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Equivocal Thyroid Hormone Results:
What Do They Mean?

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1. KEY FACTS
   a. Hyperthyroidism (FHT) is the most common endocrinopathy of senior cats.
   b. FHT is due to the autonomous oversecretion of thyroid hormones (T₄ and T₃), resulting in
      an increased metabolic rate.
   c. 98% of cases of FHT result from benign adenomatous hyperplasia of the thyroid glands;
      2% results from thyroid carcinoma.
   d. Clinical signs in well-established cases include weight loss, polyphagia, increased
      activity, and polydipsia/polyuria.
   e. A common scenario in practice is to suspect FHT based on weight loss since the last
      wellness examination, the palpation of one or both enlarged thyroid lobe(s), and the
      detection of a new cardiac murmur and resting tachycardia.
   f. The diagnosis of FHT is confirmed by documentation of increased thyroid hormones –
      total T₄ (tT₄), and free T₄ (fT₄) when tT₄ levels are equivocal. When the results of both
      hormone tests are suspicious, but not confirmatory, a low serum TSH level can support
      the diagnosis.
   g. tT₄ levels may be in the high-normal range in early FHT and in hyperthyroid cats with
      concurrent illnesses. fT₄ levels usually are increased in both of these scenarios.
   h. fT₄ levels may be increased in some cats with euthyroid sick syndrome, in the absence of
      FHT. tT₄ levels will be in the low-normal range in these cats, thereby differentiating
      them from cats with FHT. Therefore, FHT should never be diagnosed on the basis of
      increased fT₄ levels alone.
   i. FHT can mask the presence of chronic kidney disease (CKD) by artificially decreasing
      BUN and serum creatinine. Conversely, CKD can cause tT₄ levels to be lowered in cats
      with FHT because of euthyroid sick syndrome. The relationship between thyroid
      function and kidney function is complicated.
   j. Euthyroid goiter (nonfunctional enlargement of the thyroid gland) is being identified with
      increasing frequency in older cats presented for routine wellness examinations. In some
      cats, this change is a precursor to overt hyperthyroidism at a later date.
   k. Oral methimazole is a reversible (non-curative) treatment option for FHT.
   l. Hill’s prescription diet y/d, introduced in 2011, is an iodine-depleted food that is a
      reversible treatment for FHT.
   m. Permanent treatments include ¹³¹I radiation (preferable) and bilateral thyroidectomy.
   n. Cats with FHT who are being considered for a permanent treatment option may need to
      undergo a methimazole trial to evaluate kidney function.
   o. Cats with FHT who also have CKD should be treated with methimazole.
      i. Thyrotoxic substances in the environment (not proven)
         (1) Lining of pop-top cat-food cans
         (2) Fire retardants (polybrominated diphenyl ethers) in fabrics and carpets
2. CLINICAL SIGNS
   a. Age range - 3 to 20 years. Most cases occur in cats >10 years of age
   b. Unilateral thyroid lobe enlargement in 30% of patients, and bilateral in 70%
      i. The normal thyroid lobes are not palpable in cats
      ii. Enlarged lobes vary in size, are mobile, and may be difficult to identify
      iii. Palpate for enlarged lobes along the trachea ventral to the larynx and down to the thoracic inlet
   c. Some owners may interpret clinical signs as favorable changes in their older cats and not report them as problems
   d. Weight loss
      i. May be dramatic in advanced cases with a cachectic appearance
      ii. May have low muscle condition score
      iii. Affected cats may have normal body weight if they were obese at the onset of disease
   e. Polyphagia
      i. May be pronounced early in course of disease but may decrease as disease progresses
   f. Hyperactivity
      i. Night-time wandering and disrupted sleep patterns
      ii. Increased vocalization
      iii. Anxiousness and irritability
      iv. Self-induced alopecia
   g. Tachycardia at rest on examination table (often >200 bpm)
   h. Polyuria and polydipsia
      i. Mechanisms unclear – may be partly psychogenic
      ii. Often have concurrent polyuric diseases such as CKD
   i. Auscultation abnormalities
      i. Cardiac murmur or gallop sound
      ii. Due to thyrotoxic cardiomyopathy
   j. Vomiting
      i. Often occurs after eating voraciously
      ii. Due to gastric hypermotility
   k. Diarrhea and increased fecal volume
      i. Due to intestinal hypermotility and malabsorption
   l. Unkempt greasy haircoat or dry scaly haircoat (seborrhea sicca)
   m. Anorexia
      i. Sign of late-stage disease (apathetic hyperthyroidism)
   n. Polypnea (panting)
   o. Muscle weakness and muscle tremors

3. LABORATORY FINDINGS
   a. Complete blood count
      i. Patients with uncomplicated FHT usually have hematocrits at the high end of normal reference range due to trophic effects of thyroid hormones on erythropoiesis
      ii. Concurrent illnesses should be suspected in cats with FHT and hematocrits at the low end of the reference range
b. Serum chemistries
   i Liver enzymes (ALT and ALP) are increased in up to 75% of affected cats
   ii Blood urea nitrogen and creatinine
      (1) An increased BUN:creatinine ratio may be noted in some cats with FHT (due to polyphagia and muscle wasting)
      (2) Hyperthyroidism can mask the presence or severity of CKD
         (a) Increased cardiac output causes increased renal perfusion
         (b) This results in an artificially increased GFR leading to enhanced excretion of nitrogenous wastes (BUN and creatinine)
         (c) Thyroid hormones also may affect creatinine clearance in the renal tubules
         (d) BUN and creatinine return to real levels once patient is made euthyroid with treatment, revealing true stage of CKD
      (3) Conversely, euthyroid sick syndrome from concurrent CKD can depress tT4 levels in hyperthyroid cats into the upper part of normal range, making diagnosis of FHT more challenging
   c. Urinalysis
      i Specific gravity is usually <1.035
         (1) Probably due to medullary washout from increased GFR and psychogenic polydipsia
         (2) Many affected cats have concurrent polyuric diseases such as CKD
      ii Urine specific gravity cannot be used to determine which cats will become azotemic after 131I treatment or thyroidectomy

4. THYROID FUNCTION TESTING
   a. Serum thyroxine (total T4 [tT4])
      i Screening test of choice for FHT
         (1) Cheap, quick turn-around, and very few false-positive results
      ii Increased value in older cat with appropriate clinical signs confirms the diagnosis
      iii About 40% of cats with early FHT and 10% of all cats with FHT have tT4 levels in the upper part of reference range.
         (1) Concurrent diseases may suppress tT4 into the upper part of reference range in cats with FHT
      iv A second confirmatory test (fT4) should be performed on older cats with suspicious clinical signs and high-normal tT4 values
   b. Free T4 (fT4)
      i Small fraction of total circulating thyroid hormone that is not protein-bound
      ii Metabolically active and enters cells
      iii Higher than reference range in 98% of cats with FHT
         (1) More consistently increased than tT4 in early FHT and in cats with FHT and concurrent illnesses
      iv Should never be used as initial screening test. Only use in cats with suspicious clinical signs and equivocal tT4 results
         (1) About 10% of sick cats have elevated fT4 levels as a manifestation of atypical euthyroid sick syndrome, in the absence of any primary thyroid disease
            (a) These cats have low-normal or low tT4 levels, differentiating them from cats with FHT
c. **T₃ suppression test**
   i. Has been used in cats with equivocal tT₄ and fT₄ results
   ii. Requires measurement of tT₄ before and after 7 oral q8h doses of T₃

d. **Thyroid scan (technetium ⁹⁹M)**
   i. Normal vs. abnormal
   ii. Size of abnormal lobe(s)
   iii. Unilateral vs. bilateral
   iv. Benign vs. locally malignant
   v. Benign ectopic tissue
   vi. Distant metastases

e. **Endogenous serum thyroid stimulating hormone (TSH)**
   i. A specific assay for feline TSH is not available; however, the canine assay (cTSH) may provide useful information in some patients
   ii. The predictive value of the cTSH assay in cats is reduced at the lower end of the reference range (where it is most useful)
   iii. However, a very low or undetectable cTSH level with high normal tT₄ and mildly elevated fT₄ levels in an older cat who is asymptomatic or who has minimal clinical signs except a small palpable thyroid lobe, may provide supportive evidence of subclinical or very early hyperthyroidism

5. **OTHER DIAGNOSTIC TESTS**
   a. **Thoracic radiography**
      i. Indicated for detection of cardiomegaly, metastatic thyroid disease, or unrelated concurrent diseases that may affect treatment options
   b. **Echocardiography**
      i. Indicated for patients with cardiomegaly that may be associated with primary cardiomyopathies (and not due to FHT) and for patients with arrhythmias or gallop sounds
   c. **Systolic blood pressure measurement**
      i. FHT is an important cause of hypertension in cats
      ii. Compared to hypertension associated with CKD, the degree of hypertension in FHT generally is mild
         (1) Overt evidence of target organ damage is uncommon
      iii. Significant hypertension may occur after ¹³¹I treatment or thyroidectomy in cats who were normotensive before treatment
         (1) Blood pressure should be monitored every 2 to 3 months after treatment for the first year to detect this complication
Gastrointestinal Function Tests:
How Do I Interpret the Results?

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The Role of Gastrointestinal Function Tests

Weight loss, decreased appetite, vomiting, and chronic diarrhea are common presenting complaints in feline practice. Unfortunately, the underlying cause of these problems often is not identified through physical examination, hematological and biochemical evaluation, urinalysis, fecal examination, and routine radiography.

Owners may be reluctant to spend significant amounts of money on more advanced diagnostic testing such as abdominal ultrasound and gastrointestinal endoscopy unless there is a high likelihood of obtaining meaningful diagnostic and prognostic information. Similarly, practicing veterinarians would prefer a high degree of confidence that these procedures will be productive when referring their patients and clients to specialists for further evaluation.

The availability of non-invasive tests (serum cobalamin, folate, feline trypsin-like immunoreactivity, and feline pancreatic lipase immunoreactivity) provides the practicing veterinarian with important opportunities to evaluate gastrointestinal function in cats with chronic, vague clinical signs. Test results can guide recommendations about the appropriateness of more invasive and expensive diagnostic testing, and can provide valuable information about locations to examine and biopsy during ultrasound and endoscopy.

Serum Cobalamin

Cobalamin is a water-soluble, cobalt-containing vitamin that acts as a co-factor for several enzymes involved in important metabolic processes in all tissues of the body. Cobalamin initially is derived from bacterial synthesis; however, cats meet their cobalamin needs through the ingestion of herbivore tissues in their food that are rich in this vitamin.

Cobalamin is absorbed from the intestines via receptors on enterocytes in the normal ileal mucosa. After ingestion, free cobalamin is bound to R protein in the stomach, which precludes its absorption. Pancreatic proteases in the small intestine digest the R protein, releasing the cobalamin. Free cobalamin then binds with intrinsic factor, and these complexes are absorbed
through the receptors on the ileal mucosa. Intrinsic factor in cats is produced almost exclusively by the exocrine pancreas; therefore, hypocobalaminemia can occur due to distal or diffuse small intestinal disease or exocrine pancreatic insufficiency.

Serum hypocobalaminemia occurs after body stores of the vitamin become depleted and reflects long-standing intestinal or exocrine pancreatic disease. Serial monitoring of sick cats over time may be necessary to document the condition.

Serum Folate
Dietary folate, a water-soluble B vitamin, is absorbed only in the proximal small intestine of cats. Folate polyglutamate that is ingested in a non-absorbable form is deconjugated to absorbable monoglutamate in the proximal small intestine by folate deconjugase. Severe, long-standing proximal small intestinal disease may lead to low serum folate levels. Proximal small intestinal bacterial dysbiosis may result in increased levels.

Cobalamin is important in the cellular uptake and utilization of folate. Chronic hypocobalaminemia may lead, therefore, to impaired cellular uptake of folate, causing it to pool in the circulation. Serum folate levels may drop when hypocobalaminemia is corrected, unmasking evidence of proximal small intestinal disease that was not detected during initial testing. Therefore, serum folate should be re-tested 4 or more weeks into cobalamin supplementation, especially if serum folate levels initially were in the low-normal range.

Serum Feline Trypsin-Like Immunoreactivity (fTLI)
This assay mainly measures serum trypsinogen in the serum, although some trypsin also is detected. Pancreatic inflammation can enhance the amount of trypsinogen that is released into the vascular space; however, increased fTLI levels are noted in only 30-40% of cats with pancreatitis, presumably because of the short half-life of the molecule.

The earlier the patient is sampled during the course of an exacerbation of pancreatic inflammation, the more likely fTLI levels will be elevated. Other causes of modest increases in serum fTLI include small intestinal disease, advanced chronic kidney disease, and a variety of other pancreatic acinar tissue pathologies.

