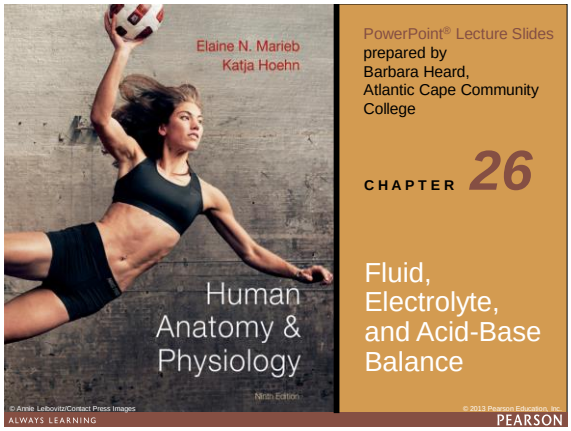


PowerPoint® Lecture Slides
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CHAPTER 26

# Fluid, Electrolyte, and 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)
  - Adipose tissue least hydrated of all
- Water content declines to ~45% in old age

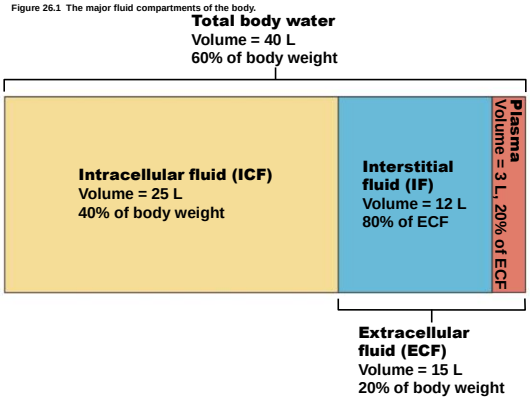


## Fluid Compartments

- Total body water = 40 L
- Two main fluid compartments
  - **Intracellular fluid (ICF) compartment:** 2/3 in cells
  - **Extracellular fluid (ECF) compartment:** 1/3 outside cells
    - **Plasma**: 3 L
    - **Interstitial fluid** (IF): 12 L in spaces between cells
      - Usually considered part of IF: lymph, CSF, humors of the eye, synovial fluid, serous fluid, and gastrointestinal secretions



Figure 26.1 The major fluid compartments of the body.






## Electrolyte Concentration

- Expressed in milliequivalents per liter (mEq/L), measure of number of electrical charges per liter of solution

$$\text{mEq/L} = \frac{\text{ion concentration (mg/L)}}{\text{atomic weight of ion (mg/mmol)}} \times \text{no. of electrical charges on one ion}$$



## Electrolyte Concentration

- For single charged ions (e.g. $Na^+$), 1 mEq = 1 mOsm
- For bivalent ions (e.g. $Ca^{2+}$), 1 mEq = 1/2 mOsm
- 1 mEq of either provides same amount of charge

## Extracellular and Intracellular Fluids

- Each fluid compartment has distinctive pattern of electrolytes
- ECF
  - – All similar
    - Major cation: $Na^+$
    - Major anion: $Cl^-$
  - – Except: higher protein, lower $Cl^-$ content of plasma



## Extracellular and Intracellular Fluids

- ICF:
  - – Low $Na^+$ and $Cl^-$
  - – Major cation: $K^+$
  - – Major anion $HPO_4^{2-}$
  - – More soluble proteins than in plasma



Figure 26.2 Electrolyte composition of blood plasma, interstitial fluid, and intracellular fluid.

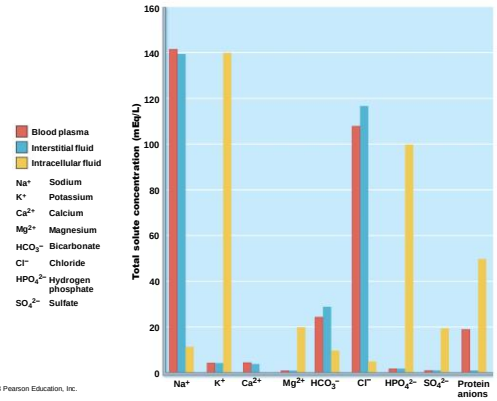
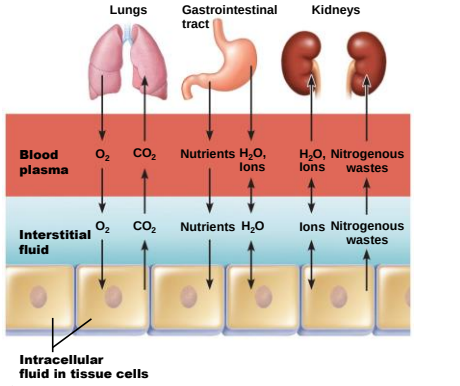





Figure 26.3 Exchange of gases, nutrients, water, and wastes between the three fluid compartments of the body.



