Physiol-03B11 Briefly describe the potential causes of a difference between measured end-tidal and arterial partial pressure of carbon dioxide.

**Background**

Classically, Bohr equation estimates physiological dead space

\[ V_D = \frac{P_A CO_2 - P_ME CO_2}{P_A CO_2} \times V_T \]

Where,
- \( P_A \) is CO\(_2\) partial pressure at alveolar level (i.e. equilibrate with blood)
- \( P_ME \) is CO\(_2\) partial pressure at mouth level (i.e. mixed with upper airway expired gas)
- \( V_D \) is physiological dead space
- \( V_T \) is tidal volume

Anatomical dead space is then measured using Fowler’s method

Alveolar dead space is calculated from *physiological dead space minus anatomical dead space*

However, as \( P_A \) is difficult to obtain and \( P_ME \) is variable throughout expiratory phase, subsequent modifications by Enghoff et al led to the following approximation:

\[ V_{DALv} = \frac{P_A CO_2 - P_{ET} CO_2}{P_A CO_2} \times V_T \]

where,
- \( P_A CO_2 \) is the arterial CO\(_2\) partial pressure
- \( P_{ET} CO_2 \) is the end-tidal CO\(_2\) partial pressure
- \( V_{DALv} \) is the *alveolar dead space*

**Arterial-End Tidal Difference**

Rearranging above equation gives

\[ P_A CO_2 - P_{ET} CO_2 = \frac{V_{DALv}}{V_T} \times P_A CO_2 \]

*i.e. the arterial-end tidal CO\(_2\) partial pressure difference is directly related to the alveolar dead space fraction*

The normal value for a-ET difference is usually 3 ~ 5 mmHg for healthy individuals

This is due to:
- inherent V/Q mismatch within the lung due to gravity resulting in alveolar dead space → this is physiological!
- approximate nature of Enghoff modification
Non-physiological causes for a-ET difference

1. Increased alveolar dead space fraction
   - increase in high V/Q units (i.e. West zone 1, $P_{alv} > P_a$)
   - increase in $P_{alv}$ – positive pressure ventilation, PEEP, positioning, etc
   - decrease in $P_a$ – hypoperfusion (esp. cardiac arrest), HPV, pulmonary embolus, etc

2. Increased $PaCO_2$
   - $P_a - P_{ET}$ difference is proportional to absolute magnitude of $P_a$
   - $↑P_a$ with $↑$production (e.g. malignant hyperpyrexia)
   - $↑P_a$ with $↑$absorption (e.g. circuit rebreathing, pneumoperitoneum)
   - $↑P_a$ with $↓$elimination (i.e. hypoventilation)

3. Measurement errors for $PaCO_2$
   - gas machine problem (uses Severinghaus electrode)
   - collection problem (e.g. sampling delay, non-arterial puncture, contamination with heparin)
   - temperature correction

4. Measurement errors for $P_{ETCO_2}$
   - infrared spectrophotometer problem (e.g. fluid in sample line, calibration error, etc)
   - collision broadening (e.g. $N_2O$)
   - not true end-tidal value (e.g. obstructive disease with very slow alveoli)

Examiner’s comments – 43% passed.

The answer required consideration of both patient factors, and those errors and limitations of the equipment and techniques used to monitor CO2 both in expired gas and arterial blood.

The effect of alveolar dead space on the difference between end-tidal and arterial CO2 was recognised by most candidates, and many included satisfactory explanations of the causes and potential underlying pathologies, gaining extra marks. The commonly observed clinical effect of delayed alveolar emptying with slow rise in expired CO2 leading to failure to obtain a true plateau, was usually overlooked, but attracted extra marks when included.

Equipment problems expected to be included were leaks and occlusions, effect of sampling site, other gases, calibration errors. This area was overlooked by many candidates, leading to the low pass rate. Most candidates that addressed both the patient and equipment components obtained a good pass.