Chapter 2
Fluid, Electrolyte, and Acid-Base Imbalances
Review of Concepts and Processes

- The major component of the body is water.

- Water is located in these compartments:
  - Intracellular fluid (ICF) compartment
  - Extracellular fluid (ECF) compartment

- Balance of water between these compartments is essential for homeostasis.
Fluid Compartments (cont’d.)

- Intracellular compartment (ICF)
- Extracellular compartment (ECF)

  - Intravascular fluid (IVF) or blood
  - Interstitial fluid (ISF) or intercellular fluid
  - Cerebrospinal fluid (CSF)
  - Transcellular fluids // Present in various secretions // e.g. Pericardial cavity & Synovial cavities
Water Movement Between Fluid Compartments

- Digestive tract
- Intracellular fluid
- Bloodstream
- Tissue fluid
- Lymph
- Bloodstream
Fluid Compartments

- About 60% of an adult’s body weight is water.
- About 70% of an infant’s body weight is water.
- Females - higher percentage of fatty tissue = lower water content than males.
- Older adults and obese persons - lower proportion of water.
- Individuals with less fluid reserve are more likely to be adversely affected by any fluid or electrolyte imbalance. e.g. dehydration from extreme heat.
### TABLE 2-1  Fluid Compartments in the Body

<table>
<thead>
<tr>
<th></th>
<th>Volume</th>
<th>Approximate Percentage of Body Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Adult Male (L)</td>
<td>Male (%)</td>
</tr>
<tr>
<td>Intracellular fluid</td>
<td>28</td>
<td>40</td>
</tr>
<tr>
<td>Extracellular fluid</td>
<td>15</td>
<td>20</td>
</tr>
<tr>
<td>Plasma</td>
<td>(4.5)</td>
<td>(4)</td>
</tr>
<tr>
<td>Interstitial fluid</td>
<td>(10.5)</td>
<td>(15)</td>
</tr>
<tr>
<td>Other</td>
<td>(1)</td>
<td>(1)</td>
</tr>
<tr>
<td>Total water</td>
<td>43</td>
<td>60</td>
</tr>
</tbody>
</table>

Note: In elderly women, water content is reduced to approximately 45% of body weight.
Intake and Output of Water

- The amount of water entering the body should equal the amount of water leaving the body.

- Water Balance
Fluid Balance

Intake 2,500 mL/day

Metabolic water 200 mL

Food 700 mL

Drink 1,600 mL

Output 2,500 mL/day

Feces 200 mL

Expired air 300 mL

Cutaneous transpiration 400 mL

Sweat 100 mL

Urine 1,500 mL
<table>
<thead>
<tr>
<th>Sources (mL)</th>
<th>Losses (mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liquids</td>
<td>Urine</td>
</tr>
<tr>
<td>1200</td>
<td>1400</td>
</tr>
<tr>
<td>Solid foods</td>
<td>Feces</td>
</tr>
<tr>
<td>1000</td>
<td>200</td>
</tr>
<tr>
<td>Cell metabolism</td>
<td>Insensible losses</td>
</tr>
<tr>
<td>300</td>
<td>Lungs</td>
</tr>
<tr>
<td></td>
<td>Skin</td>
</tr>
<tr>
<td>Total</td>
<td></td>
</tr>
<tr>
<td>2500</td>
<td>2500</td>
</tr>
</tbody>
</table>

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Movement of Water

- “Water” compartments are created by semi permeable membranes

- Fluid circulates throughout the body crossing membranes via filtration and osmosis.

- Water moves between compartments via:
  - Hydrostatic pressure
  - Osmotic pressure
Movements of Water between Compartments

A. Filtration

- Movement of water and solutes from blood (high pressure) to ISF (low pressure) area.
- Hydrostatic pressure — IVF (e.g., 30 mm Hg).
- Hydrostatic pressure — ISF (e.g., 2 mm Hg).

