## McCance: Pathophysiology, 6th Edition

### Chapter 03: The Cellular Environment: Fluids and Electrolytes, Acids and Bases

### Test Bank

### TRUE/FALSE

1. The two main intracellular fluid (ICF) compartments are the interstitial fluid contained in the nucleus and the intravascular fluid contained in the cell body.

ANS: F
Two thirds of the body's water is **ICF** and one third is in the **extracellular fluid (ECF)** compartments. The two main ECF compartments are the **interstitial fluid** and the **intravascular fluid,** which is the blood plasma. Other ECF compartments include the lymph and the transcellular fluids, such as the synovial, intestinal, biliary, hepatic, pancreatic, and cerebrospinal fluids; sweat; urine; and pleural, synovial, peritoneal, pericardial, and intraocular fluids.

REF: p. 96

2. Infants have the highest overall percentage of body water.

ANS: T
In newborn infants, TBW is about 75% to 80% of body weight because infants store less fat. The percentage of TBW decreases to about 67% of body weight during the first year of life (Table 3-2).

REF: p. 97

3. Movement of water from the ICF to the ECF is primarily accomplished by active transport.

ANS: F
The movement of water between ICF and ECF compartments is primarily a function of osmotic forces. (Osmosis and other mechanisms of passive transport are discussed in Chapter 1.)

REF: p. 97

4. Loss of sodium and gain of water can cause hyponatremia.

ANS: T

Hypotonic fluid imbalances occur when the osmolality of the ECF is less than normal. The most common causes are sodium deficit **(hyponatremia)** or free water excess **(water intoxication).**

REF: p. 104

5. Insulin and glucose can be given to correct hyperkalemia.

ANS: T
*Insulin contributes to the regulation of plasma potassium levels* by stimulating the $Na^+$, $K^+$-ATPase pump, thereby promoting the movement of potassium into liver and muscle cells simultaneously with glucose transport after eating. Insulin also can be used to treat hyperkalemia. However, dangerously low levels of plasma potassium can result from the administration of insulin when potassium levels are depressed.

REF: p. 107

6. Hypokalemia as well as hyperkalemia can cause muscle weakness.

ANS: T
Symptoms of hyperkalemia vary, but common characteristics are muscle weakness or paralysis and dysrhythmias with changes in the electrocardiogram. A wide range of metabolic dysfunctions may result from potassium deficiency. Neuromuscular excitability is decreased, causing skeletal muscle weakness, smooth muscle atony, and cardiac dysrhythmias.

REF: pp. 108, 110

7. When buffers, such as carbonic acid-bicarbonate, absorb excessive $H^+$ (acid) or $OH^-$ (base), a significant change in the plasma pH results.

ANS: F
**Buffers** can absorb excessive $H^+$ (acid) or $OH^-$ (base) without a significant change in pH.

REF: p. 115

8. Proteins are primarily an intracellular buffer.

ANS: T
Phosphate and protein are the most important intracellular buffers.

REF: p. 115

**MULTIPLE CHOICE**

1. Why are infants susceptible to significant losses in total body water (TBW)?
a. Because more than half of an infant’s body weight is water
b. Because infants have a slow metabolic rate
c. Because an infant’s kidneys are not mature enough to counter fluids losses
d. Because they are unable communicate adequately when they are thirsty

ANS: C
Infants are particularly susceptible to significant changes in TBW because of their high metabolic rate and the accelerated turnover of body fluids caused by their greater body surface area in proportion to total body size. Loss of fluids from diarrhea can represent a significant proportion of body weight. Renal mechanisms that regulate fluid and electrolyte conservation may not be mature enough to counter the losses, so dehydration may develop rapidly.