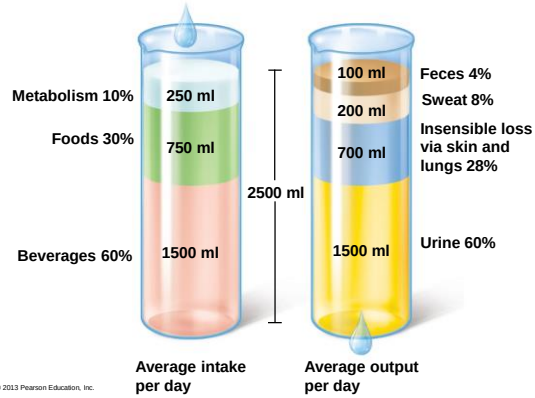



## Water Balance and ECF Osmolality

- Water intake must = water output = ~ 2500 ml/day
- Water intake: beverages, food, and metabolic water
- Water output: urine (60%), **insensible water loss** (lost through skin and lungs), perspiration, and feces



Figure 26.4 Major sources of water intake and output.

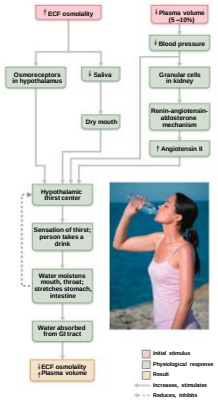


## Maintenance of Body fluid Osmolality

- Osmolality maintained at ~ 280 – 300 mOsm
- Rise in osmolality →
  - Stimulates thirst
  - ADH release
- Decrease in osmolality →
  - Thirst inhibition
  - ADH inhibition



Figure 26.5 The thirst mechanism for regulating water intake.



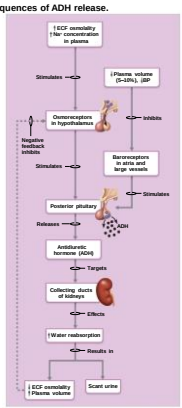



## Regulation of Water Output: Influence of ADH

- Other factors may trigger ADH release
  - Large changes in blood volume or pressure
    - E.g., ↓ BP → ↑ ADH release due to blood vessel baroreceptors and renin-angiotensin-aldosterone mechanism
    - Factors lowering blood volume: intense sweating, vomiting, or diarrhea; severe blood loss; traumatic burns; and prolonged fever



Figure 26.6 Mechanisms and consequences of ADH release.






## Disorders of Water Balance

- Principal abnormalities of water balance
  - Dehydration
  - Hypotonic hydration
  - Edema

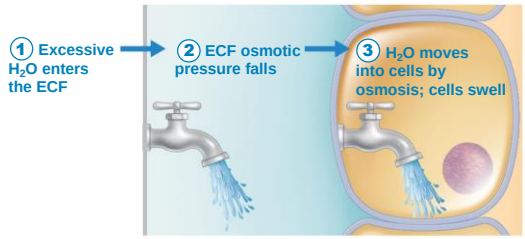


## Disorders of Water Balance: Hypotonic Hydration

- Cellular overhydration, or water intoxication
- Occurs with renal insufficiency or rapid excess water ingestion
- ECF osmolality ↓ → hyponatremia → net osmosis into tissue cells → swelling of cells → severe metabolic disturbances (nausea, vomiting, muscular cramping, cerebral edema) → possible death
- Treated with hypertonic saline

Figure 26.7b Disturbances in water balance.

