




ELECTROLYTES,



Fluids, and the Acid–Base Balance

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Electrolytes are chemical substances that separate in solution (usually water [H₂O]) to form electrically charged particles or ions.¹ Electrolytes are measured in millimoles/liter (mmol/L) or milliequivalents/liter (mEq/L).¹ When negatively charged, these ions are called *anions*; when positively charged, *cations*.¹

The most important anions involved in chemical activities within the body are chloride (Cl⁻), bicarbonate (HCO₃⁻), and phosphate (HPO₄²⁻). The principal cations are sodium (Na⁺), potassium (K⁺), calcium (Ca²⁺), and magnesium (Mg²⁺). Several pairs of opposite-charged ions are so closely linked that when one particle is affected, it directly impacts the other (e.g., sodium/chloride, calcium/phosphate).²

Function of Electrolytes

Because electrolytes carry an electrical charge, they are involved in many bodily functions. These include changes in ion concentration gradients (e.g., osmosis, diffusion, Na⁺–K⁺ pump), energy production, enzymatic reactions (e.g., digestion, metabolism), acid–base balance, and any process that requires a muscle contraction or relaxation.^{1,2}

Distribution Within the Body

The body is divided into three primary compartments for fluid distribution (intravascular, interstitial, intracellular). The intravascular compartment, located inside blood vessels, consists of fluid (plasma) and cellular components (erythrocytes, leukocytes, platelets). The interstitial compartment consists of the fluid surrounding the cells, excluding the fluid within the blood vessels. The intravascular and interstitial compartments combine to make up the extracellular fluid. Conversely, the intracellular compartment consists of fluid found inside the cell walls (Figure 1; Table I).^{1,2}

Intracellular fluid contains a primary cation (potassium), a leading cation (magnesium), and a primary anion (phosphate). Interstitial fluid contains a primary anion (chloride), a leading anion (bicarbonate), and a primary cation (sodium). Calcium is present in almost equivalent amounts in intracellular and interstitial fluids (Table I).^{1,2}

Acid–Base Balance

The pH level is used to express a solution's hydrogen-ion concentration. Acids (e.g., carbonic acid [H₂CO₃]) are chemical substances that give up hydrogen

KEY POINTS

- *Electrolytes have multiple and varied responsibilities but are unable to function on their own.*
- *Positively charged ions are called cations; negatively charged ions are called anions.*
- *The body is divided into three primary compartments for fluid distribution.*

molecules. Bases (e.g., bicarbonate) are substances that accept these donated hydrogen molecules.² As is true of most chemical reactions, the body functions best at a specific pH level. For example, the pH level in arterial blood in dogs and cats should range from 7.35 to 7.45.^{1,2} This level is achieved by the interaction of acids and bases within the animal's body. Generally, pH level is maintained in a bicarbonate:carbonic acid ratio of 20:1. A pH level lower than 6.8 (acidotic) or higher than 7.8 (alkalotic) is usually fatal. Acid–base adjustments can be made through changes in the bicarbonate level in the blood, through respiration (hypoventilation or hyperventilation), through the use of chemical buffers (e.g., phosphorus, proteins), and via kidney excretion or retention of acids or bases.²

Anions Bicarbonate

Although it is considered an electrolyte, the primary activity of bicarbonate is regulation of the acid–base balance,¹ which is accomplished when carbon dioxide (CO₂; in the form of bicarbonate) is transported from the lungs to tissue. Normal bicarbonate levels in arterial blood range from 20 to 25 mEq/L in dogs and 17 to 21 mEq/L in cats (Table II). This level can also be expressed as total CO₂ serum content, with a normal range of 16 to 26 mEq/L in dogs and 15 to 21 mEq/L in cats.² As bicarbonate and serum pH levels decrease, animals become acidotic. Alkalosis occurs when bicarbonate and serum pH levels rise.

