

# Diabetic Ketoacidosis

**Brandy Tabor, CVT, VTS (ECC)**

Animal Emergency and Specialty Center  
Parker, Colorado

**D**iabetic ketoacidosis (DKA) is a serious metabolic disorder that can occur in patients with diabetes mellitus (DM).<sup>1,2</sup> Veterinary technicians play an integral role in managing and treating patients with this life-threatening condition. In addition to recognizing the clinical signs of this disorder and evaluating the patient's response to therapy, technicians should understand how this disorder occurs. Technicians must also educate owners about the long-term care of diabetic pets.

## Pathophysiology

DM is caused by a relative or absolute lack of insulin production by the pancreatic  $\beta$  cells or by inactivity or loss of insulin receptors, which are usually found on membranes of skeletal muscle, fat, and liver cells.<sup>1,3</sup> In dogs and cats, DM is classified as either type I (insulin dependent; the body is unable to produce sufficient insulin) or type II (non-insulin dependent; the body produces insulin, but the body's tissues are resistant to insulin).<sup>4</sup> Most dogs that develop DM have insulin deficiency, while cats that develop DM tend to have insulin resistance.<sup>5</sup> DKA occurs when the body cannot use glucose for energy because of a lack of, or resistance to, insulin. When this happens, the body uses alternative energy sources, resulting in ketone production and subsequent acidosis.<sup>1</sup>

Insulin has many functions, including the enhancement of glucose uptake by the cells for energy.<sup>1</sup> Without insulin, cells cannot use glucose, causing them to starve.<sup>2</sup> The unused glucose remains in the circulation, resulting in hyperglycemia. To provide cells with an alternative energy source, the body breaks down adipocytes, releasing free fatty acids (FFAs) into the bloodstream. The liver subsequently converts FFAs to triglycerides and ketone bodies. These ketone bodies (i.e., acetone, acetoacetic acid,  $\beta$ -hydroxybutyric acid) can be used as energy by the tissues when there is a lack of glucose or nutritional intake.<sup>1,2</sup> The breakdown of fat, combined with the body's inability to use glucose, causes many pets with diabetes to present with weight loss,

despite having a ravenous or normal appetite.

Insulin is also important for moving electrolytes (e.g., potassium, magnesium, phosphorus) and amino acids into cells.<sup>5</sup> An electrolyte imbalance can lead to complications such as cardiac arrhythmia, muscle weakness, and hemolysis.

If diabetes is undiagnosed or uncontrolled, a series of metabolic events can lead to DKA. In patients with DM, predisposing disease processes (e.g., infection, pancreatitis, heart disease), trauma, or exposure to a stressful situation (e.g., being boarded or relocated) can also lead to this condition. Normally, insulin suppresses hepatic glucose production during a state of hyperglycemia. In the absence of insulin, the liver continues to release glucose, exacerbating hyperglycemia. At the same time, cellular demand for glucose stimulates the release of the hormone glucagon from the pancreas. This hormone, together with the hormones cortisol, epinephrine, and growth hormone, triggers the liver to produce even more glucose and ketone bodies.<sup>1</sup>

When the production of ketone bodies exceeds the body's ability to use them, they build up in the circulation, resulting in ketosis.<sup>2</sup> Although acetone is chemically neutral, acetoacetic and  $\beta$ -hydroxybutyric acids are acidic. Therefore, when these acids accumulate in the blood, they cause the blood pH to decrease, resulting in metabolic acidosis.<sup>4</sup>

At the same time, the rising glucose level begins to cause osmotic diuresis.<sup>6</sup> The renal tubular threshold for total reabsorption of ketone bodies and glucose is quickly exceeded, causing these substances to spill into the urine for excretion.<sup>2</sup> The negative charge of the ketones draws positively charged electrolyte ions, such as sodium and potassium, into the urine to maintain a neutral state.<sup>2</sup> Lack of fluid intake, combined with vomiting, diarrhea, and increased urine production, which commonly occur in patients with DKA, can cause an increased loss of electrolytes and fluid through the urine. This fluid loss can lead to dehydration and decreased tissue perfusion while reducing the glomeru-