More importantly, low serum fTLI levels, in combination with low serum cobalamin levels, are diagnostic for exocrine pancreatic insufficiency in cats. However, overt exocrine pancreatic
insufficiency only occurs when greater than 85-90% of exocrine acinar tissue function has been lost.

*Serum Feline Pancreatic Lipase Immunoreactivity (fPLI)*

Serum pancreatic lipase immunoreactivity detects lipase derived specifically from pancreatic acinar exocrine tissue, in comparison to serum lipase which is secreted by a wide variety of tissues thereby decreasing its predictive value in the diagnosis of pancreatitis.

Serum fPLI levels increase in early pancreatic inflammation, but stay elevated longer than serum fTLI levels, making fPLI a much more sensitive test for pancreatitis in cats. Mild increases in fPLI over reference range may be associated with small intestinal disease without overt clinical evidence of pancreatitis.

**Key Therapeutic Points**

While the detection of hypocobalaminemia has important diagnostic implications, correction of the deficiency is essential for optimal response to treatment. Chronic hypocobalaminemia exerts detrimental effects on a wide range of body tissue, impairing metabolic function.

Severe distal small intestinal disease may result in hypocobalaminemia which, in turn, further affects enterocyte function and leads to a vicious cycle of worsening cobalamin deficiency. Failure to correct hypocobalaminemia in cats with chronic small intestinal disease is an important cause of unsatisfactory response to treatments directed specifically at the primary enteropathy.

Non-invasive gastrointestinal function tests provide important diagnostic and therapeutic information about cats with chronic diseases, which often have limited localizing signs. Inclusion of these tests in the evaluation of selected feline patients can help veterinarians to make prudent decisions about further diagnostic testing or about choice of treatments if finances preclude further evaluation.
INTRODUCTION

Blood-sucking arthropods are common causes of disease in pets both directly (e.g., itching, anemia) and indirectly (e.g., pathogen transmission). Mosquitoes, flies, fleas, and ticks are each capable of transmitting a variety of microbes including parasitic worms, protozoa, viruses, and bacteria. Cats are susceptible to a number of vector-borne diseases (VBD). These include heartworm infection, hemotropic mycoplasmosis, leishmaniasis, anaplasmosis, ehrlichiosis, babesiosis, rickettsiosis, hepatozoonosis, bartonellosis, cytauxzoonosis, Q fever, tularemia, plague, trypanosomiasis, and borreliosis.

Of course, not all vector transmitted microbes are potential pathogens, and not all potential pathogens cause disease. Many microbes are well-adapted to a reservoir host and only rarely cause disease in the reservoir species (e.g., Bartonella henselae rarely causes obvious/overt disease in cats). Disease may be more likely when the potential pathogen infects a non-reservoir host (e.g., Cytauxzoon felis causes death in the vast majority of infected domestic cats but often only a mild, short lived illness in the reservoir host, the bobcat).

The mechanism of pathogen transmission varies depending on the pathogenic organism and vector. Ticks feed for extended periods of time in a single site. During the long period of feeding, they not only “suck” blood from the host, but they also “spit” into the host. The salivary fluid inoculated during tick feeding includes anticoagulants, analgesics and antipruritics, immunomodulatory molecules, and pathogens. Many pathogens must be “activated” to move from the tick’s mid-gut to the salivary gland prior to inoculation. Although this movement may take some time, it is unwise to assume that there is always a risk-free period to mechanically remove ticks prior to pathogen transmission. Fleas can regurgitate during feeding, but flea feces (frass) is the more important cause of pathogen transmission. Pruritic animals scratch the dermis thereby helping to inoculate the frass on the skin surface.

Heartworm disease and cytauxzoonosis will be covered in separate presentations at this conference. This talk will focus on a few VBD of cats relevant to practice in the Lone Star State. These include leishmaniasis, hemotropic mycoplasmal, bartonellosis, and Q fever.

Leishmaniasis
*Leishmania* are obligate intracellular protozoan that are important in zoonotic infections world-wide. The organisms is transmitted by phlebotomine bites. *Leishmania infantum*, perhaps the most important of the *Leishmania* spp. in dogs and humans, causes visceral, cutaneous, and mucocutaneous disease. Although cats can become diseased, this species is not endemic in Texas. In Texas, *Leishmania mexicana* is well recognized as an uncommon autochthonous infection. While it is zoonotic, human infection is also uncommon and it is unlikely that cats are an important reservoir for disease. This pathogen has an extreme tropism for the dermal epithelial cells. Nodular, ulcerative lesions are typical, and usually are found on the pinnae or less commonly periorbital region, muzzle, or other areas of the skin. The diagnosis is typically established by histopathology and/or cytology. Hyperkeratotic, hyperplastic ulcerated skin contains amastigotes within the interstitium or in macrophage cells. These stain poorly, and are hard to distinguish from other organisms on histologic exam. Cytology to demonstrate kinetoplasts is ideal in combination with histopathology. There is no commercial polymerase chain reaction (PCR) test [note that Texas A&M does have primers for PCR used in investigational settings]. Serology is of little use for the dermatologic form of infection. Treatment is primarily via excision of affected skin, however, recurrent lesions or spread is possible. Allopurinol has been used as an adjunctive treatment for infected cats, but even with this treatment at least several cats experienced recurrent lesions. Other anti-Leishmania drugs are either toxic for cats or not readily available in the USA, and none are proven efficacious. Prevention is likely key, as for other VBD. In dogs, the Seresto® collar is quite effective in preventing *L. infantum* and is likely to be effective to prevent *L. mexicana* in cats too. Fluralaner is approved for cats, and in dogs at least is effective against the phlebotomine sandflies that transmit infection.

### Hemotropic mycoplasma

Hemoplasmas (haemotropic *Mycoplasma* spp) are gram negative, epicellular bacteria lacking a cell wall. While there are several species that infect cats, the most pathogenic is *M. haemofelis* (formerly known as *Hemobartonella felis*). Found world-wide, fleas are thought to be the predominant arthropod vector although ticks, flies, or other such arthropods might also transmit infection (mosquitoes do not seem to be an important vector). Blood transfusion, fighting behavior, and perinatal transmission are alternative routes of transmission. In addition to these risk factors, retroviral infections FeLV and FIV predispose cats to infection, or at least to
illness from infection. *M. haemofelis* generally causes an acute hemolytic anemia that can be life-threatening or mild. Cats that survive this acute stage often become subclinical carriers with mild anemia, or may have a waxing and waning course of chronic, recurrent anemia. Clinical signs are all related to hemolytic anemia and can include lethargy, weakness, anorexia, fever, pallor, icterus, splenomegaly, lymphadenomegaly, and physiologic heart murmur. Typically anemia is regenerative (variable severity) and is Coomb’s positive. Chemistry abnormalities often include hyperglobulinemia, hyperbilirubinemia, and increased liver enzymes. The most common point-of-care diagnostic test is a simple blood smear, but both false negative and false positives occur. The organism is epicellular not intracellular, and cell attachment is mediated by calcium. Blood collected in EDTA allows the organisms to “fall off” the cells when calcium is chelated thus causing a false negative. It is best to make fresh smears from blood not treated with anticoagulants. Even then, the parasite load is variable and absence of visible parasites does not rule out infection. Stain precipitate, Howell-Jolly bodies, siderotic inclusions, basophilic stippling, or other parasites can be confused for *M. haemofelis* organisms microscopically. PCR is very sensitive, and all blood donors should be screened by PCR before use rather than relying solely on blood smear review. The species *M. hemominutum* is actually more prevalent than *M. haemofelis*, but it is less pathogenic. This, and several other hemotropic mycoplasma, are only likely to cause illness in cats with compromised immune systems. Regardless of the specific pathogen, treatment is doxycycline (or minocycline) as a first line at 5-10 mg/kg BID for 28 days. Fluoroquinolones are an alternative. Generally, it is assumed that treatment improves illness but does not eliminate infection entirely. Infection has apparently been cleared by using first a course of tetracycline followed by a further 2 weeks treatment with fluoroquinolone. Supportive care including blood transfusion plus/minus a short course of prednisolone to suppress immune mediated destructions of RBC may also be required. Prognosis for clinical recovery is fair to good with such treatment, but such recovered cats should not be used as blood donors. And of course, prognosis also depends on co-morbid conditions; a cat that developed *Mycoplasma* related hemolysis because it was immunosuppressed/ill is less likely to have a good outcome than would be an otherwise healthy cat.

**Bartonellosis**

Bartonella are gram negative intracellular bacteria. There are >38 different species in this genera, and they infect many, many species of animals. Those that are best recognized in cats are
B. henselae, B. koehlerae, B. clarridgeiae, B. bovis, B. quintana, B. vinsonii sub. Berkoff. Cats are the established reservoir host for B. henselae and B. koehlerae, and likely for several other species as well. As for other VBD, the reservoir host is often well adapted to the pathogen. Cats with exposure to fleas, thought to be the likely primary means of transmission for Bartonella, are very commonly infected (up to 98% in several prevalence studies from warmer climates such as Brazil, and 0% in Norway, with ~30% on average for temperate climes). However, overt clinical disease is rare in the species. Experimental infection of cats with B. henselae can cause transient mild fever, lethargy, lymphadenomegaly, and rarely CNS signs, reproductive failure, or anemia. Naturally infected cats have a higher leukocyte count and globulin than uninfected cats. Occasionally, myocarditis, endocarditis, or pyogranulomatous inflammation follow infection. And yet, most infected cats remain well. The greatest import for B. henselae is as a zoonotic pathogen rather than as a cause of feline disease.

Humans with bartonellosis can develop a variety of illnesses ranging from subclinical to self-limiting to fatal in outcome. The most common illness in immunocompetent people is “Cat Scratch Disease” (CSD). CSD results in variable mild fever and regional painful lymphadenomegaly that usually resolves over many months. Antimicrobial therapy does not speed recover, and organism is usually not viable leading to the proposal that an immunologic reaction to the pathogen causes disease. Bacillary angiomatosis is a more severe and often fatal form of illness related to B. henselae infection of immunocompromised people. A large variety of other manifestations of human infection has been documented including cancer and neurologic disease. Children are more likely to develop CSD than are adults, perhaps because they are more likely to play with cats in a way that leads to scratches. Kittens are more likely to be bacteremic than adult cats, and more likely to scratch and bite as well, meaning that kittens pose a greater risk of zoonosis than adult cats. Infection can be transmitted by dogs as well as cats, and human infection does not have to be preceded by a known scratch or bite. It is believed it is not the cat that causes infection directly, but rather the cat acts as a reservoir for fleas and pathogen, and that a scratch/bit inoculates flea frass mechanically.

Options for diagnostic testing include serology, specialized blood culture with cell lysis or tissue culture, and a variety of PCR tests, including PCR testing after enrichment on a specialized media. All of these methods have major pitfalls, but enrichment PCR is likely the most sensitive and specific option (culture is considered the most specific method, but sensitivity
is poor). Overt illness in cats due to bartonellosis is uncommon, but if no other explanation exists for clinical signs such as fever, testing may be worthwhile despite the pitfalls. In such a cat, treatment would be warranted if there is a positive PCR or a positive culture. Diagnostic testing is necessary for potential blood donor cats, with negative PCR as a minimal standard for donor use and negative serology, PCR, and culture as the optimal standard suggested in the ACVIM Consensus Statement on screening blood donors (JVIM 30:15-35, 2016).

No known treatment clears the pathogen. For the rare cat with clinical illness believed to be due to bartonellosis, a combination of a fluoroquinolone and doxycycline for 28 days is often suggested. Due to rapid resistance, use of azithromycin is not recommended. Such cats should still not be used as blood donors. Healthy cats that are rejected as blood donors because of a positive test result do not require antimicrobial treatment simply because of the positive test result.

Sometimes, pet owners come to veterinarians with a request to test a healthy cat because of a diagnosis of CSD in a person in the home, or because there is an immunosuppressed person. Although there is some disagreement among experts, this is generally NOT warranted. A cat that tests negative may still have harbored the pathogen, and a cat that tests positive will often self-resolve high level bacteremia as the kitten/cat ages. As mentioned, there are a multitude of pitfalls of testing that can lead to false positive and negative tests, and the cat itself is not usually the reason for infection as much as are the cat’s fleas. Furthermore, there is no treatment known to eliminate the pathogen. Instead of testing, the owner should be educated regarding preventive measures for future exposure. This includes religious use of ectoparasites control products to eliminate fleas (and ideally keeping the cat indoors), avoiding rough play, trimming the cat’s nails or using SoftPaws nail covers, and washing any scratches or bites well with soap and water. For immunocompromised persons, it may be wise to adopt cats rather than kittens as they are less likely to 1) scratch/bite, and 2) have a high level bacteremia. Also, choosing cats from sources such as a breeder or a friend where flea exposure is less likely than from an animal shelter cat may be wise. I should point out that this opinion is not held uniformly, and a few experts would recommend testing and even antimicrobial administration for healthy, positive cats that live with immunocompromised persons or very young children. Often, veterinarians know far more about the zoonotic risk of disease than do physicians, and it may be helpful to ask
that the owner’s physician get in touch either with you, with a veterinarian with expertise in infectious disease, or with a physician specialist in infectious disease.