B. Osmosis

- Movement of water from low solute concentration (ISF) to high concentration (blood).
- Osmotic pressure — blood (e.g., 25 mm Hg).
- Osmotic pressure — ISF (e.g., 3 mm Hg).
Control of Fluid Balance

- Thirst mechanism // Osmoreceptors in the hypothalamus

- Antidiuretic hormone // Promotes resorption of water into blood from kidney tubules

- Aldosterone // Determines resorption of sodium ions and water

- Atrial natriuretic peptide // Regulates fluid, sodium, and potassium levels
Fluid Excess - Edema

- excessive amount of fluid in the interstitial compartment
  - Causes swelling or enlargement of tissue
  - May be localized or throughout the body
  - May impair tissue perfusion
  - May trap drugs in ISF
Capillary Exchange

• Filtration
  - Hydrostatic pressure - IVF (e.g., 30 mm Hg)
  - Hydrostatic pressure - ISF (e.g., 2 mm Hg)

• Diffusion
  - Osmotic pressure - ISF (e.g., 3 mm Hg)
  - Osmotic pressure - blood (e.g., 25 mm Hg)

• Osmosis
  - ICF
  - C - E

• Active Transport
  - P - Protein
  - X - Waste (e.g., urea)
  - C - Carrier
  - E - Energy or ATP

Legend:
- ISF - Interstitial fluid
- ICF - Intracellular fluid
- + - Solute (e.g., Na+, glucose)
- P - Protein
- X - Waste (e.g., urea)
- C - Carrier
- E - Energy or ATP

Causes of Edema

- Increased capillary hydrostatic pressure
  - Caused by higher blood pressure or increased blood volume
  - Forces increased fluid out of capillaries into tissue
  - Cause of pulmonary edema

- Loss of plasma proteins
  - Particularly albumin
  - Results in decreased plasma osmotic pressure
Causes of Edema

From Capalbo-Kirkhorn LC: Pathophysiology, ed 4, St. Louis, 2009, Mosby.
Causes of Edema

- Obstruction of lymphatic circulation // Causes localized edema
  - Excessive fluid and protein not returned to general circulation

- Increased capillary permeability // Usually causes localized edema
  - May result from an inflammatory response or infection
  - Histamines and other chemical mediators increase capillary permeability.

- Can also result from some bacterial toxins or large burn wounds and result in widespread edema
Effects of Edema

● Swelling  //  Pale or red in color

● Pitting edema  //  Presence of excess interstitial fluid
  - Moves aside when pressure is applied by finger
  - Depression—“pit” remains when finger is removed

● Increase in body weight  //  With generalized edema
Effects of Edema (cont’d.)
Effects of Edema

- Functional impairment
  - Restricts range of joint movement
  - Reduced vital capacity
  - Impaired diastole

- Pain
  - Edema exerts pressure on nerves locally.
  - Headache with cerebral edema
  - Stretching of capsule in organs (kidney, liver)

- Impaired arterial circulation  //  Ischemia leading to tissue breakdown
Effects of Edema (cont’d.)

- Dental practice
  - Difficult to take accurate impressions
  - Dentures do not fit well

- Edema in skin
  - Susceptible to tissue breakdown from pressure
Fluid Deficit - Dehydration

- Insufficient body fluid
  - Inadequate intake
  - Excessive loss
  - Both

- Fluid loss often measured by change in body weight

- Dehydration more serious in infants and older adults

- Water loss may be accompanied by loss of electrolytes and proteins (e.g., diarrhea).
Causes of Dehydration

- Vomiting and diarrhea
- Excessive sweating with loss of sodium and water
- Diabetic ketoacidosis // Loss of fluid, electrolytes, and glucose in the urine
- Insufficient water intake in older adults or unconscious persons
- Use of concentrated formula in infants
Effects of Dehydration

- Dry mucous membranes in the mouth
- Decreased skin turgor or elasticity
- Lower blood pressure, weak pulse, and fatigue
- Decreased mental function, confusion, loss of consciousness
Manifestations of Dehydration

