Timely Topics in Nutrition

Thiamine deficiency in dogs and cats

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In past years, nutritional deficiencies were considered to be an important problem in dogs and cats. Currently, most pet owners in many countries, such as the United States and Canada, feed nutritionally balanced commercial pet foods. As a result, nutritional deficiencies have become uncommon because reputable pet food manufacturers regularly test their products to ensure that they contain adequate amounts of all nutrients. Anecdotally, most reported deficiencies currently arise from animals eating incomplete or unbalanced homemade, vegetarian, or raw meat diets. Therefore, veterinarians may not consider deficiencies as differential diagnoses in animals eating traditional commercial diets. However, thiamine (vitamin B₁) deficiency is of clinical concern even today. Since 2009, there have been 5 major voluntary pet food recalls involving thiamine-deficient pet foods in the United States that ultimately involved 9 brands of cat foods and at least 23 clinically affected cats. Most of these recalls were instituted in response to a report from a consumer or veterinarian after treating a cat that had clinical signs consistent with thiamine deficiency.

In addition to the possibility of a deficiency in commercial pet foods, there are a variety of situations in which a deficiency may arise in dogs or cats with medical conditions. Clinical manifestations of a deficiency of thiamine are variable, and the disease is likely under-reported because of the wide array of clinical signs in combination with a lack of specific clinicopathologic changes detected via laboratory analysis.

Thiamine has received much attention as a vitamin deficiency that is common in ruminants, primarily as a result of rumen bacterial inactivation of the vitamin, which results in characteristic cerebrocortical necrosis and neurologic signs. Dogs and cats can also be affected by deficiency of this vitamin because of an inability to endogenously synthesize large quantities of thiamine. Therefore, both cats and dogs need to have a consistent dietary supply of thiamine. As with all B vitamins, thiamine is water soluble, stored in the body in small amounts, and subject to urinary losses. Thiamine is also particularly labile and easily destroyed by typical food-processing techniques. In fact, many early experiments on thiamine deficiency were performed by autoclaving a pet food and then feeding it. Most pet food manufacturers add additional sources of thiamine to compensate for thiamine lost through processing. However, despite best efforts, thiamine-deficient commercial pet foods sometimes are still produced. The information provided here is intended to describe the clinical syndrome of thiamine deficiency in companion animals as well as to highlight several causes of thiamine deficiency that have been described in the literature.

Physiologic Functions of Thiamine

The B vitamins are numbered in order of their isolation. In 1911, thiamine, as the name vitamin B₁ implies, was the first B vitamin isolated. Thiamine is found naturally in many plants, particularly whole grains and grain products (eg, rice and wheat germ) as well as yeast and legumes. Thiamine is also found in meat products, often concentrated in the liver, heart, and kidneys. After ingestion, thiamine is absorbed in the small intestine, primarily the jejunum and ileum. Studies in rats, humans, and dogs have revealed that the amount of thiamine in the diet dictates the amount and process of absorption because there are both active and passive carrier transport processes. When there are excessive amounts of thiamine in the diet, thiamine is absorbed from the intestinal lumen via a passive diffusive process, whereas when there is a paucity of thiamine in the diet, thiamine is actively absorbed from the intestinal tract. In contrast to cobalamin, a genetic defect of the intestinal thiamine transporter has not been identified in dogs or cats. Once absorbed, most of the thiamine is carried within RBCs, whereas the remaining thiamine is bound to plasma proteins or is free in plasma. Thiamine participates in several vital biochemical pathways within...
the body, namely the TCA cycle in the process of carbohydrate metabolism and the pentose phosphate pathway (Figure 1). It is these important roles in carbohydrate metabolism that dictate the pathological findings and clinical signs seen in animals with thiamine deficiency.

