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Veterinary Referral News from Angell Animal Medical Center

AAHA ACCREDITED The Standard of Veterinary Excellence

Spring 2013
Volume 7:2

Iron Deficiency in Dogs and Cats

By Patty Ewing,
DVM, MS, DACVP
(Anatomic and Clinical Pathology)

angell.org/lab
pathology@angell.org
617 541-5014

INTRODUCTION

Iron is an essential trace mineral that has a variety of critical biologic functions. Iron is a component of hemoglobin, myoglobin, and cytochrome enzymes and is stored in the liver as ferritin or transferrin, which can be measured in the serum to assess iron status. The total amount of iron in the body is closely regulated by coordinating daily loss and intestinal absorption of iron. Iron deficiency results when iron demand by the body is not met by iron absorption by the intestine from the diet. Because iron is a critical component of hemoglobin, iron deficiency can result in life-threatening anemia. The major causes of iron deficiency include loss of iron via hemorrhage, and inadequate intake or absorption of iron (Table 1).[1] Clinical signs of iron deficiency may include weakness, decreased activity, dyspnea (with severe anemia), altered behavior, gastrointestinal disorders secondary to decreased gastric acid secretion and intestinal malabsorption, and increased risk of infection.[2,3]

IRON DEFICIENCY IN DOGS

At Angell Animal Medical Center, most cases of iron deficiency anemia (IDA) are due to chronic hemorrhage from gastrointestinal tumors. Diagnosis of uncomplicated and chronic iron deficiency is based on classic hematologic and biochemical abnormalities such as changes in erythrocyte indices (microcytic, hypochromic anemia) and decreased serum ferritin concentration (Table 2). Detection of early iron deficiency, before the appearance of those abnormalities, or iron deficiency in the presence of other diseases (e.g., neoplasia and inflammation) is a diagnostic challenge.[4,5] In uncomplicated iron deficiency, the serum iron concentration and ferritin are typically low and the total iron binding capacity (TIBC) is usually high. Because iron is preferentially shunted to hemoglobin (Hb) formation, typical hematologic changes do not occur until late in iron deficiency, long after detrimental effects have occurred. Also, serum ferritin is an acute-phase reactant and concentrations may be increased with certain neoplastic and inflammatory diseases, which may make diagnosis of concurrent iron deficiency challenging. Although the immunologic assay for canine serum ferritin has been available for over a decade, its value in detecting IDA in chronically ill dogs is not reported. Many clinicians consider the evaluation of bone-marrow aspirates or biopsies (for the presence or absence of stainable iron) as a sensitive and reliable test for iron status assessment in dogs. However, some hematologists consider this invasive procedure to be subjective and imprecise. Thus, the true prevalence of iron deficiency may be unknown, and dogs with early iron deficiency masked by concurrent disease may remain undiagnosed and untreated.

Table 1: Causes of Iron Deficiency in Dogs and Cats
Transient iron deficiency anemia (IDA) in neonates on all-milk diet
Blood-sucking parasites
Intestinal bleeding (tumors, ulcers, etc.) or intestinal malabsorption
Chronic kidney disease
Urinary bleeding (bladder tumors)
Hemorrhagic disorders (diathesis, coagulopathy)
Latrogenic (blood donors)

> Continued on Page 2

Inside

Update on Pimobendan 4
Progressive Rod Cone Degeneration in Dogs 5
Proximal Tibial Epiphysiodesis (PTE) 6

Courtesy Consultations

Angell specialists are available for consultation Monday–Friday, 9:00 a.m.–5:00 p.m.

Additionally, Angell emergency doctors are available for consultation on weekends and after hours (7:00 a.m.–11:00 p.m.).