## Glossary

**Anuria**—Lack of urine production

**Extravasation**—Leakage outside the vein

**Glomerular filtration rate**—Kidney function test that estimates the filtering capacity of the kidneys

**Hemoconcentration**—Increased concentration of red blood cells in the blood

**Hypotonic**—Having a lower osmotic pressure than another solution

**Ketonemia**—Excessive amount of ketones in the blood

**Ketosis**—Accumulation of large amounts of ketone bodies in the blood and tissues

**Oliguria**—Reduced daily urine production

**Polydipsia**—Excessive thirst manifested by excessive water intake

**Polyphagia**—Excessive food ingestion

**Polyuria**—Formation and excretion of large volumes of urine

lar filtration rate (GFR), which can cause renal failure.<sup>2</sup> As the GFR decreases, so does the patient's ability to excrete glucose and ketones, both of which accumulate in the vascular space.<sup>2</sup>

## Clinical Signs

Patients that present with DKA may have been previously diagnosed with and treated for DM, or clients may have observed clinical signs of DM in their pet. Signs of DM include weight loss, polydipsia, polyuria, and polyphagia. Technicians should obtain a thorough patient history in case owners observed these signs but thought that they were unimportant.<sup>1,2</sup> Questions asked of the owners should be phrased carefully, and leading questions should be avoided.

Patients that develop DKA are seriously ill. They may experience vomiting, anorexia, or lethargy, which are often caused by dehydration, electrolyte abnormalities, and acidemia.<sup>1</sup> When a diabetic patient presents with vomiting and anorexia, it is likely that ketonemia, ketonuria, and metabolic acidosis have already developed; therefore, severe illness may occur within a week or less.<sup>2</sup>

On physical examination, the pet may present with a thin body condition, muscle wasting, dehydration, depression, an unkempt haircoat, or hypothermia.<sup>1</sup> Other signs of DM, such as cataracts in dogs or plantigrade stance due to diabetic neuropathy in cats, may also be noted.<sup>2</sup> Plantigrade stance is characterized by walking with flattened

hocks rather than on the toes, as dogs and cats typically do. Clinicians may detect ketone breath,<sup>2</sup> which smells like acetone or nail polish remover. Patients with severe metabolic acidosis may also exhibit slow, deep Kussmaul breathing patterns.<sup>2</sup>

## Diagnosis

The four classic laboratory findings consistent with DKA are hyperglycemia, glucosuria, ketonuria, and metabolic acidosis.<sup>1</sup> Therefore, if DKA is suspected, a complete blood count, full chemistry profile, urinalysis, urine culture, and blood gas analysis should be conducted.<sup>1,2</sup>

If possible, urine should be collected via cystocentesis. Urine reagent test strips that measure glucose and ketones should detect glucosuria and ketonuria.<sup>1</sup> Because azotemia (elevated blood urea nitrogen [BUN]) is a common finding in patients with DKA, it is important to assess the urine specific gravity and the serum BUN level before therapy is initiated. A dehydrated patient with azotemia and a urine specific gravity of >1.030 is most likely exhibiting prerenal azotemia, with normal kidney function. Prerenal azotemia occurs when filtration in the kidneys decreases secondary to dehydration. The associated high urine specific gravity indicates that the kidneys can reabsorb water and concentrate the urine. When the kidneys reabsorb water, they also reabsorb BUN and creatinine, causing prerenal azotemia. However, patients with a urine specific gravity of <1.020 may have primary renal failure.<sup>2</sup> Evaluation of renal failure is important not only for fluid therapy but also for monitoring. For patients suspected of having oliguric or anuric renal failure, monitoring of urine output is crucial.<sup>2</sup> A urine culture should also be conducted to detect a urinary tract infection, which can complicate treatment. The complete blood count most commonly reveals an elevation in packed cell volume (PCV) and the total protein level, due to hemoconcentration caused by dehydration.<sup>1,2</sup>