A decrease in serum bicarbonate caused by metabolic problems (e.g., increased loss through diuresis) results in metabolic acidosis with a pH level less than 7.35 (see the Glossary; Table III). An increased respiratory rate may help the body compensate by “blowing off” CO₂ and increasing serum pH. Increased respiration without a metabolic component (e.g., anxious panting) may result in respiratory acidosis if enough CO₂ is lost

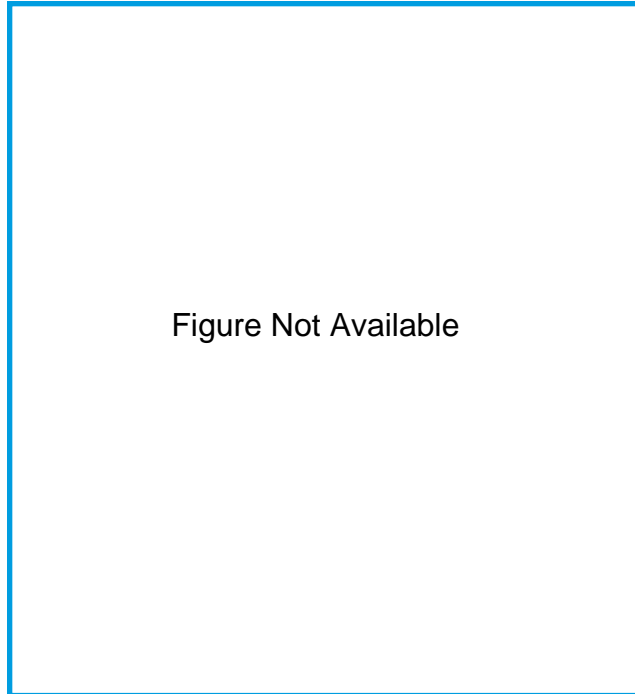


Figure 1—Relative distribution of ions within the fluid compartments of the body. The intravascular and interstitial compartments combine to form the extracellular compartment. (Data from Thurman JC, Tranquilli WJ, Benson GJ (eds): *Lumb and Jones' Veterinary Anesthesia*, ed 3. Baltimore, Lea & Febiger, 1996, p 573.)

that it affects the serum pH level.²

An increased serum bicarbonate level caused by a metabolic problem (e.g., anuric renal failure, Addison's disease) will result in metabolic alkalosis (pH over 7.45) and may be followed by a decreased respiratory rate to conserve CO₂. Decreased respiration without a metabolic component (e.g., an animal under anesthesia) could result in respiratory alkalosis if CO₂ is allowed to accumulate in the serum.^{1,2}

Carbon dioxide is the end-product of carbohydrate oxidation. As carbohydrates are metabolized, glucose is produced and then broken down into H₂O and CO₂. In tissue, CO₂ in the presence of the enzyme carbonic anhydrase reacts with H₂O to form carbonic acid. The carbonic acid breaks down to form bicarbonate and a free hydrogen (H⁺) ion.

Chloride is the most abundant anion found in extracellular fluid. Normal serum levels range from 105 to 116 mEq/L in dogs and 113 to 121 mEq/L in cats, but actual levels within the cells are only 4 mEq/L.¹ Chloride can move in and out of cells and combines readily with other

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TABLE I
Electrolyte Distribution Across Fluid Compartments^a

Table Not Available

cations (e.g., sodium to form sodium chloride [salt; NaCl]).² Considered corresponding chemicals, chloride and sodium help form the cerebrospinal fluid as well as control serum osmolality and H₂O balance in the body.

Chloride is secreted with hydrogen in the stomach to form hydrochloric acid (HCl). Chloride is needed to maintain the acid–base balance. This compound is reabsorbed and excreted in direct opposition to bicarbonate. When chloride levels become elevated, bicarbonate levels decrease, resulting in acidosis. When chloride levels decrease, bicarbonate levels rise, resulting in alkalosis. Chloride also assists in CO₂ transport within erythrocytes.²

Hypochloremia results when chloride levels decrease to less than 105 mEq/L. This condition can be caused by decreased intake or absorption of chloride; increased loss of the electrolyte through the skin, gut, or kidneys; or changes in sodium levels or acid–base balances. Hypochloremia can also result from prolonged vomiting or diarrhea, draining fistulas, metabolic alkalosis,