REF: p. 97

2. Why are obese people at greater risk for dehydration than lean people?
a. Because adipose cells contain little water; fat is water repelling
b. Because the metabolic rate of obese adults is slower than lean adults
c. Because the rate of urine output of obese adults is higher than lean adults
d. Because the thirst receptors of the hypothalamus do not function effectively

ANS: A
The percentage of TBW varies with the amount of body fat and age. Because fat is water repelling (hydrophobic), very little water is contained in adipose cells. Individuals with more body fat have proportionately less TBW and tend to be more susceptible to fluid imbalances that cause dehydration.

REF: p. 97

3. Which groups are at risk for fluid imbalance?
a. Older adults, thin women, and infants
b. Infants, children, and obese persons
c. Thin women, obese persons, and older adults
d. Obese persons, older adults, and infants

ANS: D
Kidney function, surface area, TBW, and the hydrophobic nature of fat cells all contribute to the increased risk for fluid imbalance among these populations.

REF: p. 97

4. Water movement between the intracellular fluid compartment and the extracellular compartment is primarily a function of:

a. osmotic forces.
b. plasma oncotic pressure.
c. antidiuretic hormone.
d. hydrostatic forces.

ANS: A
The movement of water between ICF and ECF compartments is primarily a function of osmotic forces. (Osmosis and other mechanisms of passive transport are discussed in Chapter 1.)

REF: p. 97

5. How does the body reestablish equilibrium when solute is added to extracellular fluid making it hypertonic?
a. Water is drawn from the extracellular space to the intracellular space.
b. Sodium is drawn from the extracellular space to the intracellular space.
c. Sodium is drawn from the intracellular space to the extracellular space.
d. Water is drawn from the intracellular space to the extracellular space.

ANS: D
ECF volume deficit increases the ECF osmotic pressure, and intracellular water is attracted to the ECF space (see *E*). Water from the intracellular space has moved to the extracellular space until the osmotic forces are equal. The consequence is a decrease in ICF water volume and cell size.

REF: p. 98; Figure 3-1 (D and E)

6. Retention of sodium and water is a cause of edema because of an increase in which pressure?
a. Capillary hydrostatic pressure
b. Interstitial hydrostatic pressure
c. Capillary oncotic pressure
d. Interstitial oncotic pressure

ANS: A
An *increase in hydrostatic pressure* can result from venous obstruction or salt and water retention. *Venous obstruction* can increase the hydrostatic pressure of fluid within the capillaries enough to cause fluid to escape into the interstitial spaces.

REF: p. 99

7. At the arterial end of capillaries, fluid moves from the intravascular space into the interstitial space because the:
a. interstitial hydrostatic pressure is higher than the capillary hydrostatic pressure.
b. capillary hydrostatic pressure is higher than the capillary oncotic pressure.
c. interstitial oncotic pressure is higher than the interstitial hydrostatic pressure.

d. capillary oncotic pressure is lower than the interstitial hydrostatic pressure.

ANS: B
At the arterial end of capillaries, fluid moves from the intravascular space into the interstitial because capillary hydrostatic pressure is higher than the capillary oncotic pressure.

REF: pp. 99-100

8. Low plasma albumin causes edema as a result of a reduction in which pressure?
a. Capillary hydrostatic pressure
b. Interstitial hydrostatic pressure
c. Capillary oncotic pressure
d. Interstitial oncotic pressure

ANS: C
*Losses or diminished production of plasma albumin* contributes to a decrease in plasma oncotic pressure.

REF: p. 100

9. Secretion of antidiuretic hormone (ADH) and the perception of thirst are stimulated by a(n):
a. decrease in serum sodium.
b. increase in plasma osmolality.
c. increase in glomerular filtration rate.
d. decrease in osmoreceptor stimulation.

ANS: B
Secretion of ADH and perception of thirst are primary factors in the regulation of water balance. Thirst is a sensation that stimulates water-drinking behavior. Thirst is experienced when water loss equals 2% of an individual's body weight or when there is an increase in osmolality.