Not surprisingly, blood chemistry findings show an increase in blood glucose level. Although the average blood glucose level for a patient with DKA is 500 mg/dL, readings may range from 200 to >1000 mg/dL. Other abnormal findings may include elevated liver values and increased levels of BUN, creatinine, cholesterol, and triglycerides.<sup>1</sup> Decreased values for many electrolytes, including sodium, potassium, chloride, phosphorus, and magnesium, are also common.<sup>1</sup> Electrolyte abnormalities may lead to cardiac arrhythmias that can be detected on a lead II electrocardiogram strip. For instance, the most common indication of hypokalemia on an electrocardiogram is a prolongation of the Q-T interval, with a lowering of the ST segment and a decrease in the T wave. Premature atrial and ventricular contractions may occur. Hyperkalemia, on the other hand, can result in

spiking of the T wave, widening of the QRS interval, and a decrease in the P wave. Patients with hyperkalemia may exhibit bradycardia and ventricular arrhythmias.<sup>2</sup>

The anion gap, which is the difference between measured anions and cations, can be calculated from the chemistry panel and electrolyte results (**BOX 1**). An elevated anion gap can indicate ketoacidosis. However, there are other causes of an increased anion gap, including ethylene glycol toxicosis and renal failure.<sup>2</sup> An elevated anion gap should be evaluated in conjunction with blood work results and the patient's history to determine the cause of the increase. The anion gap in healthy dogs and cats ranges from 12 to 15 mEq/L; most patients with ketoacidosis have an anion gap ranging from 20 to 35 mEq/L.<sup>2</sup>

Arterial blood gas analysis is necessary to document the degree of metabolic acidosis.<sup>1,2</sup> The body's buffering system may not be able to maintain a normal pH as ketones build up in the circulation.<sup>2</sup> A pH <7.0 can correlate with a grave prognosis.<sup>2</sup> If the clinic has limited resources and an arterial blood gas analysis cannot be conducted, treatment can be initiated based on the presence of hyperglycemia, glucosuria, and ketonuria.<sup>2</sup>

Treatment of underlying disease processes is essential to successfully resolve DKA. For this reason, it is also beneficial to obtain an electrocardiogram, thoracic and abdominal radiographs, and an abdominal ultrasonogram. These diagnostics may reveal heart disease, pancreatitis, or liver disease. If electrocardiography is not available, the heart can be evaluated by thoracic radiography and auscultation. Similarly, if ultrasonography is not available, the blood chemistry findings can reveal problems with the liver, and pancreatitis can be diagnosed in-house or through an outside laboratory.

## Treatment

In patients with DKA, treatment should include the following steps, in order of importance:

- Fluid therapy to restore fluid volume and enhance perfusion
- Insulin therapy to lower glucose and ketone concentrations while reversing metabolic acidosis
- Correction of electrolyte abnormalities

The goal of treatment is for all parameters to return to normal over 36 to 48 hours.<sup>2</sup>

## Fluid Therapy

Rapid initiation of fluid therapy is a cornerstone of DKA treatment. Placement of a central venous catheter eliminates the need for frequent venipuncture to monitor blood

### BOX 1

#### Calculating the Anion Gap<sup>a</sup>

$$\text{Anion gap} = [\text{Sodium (mEq/L)} + \text{Potassium (mEq/L)}] - [\text{Chloride (mEq/L)} + \text{Bicarbonate ion (mEq/L)}]$$

<sup>a</sup>Rose DB, Post TW. Metabolic acidosis. In: *Clinical Physiology of Acid-Base and Electrolyte Disorders*. 5th ed. New York: McGraw Hill; 2001:583-585.

glucose, electrolytes, and venous blood gases.<sup>6</sup> It also allows central venous pressure to be measured to reduce the risk of overhydration.