There are 4 naturally occurring forms or derivatives of thiamine in the body. Most of the thiamine in an animal is found in a phosphorylated form, TPP, whereas the nonphosphorylated forms are more common in plants. Thiamine pyrophosphate, also known as thiamine diphosphate, is the most biologically active form within the body because TPP is the form of thiamine that is a cofactor in both the TCA cycle and the pentose phosphate pathway (Figure 1). Within the TCA cycle, TPP is an integral cofactor in the conversion of pyruvate to acetyl-CoA. Without TPP, an excess amount of pyruvate and lactate is produced, which can lead to type B lactic acidosis.

Cats are more susceptible to thiamine deficiency than dogs because cats have approximately a 3-fold higher requirement for the vitamin than their canine counterparts. For example, the NRC-recommended allowance for adult cats is 1.4 mg of thiamine/1,000 kcal of metabolizable energy, whereas the NRC-recommended allowance for adult dogs is 0.56 mg of thiamine/1,000 kcal of metabolizable energy. Although AAFCO does not adjust minimum amounts of thiamine on the basis of life stage, the NRC-recommended allowance for thiamine is higher for reproduction, compared with the allowance for adult maintenance, for cats. Interestingly, although AAFCO and NRC have no specific guidelines for vitamin or other nutrient requirements for geriatric animals, older people appear to be more susceptible to thiamine deficiency than are younger individuals, irrespective of health status.

Figure 1—Schematic diagram of the role of TPP as an integral cofactor in both the TCA cycle and the pentose phosphate (PP) pathway for glucose metabolism. The important roles of TPP in these pathways dictate the clinicopathologic findings and clinical importance of thiamine deficiency. Large black arrows indicate that multiple steps were condensed. NADPH = Reduced form of nicotinamide adenine dinucleotide phosphate.
Mechanisms of Thiamine Deficiency

Systemic disease states can affect absorption, retention, and metabolism of thiamine in the body. In terms of absorption, intestinal malabsorption or other chronic enteropathy conditions can affect the uptake of thiamine from the diet, whereas medications (eg, diuretics) that increase urinary losses can potentiate the excretion of vitamin B1.14 Furthermore, the number and efficiency of thiamine transporters in the small intestines, heart, liver, and brain correlate with the overall absorption and usage of thiamine within the body. Investigators of a recent study14 found that rats with chronic kidney disease (5/6 nephrectomy scale) developed downregulation of thiamine and folate transporters in the small intestines, heart, liver, and brain. Thiamine primarily undergoes renal excretion in a nonlinear manner that is largely dependent on a combination of GFR, tubular secretion, and tubular reabsorption.14 As the GFR is reduced as a result of kidney disease, plasma concentrations of thiamine may actually increase.16 In contrast, in the physiologic normal state, medications such as diuretics or other diseases that cause an increase in GFR may also cause total body depletion of thiamine.14,17

Clinical Signs of Thiamine Deficiency

Clinical signs associated with thiamine deficiency are variable and nonspecific, and may involve several body systems2,3,9,18–22 (Table 1). Gastrointestinal signs are typically the first manifestation, but neurologic signs are often the ones most commonly associated with thiamine deficiency.3,18 In cats, spastic cervical ventroflexion may be detected that initially resembles marked hypokalemia; however, in cats with thiamine deficiency, the ventroflexion worsens and the cats will continue to look at the ground when held upside down (eg, wheelbarrow position).2,18 Ocular examination often reveals anisocoria or mydriasis with decreased to absent pupillary light reflexes and lack of menace responses but no abnormalities evident during fundic examination.2 Nystagmus or blindness may also be evident. It is important to remember that in the early stages of thiamine deficiency, clinical signs are often nonspecific and can therefore lead to a misdiagnosis until overt neurologic signs are evident.