Referral Contact Information

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Online Referral Forms

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Upcoming CE Schedule

Please see Page 6

Available at **angell.org/CE**

> Iron Deficiency in Dogs and Cats (Continued from Page 1)

Table 2: Features of Severe Iron Deficiency Anemia in Dogs

Parameter	Finding
Serum iron	Decreased
Total Iron Binding Capacity (TIBC)	Normal
Ferritin	Decreased
Transferrin saturation	<20%
Hematocrit (HCT)	Low
Mean Corpuscular Volume (MCV)	Decreased
Mean Corpuscular Hb Content (MCHC)	Decreased
Red Blood Cell (RBC) morphology	Hypochromasia, microcytosis, and RBC fragmentation
Red-cell distribution width (RDW)	Increased anisocytosis
Platelets	Thrombocytosis (increased platelet count)
Chemistry panel	Low total protein related to concomitant intestinal losses of albumin and globulin

Accurate diagnosis of early or complicated iron deficiency is made more difficult by the highly variable results obtained for biochemical markers of iron in serum; including serum iron (Fe) concentration, TIBC, and percent saturation of transferrin. Serum Fe concentration is affected by several factors such as time of day, corticosteroid administration, and consumption of meat.[4] Reticulocyte indices may be helpful in evaluating for possible causes of anemia. Fry *et al.* demonstrated that reticulocyte indices differed by greater than threefold between healthy dogs and dogs with IDA.[5] Steinberg *et al.* demonstrated that low mean Hb content of reticulocytes and low reticulocyte mean corpuscular volume (MCV) are associated with hematologic and serum biochemical abnormalities indicative of iron deficiency.[4] Both these indices hold promise as non-invasive, cost-effective measures of iron status assessment in dogs.[4]

Small-intestinal malabsorption can also promote IDA. Iron deficiency anemia secondary to inflammatory bowel disease (IBD) has been reported in dogs; therefore, determination of serum iron, ferritin, and TIBC may be worthwhile in anemic dogs with IBD.[6] In one study, serum iron concentrations in three of six dogs and three of seven cats with chronic renal failure (CRF) were below the reference interval (transferrin saturation less than 20%).[7] Whether this is related primarily to inadequate intake and absorption of iron, or increased losses of iron due to GI blood loss, is unclear.[7] It is important to distinguish anemia secondary to iron deficiency from anemia of inflammation, since only iron deficiency should be supplemented with iron. When erythropoietin (EPO) therapy is used in CRF patients, the demand for iron during stimulated erythropoiesis is high; therefore, iron supplementation is recommended for patients receiving EPO.[8]

IRON DEFICIENCY IN CATS

Iron deficiency may occur if insufficient stores are accumulated during the last week of pregnancy.[9] In the 1980s, iron deficiency anemia was reported to be common in five-week-old kittens. Kittens at this age had the highest frequency of microcytosis. At seven weeks of age, the kittens stopped producing microcytic cells. The age-related change implicated the all-milk diet in a rapidly growing animal as the cause.[9] Milk is a poor source of iron, and kitten requirements are usually higher than the intake. Now that kittens are transitioned to commercial diets typically by three weeks of age, nutritional iron deficiency is infrequently reported, and the most common cause for iron deficiency in cats is parasitism (internal and external).

Iron deficiency anemia in adult cats has rarely been reported. Although red blood cell (RBC) fragmentation may occur in cats with iron deficiency anemia, hypochromasia is not reported as a common feature. Serum iron and percent transferrin saturation values decrease.[2] An assay for feline ferritin is commercially available. Determination of concentrations of serum iron and serum ferritin may be useful in the differentiation of anemia of chronic inflammation from IDA in cats.[10]

Cats may have greater gastrointestinal iron-absorptive capacities than other species.[11] Additionally, detection of classic hematologic lesions of iron deficiency is more challenging than in dogs. This challenge is due to the lack of stainable iron in normal feline bone marrow, infrequency of hypochromasia, and the difficulty of detecting microcytosis by traditional hematologic methods in cats.[2]

TREATMENT

Transfusion of red blood cells (whole blood or packed red blood cells) should be considered for patients with IDA clinical for anemia.[1] The patient hematocrit should be raised to 15–20%. The goal of calculating the transfusion volume is to avoid reaching a higher hematocrit that will dampen the bone marrow erythrocyte production (Table 3).[12]

> Continued on Next Page

Table 3: Calculation of approximate volume of blood required to raise hematocrit levels

Volume of Donor Blood (ml)	=	Recipient Body Weight	X	90 (Dog) 70 (Cat)	X	PCV Desired – POV Recipient PCV of Donor in Anticoagulant

> Iron Deficiency in Dogs and Cats (Continued from Previous Page)

