Abnormal Ventilation, Abnormal Gas Exchange

Ventilation and 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:
Hypoxemia

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   ⇔ Hypoxia (metabolic O2 deficiency)

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   ⇔ Low O2 carrying capacity (1.34 ml O2/gm Hgb)
Hypoxemia

Low partial pressure of O2 in blood (PaO2)

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 AaDO$_2$
- The hypoxemia may be readily ameliorated with supplemental O$_2$
Alveolar Gas Equation

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

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

Alveolar Gas Equation

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

\[ \text{PIO}_2: \text{FIO}_2 \text{ (Patm-PH}_20) \]
Alveolar Gas Equation

\[ PAO_2 = PIO_2 - PACO_2 / R \]

- \( PIO_2 \): FIO2 (Patm-PH20)
- \( PACO_2 = PaCO_2 \)
- \( R \): Respiratory Exchange Ratio: (gas \( R = CO_2 \) added to alveolar gas by blood/amount of \( O_2 \) removed from alveolar gas by blood; low \( V/Q \) = 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

\[ \text{PaO}_2 = 30 \text{ mmHg, PaCO}_2 = 90 \text{ mmHg, pH} = 7.08 \]
\[ \text{PAO}_2 = 0.21 \ (760-47) - 90/0.8 \]
\[ \text{PAO}_2 = 150 - 112.5 = 37.5 \]
\[ \text{AaDO}_2 = 7.5 \text{ mmHg} \]

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

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Everest

$P_{atm} = 250 \text{ mmHg}$
Everest

\[ P_{atm} = 250 \text{ mmHg} \]

\[ PaCO_2 = 18 \text{ mmHg}; \quad R = 1 \]

\[ PAO_2 = PIO_2 - PCO_2 / R \]

\[ PAO_2 = 0.21 \times (250 - 47) - 18 / 1 = 24.6 \]

Case History

\[ RA: \quad PaO_2 = 70, \quad PaCO_2 = 30 \text{ mmHg} \]
Case History

- RA: PaO₂=70, PaCO₂=30 mmHg
- No treatment: RA PaO₂=50 mmHg, PaCO₂=28 mmHg

Alveolar Gas Equation

- \( PAO₂ = PIO₂ - \frac{PACO₂}{R} \)
- 0.21 FIO₂, PaO₂=50 mmHg, PaCO₂=28 mmHg
- \( PAO₂ = 0.21(713) - 28/0.8 = 150 - 35 = 115 \) mmHg
- \( AaDO₂ = 115 - 50 = 65 \) mmHg
AaDO2 and Hypoxemia

- Widened in diffusion disorder, V/Q mismatch, and shunt
- Not widened in alveolar hypoventilation and decreased PIO2
- 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

\( \leftarrow \) Low relationship of \( V \) to \( Q \); NOT low ventilation
\( \leftarrow \) That is, hypoventilation NOT low V/Q
\( \leftarrow \) Low V/Q NOT hypoventilation

Diffusing Abnormality

\( \leftarrow \) Alveolar-capillary membrane thickening (pulmonary hypertension, pulmonary vasculitis, pulmonary embolism)
\( \leftarrow \) Alveolar-capillary membrane destruction (emphysema)
\( \leftarrow \) Pulmonary interstitial thickening (pulmonary fibrosis)
\( \leftarrow \) 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
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 (e.g. shunt or dead space)
“If you can’t breathe, nothing else matters…”