TABLE II
Normal Electrolyte Values in Dogs and Cats^a

Table Not Available

diabetic ketoacidosis, Addison's disease, rapid removal of ascitic fluid (Figure 2), or congestive heart failure.² Muscle cramping, twitching, weakness, cardiac arrhythmia, seizure, coma, and respiratory arrest are signs of hypochloremia.²

Hyperchloremia occurs when chloride levels increase to higher than 121 mEq/L and is usually associated with an acid–base imbalance, which may occur when bicarbonate levels decrease and sodium and chloride levels increase. The condition can be caused by dehydration, renal tubular acidosis, renal failure, metabolic acidosis, aspirin toxicity, hyperparathyroidism, or hyperaldosteronism.² Common signs of hyperchloremia are the same as those associated with metabolic acidosis: tachycardia; lethargy; weakness; deep, rapid respirations progressing to cardiac arrhythmia; decreased cardiac output; coma; and eventually death.²

The primary source of chloride is salt. Chloride is also abundant in igneous rock formed from solidified molten rock. Little information is available about chloride content in pet foods because of the difficulty in isolating this compound from sodium. In diets in which NaCl has been removed (e.g., specifically purified or concentrated diets), a sodium deficiency will occur before a chloride deficiency does.³

Phosphorus

Phosphorus, in the form of phosphate, is the primary anion in intracellular fluid. Of the body's total phosphorus content, 85% can be found in bones and teeth, 14% in soft tissue (e.g., muscle),

GLOSSARY

- Acidosis**—Condition resulting from acid accumulation or bicarbonate depletion in the blood and tissue; characterized by decreased pH
- Addison's disease**—Disorder resulting from a deficiency in the secretion of adrenocortical hormones
- Alkalosis**—Condition resulting from the accumulation of base or loss of acid without comparable loss of base in body fluids; characterized by increased pH
- Anuric renal failure**—Condition that occurs when the kidneys fail to secrete sufficient urine or the secreted urine fails to reach the bladder
- ATP**—Adenosine triphosphate; energy-rich compound involved in many metabolic reactions
- Calcitonin**—Hormone essential to bone and calcium metabolism
- Cushing's syndrome**—Condition resulting from hypersecretion of the adrenal cortex, causing excessive production of glucocorticoids
- Eclampsia**—Toxemia associated with pregnancy that is often accompanied by hypertension, seizures, diminished urine production, and coma
- Halogens**—Elements that combine with hydrogen ions to form acids and with metals to form salts
- Metabolic**—Pertaining to changes that occur within living cells as a result of energy or material transformation
- Osmolality**—The characteristic of a solution determined by the ionic concentration of the dissolved substance per unit of solvent
- Osmotic pressure**—Potential force that develops when two solutions of different concentrations are separated by a semipermeable membrane⁴
- Refeeding syndrome**—Condition that occurs because of excessive dietary carbohydrate ingestion after a period of starvation that can lead to potentially fatal insulin-induced phosphorus and potassium transport into cells

TABLE III
Electrolyte Disturbances and Causes^{1,2}

<i>Electrolyte Disturbance</i>	<i>Possible Causes</i>
Low bicarbonate level	Chronic renal failure, diabetic ketoacidosis, respiratory and/or metabolic acidosis, hyperparathyroidism, hyperaldosteronism
Elevated bicarbonate level	Anuric renal failure, respiratory and/or metabolic alkalosis, chronic vomiting with loss of hydrochloric acid, draining fistulas with protein loss, Addison's disease
Low sodium level	Addison's disease, excessive use of diuretics, draining wounds, vomiting or diarrhea
Elevated sodium level	Coma, diabetes insipidus, excessive sodium supplementation (e.g., hypertonic saline, sodium bicarbonate) in intravenous fluids, Cushing's syndrome
Low chloride level	Chronic vomiting with loss of hydrochloric acid, draining fistulas with protein loss, Addison's disease, metabolic alkalosis, rapid removal of ascetic fluid
Elevated chloride level	Metabolic acidosis, renal tubular necrosis, aspirin toxicity, hyperparathyroidism, hyperaldosteronism, dehydration
Low calcium level	Hypoparathyroidism, low serum albumin level, eclampsia, decreased magnesium intake, increased phosphorus intake (all-meat diet)
Elevated calcium level	Hyperparathyroidism, malignancy, increased serum albumin level
Low phosphorus level	Respiratory alkalosis, sepsis, heat stroke, refeeding syndrome, malabsorption and/or starvation
Elevated phosphorus level	Respiratory acidosis, increased phosphorus intake (all-meat diet), trauma with cellular damage, chronic renal failure
Low potassium level	Chronic vomiting, draining fistulas with protein loss, diabetes mellitus (osmotic diuresis), Cushing's syndrome
Elevated potassium level	Oversupplementation in fluids, urethral obstruction, Addison's disease, metabolic acidosis
Low magnesium level	Pancreatic exocrine insufficiency, decreased calcium intake, pancreatitis, chronic diarrhea
Elevated magnesium level	Addison's disease, chronic renal failure, diabetic ketoacidosis