Table 4: Iron Products for Treatment of Iron Deficiency in Dogs and Cats

Product	Route	Dose	Comments
Iron dextran 50 mg/ml	IM	Cats (adjunct to EPO treatment): 50 mg IM every 3–4 weeks Dogs: 10–20 mg/kg IM once, and continue treatment with oral ferrous sulfate	A small dose should be injected IM to test for hypersensitivity reaction. A maximal dose of 2 ml is administered daily.
Ferrous sulfate	PO	Cats: 50–100 mg/cat/day Dogs: 100–300/dog/day	Exists in two forms (regular and dried). Doses are given for regular ferrous sulfate and not for elemental iron. A dose of 300 mg of iron sulfate provides 60 mg of elemental iron. Side effects: anorexia, vomiting. If bad GI side effects develop, it is recommended to give IM Fe dextran.
Ferrous gluconate	PO		A dose of 325 mg of iron gluconate provides 36 mg of elemental iron.
Pet-Tinic® (Pfizer Animal Health)	PO		Contains 12.5 mg of iron per 1 tablespoon. For adequate iron supplementation at time of iron deficiency, a cat would need to ingest 4–8 tablespoons of the liquid per day (which is not realistic).
Vitamin C	PO	Dogs: 500–1000 mg per day Cats: 125 mg BID	Can be used to increase GI absorption of oral iron. Side effects may include GI disturbances and increased risk for urate, oxalate, and cystine stone formation.

The first line of therapy for iron deficiency anemia is parenteral iron administration. Iron preparations administered intravenously may cause anaphylactic reactions, thus the intramuscular (IM) route is preferred.[13] A small dose should be injected IM to test for hypersensitivity reactions. A maximal dose of 2 ml can be administered daily.[14] Large doses of injectable iron may discolor the serum brown, which can cause falsely elevated serum bilirubin values and falsely decrease serum calcium values.[13] Iron deficiency in dogs is addressed first by administering iron dextran once at 10–20 mg/kg IM and then continuing therapy with oral iron. In cats the dose for prevention of transient iron deficiency anemia in kittens is 50 mg of iron dextran IM at 18 days of age. The dose for adjunctive therapy with EPO treatment is 50 mg of iron dextran IM every three to four weeks or daily oral supplementation. Oral iron therapy usually follows injectable iron (Table 4). Oral iron absorption varies widely based on the type of diet and other factors. Sustained-release iron formulations are not recommended as initial therapy, because they reduce the amount of iron that is presented for the absorption by the duodenal villi.[1] Gastrointestinal absorption of elemental iron is enhanced in the presence of an acidic gastric environment. This can be accomplished through concurrent intake of ascorbic acid (Vitamin C).[1, 13]

Most common side effects of oral iron supplementation are gastrointestinal upset (mostly vomiting) and constipation. Dividing the daily dose may reduce gastrointestinal upset. Although iron absorption occurs more readily when taken on an empty stomach, this increases the likelihood of stomach upset. Oral iron may result in black discoloration of feces and false-positive reactions with the guaiac occult blood test.[13] A common product used for iron supplementation is Pet-Tinic (Pfizer Animal Health). It contains 12.5 mg of iron per tablespoon. For adequate iron supplementation at time of iron deficiency, a cat would need to ingest 4–8 tablespoons of the liquid per day. Many cats will not tolerate oral supplementation; thus injections of iron dextran may be required in some cases.[13]

CONCLUSION

Chronic blood loss from bleeding gastrointestinal tumors and intestinal parasitism are the most common causes of iron deficiency. Early recognition and treatment of iron deficiency can prevent life-threatening anemia. IDA must be differentiated from anemia of chronic disease so that appropriate treatment can be initiated.

For more information about iron deficiency, please contact Angell's Pathology service at **617 541-5014** or **pathology@angell.org**. ■

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An Update on Pimobendan

By Adam Kane, DVM

angell.org/cardiology
cardiology@angell.org
617 541-5038

Medical management of cardiac disease in veterinary patients is constantly evolving and improving. Either through the introduction of new medications or updated applications of existing medications, we have been able to afford our patients both improved quality and quantity of life. The advent of pimobendan (Vetmedin®) is one of the more recent examples of this beneficial evolution. In general, it is a medication that has been found to have great benefits with typically minimal side effects. However, as with any medication or therapy, appropriate use is paramount and guidelines established by evidence-based medicine and the subsequent peer-reviewed literature should be followed.

