Fluid, Electrolyte, and Acid-Base Balance
Outline

26.1 Body fluids consist of water and solutes in three main compartments (pp. 999–1001; Figs. 26.1–26.3)

A. Body Water Content (p. 999)
   1. Total body water is a function of age, body mass, gender, and body fat.
      a. Due to their low body fat and bone mass, infants are about 73% water.
      b. The body water content of men is about 60%, but because women have relatively more body fat and less skeletal muscle than men, theirs is about 50%.
   2. Body water declines throughout life, ultimately comprising about 45% of total body mass in old age.

B. Fluid Compartments (p. 999; Fig. 26.1)
   1. There are two main fluid compartments of the body: the intracellular compartment, containing slightly less than two-thirds by volume, and the remaining third, distributed in the extracellular fluid.
   2. There are two subcompartments of the extracellular fluid: blood plasma and interstitial fluid.

C. Composition of Body Fluids (pp. 999–1001; Fig. 26.2)
   1. Nonelectrolytes include most organic molecules, do not dissociate in water, and carry no net electrical charge.
   2. Electrolytes dissociate in water to ions and include inorganic salts, acids and bases, and some proteins.
   3. Electrolytes have greater osmotic power because they dissociate in water and contribute at least two particles to solution.
   4. The major cation in extracellular fluids is sodium, and the major anion is chloride.
   5. The major cation in intracellular fluid is potassium, and the major anion is phosphate.
   6. Cells contain substantially more soluble proteins than extracellular fluids.
   7. Electrolytes are the most abundant solutes in body fluids, but proteins and some nonelectrolytes account for 60–97% of dissolved solutes.

D. Fluid Movement Among Compartments (p. 1001; Fig. 26.3)
   1. Anything that changes solute concentration in any compartment leads to net water flows.
   2. Substances must pass through both the plasma and interstitial fluid in order to reach the intracellular fluid, and exchanges between these compartments occur almost continuously, leading to compensatory shifts from one compartment to another.
      a. Exchanges between plasma and IF occur across capillaries, with hydrostatic pressure forcing nearly protein-free fluid to the IF, followed by nearly complete reabsorption into the blood from the IF.
      b. Exchanges between the IF and ICF are dependent on the exact permeabilities of the membrane; two-way water flow is substantial, but
ion movements are restricted, and nutrients, respiratory gases, and wastes typically occur in one direction.

26.2 Both intake and output of water are regulated (pp. 1001–1005; Figs. 26.4–26.7)

A. For the body to remain properly hydrated, water intake must equal water output. (pp. 1001–1002; Fig. 26.4)
   1. Most water enters the body through ingested liquids and food, but is also produced by cellular metabolism.
   2. Water output is due to evaporative loss from lungs and skin (insensible water loss), sweating, defecation, and urination.

B. Regulation of Water Intake (pp. 1002–1003; Fig. 26.5)
   1. The thirst mechanism is triggered by a decrease in plasma osmolality, which results in a dry mouth and excites the hypothalamic thirst center.
   2. Thirst is quenched as the mucosa of the mouth is moistened and continues with distention of the stomach and intestines, resulting in inhibition of the hypothalamic thirst center.

C. Regulation of Water Output (p. 1003)
   1. Drinking is necessary because there is obligatory water loss due to the insensible water losses, water lost with food residues and feces, and a minimum 500 ml sensible water loss in urine, due to the demand to flush urine solutes.
   2. Beyond obligatory water losses, solute concentration and volume of urine depend on fluid intake, diet, and water losses by other routes.

D. Influence of Antidiuretic Hormone (ADH) (pp. 1003–1004; Fig. 26.6)
   1. The amount of water reabsorbed in the renal collecting ducts is proportional to ADH release.
      a. When ADH levels are low, most water in the collecting ducts is not reabsorbed, resulting in large quantities of dilute urine.
      b. When ADH levels are high, filtered water is reabsorbed, resulting in a lower volume of concentrated urine.
   2. ADH secretion is promoted or inhibited by the hypothalamus in response to changes in solute concentration of extracellular fluid, large changes in blood volume or pressure, or vascular baroreceptors.

E. Disorders of Water Balance (p. 1005; Fig. 26.7)
   1. Dehydration occurs when water output exceeds water intake and may lead to weight loss, fever, mental confusion, or hypovolemic shock.
   2. Hypotonic hydration is a result of renal insufficiency, or intake of an excessive amount of water very quickly.
   3. Edema is the accumulation of fluid in the interstitial space, which may impair tissue function.

26.3 Sodium, potassium, calcium, and phosphate levels are tightly regulated (pp. 1005–1011; Figs. 26.8–26.10; Tables 26.1–26.2)

A. The Central Role of Sodium in Fluid and Electrolyte Balance (pp. 1005–1007; Table 26.1)
   1. Sodium is the most important cation in regulation of fluid and electrolyte balance in the body due to its abundance and osmotic pressure.
   2. Because all body fluids are in chemical equilibrium, any change in sodium levels causes a compensatory shift in water, affecting plasma volume, blood pressure, and intracellular and interstitial fluid volumes.
3. Both the concentration of \( \text{Na}^+ \) in the body and total body content of \( \text{Na}^+ \) are important.
   a. The concentration of sodium ions in the ECF influences neural and muscular excitability.
   b. Total body content of sodium determines ECF volume and blood pressure.

