Clinically important hypophosphatemia in small animal practice has not been commonly recognized. However, because hypophosphatemia can result in a variety of clinical signs, some of which are life threatening, persistent moderate hypophosphatemia and severe hypophosphatemia should be considered problems that necessitate treatment. This article presents an overview of the cause, pathogenesis, clinical implications, and therapeutic approach for hypophosphatemic patients.

Organic phosphate is present in phospholipids, phosphoproteins, nucleic acids, enzymes, ATP, and cAMP. Inorganic phosphate is a substrate in many vital functions of the body, including oxidative phosphorylation, production of 2,3-diphosphoglycerate (2,3-DPG) in erythrocytes, and production of glycogen in the liver and kidneys. Phosphatase also stimulates glycolysis by stimulating glycolytic enzymes and participating in phosphorylation of many glycolytic intermediates. Phosphate ions play a role in the acid–base balance in serum and in excretion of acids in urine because of their ability to buffer hydrogen ions (H+).

Phosphorus in the mammalian body is predominantly (85% to 90%) present as hydroxyapatite in the mineralized matrix of bone, with most of the remaining 10% to 15% occurring in the cytoplasm of cells. Only a small amount (less than 1%) of nonosseous inorganic phosphate is extracellular and accessible for routine laboratory measurements in plasma or serum. Thus the plasma phosphorus concentration reflects only a small proportion of the total body stores. Depletion of these phosphorus stores is not always reflected in a decrease in the plasma phosphorus concentration. On the other hand, a decreased plasma phosphorus concentration does not always indicate decreased total body phosphorus. Rapid translocations can occur between intracellular and plasma phosphorus pools that can dramatically change the plasma phosphorus concentration. For example, alkalinization of the blood or influx of glucose into cells causes translocation of phosphorus from plasma into cytosol.

REGULATION OF TOTAL BODY PHOSPHORUS

The plasma phosphorus concentration is primarily regulated by the kidneys. Renal excretion of phosphorus is determined by the glomerular filtration rate and the maximum tubular reab-
Approximately 90% of plasma phosphorus is filtered by the glomerulus. Of this amount, 80% to 90% is reabsorbed in the renal tubules. Most renal phosphorus reabsorption occurs in the proximal convoluted tubule, with small amounts of phosphorus being reabsorbed in the distal nephron. Reabsorption is sodium dependent because phosphorus transport is performed by a brush border sodium–phosphate cotransporter. The sodium gradient is maintained by a sodium–phosphate adenosine triphosphatase and makes phosphorus reabsorption indirectly energy dependent. Maximal tubular reabsorption of phosphorus can normally be saturated, resulting in phosphaturia when excess phosphorus enters tubular fluid.

The regulatory mechanism for renal phosphorus reabsorption can adapt to the body’s need for phosphorus through parathyroid hormone (PTH), the major hormonal regulator. PTH reduces reabsorption of phosphorus by decreasing the maximal tubular reabsorption rate in the proximal tubule while it enhances calcium reabsorption in the distal tubule. Thus PTH release results in an increased calcium concentration and phosphaturia. Low blood levels of PTH result in the opposite. The major stimulus for increased PTH synthesis and secretion is a reduced plasma calcium concentration. The major inhibitors of PTH synthesis and secretion are increased concentrations of plasma calcium and 1,25-dihydroxycholecalciferol, the active form of vitamin D. In the presence of phosphorus depletion, the kidneys conserve phosphorus, and the renal response to the phosphaturic effect of PTH is blunted.

Although the kidneys are the major regulator of the plasma phosphorus concentration, ultimately, the concentration is the combined effect of intestinal absorption and excretion, bone resorption and accretion, and renal excretion and reabsorption (Figure 1). The skeleton functions as a reservoir from which phosphorus is mobilized during states of hypophosphatemia. Absorption of dietary phosphorus is approximately 80% and occurs by passive diffusion and by active transport using a sodium–phosphate cotransporter. Absorption occurs in the small intestine, primarily in the mid-jejunum. Because of intestinal phosphorus excretion, a total of about 30% to 40% of ingested phosphorus is excreted in the feces.

