LECTURE 8: ACID BASE BALANCE

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Acid-Base Balance

Acid-Base Homeostasis

- The pH of body fluids is maintained within a narrow range despite the ability of the kidneys to generate large amounts of HCO_3^- and the normal large acid load produced by metabolism.
- This endogenous acid load is efficiently neutralized by buffer systems and ultimately excreted by the lungs and kidneys.
- Important buffers include intracellular proteins and phosphates and the extracellular bicarbonate–carbonic acid system.
- Compensation for acid-base derangements can be by respiratory mechanisms (for metabolic derangements) or metabolic mechanisms for respiratory derangements).
- Changes in ventilation in response to metabolic abnormalities are mediated by hydrogen sensitive chemoreceptors found in the carotid body and brain stem.
- Acidosis stimulates the chemoreceptors to increase ventilation، whereas alkalosis decreases the activity of the chemoreceptors and thus decreases ventilation.
- The kidneys provide compensation for respiratory abnormalities by either increasing or decreasing bicarbonate reabsorption in response to respiratory acidosis or alkalosis, respectively.
- Unlike the prompt change in ventilation that occurs with metabolic abnormalities، the compensatory response in the kidneys to respiratory abnormalities is delayed.
- Significant compensation may not begin for 6 hours and then may continue for several days.
- The predicted compensatory changes in response to metabolic or respiratory derangements are listed in Table below.

Predicted changes in acid-base disorders

DISORDER PREDICTED CHANGE

DISORDER	PREDICTED CHANGE
Metabolic	
Metabolic acidosis Metabolic alkalosis	$Pco2 = 1.5 \times HCO_3^- + 8$ $Pco2 = 0.7 \times HCO_3^- + 21$
Respiratory	
Acute respiratory acidosis Chronic respiratory acidosis Acute respiratory alkalosis Chronic respiratory alkalosis	$\Delta pH = (Pco2 - 40) \times 0.008$ $\Delta pH = (Pco2 - 40) \times 0.003$ $\Delta pH = (40 - Pco2) \times 0.008$ $\Delta pH = (40 - Pco2) \times 0.017$

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Metabolic Derangements

Metabolic Acidosis.

Etiology of metabolic acidosis

Increased Anion Gap Metabolic Acidosis

1. Exogenous acid ingestion
    a. Ethylene glycol
    b. Salicylate
    c. Methanol
2. Endogenous acid production
    a. Ketoacidosis
    b. Lactic acidosis
    c. Renal insufficiency

Normal Anion Gap

1. Acid administration (HCL)
2. Loss of bicarbonate
3. GI losses (diarrhea, fistulas)
4. Ureterosigmoidostomy
5. Renal tubular acidosis.
6. Carbonic anhydrase inhibiton.

- The body responds by several mechanisms, including producing buffers (extracellular bicarbonate and intracellular buffers from bone(phosphate) and muscle(protein)) by
    1. increasing ventilation (Kussmaul's respirations).
    2. increasing renal reabsorption and generation of bicarbonate.
- The kidney also will increase secretion of hydrogen and thus increase urinary excretion of NH_4^+ ($H^+ + NH_3^+ = NH_4^+$).
- Evaluation of a patient with a low serum bicarbonate level and metabolic acidosis includes determination of the anion gap (AG), an index of unmeasured anions.

Anion Gap(AG) = (Na) ‑ (Cl + HCO3)

- **The normal AG is <12 mmol/L** and is due primarily to the albumin effect, so that the estimated AG must be adjusted for albumin (hypoalbuminemia reduces the AG).
- Corrected AG = actual AG ‑ (2.5[4.5 ‑ albumin])
- A common cause of severe metabolic acidosis in surgical patients is **lactic acidosis.**

- In hypovolemic shock, lactate is produced in the presence of hypoxia from inadequate tissue perfusion.
- The treatment is to restore perfusion with volume resuscitation rather than to attempt to correct the abnormality with exogenous bicarbonate.
- With adequate perfusion, the lactic acid is rapidly metabolized by the liver, and the pH level returns to normal.
- In these settings، the bicarbonate loss is accompanied by a gain of chloride; thus، the AG remains unchanged.
- To determine whether the loss of bicarbonate has a renal cause, the urinary (NH_4^+) can be measured.
- A low urinary (NH_4) in the face of hyperchloremic acidosis would indicate that the kidney is the site of loss, and evaluation for renal tubular acidosis should be undertaken.
- Proximal renal tubular acidosis results from decreased tubular reabsorption of HCO_3^- whereas distal renal tubular acidosis results from decreased acid excretion.

