Abnormal Ventilation, Abnormal Gas Exchange

Objective: to achieve adequate tissue oxygenation and remove metabolically produced CO2.

Ventilation: concerned with delivery of fresh volume of air to gas exchanging units, and the removal of a sufficient volume of mixed gas out

Gas Exchange: the ability to move gas across the alveolar-capillary membrane
Ventilation and Gas Exchange

- The failure of either or both results in impaired arterial blood gases and ultimately to respiratory failure.
- Ventilatory failure: Hypercapnic respiratory failure
- Gas exchange failure: Hypoxemic respiratory failure
- Hypoxemia is the inevitable result of both

Hypoxemia

- Low partial pressure of O2 in blood (PaO2)
- Hypoxemia is not synonymous with:
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− Hypoxemia is not synonymous with:
  − Hypoxia (metabolic O2 deficiency)
  − Low O2 carrying capacity (1.34 ml O2/gm Hgb)
  − Low O2 content (CaO2:SaO2 x O2 carrying capacity +.003 ml O2/100 ml/mmHg PaO2)

− Low O2 delivery (CaO2 x C.O.)
Physiologic Causes of Hypoxemia

Alveolar Hypoventilation
Decreased PIO2
Diffusion Abnormality
V/Q mismatch
Shunt

Ventilation

Minute Ventilation (VE) = tidal volume (VT) x respiratory frequency
Alveolar ventilation (VA) = that part of minute ventilation which participates in gas exchange
Alveolar ventilation = alveolar volume (tidal volume - dead space volume) x respiratory frequency
**Ventilation**

- Alveolar PCO2 (PACO2) = VCO2/VA x K
- VCO2 = CO2 production
- VA = alveolar ventilation
- Normal: VCO2/VA = 1/21.6; K = 863 mmHg
- Alveolar PCO2 = CO2 leaving lungs after gas exchange; directly reflects arterial PCO2
- E.g., halving alveolar ventilation with constant CO2 production will double the alveolar PCO2
- E.g., doubling the alveolar PCO2 reflects halved alveolar ventilation

**Hypoventilation**

- Inability to inspire and expire a volume of air/gas sufficient to meet metabolic demands
- Inability to bring a fresh volume of O2 with each breath to the gas exchanging unit, and inability to remove CO2 produced by metabolism.
- *Sine qua non:* Increased arterial PCO2 (PaCO2); decreased arterial PO2 (PaO2) breathing room air (*parallel changes!!*)
Hypoventilation/
Alveolar hypoventilation

- All hypoventilation concerns either increased dead space/tidal volume (anatomic or physiologic): ALVEOLAR HYPOVENTILATION; or
- Decreased MINUTE ventilation (decreased tidal volume, decreased respiratory rate)
- Increased minute ventilation may make up for impaired alveolar ventilation; opposite not true…

Alveolar Hypoventilation:
2 Clinical Pearls

- Does not widen the AaDO₂
- The hypoxemia may be readily ameliorated with supplemental O₂
Alveolar Gas Equation

\[ \text{PAO}_2 = \text{PIO}_2 - \frac{\text{PACO}_2}{R} \]

\[ \text{PAO}_2 = \text{PIO}_2 - \frac{\text{PACO}_2}{R} + \left[ \text{PCO}_2 \times \text{FIO}_2 \times \frac{1-R}{R} \right] \]
Alveolar Gas Equation

\[ \text{PAO}_2 = \text{PIO}_2 - \frac{\text{PACO}_2}{R} \]

- \( \text{PIO}_2 \): \( \text{FIO}_2 \) (\text{Patm-PH}_20)
- \( \text{PACO}_2 = \text{PaCO}_2 \)
- \( R = \text{Respiratory Exchange Ratio:} \) (gas \( \text{R} = \text{CO}_2 \) added to alveolar gas by blood/amount of \( \text{O}_2 \) removed from alveolar gas by blood; low \( \text{V/Q} = \text{low R} \); normal=0.8)
Case History

- Room air: PaO2=30 mmHg, PaCO2=90 mmHg, pH=7.08
- PAO2 = 0.21 (760-47) –90/0.8

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- PAO2 = 150-112.5 = 37.5
Case History

\[ \text{PaO}_2 = 30 \text{ mmHg}, \text{PaCO}_2 = 90 \text{ mmHg}, \text{pH} = 7.08 \]

\[ \text{PAO}_2 = 0.21 (760 - 47) - 90/0.8 \]

\[ \text{PAO}_2 = 150 - 112.5 = 37.5 \]

\[ \text{AaDO}_2 = 7.5 \text{ mmHg} \]

Alveolar Hypoventilation

\[ \text{CNS: central hypoventilation; infectious, traumatic, vascular damage to medullary centers; pharmacologic and sleep suppression of ventilatory drive} \]
Alveolar Hypoventilation

- CNS: central hypoventilation; infectious, traumatic, vascular damage to medullary centers; pharmacologic and sleep suppression of ventilatory drive
- Peripheral nervous system/myoneural junction: poliomyelitis, Guillain-Barre, myasthenia gravis