Sunken eyes

- Sunken fontanelles in infant
- Lower blood pressure, rapid weak pulse
- Increased hematocrit
- Increased temperature
- Decreasing level of consciousness
- Urine—low volume and high specific gravity
Dehydration, Thirst, and Rehydration
### TABLE 2-3 Comparison of Signs and Symptoms of Fluid Excess (Edema) and Fluid Deficit (Dehydration)

<table>
<thead>
<tr>
<th>Fluid Excess (Edema)</th>
<th>Fluid Deficit (Dehydration)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Localized swelling (feet, hands, periorbital area, ascites)</td>
<td>Sunken, soft eyes</td>
</tr>
<tr>
<td>Pale, gray, or red skin color</td>
<td>Decreased skin turgor, dry mucous membranes</td>
</tr>
<tr>
<td>Weight gain</td>
<td>Thirst, weight loss</td>
</tr>
<tr>
<td>Slow, bounding pulse; high blood pressure</td>
<td>Rapid, weak, thready pulse, low blood pressure, and orthostatic hypotension</td>
</tr>
<tr>
<td>Lethargy, possible seizures</td>
<td>Fatigue, weakness, dizziness, possible stupor</td>
</tr>
<tr>
<td>Pulmonary congestion, cough, rales</td>
<td>Increased body temperature</td>
</tr>
<tr>
<td>Laboratory values:</td>
<td>Laboratory values:</td>
</tr>
<tr>
<td>Decreased hematocrit</td>
<td>Increased hematocrit</td>
</tr>
<tr>
<td>Decreased serum sodium</td>
<td>Increased electrolytes (or variable)</td>
</tr>
<tr>
<td>Urine: low specific gravity, high volume</td>
<td>Urine: high specific gravity, low volume</td>
</tr>
</tbody>
</table>

Note: Signs may vary depending on the cause of the imbalance.

Attempts to Compensate for Fluid Loss

- Increasing thirst
- Increasing heart rate
- Constriction of cutaneous blood vessels
- Producing less urine
- Concentration of urine
Third-Spacing of Fluid

- Fluid shifts out of the blood into a body cavity or tissue and can no longer re-enter vascular compartment.
  - High osmotic pressure of ISF, as in burns
  - Increased capillary permeability, as in some gram-negative infections
### TABLE 2-4 Distribution of Major Electrolytes

<table>
<thead>
<tr>
<th>Ions</th>
<th>Intracellular (mEq/L)</th>
<th>Blood (mEq/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cations</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodium (Na)</td>
<td>10</td>
<td>142</td>
</tr>
<tr>
<td>Potassium (K)</td>
<td>160</td>
<td>4</td>
</tr>
<tr>
<td>Calcium (Ca)</td>
<td>Variable</td>
<td>5</td>
</tr>
<tr>
<td>Magnesium (Mg)</td>
<td>35</td>
<td>3</td>
</tr>
<tr>
<td><strong>Anions</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bicarbonate (HCO₃⁻)</td>
<td>8</td>
<td>27</td>
</tr>
<tr>
<td>Chloride (Cl⁻)</td>
<td>2</td>
<td>103</td>
</tr>
<tr>
<td>Phosphate (HPO₄²⁻)</td>
<td>140</td>
<td>2</td>
</tr>
</tbody>
</table>

Note: There are variations in “normal” values among individuals. The concentration of electrolytes in plasma varies slightly from that in the interstitial fluid or other types of extracellular fluids. The number of anions, including those present in small quantities, is equivalent to the concentration of cations in the intracellular compartment (or the plasma) so as to maintain electrical neutrality (equal negative and positive charges) in any compartment.
Movements of Electrolytes Between Compartments

From Copstead-Kirkom LC: Pathophysiology, ed 4, St. Louis, 2009, Mosby.
Sodium Imbalance

- Review of sodium
  - Primary cation in ECF
  - Sodium diffuses between vascular and interstitial fluids.
  - Transport out of cells by sodium-potassium pump (3 Na out and 2 K in)
  - Actively secreted into mucus and other secretions
  - Exists in form of sodium chloride and sodium bicarbonate
  - Ingested in food and beverages
Sodium