Thiamine deficiency can result in several electrocardiographic abnormalities, which may be evident as bradycardia or tachycardia.23 In 1 study, investigators found an excitement syndrome that consisted of a brief period of tachycardia, followed by severe bradycardia and rhythm irregularity in nearly all of the cats during the end stages of thiamine deficiency. Electrocardiographic changes can include flattening or inversion of the T wave, QRS prolongation, or prolongation of the ST segment.23

Three progressive stages associated with thiamine deficiency have been described: induction, critical, and terminal.2,3 As described in a controlled study2 and a retrospective report,4 the induction stage generally develops within 1 week after animals begin eating a diet severely deficient in thiamine and is characterized by hyporexia, vomiting, or both. Typically, an animal must be thiamine deficient for slightly more than 1 month before the terminal stage is reached.3 However, once the terminal stage has started, an animal will die within a few days if the deficiency is not corrected immediately.5 Despite this, nearly all reported cases of thiamine deficiency in dogs and cats have been diagnosed in the third or terminal stage. Although these time periods were described in a study6 performed in 1944, and cats with experimentally induced thiamine deficiency were included in that study, there is much more variation in clinical signs, which makes it more challenging to arrive at the diagnosis. Typically, it can take weeks to months for the development of clinical signs, which are attributable to subchronic deficiency because most diets are not entirely devoid of thiamine. Mitigating factors include the amount of thiamine in the food, nutrient composition of the diet, whether the animal eats a consistent diet, and species and health status of the animal.

Risk Factors for Developing Thiamine Deficiency

Diet factors that have low concentrations of thiamine are a clear risk factor for the development of deficiency. However, a variety of other diet factors can increase or decrease an animal's risk for developing a deficiency of thiamine. In particular, animals that are fed unconventional diets (eg, raw food diets, nutritionally incomplete or unbalanced commercial pet foods, or home-prepared diets) have a number of unique risks.

The composition of the diet can affect an animal's dietary thiamine requirements. Diets that are high in protein and fat can have a thiamine-sparing effect, whereas those that are high in carbohydrates can actually increase thiamine requirements and worsen the effects of thiamine deficiency.19,24 The reason for this discrepancy is that high-carbohydrate diets require greater use of the TCA cycle, which requires thiamine to complete the conversion of glucose into energy (Figure 1). Thus, when there are more carbohydrates in the diet, thiamine is depleted more rapidly.

In addition, the rate of metabolism and the requirement of the TCA cycle for energy differ among tissue types within the body. Tissues with a higher requirement for glucose as the primary source of energy (eg, neural tissues in the brain) are more affected by thiamine deficiency than are other tissues in the body that have the ability to use lactate or pyruvate for energy.

Table 1—Common clinical signs of thiamine deficiency in dogs and cats.2,3,5,18–22

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<th>Neurologic</th>
<th>Ocular</th>
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<td>Altered mentation</td>
<td>Acute blindness</td>
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<tr>
<td>Acute blindness</td>
<td>Mydriasis or anisocoria</td>
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<td>Proprioceptive deficits</td>
<td>Nystagmus</td>
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<td>Ataxia</td>
<td>Gastrointestinal tract</td>
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<tr>
<td>Polyneuropathy</td>
<td>Anorexia or hyporexia</td>
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<tr>
<td>Spastic ventroflexion of the head and neck</td>
<td>Weight loss</td>
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<tr>
<td>Extensor rigidity</td>
<td>Vomiting</td>
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<td>Vestibular signs</td>
<td>Constipation</td>
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<td>Paresis</td>
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<td>Hyperesthesia</td>
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<td>Seizures</td>
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Furthermore, some areas of the brain are more affected than other areas, depending on the metabolic requirement for glucose. Those areas that are most affected define the primary neurologic signs associated with thiamine deficiency.

Although dry foods can be deficient in thiamine, it is more common in canned foods for a number of reasons. Canned food production is a multistep process that involves grinding and mixing of the food, filling and sealing of the cans, and sterilizing the food within the cans. The sterilization (retort) step is important for destroying common pathogenic bacteria. However, thiamine is a heat-labile vitamin, and losses of > 50% of the thiamine content have been considered to be a result of processing. In addition, some canned diets include alkalinizing gelling agents that can alter the pH and therefore the availability of thiamine. Manufacturers should consider all of these factors, use analytic methods to estimate the amount of thiamine lost to processing or inactivated because of pH, and supplement the diet with additional sources of thiamine prior to the sterilization process to compensate for impending losses. In addition, reputable manufacturers will analyze the final diet to determine the content of thiamine and other nutrients to ensure that they meet minimum values.

