Chapter 26
Fluid, Electrolyte & Acid-Base Balance
Body Water Content

- Infants: 73% or more water (low body fat, low bone mass)
- Adult males: ~60% water
- Adult females: ~50% water (higher fat content, less skeletal muscle mass)
- Water content declines to ~45% in old age
Fluid Compartments

- Total body water = 40 L
  1. Intracellular fluid (ICF) compartment: 2/3 or 25 L in cells
  2. Extracellular fluid (ECF) compartment: 1/3 or 15 L
    - Plasma: 3 L
    - Interstitial fluid (IF): 12 L in spaces between cells
    - Other ECF: lymph, CSF, humors of the eye, synovial fluid, serous fluid, and gastrointestinal secretions
Figure 26.1

Total body water
Volume = 40 L
60% body weight

Extracellular fluid (ECF)
Volume = 15 L
20% body weight

Intracellular fluid (ICF)
Volume = 25 L
40% body weight

Interstitial fluid (IF)
Volume = 12 L
80% of ECF
Composition of Body Fluids

• Water: the universal solvent
• Solute: nonelectrolytes and electrolytes
  – Nonelectrolytes: most are organic
    • Do not dissociate in water: e.g., glucose, lipids, creatinine, and urea
Composition of Body Fluids

• Electrolytes
  – Dissociate into ions in water; e.g., inorganic salts, all acids and bases, and some proteins
  – The most abundant solutes
  – Have greater osmotic power than nonelectrolytes, so they contribute to fluid shifts
  – Determine the chemical and physical reactions of fluids
Extracellular and Intracellular Fluids

• Each fluid compartment has a distinctive pattern of electrolytes

• ECF
  – All similar, except higher protein content of plasma
    • Major cation: Na$^+$
    • Major anion: Cl$^-$
Extracellular and Intracellular Fluids

- ICF:
  - Low $\text{Na}^+$ and $\text{Cl}^-$
  - Major cation: $\text{K}^+$
  - Major anion $\text{HPO}_4^{2-}$
Extracellular and Intracellular Fluids

- Proteins, phospholipids, cholesterol, and neutral fats make up the bulk of dissolved solutes
  - 90% in plasma
  - 60% in IF
  - 97% in ICF
Blood plasma
Interstitial fluid
Intracellular fluid

**Sodium** (\(\text{Na}^+\))
**Potassium** (\(\text{K}^+\))
**Calcium** (\(\text{Ca}^{2+}\))
**Magnesium** (\(\text{Mg}^{2+}\))
**Bicarbonate** (\(\text{HCO}_3^-\))
**Chloride** (\(\text{Cl}^-\))
**Hydrogen phosphate** (\(\text{HPO}_4^{2-}\))
**Sulfate** (\(\text{SO}_4^{2-}\))

Figure 26.2
Fluid Movement Among Compartments

• Regulated by osmotic and hydrostatic pressures
• Water moves freely by osmosis; osmolalities of all body fluids are almost always equal
• Two-way osmotic flow is substantial
• Ion fluxes require active transport or channels
• Change in solute concentration of any compartment leads to net water flow
Figure 26.3

- **Lungs**: Blood plasma, O₂, CO₂, Nutrients, H₂O, Ions, O₂, CO₂, Nutrients, H₂O, Ions, Nitrogenous wastes.
- **Gastrointestinal tract**: Blood plasma, O₂, CO₂, Nutrients, H₂O, Ions, O₂, CO₂, Nutrients, H₂O, Ions, Nitrogenous wastes.
- **Kidneys**: Blood plasma, O₂, CO₂, Nutrients, H₂O, Ions, O₂, CO₂, Nutrients, H₂O, Ions, Nitrogenous wastes.
Water Balance and ECF

Osmolality

• Water intake = water output = 2500 ml/day
• Water intake: beverages, food, and metabolic water
• Water output: urine, insensible water loss (skin and lungs), perspiration, and feces
Metabolism 10%
Foods 30%
Beverages 60%

Average intake per day:
- Metabolism: 250 ml
- Foods: 750 ml
- Beverages: 1500 ml