and 1% in the extracellular fluid.² Normal serum levels of phosphorus range from 2.5 to 5.79 mg/dl in dogs and 3.18 to 5 mg/dl in cats.¹

Phosphorus is involved in cell membrane integrity (the phospholipid layer); muscle and neurologic function; carbohydrate, fat, and protein metabolism; oxygen delivery from the erythrocytes to tissue; and acid–base buffering. Phosphorus also aids in the transfer of energy to cells through the formation of ATP and is an essential component of bones and teeth.

Although hypophosphatemia occurs when phosphorus levels decrease to less than 2.5 mg/dl, this condition may not be caused by an actual phosphorus deficiency in the body. Only a small amount of phosphorus is available for measure in serum. Serum deficiency may occur secondary to a shift of phosphorus from the extracellular to the intracellular fluid or may result because of decreased intestinal absorption of phosphorus or increased loss of phosphorus

through the kidneys.² A serum phosphorus level lower than 1 mg/dl will cause the body to be unable to support its energy needs, resulting in organ failure. Phosphorus shifts may result from respiratory alkalosis (e.g., sepsis, heat stroke, acute aspirin poisoning), refeeding syndrome, malabsorption, starvation, or diarrhea (Figure 3).²

Because of the phosphorus requirements in ATP, signs of hypophosphatemia are often related to decreased energy stores² and may include muscle weakness, anorexia, dysphagia, and respiratory failure caused by decreased diaphragmatic contractility and decreased cardiac output. Decreased oxygen delivery to cells, depleted cell energy stores, seizures, and coma may also result.²

Conversely, hyperphosphatemia usually occurs when phosphorus levels are above 5 mg/dl. Although commonly caused by renal failure with impaired renal excretion, this condition can also occur with increased dietary intake (e.g., all-meat diets) and decreased parathyroid hormone

(PTH) synthesis.² Respiratory acidosis and increased cellular destruction (e.g., trauma) can increase phosphorus shifts from intracellular to extracellular fluid, resulting in increased serum phosphorus levels but not total-body phosphorus levels.² Because of the close link between phosphorus and calcium, hyperphosphatemia may result in signs consistent with hypocalcemia (see Calcium section), including muscle spasms, cramps, tetany, and tissue calcification.²

Phosphorus compounds are found in nature as phosphates and always occur in the form of orthophosphates on the earth's surface. Igneous rock formed by solidified molten rock is a major source of phosphate.³

The two most important sources of phosphorus in feed production are pelleted phosphorite and guano. Guano phosphate originates from the excrement of birds and bats living on limestone beds. Guano phosphate may be used as a feed supplement or as a soil fertilizer, thereby increasing phosphorus levels in feed.³ Phosphate is present in all common feeds that contain seeds and seed by-products (e.g., bran, oils) that have higher levels than do forages.³ Most of the phosphate found in pet foods comes from organic sources (e.g., poultry, meat, poultry meal, bone, grains). The phosphate in protein meals of animal origin (e.g., fishmeal, blood meal) is considered to have more bioavailability than do meals of plant origin (e.g., corn and soybean meal).

