(a-ET)PCO$_2$ reflects Alveolar Dead Space

(a-ET)PCO$_2$ reflects alveolar dead space as a result of a temporal, a spatial and an alveolar mixing defect in the normal lung.

Normal values of (a-ET)PCO$_2$ is 2-5 mm Hg.

(a-ET)PCO$_2$ as an index of alveolar dead space

There is a positive relationship between alveolar dead space and (a-ET)PCO$_2$. There is an exception to this rule (See text below)

(a-ET)PCO$_2$ increases with age, emphysema, and in circumstances where alveolar dead space increases such as in low cardiac output states, hypovolemia, and pulmonary embolism.

(a-ET)PCO$_2$ decreases in pregnancy and children (-0.65-3 mm Hg).
Decreased cardiac output increases alveolar dead space and thus increases (a-ET)PCO_2

Air embolism increases alveolar dead space and thus increases (a-ET)PCO_2
Pulmonary thrombo-embolism increases alveolar dead space and thus increases \((a\text{-ET})PCO_2\)
(a-ET)PCO₂ may not reflect Alveolar Dead Space when phase III has a steeper slope

(a-ET)PCO₂ could be zero or negative even in the presence of alveolar dead space
This animation has been based on the concept as in reference 2.

**(a-ET)CO₂ gradient** as an index of alveolar dead space:

Under normal circumstances, the PETCO₂ (the CO₂ recorded at the end of the breath which represents PCO₂ from alveoli which empty last) is lower than PaCO₂ (average of all alveoli) by 2-5 mmHg, in adults.¹⁻⁸ The (a-ET)PCO₂ gradient is due to the V/Q mismatch in the lungs (alveolar dead space) as a result of temporal, spatial, and alveolar mixing defects. In healthy children, the (a-ET)PCO₂ gradient is smaller (-0.65-3 mm Hg) than in adults.⁹⁻¹⁴ This is due to a better V/Q matching, and hence a lower alveolar dead space in children than in the adults.⁹ The (a-ET)PCO₂ / PaCO₂ fraction is a measure of alveolar dead space, and changes in alveolar dead space correlate well with changes in (a-ET) PCO₂.⁴ An increase in (a-ET)PCO₂ suggests an increase in dead space ventilation. Hence (a-ET)PCO₂ is an indirect estimate of V/Q mismatching of the lung.

However, (a-ET)PCO₂ does not correlate with alveolar dead space in all circumstances. Changes in alveolar dead space correlate with (a-ET)PCO₂ only when phase III is flat or has a minimal slope. In this case, the area (blue shaded color in the figure above) rectangular and PaCO₂ > PETCO₂. However, if phase III has a steeper slope, the terminal part of phase III may intercept the line representing PaCO₂, resulting in either zero or negative (a-ET)PCO₂ even in the presence of alveolar dead space. Therefore, the (a-ET)PCO₂ is dependent both on alveolar dead space as well as factors that influence the slope of phase III. This implies that an increase in the alveolar dead space need not be always be associated with an increase in the (a-ET)PCO₂. The (a-ET)PCO₂ may remain the same if there is an associated increase in the slope of the phase III. For example, it has been observed during cardiac surgery that alveolar dead space was increased at the end of cardiopulmonary bypass but as the slope of phase III was also increased, there was no change in (a-ET)PCO₂.¹⁵,¹⁶

**Cardiac output and (a-ET)PCO₂**
Reduction in cardiac output and pulmonary blood flow result in a decrease in PETCO$_2$ and an increase in (a-ET)PCO$_2$. Increases in cardiac output and pulmonary blood flow result in better perfusion of the alveoli and a rise in PETCO$_2$. Consequently alveolar dead space is reduced as is (a-ET)CO$_2$. The decrease in (a-ET)PCO$_2$ is due to an increase in the alveolar CO$_2$ with a relatively unchanged arterial CO$_2$ concentration, suggesting better excretion of CO$_2$ into the lungs. The improved CO$_2$ excretion is due to better perfusion of upper parts of the lung. Askrog found an inverse linear correlation between pulmonary artery pressure and (a-ET)PCO$_2$. Thus, under conditions of constant lung ventilation, PETCO$_2$ monitoring can be used as a monitor of pulmonary blood flow.

Reference:


