

Respiratory Alkalosis (primary hypocapnia)  
Dibartola p 292-end

Characterized by:

- Decreased PCO<sub>2</sub>
- Increased pH
- Decrease in [HCO<sub>3</sub><sup>-</sup>]

Metabolic Compensation in Respiratory Alkalosis

- Acute resp alkalosis: decrease of 0.25mEq/L in [HCO<sub>3</sub><sup>-</sup>] for each 1mmHg decrease in PCO<sub>2</sub> is expected
- Chronic resp alkalosis: a 0.55mEq/L decrease in [HCO<sub>3</sub><sup>-</sup>] is expected for each 1mmHg decrease in PCO<sub>2</sub>
  - pH is normal or near normal, but normalization of pH can take up to 4 weeks to be achieved

Causes of Respiratory Alkalosis

1. Hypoxemia
  - When pO<sub>2</sub> decreases to <60mmHg the peripheral chemoreceptors mediate an increase in rate and depth of breathing → resulting in hypocapnia
  - Decreased oxygen *delivery* also results in hypocapnia
2. Pulmonary disease
  - Hyperventilation can be a result of concurrent hypoxemia
  - Pulmonary disease may cause hyperventilation *without hypoxemia* as a result of stimulation of *stretch receptors* and *nociceptive receptors* that respond to irritants, interstitial edema, fibrosis or pulmonary capillary congestion
    - i. Stretch receptors: located in smooth muscle of tracheobronchial tree
    - ii. Nociceptive receptors: include irritant receptors in the epithelium of small airways and juxtacapillary receptors lining capillaries in interstitium
3. Centrally mediated hyperventilation
4. Overzealous mechanical ventilation
5. Muscle metaboreceptor overactivity
6. Situations causing pain, fear or anxiety

Clinical Features

- Alkalemia results in arteriolar vasoconstriction that decreases cerebral and myocardial perfusion
- Hyperventilation (PCO<sub>2</sub> < 25mmHg) causes decreased cerebral blood flow
- Hypocapnia decreases blood pressure and cardiac output in *anesthetized* but not awake subjects
  - Anesthetics blunt reflex tachycardia
- Alkalemia predisposes to refractory supraventricular and ventricular arrhythmias (especially in patients with preexisting heart disease)
- Acute alkalemia shifts oxygen hemoglobin dissociation curve to the left, reducing release of oxygen to the tissues by increasing affinity of hemoglobin for oxygen
  - Chronic alkalemia negates this effect by increasing concentration of 2,3-DPG in red cells
- Hypokalemia may occur as a result of translocation of potassium into cells and renal and extrarenal losses in patients with acute respiratory alkalosis

**Box 11-4****Causes of Respiratory Alkalosis****Hypoxemia (Stimulation of Peripheral Chemoreceptors by Decreased Oxygen Delivery)**

Right-to-left shunting  
Decreased  $P_{IO_2}$  (e.g., high altitude)  
Congestive heart failure  
Severe anemia  
Severe hypotension  
Decreased cardiac output  
Pulmonary diseases with ventilation-perfusion mismatch  
    Pneumonia  
    Pulmonary thromboembolism  
    Pulmonary fibrosis  
    Pulmonary edema  
    Acute respiratory distress syndrome

**Pulmonary Disease (Stimulation of Stretch/nociceptors Independent of Hypoxemia)**

Pneumonia  
Pulmonary thromboembolism  
Interstitial lung disease  
Pulmonary edema  
Acute respiratory distress syndrome

**Centrally Mediated Hyperventilation**

Liver disease  
Hyperadrenocorticism  
Gram-negative sepsis  
Drugs  
    Salicylates  
    Corticosteroids  
    Progesterone (pregnancy)  
    Xanthines (e.g., aminophylline)  
Recovery from metabolic acidosis  
Central neurologic disease  
    Trauma  
    Neoplasia  
    Infection  
    Inflammation  
    Cerebrovascular accident  
Exercise  
Heatstroke

**Muscle Metaboreceptor Overactivity**

Heart failure

**Overzealous Mechanical Ventilation****Situations Causing Pain, Fear, or Anxiety**

## Questions

1. Name 3 causes of respiratory alkalosis
2. Fill in the blanks. Acute alkalemia shifts oxygen hemoglobin dissociation curve to the \_\_\_\_\_, but chronic alkalemia negates this effect by increasing concentration of \_\_\_\_\_ in \_\_\_\_\_ cells.