Arterial Blood Gases

- Reflect oxygenation, gas exchange, and acid-base balance
- PaO₂ is the partial pressure of oxygen dissolved in arterial blood
- SaO₂ is the amount of oxygen bound to hemoglobin
- Oxygen is transported from the alveoli into the plasma

Arterial Blood Gases

- Ranges
  - PaO₂: 80 - 100 mm Hg at sea level
    - < 80 mm Hg = hypoxemia
    - < 60 mm Hg may be seen in COPD patients
    - < 40 mm Hg is life threatening
  - SaO₂: 93 - 100 % is a normal saturation
    - Hypoxia is decreased oxygen at the tissue level

Arterial Blood Gas Interpretation

- pH: negative log of H⁺ concentration
- In blood:
  - Normal range: 7.35 - 7.45
  - Acidosis = pH less than 7.35
  - Alkalosis = pH greater than 7.45
  - A pH < 7.0 or > 7.8 can cause death
Arterial Blood Gas Interpretation

- **PaCO₂**: partial pressure of carbon dioxide dissolved in the arterial plasma
  - Normal: 35 - 45 mm Hg
  - Is regulated in the lungs
  - A primary respiratory problem is when PaCO₂ is:
    - > 45 mm Hg = respiratory acidosis
    - < 35 mm Hg = respiratory alkalosis
  - HCO₃ will be normal (22 - 26 mEq/L)

Arterial Blood Gas Interpretation

- **HCO₃** (bicarbonate)
  - Normal: 22 - 26 mEq/L
  - Is regulated by the kidneys
  - A primary metabolic or renal disorder is when the HCO₃
    - is < 22 = metabolic acidosis or
    - > 26 = metabolic alkalosis
  - PaCO₂ is normal

Arterial Blood Gas Interpretation

- Compensation:
  - Body attempts to recover from primary problem and return to homeostasis
  - Primary metabolic acidosis can cause the patient to breathe faster to compensate (blow off CO₂) by creating a respiratory alkalosis state.
  - This would be labeled as: **Metabolic acidosis with a compensatory respiratory alkalosis**
    - pH 7.30, PaCO₂ = 26 & HCO₃ = 15
  - Are PaCO₂ & HCO₃ below normal? Yes! Compensation
Metabolic Acidosis

- Risk factors: >ingestion of acids or < production of HCO3
- Etiology: lactic acidosis, ketoacidosis, uremic acidosis
- Patho: compensatory hyperventilation
  - Hyperkalemia: shift of acid from plasma to ICF
  - <pH, <HCO3, PaCo2 normal or low if compensation is occurring
  - cardiac dysrhythmias & CNS dysfunction
  - headache, diarrhea, tremors

Metabolic Alkalosis

- Risk factors:
  - Hypovolemia
  - Excess aldosterone
  - Iatrogenic base administration
- Etiology:
  - Acid loss or base gain
  - Renal excretion of HCO3 will fix the problem
  - Prolonged vomiting (loss of HCl)

Metabolic Alkalosis

- Patho: respiratory compensation is limited
- Hypokalemia: K+ moves from ECF to ICF due to hydrogen ions moving out of the cell to ECF
- Depleted body stores (K+):
  - Loop diuretics? NGT?
- Signs and symptoms:
  - cardiac dysrhythmias; seizures; confusion; muscle twitching, agitation
  - >pH; >HCO3; normal PaCo2 or elevated if compensation occurs
Respiratory Acidosis

- Risk factors:
  - Excess acid in body fluids

- Etiology:
  - Hypoventilation
  - COPD; Cystic Fibrosis; airway obstruction; spinal cord injury; CVA; depressant drugs; inadequate mechanical ventilation

- Patho:
  - Hypercapnia; CO2 diffuses easily across biological membranes
  - Clinical:
    - Decreased pH
    - $\text{PaCO}_2$
    - HCO3 is normal or increased in renal compensation

- Signs and Symptoms
  - Vasodilatation
  - Cardiac arrhythmias
  - Tachycardia
  - Somnolence & decreased ventilation

Respiratory Alkalosis

- Risk factors:
  - Relative excess of base in body fluids secondary to $>$ ventilatory elimination of CO2; pneumonia; shock; severe anemia

- Etiology:
  - Hypoxemia ($<$PaO2) causing rate & depth of ventilation to increase in an attempt to raise CO2

- Patho: buffer response is to shift acid from ICF to the blood by moving HCO3 into the cells in exchange of chloride
  - $<$pH; $<$PaCO2; HCO3 normal or low due to compensation
  - Nausea, vomiting, tingling of fingers
References

- [http://www.pathoplus.com](http://www.pathoplus.com)