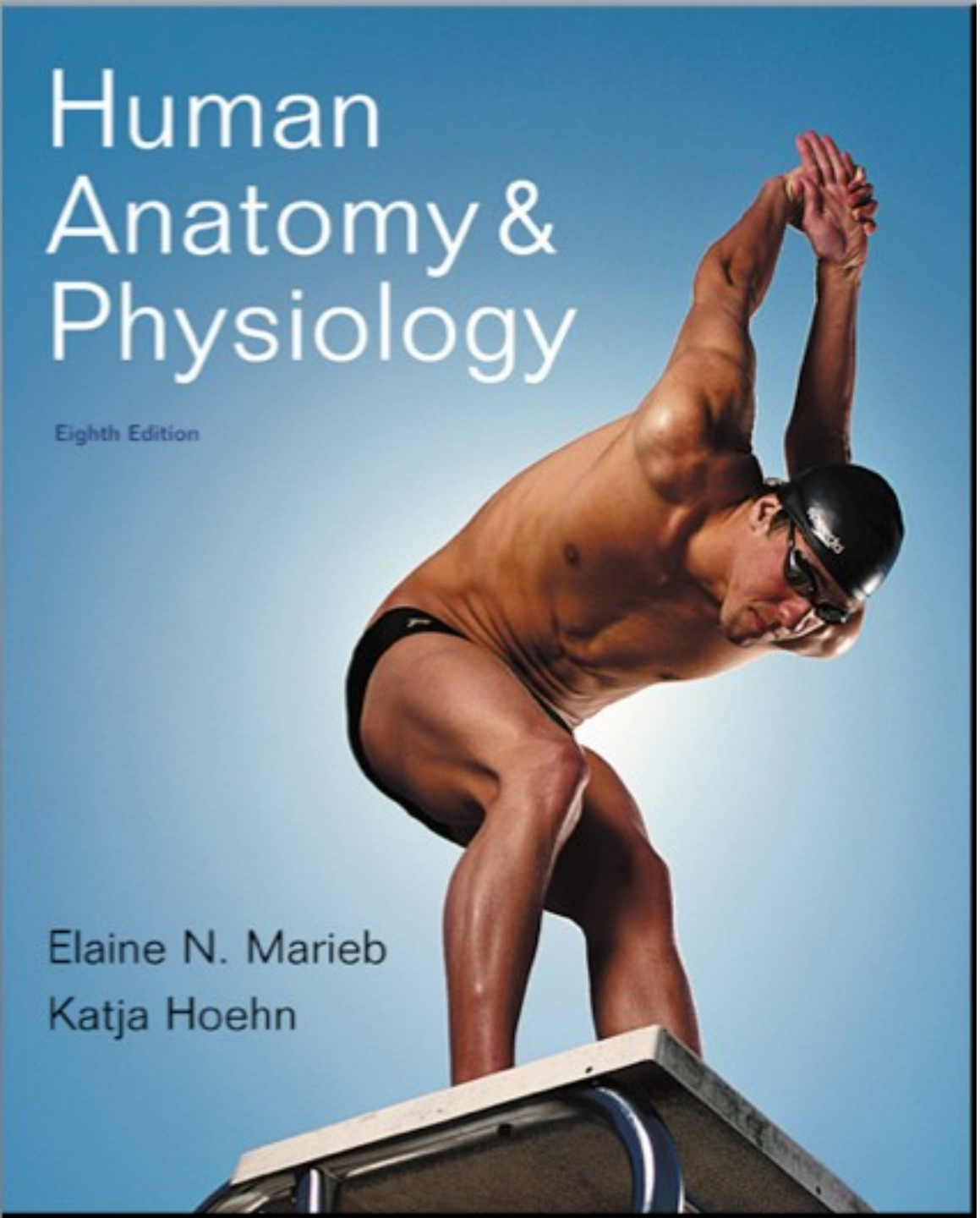


Chapter 26

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

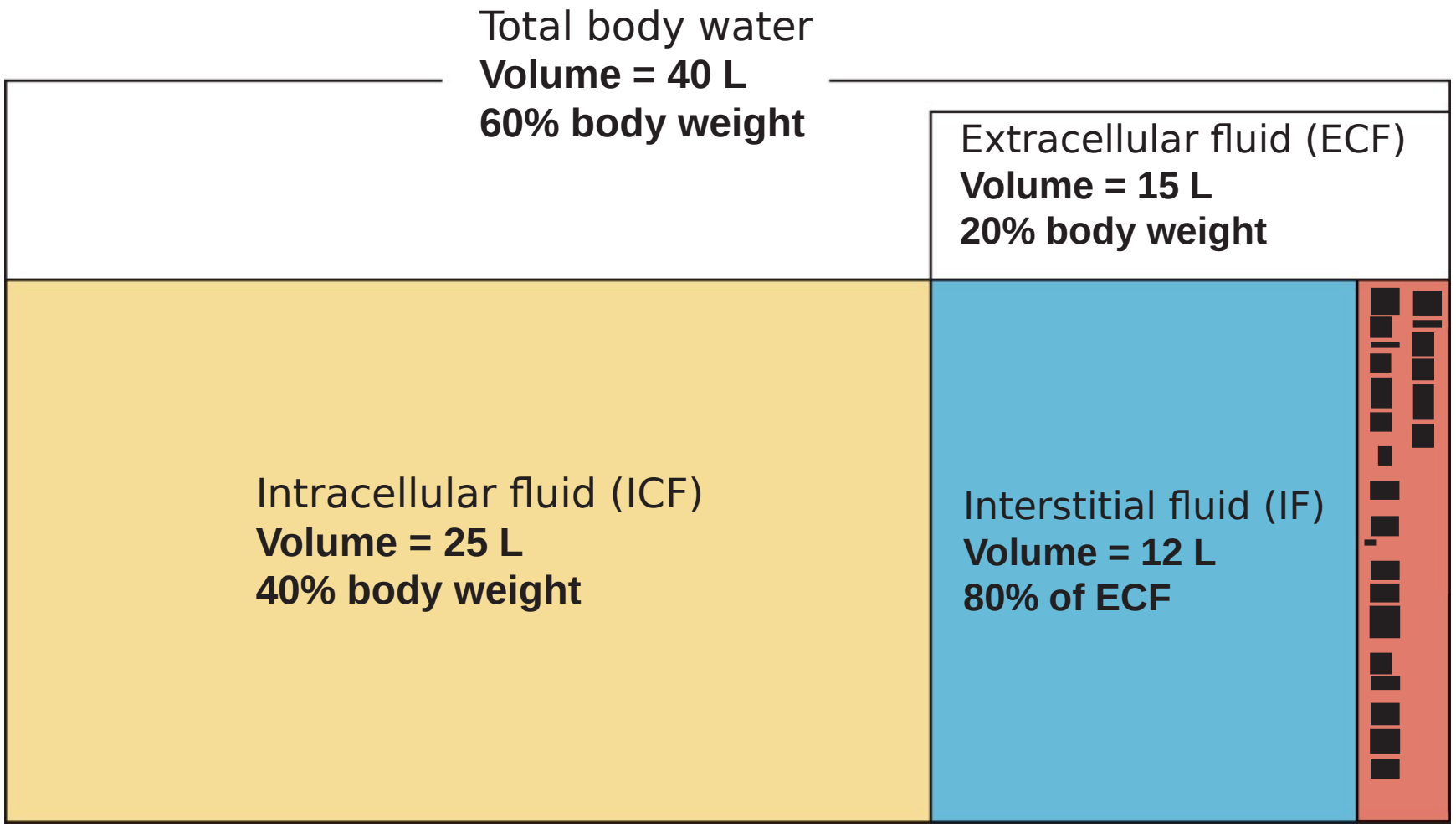

Total body water
Volume = 40 L
60% body weight
Extracellular fluid (ECF)
Volume = 15 L
20% body weight
Intracellular fluid (ICF)
Volume = 25 L
40% body weight
Interstitial fluid (IF)
Volume = 12 L
80% of ECF

Figure 26.1

Composition of Body Fluids

- Water: the universal solvent
- Solutes: 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 Na^+ and Cl^-
  - Major cation: K^+
  - Major anion 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