Cations

Sodium

Sodium is the most osmotically active extracellular particle. By attracting fluid, sodium helps preserve extracellular fluid volume and distribution within the body. This fluid preservation helps create osmotic pressure on the vessel walls, thereby preventing fluid loss. Normal sodium levels range from 145 to 154 mEq/L in dogs and 151 to 158 mEq/L in cats.^{1,2} Sodium also aids in the transmission of electrical impulses in nerve and muscle fibers and combines with chloride and bicarbonate to regulate acid-base balances.



Figure 2—Abdominal centesis is used to remove excess fluid from the abdomen. Removing too much fluid can cause sodium and chloride levels to drop.

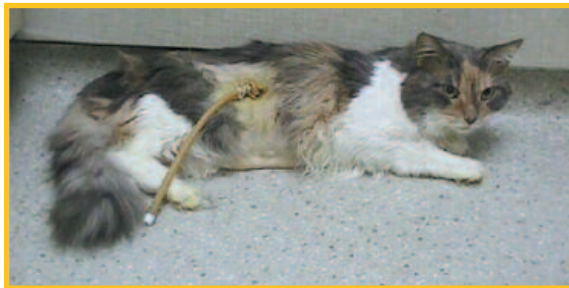


Figure 3—Thin body condition is typical of cats with hepatic lipidosis. Because animals with this condition go for long periods without eating, phosphorus levels are often low after feedings are reinitiated. Serum levels must be monitored closely to correct the problem early.

When sodium levels decrease to lower than 135 mEq/L, hyponatremia may result. This condition may occur from excessive diuretic use, Addison's disease, draining wounds, or hydrochloric acid loss from chronic vomiting. Primary neurologic signs result when fluid shifts within the brain. The rate of fluid loss affects the severity of signs, with rapid fluid loss causing more severe signs than progressive losses. Muscle twitching, changes in mentation, tremors, and cramps may occur with a slow fluid loss. When fluid loss is too rapid or severe, seizures and coma may occur.² The electrocardiogram (ECG) would show a widened QRS complex with an elevated ST segment and ventricular tachycardia progressing to fibrillation and death.¹

Hypernatremia, which results when sodium levels exceed 145 mEq/L, occurs less frequently than does hyponatremia and can potentially be caused by coma, diabetes insipidus, excessive

sodium supplementation (via intravenous fluids), and Cushing's syndrome. Elevated sodium levels can lead to seizures, coma, and permanent neurologic damage. Thirst is the body's main defense against hypernatremia. Because the need to respond to thirst is so strong, severe, persistent hypernatremia occurs only in animals that cannot drink voluntarily (e.g., coma, neglect).

The primary natural source of sodium is in the form of salt. Sodium cannot be found freely in nature but combines readily with other halogens (e.g., chloride).³

Potassium

Potassium is the primary intracellular cation, with at least 90% of this element located in the intracellular compartment. Normal serum potassium levels range from 4.1 to 5.3 mEq/L in dogs and 3.6 to 4.9 mEq/L in cats. Extracellular potassium makes up only about 2% of the total-body potassium; therefore, serum levels often do not accurately represent the extent or severity of a potassium disorder, especially in cases of chronic disease.^{1,2}

Potassium has a direct impact on cell, nerve, and mus-

cle function by maintaining the cell's electrical neutrality and osmolality, aiding in neuromuscular transmissions, assisting skeletal and cardiac muscle contractility, and affecting acid–base balance.² These activities are mostly accomplished through the action of the $\text{Na}^+\text{--K}^+$ pump, which is an active transport system that moves sodium out of the cells and puts potassium into the cells against the natural flow of diffusion. ATP aids as the energy source in this process.^{1,2}