The duration and environmental conditions associated with storage of a commercial cat or dog food after manufacturing can further affect the amount of vitamin loss over time. Although B vitamins are not as susceptible to loss during storage as are fat-soluble vitamins, thiamine is one of the B vitamins most susceptible to loss during storage. For dry pet foods, overall vitamin B loss has been estimated to be as high as 2% to 4%/mo under ideal environmental conditions. It has been suggested that thiamine losses can be as great as 57% in dry dog food and 34% in dry cat food after 18 months of storage; however, the loss of thiamine appears to be minimal in canned food. Heat, humidity, and exposure to air can affect the amount of nutrient losses in a diet, and owners should be questioned and educated as to their techniques for storage of pet foods at home. In a recent study, investigators found that 30% of owners do not store pet food in an air-conditioned environment, despite the fact that they live in a warm climate. Improper storage or feeding techniques could include food that is fed after the manufacturer's expiration date, bags that have been opened and not resealed between meals, or food that has been allowed during the summer to remain in the trunk of a car or in a garage prior to feeding.

In addition to nutrient losses from processing and storage, specific diet ingredients can also affect thiamine availability. Fish and shellfish, in particular, are known sources of thiaminases. Thiaminases are enzymes that degrade and inactivate thiamine in fish or other ingredients with which the fish are combined. Species of fish and shellfish differ in the amounts of thiaminase activity, as do parts within the same fish. The viscera of thiamine-containing fish species tend to contain more thiaminase than that found in the muscles or skeleton of fish. Therefore, potentially inadequate amounts of thiamine would be of particular concern for owners feeding raw fish— or shellfish-based diets or any time substantial amounts of raw fish are included in the diet. Raw fish are not the only sources of thiaminase, which can also be found in some species of plants, bacteria, and fungi. These could potentially be an issue for pet food when added as ingredients or indirectly through contamination. Although thiaminases are heat labile and are destroyed by cooking and standard food-processing techniques, the amount of time that thiaminase is in contact with thiamine-containing foods within a diet prior to cooking plays a role in the amount of thiamine that is ultimately destroyed. Therefore, contact between thiamine-containing foods and thiaminase-containing foods can lead to inactivation of thiamine. This issue may be important in fish-based commercial pet foods produced by manufacturers that do not routinely test their ingredients and end-products for thiamine. Processed fish meal is heat treated; thus, it would pose no risk because the thiaminase has been denatured.

The use of certain food preservatives can also impact the availability of thiamine, as indicated by several outbreaks of thiamine deficiency in Australia secondary to the use of sulfur-based preservatives. Sulfur dioxide can cause concentration-dependent inactivation of thiamine. Sulfur derivatives are used for preserving meat through inhibition of bacterial growth, extending the storage life, and maintaining the red color of the meat. Many countries have banned the use of sulfur preservatives in the production of meat products intended for human consumption; however, in some countries, the same bans do not apply to meat intended for pet foods or to all types of meats available for human consumption. In the United States, sulfur dioxide is specifically prohibited for use as a preservative in meats or in products that serve as sources of vitamin B.

Not surprisingly, incomplete or unbalanced commercial diets are also a cause of vitamin deficiencies. Diets intended for supplementary or intermittent feeding (ie, supplementary diets) are not nutritionally complete and balanced. This is not always readily apparent to consumers from the label and can result in a number of nutritional deficiencies, including thiamine deficiency, when a supplementary diet is fed as a sole or primary diet. Although some veterinary therapeutic diets may be labeled for intermittent or supplemental feeding to specifically address certain disease conditions, over-the-counter commercial foods should always be complete and balanced because these products are not dispensed under the guidance of a veterinarian. Thiamine deficiency was identified in a published report of a colony of cats fed 2 supplementary diets over a course of 6 months, which resulted in the death of 5 cats and clinical signs in 23 others. When in doubt, pet owners should bring the food label for their pet's food to their veterinarian so that it can be evaluated to help identify this potential cause of thiamine deficiency. It is also important for veterinarians to proactively educate owners that an over-the-counter food label that states "for intermittent or supplemental use" indicates the diet is not complete and balanced and should be fed in only small amounts as a treat, rather than as a sole or primary diet.