Fluid therapy is geared toward correcting cellular dehydration, poor tissue perfusion, and electrolyte abnormalities.<sup>6</sup> Fluids can also decrease the blood glucose level, even without insulin, through dilution and by increasing the GFR, which allows increased excretion of glucose through the urine.<sup>6</sup> However, fluid therapy alone cannot reduce the ketone concentration.<sup>2</sup>

The type of fluid chosen should be based on the patient's electrolyte status.<sup>2</sup> Because most patients with DKA have a deficit in total body sodium,<sup>2,6</sup> a common fluid choice is 0.9% sodium chloride. Other fluid options include Normosol-R (Abbott Laboratories), Plasma-Lyte 148 (Baxter), and lactated Ringer's solution.<sup>2,6</sup> Hypotonic fluids (e.g., 0.45% sodium chloride) usually do not provide enough sodium, and rapid infusion can result in cerebral edema, which can lead to coma.<sup>2,6</sup>

The volume and rate of fluids infused during the first 24 hours should be designed to correct dehydration while supplying maintenance needs and replacing ongoing fluid losses from vomiting or diarrhea.<sup>2,6</sup> There are several methods of determining dehydration (e.g., assessing skin turgor, mucous membrane moisture, and urine specific gravity) while monitoring the PCV and the total protein level. Dogs and cats with DKA are typically 6% to 12% dehydrated.<sup>2</sup> The amount of fluid to be infused can be calculated using the formula in **BOX 2**. The initial fluid rate is determined by the clinician based on many factors, including the presence of underlying diseases (e.g., congestive heart failure). If the patient is in shock during presentation, the clinician may choose to administer a crystalloid fluid bolus.<sup>7</sup>

Because of the high fluid rate required to correct dehydration, replace losses, and maintain normal requirements, the patient should be monitored closely for signs of fluid overload (e.g., nasal discharge, coughing, peripheral edema, ascites, increased respiratory rate and effort). After dehydration has been corrected, the fluid rate can be decreased to a maintenance rate that includes replacement of ongoing losses.<sup>2</sup>

## BOX 2

**Estimating Replacement Fluids****To estimate fluids to be infused in the first 24 hours<sup>6</sup>:**

Maintenance fluid requirements = 55–65 mL/kg/d  
(Includes insensible losses of 20 mL/kg/d and urinary losses of 35–45 mL/kg/d)

plus

Volume to correct dehydration:

Body weight (kg) × % Dehydration (0.00) × 1000 mL/kg  
= Fluid deficit (mL)

plus

Estimated losses from vomiting and/or diarrhea

The fluid volume is usually replaced over 24 hours.

**To calculate dextrose supplementation<sup>6</sup>:**

(Desired % of dextrose solution ÷ Strength [%] of available dextrose solution) × Amount of fluid to which dextrose will be added = Amount of available dextrose solution to add to fluid

*Example*—Making a 5% dextrose solution in a 750-mL fluid bag when a 50% dextrose solution is available:

$$(5\% \div 50\%) \times 750 = (0.05 \div 0.5) \times 750 = 75 \text{ mL}$$

Therefore, 75 mL of 50% dextrose solution must be added to a 750-mL fluid bag to make a 5% dextrose solution.

*Example*—Making a 2.5% dextrose solution in a 1000-mL fluid bag when a 50% dextrose solution is available:

$$(2.5\% \div 50\%) \times 1000 = (0.025 \div 0.5) \times 1000 = 50 \text{ mL}$$

Therefore, 50 mL of a 50% dextrose solution must be added to a 1000-mL fluid bag to make a 2.5% dextrose solution.

<sup>6</sup>This is the author's method.

**Insulin Therapy**

Insulin therapy is crucial in treating patients with DKA. Insulin lowers the blood glucose level as well as reduces the ketone concentration, which can improve metabolic acidosis. Insulin also decreases the serum potassium level by promoting potassium entry into the liver and skeletal muscle.<sup>8</sup> The initiation of insulin therapy is often delayed to allow fluid therapy to correct dehydration and normalize electrolyte levels.<sup>2</sup> The clinician should decide when it is best to start insulin therapy for an individual patient.

Several types of insulin are available: short acting, intermediate acting, and long acting. Short-acting insulin includes regular human insulin. Depending on how short-acting insulin is administered, it can have an onset of action of 10 to 30 minutes, and the effects can last 3 to 10 hours. Only regular human insulin should be used for the initial treatment of DKA.<sup>6</sup>

Intermediate-acting insulin, such as 3-nitropropionic acid (NPH), contains zinc and proteins that are absorbed more slowly; therefore, this insulin has a slower onset of action, and the effects last longer than those of short-acting insulin.