Slide 1




**(b) Consequences of hypotonic hydration (water gain). If more water than solutes is gained, cells swell.**



## Disorders of Water Balance: Edema

- Atypical accumulation of IF → tissue swelling (not cell swelling)
- Result of ↑ fluid out of blood or ↓ fluid into blood
- ↑ fluid out of blood caused by
  - Increased capillary hydrostatic pressure or permeability
    - Capillary hydrostatic pressure increased by incompetent venous valves, localized blood vessel blockage, congestive heart failure, ↑ blood volume
    - Capillary permeability increased by ongoing inflammatory response



## Edema

- ↓ fluid returning to blood result of
  - Imbalance in colloid osmotic pressures, e.g., **hypoproteinemia** (↓ plasma protein levels → low colloid osmotic pressure)
    - Fluids fail to return at venous ends of capillary beds
    - Results from protein malnutrition, liver disease, or glomerulonephritis



## Electrolyte Balance

- Electrolytes are salts, acids, bases, some proteins
- **Electrolyte balance** usually refers only to salt balance
- Salts control fluid movements; provide minerals for excitability, secretory activity, membrane permeability
- Salts enter body by ingestion and metabolism; lost via perspiration, feces, urine, vomit



## Central Role of Sodium

- Most abundant cation in ECF
  - Sodium salts in ECF contribute 280 mOsm of total 300 mOsm ECF solute concentration
- Only cation exerting *significant* osmotic pressure
  - **Controls ECF volume and water distribution**
  - Changes in $Na^+$ levels affects plasma volume, blood pressure, and ECF and IF volumes



Table 26.2 Sodium Concentration and Sodium Content

**Table 26.2 Sodium Concentration and Sodium Content**

| | ECF $Na^+$ CONCENTRATION | BODY $Na^+$ CONTENT |
|---|---|---|
| **Homeostatic Importance** | ECF osmolality | Blood volume and blood pressure |
| **Sensors** | Osmoreceptors | Baroreceptors |
| **Regulation** | ADH and thirst mechanisms | Renin-angiotensin-aldosterone and ANP hormone mechanisms* |

*ADH and thirst are also required to maintain blood volume and for long-term control of blood pressure.

## Regulation of Sodium Balance: Aldosterone

- Regardless of aldosterone presence
  - 65% $Na^+$ reabsorbed in proximal tubules; 25% reclaimed in nephron loops
  - $Na^+$ *never* secreted into filtrate
- Water in filtrate follows $Na^+$ *if ADH is present*
  - ↑ $Na^+$ in urine → ↑ water loss



## Aldosterone

- **Aldosterone** → decreased urinary output; increased blood volume
  - By active reabsorption of remaining $Na^+$ in distal convoluted tubule and collecting duct
- Also causes increased $K^+$ secretion



## Regulation of Sodium Balance: Aldosterone

- Renin-angiotensin-aldosterone mechanism main trigger for aldosterone release
  - Granular cells of JGC secrete renin in response to
    - Sympathetic nervous system stimulation
    - ↓ filtrate NaCl concentration
    - ↓ stretch (due to ↓ blood pressure) of granular cells



## Regulation of Sodium Balance: Aldosterone

- Renin catalyzes production of **angiotensin II**
  - Prompts aldosterone release from adrenal cortex
  - ↑ $Na^+$ reabsorption by kidney tubules
- Aldosterone release also triggered by elevated $K^+$ levels in ECF
- Aldosterone brings about its effects slowly (hours to days)



**Figure 26.8 Mechanisms and consequences of aldosterone release.**

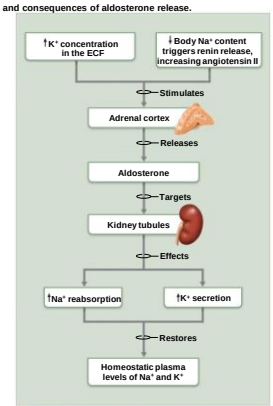
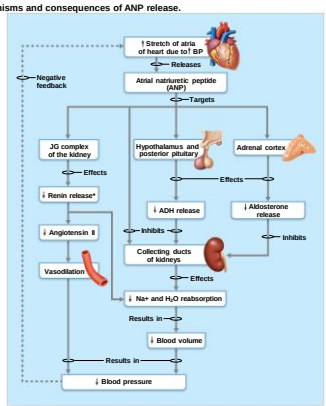





