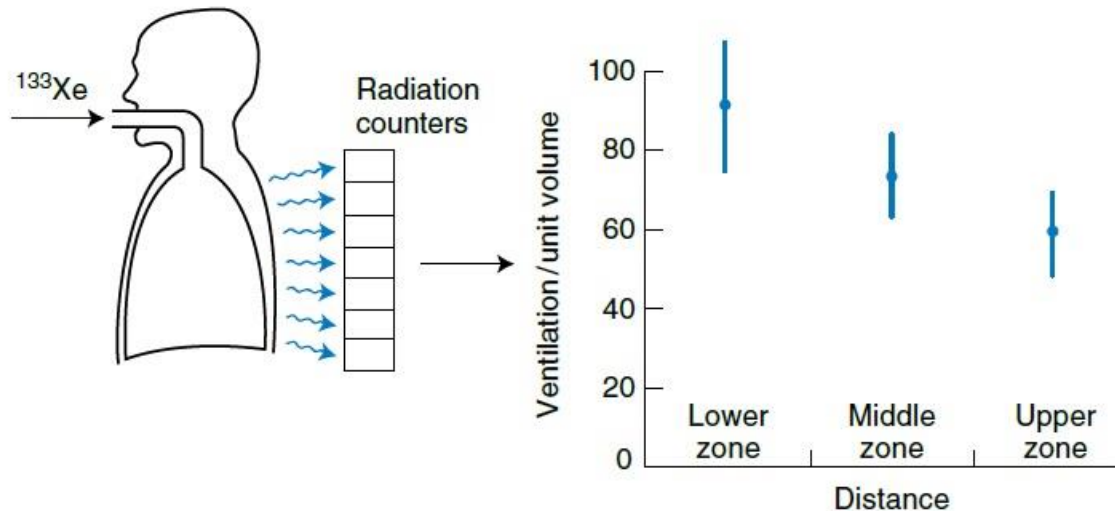
Measurement of regional differences in ventilation with radioactive xenon. When the gas is inhaled, its radiation can be detected by counters outside the chest. Note that the ventilation decreases from the lower to upper regions of the upright lung.

⦿ In upright position, ventilation per unit volume is greatest <u>near the bottom of the lung</u> and becomes progressively smaller toward the top.
⦿ In the supine position, this difference disappears, with the result that apical and basal ventilations become the same. However, the ventilation of the lowermost (posterior) lung exceeds that of the uppermost (anterior) lung.
⦿ The lower regions of the lung are better ventilated (alveoli expand more) than the upper regions because of the **effects of gravity on the lung**.

Alveolar O_2 and CO_2 tensions

1. Alveolar CO_2 tension ($\textbf{P}_{\textbf{ACO}_2}$) is equal to arterial CO_2 tension because of the high diffusibility of CO_2. It is about 40 ± 4 mmHg. Regulation of this tension is <u>very important</u>.

   - With constant rate of CO_2 production, there is **inverse relationship** between alveolar ventilation and P_{aCO_2}. If alveolar ventilation is halved, P_{aCO_2} will double. So;

   - **Hyperventilation** (with constant rate of CO_2 production) → **Hypocapnia** (↓alveolar CO_2 tension) → **Respiratory alkalosis**

   - **Hypoventilation** → **Hypercapnia** (↑alveolar CO_2 tension), which if it exceeds 45 mmHg → **Respiratory acidosis**. Hypercapnia can lower the O_2 tension in the alveoli by displacing the O_2.

Note: It is important to differentiate between *Hyperventilation* and *Hyperpnea*. Hyperpnea is increased alveolar ventilation with <u>no change</u> in P_{aCO_2} as in exercise and fever. This is due to increased CO_2 production by metabolism.

Alveolar O_2 and CO_2 tensions

2. Alveolar O_2 tension ($\mathbf{P_{AO_2}}$). Inspiration brings fresh air into the alveoli, which normally maintains the alveolar O_2 tension at about 100 mmHg.

    An increase in alveolar ventilation $\rightarrow P_{aO_2}$ will increase in non-linear relationship, as more oxygen is removed from the alveolar gas per unit time than CO_2 is added.