Average output per day:
- Feces: 100 ml
- Sweat: 200 ml
- Insensible losses via skin and lungs: 700 ml
- Urine: 1500 ml

Total:
Average intake per day: 2500 ml
Average output per day: 2500 ml
Regulation of Water Intake

• Thirst mechanism is the driving force for water intake
• The hypothalamic thirst center osmoreceptors are stimulated by
  – ↓ Plasma osmolality of 2–3%
  – Angiotensin II or baroreceptor input
  – Dry mouth
  – Substantial decrease in blood volume or pressure
Regulation of Water Intake

• Drinking water creates inhibition of the thirst center
• Inhibitory feedback signals include
  – Relief of dry mouth
  – Activation of stomach and intestinal stretch receptors
Figure 26.5

(*Minor stimulus)

Granular cells
in kidney

Osmoreceptors
in hypothalamus

Hypothalamic
thirst center

Sensation of thirst;
person takes a
drink

Water moistens
mouth, throat;
stretches stomach,
intestine

Water absorbed
from GI tract

Plasma osmolality

Dry mouth

Saliva

Plasma volume*

Blood pressure

Renin-angiotensin
mechanism

Angiotensin II

Plasma osmolality

Initial stimulus

Physiological response

Result

Increases, stimulates

Reduces, inhibits

(*Minor stimulus)
Regulation of Water Output

• Obligatory water losses
  – Insensible water loss: from lungs and skin
  – Feces
  – Minimum daily sensible water loss of 500 ml in urine to excrete wastes

• Body water and Na\(^+\) content are regulated in tandem by mechanisms that maintain cardiovascular function and blood pressure
Regulation of Water Output: Influence of ADH

- Water reabsorption in collecting ducts is proportional to ADH release.
- ↓ ADH $\rightarrow$ dilute urine and ↓ volume of body fluids.
- ↑ ADH $\rightarrow$ concentrated urine.
Regulation of Water Output: Influence of ADH

• Hypothalamic osmoreceptors trigger or inhibit ADH release
• Other factors may trigger ADH release via large changes in blood volume or pressure, e.g., fever, sweating, vomiting, or diarrhea; blood loss; and traumatic burns
Figure 26.6

- **Osmolality**
  - **Na⁺ concentration in plasma**
    - Stimulates
  - **Osmoreceptors in hypothalamus**
    - Negative feedback inhibits
    - Stimulates
    - **Posterior pituitary**
      - Releases
      - **Antidiuretic hormone (ADH)**
        - Targets
        - **Collecting ducts of kidneys**
          - **Water reabsorption**
            - Results in
            - **↓ Osmolality**
            - **Plasma volume**
            - **Scant urine**
  - **Plasma volume**
    - **BP (10–15%)**
      - Inhibits
      - **Baroreceptors in atrium and large vessels**
        - Stimulates
Disorders of Water Balance: Dehydration

• Negative fluid balance (net water loss)
  – ECF water loss due to: hemorrhage, severe burns, prolonged vomiting or diarrhea, profuse sweating, water deprivation, diuretic abuse
  – Signs and symptoms: thirst, dry flushed skin, oliguria
  – May lead to weight loss, fever, mental confusion, hypovolemic shock, and loss of electrolytes
(a) Mechanism of dehydration

1. Excessive loss of $H_2O$ from ECF
2. ECF osmotic pressure rises
3. Cells lose $H_2O$ to ECF by osmosis; cells shrink
Disorders of Water Balance: Hypotonic Hydration

• Cellular overhydration, or water intoxication
• Occurs with renal insufficiency or rapid excess water ingestion
• ECF is diluted → hyponatremia → net osmosis into tissue cells → swelling of cells → severe metabolic disturbances (nausea, vomiting, muscular cramping, cerebral edema) → possible death
(b) Mechanism of hypotonic hydration

1. Excessive \( \text{H}_2\text{O} \) enters the ECF
2. ECF osmotic pressure falls
3. \( \text{H}_2\text{O} \) moves into cells by osmosis; cells swell
Disorders of Water Balance: Edema