Pimobendan is a benzimidazole-pyridazinone derivative, approved by the FDA in 2007 for use in the treatment of congestive heart failure in canine patients. Because its mechanism of action does not affect endogenous catecholamines, stimulate cardiac-adrenergic receptors, or inhibit the Na+/K+-ATPase (sodium potassium) pump, it is classified as a nonsympathomimetic, nonglycoside, inotropic drug. Pimobendan is an inodilator that has both positive inotropic and vasodilatory effects.

In the failing heart, pimobendan exerts its positive inotropic effects primarily by increasing the binding affinity of cardiac troponin C for calcium. Calcium cycling is one of the primary determinants of myocardial contractility. The binding of calcium to cardiac troponin C allows for interaction between the contractile proteins of the cardiac myocytes, resulting in force generation and myocardial contraction. The increased binding sensitivity for calcium results in an increased strength of contraction. Positive inotropy is also conferred by the medication's phosphodiesterase-3 inhibitory effects within cardiac muscle cells. Phosphodiesterase-3 inhibition leads to phosphorylation of several cellular proteins involved in calcium uptake and release. The net result is a more rapid sequestration of calcium during diastole, and more profound release of calcium during systole, both of which contribute to an overall positive inotropic effect. Because the total intracellular concentration of calcium is not altered by pimobendan, this positive inotropy is achieved without increasing the myocardial oxygen demand.

Pimobendan is a mixed vasodilator (arterial and venodilator). This effect is also mediated by the medication's phosphodiesterase-3 inhibitory properties. Through various signaling mechanisms in vascular smooth muscle cells, this causes an increase in sequestration of intracellular calcium, making less calcium available for smooth muscular contraction within the blood vessels. Pimobendan also has several other biologic activities of lesser importance, including mild immunomodulatory effects by decreasing pro-inflammatory cytokines, decreased catecholamine synthesis, and antithrombotic effects.

Side effects with the medication are uncommon, but typically include gastrointestinal signs such as decreased appetite and diarrhea. There has been concern surrounding possible arrhythmogenicity of pimobendan, due to prolongation of the cardiac action potential, but this concern has not been supported in various studies.

↘ Angell cardiology patient with dilated cardiomyopathy

Pimobendan's use in the treatment of canine chronic degenerative valvular disease has been evaluated in multiple studies involving both pre- and post-heart-failure patients. Unfortunately, no clear benefit has been established for patients with valvular disease prior to the onset of congestive heart failure. A study of asymptomatic dogs with valvular disease treated with pimobendan or benazepril showed worsening of mitral regurgitation, with corresponding valvular and endocardial lesions in those dogs treated with pimobendan, despite an improvement in systolic function. These findings were corroborated by a case report of two dogs that developed similar lesions with long-term pimobendan therapy; the lesions resolved after discontinuation of the medication.

Conversely, several studies have underscored the benefits of pimobendan after the onset of heart failure in patients with valvular disease, and its use at this stage is supported by the ACVIM consensus statement for canine chronic valvular disease. The VetSCOPE study compared the use of pimobendan to that of benazepril in heart-failure patients. Those treated with pimobendan had a prolonged time to death or treatment failure, with an average of 415 days compared to 128 days for those in the benazepril treatment group. The QUEST study further supported these results, showing that the pimobendan-treated group had significantly longer time to the primary endpoint than the benazepril group (267 days versus 140 days).

Canine patients with dilated cardiomyopathy (DCM) may be the group that has benefited the most from pimobendan therapy. Its use in dogs with DCM and subsequent congestive heart failure has been supported for several years. One study compared the survival times of Cocker Spaniels and Doberman Pinschers treated

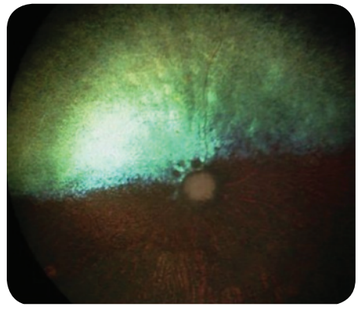
> Continued on Page 7

Progressive Rod Cone Degeneration in Dogs

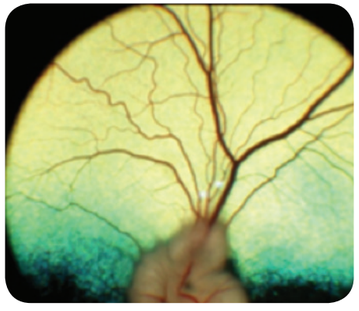
By Dan Biros, DVM, DACVO

angell.org/eyes
ophthalmology@angell.org
617 541-5095

One of the more interesting causes of vision loss in dogs is due to progressive rod cone degeneration (PRCD), a hereditary loss of photoreceptor function (often at times mistaken for cataract formation by clients). The disease is caused by a genetic mutation in the photoreceptors of the retina. The cells develop normally, but in adolescence or early adulthood the mutation causes the retinas to degenerate over time, leading to blindness.



↘ Figure 1



↘ Figure 2: Normal dog fundus

When a patient presents for vision loss and the visual axis is clear, the attention turns to diseases causing retinal dysfunction including glaucoma, sudden acquired retinal degeneration (SARD), optic neuritis, other central nervous system (CNS) disease, retinal dysplasia or detachment, or one of the many varieties of retinal degeneration. The key points of differentiating photoreceptor degeneration from other types of retinal disease are a) the slow rate of progression; b) the characteristic nyctalopia, or night vision loss, as the early indicator of a problem (daytime vision often remains normal for a while); c) the absence of inflammation or ocular hypertension; and d) the often normal appearance of the retina early in the disease. Quite often the eyes are not red, and the pupillary light reflexes (PLRs) may be normal early on, with a gradually diminished response as the condition advances. In later stages we will see signs of retinal thinning, namely the retinal blood vessel attenuation and tapetal hyperreflectivity characteristic in the end-stage disease (Figure 1). As the retina degenerates over months to years, the optic nerve also atrophies, leaving a smaller and often darkened optic disk. By contrast the normal dog fundus has a well developed retinal vasculature, a large myelinated optic nerve, and a tapetum that is not hyperreflective (Figure 2). Both eyes usually progress equally, but it is not unusual to have unequal PLRs as the degeneration takes place. In later stages the condition may also be associated with cataract development. The relationship of the cataract development to the retinal disease is not clear, but it may be associated with the release of metabolic toxins in the ocular microenvironment that appear as the retina degenerates.

As mentioned, PRCD does not appear suddenly and can be contrasted to SARD, which is a rapid loss of vision due to loss of retinal function. While most PRCD cases are hereditary, the SARD condition is not. (The cause of SARD is currently unknown.) The mode of PRCD inheritance is usually autosomal recessive, but in select breeds is autosomal dominant (Mastiffs) or even x-linked (Samoyeds and Siberian Huskies). Other categories of disease can be further broken down to early- and late-onset. Classic early-onset PRCD can be evident as early as a few weeks of age, with late-onset more typically appearing starting at 5–7 years of age. Breeds affected by early onset include Irish Setters, Collies, Tibetan Terriers, and Cardigan Welsh Corgis. Breeds with late-onset disease include Miniature and Toy Poodles, English and American Cocker Spaniels, English Springer Spaniels, and Labrador Retrievers. Both SARD and retinal degeneration are confirmed by the characteristic appearance of the retinas, either in the later stages or early stages, by electroretinography. Also, the Optigen independent research laboratory (optigen.com), run in consultation with Penn and Cornell researchers, can provide blood testing for many of the hereditary conditions, which has proven helpful in screening for carriers of the disease. This is important since many of the conditions are autosomal recessive. With genetic lineage it can be beneficial to identify carriers and limit propagation of the genetic disease.

There is no cure for PRCD and no accepted treatment for slowing the disease or prolonging vision. Causes for the disease have been theorized, and among those proposed ideas are changes in plasma lipid levels (docosahexaenoic acid, 22:6n-3). A few veterinary ophthalmologists have developed a neutraceutical product, Ocu-Glo™ (ocuglo.com), containing a mixture of antioxidants and vitamins that may support ocular health in the face of retinal disease. There is no proof that it slows down or reverses the damage caused by the genetic changes, but it may promote the optimal conditions for vision during disease progression. Large, randomized studies are needed to support any testimony that the product actually stabilizes or preserves retinal function and vision. We offer this product to clients who want to try something that may help, but we say up front there is no documented medicinal benefit by way of peer-reviewed papers at this time.