● DRI is 0.5 g/day

● Typical American consumes 5 to 7 mg/day

● Adult male secretes 5 g of sodium per day

● Under influence of aldosterone the urine can be sodium free

● Antiport exchanges sodium for potassium
Hyponatremia

- Causes

- Losses from excessive sweating, vomiting, diarrhea
- Use of certain diuretic drugs combined with low-salt diet
- Hormonal imbalances
  - Insufficient aldosterone
  - Adrenal insufficiency
  - Excess ADH secretion
- Diuresis
- Excessive water intake
Effects of Hyponatremia

- Low sodium levels // Cause fluid imbalance in compartments
  - Fatigue, muscle cramps, abdominal discomfort or cramps, nausea, vomiting

- Decreased osmotic pressure in ECF compartment
  - Fluid shift into cells // Hypovolemia and decreased blood pressure
  - Cerebral edema // Confusion, headache, weakness, seizures
Hyponatremia and Fluid Shift into Cells

1. Low sodium concentration in blood
2. Low osmotic pressure in extracellular fluids
3. Water shifts out of blood
4. More water shifts into cell (from low to high osmotic pressure)
5. Cell swells, function decreases, and then cell ruptures

Low sodium concentration in ISF
Low osmotic pressure in ISF
Interstitial fluid

Hypernatremia

• Cause is imbalance in sodium and water

  ➢ Insufficient ADH (diabetes insipidus) // Results in large volume of dilute urine

  ➢ Failure of the thirst mechanism

  ➢ Watery diarrhea & prolonged periods of rapid respiration

  ➢ Ingestion of large amounts of sodium without enough water
Effects of Hypernatremia

- Weakness & agitation
- Dry, rough mucous membranes
- Edema
- Increased thirst (if thirst mechanism is functional)
- Increased blood pressure
Review of Potassium Imbalance

- Major intracellular cation
- Serum levels are low, with a narrow range.
- Excreted primarily in urine / but retained by antiport when kidneys excrete H+
- Insulin promotes movement of potassium into cells
- Level influenced by acid-base balance
- Excess potassium ions in interstititial fluid may lead to hyperkalemia.
- Abnormal potassium levels (high or low) cause changes in cardiac conduction and are life-threatening!
<table>
<thead>
<tr>
<th>Hypokalemia</th>
<th>Hyperkalemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac arrhythmias, cardiac arrest</td>
<td>Arrhythmias, cardiac arrest</td>
</tr>
<tr>
<td>Anorexia, nausea, constipation</td>
<td>Nausea, diarrhea</td>
</tr>
<tr>
<td>Fatigue, muscle twitch, weakness, leg cramps</td>
<td>Muscle weakness, paralysis beginning in legs</td>
</tr>
<tr>
<td>Shallow respirations, paresthesias</td>
<td>Paresthesias—fingers, toes, face, tongue</td>
</tr>
<tr>
<td>Postural hypotension, polyuria, and nocturia</td>
<td>Oliguria</td>
</tr>
<tr>
<td>Serum pH elevated—7.45 (alkalosis)</td>
<td>Serum pH decreased—7.35 (acidosis)</td>
</tr>
</tbody>
</table>
Role of sodium and potassium ions in the conduction of an impulse (action potentials)

1. Polarization
   Resting state of semipermeable membrane

2. Depolarization
   Stimulus opens Na⁺ channels, Na⁺ moves into cell

3. Repolarization
   As impulse moves along membrane, Na⁺ channels close and K⁺ channels open, allowing K⁺ to move outward

4. Return to Resting State
   Channels close. Sodium-potassium pump returns Na⁺ outside cell and K⁺ inside cell
Causes of Hypokalemia

- Definition of hypokalemia // Serum K⁺ < 3.5 mEq/L

- Causes
  - Excessive losses caused by diarrhea
  - Diuresis associated with some diuretic drugs
  - Excessive aldosterone or glucocorticoids // Example: Cushing syndrome
  - Decreased dietary intake // May occur with alcoholism, eating disorders, starvation
  - Treatment of diabetic ketoacidosis with insulin
Effects of Hypokalemia