Hypokalemia occurs when potassium levels fall below 4.1 mEq/L in dogs and 3.6 mEq/L in cats. Decreased potassium intake or increased renal or gastrointestinal (GI) losses (e.g., from chronic vomiting or diabetes mellitus) result in this condition. Draining fistulas with protein loss and Cushing's syndrome can also cause potassium loss. Because of its use in the $\text{Na}^+\text{--K}^+$ pump, decreased potassium levels usually result in muscle weakness and decreased GI motility. An ECG will show prolonged repolarization (the interval in which the $\text{Na}^+\text{--K}^+$ pump moves the potassium back into the cell and the sodium out), causing prolonged PR, QRS, and QT intervals; a decreased ST segment; and a flattened or inverted T wave. When the deficiency is severe, sinus bradycardia with heart block and atrioventricular dissociation can be seen.²

Hyperkalemia occurs when potassium levels exceed 5.3

mEq/L in dogs and 4.9 mEq/L in cats. The condition may be iatrogenic (caused by oversupplementation via intravenous fluids) or caused by urethral obstruction, metabolic acidosis, or Addison's disease.² Signs may include decreased muscle contractility, delayed neuromuscular impulse transmission, intracellular swelling caused by the buildup of intracellular ions, and bradycardia. An ECG would show peaked T waves and decreased P-wave amplitude, progressing to asystole or ventricular fibrillation.²

Although potassium cannot be found as a free molecule in nature, it does appear in combined substances (e.g., potassium chloride [KCl]). In contrast to calcium and phosphorus, potassium is not readily stored in the body; therefore, it must be supplied daily in the diet. The highest concentration of potassium can be found in plant leaves rather than in stems or seeds. The amount of potassium found in feeds is affected by the maturity of the feed; the potassium level in the feed decreases with age and soil fertility. The highest levels of potassium can be found in products of animal origin, especially meat and blood meals.³

Calcium

Calcium plays an important role in the physiology of neuromuscular function, cell membrane permeability,

muscle contraction, hemostasis, and clot formation.^{1,2} Normal calcium levels range from 8.9 to 12.2 mg/dl for dogs and 8.9 to 9.13 mg/dl for cats.¹ Calcium and phosphorus are responsible for bone formation. In fact, the majority of calcium in the body (99%) is located in the bones; only 1% can be found in the serum. Roughly 40% of the 1% in the serum is found in ionized form, roughly 50% is protein bound, and 10% is bound to phosphorus and bicarbonate.¹ The calcium that is usually measured in the serum is the ionized or unbound form. Because roughly 50% of the calcium is protein bound, changes in protein levels can affect total calcium levels without affecting a change in ionized calcium levels.

Changes in body stores and dietary intake can affect serum levels. Vitamin D promotes increased calcium absorption in the small intestine, whereas phosphorus inhibits calcium absorption. Changes in pH can also affect the amount of ionized calcium that is available. When an increase in pH (alkalosis) occurs, more calcium binds with protein, causing a decrease in ionized serum calcium. When a decrease in pH (acidosis) occurs, less calcium binds with protein, increasing the level of ionized serum calcium.² With a lowered calcium level, PTH is produced and causes calcium to be released from bones. In animals with higher calcium levels, PTH levels are suppressed by the release of calcitonin from the thyroid gland.²

Hypocalcemia occurs with calcium levels less than 8.9 mg/dl and can be caused by decreased calcium intake or absorption, decreased vitamin D intake, increased calcium loss, and, in particular, PTH abnormalities. Increased calcium loss is often experienced with parathyroid damage during thyroid surgery causing secondary hypoparathyroidism, low albumin levels, or eclampsia. In animals, total serum calcium levels can be adjusted with low levels of albumin as follows^{1,2}:

$$\text{Serum calcium (mg/dl)} - \text{Serum albumin (g/dl)} + 3.5 = \text{Adjusted serum calcium}$$

Calcium levels below 6.5 mg/dl are generally metabolic in origin.¹ Decreased magnesium levels and increased phosphorus levels that occur from eating primarily an all-meat diet can also decrease serum calcium.² Animals with hypocalcemia often show signs of restlessness, muscle fasciculations, tetany, or convulsions. Prolonged QT and ST segments secondary to prolonged myocardial action potentials may be present on ECGs.¹