Nutritional imbalances are of major concern for owners who prefer to feed a home-prepared diet.
Anecdotally, home-prepared diets fed by most owners include a protein and carbohydrate source with or without other ingredients, but all-meat diets are not uncommon. Although it might be expected that a diet consisting primarily of meat would be sufficient in thiamine, the amount of thiamine differs among cuts of meat and the species from which the meat is obtained; thus, these diets are not consistent and may not provide adequate thiamine intake. More importantly, as mentioned previously, cooking will destroy between 73% and 100% of the thiamine content in most meat. Additionally, the thiamine in the meat and other ingredients is likely to be destroyed (regardless of any effects from cooking) in home-prepared diets that are high in thiaminase-containing fish or sulfite-preserved meat.

Certain medications have also been associated with the induction of thiamine deficiency. Amprolium is a coccidiostat medication that has been associated with thiamine deficiency in ruminants and dogs. Amprolium works within parasites by mimicking the action of thiamine; however, prolonged administration or excessive doses can lead to thiamine deficiency in the host. Conversely, diets high in thiamine or dietary supplementation with thiamine can render amprolium less effective, and this factor should be considered for animals to which amprolium is prescribed. A similar medication is pyrimethamine, which is an inhibitor of folic acid synthesis. Pyrimethamine is often prescribed to treat protozoal infections such as toxoplasmosis, neosporosis, or hepatozoonosis. Pyrimethamine can induce thiamine deficiency in dogs and cats and has been used to experimentally induce thiamine deficiency in cats and rats.

The aforementioned risk factors, many of which are dietary factors, highlight the importance of collecting a good diet history as the key to determining the various components of an animal’s diet, the duration of feeding of that diet, and clinical signs. The American Animal Hospital Association and World Small Animal Veterinary Association have developed nutritional assessment guidelines that include collection of a thorough diet history on every animal at every visit. A diet history should include the type and amount of diet fed, duration of feeding of that diet, frequency of feeding, treats, supplement-type products, foods used for medication administration, and appetite of the animal. This information will be instrumental in identifying risk factors for thiamine deficiency as well as other deficiencies, excesses, or other forms of suboptimal nutritional status.

**Diagnosis of Thiamine Deficiency**

The diagnosis of thiamine deficiency is initiated by recognition of relevant clinical signs and evaluation of the medical and diet history. Results of serum biochemical analyses, CBCs, and urinalyses are often within reference limits in dogs and cats with thiamine deficiency and may not aid in the diagnosis. Similarly, evaluation of CSF is unhelpful in obtaining a diagnosis, but it may aid in ruling out other infectious and inflammatory causes. Magnetic resonance imaging can provide characteristic findings suggestive of thiamine deficiency, namely bilateral hyperintense areas that correlate with several brainstem nuclei (Figure 2). A definitive diagnosis is obtained by determining a reduction in blood or tissue thiamine concentrations.

Several forms of thiamine exist within the body; however, there currently is no single test that measures all forms of thiamine. The 2 most commonly used methods for assessing thiamine status in vivo are the erythrocyte transketolase activity assay and HPLC. The erythrocyte transketolase activity assay is a functional assay that measures the activity of TPP in erythrocytes. Because this method measures transketolase activity rather than measuring thiamine directly, it can be affected by other systemic processes (e.g., diabetes mellitus or liver disease) in an animal. By contrast, the HPLC method directly measures the TPP form of thiamine within RBCs. The HPLC method is more sensitive, specific, and stable than the erythrocyte transketolase activity assay for the measurement of thiamine. In humans, HPLC (which is typically performed on whole blood) has the additional benefit of better representing total body status. However, neither method can measure the small percentage of thiamine in the plasma; they only measure the thiamine in the erythrocytes or tissue. Furthermore, these assays are not available at all diagnostic laboratories, and specific procedures for collection and transport of samples may apply.