• Atypical accumulation of IF fluid $\rightarrow$ tissue swelling
• Due to anything that increases flow of fluid out of the blood or hinders its return
  – $\uparrow$ Blood pressure
  – $\uparrow$ Capillary permeability (usually due to inflammatory chemicals)
  – Incompetent venous valves, localized blood vessel blockage, impaired lymphatic system
  – Congestive heart failure, hypertension, $\uparrow$ blood volume
Edema

• Hindered fluid return occurs with an imbalance in colloid osmotic pressures, e.g., hypoproteinemia (↓ plasma proteins)
  – Fluids fail to return at the venous ends of capillary beds
  – Results from protein malnutrition, liver disease, or glomerulonephritis
Edema

• Blocked (or surgically removed) lymph vessels
  – Cause leaked proteins to accumulate in IF
  – ↑ Colloid osmotic pressure of IF draws fluid from the blood
  – Results in low blood pressure and severely impaired circulation
Electrolyte Balance

- Electrolytes are salts, acids, and bases
- Electrolyte balance usually refers only to salt balance
- Salts enter the body by ingestion and are lost via perspiration, feces, and urine
Electrolyte Balance

• Importance of salts
  – Controlling fluid movements
  – Cellular Excitability
  – Secretory activity
  – Membrane permeability
Central Role of Sodium

• Most abundant cation in the ECF
• Sodium salts in the ECF contribute 280 mOsm of the total 300 mOsm ECF solute concentration
• Na\(^+\) leaks into cells and is pumped out against its electrochemical gradient
• Na\(^+\) content may change but ECF Na\(^+\) concentration remains stable due to osmosis
Central Role of Sodium

• Changes in plasma sodium levels affect
  – Plasma volume, blood pressure
  – ICF and IF volumes

• Renal acid-base control mechanisms are coupled to sodium ion transport
Regulation of Sodium Balance

- No receptors are known that monitor Na\(^+\) levels in body fluids
- Na\(^+\)-water balance is linked to blood pressure and blood volume control mechanisms
Regulation of Sodium Balance: Aldosterone

• $\text{Na}^+$ reabsorption
  – 65% is reabsorbed in the proximal tubules
  – 25% is reclaimed in the loops of Henle

• Aldosterone $\rightarrow$ active reabsorption of remaining $\text{Na}^+$

• Water follows $\text{Na}^+$ if ADH is present
Regulation of Sodium Balance: Aldosterone

• Renin-angiotensin mechanism is the main trigger for aldosterone release
  – Granular cells of JGA secrete renin in response to
    • Sympathetic nervous system stimulation
    • ↓ Filtrate osmolality
    • ↓ Stretch (due to ↓ blood pressure)
Regulation of Sodium Balance: Aldosterone

- Renin catalyzes the production of angiotensin II, which prompts aldosterone release from the adrenal cortex.
- Aldosterone release is also triggered by elevated K⁺ levels in the ECF.
- Aldosterone brings about its effects slowly (hours to days).
Figure 26.8

**K^+** (or **Na^+**) concentration in blood plasma

- **Stimulates**
  - **Adrenal cortex**
    - **Releases**
      - **Aldosterone**
        - **Targets**
          - **Kidney tubules**
            - **Effects**
              - **\( \downarrow \text{Na^+ reabsorption} \)**
              - **\( \uparrow \text{K^+ secretion} \)**
            - **Restores**
              - **Homeostatic plasma levels of Na^+ and K^+**

- **Negative feedback inhibits**

**Renin-angiotensin mechanism**
Regulation of Sodium Balance: ANP

• Released by atrial cells in response to stretch (↑ blood pressure)
• Effects
• Decreases blood pressure and blood volume:
  – ↓ ADH, renin and aldosterone production
  – ↑ Excretion of Na⁺ and water
  – Promotes vasodilation directly and also by decreasing production of angiotensin II
Figure 26.9

Stretch of atria of heart due to BP

Atrial natriuretic peptide (ANP)