- Cardiac dysrhythmias // Caused by impaired repolarization leading to cardiac arrest
- Interference with neuromuscular function // Muscles less responsive to stimuli
- Paresthesias—“pins and needles”
- Decreased digestive tract motility
- Severe hypokalemia results in: Shallow respirations // Failure to concentrate urine and polyuria
Causes of Hyperkalemia

- Definition of hyperkalemia // Serum K⁺ > 5 mEq/L

- Causes
  - Renal failure
  - Deficit of aldosterone
  - “Potassium-sparing” diuretics
  - Leakage of intracellular potassium into extracellular fluids // In patients with extensive tissue damage
  - Displacement of potassium from cells by prolonged or severe acidosis
Relationship of Hydrogen and Potassium Ions
Effects of Hyperkalemia

- Cardiac dysrhythmias // May progress to cardiac arrest (how dogs are killed)

- Muscle weakness common
  - Progresses to paralysis
  - May cause respiratory arrest
  - Impairs neuromuscular activity

- Fatigue, nausea, paresthesias
Calcium Imbalance

- Review of calcium
  - Important extracellular cation
  - Ingested in food
  - Stored in bone
  - Excreted in urine and feces
  - Balance controlled by parathyroid hormone (PTH) and calcitonin
  - Vitamin D promotes calcium absorption from intestine // Ingested or synthesized in skin in the presence of ultraviolet rays // Activated in kidneys
Functions of Calcium

- Provides structural strength for bones and teeth
- Maintenance of the stability of nerve membranes
- Required for muscle contractions
- Necessary for many metabolic processes and enzyme reactions
- Essential for blood clotting
Causes of Hypocalcemia

- Hypoparathyroidism
- Malabsorption syndrome
- Deficient serum albumin
- Increased serum pH level
- Renal failure
Effects of Hypocalcemia

- Increase in the permeability and excitability of nerve membranes
  - Spontaneous stimulation of skeletal muscle
    - Muscle twitching
    - Carpopedal spasm
  - Tetany

- Weak heart contractions
  - Delayed conduction // Leads to dysrhythmias and decreased blood pressure
Causes of Hypercalcemia

- Uncontrolled release of calcium ions from bones // Neoplasms—malignant bone tumors

- Hyperparathyroidism

- Demineralization caused by immobility // Decrease stress on bone > decrease osteoblast activity

- Increased calcium intake // Excessive vitamin D // or excess dietary calcium

- Milk-alkali syndrome
Effects of Hypercalcemia

- Depressed neuromuscular activity
  - Muscle weakness, loss of muscle tone
  - Lethargy, stupor, personality changes
  - Anorexia, nausea

- Interference with ADH function // less absorption of water // decrease in renal function

- Increased strength in cardiac contractions // dysrhythmias may occur.
Magnesium Imbalances

- **Intracellular ion**

- **Hypomagnesemia**
  - Results from malabsorption or malnutrition; often associated with alcoholism
  - Caused by use of diuretics, diabetic ketoacidosis, hyperthyroidism, hyperaldosteronism

- **Hypermagnesemia**
  - Occurs with renal failure
  - Depresses neuromuscular function
  - Decreased reflexes
Phosphate Imbalances

- Bone and tooth mineralization
- Important in metabolism—ATP
- Phosphate buffer system—acid-base balance
- Integral part of the cell membrane
- Reciprocal relationship with serum calcium
- Hypophosphatemia // Malabsorption syndromes, diarrhea, excessive antacids
- Hyperphosphatemia // From renal failure
Chloride Imbalance

- Major extracellular anion
- Chloride levels related to sodium levels
- Chloride and bicarbonate ions can shift in response to acid-base imbalances.
- Hypochloremia // Usually associated with alkalosis // Early stages of vomiting—loss of hydrochloric acid
- Hyperchloremia // Excessive sodium chloride intake
Hydrogen Ion and pH Scale