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For more information about Angell's Ophthalmology service, please visit angell.org/eyes. Drs. Coster and Biros are available for consultations or referrals at **617 541-5095**, or e-mail at **ophthalmology@angell.org**. ■

Proximal Tibial Epiphysiodesis (PTE): An Alternative Technique to Correcting the Cranial Cruciate Ligament Deficient Stifle in Growing Dogs

By Nick Trout,
MA, VET MB, DACVS, ECVS

angell.org/surgery
surgery@angell.org
617 541-5048

Canine cruciate disease is arguably one of the most common orthopedic problems facing veterinarians today. In part, our awareness of stifle abnormalities has been heightened by an appreciation of effective surgical options for athletic or energetic dogs, in the form of tibial plateau leveling osteotomy (TPLO) and tibial tuberosity advancement (TTA). These procedures tend to be used on skeletally mature dogs of any breed, but what if your patient is still growing? Cruciate ligament injuries can occur at any age and, from time to time, four- to eight-month-old dogs will present with partial or complete cruciate tears. Should we wait until the proximal tibial growth plates are closed, or are we limited to stifle stabilization with a lateral suture technique?

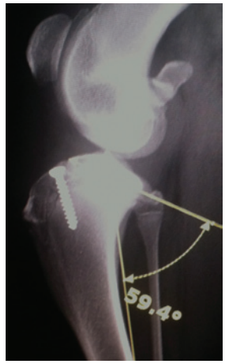
Proximal tibial epiphysiodesis (PTE) offers an alternative technique to correcting the cranial cruciate ligament deficient stifle in growing dogs. Think of it as a "slow TPLO." By prematurely closing the cranial portion of the tibia plateau using a carefully positioned cancellous screw, growth can be impeded while allowing the caudal portion to continue growing. As long as the dog has sufficient residual growth potential, the result is a progressive decline in the tibia plateau angle. In the same way as a TPLO, PTE can eliminate the need for a cranial cruciate ligament to counteract tibial thrust, thereby achieving dynamic stifle stabilization.

Accurate screw placement using a small lateral arthrotomy can be achieved intra-operatively, using plain radiographs or fluoroscopic guidance. Eccentric screw placement has been reported to produce tibial valgus deviation, though this did not cause clinical signs or require corrective surgery. The technique is minimally invasive and can be performed on an outpatient basis. Standard postoperative restrictions apply and dogs typically require follow-up radiographs every four to six weeks until growth plates close.

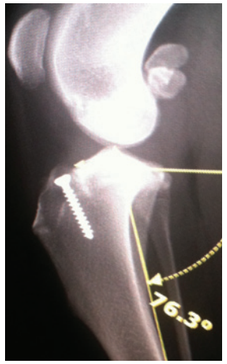
PTE is a simple, practical, well-tolerated procedure that answers the question of how to treat cruciate disease in young dogs. Owners should be aware that resolution of lameness requires growth and this takes several weeks to achieve. They should also appreciate that this technique may not work in dogs over eight months of age or where the growth plate closes early. Screw removal has been reported to prevent overcorrection of the tibial plateau angle, though in my experience this has not proven necessary.

For large breeds or dogs that are disposed to be active, PTE offers dog owners a fantastic alternative to a lateral suture technique or the frustration of waiting until skeletal growth is complete.

For more information about this technique, please contact Angell's Surgery service at **617 541-5048** or **surgery@angell.org**. ■



↘ Figure 1: Immediate postoperative lateral stifle radiograph in a seven-month-old male Giant Schnauzer with a tibial plateau angle of just over 30 degrees.



↘ Figure 2: Three months later, the tibial plateau angle is less than 14 degrees and clinical lameness has resolved.

Upcoming Angell CE Seminars

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Upper Airway Stenting: Indications, Complications, and Case Reports

Wednesday, October 9, 2013
6:15 p.m.–8:45 p.m.
2 CE Credits (pending R.A.C.E. approval)

CE Location:
Hilton Hotel–Dedham, 25 Allied Drive, Dedham, MA
(at the intersection of Route 128 and Route 1)

Speakers:
Maureen Carroll, DVM, DACVIM; Erika de Papp, DVM, DACVIM; Shawn Kearns, DVM, DACVIM; and Kirstin Johnson, DVM, DACVIM

Complimentary dinner and lecture for veterinarians.