**Q fever**

Caused by *Coxiella burnetii*, Q fever is more of an issue for livestock (especially small ruminants) and humans than for cats, although cats are certainly susceptible to infection. The bacterium is found at high concentrations in the birth products of infected ruminants as well as in the urine, feces, and milk. It is also found in the dust from pastures contaminated with these fluids. It is a VBD because it can be transmitted by tick bite, especially the brown dog tick *Rhipicephalus sanguineus*. Non-vector transmission seems to be more common, however, as a source of human exposure to this zoonotic pathogen. Farm cats are likely exposed via shed placenta or aborted fetuses, but infection of cats from non-rural areas are also reported. Most infected cats remain healthy. Lethargy, fever, an anorexia can occur. In intact queens, abortion and still births are common. Pregnancy loss and parturition are important routes of zoonotic transmission to humans because of the exposure to the products of conception from a pet cat that experiences abortion (always wear gloves and protective clothing when handling cats or dogs experiencing abortion…don’t forget brucellosis is also zoonotic), or when trying to resuscitate weak kittens at parturition. Culture of the bacteria is possible but not advised unless biosafety level 3 facilities are available. Diagnosis in cats is usually based on a fourfold increase in IgG on paired serum samples, but there can be cross reaction with *Bartonella* genera organisms. Treatment for cats is not well described. Tetracyclines, fluoroquinolones, and chloramphenicol are the treatments of choice for Q fever in humans and are likely effective for cats.

**Prevention of Vector Borne Disease**

Very few vector borne infections have available vaccines for disease prevention. For most VBD, the best prevention is prevention of ectoparasites. Even in the case of heartworms this may be true, with increased emphasis on prevention of mosquito bite in addition to simply killing the immature parasite once transmitted.

Flea and tick prevention should be used on all cats year round. Not all products have equal efficacy on all arthropod vectors, and even those effective for ticks do not possess equal efficacy against all species of ticks. For example, the Lone Star Tick (*Amblyomma americanum*) is especially difficult to kill. A variety of considerations should apply when recommending these products to pet owners, including:
• Safety for use on cats (many dog products are very toxic to cats)
• Spectrum of efficacy
• Repellency
• Speed of Kill
• Owner Compliance
  ▪ Ease of application
  ▪ Frequency of application
  ▪ Aesthetic considerations
  ▪ Cost
• Efficacy throughout treatment interval

At present, there is no oral product available for use in cats. Collars and topical products are readily available but must be applied correctly and at the correct time. Please see https://capcvet.org/parasite-product-applications for product details.

Vectors and Diseases They Transmit

<table>
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<th><strong>Lone Star ticks</strong></th>
<th><strong>Brown dog ticks</strong></th>
<th><strong>American Dog ticks</strong></th>
<th><strong>Black legged ticks</strong></th>
<th><strong>Soft bodied ticks</strong></th>
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<tr>
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<td>Canine monocytic ehrlichiosis</td>
<td>Rocky Mountain spotted fever</td>
<td>Lyme disease</td>
<td>Tick borne relapsing fever</td>
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<td>Q fever</td>
<td>Cyclic thrombocytopenia</td>
<td>Bartonellosis</td>
<td>Granulocytic anaplasmosis</td>
<td></td>
</tr>
<tr>
<td>Tularemia</td>
<td>Rocky Mountain spotted fever</td>
<td>Tularemia</td>
<td></td>
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Suggested Readings

CDC.gov (Q fever, Bartonellosis, Leishmaniasis)


Cytauxzoonosis
Leah A. Cohn, DVM, PhD, DACVIM (SAIM)
Professor, University of Missouri

_Cytauxzoon felis_ is a protozoal organism transmitted by ticks that causes potentially fatal illness in domestic cats. _C. felis_ infects only domestic and wild cats, with no age or sex predisposition. Outdoor cats with tick exposure in endemic areas are at risk. Specific risk factors include urban-edge habitats and close proximity to wooded or unmanaged areas. It is common for multiple cats from the same household or neighborhood to become infected.

_C. felis_ infection has been reported in the south central and southeastern United States, but its range appears to be expanding north and east, corresponding to changes in distribution of the tick *Amblyomma americanum*. Most cases occur between March and September, with a peak incidence between March and June and a second wave of infections occurring in August and September.

The natural host is thought to be the eastern bobcat (*Lynx rufus rufus*), which develops a mild or subclinical infection compared to the rapidly progressive and usually fatal disease seen in domestic cats. The organism is transmitted by _A. americanum_ (suspected predominant vector) or _Dermacentor variabilis_ ticks during feeding. Sporozoites released from the tick salivary glands infect macrophages. Asexual reproduction occurs within the host macrophage during the schizogenous phase, causing infected cells to grow to enormous size (≥250 microns in diameter). These schizont-laden macrophages then occlude arterioles, venules, and capillaries, causing organ failure and clinical illness. When the schizonts rupture, merozoites are released to infect erythrocytes. Merozoites are minimally pathogenic but may cause initial hemolysis. Healthy, recovered cats can harbor erythrocyte piroplasms for years; they can transmit the pathogen to ticks during feeding.

Domestic cats usually develop the disease, acute cytauxzoonosis (classic severe illness), about 2 weeks after the bit of an infected tick. Recovered cats (and a few that are acutely infected) harbor the pathogen without illness. These chronic carrier cats may be discovered to
have incidental erythroparasitemia. Importantly, recovered domestic cats can act as reservoirs of infection.

Clinical signs of illness are acute and nonspecific and include acute onset of anorexia, lethargy, dyspnea, icterus, and pallor. Cats may be reported to seem as if in pain and often the third eyelid is elevated. Affected cats are usually febrile (103°F-107°F [39.4°C-41.7°C]), but hypothermia is seen in moribund cats. Icterus and/or pallor are common, as is elevation of the nictitans. Abdominal palpation reveals splenomegaly and hepatomegaly. Tachypnea, tachycardia, altered mentation, vocalization, seizures, and coma can be seen in the later stages of disease. Most cases exhibit a rapid course, with death occurring within 1 week of onset of signs if left untreated.

Diagnosis is usually through blood smear examination when a clinical suspicion exists. Although most often diagnosed by microscopic identification of piroplasms in red blood cells (RBCs) on blood smear, illness can occur before appearance of piroplasms, and piroplasms may be seen in low number in chronic carriers. On the other hand, identification of schizont-laden macrophage on the feathered edge of a blood smear or by cytology of fine-needle aspirates is pathognomonic for disease. Pleomorphic (round, oval, anaplasmoid, bipolar [binucleated], or rod-shaped) organisms in RBC are often seen in high numbers; the round and oval piroplasm forms are most common (0.8-2.2 microns in diameter; typical erythrocyte diameter ≈8 microns). Infected macrophages may occasionally be seen on the feathered edge and may be mistaken for platelet clumps at low power.

If cytauxzoonosis is suspected but not immediately detected by examination of a blood smear, tissue aspirates of lymph node, liver, and spleen are indicated to identify infected macrophages. These cells range in size from 15-250 microns in diameter, typically have a large distinct nucleolus, and their cytoplasm is filled with numerous small (1-2 micron) basophilic particles (i.e., developing merozoites).

CBC findings include pancytopenia (normocytic, normochromic, nonregenerative anemia, leukopenia, and thrombocytopenia), but monocytopenias or bicytopenias may occur. Serum biochemistry profile often demonstrates elevated liver enzymes (frequently lower than expected
for the degree of hyperbilirubinemia), hyperbilirubinemia (mild to moderate), and
hyperglycemia. Azotemia is rare. Urinalysis reveals bilirubinuria. Coagulation testing may be
consistent with disseminated intravascular coagulation (DIC)

Imaging studies do not contribute directly to the diagnosis. Abdominal imaging:
splenomegaly and hepatomegaly common. Thoracic radiographs: ± pleural effusion, pulmonary
infiltrates

Polymerase chain reaction (PCR) testing can confirm infection before appearance of
schizonts or piroplasms but will also be positive in chronic carriers. This send-out test means
result often come “too late” to change the course of treatment, even if only delayed by a few
days. Necropsy with histopathology can confirm the diagnosis and is usually how
cytauxzoonosis is first recognized in regions that were previously considered nonendemic.

New treatments with antiprotozoal therapy have proved effective along with supportive care
in this disease, which was previously considered universally fatal. Because the disease
progression is very rapid, specific treatment should be instituted immediately in suspected cases.
Minimal stress and handling is recommended; early placement of nasogastric tube may facilitate
administration of medication and nutrition with less stress. Crystalloid fluids are provided to
correct dehydration, restore intravascular volume, and maintain perfusion. In anemic animals,
oxygen delivery to tissues must be restored with a transfusion of whole blood (20 mL/kg IV
administered over 4 hours) or packed RBCs (20 mL/kg IV administered over 4 hours)). Some
clinicians treat/prevent DIC with heparin 100-300 IU/kg SQ q 8h or 18 IU/kg/h IV constant-rate
infusion (CRI). Animals with respiratory compromise may require supplemental oxygen or
thoracocentesis if pleural effusion is identified. Analgesia is recommended. The author prefers
Buprenorphine 0.01-0.02 mg/kg SQ, IM, or IV q 6-12h prn over meloxicam. While the later can
reduce fever, this is not always a good thing in my experience. Placement of a feeding tube eases
administration of drugs and provision of nutritional support.

Antiprotozoal therapy should be started immediately. The preferred option is Atovaquone
15 mg/kg PO q 8h administered with a fatty meal for 10 days combined with azithromycin
10 mg/kg PO q 24h for 10 days; this treatment is associated with survival rates of approximately
65%. The treatment is facilitated by placement of a feeding tube. Atovaquone is very viscous and therefore difficult to accurately dose. Fill the dosing syringe a few minutes before administration to allow the drug to settle in order to be sure that the entire dose is present (the thick drug can coat the sides of the syringe causing inadvertent underdosage).

There are a few alternative antimalarials. One such drug is Imidocarb dipropionate (Imizol) 3-5 mg/kg IM then repeated in 7-14 days. Pretreat with atropine 0.04 mg/kg SQ to minimize cholinergic side effects. Survival with imidocarb treatment ≈25% although some claim higher success rates. Another option is Diminazene aceturate (Ganaseg) 2 mg/kg IM repeated in 7 days. A small case series showed this to be a promising treatment, but it is not approved by the U.S. Food and Drug Administration (FDA) or available in the United States. Recently, the combination antimalarial Coartem was about 45% efficacious in a prospective study of naturally infected cats. The advantage of coartem is ease of administration for this pill with a course of treatment completed in three days. Doxycycline 5 mg/kg PO q 12-24h × 14 days may be considered as treatment for co-infection with other tick-borne pathogens.

Without aggressive treatment, 95% of cats will die within a few days. Even with early diagnosis and atovaquone/azithromycin treatment, many cats will die within a day or two. Survivors may have a difficult course with severe clinical disease that lasts for 5-7 days before finally improving. The likelihood of survival varies regionally. In some areas, incidental discovery of erythroparasitemia is common (indicating that the cat survived acute infection), and in other areas, survival is rare even with aggressive treatment. All clinical, hematologic, and biochemical abnormalities resolve in 4-6 weeks. Chronic infection does not appear to be associated with decreased life span. Recurrent severe infections appear extremely rare in surviving animals, suggesting some degree of protective immunity, but re-infection is possible. Hypothermia, icterus, severe anemia, and seizures warrant a poor prognosis, and euthanasia should be discussed as a possibility for the most severely affected cats.

In endemic areas, an effective acaricides and tick repellent should be used to prevent exposure to ticks. Both the Seresto collar (Bayer) and Revolution Plus (Zoetis) have been demonstrated to help prevent infection. The author has personally treated many cats that received
other types of ectoparasites control. Because infections have occurred despite acaricides
treatment, keeping cats indoors is strongly recommended.

**Suggested Readings**

Feline Infectious Peritonitis
Leah A. Cohn, DVM, PhD, DACVIM (SAIM)
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Feline infectious peritonitis (FIP) is a typically fatal disease of cats often characterized by body cavity effusions, icterus, neurologic signs, or uveitis. It is caused by a common feline coronavirus (FCoV) that in clinically affected cats has spontaneously mutated from a benign, minimally pathogenic virus to an aggressive, lethal virus. FCoV is spread via a fecal-oral route, and is ubiquitous in the environment. Cat-to-cat transmission of the mutated FIP virus does not occur under natural circumstances; instead, cats contract ordinary FCoV and it will either mutate inside the cat to cause FIP, or it will not. Cats can either stop shedding FCoV or may be persistent shedders.

