

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 = 28 & HCO₃ = 15**
 - Are PaCO₂ & HCO₃ below normal? Yes! Compensation:

Metabolic Acidosis

- Risk factors: >ingestion of acids or < production of HCO_3
- Etiology: lactic acidosis, ketoacidosis, uremic acidosis
- Patho: compensatory hyperventilation
 - Hyperkalemia: shift of acid from plasma to ICF
 - pH , HCO_3 , PaCO_2 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 HCO_3 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 ; HCO_3 ; normal PaCO_2 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

Respiratory Acidosis

- Patho:
 - Hypercapnia; CO₂ diffuses easily across biological membranes
- Clinical:
 - Decreased pH
 - $>PaCO_2$
 - 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 ($<PaO_2$) 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
 - $>pH$; $<PaCO_2$; HCO₃ normal or low due to compensation
 - nausea, vomiting, tingling of fingers

References

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- Huether, S. E., & McCance, K. L. (2002). *Pathophysiology*. St. Louis: Mosby.
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