Targets

Hypothalamus and posterior pituitary

Adrenal cortex

Collecting ducts of kidneys

JG apparatus of the kidney

Negative feedback

Effects

Renin release*

Angiotensin II

Vasodilation

Inhibits

Collecting ducts of kidneys

Na⁺ and H₂O reabsorption

Results in

Blood volume

Blood pressure

Signals

Blood pressure

Releases

ADH release

Aldosterone release

Inhibits

Effects

Results in
Influence of Other Hormones

• Estrogens: \( \uparrow \) NaCl reabsorption (like aldosterone)
  \( \rightarrow \) H\(_2\)O retention during menstrual cycles and pregnancy
• Progesterone: \( \downarrow \) Na\(^+\) reabsorption (blocks aldosterone)
  \( \rightarrow \) Promotes Na\(^+\) and H\(_2\)O loss
• Glucocorticoids: \( \uparrow \) Na\(^+\) reabsorption and promote edema
Cardiovascular System

Baroreceptors

- Baroreceptors alert the brain of increases in blood volume and pressure
  - Sympathetic nervous system impulses to the kidneys decline
  - Afferent arterioles dilate
  - GFR increases
  - $\text{Na}^+$ and water output increase
Figure 26.10

- **Stretch in afferent arterioles**
  - (+)
  - Inhibits baroreceptors in blood vessels

- **Angiotensinogen** (from liver)
  - (+)
  - Released by granular cells of kidneys
  - Catalyzes conversion to Angiotensin I

- **Angiotensin I**
  - (+)
  - Converted to Angiotensin II in systemic arterioles

- **Angiotensin II**
  - (+)
  - Causes vasoconstriction
  - Inhibits baroreceptors in blood vessels
  - Released by posterior pituitary

- **Systemic blood pressure/volume**
  - (+)

- **ADH (antidiuretic hormone)**
  - (+)
  - Reabsorption of water (H₂O) by collecting ducts of kidneys

- **Blood volume**
  - (+)

- **Blood pressure**
  - (+)

Legend:
- Green: Renin-angiotensin system
- Yellow: Neural regulation (sympathetic nervous system effects)
- Purple: ADH release and effects

(+): stimulates
Regulation of Potassium Balance

• Importance of potassium:
  – Affects resting membrane potential in neurons and muscle cells (especially cardiac muscle)
  • \( \uparrow \text{ECF } [K^+] \rightarrow \downarrow \text{RMP} \rightarrow \text{depolarization} \& \text{inactivation of Na}^+ \text{ channels.} \)
  • \( \downarrow \text{ECF } [K^+] \rightarrow \text{hyperpolarization and nonresponsiveness} \)
Regulation of Potassium Balance

• H⁺ shift in and out of cells
  – Leads to corresponding shifts in K⁺ in the opposite direction to maintain cation balance
  – Interferes with activity of excitable cells
Regulation of Potassium Balance

- $\text{K}^+$ balance is controlled in the cortical collecting ducts by changing the amount of potassium secreted into filtrate
- High $\text{K}^+$ content of ECF favors principal cell secretion of $\text{K}^+$
- When $\text{K}^+$ levels are low, duct cells reabsorb some $\text{K}^+$ left in the filtrate
Regulation of Potassium Balance

• Influence of aldosterone
  – Stimulates $K^+$ secretion (and $Na^+$ reabsorption) by duct cells

• Increased $K^+$ in the adrenal cortex causes
  • Release of aldosterone
  • Potassium secretion
Regulation of Calcium

• Ca\(^{2+}\) in ECF is important for
  – Neuromuscular excitability
  – Blood clotting
  – Cell membrane permeability
  – Secretory activities (release of secretory granules)
Regulation of Calcium

• Hypocalcemia $\rightarrow$ $\uparrow$ excitability and muscle tetany
• Hypercalcemia $\rightarrow$ Inhibits neurons and muscle cells, may cause heart arrhythmias
• Calcium balance is controlled by parathyroid hormone (PTH) and calcitonin
Influence of PTH

• Bones are the largest reservoir for Ca$^{2+}$ and phosphates
• PTH promotes increase in plasma calcium levels by targeting bones, kidneys, and small intestine (indirectly through vitamin D)
• Calcium reabsorption and phosphate excretion go hand in hand
Hypocalcemia (low blood Ca\(^{2+}\)) stimulates parathyroid glands to release PTH.