$\mathbf{P_{AO_2}}$ is affected by;

- Alveolar CO_2 tension
- Changes in barometric pressure
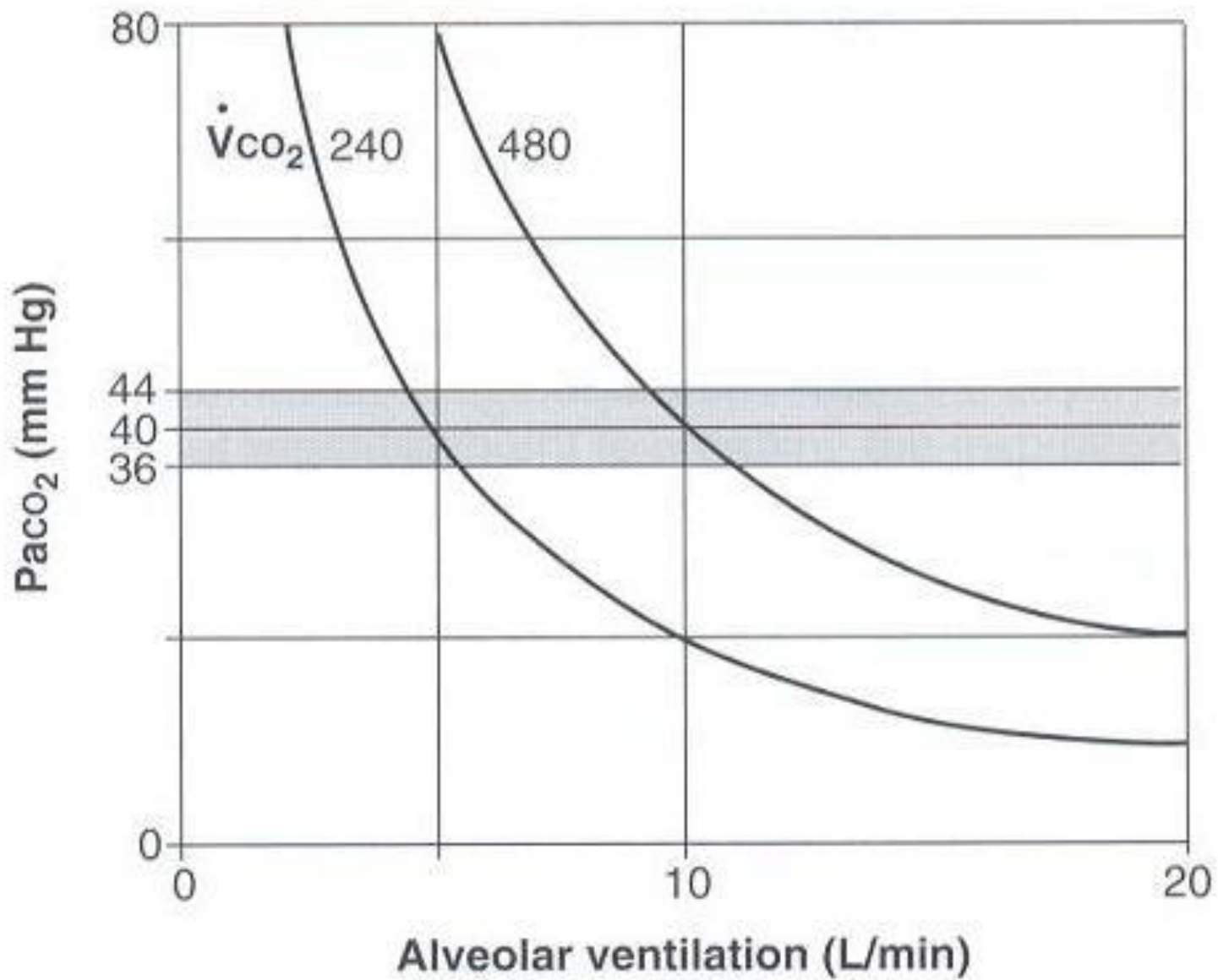- Fraction of O_2 inspired

$\mathbf{P_{AO_2}}$ can be calculated using the ***Alveolar Gas Equation***

$$P_{AO_2} = F_I O_2 (P_{atm} - P_{H_2O}) - \frac{P_{aCO_2}}{R}$$

$F_I O_2$ = Fraction of oxygen in the inspired gas

R = 0.8 (respiratory exchange ratio)

Note: Water vapor pressure at body temperature of 37°C is 47 mmHg regardless of the external barometric pressure.

$\dot{V}_{CO2}$ = amount of carbon dioxide eliminated in ml/min

Test Question:

Respiratory dead space:

A. Saturates inspired air with water vapor before it reaches the alveoli.

B. Removes all particles from inspired air before it reaches the alveoli.

C. Decreases when blood catecholamine levels rise.

D. Decreases during a deep inspiration.

E. Increases during a cough.