Decreased intestinal absorption of phosphorus occurs in vitamin D deficiency, in malabsorption syndromes (e.g., steatorrhea, pancreatitis, lymphangiectasia), and with a phosphorus-deficient diet. In addition, substances containing iron, aluminum (e.g., aluminum hydroxide), or unsaturated fatty acids interfere with intestinal phosphorus absorption. The active form of vitamin D, produced by epithelial cells of the proximal convoluted tubules in the kidneys, increases intestinal absorption of phosphorus. It also increases plasma phosphorus concentrations by stimulating bone resorption and possibly by some small contribution of increased renal tubular resorption. Renal production of active vitamin D is increased by PTH and a low dietary content of phosphorus and/or hypophosphatemia. Renal synthesis is also increased by growth hormone, estrogen, and prolactin, which are important hormones during growth, pregnancy, and lactation, respectively. Renal production of active vitamin D is inhibited by hyperphosphatemia, hypercalcemia, and renal diseases characterized by loss of renal tubular mass.

Hypomagnesemia has been associated with hypophosphatemia, but there does not seem to be a real
cause-and-effect relationship between magnesium and phosphorus; they most often seem to occur conjunctively in diseases in which depletion of elements is the problem.\textsuperscript{7,13}

**MEASURING PHOSPHORUS LEVELS**

Phosphorus is measured in plasma or serum as inorganic orthophosphate by colorimetric assays after reduction of a phosphomolybdate compound.\textsuperscript{14} Phosphate circulates mostly as a free anion but can be bound to sodium ions (Na\textsuperscript{+}), magnesium ions (Mg\textsuperscript{2+}), or calcium ions (Ca\textsuperscript{2+}) or to protein (10% to 15% of total plasma phosphorus).\textsuperscript{6} Most (80%) plasma inorganic phosphate is in the dibasic form (HPO\textsubscript{4}\textsuperscript{2–}), and the remaining 20% is primarily in the monobasic form (H\textsubscript{2}PO\textsubscript{4}\textsuperscript{–}), with only a minimal amount as phosphate ions (PO\textsubscript{4}\textsuperscript{3–}).\textsuperscript{6} This ratio depends on the blood pH; at physiologic pH, the average valence to plasma inorganic phosphate is −1.8, indicating that the milliequivalence of plasma phosphorus can be estimated by the following calculation:

\[
1 \text{ mmol/L of Phosphate} = 1.8 \text{ mEq/L of Phosphate}
\]

When alkalalemia occurs, more of the dibasic form is present, whereas when acidemia occurs, more monobasic phosphate is present.\textsuperscript{15} Therefore, plasma phosphorus levels are usually expressed in milligrams per deciliter for elemental phosphorus or millimoles per liter for phosphate ions because both units, unlike milliequivalents, are independent of the blood pH (3.1 mg of phosphorus/dl = 1 mmol/L of phosphate).

Normal values for dogs range from 2.5 to 6 mg/dl. It may be higher in younger animals (i.e., younger than 1 year of age) because growth hormone increases renal phosphorus reabsorption. The magnitude of plasma phosphorus elevation is similar in puppies of giant and small breeds and is approximately 8.5 mg/dl. Adult cats tend to have higher mean plasma phosphorus values than adult dogs. Mean plasma phosphorus values in cats younger than 1 year of age are not as elevated as those in young dogs.\textsuperscript{8} Clinicians should always be mindful that plasma phosphorus is an unreliable indicator of body stores: Hypophosphatemia does not always imply that phosphorus depletion exists. In contrast, severe phosphorus depletion may be present despite a normal or elevated plasma phosphorus concentration.\textsuperscript{7} Phosphorus may rapidly shift between the extracellular and intracellular compartments.\textsuperscript{15}