Metabolic Alkalosis

- Normal acid-base homeostasis prevents metabolic alkalosis from developing unless both an increase in bicarbonate generation and impaired renal excretion of bicarbonate occur.

Etiology of metabolic alkalosis

1. **Increased bicarbonate generation**
   - **a.** Chloride losing (urinary chloride >20 mEq/L)
     - **i.** Mineralocorticoid excess
     - **ii.** Profound potassium depletion.
   - **b.** Chloride sparing (urinary chloride <20 mEq/L)
     - **i.** Loss from gastric secretions (emesis or nasogastric suction)
     - **ii.** Diuretics.
   - **c.** Excess administration of alkali
     - **i.** Acetate in parenteral nutrition
     - **ii.** Citrate in blood transfusions
     - **iii.** Antacids
     - **iv.** Bicarbonate
2. **Impaired bicarbonate excretion**
   - **a.** Decreased glomerular filtration
   - **b.** Increased bicarbonate reabsorption

- Metabolic alkalosis results from the loss of fixed acids or the gain of bicarbonate and is worsened by potassium depletion.

- The majority of patients also will have hypokalemia because extracellular potassium ions exchange with intracellular hydrogen ions and allow the hydrogen ions to buffer excess $HCO3^-$.
- Hypochloremic and hypokalemic metabolic alkalosis can occur from isolated loss of gastric contents in infants with pyloric stenosis or adults with duodenal ulcer disease.
- Unlike vomiting associated with an open pylorus، which involves a loss of gastric as well as pancreatic, biliary, and intestinal secretions, vomiting with an obstructed pylorus results only in the loss of gastric fluid, which is high in chloride and hydrogen, and therefore results in a hypochloremic alkalosis.
- Initially, the urinary bicarbonate level is high in compensation for the alkalosis.
- Hydrogen ion reabsorption also ensues، with an accompanied potassium ion excretion.

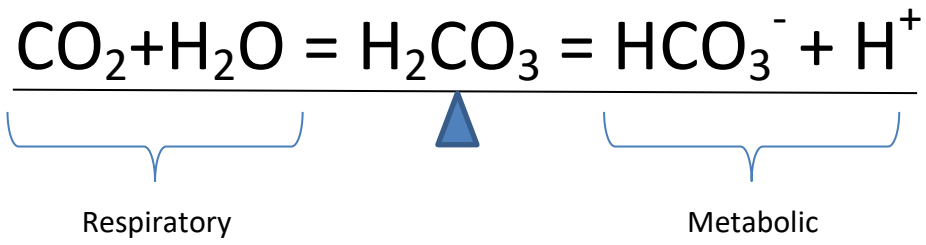
Respiratory Derangements.

- Under normal circumstances، blood Pco2 is tightly maintained by alveolar ventilation, controlled by the respiratory centers in the pons and medulla oblongata.
- **Respiratory acidosis** is associated with the retention of CO2 secondary to decreased alveolar ventilation.
- **Etiology of respiratory acidosis: hypoventilation**
    1. Narcotics
    2. Central nervous system injury
    3. Pulmonary: significant
        - Secretions
        - Atelectasis
        - Mucus plug
        - Pneumonia
        - Pleural effusion
    4. Pain from abdominal or thoracic injuries or incisions
    5. Limited diaphragmatic excursion from intra-abdominal pathology
        - Abdominal distention
        - Abdominal compartment syndrome
        - Ascites
- Because compensation is primarily a renal mechanism, it is a delayed response.

- Treatment of acute respiratory acidosis is directed at the underlying cause.
- Measures to ensure adequate ventilation are also initiated.
- This may entail patient-initiated volume expansion using noninvasive bi-level positive airway pressure or may require endotracheal intubation to increase minute ventilation.
- In the chronic form of respiratory acidosis, the partial pressure of arterial CO2 remains elevated, and the bicarbonate concentration rises slowly as renal compensation occurs.

Respiratory Alkalosis

- In the surgical patient, most cases of respiratory alkalosis are acute and secondary to alveolar hyperventilation.
- Causes include
    1. pain, anxiety, and neurologic disorders، including central nervous system injury and assisted ventilation.
    2. Drugs such as salicylates.
    3. gram-negative bacteremia, thyrotoxicosis, and hypoxemia



Acidosis ↑CO_2 , ↑H,↓PH

Alkalosis ↓CO_2 ↓H^+ ↑

Acidosis ↑H^+, ↓HCO_3^-,↓PH

Alkalosis ↓H^+, ↑HCO_3^-,↑PH