B. Regulation of Sodium Balance (pp. 1007–1009; Figs. 26.8–26.10; Table 26.2)
1. When aldosterone secretion is high, nearly all the filtered sodium is reabsorbed in the distal convoluted tubule and the collecting duct.
   a. Absorption of sodium ions creates osmotic absorption of water, while sodium excretion also causes water loss to the urine.
2. The most important trigger for the release of aldosterone is the renin-angiotensin-aldosterone mechanism, initiated in response to sympathetic stimulation, a decrease in filtrate osmolality, or decreased blood pressure.
   a. Low blood volume and pressure trigger renin release from granular cells of the juxtaglomerular complex.
   b. Renin catalyzes the initial step in the conversion of angiotensin II.
   c. Angiotensin II, produced by the renin-angiotensin-aldosterone mechanism, causes the adrenal cortex to release aldosterone and also directly causes kidney tubules to increase \( \text{Na}^+ \) retention as part of a mechanism regulating systemic blood pressure.
3. Atrial natriuretic peptide, released in response to increased stretch of atrial cells, reduces blood pressure and blood volume by inhibiting release of ADH, renin, and aldosterone and directly causing vasodilation.
4. Estrogens are chemically similar to aldosterone and enhance reabsorption of salt by the renal tubules.
5. Progesterone decreases \( \text{Na}^+ \) absorption by blocking the action of aldosterone on renal tubules.
6. Glucocorticoids enhance tubular reabsorption of sodium, but increase glomerular filtration.
7. Cardiovascular baroreceptors monitor vessels in the heart, neck, and thorax; when blood pressure drops, these signal the cardiovascular centers in the brain, resulting in reduced sympathetic stimulation of the kidneys, allowing afferent vessels to dilate and increase glomerular filtration.

C. Regulation of Potassium Balance (pp. 1010; Table 26.1)
1. Potassium is critical to the maintenance of the membrane potential of neurons and muscle cells and is a buffer that compensates for shifts of hydrogen ions in or out of the cell.
   a. Since shifts of \( \text{H}^+ \) into or out of the cell result in compensatory shifts of \( \text{K}^+ \) in the opposite direction, ECF potassium levels rise with acidosis, and fall with alkalosis.
2. Potassium balance is chiefly regulated by renal mechanisms, which control the amount of potassium secreted into the filtrate.
   a. 60–80% of filtered \( \text{K}^+ \) is absorbed in the proximal tubule, the thick ascending limb absorbs a further 10–20%, regardless of need; final \( \text{K}^+ \)
levels are determined by changing $K^+$ secretion by principal cells of the collecting ducts.

3. Blood plasma levels of potassium are the most important factor regulating potassium secretion.

4. Aldosterone influences potassium secretion because potassium secretion is simultaneously enhanced when sodium reabsorption increases.

D. Regulation of Calcium and Phosphate Balance (pp. 1010–1011; Table 26.1)
1. Calcium ion levels are closely regulated by parathyroid hormone and calcitonin; about 98% is reabsorbed.
   a. Parathyroid hormone is released when blood calcium levels decline and targets the bones, small intestine, and kidneys.
   b. Calcitonin is an antagonist to parathyroid hormone and is released when blood calcium rises, targeting bone.

E. Regulation of Anions (p. 1011)
1. Chloride is the major anion reabsorbed with sodium and helps maintain the osmotic pressure of the blood.
2. When acidosis occurs, less $Cl^-$ is absorbed, because $Na^+$ absorption is then coupled to $HCO_3^-$ absorption to buffer pH.

24.4 Chemical buffers and respiratory regulation rapidly minimize pH changes (pp. 1011–1014; Fig. 26.11)

A. Because of the abundance of hydrogen bonds in the body’s functional proteins, they are strongly influenced by hydrogen ion concentration (pp. 1011–1012).
1. When arterial blood pH rises above 7.45, the body is in alkalosis; when arterial pH falls below 7.35, the body is in physiological acidosis.
2. Although $H^+$ can enter the body via ingested foods, most hydrogen ions originate as metabolic by-products:
   a. Protein breakdown releases phosphoric acid.
   b. Anaerobic respiration of glucose produces lactic acid.
   c. Fat metabolism yields fatty acids and ketones.
   d. Transport of $CO_2$ as bicarbonate releases $H^+$.