Serum or heparinized plasma can be used for measurements; citrate, oxalate, or EDTA should not be used as anticoagulants because they interfere with the assay. Fasting blood samples should be taken because a phosphorus-rich meal (e.g., meat) can increase plasma phosphorus levels, whereas a carbohydrate-rich meal can reduce them. Preventing hemolysis of blood samples, which artificially increases the measurement because of release of intracellular stores of inorganic phosphate, is important. To prevent leakage of cellular phosphorus,

**Rapid translocations can occur between intracellular and plasma phosphorus pools and dramatically change plasma phosphorus levels.**

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feeding high-carbohydrate diets). Insulin promotes intracellular uptake of phosphorus needed for increased glycolysis and phosphorylation of ADP to ATP. Rapid repair and regeneration of damaged tissue may cause uptake of extracellular phosphorus, resulting in hypophosphatemia. Chronic administration of glucocorticoids or Cushing’s disease can cause decreased phosphorus levels by transcellular shift into cells and increased phosphorus excretion into urine.

Changes in pH (e.g., respiratory alkalosis, diabetic ketoacidosis) also lead to transcellular shift of phosphorus. Respiratory alkalosis leads to rapid diffusion of carbon dioxide from the intracellular space into the extracellular fluid. The rise in intracellular pH accelerates phosphorylation of glucose, and inorganic phosphate is moved into the cell, resulting in hypophosphatemia. Respiratory alkalosis can be due to hyperventilation, which may be associated with fear, pain, septicemia, and central nervous system disorders (e.g., seizures). Hypophosphatemia of a modest degree is common in patients with bacteremia due to gram-negative organisms (presumably the result of hyperventilation and respiratory alkalosis).

In the veterinary literature, if hypophosphatemia is mentioned, it is usually in conjunction with diabetes mellitus. Phosphorus depletion is common in diabetic ketoacidosis and is due to excessive phosphorus loss in urine. The loss is caused by polyuria (i.e., osmotic diuresis) and decreased tubular reabsorption of phosphorus due to the presence of glucose (and ketones) in the tubular fluid. Metabolic acidosis enhances phosphaturia due to decomposition of intracellular organic phosphate compounds, causing phosphorus to move into the plasma and then be excreted in urine. There can be concurrent decreased intake from anorexia and vomiting. Despite the presence of phosphorus depletion, the plasma phosphorus concentration at presentation can be normal or even high because both insulin deficiency and metabolic acidosis can cause a shift of phosphorus out of the cells. This transcellular shift can be reversed and the plasma phosphorus concentration can rapidly decline after treatment with insulin, which promotes phosphorus (and glucose) entry into the cells under the influence of the enzyme hexokinase.

Calcium may also play a role in phosphorus losses in animals during the early stages of starvation and in association with total pa-
renteral nutrition. Increasing calcium levels in total parenteral nutrition solutions lowered urinary phosphorus losses and the degree of hypophosphatemia in rats fasted for 3 days.22

Hypovitaminosis D alone is an unlikely cause of low plasma phosphorus levels but may be contributory. Vitamin D deficiency may be caused by lack of exposure to sunlight, inadequate amounts of vitamin D in the diet, or steatorrhea, which results in intestinal malabsorption of vitamin D.23 If overused, gastrointestinal protectants and antacids containing aluminum hydroxide or magnesium hydroxide may combine with phosphorus, thereby limiting its absorption from the intestine. In humans, it has been reported that this causes hypophosphatemia but only when a phosphorus-deficient diet is being fed or when phosphorus depletion already exists.24,25