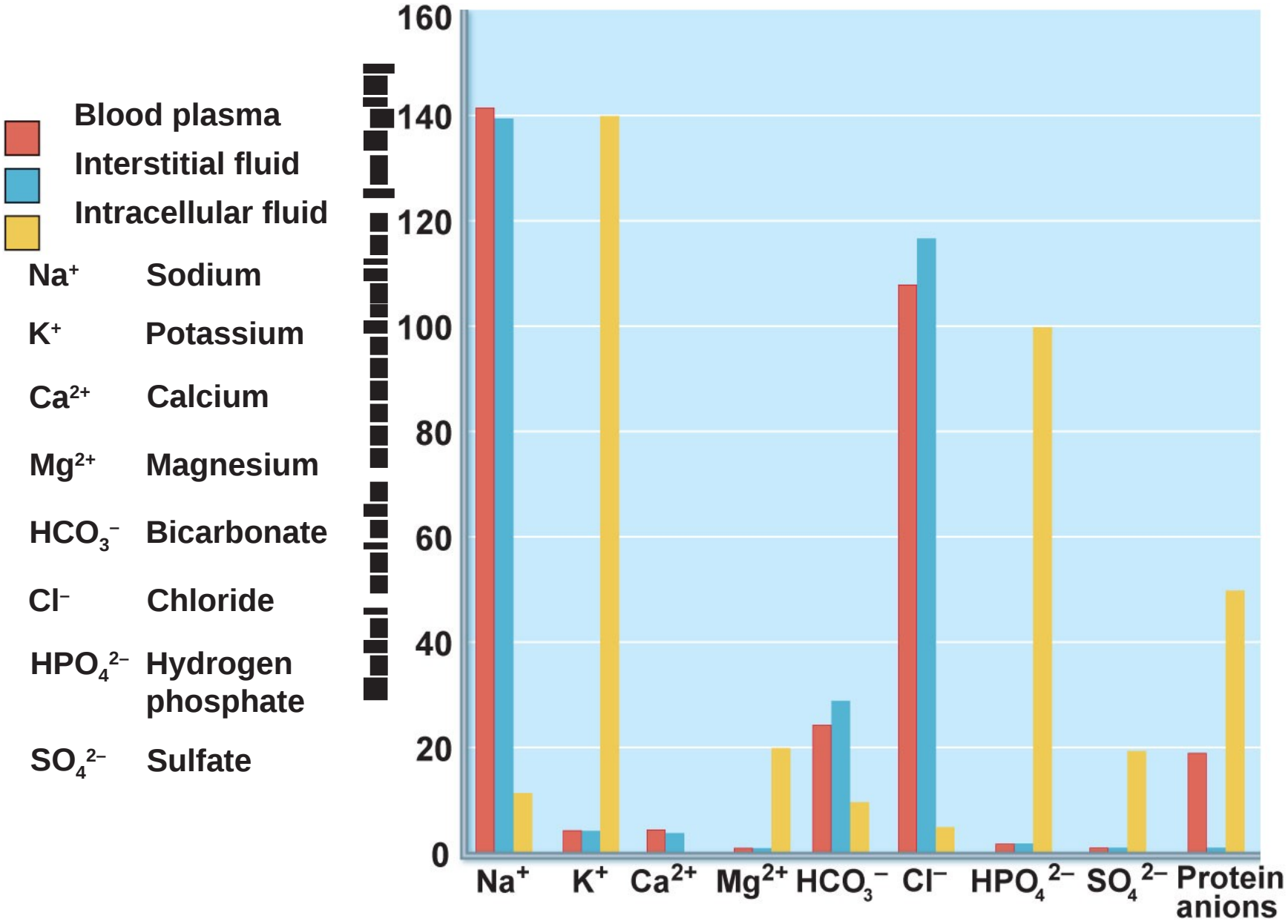

Blood plasma
Interstitial fluid
Intracellular fluid
Na+ Sodium
K+ Potassium
Ca2+ Calcium
Mg2+ Magnesium
HCO3− Bicarbonate
Cl− Chloride
HPO4 2− Hydrogen phosphate
SO4 2− Sulfate
160
140
120
100
80
60
40
20
0
Na+
K+
Ca2+
Mg2+
HCO3−
Cl−
HPO4 2−
SO4 2−
Protein anions

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

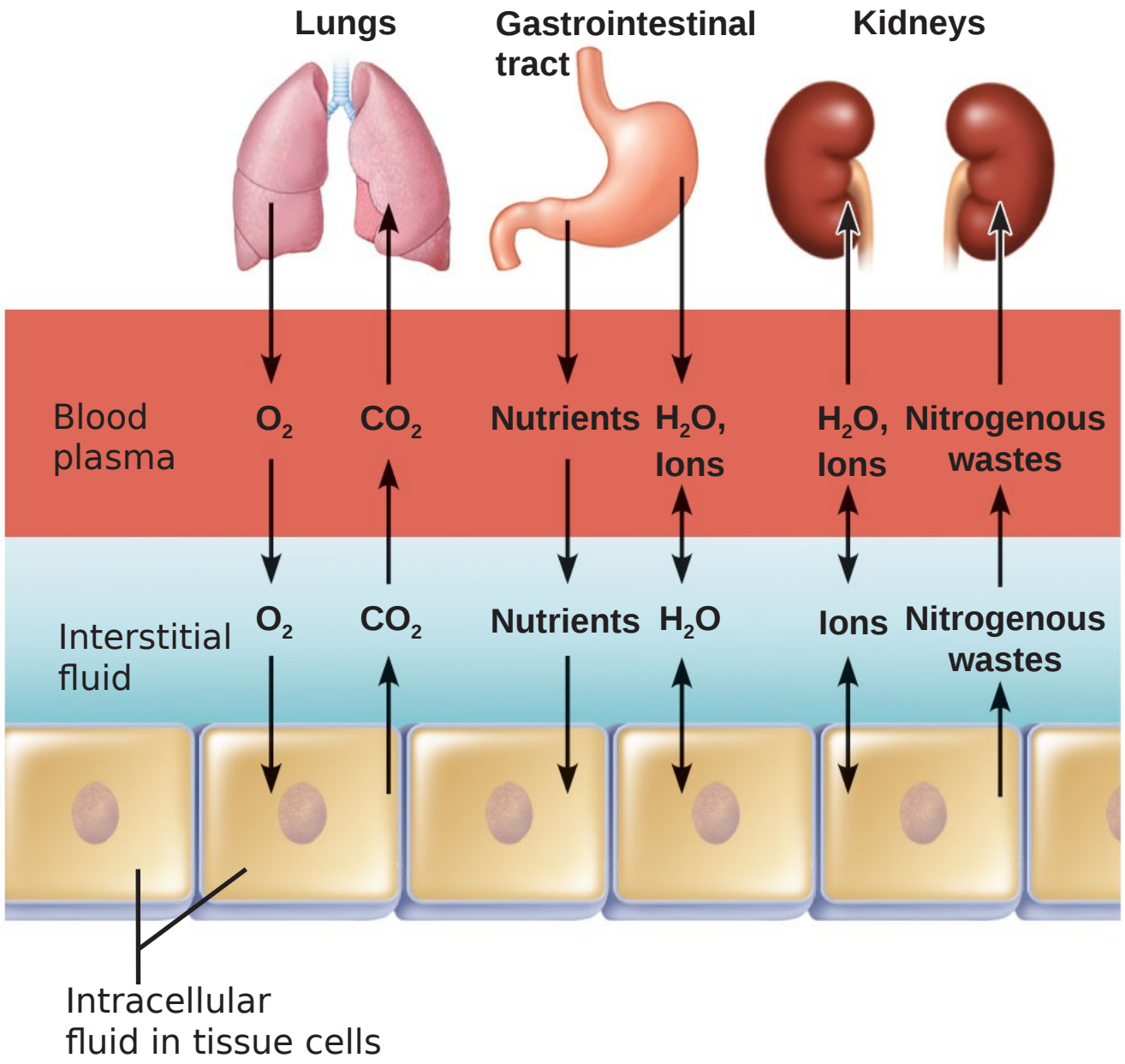

Lungs
Gastrointestinal tract
Kidneys
Blood plasma
O_2
CO_2
Nutrients
H_2O, Ions
H_2O, Ions
Nitrogenous wastes
Interstitial fluid
O_2
CO_2
Nutrients
H_2O
Ions
Nitrogenous wastes
Intracellular fluid in tissue cells