**Figure 26.9 Mechanisms and consequences of ANP release.**

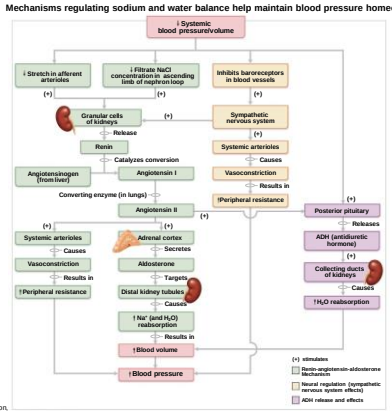


## Influence of other Hormones

- Female sex hormones
  - Estrogens: ↑ NaCl reabsorption (like aldosterone)
    - → $H_2O$ retention during menstrual cycles and pregnancy
  - Progesterone: ↓ $Na^+$ reabsorption (blocks aldosterone)
    - Promotes $Na^+$ and $H_2O$ loss
- Glucocorticoids: ↑ $Na^+$ reabsorption and promote edema



Figure 26.10 Mechanisms regulating sodium and water balance help maintain blood pressure homeostasis.






## Regulation of Potassium Balance

- Importance of potassium
  - Affects RMP in neurons and muscle cells (especially cardiac muscle)
    - ↑ ECF $[K^+]$ → ↓RMP → depolarization → reduced excitability
    - ↓ ECF $[K^+]$ → hyperpolarization and nonresponsiveness



## Regulation of Potassium Balance

- **Hyperkalemia** - too much $K^+$
- **Hypokalemia** - too little $K^+$
- Both disrupt electrical conduction in heart →
  - Sudden death



## Regulation of Potassium Balance

- $K^+$ part of body's buffer system
- $H^+$ shifts in and out of cells in opposite direction of $K^+$ to maintain cation balance, so
  - ECF $K^+$ levels rise with acidosis
  - ECF $K^+$ levels fall with alkalosis
    - Interferes with activity of excitable cells



## Influence of Plasma Potassium Concentration

- Most important factor affecting $K^+$ secretion is its concentration in ECF
- High $K^+$ diet → ↑ $K^+$ content of ECF → $K^+$ entry into principal cells → $K^+$ secretion
- Low $K^+$ diet or accelerated $K^+$ loss reduces its secretion

## Regulation of Potassium Balance

- Influence of aldosterone
  - Stimulates $K^+$ secretion (and $Na^+$ reabsorption) by principal cells
  - Adrenal cortical cells directly sensitive to $K^+$ content of ECF
    - Increased $K^+$ in adrenal cortex causes
      - Release of aldosterone $\rightarrow$ $K^+$ secretion
- Abnormal aldosterone levels severely influence $K^+$ levels



## Regulation of Calcium

- 99% of body's calcium in bones
  - Calcium phosphate salts
- $Ca^{2+}$ in ECF important for
  - Blood clotting
  - Cell membrane permeability
  - Secretory activities
  - Neuromuscular excitability - most important



## Regulation of Calcium

- **Hypocalcemia** $\rightarrow \uparrow$ excitability and muscle tetany
- **Hypercalcemia** $\rightarrow$ inhibits neurons and muscle cells, may cause heart arrhythmias
- Calcium balance controlled by parathyroid hormone (PTH) from parathyroid gland
  - Rarely deviates from normal limits



## Influence of PTH

- PTH promotes increase in calcium levels by targeting
  - Bones – osteoclasts break down matrix, releasing calcium and phosphate to blood
  - Kidneys – increases calcium reabsorption; decreases phosphate ion reabsorption
  - Small intestine – increases calcium absorption (indirectly through stimulation of kidney to activate vitamin D precursor)

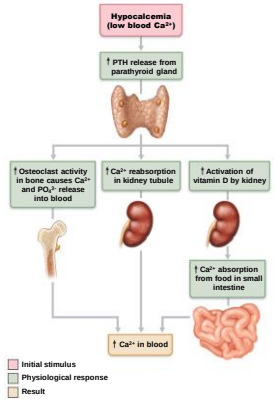


## Influence of PTH

- 98% filtered calcium reabsorbed due to PTH
- If ECF calcium levels normal PTH secretion inhibited
- 75% of filtered phosphates reabsorbed in PCT
  - PTH inhibits this by decreasing the $T_m$
- Phosphate reabsorption also affected by insulin (increases it) and glucagon (decreases it)



**Figure 16.13 Effects of parathyroid hormone on bone, the kidneys, and the intestine.**



## Regulation of Anions

- $Cl^-$ is major anion in ECF
  - Helps maintain osmotic pressure of blood
  - 99% of $Cl^-$ is reabsorbed under normal pH conditions
- When acidosis occurs, fewer chloride ions are reabsorbed
- Other anions have transport maximums and excesses are excreted in urine



