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 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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- 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)
  - 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 PCO$_2$ (PACO$_2$) = VCO$_2$/VA x K
- VCO$_2$ = CO$_2$ production
- VA = alveolar ventilation
- Normal: VCO$_2$/VA = 1/21.6; K = 863 mmHg
- Alveolar PCO$_2$ = CO$_2$ leaving lungs after gas exchange; directly reflects arterial PCO$_2$
- e.g., halving alveolar ventilation with constant CO$_2$ production will double the alveolar PCO$_2$
- e.g., doubling the alveolar PCO$_2$ 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 O$_2$ with each breath to the gas exchanging unit, and inability to remove CO$_2$ produced by metabolism.
- *Sine qua non:* Increased arterial PCO$_2$ (PaCO$_2$); decreased arterial PO$_2$ (PaO$_2$) 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

- $PAO_2 = PIO_2 - PACO_2/R$
- $PAO_2 = PIO_2 - PACO_2/R + [PCO_2 \times FIO_2 \times 1 - R/R]$

Alveolar Gas Equation

- $PAO_2 = PIO_2 - PACO_2/R$
- $PIO_2: FIO_2 (Patm-PH20)$
Alveolar Gas Equation

- \( PAO_2 = PIO_2 - PACO_2 / R \)
- \( PIO_2: FIO_2 (P_{atm} - PH_{20}) \)
- \( PACO_2 = PaCO_2 \)

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

- Room air: PaO2=30 mmHg, PaCO2=90 mmHg, pH=7.08
- PAO2= 0.21 (760-47) –90/0.8
- PAO2=150-112.5=37.5
Case History

- PaO2=30 mmHg, PaCO2=90 mmHg, pH=7.08
- PAO2= 0.21 (760-47) –90/0.8
- PAO2=150-112.5=37.5
- AaDO2=7.5 mmHg

Alveolar Hypoventilation

- 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
- Hypoxemia MUST result
Physiologic Causes of Hypoxemia

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

Climbing Everest (Decreased PIO2)

P atm = 250 mmHg
PaCO2 = 18 mmHg; R = 1
PAO2 = PIO2 - PCO2 / R
PAO2 = 0.21 (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?

- \( \text{PAO}_2 = \text{PIO}_2 - \text{PACO}_2 / R \)
- \( \text{0.21 FIO}_2, \text{PaO}_2 = 50 \text{ mmHg}, \)
  \( \text{PaCO}_2 = 28 \text{ mmHg} \)
- \( \text{PAO}_2 = 0.21(713) - 28/0.8 = 150 - 35 = 115 \text{ mmHg} \)
- \( \text{AaDO}_2 = 115 - 50 = 65 \text{ mmHg} \)

AaDO2 and Hypoxemia

- 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 AaDO$_2$: hypoventilation, low PIO$_2$.
- Widened AaDO$_2$: shunt, low V/Q, low diffusing capacity
- Hypoxemia of each may be overcome with supplemental O$_2$ except: shunt.
- Note: no gas exchange=no amelioration of hypoxemia with O$_2$, 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 PaO2 with large shunt
- Clinical examples: ARDS, other severe pneumonia, cardiogenic pulmonary edema
- May also be cardiogenic R-L shunt
RESPIRATORY PHYSIOLOGY

\[
\frac{Q_S}{Q_T} = \frac{Cc'O_2 - CaO_2}{Cc'O_2 - C\bar{V}O_2}
\]

\[Q_S - CO_2\text{ diagram}
\]

Note: \( \bar{V}A = V_{A/0} \)
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 (e.g., shunt)