Figure 26.3

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

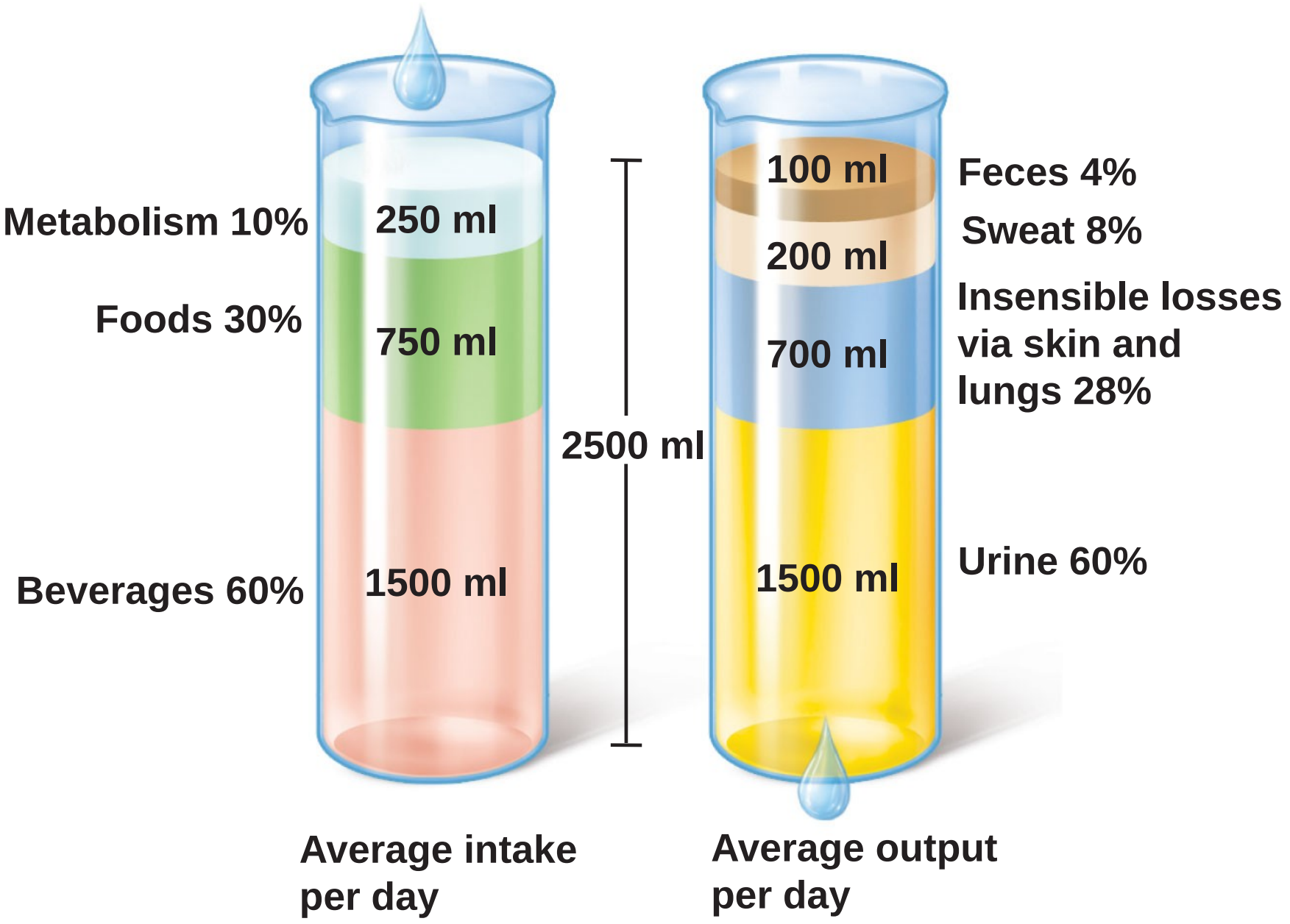

Metabolism 10%
250 ml
Foods 30%
750 ml
Beverages 60%
1500 ml
2500 ml
Average intake per day
100 ml
Feces 4%
Sweat 8%
200 ml
Insensible losses via skin and lungs 28%
700 ml
Urine 60%
1500 ml
Average output per day

Figure 26.4

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

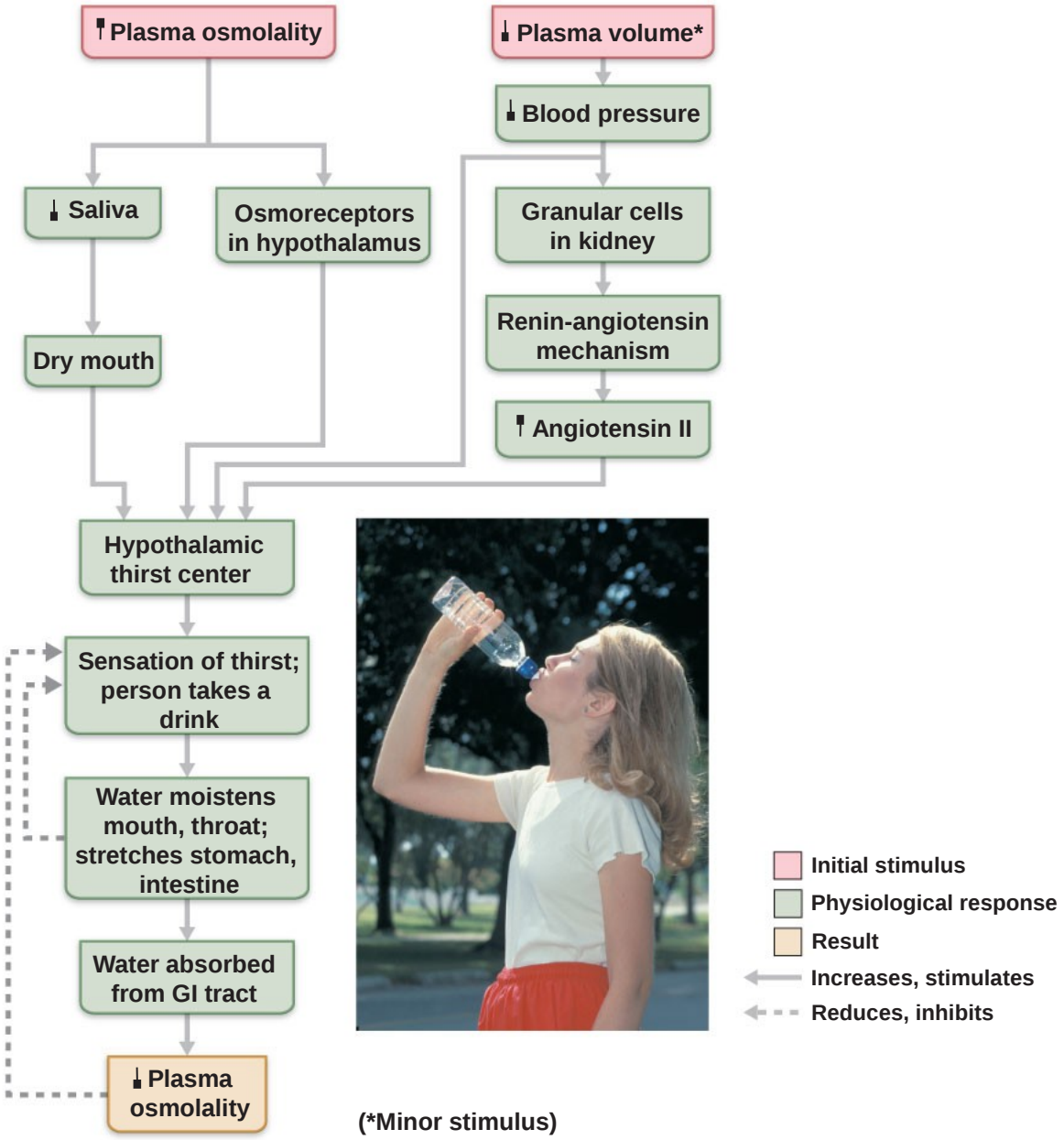

Plasma osmolality
Plasma volume*
Blood pressure
Saliva
Osmoreceptors in hypothalamus
Granular cells in kidney
Dry mouth
Renin-angiotensin mechanism
Angiotensin II
Hypothalamic thirst center
Sensation of thirst; person takes a drink
Water moistens mouth, throat; stretches stomach, intestine
Water absorbed from GI tract
Plasma osmolality
(*Minor stimulus)
Initial stimulus
Physiological response
Result
Increases, stimulates
Reduces, inhibits

Figure 26.5

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 → dilute urine and ↓ volume of body fluids
- ↑ ADH → 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