Serum pH

7.35  7.4  7.45

NORMAL RANGE

Acidosis
Increased H⁺

Alkalosis
Decreased H⁺

6.8  7.8
Death  Death
Control of Serum pH

- Buffer pairs in the blood respond to pH changes immediately. (chemical buffers)

- Respiratory system can alter carbonic acid levels to change pH. (physiologic buffer)

- Kidneys can modify the excretion rate of acids and absorption of bicarbonate ions to regulate pH. (physiologic buffer)

  - Most significant control mechanism // move pH more

  - Slowest mechanism
Buffer Systems

- Sodium bicarbonate–carbonic acid system
  - Major ECF buffer
  - Controlled by the respiratory system and kidneys

- Other buffering systems:
  - Phosphate
  - Serum protein / hemoglobin
Compensation Mechanisms for pH Imbalance

- Compensation limited, usually short term
- *Does not remove the cause of imbalance*

- Compensation occurs to balance the *relative proportion* of hydrogen ions and bicarbonate ions in circulation:
  - Buffers
  - Change in respiration
  - Change in renal function
Decompensation

- Occurs when:
  - Causative problem becomes more severe
  - Additional problems occur
  - Compensation mechanisms are exceeded or fail

- Requires intervention to maintain homeostasis

- LIFE-THREATENING!
Acid-Base Imbalance

- Acidosis (below pH 7.35)
  - Excess hydrogen ions
  - Decrease in serum pH

- Alkalosis (above pH 7.45)
  - Deficit of hydrogen ions
  - Increase in serum pH
<table>
<thead>
<tr>
<th>Respiratory</th>
<th>Acidosis</th>
<th>Alkalosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Causes</td>
<td>Slow shallow respirations (e.g., drugs)</td>
<td>Hyperventilation (anxiety, aspirin overdose)</td>
</tr>
<tr>
<td></td>
<td>Respiratory congestion</td>
<td></td>
</tr>
<tr>
<td>Effect</td>
<td>Increased PCO₂</td>
<td>Decreased PCO₂</td>
</tr>
<tr>
<td>Compensation</td>
<td>Kidneys excrete more hydrogen ion and reabsorb more bicarbonate</td>
<td>Kidneys excrete less hydrogen ion and reabsorb less bicarbonate</td>
</tr>
<tr>
<td>Laboratory</td>
<td>Elevated PCO₂</td>
<td>Low PCO₂</td>
</tr>
<tr>
<td></td>
<td>Elevated serum bicarbonate</td>
<td>Low serum bicarbonate</td>
</tr>
<tr>
<td></td>
<td>Compensated—serum pH = 7.35 to 7.4</td>
<td>Compensated—serum pH = 7.4 to 7.45</td>
</tr>
<tr>
<td></td>
<td>Decompensated—serum pH &lt; 7.5</td>
<td>Decompensated—serum pH &gt; 7.45</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Metabolic</th>
<th>Acidosis</th>
<th>Alkalosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Causes</td>
<td>Shock</td>
<td>Vomiting (early stage)</td>
</tr>
<tr>
<td></td>
<td>Diabetic ketoacidosis</td>
<td>Excessive antacid intake</td>
</tr>
<tr>
<td></td>
<td>Renal failure</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Diarrhea</td>
<td></td>
</tr>
<tr>
<td>Effect</td>
<td>Decreased serum bicarbonate ion</td>
<td>Increased serum bicarbonate ion</td>
</tr>
<tr>
<td>Compensation</td>
<td>Rapid, deep respirations</td>
<td>Slow, shallow respirations</td>
</tr>
<tr>
<td></td>
<td>Kidneys excrete more acid and increase bicarbonate absorption</td>
<td>Kidneys excrete less acid and decrease bicarbonate absorption</td>
</tr>
<tr>
<td>Laboratory</td>
<td>Low serum bicarbonate</td>
<td>Elevated serum bicarbonate</td>
</tr>
<tr>
<td></td>
<td>Low PCO₂</td>
<td>Elevated PCO₂</td>
</tr>
<tr>
<td></td>
<td>Compensated—serum pH = 7.35 to 7.4</td>
<td>Compensated—serum pH = 7.4 to 7.45</td>
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<tr>
<td></td>
<td>Decompensated—serum pH &lt; 7.35</td>
<td>Decompensated—serum pH &gt; 7.45</td>
</tr>
</tbody>
</table>
Respiratory Acidosis