Rising Ca\(^{2+}\) in blood inhibits PTH release.

1. PTH activates osteoclasts: Ca\(^{2+}\) and PO\(_{4}^{3-}\) released into blood.
2. PTH increases Ca\(^{2+}\) reabsorption in kidney tubules.
3. PTH promotes kidney’s activation of vitamin D, which increases Ca\(^{2+}\) absorption from food.
Influence of PTH

• Normally 75% of filtered phosphates are actively reabsorbed in the PCT
• PTH inhibits this reabsorption.
• Prevents crystals of calcium phosphate from forming in the kidneys and blood.
Acid-Base Balance

• pH affects all functional proteins and biochemical reactions
• Normal pH of body fluids
  – Arterial blood: pH 7.4
  – Venous blood and IF fluid: pH 7.35
  – ICF: pH 7.0
• Alkalosis or alkalemia: arterial blood pH > 7.45
• Acidosis or acidemia: arterial pH < 7.35
Acid-Base Balance

• Most $H^+$ is produced by metabolism
  – Phosphoric acid from breakdown of phosphorus-containing proteins in ECF
  – Lactic acid from anaerobic respiration of glucose
  – Fatty acids and ketone bodies from fat metabolism
  – $H^+$ liberated when $CO_2$ is converted to $HCO_3^-$ in blood
Acid-Base Balance

• Concentration of hydrogen ions is regulated sequentially by
  – Chemical buffer systems: rapid; first line of defense
  – Brain stem respiratory centers: act within 1–3 min
  – Renal mechanisms: most potent, but require hours to days to effect pH changes
Acid-Base Balance

• Strong acids dissociate completely in water; can dramatically affect pH
• Weak acids dissociate partially in water; are efficient at preventing pH changes
• Strong bases dissociate easily in water; quickly tie up H⁺
• Weak bases accept H⁺ more slowly
(a) A strong acid such as HCl dissociates completely into its ions.

(b) A weak acid such as H$_2$CO$_3$ does not dissociate completely.
Chemical Buffer Systems

• Chemical buffer: system of one or more compounds that act to resist pH changes when strong acid or base is added
  1. Bicarbonate buffer system
  2. Phosphate buffer system
  3. Protein buffer system
Bicarbonate Buffer System

• Mixture of $\text{H}_2\text{CO}_3$ (weak acid) and salts of $\text{HCO}_3^-$ (e.g., NaHCO$_3$, a weak base)
• Buffers ICF and ECF
• The only important ECF buffer
Bicarbonate Buffer System

• If strong acid is added:
  – HCO$_3^-$ ties up H$^+$ and forms H$_2$CO$_3$
    • HCl + NaHCO$_3$ → H$_2$CO$_3$ + NaCl
  – pH decreases only slightly, unless all available HCO$_3^-$ (alkaline reserve) is used up
  – HCO$_3^-$ concentration is closely regulated by the kidneys
Bicarbonate Buffer System

• If strong base is added
  – It causes H₂CO₃ to dissociate and donate H⁺
  – H⁺ ties up the base (e.g. OH⁻)
    • NaOH + H₂CO₃ → NaHCO₃ + H₂O
  – pH rises only slightly
  – H₂CO₃ supply is almost limitless (from CO₂ released by respiration) and is subject to respiratory controls
Phosphate Buffer System

• Action is nearly identical to the bicarbonate buffer
• Components are sodium salts of:
  – Dihydrogen phosphate (H$_2$PO$_4^-$), a weak acid
  – Monohydrogen phosphate (HPO$_4^{2-}$), a weak base
• Effective buffer in urine and ICF, where phosphate concentrations are high
Protein Buffer System