Hypophosphatemia can also result from increased renal excretion of phosphorus. Primary hyperparathyroidism and pseudohyperparathyroidism cause increased circulating plasma PTH or PTH-related protein levels, which enhance phosphaturia.8 Hypercalcemia has a direct effect on tubular cells, which enhances phosphaturia, thereby contributing to the development of hypophosphatemia.8 If renal function is impaired, hypophosphatemia might not be present. Renal secondary hyperparathyroidism is stimulated by transient or persistent elevations of plasma phosphorus levels as a result of a decreased glomerular filtration rate. Thus this condition is not associated with hypophosphatemia. Renal tubular loss of phosphorus due to primary renal disease (e.g., Fanconi syndrome) is an uncommon cause of hypophosphatemia. Diuretics may cause hypophosphatemia secondary to renal phosphorus loss.9

In cases of puerperal tetany (eclampsia), plasma calcium levels of less than 7 mg/dl often coexist with hypophosphatemia.10 Hemodialysis can deplete too much phosphorus from the blood.8 Severe hypothermia from environmental exposure has reportedly caused hypophosphatemia and hypercalcemia in a dog and cat.26 The plasma phosphorus values were less than 2 mg/dl in both instances but rapidly returned to normal following rewarming and parenteral fluid support. The mechanism for the hypophosphatemia was not explained.26 In human medicine, chronic alcoholism and thermal burns have also been described as causes of hypophosphatemia.31

**CONSEQUENCES**

Mild hypophosphatemia is characterized by plasma phosphorus levels of 2 to 2.5 mg/dl. It is rarely clinically significant, and the reason for the hypophosphatemia is usually undetermined.8 Clinical signs of hypophosphatemia in small animals, as in humans, vary, and many severely hypophosphatemic patients do not have clinical signs of hypophosphatemia. Signs in humans are generally unrecognized unless the plasma phosphorus level is less than 2 mg/dl. Serious side effects often are not seen until the plasma phosphorus level falls below 1 mg/dl.

Phosphorus is an important component of ATP and is therefore critical in certain energy-dependent physiologic processes. ATP is also required to maintain the integrity of cell membranes and cell shape and deformability.27–29 This makes nearly every organ system susceptible to the effects of hypophosphatemia. Hypophosphatemia mainly affects body cells that are high-energy users, including erythrocytes, skeletal muscle cells, and brain cells. Intracellular inorganic phosphate is a cofactor in anaerobic glycolysis, the sole pathway for erythrocyte synthesis of ATP and 2,3-DPG.11 In erythrocytes, the lack of ATP and decreased production of intracellular 2,3-DPG can cause both structural and functional abnormalities.30 Decreased 2,3-DPG increases the affinity of hemoglobin for oxygen. This shifts the oxyhemoglobin dissociation curve to the left, impairing oxygen delivery to peripheral tissue. Subsequent hypoxia is thought to be the cause of many of the clinical manifestations of hypophosphatemia.1,7,27 In patients with diabetic ketoacidosis, as long as acidosis is present, this effect is counterbalanced by the opposite effect of acidosis on the oxygen dissociation of hemoglobin.

Hemolysis is the most common complication of hypophosphatemia.1,2,28 Hemolysis occurs because of depleted ATP levels in erythrocytes. The concentration of erythrocyte ATP closely correlates with membrane deformability. In humans, hypophosphatemia-induced hemolytic anemia is rare and has been reported only in patients with severe hypophosphatemia (i.e., <0.25 mg/dl).27,31 In experimental models in dogs, hemolysis

**Many severely hypophosphatemic patients do not show clinical signs of hypophosphatemia.**
was induced only at very low plasma phosphorus concentrations (i.e., <0.5 mg/dl). However, cats may be more sensitive than other species because hypophosphatemia-induced hemolysis has been described in cats with plasma phosphorus concentrations above 1 mg/dl. In hemolytic cases, a decrease in hematocrit was seen within 24 to 48 hours after documented hypophosphatemia.

Severe hypophosphatemia can also impair leukocyte function secondary to depletion of cellular ATP. Leukocytes may show impaired chemotaxis, phagocytosis, and intracellular killing functions, making patients more susceptible to infections. In addition, platelets may function poorly, causing impaired clot retraction and cutaneous hemorrhage, and the platelets may have a decreased survival time in the blood. Thrombocytopenia and large-diameter platelets have been observed.