FIP can occur in all Felidae family members. Young cats during post-weaning periods are most susceptible (peak age, 3 months to 2 years), but cats of all ages can be affected. It is more common in purebred cats, likely at least in part because they often come from multi-cat environments. Genetic predisposition is suspected but specific genetic susceptibility is not known. Risk factors include young (or old) age, multi-cat environments (catteries, shelters, pet-hoarding environments), stressful situations, and immunosuppression.

Historically, there have been two clinical forms of FIP: dry form (non-effusive) and wet form (effusive). The most important difference in the forms is that the wet form is much easier to recognize than is the dry form. As a general rule, however, the wet form occurs at a somewhat younger age and progresses more rapidly than the dry form. Every cat with body cavity effusion also has microgranulomas, and cats with the dry form will commonly develop effusion later. Therefore, the differentiation is really one describing clinical presentation and not a difference in pathogenicity.

Signs of FIP are often vague, and typically include lethargy, hyporexia, and weight loss. Abdominal distention due to abdominal effusion or respiratory distress due to pleural effusion are common, especially in younger kittens. Gastrointestinal (GI) signs are common, and neurologic signs from central nervous system (CNS) involvement also occur. Physical examination often reveals fever, abdominal fluid wave, and/or tachypnea and muffled lung and heart sounds due to pleural effusion. Uveitis, icterus, or CNS deficits (ataxia, altered mentation,
nystagmus, etc) are sometimes identified, as may be lymphadenopathy, organomegaly, or irregular kidneys.

There are classic findings suggestive of FIP on routine laboratory testing. These include lymphopenia, neutrophilia without left shift (stress leukogram), mild nonregenerative anemia of chronic inflammation, and increased total protein/total solids on CBS. Serum biochemistry profile often demonstrates hyperglobulinemia (and low albumin/globulin ratio) and increased bilirubin. Serum protein electrophoresis is not usually performed, but should demonstrate a polyclonal gammopathy. Fluid analysis from effusion is usually straw-colored and viscous, with high protein (often > 3.5 mg/dL) but low cell counts. The fluid is not septic. A simple and yet useful test is the Rivalta test. Simply add 1 drop effusion to water-acetic acid mixture, watching for coagulation (lava lamp look); sensitivity 91%, specificity 65%. If the Rivalta test is negative, wet FIP is extremely unlikely. Cerebrospinal fluid (CSF) analysis can be unremarkable or may demonstrate cytoalbuminologic disassociation (ie, high protein with low cell count). In addition to lab testing, imaging studies can also be suggestive, be that by identifying fluid accumulations or granulomas in parenchymal tissues.

The only way to definitively confirm a diagnosis of FIP is via immunohistochemical demonstration of FCoV antigen in macrophages, but this requires tissue biopsy or necropsy. An alternative method is detection of FCoV antigen in macrophages collected from CSF, tissue aspirates, or aqueous humor (but not blood) by immunofluorescence or immunocytoLOGY staining; this can result in false positives but has a rather high specificity (72%) and sensitivity (85%). There exist several PCR tests for FIP, some of which are better than others but none are perfect. PCR to detect specific mutations in the spike protein that leads to FIP-inducing FCoV reportedly has a very good specificity (96%) and fair sensitivity (69%) for effusion but it is not useful for blood. Routine PCR for FCoV is not useful. Serum antibody tests for FCoV have very little use as they only demonstrate exposure to a coronavirus (including FIP vaccine virus), not FIP, and most corona antibody titer positive cats will never develop FIP. Additionally, the text can’t be used to rule out FIP as about 10% of infected cats do not develop antibodies or stop making antibodies when they are ill from FIP. Realistically, a kitten from a multicat environment with typical history, exam, and routine lab tests can be comfortably diagnosed as having FIP. Cats without effusion or that don’t fit the norms of age, exposure, etc, are more problematic.
Differentials should be ruled out as best as possible (eg, toxoplasma testing as a differential for CNS deficits) with consideration of biopsy or antibody testing.

To date, FIP is considered incurable and fatal with rare exceptions of spontaneous recovery. The treatment goal is to provide comfort and some additional time. For cats with effusion, centesis may improve clinical signs, and especially improve respiratory compromise. Traditional therapy is immunosuppression, usually with glucocorticoids. Prednisolone 2 mg/kg PO q 24h, which may be reduced if the cat is stable for a few months, plus or minus cavitary injections of dexamethasone are typical. Immunomodulating drugs have been used with little efficacy. Feline interferon-omega showed no efficacy in a placebo controlled trial. In a case report, human interferon-alpha 30 U/CAT PO q 24h on alternating weeks was used with no obvious ill effects, but there are no proven benefits. Polyprenyl immunostimulant was able to prolong life of a few cats with FIP without effusion in a case series but controlled studies are missing. Readily available antiviral drugs show no benefit and several (eg, ribavirin) cause severe adverse effects. Supportive care is important, such as provision of adequate nutrition, antibiotics for secondary infections, or treatment of uveitis.

Recently, there is more reason to hold out hope for recovery. A new antiviral drug now called GC376 has resulted in some apparent cure for cats with FIP (Pedersen 2018). This is a 3C-line protease inhibitor antiviral drug that resulted in improvement in 19/20 naturally infected cats. Of these, 5 kittens with wet FIP were in remission for anywhere from 5 to 14 months, with at least one cat apparently cured two years after starting treatment. This drug is not yet FDA approved, but the approval process has begun. The same authors also published in vitro and in vivo efficacy studies of a nucleoside analog GS-441524 small molecule viral inhibitor. In 10/10 kittens experimentally infected, treatment result in apparent cure. In a study of that same compound in naturally infected cats 26 with wet form, 5 with dry), an impressive 18 were apparently cured with one round of treatment, another 5 with a second round, and 2 more with a third round. Only 6 of 31 cats died of FIP, and 5 of those died within days so likely too quickly for anything to have worked. While some people “buy” this compound from China, the safety and efficacy is of black-market unapproved drug is questionable at best.

Prevention of FIP would be ideal. Unfortunately, the vaccine, while safe, appears to have limited efficacy. Strict hygiene and reduction of stress can be helpful. Theoretically, removing continuous or high FCoV shedders would helpful but it is difficult to identify such cats. If
catteries have only FCoV antibody titer negative healthy cats, it demonstrates that they have likely not been exposed to FCoV, which in turn would make the risk of FIP extremely small. There is no need to handle cats sick with FIP as if they are contagious to other cats.

**Suggested Readings**


Feline Leukemia Virus Screening and Diagnostic Testing
Leah A. Cohn, DVM, PhD, DACVIM (SAIM)
Professor, University of Missouri

Feline Leukemia virus (FeLV) is an oncornviridae Gammaretrovirus of cats. Exogenous FeLV is the virus that causes disease. It is competent for replication by taking over a host cell, where the viral single stranded RNA becomes integrated into the host’s own DNA. This allows the virus to make new virus that can be shed in the saliva, feces, milk, and found in the blood. The vast majority of transmission is horizontal, via close contacts such as a cat grooming her kittens. The other type of FeLV is endogenous FeLV. Endogenous FeLV is actually replication-defective bits of provirus that are remnants of ancient exogenous viruses that have become integrated into the feline genome. All cats have these bits of FeLV in their genome, and they don’t cause disease on their own. It is possible in a cat with exogenous FeLV (the virus) for the endogenous bits (in the genome) to recombine together and result in either protection from or worsening of disease, but without exogenous FeLV, endogenous FeLV does NOT cause illness. It can, however, impact diagnostic testing when it is detected in certain kinds of PCR tests. This discussion will focus on exogenous FeLV unless stated otherwise.

When an uninfected cat is exposed to an infected cat shedding the exogenous virus, there are several potential outcomes. The most basic possible outcomes are that the naïve cat can remain uninfected, or can become infected. Infected cats may or may not become ill. If no infection ensues after exposure, it is described as an “abortive infection” because the exposed cat stopped the virus before it could ever become established. Cats with an abortive infection will not become ill and cannot spread the virus – they are essentially as if unexposed. If the exposed cat becomes infected but is able to contain the viremia, it is described as a “regressive infection”. If the cat becomes infected and the viremia is not contained, it is a “progressive infection”. Factors such as the cat’s age at exposure, intensity of exposure, prior vaccination, the cat’s immune status, and others, all affect the outcome of the exposure and the course of infection. Young kittens are by far the most likely to develop progressive infection while vaccinated cats are more likely to have an abortive infection, for example.

Cats with progressive infection become ill. The illness is often a secondary infection, but might be cancer or anemia, or any of several other conditions (neurologic signs, reproductive failure, etc). These conditions often respond well to treatment, and cats with FeLV progressive infection can live for years if provided with good health care. The old thinking that 30% of FeLV infected cats will die each year after infection is likely not true now, but there is no question that progressive infection can lead to death. These progressively infected cats are the source of virus to infect naïve cats and spread the virus, and so they should be kept indoors and away from naïve cats (relative isolation also protects the infected cat from secondary pathogens).

Regressively infected cats are able to contain the virus but the virus stays dormant in the body. In most cases, it remains dormant for the cat’s entire lifetime and causes no illness. There is some evidence that even cats with regressive FeLV infection may be at increased risk of lymphoma. It is also possible that the dormant virus become reactivated and the cat moves from
the regressive to the progressive state. When you see a cat that was tested negative as a kitten but now has FeLV related disease at 10 years later, after a life-time of indoor living and no exposure, it is probably a cat that moved from the regressive to the progressive state of infection. There is recent evidence that at least on occasion the opposite can occur too, with cats moving not only from regressive to progressive but also from progressive to regressive infection. Regressive infection is not transmitted to other cats as long as it stays regressive.

Focal infections are not well understood and most were described prior to the advent of the currently used molecular tests. These cats have virus that remains localized to only a specific tissue, such as the mammary gland. While the cat is unlikely to become ill, a focal mammary infection could pass the virus on when kittens nurse.

There is no single test that can be run at a single time point that can define a cat’s true FeLV infection status. The AAFP has a set of guidelines on screening and testing, and these will be updated soon. The current guidelines call for testing of all sick cats regardless of prior test results, all cats that are soon to be adopted, all cats with a risk of exposure more than 30 days prior, and all cats prior to FeLV vaccination.

Screening tests should be sensitive to find all the infected cats. Remember that there is no perfect test. Even tests with very, very good sensitivity and specificity will have false results. Sensitivity looks only at infected animals, and is the proportion of infected patients that test positive. Specificity looks only at uninfected animals, and is the proportion of uninfected animals that test negative. What is much more important than population proportions to you as a practicing veterinarian is the accuracy of the test, and what the test result means for an individual cat. You want to know the positive predictive value (PPV), or “How likely is a an animal with a positive test to be infected?” You also want to know the negative predictive value (NPV), or “How likely is an animal with a negative test to be uninfected?” This depends on the prevalence of infection. If prevalence is low, the PPV will always be worse than if the prevalence is high. Even though the most commonly used screening tests have sensitivity and specificity of ~98%, there will be false tests. The prevalence of FeLV infection in all cats is about 2%. On the other hand, the prevalence in cats with thymic lymphoma is more like 92%. This means that you are justified to believe the positive screening test in a cat with lymphoma while you must confirm the positive test in a cat “off the street”.

There are several options for screening and testing cats. These include serology, polymerase chain reaction (PCR), and viral isolation. Viral isolation is rarely performed and is primarily a research technique. Serologic tests that detect the p27 viral antigen are almost always the first line for screening and diagnosis. Serologic testing options include immunochomatographic point-of-care (POC) tests such as ELISA, and immunoflourescentantibody (IFA) tests used for disease confirmation.

The POC p27 tests detect soluble viral capsid antigen. The test becomes positive 21-60 days after infection, and reflect virus in the sample but not necessarily in the cells (that is, virus might be free in the blood). Whole blood is the best test sample although plasma and other fluids can be used; samples from multiple cats should not be pooled as this reduces sensitivity for a screening
test. The most commonly used of these tests are the SNAP, Witness, Anigen, and VetScan. Publications compare the tests from various manufacturers, and all are slightly more specific than they are sensitive. A cat exposed to an infected cat might test negative before 60 days, and will continue to test negative if the infection is abortive. If the cat develops a regressive infection it will become positive for a while, but will then revert to negative status. This is the reason for the long held recommendation to re-test cats that test positive on an initial POC screen either with the same test 6-8 weeks later, or by another methodology such as IFA. Once a progressively infected cat tests positive on a POC test, it is expected to remain positive.