• Intracellular proteins are the most plentiful and powerful buffers; plasma proteins are also important
• Protein molecules are amphoteric (can function as both a weak acid and a weak base)
  – When pH rises, organic acid or carboxyl (COOH) groups release $H^+$
  – When pH falls, $NH_2$ groups bind $H^+$
Physiological Buffer Systems

• Respiratory and renal systems
  – Act more slowly than chemical buffer systems
  – Have more capacity than chemical buffer systems
Respiratory Regulation of H⁺

• Respiratory system eliminates CO₂
• A reversible equilibrium exists in the blood:
  \[ \text{CO}_2 + \text{H}_2\text{O} \leftrightarrow \text{H}_2\text{CO}_3 \leftrightarrow \text{H}^+ + \text{HCO}_3^- \]
• During CO₂ unloading the reaction shifts to the left (and H⁺ is incorporated into H₂O)
• During CO₂ loading the reaction shifts to the right (and H⁺ is buffered by proteins)
Respiratory Regulation of $H^+$

- Hypercapnia (elevated $CO_2$) activates medullary chemoreceptors
- Rising plasma $H^+$ activates peripheral chemoreceptors
  - Increases ventilation rate
  - More $CO_2$ is removed from the blood
  - $H^+$ concentration is reduced
Respiratory Regulation of H⁺

• Alkalosis depresses the respiratory center
  – Respiratory rate and depth decrease
  – H⁺ concentration increases

• Respiratory system impairment causes acid-base imbalances
  – Hypoventilation → respiratory acidosis
  – Hyperventilation → respiratory alkalosis
Acid-Base Balance

• Chemical buffers cannot eliminate excess acids or bases from the body
  – Lungs eliminate volatile carbonic acid by eliminating CO$_2$
  – Kidneys eliminate other fixed metabolic acids (phosphoric, uric, and lactic acids and ketones) and prevent metabolic acidosis
Renal Mechanisms of Acid-Base Balance

• Most important renal mechanisms
  – Conserving (reabsorbing) or generating new HCO$_3^-$
  – Excreting HCO$_3^-$

• Generating or reabsorbing one HCO$_3^-$ is the same as losing one H$^+$

• Excreting one HCO$_3^-$ is the same as gaining one H$^+$
Renal Mechanisms of Acid-Base Balance

• Renal regulation of acid-base balance depends on secretion of H⁺
• H⁺ secretion occurs in the PCT and in collecting duct:
  – The H⁺ comes from H₂CO₃ produced in reactions catalyzed by carbonic anhydrase inside the cells
  – See Steps 1 and 2 of the following figure
**1** CO₂ combines with water within the tubule cell, forming H₂CO₃.

**2** H₂CO₃ is quickly split, forming H⁺ and bicarbonate ion (HCO₃⁻).

**3a** H⁺ is secreted into the filtrate.

**3b** For each H⁺ secreted, a HCO₃⁻ enters the peritubular capillary blood either via symport with Na⁺ or via antiport with Cl⁻.

**4** Secreted H⁺ combines with HCO₃⁻ in the filtrate, forming carbonic acid (H₂CO₃). HCO₃⁻ disappears from the filtrate at the same rate that HCO₃⁻ (formed within the tubule cell) enters the peritubular capillary blood.

**5** The H₂CO₃ formed in the filtrate dissociates to release CO₂ and H₂O.

**6** CO₂ diffuses into the tubule cell, where it triggers further H⁺ secretion.
Excretion of Buffered H⁺

• Dietary H⁺ must be balanced by generating new HCO₃⁻
• Most filtered HCO₃⁻ is used up before filtrate reaches the collecting duct
Excretion of Buffered H⁺

• Collecting duct cells actively secrete H⁺ into urine, which is buffered by phosphates and excreted
Ammonium Ion Excretion

• Ammonium is an acid.
• Involves metabolism of glutamine in PCT cells
• Each glutamine produces 2 $\text{NH}_4^+$ and 2 “new” $\text{HCO}_3^-$
• $\text{HCO}_3^-$ moves to the blood and $\text{NH}_4^+$ is excreted in urine
**1** PCT cells metabolize glutamine to \( \text{NH}_4^+ \) and \( \text{HCO}_3^- \).