- Acute problems
  - Pneumonia, airway obstruction, chest injuries
  - Drugs that depress the respiratory control center

- Chronic respiratory acidosis // Common with chronic obstructive pulmonary disease

- Decompensated respiratory acidosis // May develop if impairment becomes severe or if compensation mechanisms fail
Metabolic Acidosis

• Excessive loss of bicarbonate ions to buffer hydrogen // Diarrhea—loss of bicarbonate from intestines

• Increased use of serum bicarbonate

• Renal disease or failure
  - Decreased excretion of acids
  - Decreased production of bicarbonate ions

• Decompensated metabolic acidosis // Additional factor interferes with compensation.
Effects of Acidosis

- Impaired nervous system function
  - Headache
  - Lethargy
  - Weakness
  - Confusion
  - Coma and death

- Compensation
  - Deep rapid breathing
  - Secretion of urine with a low pH
Changes in Blood Gases with Acidosis
Changes in Blood Gases with Acidosis
Changes in Blood Gases with Acidosis

1. Metabolic balance before onset of acidosis
   - $\text{H}_2\text{CO}_3$: Carbonic acid
   - $\text{HCO}_3^-$: Bicarbonate ion
   - (Na⁺ - HCO₃⁻)
   - (K⁺ - HCO₃⁻)
   - (Mg⁺⁺ - HCO₃⁻)
   - (Ca²⁺ - HCO₃⁻)

2. Respiratory acidosis
   - Breathing is suppressed, holding CO₂ in body

3. Body's compensation
   - Kidneys conserve HCO₃⁻ ions and eliminate H⁺ ions in acidic urine

4. Therapy required to restore metabolic balance
   - Lactate solution used
1. Metabolic balance before onset of alkalosis

\[
\text{H}_2\text{CO}_3 \quad \text{HCO}_3^- \\
\text{Carbonic acid} \\
\text{HCO}_3^- \quad \text{Bicarbonate ion} \\
(\text{Na}^+ \cdot \text{HCO}_3^-) \\
(\text{K}^+ \cdot \text{HCO}_3^-) \\
(\text{Mg}^{2+} \cdot \text{HCO}_3^-) \\
(\text{Ca}^{2+} \cdot \text{HCO}_3^-)
\]

2. Respiratory alkalosis

Hyperactive breathing "blows off" \( \text{CO}_2 \)

3. Body's compensation

Kidneys conserve \( \text{H}^+ \) ions and eliminate \( \text{HCO}_3^- \) in alkaline urine

4. Therapy required to restore metabolic balance

HCO_3^- ions are replaced by Cl^- ions

From Patton KT, Thibodeau GA. Anatomy & Physiology, ed 8, St. Louis, 2013. Mosby
# TABLE 2-9 Examples of Acidosis

<table>
<thead>
<tr>
<th>Respiratory Acidosis—Individual with Emphysema Retaining CO₂</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1: Kidneys compensate for slight increase in PCO₂ by increasing excretion of acids and production of bicarbonate</td>
<td>No change in serum levels</td>
</tr>
<tr>
<td>Stage 2: Increased retention of CO₂. Respiratory acidosis</td>
<td>Elevated PCO₂</td>
</tr>
<tr>
<td>Stage 3: Compensation. Kidneys reabsorb more bicarbonate</td>
<td>Elevated serum ions. bicarbonate</td>
</tr>
<tr>
<td>Stage 4: Compensated respiratory acidosis: Abnormal serum values indicate problem and compensation adequate to maintain ratio and normal serum pH.</td>
<td>Serum pH = 7.35</td>
</tr>
<tr>
<td>Stage 5: Decompensated respiratory acidosis: Patient acquires pneumonia, and much more CO₂ is retained. Also, kidneys cannot maintain compensation. Ratio is no longer normal, CNS depression, coma, and serum pH drops below the normal range.</td>
<td>Serum pH = 7.31</td>
</tr>
</tbody>
</table>
Alkalosis