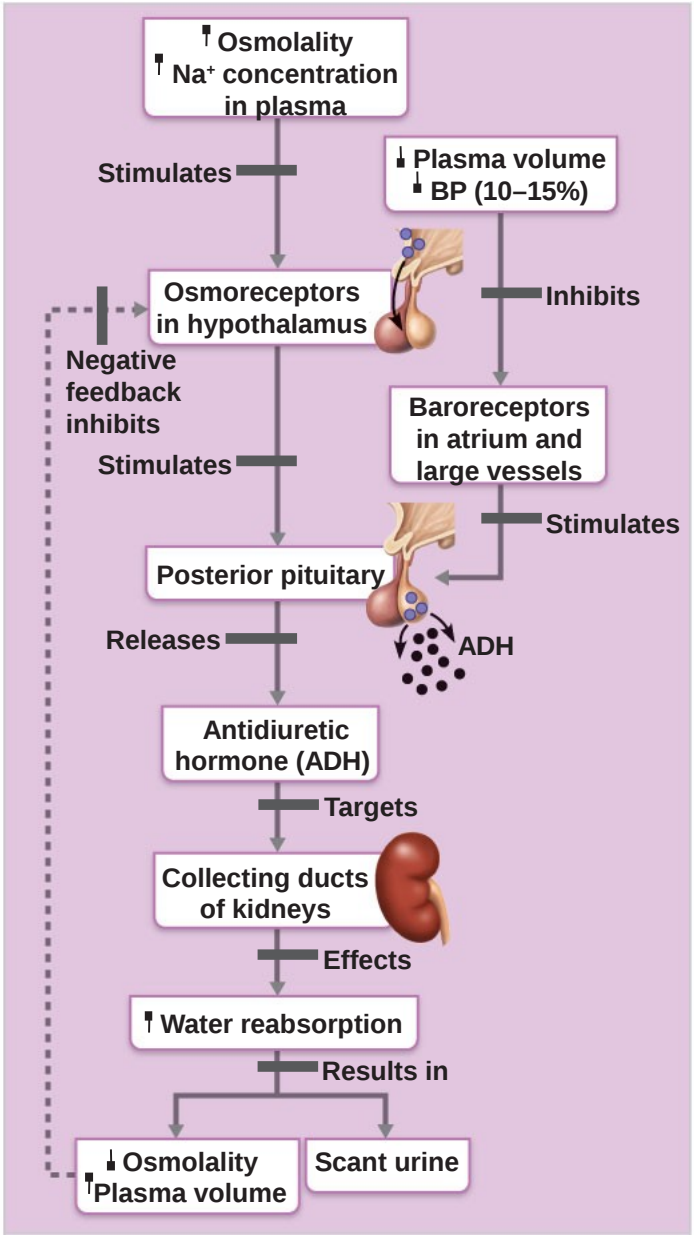
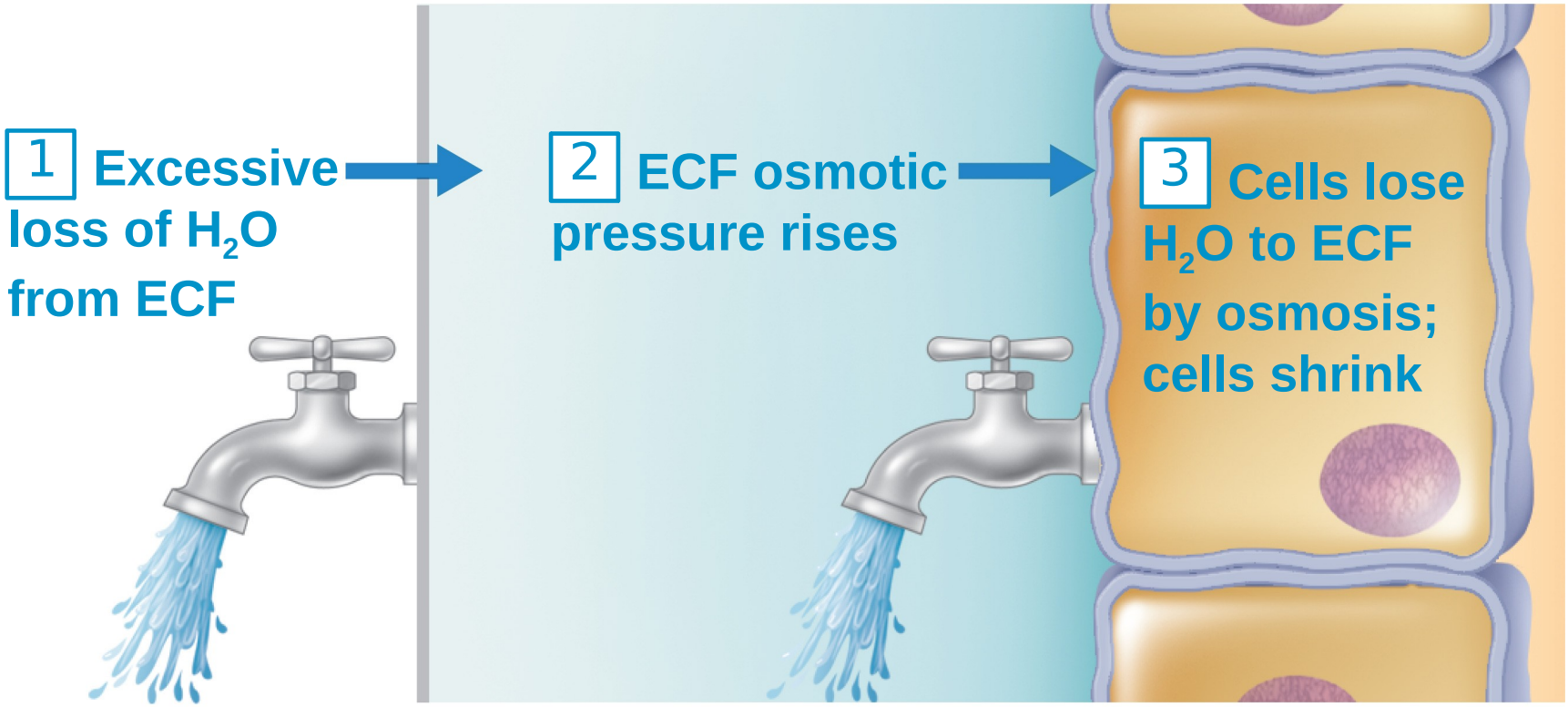

↑ Osmolality
↑ Na+ concentration in plasma
Stimulates
↓ Plasma volume
↓ BP (10–15%)
Osmoreceptors in hypothalamus
Inhibits
Negative feedback inhibits
Stimulates
Baroreceptors in atrium and large vessels
Stimulates
Posterior pituitary
Releases
ADH
Antidiuretic hormone (ADH)
Targets
Collecting ducts of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