The IFA is a mail out confirmatory serologic test that detects p27 inside the cells, therefore it is only positive after the virus is integrated into the bone marrow progenitor cells. It takes longer after infection to become positive than do the POC p27 tests. IFA can be run on whole blood or on tissue such as bone marrow, but not on plasma, saliva, or the like. The test is less sensitive than the POC p27 serology. Cats with either abortive or regressive infection are expected to be negative at all times, while cats with progressive infection convert from negative to positive and remain positive. There is a period of time during which the POC test can be positive but the IFA has not yet converted, so while a positive IFA after a positive POC can confirm infection, a negative IFA after a positive POC cannot rule out infection. Once a cat is IFA positive, it should always be IFA positive.

There are many different types of PCR testing that each tell you different things. Most importantly, there are PCR tests for proviral DNA and PCR tests for viral RNA. A viral RNA test detects viral RNA and viremia; this is not readily available in commercial labs and has little advantage over POC serology. The test that is offered commercially by several labs is a rt-PCR for proviral DNA that only occurs in the exogenous virus (endogenous proviral DNA complicated development of PCR tests, but is not detected in the commercial tests). Recognizing that FeLV is an RNA virus, this test looks for the virus after it has integrated itself into the host DNA and is there for good. Theoretically, a cat should never convert from proviral DNA PCR positive to PCR negative. Cats with abortive infections stay negative always, while cats with both regressive and progressive infection will stay positive forever. PCR is not a perfect test. While some 5 to 10% of cats with a negative POC serology were found in one study to be rt-PCR positive, other cats that are positive by POC tests persistently have been rt-PCR negative. The moral again is that there is no perfect test! The sample quality may be poor, the strain may be atypical, or there may simply be a low quantity of DNA in the sample causing a false negative.

Proviral DNA rt-PCR testing is the only way known to detect a regressive infection in a healthy cat. This is a crucial bit of information for blood donors. Cats with regressive infection not only can pass on the virus via blood transfusion, but the recipient cat can develop progressive infection and disease. Since most cats that require blood transfusion are at least temporarily immunosuppressed, this is crucially important. ALL BLOOD DONOR CATS SHOULD BE SCREENED FIRST WITH A POC TEST, THEN WITH PROVIRAL DNA PCR.
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<th>REGRESSIVE</th>
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**Suggested readings**

Feline retrovirus management guidelines.  
Feline lungworms

Parasitic pulmonary disease is caused by both lungworms and non-lungworms. Some intestinal worms, especially *Toxocara* (roundworms) but also *Ancylostoma* (hookworms), undergo pulmonary migration before the adult worm reaches its final destination in the intestine. Usually, pulmonary migration causes little disease and few (if any) respiratory signs. However, massive larval migration can result in both direct and indirect inflammatory damage to the lung, causing verminous pneumonia. In contrast to intestinal worms that migrate through the lungs on their way to a final destination, for lungworms the final destination is the respiratory tract. These parasites can reside primarily in the pulmonary parenchyma, in the airways, or both. Parasitic lung disease easily can be confused with other conditions such as bronchopneumonia, eosinophilic pneumonia, asthma, pulmonary granulomatosis, or even pulmonary neoplasia. Unfortunately, intermittent fecal shedding of parasite ova or larvae after expectoration means that fecal examination is an insensitive diagnostic method. For this reason, therapeutic trials often are employed when lungworms are suspected. High-dosage fenbendazole (50 mg/kg PO q 24 h for 10-14 days) or ivermectin usually are used.

*A. abstrusus* is a common feline lungworm found throughout the world; in the United States, it is most common in northeastern states. Although most infected cats remain well, infection can produce clinical signs that mimic feline bronchopulmonary disease. Mature worms reside in the bronchioles; inflammation of these small airways can result in cough, wheezing, and/or respiratory distress. Thoracic radiographs of parasitized cats can appear unremarkable or can demonstrate a diffuse interstitial nodular and/or peribronchiolar pattern, or sometimes an alveolar pattern. In endemic regions, *A. abstrusus* should be considered an important differential diagnosis for feline “asthma,” especially in cats with outdoor exposure, which are more likely to ingest the mollusk intermediate host of the parasite. Diagnosis is based on detection of larvae in either airway lavage samples or the feces via Baermann sedimentation. Baermann is more sensitive than fecal flotation by several fold. A very sensitive and specific diagnostic PCR using either feces or pharyngeal swab material has been developed but is not currently commercially available. A point-of-care test is currently in development. Fenbendazole (25-50 mg/kg PO q 24 h for 10-14 days), ivermectin (300-400 mcg/kg SC), or selamectin (6 mg/kg applied topically) can be used for treatment. Oral or inhaled antiinflammatory dosages of glucocorticoids can be useful during therapy, as can bronchodilators for cats with increased respiratory effort.

Feline bronchitis

Although terminology describing feline asthma and bronchitis is often used interchangeably, including terms such as asthmatic bronchitis, the disorders may be distinct. In human medicine asthma and chronic bronchitis are thought to be distinct conditions with considerable clinical overlap, and one person can have both conditions. Chronic bronchitis in people often occurs in conjunctions with emphysema, with the combination referred to as Chronic Obstructive Pulmonary Disease (COPD). Unlike in asthmatic people, airflow limitation due to COPD is poorly responsive to bronchodilators. Like humans, cats can develop either allergic asthma or non-allergic chronic bronchitis. Non-allergic mechanisms are likely the cause of chronic bronchitis in both cats and people. Feline bronchitis is poorly understood, but might be the result
of a previous insult (e.g., inhaled irritant, infection) initiating a self-perpetuating neutrophilic inflammatory response in the airways. Both allergic asthma and non-allergic chronic bronchitis result in similar clinical signs of cough and wheeze. However, as is the case in people, asthma is more likely to cause episodic muscular bronchospasm with airflow limitation and to respond better to bronchodilator therapy than is non-allergic bronchitis.

Thus far, the only clinical distinction between bronchitis and asthma in cats relates to the predominant cellular make up of BALF. In allergic asthma, >17% of BALF cells are made up of eosinophils while chronic bronchitis results in >7% BALF neutrophils. However, this is not an ideal discriminator as there is often an increase in both types of inflammatory cells. Further, the BALF sample represents only a single point in time and might change depending on timing of exposure to triggering allergens or irritants. Practically speaking, cats with chronic bronchitis are treated with either systemic and/or inhalational glucocorticoids in the same was as cats with asthma. And while bronchodilator drugs are likely to be of greater benefit during an asthmatic exacerbation than they would be for bronchitis, they are used in both scenarios. Perhaps the most important reason for distinguishing between bronchitis and asthma would be that hyposensitization through ASIT could lead to a cure for allergic asthma, but would likely not be efficacious for treatment of bronchitis.

Heartworm Associated Respiratory Disease
Cats are susceptible to infection with the filarial nematode *Dirofilaria immitis*, the cause of heartworm disease. Although the percentage of infective larvae (L3) that mature to adulthood in cats is markedly less than the percentage for dogs, infection can still result in substantial morbidity and mortality even without a sustained adult worm burden. Larval worms cause vascular and parenchymal inflammatory reactions as they reach the pulmonary arteries and arterioles. These reactions cause a syndrome known as Heartworm-Associated Respiratory Disease (HARD). Clinical signs of HARD are easily misdiagnosed as asthma or bronchitis. Changes on radiographs can be identical for all three disorders, and BALF eosinophilia is found in either HARD or asthma. Of note, the same sorts of clinical signs, radiographic, and BALF abnormalities are also found to a lesser degree in cats infected with *Toxacara cati*.

Some have questioned the need to differentiate between cats with HARD and allergic asthma, arguing that adulticide therapy is not used in cats, and that glucocorticoid therapy would be used in either case. A key difference between parasitized cats and those with allergic asthma seems to be the likelihood of bronchospasm. In experimental models of each, ex vivo airway ring responses are blunted. It is possible that it is not only the heartworms themselves that incite HARD, but also the endosymbiont Wolbachia. Cats seropositive to Wolbachia were found to have greater bronchoreactity via barometric whole-body plethysmography than were Wolbachia negative cats.

Discriminating between parasitism and allergy would definitely impact the option of ASIT, might impact treatment for Wolbachia with tetracyclines, and would likely impact utility of bronchodilatory therapy as well. The difficulty, unfortunately, is in making a diagnosis of parasitism. Cats with HARD are usually heartworm antigen negative and often antibody negative as well.

Feline Asthma
Feline asthma is one of the most common bronchopulmonary diseases in cats and is responsible for substantial morbidity and occasional mortality. It is an IgE mediated hypersensitivity response against what otherwise would be harmless environmental aeroallergens. Exposure to an allergen allows for production of allergen-specific IgE formation. Those IgE antibodies then bind to mast cells on respiratory mucosal surfaces. Upon re-exposure to allergen, IgE on the surface of the mast cell bind allergen and send an intracellular signal to trigger mast cell degranulation. Mediators that are either immediately released from granules or later synthesized within mast cells are major contributors to signs of asthma. Inflammation in the airways leads to cellular infiltration (mostly eosinophils), increased mucus production, bronchoconstriction, and creates permanent architectural changes in the lung called airway remodeling. All of these lead to clinical signs of asthma.

**Therapeutic Options For Feline Asthma**

Therapeutic strategies for the treatment of asthma can either focus on suppressing the inflammation and bronchoconstriction once they have developed, or can attempt to turn off the aberrant hypersensitivity reaction before it causes airway inflammation and bronchoconstriction. Cats with asthma have varied clinical presentations, and treatment is variable as well. More severe disease signs call for more aggressive therapy. Therapy can be distinguished when treating cats with mild, moderate, or severe asthma, or cats experiencing an asthmatic crisis.

**Traditional therapies**

Traditional therapy for asthmatic cats has relied on environmental modulation as well as injectable and oral corticosteroids and bronchodilators. If the allergen causing asthma can be identified, and it is possible to remove it from the environment, the driving force for the induction of asthmatic events is removed. More often than not, the allergen is either ubiquitous or the patient is sensitized to multiple allergens, making it impossible to completely remove all allergens. Hepa-type filters can be beneficial in reducing the load or indoor aeroallergens. It is also important to decrease exposure to environmental airborne irritants, especially smoke, dusts (eg, kitty litter), and aerosols.

The mainstay of therapy for asthmatic cats or people is the reductions of inflammation, most often via treatment with glucocorticosteroids (GC). The inflammatory component of asthma must be addressed to prevent progression of disease and irreparable damage to the lungs. GC should be used in the initial management of this disease and with flare-ups, but GC actions are not immediate. Because GC can produce serious adverse effects they should be tapered to the lowest effective dose to control clinical signs and may be discontinued during periods of disease remission. For routine oral use prednisolone is preferred over prednisone for cats. Inhalant GC therapy allows direct application of GC to airways with minimal systemic absorption, allowing maximal respiratory effect with minimal systemic effect. Metered dose inhalers containing GC (eg, fluticasone or flunisolide) can be adapted for use in asthmatic cats. Inhalant therapy is certainly advantageous in cats with conditions for which oral GC are relatively contraindicated, such as diabetes mellitus or cardiomyopathy.

Traditionally, veterinarians monitor therapy only by the owner’s description of clinical response. A more objective approach would be assessment of airway eosinophilic inflammation. Collection of airway lavage fluid by BAL in a “blind” fashion (ie, without a bronchoscope) is simple, inexpensive, and safe in cats with stable respiratory function. Pre-treatment with
terbutaline further reduces the risk of this procedure. It would be reasonable to evaluate airways
by BAL prior to any significant modification of anti-inflammatory drug therapy.

Bronchodilators enhance airflow to the lungs by relaxing airway smooth muscle
and allowing an increase in airway diameter. However, the use of bronchodilators as
monotherapy is not advocated. Asthma is not just a disease associated with airway
hyperreactivity; inflammation plays a key role in both clinical manifestations as well
as permanent airway remodeling. These drugs are often described as “rescue”
medications since they rescue the ability to breathe but they do not address the actual
cause of airway narrowing. Therefore, these rescue drugs should be used in
combination with other drugs to address the cause (eg, GC inhibit airway
inflammation). Bronchodilators, including methylxanthines like aminophylline and
theophylline or beta-2 agonists like terbutaline can be administered to cats orally or
parenterally. Parenteral terbutaline (0.01mg/kg SQ) can be life-saving during
asthmatic crisis. More recently, administration of the beta-2 agonist albuterol by
metered dose inhaler has been advocated. In people with asthma, overuse of
inhalant bronchodilators may increase morbidity and mortality. The most commonly
used inhalant albuterol is composed of two racemic enantiomers, one of which
causes bronchodilation while the other may cause paradoxical inflammation and
bronchoconstriction. Single enantiomer levalbuterol (Xopenex HFA) is now
available for use and may be associated with fewer adverse reactions. For now, it
seems wise to use inhaled albuterol only as needed and to focus routine treatment on
inflammation. For cats requiring routine bronchodilation, the author prefers oral
extended duration theophylline.
<table>
<thead>
<tr>
<th>Mild Asthma</th>
<th>Moderate Asthma</th>
<th>Severe Asthma</th>
<th>Asthmatic Crisis</th>
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<td>(daily cough, occasional tachypnea,</td>
<td>(respiratory distress)</td>
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<td>tachypnea)</td>
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<td>Albuterol MDI kept</td>
<td>Albuterol no more than several times</td>
<td>Theophylline orally as bronchodilator</td>
<td>Terbutaline injection (0.01mg/kg</td>
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<td>per week, or several days in a row.</td>
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<td>use if needed</td>
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<td>OR nebulized albuterol (0.5 ml of</td>
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**Alternative therapies**

Because the “standard” therapy is inadequate in some cats, additional methods of treatment are needed. Significant work has been undertaken to investigate alternative treatments for asthma using an experimental model in which an asthmatic phenotype is induced in cats. This model has been useful, with some tested therapies seeming to be “helpful”, others not so helpful, and others still requiring further investigation.