Hypercalcemia, which can be caused by hyperparathyroidism, malignancy, or increased albumin levels, occurs when the calcium level exceeds 12.2 mg/dl in dogs and 9.13 mg/dl in cats. When PTH levels increase, bones re-

lease more calcium. In addition, a malignancy may result in increased bone destruction, causing the release of a substance similar to PTH, which in turn will release more calcium into the bloodstream.^{1,2} Increased calcium levels secondary to hypophosphatemia and acidosis can also occur.¹

Animals with hypercalcemia show signs of anorexia, vomiting, generalized weakness, and hypertension. An ECG would show bradycardia with a prolonged PR interval and shortened ST segment.^{1,2}

Calcium is found naturally only in compounds, chiefly limestone (i.e., calcium carbonate), calcium fluoride, and calcium sulfate. Most grains (e.g., barley, corn, sorghum, oats, wheat) are deficient in calcium, whereas most forages (e.g., grass hay, alfalfa, clover hay) have intermittent levels of the element depending on growing conditions, fertilizers, or soil amendments used. The highest levels of calcium are present in alfalfa and clover. Milk products, ground limestone, and bone meal products provide the best sources of dietary calcium.³

Magnesium

Magnesium is the second most abundant cation in the intracellular fluid compartment. Approximately 60% of the body's magnesium can be found in the bones and teeth, 39% in intracellular fluid, and less than 1% in the extracellular fluid. Normal serum magnesium levels range from 1.7 to 2.8 mg/dl in dogs and 1.79 to 2.72 mg/dl in cats.¹ Serum magnesium levels do not accurately reflect actual body stores because of the relatively small amount of magnesium that is usually found in the blood. Approximately 30% of serum magnesium is protein bound; therefore, reduced albumin levels may falsely decrease the serum reading even if actual levels are within normal limits.²

Magnesium promotes enzymatic reactions within cells during carbohydrate metabolism and helps the body produce and use ATP. This element is involved in protein synthesis and influences vasodilation, thereby helping the cardiovascular system to function normally. Magnesium aids the Na⁺–K⁺ pump in both depolarization and repolarization, thereby affecting the irritability and contractility of cardiac and skeletal muscle. It also influences the body's calcium level by its effect on PTH.²

Hypomagnesemia can occur when magnesium levels are less than 1.7 mEq/L. This condition can be caused by decreased magnesium intake, poor GI absorption caused by low levels of calcium or high levels of phosphorus in the diet, increased GI loss (occurring with pancreatic exocrine insufficiency), and chronic diarrhea. Signs of this condition (e.g., muscle weakness, myocardial irritability and arrhythmia, dysphagia, depression, seizures) are similar to those of hypocalcemia or hypokalemia. When mag-

nesium levels fall below 1.0 mEq/L, respiratory muscle paralysis, complete heart block, and coma result.

Hypermagnesemia can be caused by increased magnesium intake or decreased renal output (e.g., Addison's disease, chronic renal failure, diabetic ketoacidosis). This condition usually occurs when magnesium levels exceed 2.7 mEq/L. Central nervous system depression may progress to coma; bradycardia may progress to heart block and cardiac arrest; and respiratory depression may progress to respiratory arrest and vasodilation. An ECG would show a prolonged R interval, widened QRS complex, and tall T wave.²

Magnesium is found naturally only in compounds (e.g., magnesite, carnallite, dolomite, epsomite, kieserite). Most cereal grains (e.g., rice, oat, wheat) are fair sources of magnesium. Plant proteins are excellent sources, whereas animal protein sources are more variable in content.³

Conclusion

Electrolytes have multiple and varied responsibilities but are unable to function on their own. It is difficult to obtain an accurate assessment of some electrolytes because of their location within the body. The veterinary team must pay careful attention to all presenting signs, not simply individual test values. For example, an electrolyte value may have changed in response to acidosis or alkalosis. In addition, the animal may have experienced trauma or starvation. Factors such as these play an important role in accurate diagnosis and proper treatment. Because technicians usually perform initial patient assessments, draw blood, and set up ECGs, they must have a clear understanding of the entire picture. Knowledge of electrolyte function as well as the ability to accurately evaluate blood work will help technicians better anticipate the needs of their patients.

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