In humans and dogs, chronic, moderate hypophosphatemia can cause proximal myopathy characterized by weakness, osteomalacia, bone pain, muscle atrophy, and normal plasma activity of creatine kinase. Striated muscles have altered membrane potentials. The abnormalities are reversible with phosphorus repletion. Acute, severe hypophosphatemia can cause generalized myopathy characterized by muscle necrosis (rhabdomyolysis), myoglobinuria, diffuse muscle pain, generalized weakness, and elevated creatine kinase values. It appears that preexisting muscle cell injury (i.e., from chronic hypophosphatemia and phosphorus depletion) is necessary for the development of muscle necrosis secondary to acute hypophosphatemia. In human medicine, acute respiratory failure has been attributed to hypophosphatemia-induced weakness of the respiratory muscles.

In dogs, hypophosphatemia has been associated with ataxia, convulsions, stupor, and death. In humans, symptoms compatible with metabolic encephalopathy (i.e., irritability, paresthesias, confusion, seizures, coma) have been observed. The neurologic abnormalities are reflected in electroencephalographic, electromyographic, and nerve conduction studies. The cause of neurologic dysfunction may be tissue hypoxia and ATP depletion subsequent to a decline in erythrocyte 2,3-DPG.

Severe hypophosphatemia may impair myocardial performance by reducing the energy-generating ability of the left ventricle. Hypophosphatemia (0.9 mg/dl) in phosphorus-deprived dogs caused decreased myocardial stroke volume independent of the Frank-Starling effect. Dietary phosphorus repletion resulted in rapid resolution of cardiac abnormalities. The myocardial consequences of severe hypophosphatemia are thought to result from depleted intracellular ATP stores and/or impaired calcium metabolism. Reversible congestive cardiomyopathy occurred in three humans with severe hypophosphatemia. The cardiomyopathy resolved when plasma phosphorus concentrations returned to normal.

Hypercalciuria is often present in hypophosphatemic patients and is augmented by increased calcium mobilization from bone, enhanced gastrointestinal absorption of calcium, and inhibition of renal tubular calcium reabsorption. All of these effects occur independently of PTH activity and may be due to direct effects of phosphorus on calcium transport processes. Other signs of acute hypophosphatemia include intestinal ileus, anorexia, and vomiting. In humans, acute liver failure has been attributed to hypophosphatemia. Phosphorus depletion may also change cellular energy potentials and may lead to insulin resistance by altering membrane proteins.

**TREATMENT**

Most cases of hypophosphatemia do not require specific treatment. Successfully managing an underlying condition, normalizing a patient’s acid–base status, and resuming a normal diet usually return plasma phosphorus levels to normal. Most foods are abundant in phosphorus. Skim or low-fat milk is an excellent source of elemental phosphorus, but many veterinary patients do not tolerate milk. Oral phosphorus supplements are available in sodium, potassium, and calcium phosphate salts. Oral supplementation is safer and thus preferable to parenteral supplementation, but dosing in veterinary medicine is empiric. For adult dogs, the phosphorus requirement is estimated to be 1.4 to 4.6 g/1,000 kcal of metabolizable energy; for adult cats, the estimated requirement is 1.25 g/1,000 kcal of metabolizable energy, according to the Association of American Feed Control Officials (AAFCO) Nutrient Profiles. Oral

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**If parenteral phosphorus replacement is attempted, plasma calcium and phosphorus levels must be carefully monitored.**
phosphorus supplements may cause diarrhea and should be given in multiple small doses.