B. Chemical Buffer Systems (pp. 1004–1012–1013; Fig. 26.11)
1. A chemical buffer is a system of one or two molecules that acts to resist changes in pH by binding $H^+$ when the pH drops or releasing $H^+$ when the pH rises.
2. The bicarbonate buffer system is the main buffer of the extracellular fluid and consists of carbonic acid and its salt, sodium bicarbonate.
   a. When a strong acid is added to the solution, carbonic acid is mostly unchanged, but bicarbonate ions of the salt bind excess $H^+$, forming more carbonic acid.
   b. When a strong base is added to solution, the sodium bicarbonate remains relatively unaffected, but carbonic acid dissociates further, donating more $H^+$ to bind the excess hydroxide.
   c. Bicarbonate concentration of the extracellular fluid is closely regulated by the kidneys, and plasma bicarbonate concentrations are controlled by the respiratory system.
3. The phosphate buffer system operates in the urine and intracellular fluid similarly to the bicarbonate buffer system: Sodium dihydrogen phosphate is its weak acid, and monohydrogen phosphate is its weak base.

4. The protein buffer system consists of organic acids containing carboxyl groups that dissociate to release $\text{H}^+$ when the pH begins to rise or bind excess $\text{H}^+$ when the pH declines.

C. Respiratory Regulation of $\text{H}^+$ (pp. 1013–1014)

1. The respiratory and renal systems are physiological buffer systems that control pH by regulating the amount of acid or base in the body.
   a. Physiological buffer systems act more slowly than chemical buffer systems, but have a much greater buffering power.

2. Carbon dioxide from cellular metabolism enters erythrocytes and is converted to bicarbonate ions for transport in the plasma.

3. In the lungs, $\text{CO}_2$ is expelled from the lungs, causing a reversal of bicarbonate to water; the result is that $\text{CO}_2$ has little effect on blood pH.

4. When hypercapnia occurs, blood pH drops, activating medullary respiratory centers, resulting in increased rate and depth of breathing and increased unloading of $\text{CO}_2$ in the lungs.

5. When blood pH rises, the respiratory center is depressed, allowing $\text{CO}_2$ to accumulate in the blood, lowering pH.

26.5 Renal regulation is a long-term mechanism for controlling acid-base balance (pp. 1014–1017; Figs. 26.12–26.14)

A. Only the kidneys can rid the body of acids generated by cellular metabolism, while also regulating blood levels of alkaline substances and renewing chemical buffer components. (pp. 1014–1015; Figs. 26.12–26.14)
   1. Bicarbonate ions can be conserved from filtrate when depleted or newly generated by the kidneys, and their reabsorption is dependent on $\text{H}^+$ secretion.
   2. Type A intercalated cells of the renal tubules can synthesize new bicarbonate ions while excreting more hydrogen ions.
   3. Ammonium ions are weak acids that are excreted and lost in urine, replenishing the alkaline reserve of the blood.
   4. When the body is in alkalosis, type B intercalated cells excrete bicarbonate and reclaim hydrogen ions.

26.6 Abnormalities of acid-base balance are classified as metabolic or respiratory (pp. 1017–1020; Table 26.3)

A. Respiratory Acidosis and Alkalosis (p. 1017; Table 26.3)
   1. Respiratory acidosis is characterized by falling blood pH and rising $P_{\text{CO}_2}$, which can result from shallow breathing or some respiratory diseases.
   2. Respiratory alkalosis results when carbon dioxide is eliminated from the body faster than it is produced due to hyperventilation.

B. Metabolic acidosis and alkalosis is related to any factor except loss of $\text{CO}_2$ in the lungs. (p. 1018; Table 26.3)
1. Metabolic acidosis is characterized by low blood pH and bicarbonate levels and is due to excessive loss of bicarbonate ions or ingestion of too much alcohol.

2. Metabolic alkalosis is indicated by rising blood pH and bicarbonate levels and is the result of vomiting or excessive base intake.

C. Effects of Acidosis and Alkalosis (p. 1018)

1. Absolute pH limits for life range from 6.8–7.8.

D. Respiratory and Renal Compensations (p. 1019–1020)

1. Respiratory rate and depth increase during metabolic acidosis and decrease during metabolic alkalosis in order to change the rate of loss of \( \text{CO}_2 \) in the lungs.

2. In renal compensation for respiratory acidosis, plasma bicarbonate ion concentrations increase as the kidneys retain \( \text{HCO}_3^- \) to compensate for elevated \( \text{P}_{\text{CO}_2} \).

3. In respiratory alkalosis, plasma \( \text{HCO}_3^- \) falls as the kidneys actively excrete it to the urine, to offset low \( \text{P}_{\text{CO}_2} \).

Developmental Aspects of Fluid, Electrolyte, and Acid-Base Balance (p. 1020)

A. An embryo and young fetus are more than 90% water, but as solids accumulate, the percentage declines to about 70–80% at birth.

B. Distribution of body water begins to change at 2 months of age and takes on adult distribution by the time a child is 2 years of age.

C. At puberty, sex differences in body water content appear as males develop more skeletal muscle.

D. During infancy, problems with fluid, electrolyte, and acid-base balance are common, due to low residual volume of infant lungs that can produce large-scale changes in \( \text{P}_{\text{CO}_2} \) and the high rate of fluid intake and output.

E. In old age, body water loss is primarily from the intracellular compartment due to decline in muscle mass and increase in adipose tissue.

F. Increased insensitivity to thirst cues makes the elderly vulnerable to dehydration and to electrolyte or acid-base imbalances.