Long-acting insulin, such as glargine, microprecipitates in subcutaneous tissue and is released very slowly. These types of insulins are not appropriate for treating DKA but can be sent home with clients for long-term management of diabetes.<sup>9</sup>

Another insulin that is used for managing diabetes is Vetsulin (Intervet/Schering-Plough Animal Health), a porcine insulin in a zinc suspension. Vetsulin is the first FDA-approved insulin for use in dogs and cats. In 2009, Vetsulin was scrutinized by the FDA because of concerns that the onset of action may be delayed and the duration of action prolonged. The FDA recommended that veterinarians switch their patients to other insulin products and has allowed Vetsulin to remain in distribution to allow time for this switch. The manufacturer has a limited supply of Vetsulin and has permission from the FDA to distribute the product through the "Vetsulin Critical Need Program" to patients that could not be switched to a different insulin product.<sup>10</sup>

Three protocols for insulin therapy exist: intramuscular injections hourly, intramuscular or subcutaneous injections every 4 to 6 hours, and a constant-rate intravenous infusion.<sup>2,6</sup> Each protocol has advantages and disadvantages; therefore, the clinician should choose the protocol based on experience and the patient's condition.<sup>6</sup> A constant-rate infusion is typically preferred.<sup>2,6</sup> If the patient is dehydrated or has poor perfusion, intermittent intramuscular or subcutaneous insulin may be less effective. Insulin therapy should be administered in a manner that lowers the blood glucose concentration gradually (50 mg/dL/h) to 200 to 250 mg/dL over 6 to 10 hours.<sup>2</sup> This slow, steady decrease prevents the osmolarity from changing too rapidly.<sup>2</sup> Once the blood glucose level has reached 200 to 250 mg/dL, 2.5% to 5% dextrose should be supplemented (**BOX 2**) in the fluids to avoid hypoglycemia while the ketosis resolves.<sup>2,6</sup> The reversal of ketogenesis and the subsequent resolution of ketosis can take as long as 48 to 72 hours.<sup>2,6</sup> Once the patient is able to eat and drink without vomiting, treatment with a longer-duration insulin may be used.<sup>6</sup>

**Electrolyte Supplementation**

One of the most common electrolyte abnormalities in patients with DKA is hypokalemia. Clinical signs of this imbalance include muscle weakness and cardiac arrhythmias. In cats, cervical ventroflexion can occur. Even if blood work reveals that the potassium concentration is normal, it should be anticipated that the concentration will decrease rapidly as dehydration is corrected and potassium moves

from the extracellular space into the intracellular space in response to insulin therapy and correction of acidemia.<sup>2</sup> Therefore, potassium chloride should be supplemented in the fluids.<sup>2,6</sup> Potassium supplementation can be based on the potassium replacement scale (**TABLE 1**) as long as the potassium supplementation does not exceed 0.5 mEq/kg/h.<sup>2,6</sup> The serum potassium level should be reevaluated every 6 to 8 hours to allow for adjustments and to avoid oversupplementation.<sup>2,6</sup>

After therapy is initiated, the patient's serum phosphorus level may decline for the same reason that the potassium level decreases.<sup>2</sup> Hypophosphatemia typically affects the hematologic and neuromuscular systems, resulting in hemolytic anemia, weakness, ataxia, and seizure activity if the phosphorus level decreases to 1.5 mg/dL.<sup>2</sup> Potassium phosphate can be added to fluids and given as a constant-rate infusion of 0.01 to 0.03 mmol/kg/h.<sup>2,6</sup> The serum phosphorus level should be reevaluated every 6 to 8 hours and supplementation adjusted as necessary.<sup>2,6</sup> Hyperphosphatemia can cause mineralization of tissue, iatrogenic hypocalcemia, and hypotension<sup>2,6</sup>; therefore, oversupplementation should be avoided.<sup>2,6</sup> If potassium phosphate is added to the fluid regimen, the amount of potassium chloride should be adjusted to account for the additional potassium.