Sponsored by Dextronix

> An Update on Pimobendan (Continued from Page 4)

with pimobendan versus placebo after the onset of heart failure. Both breeds in the pimobendan group survived for a significantly longer period of time (1,037 days versus 537 days for the Cocker Spaniels, 329 days versus 50 days for the Doberman Pinschers). Similar findings were seen in a separate study evaluating Doberman Pinschers with congestive heart failure secondary to DCM. Those treated with pimobendan and other medications appropriate for heart failure survived for 130.5 days, while those given placebo plus other heart-failure medications survived for only 14 days. More recently, the PROTECT study provided evidence for the benefits of pimobendan therapy in DCM patients prior to the onset of heart failure. The median time to the primary end point (congestive heart failure or sudden death) was 63% longer in the pimobendan group compared to the placebo group (718 days versus 441 days). The median survival time was also longer for the pimobendan-treated dogs (623 days versus 466 days).

There is very little evidence to support the use of pimobendan in feline patients. As such, its use in cats is off-label at this time. Although it has been shown to be well tolerated, a recent pharmacologic study demonstrated that it can reach serum levels up to four times those seen in dogs with standard doses, and the serum half-life is approximately three times longer than that seen in dogs. While it has empirically been reported to be of benefit for feline patients with systolic dysfunction, the use of pimobendan, or any other positive inotrope, is contraindicated in patients with systolic anterior mitral valve motion, due to the possibility of causing worsened dynamic outflow tract obstruction.

Pimobendan has clear benefits when used appropriately in the treatment of canine patients with cardiac disease. Other uses and applications in these patients, as well as its general use in feline patients, require further evaluation.

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For more information about Angell's Cardiology service, please visit **angell.org/cardiology**. For consults or referrals, please call **617 541-5038**, or e-mail **cardiology@angell.org**. ■

REFERRAL CONTACT INFORMATION

For additional information, please contact Eleanor Cousino, Angell Referral Coordinator, at 617 522-5011, or by fax at 617 989-1635. You may also find our appointment hours at angell.org/hours.

Avian & Exotic Medicine Service
Referral Contact: Stacey Escalante
Referral Line: 617 989-1561
Referral Fax: 617 989-1668
E-mail: avianexotic@angell.org
angell.org/avianandexotic

Cardiology Service
Referral Contact: Rebecca Stlaske
Referral Line: 617 541-5038
Referral Fax: 617 989-1653
E-mail: cardiology@angell.org
angell.org/cardiology

Dentistry Service
Referral Contact: Michael Johnson
Referral Line: 617 524-5643
Referral Fax: 617 989-1636
E-mail: dentistry@angell.org
angell.org/dentistry

Dermatology Service
Referral Contact: Rebecca Stlaske
Referral Line: 617 524-5733
Referral Fax: 617 989-1613
E-mail: dermatology@angell.org
angell.org/dermatology

Diagnostic Imaging
Referral Contact: Radiology Staff
Referral Line: 617 541-5139
Referral Fax: 617 989-1617
E-mail: diagnosticimaging@angell.org
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Emergency/Critical Care Service
Referral Line: 617 522-5011
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angell.org/emergency

Internal Medicine
Referral Contact: Eleanor Cousino
Referral Line: 617 522-5011
Referral Fax: 617 989-1635
E-mail: internalmedicine@angell.org
angell.org/internalmedicine

Neurology Service
Referral Contact: Lisa Canale
Referral Line: 617 541-5140
Referral Fax: 617 989-1666
E-mail: neurology@angell.org
angell.org/neurology

Nutrition
Referral Contact: Eleanor Cousino
Referral Line: 617 522-5011
Referral Fax: 617 989-1635
E-mail: nutrition@angell.org
angell.org/nutrition

Oncology Service
Referral Contact: Stacey Escalante
Referral Line: 617 541-5136
Referral Fax: 617 989-1668
E-mail: oncology@angell.org
angell.org/oncology

Ophthalmology Service
Referral Contact: Rachael Donnelly
Referral Line: 617 541-5095
Referral Fax: 617 989-1647
E-mail: ophthalmology@angell.org
angell.org/eyes