REF: p. 102

10. When thirst is experienced, how are osmoreceptors activated?
a. By an increase in the antidiuretic hormone secreted into the plasma
b. By an increase in aldosterone secreted into the plasma
c. By an increase in the hydrostatic pressure of the plasma
d. By an increase in the osmotic pressure of the plasma

ANS: D

Thirst is experienced when water loss equals 2% of an individual's body weight or when there is an increase in osmolality. Dry mouth, hyperosmolality, and plasma volume depletion activate **osmoreceptors** (neurons located in the hypothalamus that are stimulated by increased osmolality).

REF: p. 102

11. What is the action of natriuretic peptides?
a. They decrease blood pressure and increase sodium and water excretion.
b. They increase blood pressure and decrease sodium and water excretion.
c. They increase heart rate and decrease potassium excretion.
d. They decrease heart rate and increase potassium excretion.

ANS: A
**Natriuretic peptides** are hormones that include atrial natriuretic peptide (ANP) produced by the myocardial atria, brain natriuretic peptide (BNP) produced by the myocardial ventricles, and urodilatin within the kidney. Natriuretic peptides decrease blood pressure and increase sodium and water excretion.

REF: p. 102

12. ________ alterations occur when changes in TBW are accompanied by proportional changes in electrolytes.
a. Isotonic
b. Hypertonic
c. Hypotonic
d. Normotonic

ANS: A
Isotonic alterations occur when changes in TBW are accompanied by proportional changes in electrolytes and water.

REF: p. 102

13. Which enzyme is secreted by the juxtaglomerular cells of the kidney when circulating blood volume is reduced?
a. Angiotensin I
b. Angiotensin II
c. Aldosterone
d. Renin

ANS: D
When circulating blood volume or blood pressure is reduced, **renin,** an enzyme secreted by the juxtaglomerular cells of the kidney, is released in response to sympathetic nerve stimulation and decreased perfusion of the renal vasculature.

REF: p. 101

14. What mechanisms cause hypernatremia?
a. Syndrome of inappropriate antidiuretic hormone (SIADH)
b. Cushing disease and hyperaldosteronism
c. Prolonged vomiting or diarrhea
d. Excessive diuretic therapy

ANS: B
More commonly, high sodium levels occur because of (1) inadequate free water intake, (2) inappropriate administration of hypertonic saline solution (e.g., sodium bicarbonate for treatment of acidosis during cardiac arrest), (3) high sodium levels as a result of oversecretion of aldosterone (as in primary hyperaldosteronism), or (4) Cushing syndrome (caused by excess secretion of adrenocorticotropic hormone [ACTH], which also causes increased secretion of aldosterone).

REF: p. 104

15. What causes the clinical manifestations of confusion, convulsions, cerebral hemorrhage, and coma in hypernatremia?
a. The high sodium in the blood vessels pulls water out of brain cells into the blood vessels, causing brain cells to shrink.
b. The high sodium in the brain cells pulls water out of blood vessels into the brain cells, causing them to swell.
c. The high sodium in the blood vessels pulls potassium out of brain cells, which slows the synapses within the brain.
d. The high sodium in the blood vessels draws chloride into the brain cells followed by water, causing brain cells to swell.

ANS: A
Hypertonic (hyperosmolar) imbalances result in an ECF concentration greater than 0.9% salt solution, i.e., water loss or solute gain; cells shrink in a hypertonic fluid (Table 3-5).

REF: p. 103

16. Which are indications of dehydration?
a. Decreased hemoglobin and hematocrit
b. Muscle weakness and decreased deep tendon reflexes
c. Tachycardia and weight loss
d. Polyuria and hyperventilation

ANS: C

Marked water deficit is manifested by symptoms of dehydration: headache, thirst, dry skin and mucous membranes, elevated temperature, weight loss, and decreased or concentrated urine (with the exception of diabetes insipidus). Skin turgor may be normal or decreased. Symptoms of hypovolemia, including tachycardia, weak pulses, and postural hypotension, may be present.