Hypomagnesemia, which is a common finding in patients with DKA, can be life threatening and can cause refractory hypokalemia and cardiac arrhythmias. If hypomagnesemia is mild, it can usually be resolved by administering a fluid that contains magnesium (e.g., Plasma-Lyte 148).<sup>6</sup> However, if it is more severe (<1.2 mg/dL), it should be treated more aggressively with an infusion of magnesium sulfate or magnesium chloride.<sup>2,6</sup>

### Monitoring the Response to Therapy

Careful patient monitoring, especially during the first 24 to 48 hours, is vital for successful treatment outcomes. Patients must be kept warm and dry, and recumbent pets should be turned every few hours to prevent the formation of decubital ulcers. Because hematologic and physical parameters may change quickly, it is important for technicians to monitor the patient for any changes and to alert the clinician as soon as possible if any occur.

The blood glucose level should be checked every 1 to 2 hours until it is <250 mg/dL. As soon as this level is reached, dextrose should be added to the fluids to create a 2.5% to 5% dextrose solution to prevent hypoglycemia while insulin therapy continues.<sup>2,6</sup>

Continuous glucose monitoring systems (CGMSs) are an alternative to periodic blood glucose checks. CGMSs can eliminate the need for blood draws every 1 to 2 hours.

**TABLE 1 Potassium Supplementation<sup>6,a</sup>**

Current Serum Potassium Level (mEq/L)	Potassium Supplementation (mEq/L) for Fluid Therapy
3.5–5.0	20
3.0–3.4	30
2.5–2.9	40
2.0–2.4	60
<2.0	80

<sup>a</sup>Therapy must not exceed 0.5 mEq/kg/h.

Several monitoring systems are available and work in the same way. A sensor is placed subcutaneously to measure the glucose concentration in the interstitial fluid. The system has to be calibrated every 8 to 12 hours, which requires a peripheral blood draw. While this method decreases the need for blood draws, it has several pitfalls:

- Because no CGMSs are made specifically for dogs and cats, the reliability and accuracy of these systems are questionable for monitoring these species
- The performance of CGMS sensors can vary because of hemorrhage, thrombosis, or inflammation at the sensor site
- The patient's hydration status and perfusion must be considered

With the availability of glucometers made specifically for dogs and cats and the importance of accurate blood glucose measurements, clinicians must weigh the pros and cons of using a CGMS.<sup>11</sup>

Serum electrolyte levels should be measured every 6 to 8 hours.<sup>2</sup> When the serum sodium level reaches 140 to 155 mEq/L, the IV fluid should be changed from 0.9% sodium chloride to Ringer's solution or lactated Ringer's solution, both of which have a lower sodium content.<sup>2</sup> Cardiac changes secondary to electrolyte abnormalities can be further monitored with lead II electrocardiogram strips.

As insulin lowers the ketone concentration, the anion gap should return to normal, and ketonuria will resolve. Total venous carbon dioxide or arterial blood gases should be monitored every 6 to 8 hours.

It is important to evaluate hydration status every 2 to 4 hours.<sup>2</sup> Respiratory and cardiac auscultation should be normal, and mucous membranes should be pink and moist with a normal capillary refill time. Central venous pressure should remain below 10 cm H<sub>2</sub>O.<sup>2</sup> Doppler blood pressure readings should remain within normal limits. A fasted pet

should lose about 0.5% to 1.0% of body weight per day.<sup>2</sup> Abnormal weight gain may indicate overhydration. Fluids should be adjusted as needed.

Urine output should be monitored every 2 to 4 hours. Technicians should expect a minimum of 1 to 2 mL of urine/kg of body weight per hour.<sup>2</sup> Lack of urine output within a few hours of initiating fluid therapy is a medical emergency, and the clinician should be notified immediately. Urine should also be checked for glucose and ketones.

When patients are hypophosphatemic, the PCV should be checked daily for signs of hemolytic anemia.