Pain Medicine Service
Referral Contact: Lisa Canale
Referral Line: 617 541-5140
Referral Fax: 617 989-1666
E-mail: painmedicine@angell.org
angell.org/painmedicine

Pathology Service (Clinical and Anatomical)
Referral Contact: Laboratory Staff
Referral Line: 617 541-5014
Referral Fax: 617 522-7356
E-mail: pathology@angell.org
*Sample submission forms and information at angell.org/lab

Surgery Service
Referral Contact: Kim Swank
Referral Line: 617 541-5048
Referral Fax: 617 989-1660
E-mail: surgery@angell.org
angell.org/surgery

350 South Huntington Ave.
Boston, MA 02130
angell.org

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We mail one complimentary copy of our newsletter to each of our referring partners. Please circulate this copy within your practice.

Saturday/Sunday/Evening/ Early Morning Appointments:
Angell offers expanded appointment hours for many services. Please visit **angell.org/hours.**

*Are your clients new to Angell? Send them to **angell.org/directions** for detailed directions to our location. Ample free parking on site.*

We encourage you to e-mail Angell's specialists with questions. We hope you will use Angell as a resource, and we look forward to working with you as we continue our legacy of providing compassion and care for animals.

Main Phone: 617 522-7282
Veterinary Referrals: 617 522-5011

Chief of Staff
Ann Marie Manning, DVM, DACVECC
amanning@angell.org

Avian & Exotic Medicine
Elisabeth Simone-Freilicher, DVM, DABVP (Avian Practice)
esimonefreilicher@angell.org

Cardiology
Nancy Laste, DVM, DACVIM (Cardiology)
nlaste@angell.org

Rebecca Malakoff, DVM, DACVIM (Cardiology)
rmalakoff@angell.org

Rebecca Quinn, DVM, DACVIM
rquinn@angell.org

Dentistry
William Rosenblad, DVM
wrosenblad@angell.org

Dermatology
Klaus Loft, DVM
kloft@angell.org

Meghan Umstead, DVM
mumstead@angell.org

Diagnostic Imaging
Rebecca Manley, DVM, DACVR
rmanley@angell.org

Joan Regan, VMD, DACVR
jregan@angell.org

Steven Tsai, DVM, DACVR
stsai@angell.org

Emergency & Critical Care Medicine
Kiko Bracker, DVM, DACVECC
kbracker@angell.org

Roxanna Khorzad, DVM
rkhorzad@angell.org

Megan Whelan, DVM, DACVECC
mwhelan@angell.org

Internal Medicine
Doug Brum, DVM
dbrum@angell.org

Maureen Carroll, DVM, DACVIM
mccarroll@angell.org

Erika de Papp, DVM, DACVIM
edepapp@angell.org

Jean Marie Duddy, DVM
jduddy@angell.org

Kirstin Johnson, DVM, DACVIM
kcjohnson@angell.org

Shawn Kearns, DVM, DACVIM
skearns@angell.org

Susan O'Bell, DVM, MPH, DACVIM
sobell@angell.org

Neurology
Allen Sisson, DVM, MS, DACVIM (Neurology)
asisson@angell.org

Nutrition
Dana Hutchinson, DVM, DACVN
dhutchinson@angell.org

Oncology
Christine Anderson, DVM, MS, DACVIM (Oncology), DACVR (Radiation Oncology)
cranderson@angell.org

Jennifer Mahoney, DVM, DACVIM (Oncology)
jmahoney@angell.org

Carrie Wood, DVM, DACVIM (Oncology)
cawood@angell.org

Ophthalmology
Daniel Biros, DVM, DACVO
dbiros@angell.org

Martin Coster, DVM, MS, DACVO
mcoster@angell.org

Pain Medicine
Lisa Moses, VMD, DACVIM, CVMA
lmoses@angell.org

Pathology (Clinical & Anatomical)
Patty Ewing, DVM, MS, DACVP
pewing@angell.org

Pamela Mouser, DVM, MS, DACVP
pmouser@angell.org

Surgery
Sue Casale, DVM, DACVS
scasale@angell.org

Michael Pavletic, DVM, DACVS
mpavletic@angell.org

Nicholas Trout, MA, VET MB, MRCVS, DACVS, DECVS
ntrout@angell.org