## Acid-base Balance

- pH affects all functional proteins and biochemical reactions, so closely regulated
- 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 pH >7.45
- **Acidosis** or acidemia: arterial pH <7.35



## Acid-base Balance

- Most $H^+$ produced by metabolism
  - Phosphorus-containing protein breakdown releases *phosphoric acid* into ECF
  - *Lactic acid* from anaerobic respiration of glucose
  - *Fatty acids* and *ketone bodies* from fat metabolism
  - $H^+$ liberated when $CO_2$ converted to $HCO_3^-$ in blood



## Acid-base Balance

- Concentration of hydrogen ions 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

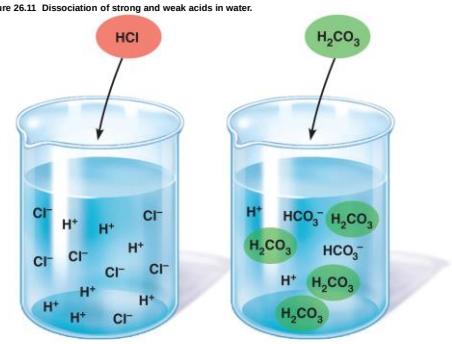


## Acid-base Balance: Chemical Buffer Systems

- 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



Figure 26.11 Dissociation of strong and weak acids in water.




(a) A strong acid such as HCl dissociates completely into its ions.

(b) A weak acid such as $H_2CO_3$ 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
  - Bind $H^+$ if pH drops; release $H^+$ if pH rises
  1. **Bicarbonate buffer system**
  2. **Phosphate buffer system**
  3. **Protein buffer system**



## Phosphate Buffer System

- Action nearly identical to bicarbonate buffer
- Components are sodium salts of:
  - Dihydrogen phosphate ($H_2PO_4^-$), a weak acid
  - Monohydrogen phosphate ($HPO_4^{2-}$), a weak base
- Unimportant in buffering plasma
- Effective buffer in urine and ICF, where phosphate concentrations are high



## Respiratory Regulation of $H^+$

- **Hypercapnia** activates medullary chemoreceptors
  - → Increased respiratory rate and depth
- Rising plasma $H^+$ activates peripheral chemoreceptors
  - → Increased respiratory rate and depth
  - More $CO_2$ is removed from the blood
  - $H^+$ concentration is reduced



## Respiratory Regulation of $H^+$

- Alkalosis depresses respiratory center
  - Respiratory rate and depth decrease
  - $H^+$ concentration increases
- Respiratory system impairment causes acid-base imbalances
  - **Hypoventilation → respiratory acidosis**
  - **Hyperventilation → respiratory alkalosis**



## Renal Mechanisms of Acid-Base Balance

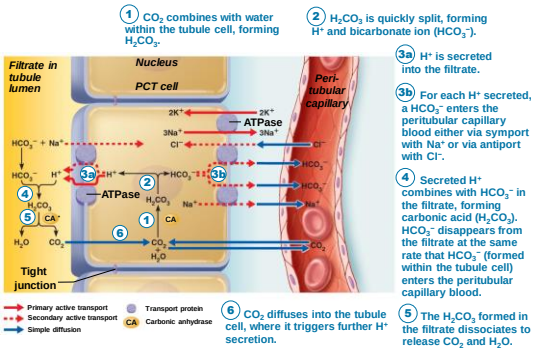
- Most important renal mechanisms
  - Conserving (reabsorbing) or generating new $HCO_3^-$
  - Excreting $HCO_3^-$
- Generating or reabsorbing one $HCO_3^-$ same as losing one $H^+$
- Excreting one $HCO_3^-$ same as gaining one $H^+$



## Renal Mechanisms of Acid-base Balance

- Renal regulation of acid-base balance depends on kidney's ability to secrete $H^+$
- $H^+$ secretion occurs in PCT and collecting duct type A intercalated cells:
  - The $H^+$ comes from $H_2CO_3$ produced in reactions catalyzed by carbonic anhydrase inside cells
  - As $H^+$ secreted, $Na^+$ reabsorbed
  - See Steps 1 and 2 of following figure

Figure 26.12 Reabsorption of filtered $HCO_3^-$ is coupled to $H^+$ secretion. Slide 1