### Complications

Complications of therapy for DKA include hypoglycemia, cerebral edema, hemolytic anemia, hypernatremia, hyperchloremia, hypokalemia, and hypophosphatemia.<sup>2</sup> The best way to avoid these complications is with frequent reassessment of the patient's electrolyte and blood glucose levels during the first 24 hours of treatment and then as the clinician deems necessary.<sup>2</sup>

### Prognosis

The prognosis for patients with DKA depends on response to treatment, the severity of the illness, and any underlying diseases that may exist.<sup>6</sup> The high mortality rate (30% to 40%) of patients with DKA is most often associated with the underlying disease that caused the development of DKA.<sup>2</sup>

### The Role of the Technician

Veterinary technicians play a crucial role in treating patients with DKA. The mortality rate increases for patients that are not intensely monitored.<sup>2</sup> Technicians are responsible for alerting the clinician immediately of blood work results and monitoring the patient for signs of neurologic problems, aspiration pneumonia (if vomiting occurs), poor urine production, anemia, overhydration (including nasal discharge), weight gain above expected, and changes in lung sounds, pulse quality, blood pressure, and mucous membrane color. Dogs should be walked frequently because existing polyuria may not resolve immediately. Patients are likely to have diarrhea. Frequent baths can prevent scalding,

keep patients comfortable, and decrease stress in patients that are meticulous about their hygiene. The intravenous catheter should be monitored closely; if there are signs of inflammation or irritation at the injection site or pain during injection, the catheter should be removed and a new one placed in a different vein. It is also important to replace these catheters as needed because extravasation of any medication (including dextrose) can cause pain, redness, and swelling. Some medications can also cause sloughing of affected tissues.

Although DKA is a serious disease, it can be treated successfully. Technicians can play an important role in client education and in helping recognize and treat this disease. Owners may be overwhelmed by the prospect of giving insulin injections to their pets and be concerned with their pets' quality of life. Technicians have the opportunity to help pet owners understand diabetes and to explain that affected pets can have a long, healthy life.

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## Article #1 FREE CE Test

The article you have read qualifies for 1.0 credit hour. To receive credit from Alfred State College, choose the best answer to each of the following questions. Take the test online at **Vetlearn.com**.

- 1. The anion gap is the difference between**
  - a. measured anions and unmeasured cations.
  - b. measured anions and cations.
  - c. unmeasured anions and measured cations.
  - d. none of the above
- 2. In patients with DKA, the body's buffering system may not be able to maintain a normal pH as \_\_\_\_\_ build up in the circulation.**
  - a. FFAs
  - b.  $\beta$  cells
  - c. ketones
  - d. none of the above
- 3. Which insulin should be used for the initial treatment of DKA?**
  - a. NPH
  - b. regular human insulin
  - c. Vetsulin
  - d. glargine
- 4. Which hormone(s) contribute(s) to DKA?**
  - a. epinephrine
  - b. cortisol
  - c. growth hormone
  - d. all of the above
- 5. Most patients with DKA have a deficit in total body sodium; therefore, \_\_\_\_\_ is a common fluid choice.**
  - a. lactated Ringer's solution
  - b. 0.45% sodium chloride
  - c. 0.9% sodium chloride
  - d. Normosol-R
- 6. In patients with insulin-dependent DM, the body**
  - a. can produce insulin but is resistant to it.
  - b. cannot produce sufficient insulin.
  - c. overproduces insulin.
  - d. none of the above
- 7. In patients with non-insulin-dependent DM, the body**
  - a. can produce insulin but is resistant to it.
  - b. overproduces insulin.
  - c. cannot produce sufficient insulin.
  - d. none of the above
- 8. The anion gap in healthy cats and dogs ranges from \_\_\_\_\_ mEq/L.**
  - a. 12 to 15
  - b. 15 to 20
  - c. 15 to 25
  - d. 20 to 35
- 9. Potassium supplementation should not exceed**
  - a. 0.5 mEq/lb/h.
  - b. 0.5 mEq/kg/h.
  - c. 0.5 mEq/lb/d.
  - d. 0.5 mEq/kg/d.
- 10. In patients with DKA, the goal of insulin therapy is to lower the blood glucose level by 50 mg/dL/h to a level of 200 to 250 mg/dL in \_\_\_\_\_ hours.**
  - a. 4 to 8
  - b. 6 to 10
  - c. 10 to 14
  - d. 12 to 15