**2a** This weak acid \( \text{NH}_4^+ \) (ammonium) is secreted into the filtrate, taking the place of \( \text{H}^+ \) on a \( \text{Na}^+ - \text{H}^+ \) antiport carrier.

**2b** For each \( \text{NH}_4^+ \) secreted, a bicarbonate ion (\( \text{HCO}_3^- \)) enters the peritubular capillary blood via a symport carrier.

**3** The \( \text{NH}_4^+ \) is excreted in the urine.
Bicarbonate Ion Secretion

• When the body is in alkalosis, collecting duct cells
  – Secrete HCO$_3^-$
  – Reclaim H$^+$ and acidify the blood
Bicarbonate Ion Secretion

• Mechanism is the opposite of the bicarbonate ion reabsorption process by type A intercalated cells
• Even during alkalosis, the nephrons and collecting ducts excrete fewer $\text{HCO}_3^-$ than they conserve
Abnormalities of Acid-Base Balance

• Respiratory acidosis and alkalosis
• Metabolic acidosis and alkalosis
Respiratory Acidosis and Alkalosis

- The most important indicator of adequacy of respiratory function is $P_{CO_2}$ level (normally 35–45 mm Hg)
  - $P_{CO_2}$ above 45 mm Hg $\rightarrow$ respiratory acidosis
  - Most common cause of acid-base imbalances
  - Due to decrease in ventilation or gas exchange
  - Characterized by falling blood pH and rising $P_{CO_2}$
Respiratory Acidosis and Alkalosis

- $P_{CO_2}$ below 35 mm Hg → respiratory alkalosis
  - A common result of hyperventilation due to stress or pain
Metabolic Acidosis and Alkalosis

- Any pH imbalance not caused by abnormal blood CO$_2$ levels
- Indicated by abnormal HCO$_3^-$ levels despite normal ventilation and gas exchange.
Metabolic Acidosis and Alkalosis

• Causes of metabolic acidosis
  – Ingestion of too much alcohol (→ acetic acid)
  – Excessive loss of $\text{HCO}_3^-$ (e.g., persistent diarrhea)
  – Accumulation of lactic acid, shock, ketosis in diabetic crisis, starvation, and kidney failure
Metabolic Acidosis and Alkalosis

• Metabolic alkalosis is much less common than metabolic acidosis
  – Indicated by rising blood pH and $\text{HCO}_3^-$
  – Caused by vomiting of the acid contents of the stomach or by intake of excess base (e.g., antacids)
Effects of Acidosis and Alkalosis

- Blood pH below 7 $\rightarrow$ depression of CNS $\rightarrow$ coma $\rightarrow$ death
- Blood pH above 7.8 $\rightarrow$ excitation of nervous system $\rightarrow$ muscle tetany, extreme nervousness, convulsions, respiratory arrest
Respiratory and Renal Compensations

• If acid-base imbalance is due to malfunction of a physiological buffer system, the other one compensates
  – Respiratory system attempts to correct metabolic acid-base imbalances
  – Kidneys attempt to correct respiratory acid-base imbalances
Respiratory Compensation

• In metabolic acidosis
  – High H\(^+\) levels stimulate the respiratory centers
  – Rate and depth of breathing are elevated
  – Blood pH is below 7.35 and HCO\(_3^-\) level is low
  – As CO\(_2\) is eliminated by the respiratory system, P\(_{CO_2}\) falls below normal
Respiratory Compensation

• Respiratory compensation for metabolic alkalosis is revealed by:
  – Slow, shallow breathing, allowing $\text{CO}_2$ accumulation in the blood
  – High pH (over 7.45) and elevated $\text{HCO}_3^-$ levels
Renal Compensation

- Hypoventilation causes elevated $P_{CO_2}$
- (respiratory acidosis)
  - Renal compensation is indicated by high $HCO_3^-$ levels
- Respiratory alkalosis exhibits low $P_{CO_2}$ and high pH
  - Renal compensation is indicated by decreasing $HCO_3^-$ levels