- Cyclosporin decreases IL-2 production, leading to inhibition of T cell proliferation. It has been used in severely asthmatic people as a GC sparing anti-inflammatory drug. In experimental feline asthma, cyclosporine did not inhibit the early phase response to allergen challenge (mediated in large part by mast cells), but it was effective at blunting airway hyperresponsiveness to acetylcholine and airway remodeling. Its routine use is not advocated because of a lack of proven efficacy, the need to monitor this expensive treatment often, and the potential for adverse effects. Nevertheless, this is a reasonable option for cats that fail to respond to standard treatments.
- Maropitant, an NK1 receptor antagonist approved for use as an antiemetic, has been touted as a possible therapy for multiple respiratory disorders including asthma. Recently, studies were completed evaluating the utility of maropitant either as an every-other-day therapy for one month, or as an immediate treatment, for experimentally induced asthma. In neither setting
was there any benefit to the use of maropitant in either airway inflammation or hyperreactivity.

- Cetirizine, a 2nd generation selective histamine receptor 1 antagonist that has effects both dependent and independent of histamine antagonism, was also evaluated for suppression of eosinophilic airway inflammation in experimentally asthmatic cats, had no significant beneficial anti-inflammatory or immunological effects in an experimental model of asthma.

- Serotonin is a mediator of smooth muscle contractility in feline airways. Antagonizing the effects of serotonin by using cyproheptadine has theoretical promise, but studies have failed to support a benefit from cyproheptadine.

- Leukotriene (LT) antagonists such as zafirlukast and montelukast are useful in many but not all people with asthma. Although these drugs have been used in cats, there is no proven utility for these drugs in feline asthma. Unlike in humans, cysteinyl LTs do not appear to be an important mediator in feline asthma. Also, administration of zafirlukast to cats with experimentally induced asthma had no beneficial effects on reducing airway inflammation or hyperreactivity.

- To date, allergen-specific immunotherapy (eg, allergy shots) is the only treatment associated with a cure of allergic disease. Identification of allergens to which the patient has been sensitized is critical but difficult in practice. Intradermal skin testing seems to be the most sensitive method of allergen identification but is hampered by many medications (especially steroids) that may be given concurrently. Certain types of blood tests may accurately identify allergens, but sensitivity of these tests is not as good as for IDST. It must also be remembered that the presence of IgE to a particular allergen does not mean that that specific allergen is responsible for disease.

- Protocols for “rush immunotherapy” delivered over two days either by subcutaneous injection or mucosal administration resulted in amelioration of airway inflammation in an experimental model of feline asthma. Although the mucosal route more closely mimics actual exposure, the SC route is easier to administer and seems to be effective without compromising safety. This form of therapy nicely reduces eosinophilic inflammation in the airways, and may permanently retrain the animal’s immune system to “ignore” the inciting allergens. Trials of hyposensitization in naturally affected cats are just beginning.

- Tricking the immune response into believing that it must deal with bacterial infection by administering CpG motifs may turn the immune system away from a Th2 response that promotes asthma. In the future, CpG motifs may be used as “adjuvants” for other forms of immunotherapy.

- Salivary peptides which affect the immune response (eg, FeG-COOH; LeukoSTAT) have been postulated to be useful for feline asthma, but clinical trials have not shown efficacy in an experimental model.

- The monoclonal antibody omalizumab (Xolair) works in people with severe asthma to block IgE receptors, but this expensive humanized product cannot be used safely in cats.

- Inhibition of tyrosine kinases, a group of proteins which regulate cell survival, growth and differentiation, has more recently been of interest for treatment of asthmatic patients. Tyrosine kinase inhibitors are small molecule inhibitors which block the ATP binding sites of kinases. In asthma, the c-KIT receptor has been associated with proliferation and degranulation of mast cells and eosinophils in humans and mice and seems to be a logical target for therapeutic intervention. There are a number of commercially available tyrosine kinase inhibitors. Unfortunately, in one study in an experimental model of feline asthma
toxicity of these small molecule inhibitors was a concern despite the potential benefits on airway inflammation and airflow limitation.

- Dietary omega-3 polyunsaturated fatty acids and luteolin (an antioxidant flavinoid) administered to experimentally asthmatic cats for 4 weeks showed no effect on airway inflammation, but did show a decrease in airway reactivity. Importantly, although this prophylactic treatment holds promise to diminish airway hyperresponsiveness, because it does not blunt eosinophilic airway inflammation (which ultimately contributes to worsening airway hyperresponsiveness and airway remodeling), it should not be given as monotherapy to treat feline asthma.

- Inhaled N-acetylcysteine a mucolytic and antioxidant medication, was also tested in experimental feline asthma and was found to increase airway resistance putting into question its safety in cats with preexisting airway disease. No studies to date have evaluated the effects of N-acetylcysteine administered either orally or by inhalation on airway inflammation or mucus quality in asthmatic cats.

- Stem cell therapies have been attempted in experimental asthma but demonstrate a very limited, if any, benefit.

References available by request
**FELINE CHRONIC ENTEROPATHY**
**PART 1: THE DIAGNOSTIC APPROACH**
Audrey K. Cook BVM&S, MRCVS, Dip ACVIM-SAIM, Dip ECVIM-CA, Dip ABVP (Feline)

**History**
Although we tend to assume that cats with chronic GI disease have changes in stool consistency and frequency, this is not always the case. If the disease is limited to the small intestine (SI) and the large intestine (LI) is healthy, stool consistency may remain normal despite substantial dysfunction. Decreased food intake, weight loss or vomiting may be the only signs noted; do not discount the possibility of GI disease just because the stools have not changed.

Patient age, vaccination status, deworming history, and environmental circumstances (i.e., indoor or outdoor, single or multiple pet household, etc.) should be verified. Specific information regarding previous and current diet(s) and the administration of any prescription or over-the-counter medications should be collected. If there are other pets in the household, it may be helpful to ask about the appearance of their stools and overall health status.

Detailed information regarding the appearance of the patient's stool and the process of defecation may help localize disease. Patients with SI disease or extra-GI disorders are more likely to have voluminous stools with little evidence of discomfort during voiding. Urgency, straining, fresh blood and mucus suggest LI/rectal disease. Narrowing of the stool may indicate recto-anal disease or extraluminal compression.

Vomiting, anorexia or polyphagia may be reported. It is important to ask about changes in appetite as increased food intake suggests extra-GI disorders (e.g., hyperthyroidism or chronic cholangitis) or malassimilation disorders (e.g., exocrine pancreatic insufficiency [EPI]). A systematic review of the patient's general health should be performed, and the owner should be asked specifically about changes in thirst, energy levels, attitude, and body weight.

It is often very helpful to determine the clinical index for disease activity at the first visit, as this can be used to track response to therapy etc. Essentially, clinical signs and their relative severity are used to calculate a number (see Jergens AL, in recommended reading section).

**Physical Examination**
The goal of the exam is to identify specific abnormalities that may direct further diagnostics, such as a palpable mass or a thyroid nodule. It is important to determine an accurate bodyweight, as changes may indicate progression or improvement of disease. If the history suggests recto-anal disease, a digital rectal examination should be (cautiously!) attempted. Abdominal palpation should be systematic and careful note should be taken of apparent discomfort as well as changes in the texture or size of the intra-abdominal organs or the presence of free fluid. The consistency of fecal material within the distal large bowel may be determined through gentle compression of the contents of the descending colon / rectum. In most patients with a chronic enteropathy, the physical examination may be essentially unremarkable or fairly non-specific (e.g., mild discomfort, soft stools in colon). Obvious cachexia, dehydration or fever usually indicate severe disease; cats with these changes may need supportive care (e.g., fluid therapy, nutritional support) whilst other diagnostics are performed.

**Fecal Diagnostics**

*General considerations*
A gross examination of the feces helps localize disease to the SI or LI and may provide additional useful information. For example, greasy mustard-colored stools suggests maldigestion; dark tarry stools suggest bleeding within the stomach or proximal SI. Microscopic fecal examinations are an essential part of the diagnostic workup. If parasitic disease seems likely (e.g., young animal, access to the outside, multiple pets in household affected) then repeated examinations may be worthwhile as some parasites may be missed on a single examination. For most purposes, a three-way fecal examination is appropriate; this includes a saline smear (aka direct exam or wet mount), a flotation, and fecal cytology.

**Saline smear**
This permits identification of motile organisms such as protozoal trophozoites (i.e., *Giardia duodenalis* and *Trichomonas blagburni / foetus*). A fresh fecal sample (ideally less than 30 minutes old and not chilled) should be used, as trophozoite motility decreases quickly following removal from the host. Mix a peppercorn sample of fresh feces with 4-5 drops of saline. Examine the slide microscopically at low and high magnification; it may take a moment to recognize trophozoites as their motion may be confused by the Brownian motion of the sample. 
*Tritrichomonads* have a long, undulating membrane and a jerky movement. *Giardia* trophozoites demonstrate a more rolling motion and are pear shaped with a concave ventral disc.

**Fecal flotation**
The goal of the flotation test is to identify parasitic ova, cysts and oocysts. Centrifugation methods improve diagnostic sensitivity and are worth the additional effort. About 1 teaspoon (5 grams) of stool should be used for a flotation examination. This stool does not need to be fresh; a refrigerated sample is acceptable, but the specimen should never be frozen. Mix the stool with 5 to 10 ml of flotation solution. The solution used should have a specific gravity between 1.18 and 1.30; most commercially available solutions are based on sugar or sodium salts and fall within this range. If the patient's history suggests giardiasis, a zinc sulfate solution may be a better choice as this is less likely to distort protozoal cysts. Centrifuge for 10 minutes at 1500 rpm (generally the speed used for a urine examination). After spinning, gently add enough additional solution to create a meniscus and then place a coverslip on top. Let the sample stand for 3-4 minutes, and then transfer the coverslip to a slide and examine the material at low magnification. As the shedding of ova, cysts and oocysts may be intermittent, a negative fecal flotation does not exclude the possibility of parasitism and many clinicians will empirically treat patients with a broad-spectrum anthelmintic such as fenbendazole even if this test is negative.

**Fecal cytology**
This examination is of limited use in most patients, but may confirm an inflammatory reaction if abundant neutrophils are noted, or suggest a Clostridial disorder if numerous large bacterial spores are evident. However, enterotoxin production must be confirmed by other methods before a diagnosis of clostridial colitis can be made, since studies have indicated poor correlation between fecal microscopy and clinical disease. Similarly, a large number of campylobacter-like organisms (recognized by their distinctive gull-wing shape) may suggest campylobacteriosis, but PCR or culture is needed to confirm this diagnosis.

**Fecal antigen testing**
ELISA-based tests may be used to detect *Giardia* antigens in fecal samples. These tests have similar sensitivity to zinc sulfate flotation methods if 3 or more serial tests are performed.
**Fecal cultures**

Routine fecal cultures are usually low yield and are rarely performed in feline patients. Specific cultures for pathogens such as *Campylobacter jejuni* and *Trichomonas* are appropriate in cases where signalment and clinical signs suggest the possibility of either infection. *C. jejuni* is associated with mucoid diarrhea with fresh blood, and is most often diagnosed in cats less than 6 months of age. Special culturing methods are needed. This organism can be cultured from healthy animals, so positive culture results should be interpreted with caution.

*Trichomonas blagburni/foetus* usually affects young cats and kittens kept in breeding or shelter environments. Clinical signs consist of waxing and waning chronic large bowel diarrhea with blood, mucus, flatulence, profound anal irritation, and fecal incontinence. As successful culture requires prompt inoculation of the medium with a fresh fecal sample, reference laboratories do not routinely offer this test, but it is easily performed in the clinic setting.