Alveolar Hypoventilation

Respiratory muscles: muscular dystrophy, ALS, increased inspiratory loading (eg emphysema)
Alveolar Hypoventilation

Respiratory muscles: muscular dystrophy, increased inspiratory loading (e.g., emphysema)

Chest wall/mechanical restriction:
kyphoscoliosis, trauma, splinting, obesity

Airway obstruction: upper airway, lower airway
**Alveolar Hypoventilation**

- Respiratory muscles: muscular dystrophy, increased inspiratory loading (e.g., emphysema)
- Chest wall/mechanical restriction: kyphoscoliosis, trauma, splinting, obesity
- Airway obstruction: upper airway, lower airway
- Increased dead space ventilation: pulmonary embolism; COPD

**Hypercapnic Respiratory Failure**

- Primary deficit = hypoventilation without gas exchange abnormality, until late
Physiologic Causes of Hypoxemia

- Alveolar Hypoventilation
- Decreased PIO₂
- Diffusion Abnormality
- V/Q mismatch
- Shunt

Climbing Everest

\[ P_{atm} = 250 \text{ mmHg} \]
\[ P_{aCO₂} = 18 \text{ mmHg}; R = 1 \]
\[ P_{aO₂} = P_{IO₂} - P_{CO₂}/R \]
\[ P_{aO₂} = .21 (250-47)-18/1 = 24.6 \]
Case History

 pérdida de Pao2=70, PaCO2=30 mmHg

No treatment: RA PaO2=50 mmHg, PaCO2=28 mmHg
What happened?

\[ \text{PAO}_2 = \text{PIO}_2 - \frac{\text{PACO}_2}{R} \]

\[ 0.21 \times \text{FIO}_2, \text{PaO}_2 = 50 \text{ mmHg}, \]
\[ \text{PaCO}_2 = 28 \text{ mmHg} \]

\[ \text{PAO}_2 = 0.21(713) - 28/0.8 = 150 - 35 = 115 \text{ mmHg} \]

\[ \text{AaDO}_2 = 115 - 50 = 65 \text{ mmHg} \]

AaDO2 and Hypoxemia

\[ \text{Widened in diffusion disorder, V/Q mismatch, and shunt} \]

\[ \text{Not widened in alveolar hypoventilation and decreased PIO2} \]

\[ \text{Normal 10-15 mmHg in young adult} \]
Hypoxemia

- No widening of AaDO₂: hypoventilation, low PIO₂.
- Widened AaDO₂: shunt, low V/Q, low diffusing capacity
- Hypoxemia of each may be overcome with supplemental O₂ except: shunt.
- Note: no gas exchange=no amelioration of hypoxemia with O₂, whether dead space, shunt, or no diffusion.

Low V/Q

- “Venous admixture”
- Alveolar filling: pneumonia, pulmonary edema (cardiogenic/non-cardiogenic)
- COPD
- Usually will involve some infinitely low V/Q (shunt) and decreased diffusion.
Low V/Q

\[ \text{Low relationship of V to Q; NOT low ventilation} \]
\[ \text{That is, hypoventilation NOT low V/Q} \]
\[ \text{Low V/Q NOT hypoventilation} \]
Diffusing Abnormality

- Alveolar-capillary membrane thickening (pulmonary hypertension, pulmonary vasculitis, pulmonary embolism)
- Alveolar-capillary membrane destruction (emphysema)
- Pulmonary interstitial thickening (pulmonary fibrosis)
- Alveolar filling

Shunt

- Infinitely low V/Q
- Supplemental O2 will not raise PaO2 with large shunt
- Clinical examples: ARDS, other severe pneumonia, cardiogenic pulmonary edema
- May also be cardiogenic R-L shunt
RESPIRATORY PHYSIOLOGY

\[ \frac{Q_T}{Q_S} = \frac{Cc_o^2 - Ca_o^2}{Cc'_o^2 - C\dot{V}_o^2} \]

\[ Q_T \]

\[ Q_S \]

\[ C\dot{V}_o^2 \]

\[ Ca_o^2 \]

\[ Cc_o^2 \]

\[ PO_2 \]

\[ PCO_2 \]

\[ VA \]

\[ V_A \]

\[ V/A \]

\[ VA/\dot{V}_A \]

**Note:** $V/A = V_A/\dot{V}_A$
Hypoxemic Respiratory Failure

- Primary deficit = hypoxemia without hypoventilation, until late
- Gas exchange abnormality: shunt, low V/Q, low diffusing capacity, all...

SUMMARY

- Hypoventilation: High PaCO2, Low PaO2, no widening of AaDO2
- Gas exchange abnormality: Low PaO2, normal to low PaCO2, widened AaDO2
- Hypoxemia of all hypoventilation and gas exchange abnormalities may be sufficiently overcome by supplemental O2 unless gas exchange abnormality or hypoventilation is absolute (eg. shunt or dead space)