Other less commonly used methods that can lead to a diagnosis include the measurement of urinary thiamine or urinary organic acid concentrations or analysis of dietary thiamine content. Because of the difficulties in obtaining a definitive diagnosis, some clinicians may elect an empirical treatment trial in an animal with a relevant medical history and clinical signs while waiting for test results or in lieu of testing.

Unfortunately, some cases of thiamine deficiency are not diagnosed until after an animal dies or is euthanatized. Gross postmortem examination of animals with thiamine deficiency may reveal bilateral symmetric focal hemorrhages clearly visible in the paraventricular gray matter. Brainstem nuclei typically involved in companion animals with thiamine deficiency include the lateral geniculate nuclei, caudal colliculi nuclei, dor-

![Figure 2—Transverse T2-weighted fluid-attenuated inversion recovery image at the level of the thalamus in an adult cat with thiamine deficiency. Notice the bilateral hyperintense lesions (white arrow) in the region of the lateral geniculate nuclei. R = Right.](image-url)
Treatment

Treatment of thiamine deficiency involves parenteral administration of thiamine for 3 to 5 days, followed by oral administration for an additional 2 to 4 weeks. In 1 veterinary formulary, dosages for parenteral administration to dogs or cats ranged from 1 to 250 mg every 12 to 24 hours. The ideal dose of thiamine is unknown; however, doses described for clinical cases range from 25 to 150 mg in cats and 100 to 600 mg in dogs. The authors typically use a dose of 50 to 100 mg every 12 hours for cats, but further research is needed to determine the optimal dose needed to correct thiamine deficiency.

Route of administration and duration of treatment differ widely. In addition, some clinicians initiate treatment with a loading dose, whereas others prefer a consistent dose throughout treatment. Irrespective of the dose, response to treatment is often noticeable within hours. Parenteral administration of thiamine is particularly important in an animal with concurrent vomiting or gastrointestinal tract disease because intestinal absorption of the vitamin may be impaired. Pure thiamine hydrochloride, which commonly is provided at a concentration of 100, 200, or 500 mg/mL, is preferred over solutions of vitamin B complex because the composition of vitamin B complex products differs widely. For example, the thiamine content of 8 commercial solutions of vitamin B complex ranged from 10 to 150 mg/mL (up to one-fiftieth the content of a solution of pure thiamine). To achieve a dose of 100 mg for a cat, between 0.66 and 10 mL of vitamin B complex would be required every 12 hours, depending on the brand of the vitamin B complex.

Intravenous administration of thiamine should be avoided because dose-dependent hemodynamic effects (eg, acute hypotension, cardiac arrhythmias, neuromuscular or ganglionic blockade, apnea, or death) could result. In 1 study, investigators found that IV administration of thiamine at doses ≥ 20 mg/kg (9.1 mg/lb) to cats and 80 mg/kg (36 mg/lb) to dogs led to neuromuscular paralysis and ganglionic blockade. Therefore, parenteral administration should be provided via SC or IM injection. In the event that an overdose of thiamine is administered IV, norepinephrine may reduce the adverse hemodynamic effects of the vitamin. It is important to mention that oral intake of an excess of thiamine via supplementation of a commercial diet or as a supplement-type product has not been found to induce adverse clinical effects.

