



# Acid-Base Disturbances

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# What is it?

- Respiratory:

- breathing is inadequate and carbon dioxide accumulates

- ↑ PCO<sub>2</sub> contributes to an acid pH

- Metabolic:

- normal metabolism is impaired - acid forms

- if severe, the patient may be in shock

# Physiology



- Carbonic acid ( $\text{H}_2\text{CO}_3$ ) is central to our understanding and evaluation of acid-base disturbances.
- The dissociation products and the ionization products are normally in equilibrium

# Physiology: The Cell Wall



- Limits transfer of substances
- Depends on pH
  - first, as the pH changes so will the degree of ionization and, hence, the concentration of ionized
  - substances; second, if the degree of ionization changes greatly, a substance may cease to be ionized and will, therefore, escape from the cell.



# Physiology: Extracellular Fluid

- Treatable volume
- Extracellular fluid is 20% of the body weight
- Provides:
  - Nutrition
  - Oxygenation
  - Waste removal
  - Temperature
  - Alkalinity



# Acid-Base Disturbances

- pH must be within small range
  - Normal is 7.4
- Large acid loads are produced by *normal* metabolism

# Some definitions...

- pH defines the blood  $[H^+]$  concentration
  - Low ( $<7.35$ ) = Acidemia
  - High ( $>7.45$ ) = Alkalemia

# Some definitions...

- [HCO<sub>3</sub>] defines the metabolic component
  - Low (<20 mmol/L) = Metabolic acidosis
  - High (>33 mmol/L) = Metabolic alkalosis



# More definitions...



- pCO<sub>2</sub> defines the respiratory component
  - Low (<35 mmHg) = Respiratory alkalosis
  - High (>45 mmHg) = Respiratory acidosis

# Basic Evaluation



- High pH ( $>7.45$ ) suggests:
  - Respiratory alkalosis -  $p\text{CO}_2 < 35\text{mmHg}$
  - Metabolic alkalosis -  $[\text{HCO}_3] > 33\text{ mmol/L}$ .

# Basic Evaluation



- Low pH ( $<7.35$ ) suggests:
  - Respiratory acidosis -  $p\text{CO}_2 > 45$  mmHg
  - Metabolic acidosis -  $[\text{HCO}_3] < 23$  mmol/L

# Normal pH?



- Normal pH (7.35-7.45) suggests:
  - No acid-base disturbance
  - Chronic respiratory alkalosis
  - Chronic respiratory acidosis (mild)
  - Mixed disturbance

# Buffer Systems



- Bicarbonate – carbonic acid system
  - Lungs excrete
- Proteins and phosphates
  - Kidneys excrete

# Respiratory Acidosis



- ↓ respiratory exchange with retention of CO<sub>2</sub> results in a ↑ pCO<sub>2</sub> which then causes renal retention of bicarbonate

# Respiratory Acidosis: Causes

- ↓ respiratory exchange
- CNS Depression
  - trauma/infections/tumor
  - cerebrovascular accidents
  - drug overdose
- Neuromuscular disorders
  - Myopathies
- Thoracic disorders
  - hydrothorax
  - pneumothorax
- Lung disorder
  - bronchial obstruction
  - emphysema (chronic obstructive airway disease)
  - severe pulmonary edema

# Respiratory Acidosis: Compensation

- Problem:  $\uparrow$  pCO<sub>2</sub> and this results in a  $\downarrow$  blood pH (high H<sup>+</sup>)
- [H<sup>+</sup>] stimulates kidney to generate and retain bicarbonate
  - respiratory acidosis is compensated for by the development of a metabolic alkalosis



# Respiratory Acidosis: Compensation

- Compensation is complete ([HCO<sub>3</sub>] levels out) in 2-4 days
- Final HCO<sub>3</sub> can be calculated from the following equation:
  - $\text{HCO}_3 \text{ mmol/L} = 0.44 \times \text{pCO}_2 \text{ mmHg} + 7.6 (+/- 2)$ .
- Limit of compensation is a HCO<sub>3</sub> of 45 mmol/L

# Respiratory Acidosis: Treatment

- Acute: correct underlying source of alveolar hypoventilation
  - Bronchodilators
  - Oxygen
  - Antibiotics/Drug therapy
  - Dialysis
- If it is chronic: try to avoid excessive supplemental oxygen

# Respiratory Alkalosis



- ↑ respiratory exchange with loss of CO<sub>2</sub> results in a ↓ pCO<sub>2</sub> which then stimulates renal excretion of bicarbonate