Figure 26.6

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

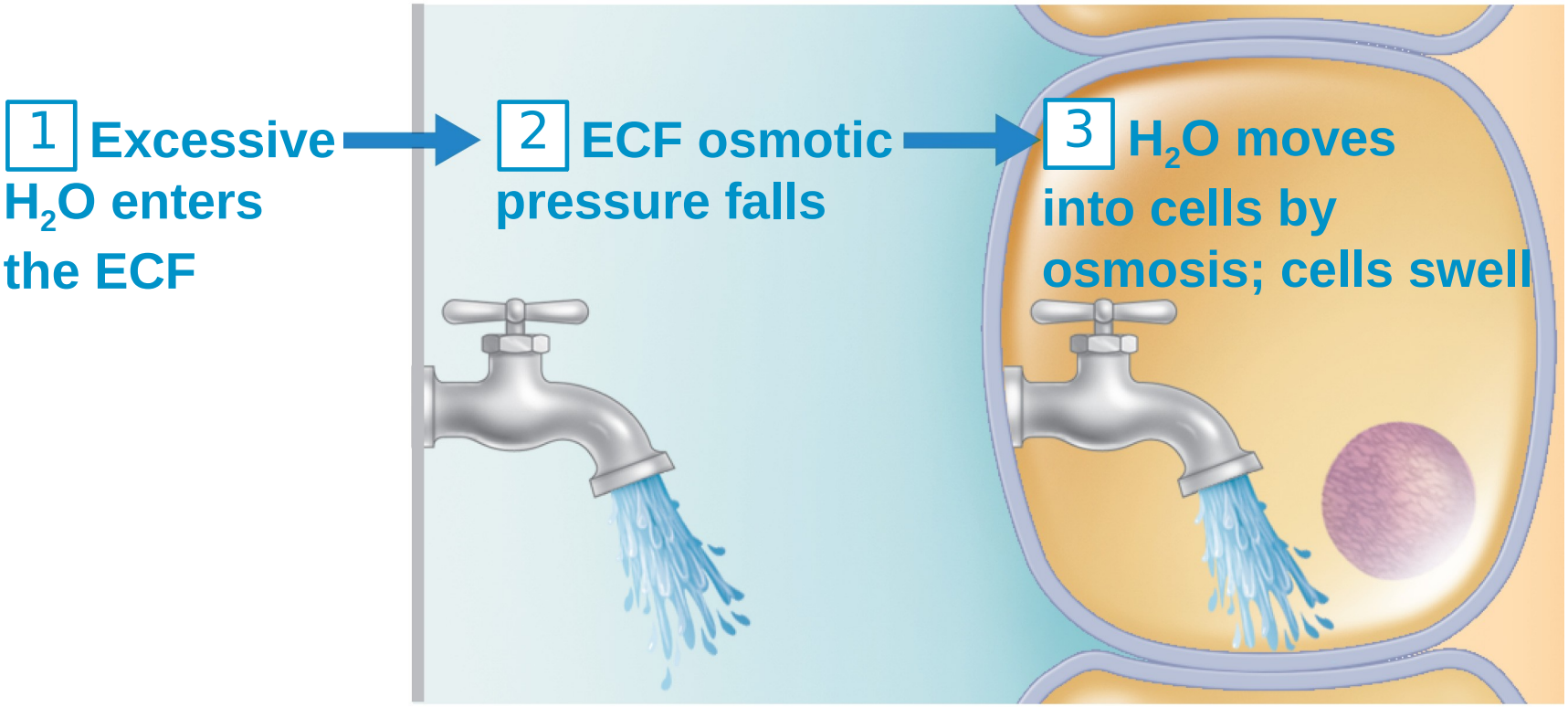


(a) Mechanism of dehydration

Figure 26.7a

Disorders of Water Balance: Hypotonic Hydration

- Cellular overhydration, or water intoxication
- Occurs with renal insufficiency or rapid excess water ingestion
- ECF is diluted $\rightarrow$ hyponatremia $\rightarrow$ net osmosis into tissue cells $\rightarrow$ swelling of cells $\rightarrow$ severe metabolic disturbances (nausea, vomiting, muscular cramping, cerebral edema) $\rightarrow$ possible death



(b) Mechanism of hypotonic hydration

Figure 26.7b

Disorders of Water Balance: Edema

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

- Na^+ reabsorption
  - 65% is reabsorbed in the proximal tubules
  - 25% is reclaimed in the loops of Henle
- Aldosterone $\rightarrow$ active reabsorption of remaining Na^+
- Water follows 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)

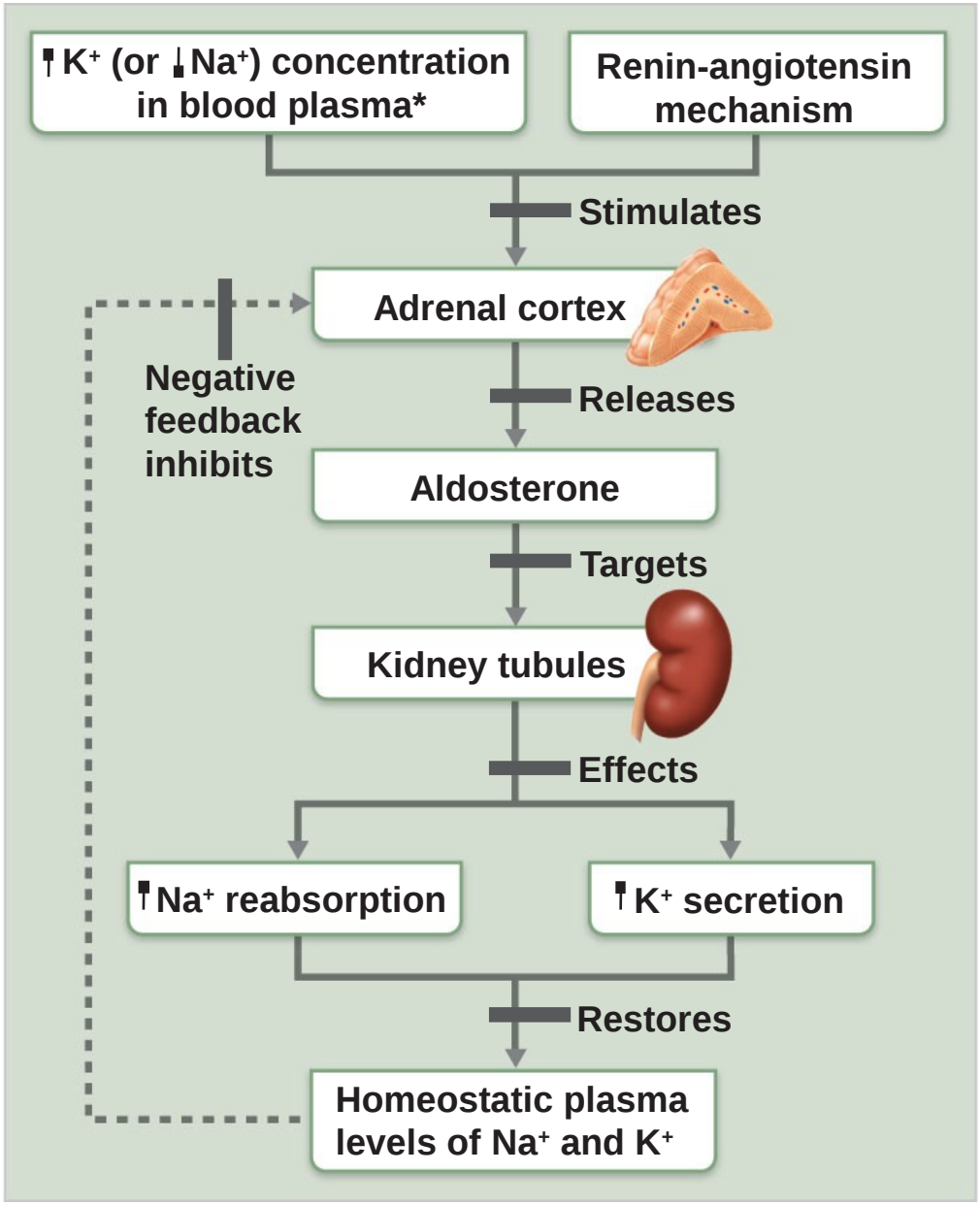

↑K+ (or ↓Na+) concentration in blood plasma*
Renin-angiotensin mechanism
Stimulates
Adrenal cortex
Negative feedback inhibits
Releases
Aldosterone
Targets
Kidney tubules
Effects
↑Na+ reabsorption
↑K+ secretion
Restores
Homeostatic plasma levels of Na+ and K+