Severe hypophosphatemia is an indication for intravenous rather than oral phosphorus supplementation. Intravenous therapy may also be required when oral intake is precluded by anorexia, vomiting, altered consciousness, or inability to absorb phosphorus from the intestine. Parenteral supplementation is available in the form of potassium phosphate and sodium phosphate. Because many severely hypophosphatemic patients are also hypokalemic, potassium phosphate solution is especially attractive as a therapeutic agent. Potassium phosphate solution contains 4.4 mEq/ml of potassium and 3 mmol/ml of phosphate. Sodium phosphate solution contains 4 mEq/ml of sodium and 3 mmol/ml of phosphate. Potassium phosphate or sodium phosphate solutions are hypertonic and require dilution before use. They should be administered in calcium-free fluids, such as 0.9% saline or 5% dextrose solutions, to prevent precipitation of insoluble calcium phosphate salts. Lactated Ringer’s solution has also been used with potassium phosphate but contains 3 mEq/L of calcium; thus fluids without calcium are preferable, when available.

There is marked variation in the therapeutic phosphorus requirement between patients. An individual patient’s phosphorus requirement depends on the underlying cause, duration, and pathogenesis of hypophosphatemia, and plasma phosphorus concentration is an unreliable indicator of total body stores. Careful monitoring of clinical efficacy and plasma phosphorus concentrations can aid in making dosage adjustments. The recommended dose is 0.01 to 0.03 mmol/kg/hr of intravenous phosphate infusion for 6 hours until the plasma phosphorus level is more than 2 mg/dl. One report recommends 0.01 to 0.06 mmol/kg/hr, with the higher dose reserved for severely affected cats with refeeding syndrome. The plasma phosphorus concentration should be monitored every 3 to 6 hours. The dosage should subsequently be adjusted according to the results of plasma phosphorus determinations. In most cases, two to four treatments of potassium phosphate at 6-hour intervals were necessary to increase the phosphorus concentration to more than 2.5 mg/dl. The plasma phosphorus concentration should be monitored continually to assess the efficacy of therapy and prevent hypophosphatemia, even after return to normophosphatemia.

Parenteral treatment of hypophosphatemia associated with diabetes mellitus in humans has been controversial. Some authors recommend treating all patients with diabetic ketoacidosis by routinely adding phosphorus to intravenous solutions because phosphorus depletion can occur in these patients in the absence of hypophosphatemia. However, routine administration of phosphorus did not reduce morbidity or speed the correction of electrolyte disturbances in controlled studies. Supplementation has been described based on the serum potassium concentration without specific regard to plasma phosphorus levels by administering the potassium requirement either as potassium phosphate alone or half as potassium phosphate and half as potassium chloride. This is no longer recommended because the potassium deficit greatly exceeds the phosphorus deficit. However, monitoring plasma phosphorus concentrations is critical during the first 12 to 24 hours after initiating insulin and fluid therapy.
The biggest concern about phosphorus therapy is that it may cause hyperphosphatemia, which in turn might lead to hypocalcemia as well as deposition of calcium phosphate in soft tissue. Phosphorus infusion lowers ionized calcium if the calcium × phosphorus product is greater than 58. In one human case report, hypocalcemia was complicated by tetany. Acute renal failure and hypotension have also been described as complications of intravenous phosphorus therapy in human medicine. Four cats were described as having developed hypocalcemia following phosphorus supplementation, but only one cat developed severe hyperphosphatemia and clinical signs of hypocalcemia while receiving potassium phosphate supplementation. If phosphorus replacement is attempted, plasma calcium and phosphorus levels must be monitored carefully. Hyperkalemia and hypernatremia may occur with the use of potassium phosphate and sodium phosphate, respectively, so potassium and sodium levels should be monitored in conjunction with phosphorus and calcium levels. Animals that are hypercalcemic, oliguric, or have evident tissue necrosis should not receive intravenous phosphorus because metastatic calcification can occur. Prevention of hypophosphatemia is the best therapy. Widespread use of phosphorus–binding antacids in conjunction with phosphorus–restricted diets in patients with chronic renal failure puts these patients at risk for phosphorus depletion, so these treatments should be used only if indicated and carefully monitored.