# Respiratory Alkalosis: Causes

- ↑ respiratory exchange
- CNS disturbances
- Psychogenic (anxiety)
- Pregnancy
- Hypoxia
- Drug toxicity / overdose
- Pulmonary disorders
  - Embolism
  - Edema
  - Asthma
  - Pneumonia

# Respiratory Alkalosis: Compensation

- Problem: ↓ pCO<sub>2</sub> causing ↑ blood pH (low H<sup>+</sup>)
- ↑ pH stimulates the kidney to excrete bicarbonate
  - respiratory alkalosis is compensated for by the development of a metabolic acidosis

# Respiratory Alkalosis: Compensation

- If the condition has been present for 7 days or more full compensation may occur.
- Compensation is complete ( $[\text{HCO}_3^-]$  levels out) in 7-10 days.
- The limit of compensation is a  $\text{HCO}_3^-$  of 12 mmol/L.



# Respiratory Alkalosis: Treatment

- Treatment aims to eradicate the underlying condition
  - removal of ingested toxins
  - treatment of fever or sepsis (toxin)
  - treatment of CNS disease
- In severe respiratory alkalosis:
  - breathing into a paper bag, which helps relieve acute anxiety and increases carbon dioxide levels

# Metabolic Acidosis



- ↑ production or renal retention of  $H^+$  results in a low pH which stimulates respiration to ↓ the  $pCO_2$



# Metabolic Acidosis: Causes



- High Anion Gap

- Renal failure
- toxins
- ketoacidosis

- Normal anion gap (hyperchloremic)

- Hyperkalemia
- obstructive uropathy
- diarrhea
- renal tubular acidosis
- Some medications

# Metabolic Acidosis: Compensation

- Problem: ↓ [HCO<sub>3</sub>] causing ↓ blood pH (high H<sup>+</sup>).
- [H<sup>+</sup>] stimulates respiration which lowers the blood pCO<sub>2</sub>
  - metabolic acidosis is compensated for by the development of a respiratory alkalosis

# Metabolic Acidosis: Compensation

- Compensation is complete (pCO<sub>2</sub> levels out) in 12-24 hours.
- The final pCO<sub>2</sub> can be calculated from the following equation:
  - $p\text{CO}_2 \text{ mmHg} = 1.5 \times [\text{HCO}_3] \text{ (mmol/L)} + 8 \text{ (+/- 2)}$ .
- The limit of compensation is a pCO<sub>2</sub> of 10 mmHg

# Metabolic Acidosis: Treatment

- Try to restore perfusion and correction of underlying disturbance
- It is rarely necessary to administer sodium bicarbonate to patients with acute metabolic acidosis
  - Not recommended for stable patients with pH 7.2 or higher

# Metabolic Alkalosis



- ↑ production or renal retention of  $\text{HCO}_3^-$  results in a high pH which inhibits respiration to increase the  $\text{pCO}_2$

# Metabolic Alkalosis: Causes

- **↓ Urinary chloride**

- Gut H<sup>+</sup> loss

- Vomiting, suction

- Renal H<sup>+</sup> loss

- Diuretic therapy
- Contraction alkalosis

- **↑ Urinary chloride**

- Mineralocorticoid excess

- Diuretic therapy

# Metabolic Alkalosis: Compensation

- Problem: ↑ [HCO<sub>3</sub>] causing ↑ blood pH (low H<sup>+</sup>)
- Low [H<sup>+</sup>] suppresses respiration which ↑ blood pCO<sub>2</sub>
  - metabolic alkalosis is compensated for by the development of a respiratory acidosis

# Metabolic Alkalosis: Compensation

- Compensation is complete (pCO<sub>2</sub> levels out) in 12-24 hours.
- The final pCO<sub>2</sub> can be calculated from the following equation:
  - $p\text{CO}_2 \text{ mmHg} = 0.9 \times [\text{HCO}_3] \text{ (mmol/L)} + 9 \text{ (+/- 2)}$
- The limit of compensation is a pCO<sub>2</sub> of 60 mmHg



# Metabolic Alkalosis: Treatment

- When metabolic alkalosis is potentially life-threatening ( $\text{pH} > 7.6$  or  $[\text{HCO}_3^-] > 40$  mEq/L):
  - the carbonic anhydrase inhibitor acetazolamide should be considered; however, this agent is associated with renal loss of potassium



## Metabolic Alkalosis: Treatment

- If acetazolamide is not effective or the metabolic alkalosis worsens:
  - exogenous acid, in the form of a 0.1N solution of hydrochloric acid (100 mEq/L), should be administered through a central venous catheter