Figure 26.8

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

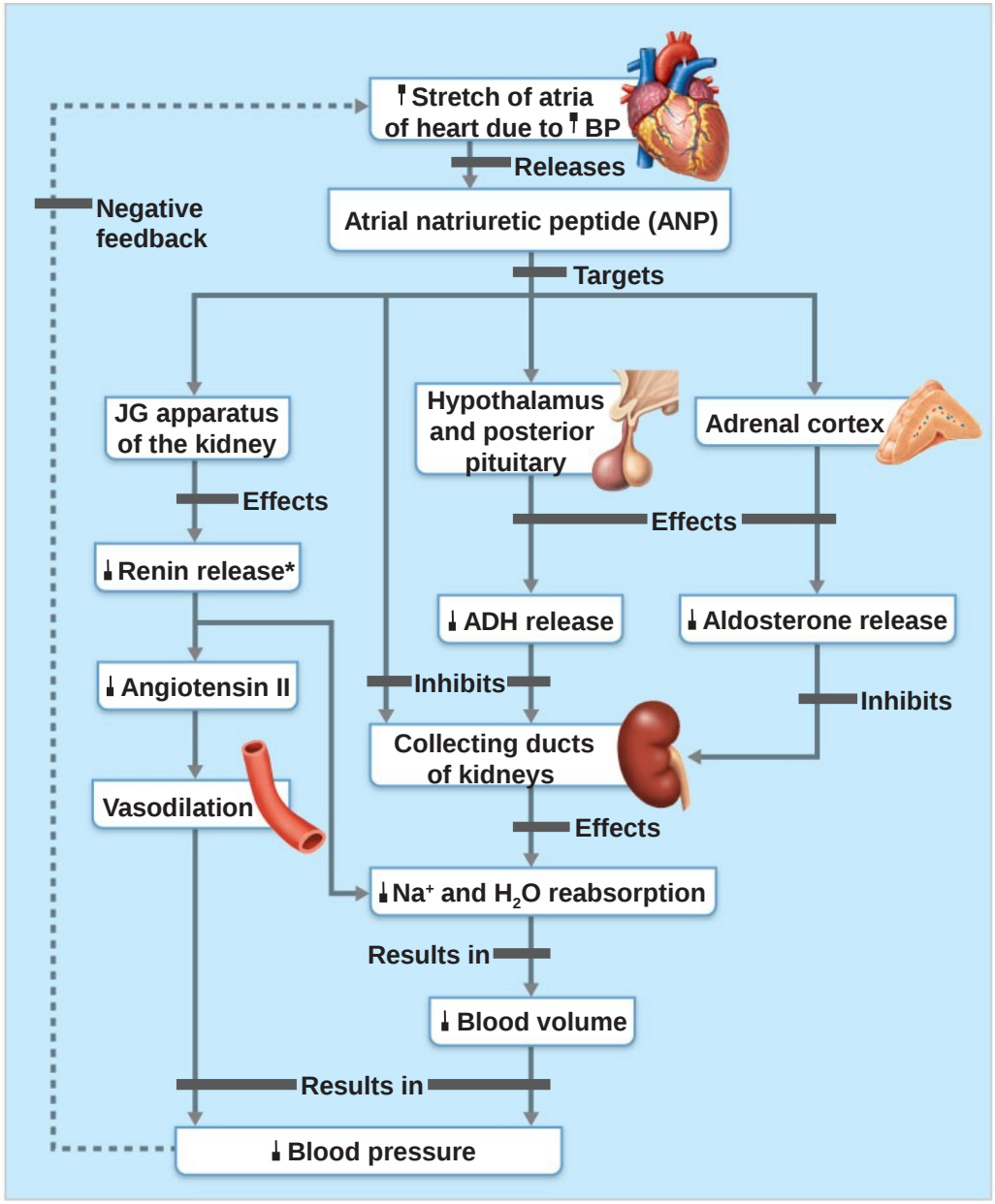

↑ Stretch of atria of heart due to ↑ BP
Releases
Atrial natriuretic peptide (ANP)
Negative feedback
Targets
JG apparatus of the kidney
Hypothalamus and posterior pituitary
Adrenal cortex
Effects
Effects
↓ Renin release*
↓ ADH release
↓ Aldosterone release
↓ Angiotensin II
Inhibits
Inhibits
Collecting ducts of kidneys
Vasodilation
Effects
↓ Na^+ and H_2O reabsorption
Results in
↓ Blood volume
Results in
↓ Blood pressure

Figure 26.9

Influence of Other 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

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
  - Na^+ and water output increase

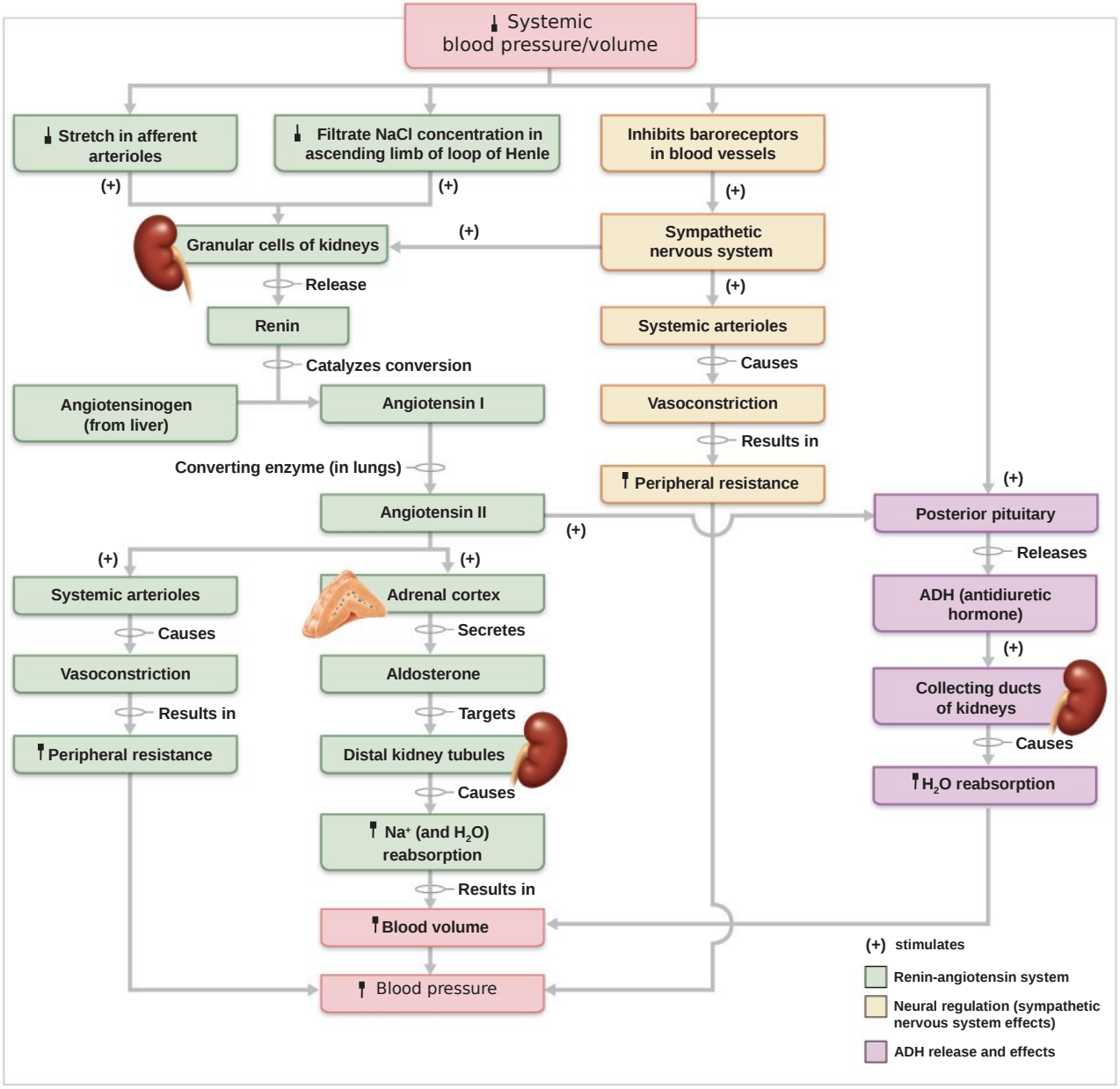

↓ Systemic blood pressure/volume
↓ Stretch in afferent arterioles
↓ Filtrate NaCl concentration in ascending limb of loop of Henle
Inhibits baroreceptors in blood vessels
(+)
(+)
(+)
Granular cells of kidneys
(+)
Sympathetic nervous system
Release
(+)
Renin
Systemic arterioles
Catalyzes conversion
Causes
Angiotensinogen (from liver)
Angiotensin I
Vasoconstriction
Results in
Converting enzyme (in lungs)
↑ Peripheral resistance
(+)
Angiotensin II
(+)
Posterior pituitary
(+)
(+)
Releases
Systemic arterioles
Adrenal cortex
ADH (antidiuretic hormone)
Causes
Secretes
(+)
Vasoconstriction
Aldosterone
Collecting ducts of kidneys
Results in
Targets
Causes
↑ Peripheral resistance
Distal kidney tubules
↑ H_2O reabsorption
Causes
↑ Na^+ (and H_2O) reabsorption
Results in
↑ Blood volume
↑ Blood pressure
(+) stimulates
Renin-angiotensin system
Neural regulation (sympathetic nervous system effects)
ADH release and effects

Figure 26.10

Regulation of Potassium Balance

- Importance of potassium:
  - Affects resting membrane potential in neurons and muscle cells (especially cardiac muscle)
    - ↑ ECF $[K^+]$ → ↓RMP → depolarization & inactivation of Na^+ channels.
    - ↓ ECF $[K^+]$→ 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