**Fecal PCR testing**

Numerous polymerase chain reaction (PCR) tests are now available for specific GI pathogens, including *Trichomonas*, *Salmonella* spp., and *Cryptosporidium* spp., and for the *Cl. perfringens* enterotoxin A gene. Although such tests have markedly improved our ability to identify these organisms, remember that many of these can be found in the feces of clinically normal cats. Results must therefore be correlated with patient age, environment, history and clinical signs.

**Systemic evaluation**

Before considering more specific diagnostics, it is important to perform a serum biochemistry profile, complete blood count including white cell differential, electrolyte panel and urinalysis. If the retroviral status of a feline patient is uncertain, appropriate testing should be performed. Thyroxine levels should be measured in any cat over 7 years of age or any adult cat with a history of polyphagia and weight loss. The purpose behind this general health assessment is three-fold: firstly, to exclude non-GI issues such as hyperthyroidism or chronic cholangitis; secondly to identify concurrent unrelated conditions; and thirdly to look for changes related to GI disease such as anemia, hypercalcemia or hypoproteinemia.

Routine blood tests are often unremarkable in cats with chronic GI disease. If changes are noted, they tend to suggest more severe or sustained intestinal compromise. For example, a microcytic or regenerative anemia suggests GI hemorrhage; hypoalbuminemia suggests lymphoma; and hypercalcemia suggests an underlying malignancy or granulomatous disease.

**Additional Laboratory Testing**

**Folate (Vitamin B9)**

Serum folate concentrations are influenced by dietary intake, small intestinal brush border enzyme activity, and the number and function of specific carriers in the proximal SI. As dietary deficiency is extremely uncommon, low serum folate concentrations suggest duodenal disease. Non-hemolyzed samples must be submitted, as red cell lysis causes spurious elevations.

**Cobalamin (Vitamin B12)**

After ingestion, cobalamin is bound first to R-protein in the stomach, and then handed over to intrinsic factor (IF), which is secreted by the exocrine pancreas. The cobalamin-IF complex is
then absorbed by specific receptors in the ileum. Low serum cobalamin concentrations therefore suggest long-standing ileal disease or EPI.

**Trypsin-like immunoreactivity assay (TLI)**
This test is a reliable way to identify EPI. A twelve-hour fast is necessary prior to sample collection. This test is particularly appropriate for patients with a robust appetite and chronic voluminous diarrhea. In many affected cats, the stool is greasy and a light mustard color.

**Pancreatic lipase immunoreactivity (fPLI)**
PLI testing provides useful information regarding pancreatic inflammation and appears to be a more sensitive and specific marker for pancreatitis than abdominal ultrasonography. Pancreatitis would be unlikely to cause chronic diarrhea but may occur concurrently with chronic GI disease.

**Histoplasma capsulatum testing**
A quantitative enzymatic immunoassay (EIA) is an excellent way to diagnose this fungal infection. This should be considered in cats in endemic areas, including those that are strictly indoors, as the infection can be acquired from contaminated shoes or houseplant soil. Testing urine and serum concurrently maximizes the chance of a positive result. Test sensitivity is high (estimated at >95%); I always run this test before endoscopy or an empirical steroid trial.

**Abdominal Imaging**

**Abdominal radiography**
Radiographs are rarely rewarding in cats with chronic GI disease, but may indicate a foreign body, obstruction, intussusception, traumatic or congenital hernia, megacolon, or mass lesion.

**Abdominal ultrasonography**
Ultrasonography is highly operator dependent, but is a more sensitive imaging modality than radiography, and provides useful information regarding mass lesions, intussusceptions, lymphadenopathy, and changes in intestinal wall thickening or abnormal layering. In addition, lesions may be aspirated under brief sedation with ultrasound guidance. For some disorders, cytological findings may be diagnostic, e.g., visceral mast cell tumors, large cell lymphoma or fungal disease. The liver, biliary system and pancreas should be carefully evaluated, as concurrent disorders of these systems may be noted and impact patient management or outcome.

**Endoscopy**
Endoscopic evaluation of the GI tract is relatively non-invasive and is an appropriate option for cats with diffuse inflammatory or infiltrative mucosal disease. In patients with SI signs, multiple (6-10) mucosal biopsies should be collected from the stomach, duodenum and ileum, even if the tissue appears normal. Colonic biopsy is necessary in those with signs of LI disease. Biopsies are small and fragile; careful orientation and prompt fixation are necessary. GI endoscopy requires specialized equipment and expertise and does have substantial limitations. Firstly, large stretches of the SI (jejunum and upper ileum) are not remain accessible. Secondly, only mucosal lesions can be identified, and focal thickening or submucosal changes will not be appreciated. Thirdly, specimen size and depth may limit their diagnostic utility.
**Exploratory Laparotomy**
Surgical evaluation of the abdomen is indicated in cats with mass lesions, jejunal disease, submucosal pathology or evidence of extra-GI pathology such as hepatobiliary or mesenteric disease. If endoscopy would be appropriate but is not available, an exploratory laparotomy should be considered, but clients should be advised of the increased risks associated with surgery v endoscopy and offered referral. If surgery is performed, the entire abdominal cavity should be evaluated carefully, and biopsies collected from each part of the GI tract +/- the liver.

**Advanced testing**
If histopathological findings suggest lymphoma, PCR for antigen receptor rearrangement (PARR) may be necessary. This test helps differentiate small cell lymphoma from lymphocytic inflammatory bowel disease. Immunophenotyping can differentiate B cell v T cell lymphoma and can guide therapeutic options. However, recent studies in clinically normal aged cats have suggested that these tests may be less definitive than originally assumed.

**Conclusion**
Appropriate testing is necessary to establish a diagnosis in a cat with a chronic enteropathy. Decisions about diagnostic options should be based on signalment, history and response to previous therapies. However, the diagnostic tools that are presently available have substantial limitations, as many of the functions of the GI tract (such as motility, secretion, digestion, absorption and immunological surveillance) cannot be adequately evaluated.
Introduction
The therapeutic plan for a cat with a chronic enteropathy is ideally based on a diagnosis. Readers are directed to current textbooks for information regarding the treatment of specific disorders, such as gastrointestinal (GI) infections or neoplasia. However, the underlying cause of the enteropathy may be ill-defined (e.g. inflammatory bowel disease [IBD]), or a definitive diagnosis may not be established. In these circumstances, a number of symptomatic or nonspecific therapies may be considered.

Therapeutic diets
A range of diets are marketed for use in cats with vomiting and diarrhea; these are generally highly digestible, high in fiber, or with limited/novel antigen. Care must always be taken when making a dietary change, as a sudden diet switch may exacerbate diarrhea. Also, cats have strong taste and texture preferences and may refuse to eat an unfamiliar diet. The new food should be provided in conjunction with the old diet for a few days, and food intake carefully monitored.

Highly digestible diets
High digestibility minimizes intestinal bacterial metabolism and facilitates nutrient assimilation when digestive function is compromised. This tends to decrease fecal volume and may limit weight loss. A highly digestible diet should contain a single protein source, no allergenic additives or flavorings, with protein digestibility >87% and carbohydrate digestibility >90%. Highly digestible diets are an appropriate choice for patients with small intestinal diarrhea in which food allergy seems unlikely. Canned products are generally more digestible than the dry equivalent and should be used whenever possible. It may also be helpful to pick a diet from a different manufacturer to the one the cat is accustomed to. Some animals appear to be intolerant of routine additives such as preservatives or colorings; most pet food companies use the same additives throughout their product line and picking a different brand is generally advisable.

High fiber diets
Dietary fiber may be soluble or insoluble; both may be beneficial in patients with colonic dysfunction. Insoluble fibers improve intestinal motility and bind fluid in the intestinal lumen. Soluble fibers decrease fecal bulk, bind non-absorbed fluid into gels, and increase the concentrations of beneficial bacteria. An addition benefit from soluble fibers is the production of short chain fatty acids; these are the preferred energy source for colonocytes and decrease pH within the colon. The more acidic environment may inhibit opportunistic pathogens such as Clostridium and Salmonella spp. If a high fiber diet is not appealing, fiber supplements containing psyllium (a mixed fiber) may be used instead, at a dose of about 250-500 mg daily.

Limited / novel antigen diets
Limited or novel antigen diets are designed for patients with abnormal immunological responses (often called 'food sensitivities' or 'dietary allergies') to one or more foodstuffs. There are many commercially available diets in this group, containing either hydrolyzed proteins or novel protein and carbohydrate sources. The term 'hydrolyzed' refers to proteins which have been cleaved in to
small fragments; these are thought to avoid triggering an immunological response. When selecting an elimination diet, it is important to choose a protein source to which the patient has not been previously exposed, as prior sensitization may have occurred. Homemade diets can be used, but they must be nutritionally balanced if long term use is planned. It may take several weeks to see a complete resolution of diarrhea, although most cats with true food allergies show substantial improvement within the first week. Ideally, a novel / hydrolyzed antigen dietary trial should last at least 4 weeks before a judgment is made, and strict adherence to the limited diet is necessary. In a patient with true dietary hypersensitivity, re-introduction of the previous diet(s) should result in a rapid onset of clinical signs; although this step is necessary to confirm the diagnosis, most owners are reluctant to risk a relapse.

Prebiotics and Probiotics
Modification of the GI microbial populations may be a helpful strategy for patients with IBD. Prebiotics and probiotics are designed to promote so-called beneficial populations and thereby confer positive effects on the host. Synbiotics are products containing both pre- and probiotics. There is minimal independent information supporting the use of these products in cats and they should not be used in place of standard therapies for infectious disorders.

Prebiotics
These are non-digestible ingredients which essentially ‘feed’ beneficial microbial species. Many prebiotics are moderately fermentable fibers such as beet pulp and psyllium and are likely to be most helpful for patients with chronic large bowel diarrhea. The other common probiotic group is the fructooligosaccharides (FOS); these are long chains of fructose molecules found in many plant species. FOS supplementation favors the growth of *Bifidobacterium*, *Lactobacillus acidophilus* and other *Lactobacilli* species, which may improve intestinal health and function.

Probiotics
Probiotic products contain live microorganisms which are purported to confer a benefit on the host when administered in appropriate amounts. Possible positive effects include a modulated immune response, improved GI function and improved fecal consistency. As probiotic products are not licensed drugs, product oversight is limited, and concerns may arise regarding their efficacy and viability. Numerous veterinary probiotic products are currently available; practitioners should recommend products made by reputable companies, and with labels describing the strain and number of live organisms present and an expiration date. It is also possible that a patient may have a positive response to one probiotic product but not to another.

Antimicrobial agents
*Tylosin*
Tylosin is a macrolide antibiotic with activity against numerous gram-positive organisms and may be used specifically for the treatment of patients with *Clostridium perfringens* enterotoxicosis. The drug also has immunomodulatory properties and may be a useful adjunctive therapy for cats with IBD or non-specific colitis. Tylosin is not licensed for small animal use but is available as a powdered formulation. This has a bitter taste, so it is often necessary to have it encapsulated for easier administration. I recommend 20 mg/kg by mouth every 12 hours. A teaspoonful of the soluble powder contains about 2600 mg; mixing the powder with confectioners’ sugar assists with appropriate dosing.
Metronidazole
Metronidazole is a nitroimidazole antimicrobial with activity against anaerobic bacteria and some protozoal species. The recommended dose for chronic diarrheal disorders such as IBD is 10-15 mg/kg by mouth every 12 hours until clinical signs improve, then tapered to the lowest effective dose needed to control clinical signs. It has been suggested that this drug may be mutagenic in cats, so it may not be the best choice for long term use in this species. Also, most cats find metronidazole unpalatable and may salivate profusely; this can be mitigated by placing partial tablets in gelatin capsules to avoid contact with the tongue.

Immunosuppressive Drugs
Although IBD is one of the most common chronic enteropathies in cats, glucocorticoids and other immunosuppressive drugs should not be started unless other causes such as infection or neoplasia have been reliably excluded, and the patient has failed to respond to non-specific therapies such as diet modification and tylosin.

Prednisone and Prednisolone
Glucocorticoids are routinely used in patients with IBD. In addition to their immunomodulatory and anti-inflammatory properties, they may also impact bile acid metabolism. As some cats are unable to convert prednisone into the active form (which is prednisolone), the latter is the preferred option in this species. Long-term use is associated with iatrogenic Cushing’s syndrome and increases the risk of diabetes mellitus. The starting dose for cats with IBD is 2 mg/kg/day; in more severe cases, this may be increased to 4 mg/kg/day, divided into two equal doses. If the clinical signs resolve, the dose should be tapered by 25% every 3-4 weeks. In some cases, the steroids can be effectively withdrawn, but others need long-term therapy. If this is the case, the goal is to find the lowest effective dose, ideally given every other day to prevent suppression of the pituitary-adrenal axis.