**RECOMMENDATIONS**

In light of the risks associated with administration and the minimal benefits in humans, the current recommendation is not to give phosphorus supplementation routinely to patients being treated for diabetic ketoacidosis. In veterinary medicine, routine administration of intravenous phosphate to patients with diabetic ketoacidosis is discouraged. It is recommended to monitor the plasma phosphate concentration every 6 to 12 hours in patients likely to become hypophosphatemic, including ketoacidotic animals with a plasma phosphorus level of less than 3.5 mg/dl before treatment and all cats receiving enteral alimentation. Treatment of documented hypophosphatemia, especially in cats, is warranted because of the apparent increased susceptibility of cats to hypophosphatemia–induced hemolysis. In cases of mild to moderate hypophosphatemia (i.e., 1.5 to 3 mg/dl), oral phosphorus supplementation should be given. Patients with severe hypophosphatemia (i.e., <1.5 mg/dl) should receive parenteral phosphate at the recommended dosage of 0.01 to 0.03 mmol/kg/hr for 6 hours, if there are no contraindications (Figure 2). The plasma phosphate concentration, in conjunction with plasma calcium, potassium, and sodium concentrations, should be evaluated every 6 hours and the dose adjusted as necessary.

**REFERENCES**


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1. Phosphate is the major ______________ in the body.
   a. extracellular anion
   b. extracellular cation
   c. intracellular anion
   d. intracellular cation

2. The plasma phosphorus concentration is mainly regulated by the
   a. liver
   b. kidneys
   c. parathyroid gland
   d. intestines

3. Which statement regarding phosphorus levels is false?
   a. Phosphorus levels should not be measured in blood samples collected in EDTA tubes.
   b. In puppies, higher phosphorus levels may be found because of an increased growth hormone level.
   c. Phosphorus levels can be influenced by recent digestion of a carbohydrate-rich meal.
   d. Hyperbilirubinemia, hyperlipemia, and hemolysis can falsely decrease the phosphorus concentration.

4. ______________ is not a reason for translocation of phosphorus from the extracellular fluid to intracellular locations.
   a. Hypercalcaemia
   b. Intravenous infusion of glucose
   c. Refeeding syndrome
   d. Diabetic ketoacidosis

5. ______________ is not a reason for phosphorus depletion in animals with diabetic ketoacidosis.
   a. Insulin deficiency
   b. Vomiting
   c. Urinary phosphorus loss
   d. Anorexia
6. _____________ is not a consequence of hypophosphatemia.
   a. Hemolysis
   b. Impaired leukocyte chemotaxis.
   c. Myopathy
   d. Hypersalivation

7. Which statement regarding hypophosphatemia is false?
   a. Most cases of hypophosphatemia do not require specific treatment.
   b. A phosphorus level less than 1 mg/dl is an indication for intravenous phosphorus supplementation.
   c. Parenteral supplementation is safer than oral supplementation.
   d. Vomiting or altered consciousness is an indication for intravenous phosphorus supplementation.

8. Which statement regarding intravenous phosphorus supplementation is correct?
   a. Potassium phosphate solutions do not require dilution before use.
   b. Solutions of 0.9% saline or 5% dextrose do not contain calcium.
   c. Because many hypophosphatemic patients are also hyponatremic, sodium phosphate solutions should be used.
   d. When phosphate solutions are diluted, lactated Ringer’s solution is the solution of choice.

9. How should patients treated with intravenous potassium phosphorus solutions be monitored?
   a. Phosphorus levels should be measured every 12 hours in combination with sodium and potassium levels.
   b. Potassium levels should be measured every 3 to 6 hours in combination with calcium and magnesium levels.
   c. Phosphorus levels should be measured every 3 to 6 hours in combination with calcium and potassium levels.
   d. Potassium levels should be measured every 12 hours in combination with calcium and magnesium levels.

10. What is the biggest concern regarding phosphorus supplementation therapy?
    a. hypocalcemia
    b. acidosis
    c. alkalosis
    d. hyperkalemia

Test answers now available at CompendiumVet.com