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.

The failure of either or both results in impaired arterial blood gases and ultimately 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:
- Hypoxia (metabolic O2 deficiency)
- 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)

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), or
  - Decreased MINUTE ventilation (decreased tidal volume, and/or decreased respiratory rate)
  - Each is considered alveolar hypoventilation if PaCO2 is elevated.

Alveolar Hypoventilation: 2 Clinical Pearls

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

Alveolar Gas Equation

- \( \text{PAO2}=\text{PIO2} – \frac{\text{PACO2}}{R} \)
- \( \text{PAO2}=\text{PIO2} – \frac{\text{PACO2}}{R} + \left[ \frac{\text{PCO2} \times \text{FIO2} \times (1-R)}{R} \right] \)

Alveolar Gas Equation

- \( \text{PAO2}=\text{PIO2} – \frac{\text{PACO2}}{R} \)
- \( \text{PIO2}: \text{FIO2} (\text{Patm-PH20}) \)
- \( \text{PACO2}=\text{PaCO2} \)

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- \( R=\text{Respiratory Exchange Ratio}: (\text{gas R=}\text{CO2 added to alveolar gas by blood}/\text{amount of O2 removed from alveolar gas by blood}; \text{low V/Q=low R}); \text{normal=}0.8 \)
Case History

Room air: PaO$_2$=30 mmHg, PaCO$_2$=90 mmHg, pH=7.08

PAO$_2$= 0.21 (760-47) – 90/0.8

PAO$_2$=150-112.5=37.5

AaDO$_2$=7.5 mmHg

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

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

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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
Hypoxemia MUST result

Physiologic Causes of Hypoxemia
Alveolar Hypoventilation
Decreased PIO2
Diffusion Abnormality
V/Q mismatch
Shunt

Climbing Everest (Decreased PIO2)

\[ P_{atm} = 250 \text{ mmHg} \]
\[ P_{aCO2}=18 \text{ mmHg}; R=1 \]
\[ P_{AO2}=P_{IO2}-P_{CO2}/R \]
\[ P_{AO2}=.21 (250-47)-18/1=24.6 \]
Case History

RA: PaO2=70, PaCO2=30 mmHg

No treatment: RA PaO2=50 mmHg, PaCO2=28 mmHg

What happened?

PAO2=PIO2 – 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 a common situation of low V/Q
Usually will involve some infinitely low V/Q (shunt) and decreased diffusion.
**Low V/Q**

- Low relationship of $V$ to $Q$; NOT low ventilation
- Low V/Q is NOT hypoventilation (unless all units are the same low V/Q)

**Diffusion 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 $PaO_2$ 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 is absolute (eg shunt)