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

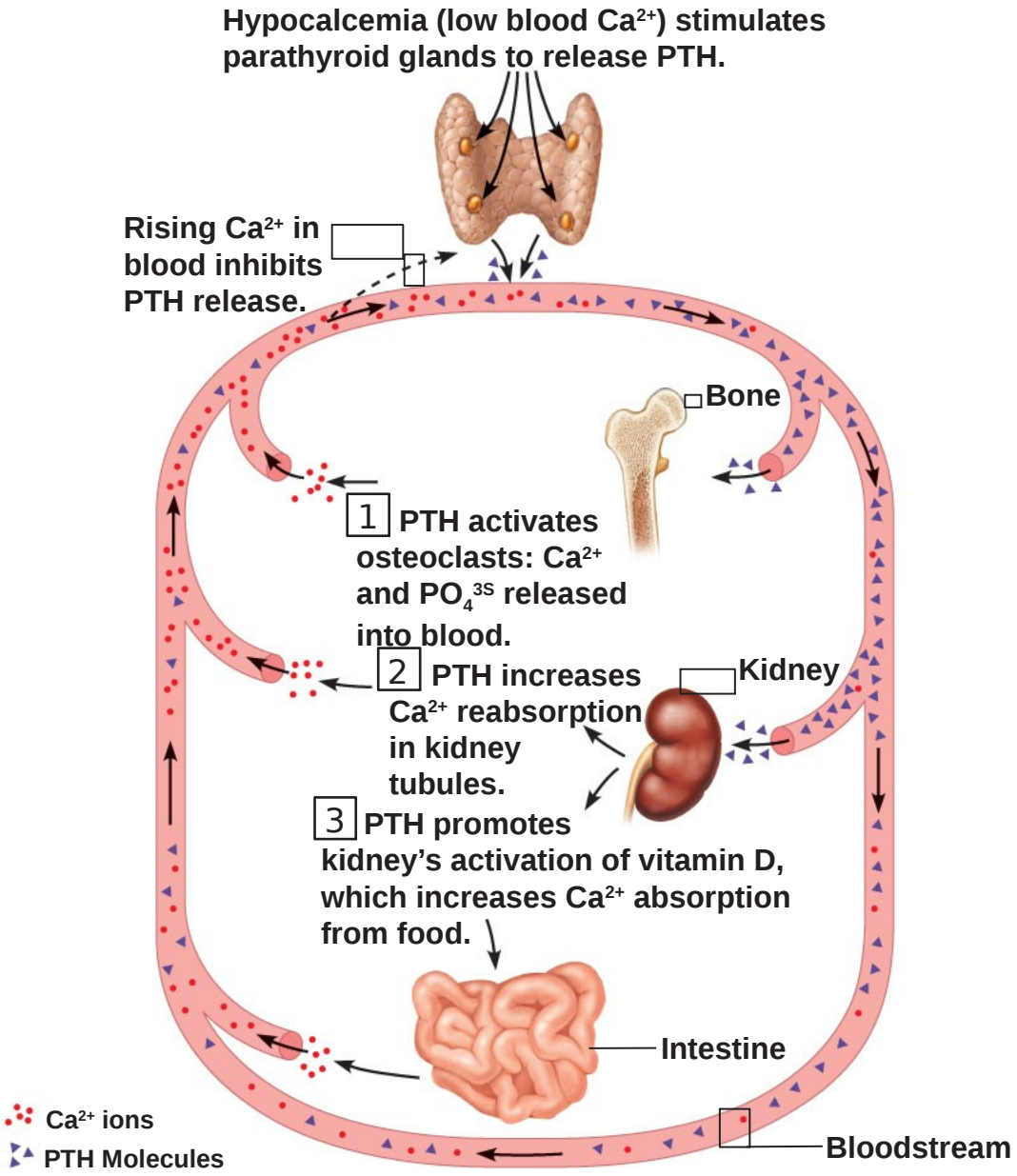

Hypocalcemia (low blood Ca^{2+}) stimulates parathyroid glands to release PTH.
Rising Ca^{2+} in blood inhibits PTH release.
1
Bone
1 PTH activates osteoclasts: Ca^{2+} and PO_4^{3S} released into blood.
2 PTH increases Ca^{2+} reabsorption in kidney tubules.
Kidney
3 PTH promotes kidney's activation of vitamin D, which increases Ca^{2+} absorption from food.
Intestine
Bloodstream
Ca^{2+} ions
PTH Molecules

Figure 16.12

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

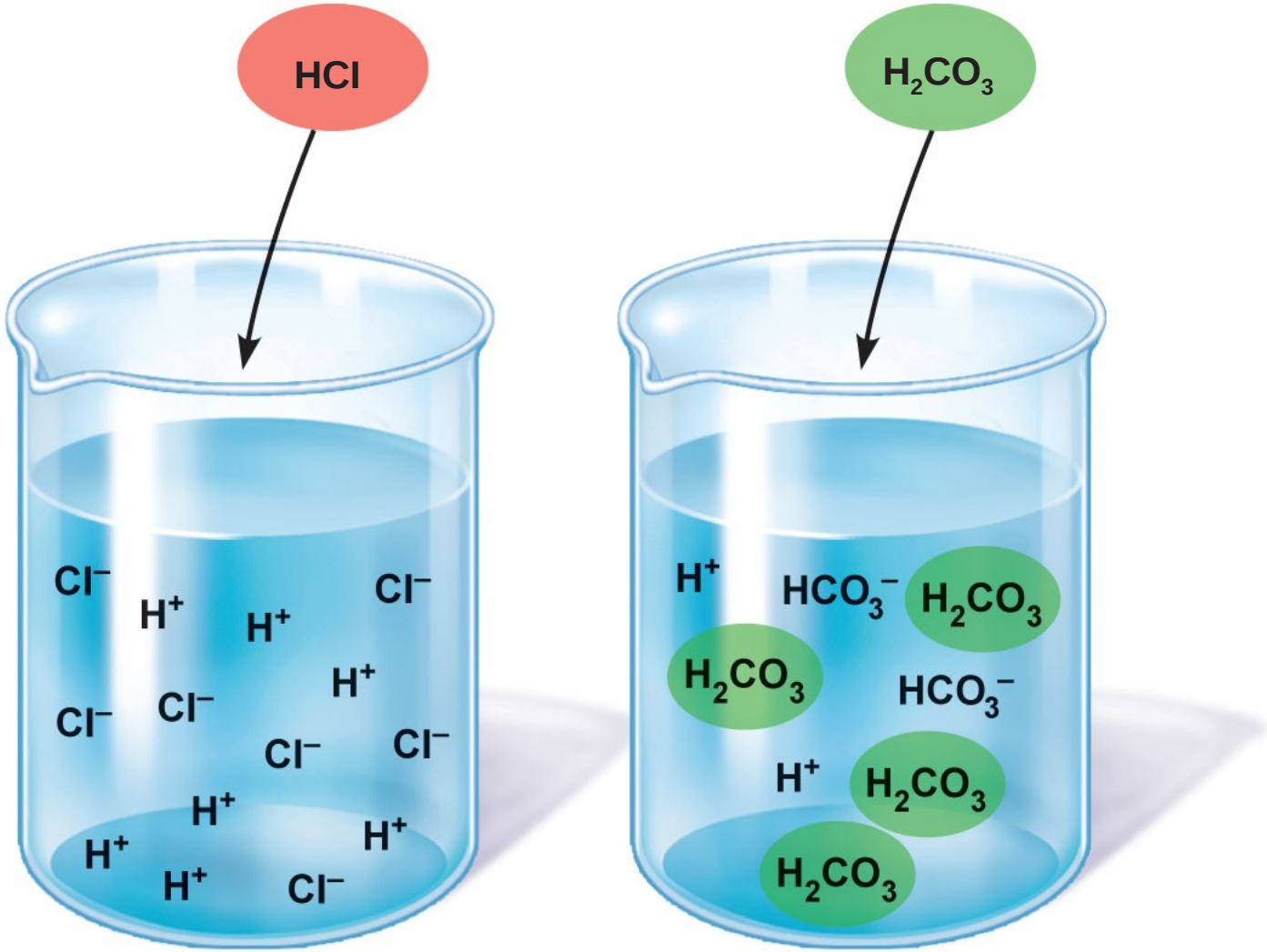
- Most H^+ is produced by metabolism
  - Phosphoric acid from breakdown of phosphorus-containing proteins in ECF
  - Lactic acid from anaerobic respiration of glucose
  - Fatty acids and ketone bodies from fat metabolism
  - H^+ liberated when CO_2 is converted to HCO_3^- in blood

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_2CO_3 does *not* dissociate completely.

Figure 26.11

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 H_2CO_3 (weak acid) and salts of 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_2CO_3
    - $HCl + NaHCO_3 \rightarrow H_2CO_3 + NaCl$
  - 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 H_2CO_3 to dissociate and donate H^+
  - H^+ ties up the base (e.g. OH^-)
    - $NaOH + H_2CO_3 \rightarrow NaHCO_3 + H_2O$
  - pH rises only slightly
  - H_2CO_3 supply is almost limitless (from CO_2 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 ($H_2PO_4^-$), 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 CO_2
- A reversible equilibrium exists in the blood:
  – $CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-$
- During CO_2 unloading the reaction shifts to the left (and H^+ is incorporated into H_2O)
- During CO_2 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 $\rightarrow$ respiratory acidosis
  - Hyperventilation $\rightarrow$ 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 H_2CO_3 produced in reactions catalyzed by carbonic anhydrase inside the cells
  - See Steps 1 and 2 of the following figure