REF: p. 104

17. How does SIADH cause excess water?
a. The increase in antidiuretic hormone causes retention of sodium that retains excessive water in the renal tubules.
b. The decrease in antidiuretic hormone increases serum glucose, which binds to water.
c. The decrease in antidiuretic hormone prevents the renal tubules from reabsorbing water.
d. The increase in antidiuretic hormone causes retention of water in the renal tubules.

ANS: D
SIADH is not caused by excess water intake but by increased renal reabsorption of water as a result of inappropriate increases in ADH. Serum sodium and osmolality are reduced by dilution. The kidney continues to excrete sodium, and urine sodium and urine osmolality are elevated; water is reabsorbed, increasing body fluid volume, and urine volume is decreased.

REF: p. 106

18. How is insulin used to treat hyperkalemia?
a. Insulin stimulates sodium to be removed from the cell in exchange for potassium.
b. Insulin binds to potassium to remove it through the kidneys.
c. Insulin transports potassium from the blood to the cell along with glucose.
d. Insulin breaks down the chemical components of potassium so that it is no longer effective.

ANS: C
*Insulin contributes to the regulation of plasma potassium levels* by stimulating the $Na^+$, $K^+$-ATPase pump, thereby promoting the movement of potassium into liver and muscle cells simultaneously with glucose transport after eating. The intracellular movement of potassium prevents an acute hyperkalemia related to food intake. Insulin also can be used to treat hyperkalemia.

REF: p. 107

19. What is a major determinant of the resting membrane potential necessary for transmission of nerve impulses?

a. The ratio between intracellular $Na^+$ and extracellular sodium
b. The ratio between intracellular $K^+$ and extracellular potassium
c. The ratio between intracellular $Na^+$ and extracellular $K^+$
d. The ratio between intracellular $K^+$ and extracellular $Na^+$

ANS: B
The ratio of $K^+$ in the ICF to $K^+$ in the ECF is the major determinant of the resting membrane potential, which is necessary for the transmission and conduction of nerve impulses, maintenance of normal cardiac rhythms, and skeletal and smooth muscle contraction.

REF: p. 106

20. During acidosis, the body compensates for the increase in hydrogen ions in the blood by shifting hydrogen ions into the cell in exchange for which electrolyte?
a. Oxygen
b. Sodium
c. Potassium
d. Magnesium

ANS: C
In states of acidosis, hydrogen ions shift into the cells in exchange for ICF potassium; hyperkalemia and acidosis therefore often occur together.

REF: p. 110

21. What are causes of hyperkalemia?
a. Hyperparathyroidism and malnutrition
b. Vomiting and diarrhea
c. Renal failure and Addison disease
d. Hyperaldosteronism and Cushing disease

ANS: C
Hyperkalemia should be investigated when there is a history of renal disease, massive trauma, insulin deficiency, Addison disease, use of potassium salt substitutes, or metabolic acidosis.

REF: p. 111

22. In hyperkalemia, cardiac rhythm changes are a direct result of:
a. cardiac cell hypopolarization.
b. cardiac cell hyperexcitability.
c. depression of the sinoatrial (SA) node.
d. cardiac cell repolarization.

ANS: A

If extracellular potassium concentration increases without a significant change in intracellular potassium, the resting membrane potential becomes more positive (i.e., changes from –90 to –80 mV) and the cell membrane is **hypopolarized** (the inside of the cell becomes less negative or partially depolarized [increase excitability]). (Electrical properties of cells are discussed in Chapter 1.)

REF: pp. 110-111

23. The calcium and phosphate balance is influenced by which three substances?
a. Parathyroid hormone, vasopressin, and vitamin D
b. Parathyroid hormone, calcitonin, and vitamin D
c. Thyroid hormone, vasopressin, and vitamin A
d. Thyroid hormone, calcitonin, and vitamin A

ANS: B
Calcium and phosphate balance is regulated by three hormones: parathyroid hormone (PTH), vitamin D, and calcitonin.