- Respiratory alkalosis
  - Hyperventilation
    - Caused by anxiety, high fever, overdose of aspirin
    - Head injuries
    - Brainstem tumor

- Metabolic alkalosis
  - Increase in serum bicarbonate ion
    - Loss of hydrochloric acid from stomach
    - Hypokalemia
    - Excessive ingestion of antacids
Effects of Alkalosis

- Increased irritability of the nervous system causes:
  - Restlessness
  - Muscle twitching
  - Tingling and numbness of the fingers
  - Tetany
  - Seizures
  - Coma
Treatment of Imbalances

- Treatment of underlying cause

- Immediate corrective measures to include fluid and electrolyte replacement or removal
  - Caution is required when adjusting fluid levels to ensure appropriate electrolyte balance.

- Addition of bicarbonate to the blood to reverse acidosis

- Modification of diet to maintain better electrolyte balance
Secretion and Effects of ADH
blood volume and fluid intake

kidneys compensate very well for excessive fluid intake, but not for inadequate fluid intake.
Electrolyte Concentrations

(a) Blood plasma

- Na⁺: 145 mEq/L
- K⁺: 4 mEq/L
- Cl⁻: 103 mEq/L
- Ca²⁺: 5 mEq/L
- Pi: < 1 mEq/L
- Osmolarity: 300 mOsm/L

(b) Intracellular fluid

- Na⁺: 12 mEq/L
- K⁺: 150 mEq/L
- Cl⁻: 4 mEq/L
- Ca²⁺: 75 mEq/L
- Pi: < 1 mEq/L
- Osmolarity: 300 mOsm/L
Acid-Base Balance

\[
\begin{align*}
&\text{H}_2\text{CO}_3 \\
&\text{HCO}_3^- \\
&\text{pH} 7.35 \\
&\text{Normal} \\
&\text{7.45} \\
&\text{Acidosis} \quad \text{Death} 6.8 \\
&\text{Alkalosis} \quad \text{Death} 8.0
\end{align*}
\]
H+ Secretion and Excretion in Kidney

2. H2CO3 decomposes into H2O and CO2, which enter the tubule cell.
3. Tubule cells acquire CO2 from blood, tubular fluid, and their own aerobic respiration.
4. Carbonic anhydrase (CAH) combines H2O and CO2 to re-form H2CO3.
5. H2CO3 ionizes to form HCO3– (which returns to the blood) and H+.
7. NaHCO3 from glomerular filtrate decomposes into Na+ and HCO3–. Na+ is pumped into tubule cell.
8. Na+ is removed by Na+–K+ pump at the base of the cell.
9. HCO3– reacts with H+ from tubule cell to form H2CO3.
10. CAH on brush border decomposes H2CO3 to form H2O and CO2.
11. CO2 enters the tubular cell and H2O passes in the urine (carrying the H+ that was originally in the blood).
(a) Acidosis leading to Hyperkalemia
(b) Alkalosis → leading to → Hypokalemia
Secretion and Effects of Aldosterone

- Hypotension
- Hyponatremia
- Hyperkalemia

Stimulates adrenal cortex to secrete aldosterone

Stimulates renal tubules

- Increases Na+ reabsorption
- Less Na+ and H2O in urine
- Supports existing fluid volume and Na+ concentration pending oral intake

Increases K+ secretion
- More K+ in urine

Negative feedback loop
Potassium & Membrane Potentials

(a) Normokalemia

- K+ concentrations in equilibrium
- Equal diffusion into and out of cell
- Normal resting membrane potential (RMP)

Hyperkalemia

- Elevated extracellular K+ concentration
- Less diffusion of K+ out of cell
- Elevated RMP (cells partially depolarized)
- Cells more excitable

Hypokalemia

- Reduced extracellular K+ concentration
- Greater diffusion of K+ out of cell
- Reduced RMP (cells hyperpolarized)
- Cells less excitable