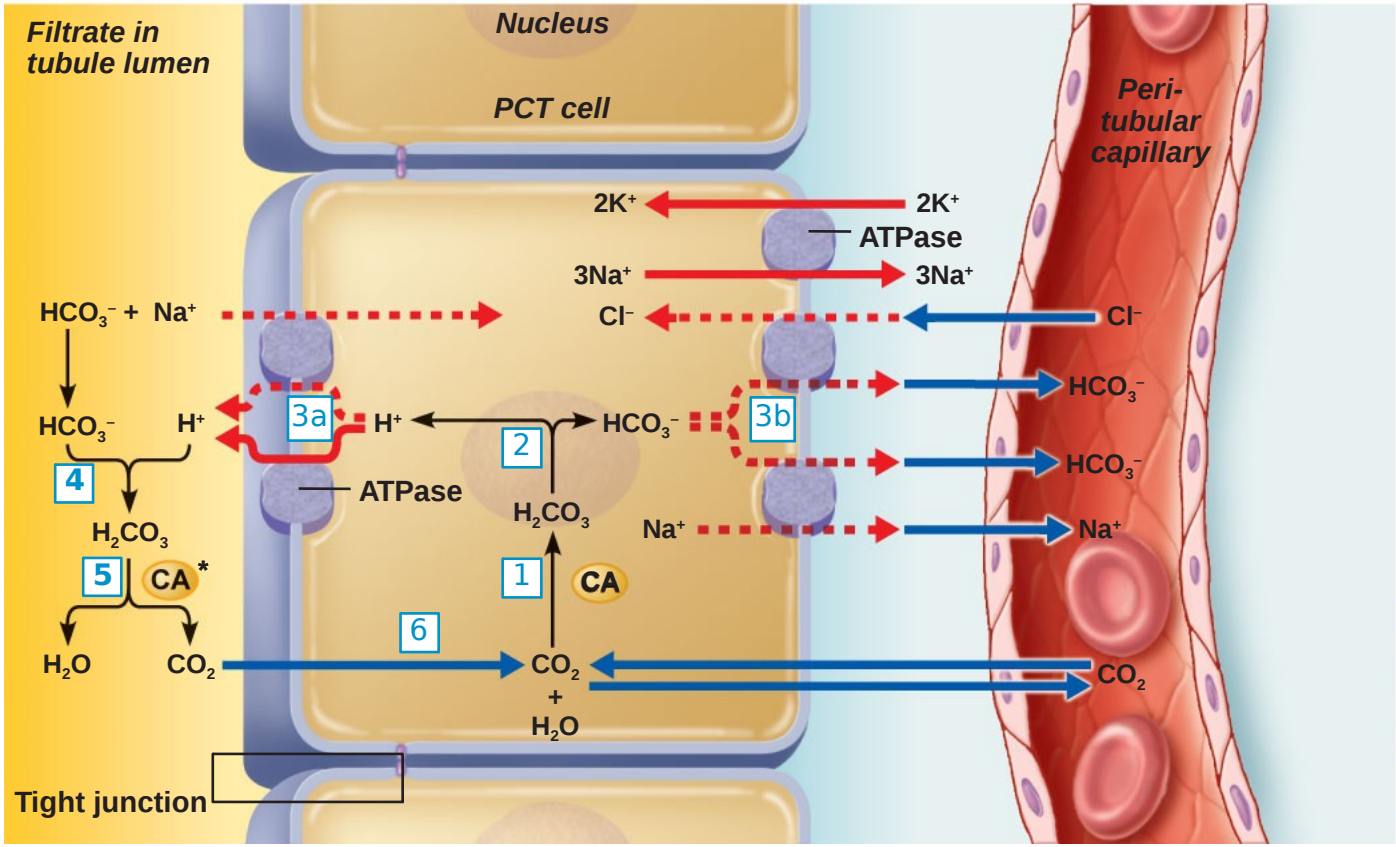
1 CO_2 combines with water within the tubule cell, forming H_2CO_3.

2 H_2CO_3 is quickly split, forming H^+ and bicarbonate ion (HCO_3^-).

3a H^+ is secreted into the filtrate.

3b For each H^+ secreted, a HCO_3^- enters the peritubular capillary blood either via symport with Na^+ or via antiport with Cl^-.

4 Secreted H^+ combines with HCO_3^- in the filtrate, forming carbonic acid (H_2CO_3). HCO_3^- disappears from the filtrate at the same rate that HCO_3^- (formed within the tubule cell) enters the peritubular capillary blood.



5 The H_2CO_3 formed in the filtrate dissociates to release CO_2 and H_2O.

6 CO_2 diffuses into the tubule cell, where it triggers further H^+ secretion.

- Primary active transport
- Secondary active transport
- Simple diffusion
- Transport protein
- CA Carbonic anhydrase

Figure 26.12

Excretion of Buffered H^+

- Dietary H^+ must be balanced by generating new HCO_3^-
- Most filtered HCO_3^- 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 NH_4^+ and 2 “new” HCO_3^-
- HCO_3^- moves to the blood and NH_4^+ is excreted in urine

1 PCT cells metabolize glutamine to NH_4^+ and HCO_3^-.

2a This weak acid NH_4^+ (ammonium) is secreted into the filtrate, taking the place of H^+ on a Na^+- H^+ antiport carrier.

2b For each NH_4^+ secreted, a bicarbonate ion (HCO_3^-) enters the peritubular capillary blood via a symport carrier.

3 The NH_4^+ is excreted in the urine.

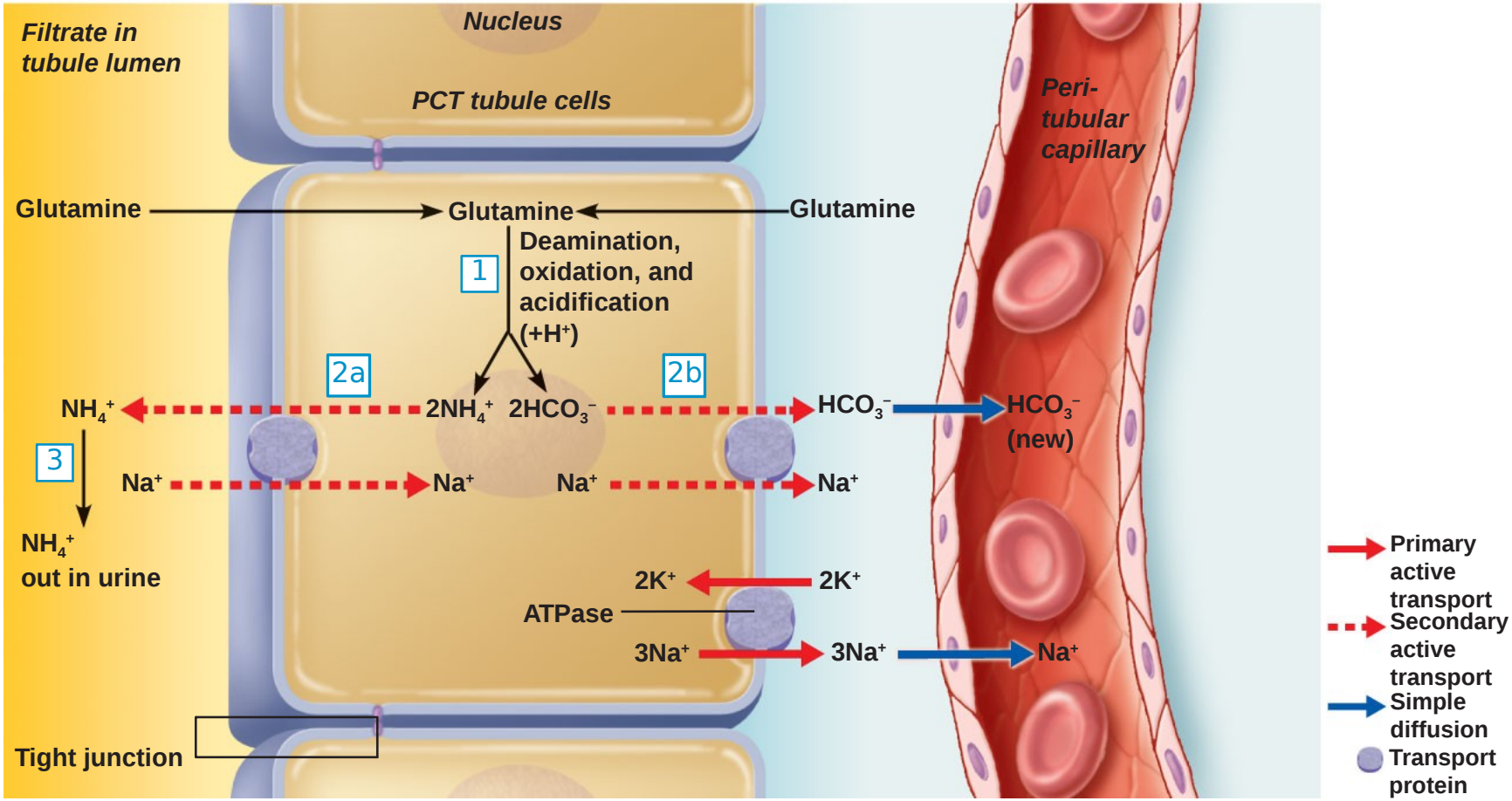


Figure 26.14

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 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 $\rightarrow$ 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 ($\rightarrow$ acetic acid)
  - Excessive loss of 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 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 CO_2 accumulation in the blood
  - High pH (over 7.45) and elevated 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