REF: p. 111

24. Which are causes of hypocalcemia?
a. Repeated blood administration or pancreatitis
b. Kidney stones and immobility
c. Decreased resorption of calcium and phosphate from bone
d. Hyperparathyroidism and bone metastasis

ANS: A
Blood transfusions are also a common cause of hypocalcemia because the citrate solution used in storing whole blood binds with calcium. Pancreatitis causes release of lipases into soft tissue spaces, so the free fatty acids that are formed bind calcium, causing a decrease in ionized calcium.

REF: p. 112

25. Chvostek sign and Trousseau sign indicate:
a. hypokalemia.
b. hyperkalemia.
c. hypocalcemia.
d. hypercalcemia.

ANS: C
Two clinical signs are Chvostek sign and Trousseau sign.

REF: p. 112

26. Excessive use of magnesium- and aluminum-containing laxatives can result in:

a. hypomagnesemia.
b. hypophosphatemia.
c. hyponatremia.
d. hypokalemia.

ANS: B
The most common causes are intestinal malabsorption and increased renal excretion of phosphate. Inadequate absorption is associated with vitamin D deficiency, use of magnesium and aluminum-containing antacids (which bind with phosphorus), long-term alcohol abuse, and malabsorption syndromes.

REF: p. 113

27. Hypermagnesemia is usually caused by:
a. hepatitis.
b. renal failure.
c. trauma to the hypothalamus.
d. pancreatitis.

ANS: B
**Hypermagnesemia,** in which magnesium concentration is greater than 2.5 mEq/L, is rare and usually is caused by renal failure.

REF: p. 114

28. Physiologic pH is maintained around 7.4 because bicarbonate ($HCO_3$) and carbonic acid ($H_2CO_3$) exist in a ratio of:
a. 20:1.
b. 1:20.
c. 10:2.
d. 10:5.

ANS: A
The relationship between bicarbonate and carbonic acid is usually expressed as a ratio. When the pH is 7.40, this ratio is 20:1 (bicarbonate/carbonic acid).

REF: p. 115

29. What initiates the formation of ammonium ($NH_4$) from ammonia ($NH_3$) in the tubular lumen of the kidney?
a. Arterial pH of 7.25
b. Arterial pH of 7.35
c. Arterial pH of 7.55
d. Arterial pH of 7.65

ANS: A

Pathophysiologic changes in the concentration of hydrogen ion or base in the blood lead to acid-base imbalances. **Acidemia** is a state in which the pH of arterial blood is less than 7.35. $NH_3$ is produced from glutamine in the epithelial cell and diffuses to the tubular lumen, where it combines with $H^+$ to form $NH_4$.

REF: p. 117

30. What is the significance of deep, rapid breathing (Kussmaul respirations) in metabolic acidosis?
a. It indicates that anxiety, with rapid breathing, is a cause of respiratory acidosis.
b. It indicates the excessive carbon dioxide is exhaled to compensate for metabolic acidosis.
c. It indicates that diabetic ketoacidosis is the cause of the metabolic acidosis.
d. It indicates that more oxygen is necessary to compensate for respiratory acidosis.

ANS: B
Deep, rapid respirations (Kussmaul respirations) are indicative of respiratory compensation for metabolic acidosis.

REF: p. 118

31. How does the loss of chloride during vomiting cause metabolic alkalosis?
a. Loss of chloride stimulates the release of aldosterone, which causes the retained sodium to bind with the chloride.
b. Loss of chloride causes hydrogen to move into the cell and exchange with potassium to maintain cation balance.
c. Loss of chloride causes retention of bicarbonate to maintain the anion balance.
d. Loss of chloride causes hypoventilation to compensate for the metabolic alkalosis.

ANS: C
When acid loss is caused by vomiting with depletion of ECF and chloride **(hypochloremic metabolic alkalosis),** renal compensation is not very effective because the volume depletion and loss of electrolytes ($Na^+$, $K^+$, $H^+$, $Cl^-$) stimulate a paradoxical response by the kidneys. The kidneys increase sodium and bicarbonate reabsorption with excretion of hydrogen. Bicarbonate is reabsorbed to maintain an anionic balance because the ECF chloride concentration is decreased.