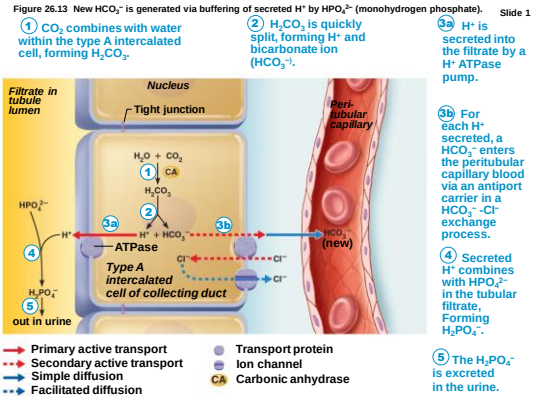



## Renal Mechanisms of Acid-base Balance

- Rate of $H^+$ secretion changes with ECF $CO_2$ levels
  - – ↑ $CO_2$ in peritubular capillary blood → ↑ rate of $H^+$ secretion
  - – System responds to both rising and falling $H^+$ concentrations



Figure 26.13 New $HCO_3^-$ is generated via buffering of secreted $H^+$ by $HPO_4^{2-}$ (monohydrogen phosphate). Slide 1



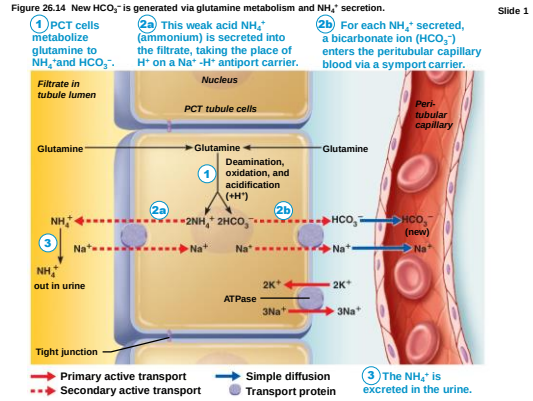



## Ammonium Ion Excretion

- More important mechanism for excreting acid
- Involves metabolism of glutamine in PCT cells
- Each glutamine produces 2 $NH_4^+$ and 2 "new" $HCO_3^-$
- $HCO_3^-$ moves to blood and $NH_4^+$ is excreted in urine
- Replenishes alkaline reserve of blood



Figure 26.14 New $HCO_3^-$ is generated via glutamine metabolism and $NH_4^+$ secretion. Slide 1






## Bicarbonate Ion Secretion

- When body in alkalosis, type B intercalated cells
  - – Secrete $HCO_3^-$
  - – Reclaim $H^+$ to acidify blood

## Bicarbonate Ion Secretion

- Mechanism is opposite of bicarbonate ion reabsorption process by type A intercalated cells
- Even during alkalosis, nephrons and collecting ducts conserve more $HCO_3^-$ than they excrete



## Respiratory Acidosis and Alkalosis

- 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 → respiratory acidosis
    - Common cause of acid-base imbalances
    - Due to decrease in ventilation or gas exchange
    - $CO_2$ accumulates in blood
    - Characterized by falling blood pH and rising $P_{CO_2}$



## Respiratory Acidosis and Alkalosis

- $P_{CO_2}$ below 35 mm Hg → respiratory alkalosis
  - Common result of **hyperventilation** often due to stress or pain
    - $CO_2$ eliminated faster than produced



## Metabolic Acidosis and Alkalosis

- Metabolic acidosis – low blood pH and $HCO_3^-$
  - Causes
    - Ingestion of too much alcohol (→ acetic acid)
    - Excessive loss of $HCO_3^-$ (e.g., persistent diarrhea)
    - Accumulation of lactic acid (exercise or shock), ketosis in diabetic crisis, starvation, and kidney failure



## Metabolic Acidosis and Alkalosis

- Metabolic alkalosis much less common than metabolic acidosis
  - Indicated by rising blood pH and $HCO_3^-$
  - Causes include vomiting of acid contents of stomach or by intake of excess base (e.g., antacids)



## Respiratory Compensation

- Changes in respiratory rate and depth
- In metabolic acidosis
  - High $H^+$ levels stimulate respiratory centers
  - Rate and depth of breathing elevated
  - Blood pH is below 7.35 and $HCO_3^-$ level is low
  - As $CO_2$ eliminated by respiratory system, $P_{CO_2}$ falls below normal