Improvement in thiamine status can be monitored via clinical signs, repeated MRI, and measurement of thiamine status via HPLC or erythrocyte transketolase activity. There may be residual neurologic deficits such as mild ataxia after treatment is completed. In addition, an animal’s diet should be evaluated and changed to a good-quality, complete, and balanced diet if a home-prepared, raw, or supplementary diet is being fed. For a cat or dog that is consuming a complete and balanced commercial diet and that has no medical reason for developing thiamine deficiency, diagnosis or suspicion of thiamine deficiency should be reported to the food manufacturer and the FDA. Concerns can be reported to the FDA by calling the FDA Consumer Complaint Coordinators for each state that are listed on the FDA website or by reporting concerns to the FDA Safety Reporting Portal online. Ideally, the lot and expiration date should be obtained from each bag or can of pet food, and multiple cans from the same lot or samples of dry food from the same bag should be retained for testing as needed.

Other Issues of Clinical Importance Related to Thiamine

Dietary thiamine supplementation should be considered when starting parenteral provision of nutrients (ie, parenteral nutrition) in dogs and cats because there have been several reported cases of the development of thiamine deficiency in humans receiving parenteral nutrition without vitamin supplementation. The authors recommend that most parenteral nutrition mixtures include B vitamins because animals in need of parenteral nutrition frequently have increased metabolic requirements for these vitamins and may have an extended history of inadequate caloric and nutrient intake as a result of illness. The provision of thiamine should precede IV administration of dextrose because thiamine is required for the conversion of glucose to energy via the TCA cycle. Therefore, dextrose administration could potentiate an underlying deficiency.

The addition of vitamin B complex products to crystalloid solutions is a popular technique to provide additional B vitamins to hospitalized dogs and cats or to animals receiving fluids administered at home (eg, pets with chronic kidney disease). Most commercial solutions of vitamin B complex contain thiamine, riboflavin, niacinamide, pyridoxine hydrochloride, d-panthothenol, and cyanocobalamin; however, the concentration of each B vitamin (including thiamine) differs widely among commercial solutions. Similar to thiamine, the optimal dose of vitamin B complex is unknown. A common standard dose in use by many veterinarians is 1 to 2 mL/L of fluids administered IV, irrespective of the rate of fluid administration or the brand of vitamin B com-
plex. However, the actual dose of B vitamins administered via this method differs widely depending on the brand of vitamin B complex selected as well as the rate of fluid administration. As mentioned previously, there may be a 15-fold difference in thiamine concentration, and the other B vitamins have similar wide variations for the various formulations. In addition, possible concerns with the use of B vitamins in IV fluid therapy include potential interactions with concurrent additives (eg, potassium chloride) and the degradation of B vitamins via light exposure. Key points to consider for the administration of vitamin B complex is the route and rate of administration and knowledge of the content of the specific vitamin B complex used so that clinicians are able to calculate an appropriate dose for each animal requiring thiamine.

A common dietary supplement, brewer’s yeast, reportedly is a rich source of B vitamins and is sometimes used as part of home-prepared diets to provide additional B vitamins to animals with diseases (eg, chronic kidney disease) or for its reported insect-repellant effects. There are differences in formulations of brewer’s yeast, but 30 mg (2 tablespoons) of brewer’s yeast provides approximately 1 mg of thiamine. This means that the typical cat would need 3,000 mg (200 tablespoons) of brewer’s yeast/d to provide the amount of thiamine used to correct thiamine deficiency. In addition, 2 prospective case-controlled clinical studies revealed that there was no flea-repellent benefit for dogs administered brewer’s yeast.

**Clinical Summary**

Thiamine deficiency is unlikely to be diagnosed on initial evaluation until overt neurologic signs develop; therefore, thiamine deficiency may be underreported in companion animals. A thorough and complete dietary history is integral to the initial identification of at-risk or affected animals. Identification and diagnosis of this disease remain the biggest challenges for clinicians, and treatment should not be delayed because of a lack of a definitive diagnosis. Treatment results in complete resolution of clinical signs in most animals, providing extensive neurologic damage has not already occurred.

References


33. Hutchinson D, Freeman LM, McCarthy R, et al. Seizures and
severe nutrient deficiencies in a puppy fed a homemade diet. 