REF: p. 119

32. A patient has a long history of smoking. He has blood studies done because he is very tired, is short of breath, and just does not feel well. His blood gases reveal the following findings: pH, 7.3; $HCO_3$ 27 mEq/L; $CO_2$, 58 mmHg. What is the interpretation of these gases?
a. Respiratory alkalosis

b. Metabolic acidosis
c. Respiratory acidosis
d. Metabolic alkalosis

ANS: C
Acute uncompensated respiratory acidosis is characterized by decreased arterial pH, elevated $PCO_2$, and normal or slightly increased bicarbonate.

REF: p. 121

**MATCHING**

*Match the electrolytes with the corresponding descriptions. Terms may be used more than once.*

a. Sodium
b. Chloride
c. Potassium
d. Magnesium
e. Phosphate

1. Regulation of osmolality
2. Inversely related to $HCO_3$ concentration
3. Major determinant of resting membrane potential
4. An intracellular metabolic form is ATP
5. Changes in hydrogen ion concentration affect this electrolyte

1. ANS: A REF: p. 98
NOT: Sodium is the most abundant ECF ion and is responsible for the osmotic balance of the ECF space. Potassium maintains the osmotic balance of the ICF space.

2. ANS: B REF: p. 120
NOT: Chloride levels are inversely related to HCO3 concentration (Figure 3-13).

3. ANS: C REF: p. 106
NOT: The ratio of K+ in the ICF to K+ in the ECF is the major determinant of the resting membrane potential, which is necessary for the transmission and conduction of nerve impulses, maintenance of normal cardiac rhythms, and skeletal and smooth muscle contraction. (Membrane transport and membrane potentials are discussed in Chapter 1.)

4. ANS: E REF: p. 111
NOT: Phosphate acts as an intracellular and extracellular anion buffer in the regulation of acid-base balance; in the form of ATP it provides energy for muscle contraction.

5. ANS: C REF: p. 110
NOT: In states of acidosis, hydrogen ions shift into the cells in exchange for ICF potassium; hyperkalemia and acidosis therefore often occur together.

*Match each electrolyte imbalance with its clinical manifestations.*
a. Hyponatremia
b. Hypernatremia
c. Hypercalcemia
d. Hypokalemia
e. Hypomagnesemia

6. Absent bowel sounds, skeletal muscle weakness, bradycardia
7. Depression, confusion, irritability, increased reflexes, tetany
8. Confusion, irritability, depression, headache, seizures
9. Fatigue, weakness, kidney stones, varying degrees of heart block
10. Hypotension, fever, tachycardia

6. ANS: D REF: p. 109
NOT: A variety of dysrhythmias may occur, including sinus bradycardia, atrioventricular block, and paroxysmal atrial tachycardia, and paralytic ileus.

7. ANS: E REF: p. 114
NOT: Signs and symptoms of hypomagnesemia are similar to those of hypocalcemia. Depression, confusion, irritability, increased reflexes, muscle weakness, ataxia, nystagmus, tetany, convulsions, and tachyarrhythmias may be observed.

8. ANS: A REF: p. 106
NOT: Behavioral and neurologic changes characteristic of hyponatremia include lethargy, headache, confusion, apprehension, seizures, and coma.

9. ANS: C REF: p. 113
NOT: Fatigue, weakness, lethargy, anorexia, nausea, and constipation are common. Behavioral changes may occur. Impaired renal function frequently develops, and kidney stones form as precipitates of calcium salts. A shortened QT segment and depressed widened T waves also may be observed on the ECG, with bradycardia and varying degrees of heart block.

10. ANS: B REF: p. 104
NOT: Thirst, fever, dry mucous membranes, hypotension, tachycardia, low jugular venous pressure, and restlessness are associated with hypernatremia as a result of water loss.