Budesonide
Budesonide is a locally active steroid: it undergoes extensive first-pass metabolism by the liver so systemic exposure is limited. If glucocorticoid-associated side effects are a concern, budesonide can be a useful alternative, and I have used it successfully in cats with IBD that respond to pred but relapse when this is tapered. Budesonide seems able to maintain these cats but is not (in my hands) effective as a first-line option. The recommended dose is 1 mg/cat by mouth every 24 hours. Budesonide is more costly than prednisolone and must be compounded.

Chlorambucil
This drug is an alkylating, antineoplastic/immunosuppressive agent. It is routinely used in cats with intestinal small cell lymphoma but may also be considered for cats with refractory IBD. Various dose schedules have been described; I routinely start at 2 mg/cat by mouth every 48-72 hours. Adverse effects include vomiting or hyporexia. Myelosuppression is a concern; a CBC should be monitored every 4 weeks for 3 months, and then every 2-3 months.

Cyclosporine
Cyclosporine is a potent immunosuppressive agent and works primarily through modification of T-cell function. It has been used successfully for dogs with steroid-refractory IBD, but there is
little information regarding the use of this drug in cats with GI disease. The recommended dose is 5 mg/kg by mouth every 12-24 hours. Side effects include vomiting and hyporexia. This drug causes diabetes mellitus in people, by decreasing insulin secretion and limiting insulin responsiveness. Although this has not been evaluated in cats, we should expect a similar effect.

**Adjunctive therapies**

*Motility modifiers*
Opioid agonists such as loperamide and diphenoxylate may cause excitatory behavior in cats and are also not a suitable choice in this species.

*Adsorbents*
Oral adsorbents and protectants are designed to bind damaging bacterial toxins and digestive juices and/or protect the mucosa from direct damage. There are numerous preparations in this category, containing various combinations of bismuth salts and kaolin, +/- charcoal or calcium salts. Some products contain subsalicylate for its anti-inflammatory effects; this should be used cautiously in cats as elimination can be slow if systemic absorption occurs.

*Cobalamin*
Hypocobalaminemia is a common finding in cats with severe or chronic ileal disease or exocrine pancreatic insufficiency. All patients a serum cobalamin concentration <400 ng/L should receive parenteral or oral supplementation with cyanocobalamin; current dosing recommendations are listed on the TAMU GI Lab website. Interestingly, it seems as though hypocobalaminemia may be both a cause and a consequence of chronic intestinal dysfunction, as cobalamin supplementation alone may improve clinical status, with firmer stools and increased food intake.

**Stem cell therapy**
In one study looking at a very small number of cats with chronic enteropathy, 5/7 cats receiving allogenic adipose-derived mesenchymal stem cells improved or had resolution of clinical signs. More information is needed regarding this interesting approach, but this report was certainly encouraging.

**Fecal transplantation**
This is now a standard approach for refractory *Cl difficile* diarrhea in people, and has been reported anecdotally to be beneficial in some dogs and cats with chronic diarrheal disorders. There is little consensus about donor selection, patient preparation, dosing volume and methodology, but (assuming the donor is healthy) this option is unlikely to be harmful.

**Conclusion**
A specific diagnosis should be established, whenever possible, in cats with chronic enteropathy, in which case appropriate, targeted therapy is indicated. However, the response to treatment may be slow and other options such as dietary modification or probiotics may hasten improvement. For other patients, the underlying cause may be elusive, or a non-specific disorder such as IBD may be diagnosed. If this is the case, a multi-modal approach may offer the best chance for successful patient management.
RADIOGRAPHIC DIAGNOSIS OF CARDIAC VS RESPIRATORY DISEASE IN CATS

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It is well documented that cats can have subclinical heart disease, and diagnosis of clinical disease can be particularly complicated. Clinical signs and physical exam findings are often nonspecific and may not become evident until after the patient is already in congestive heart failure (CHF). Cardiothoracic auscultation usually reveals a heart murmur, gallop rhythm, or arrhythmia, though heart sounds may be masked or muffled by pulmonary crackles, wheezes, and pleural effusion. The first sign of heart disease of any kind may also be particularly catastrophic, with outcomes including thromboembolism and sudden death.

These difficulties make an organized diagnostic approach to the dyspneic feline even more relevant. The use of “shotgun” therapy with diuretics, bronchodilators, and steroids is sometimes employed when a definitive diagnosis cannot be made based on the available information. While this can be very effective in an emergency setting, it is not recommended for long term management of either cardiac or respiratory disease. Common mistakes that lead to misdiagnosis (or missed diagnosis) of CHF in a cat that actually has respiratory disease include: (1) mismatching historical and physical exam findings with radiographic suspicions; (2) lack of confidence in evaluation of pulmonary vessels (3) lack of confidence in using the vertebral heart score; (4) not using follow up radiographs after diuretic trial; (5) not correlating pulmonary opacity on orthogonal projections; and (6) forgoing radiographs entirely if orthogonal projections cannot be obtained.

Signalment: Breed predispositions for heart disease have been identified for the Maine Coon, American Shorthair, British Shorthair, Ragdoll, Norwegian forest cat, Siberian, and Rex, though hypertrophic cardiomyopathy (the most common heart disease in cats) is still most frequently diagnosed in the domestic shorthair (mixed breed).

History: In contrast to dogs in CHF, cats do not typically cough as a result of heart disease. This history should help rank respiratory causes higher than cardiac causes of distress. Inquiring about heartworm prevention will determine whether early testing for this disease would be beneficial. Considering familial history of heart disease is useful, when available. Cats in CHF may be visibly dyspneic or tachypneic, but clinical signs may also be quite vague, such as decreased activity, decreased appetite, hiding, or other abnormal behaviors perceived by the owner. Clinical signs not intuitively associated with cardiac disease in cats include vomiting, and lameness, paraparesis, or abdominal pain due to thromboembolism.

Physical Exam: Cats in CHF present with some degree of respiratory compromise and systemic hypotension. Exam findings include tachypnea, dyspnea, orthopnea, and tachycardia. It is always possible that an animal can have comorbid respiratory and cardiac disease, although a recent study reported that the absence of a heart murmur has a 90-100% negative predictive value for hypertrophic cardiomyopathy (Payne et al, 2015). Lung sounds are often difficult to hear, and are
also dependent on the degree of pulmonary edema and/or pleural effusion. Thin body condition or cachexia may be apparent. Cats with primary respiratory disease may have normal to increased lung sounds.

**Ultrasonography:** If available, point-of-care ultrasound should be employed to evaluate dyspneic cats for pleural effusion. Thoracocentesis is recommended early in the diagnostic workup period, if necessary, to stabilize the patient, allow additional diagnostics (such as radiographs), and provide fluid for analysis.

Full echocardiographic studies are often indicated early in the diagnostic workup for dyspneic cats, and are recommended in combination with thoracic radiographs if CHF is suspected. In circumstances when specialist echocardiography is not an option, sonographers trained in the most basic left atrial measurement, the ratio of left atrial vs aortic diameter (LA:Ao) on short axis ultrasound of the heart could also be very helpful in supporting a diagnosis of CHF.

**Baseline Radiographs:** Thoracic radiographs remain the gold standard for diagnosis of CHF, but they have a poor negative predictive value for diagnosis of heart disease in cats. Radiographs may be normal in cats that actually have heart disease, although they are always abnormal in cats with CHF. The major limitation for diagnosis of CHF in cats via thoracic radiography alone is that singular abnormalities are not pathognomonic for CHF.

At minimum, two orthogonal radiographic projections are recommended to diagnose cardiogenic pulmonary edema. These may include right lateral and ventrodorsal (VD) or dorsoventral (DV) views, although it may be beneficial to obtain 3 or 4 views in some cases (right lateral, left lateral, VD, and DV). It is easy to misdiagnose pulmonary edema from a lateral radiograph in dyspneic cats because the pulmonary parenchyma is more likely to be poorly aerated during exposure. This will give the overall impression of “busy” caudal lungs that are diffusely increased in opacity. Practically speaking, caudodorsal interstitial or alveolar markings should almost always be confirmed on a VD or DV radiographic view. When questioning the presence of CHF, a well-positioned DV projection will highlight the caudal lung lobes, making the associated pulmonary vessels easier to assess. The sternal patient position for a DV projection may also be less stressful for the patient in respiratory distress. Nevertheless, for stable feline patients, the author prefers to obtain VD and DV views together in the same radiographic study. Given the feline propensity for cardiogenic pleural effusion, a combination of these views allows concurrent evaluation of the caudal lobar vasculature and movement of pleural effusion that might cause border effacement of the heart. The addition of a left lateral view is also often beneficial for specific evaluation of the right lung lobes and the cranial lobar vessels.

Radiographs will usually reveal cardiomegaly, but some cats will have normal radiographic heart size despite cardiomyopathy that is severe enough to lead to CHF. Cardiomegaly may also be obscured by severe alveolar disease and/or large volumes of pleural effusion. Radiographic atrial enlargement is difficult to accurately assess in cats, but best evaluated on DV/VD views. In patients with untreated CHF, expect vascular enlargement and cardiomegaly. Lesions associated with CHF are found in the pulmonary parenchyma, pleural space, or both. Distribution of
pulmonary parenchymal changes is varied and nonspecific, and cats do not maintain the perihilar and caudodorsal distribution of pulmonary edema often seen in dogs.

**Response to Therapy:** A diuretic trial is often considered a diagnostic test in its own right, particularly when trying to discern the principal contributor to a patient’s respiratory difficulty in the face of comorbid cardiac and respiratory diseases. Radiographic abnormalities caused by primary respiratory disease will not respond to diuretic therapy alone. Conversely, assuming that appropriate dose and duration of diuretic are administered in the absence of concurrent therapies (such as antibiotics), the clinical signs and radiographic signs of pulmonary edema and small volumes of pleural effusion are expected to improve.

Patients are expected to have a positive clinical response to diuretic therapy sooner than radiographic improvement. Improvements are manifested as decreased respiratory rate, respiratory effort, and reliance on supplemental oxygen. In order to help prevent severe secondary dehydration, cardiology faculty at this author’s institution recommend that diuretic therapy be titrated down when the patient shows 50% improvement in clinical parameters such as respiratory rate.

Large volumes of pleural effusion secondary to CHF (or other causes) may not resolve quickly enough with diuretic therapy alone. In tachypneic/dyspneic cats, pleural effusion should be removed via thoracocentesis unless the volume is small enough that it is unlikely to be a primary contributor to clinical signs.

**Recheck Radiographs:** When CHF is suspected and diuretics are subsequently prescribed, the timing of repeat thoracic radiographs depends on the severity of clinical signs at presentation. In less stable patients, repeat radiography may be performed as early as 12-24 hours after the initial study, and in more stable patients, the recheck interval may span 2-4 days or more. In cats with true CHF, cardiogenic pulmonary edema should improve (or resolve) following appropriate diuretic therapy. Following diuretic therapy, all patients are expected to have a reduction in pulmonary vascular size and/or heart size (whether or not they had CHF) due to iatrogenic dehydration. Primary pulmonary disease (such as bronchial wall thickening secondary to asthma) will likely remain unaffected, although certain processes (such as non-cardiogenic edema, acute respiratory distress syndrome, hemorrhage) may begin to resolve during the recheck interim without directed therapy.

Radiographs made before and after thoracocentesis are helpful for comparison purposes. Post-thoracocentesis images are especially helpful to evaluate the mediastinum and heart size, as well as the pulmonary parenchyma, which should expand as the pleural fluid volume decreases. More aerated pulmonary parenchyma provides better contrast to evaluate vascular size and interstitial opacity. If a large volume of fluid is removed from the pleural space and the radiographic appearance of the lung does not change, chronic effusion and secondary pleural fibrosis would be suspected (making congestive heart failure somewhat less likely).

Many confounding factors can influence the above generalizations. One clear example is the recent administration of diuretic or other therapies that may improve a patient’s clinical signs, normalize physical exam parameters, and change radiographic appearance of anatomic structures
like heart size, vessel diameter, and pulmonary patterns. For instance, it can be quite difficult to confirm suspicions of CHF in cats when diuretic therapy is administered in combination with antibiotics, steroids, and/or antifungals. The opportunity to monitor radiographic response to therapy is further hindered if these therapies are instituted prior to baseline thoracic radiographs.

It is a good rule of thumb to interpret diagnostic tests based on the patient in front of you rather than interpreting the patient based on the test results. The guidelines reported here are aimed to put this rule into practice, with proper emphasis focused on the patient history, physical exam, and response to therapy in conjunction with emphasis placed upon diagnostic imaging. By refining your diagnostic approach to interpret each set of findings (historical, physical, radiographic, and therapeutic) in context with the others, you will be more likely to make accurate diagnoses and appropriate treatment recommendations for your patients.

References:


Payne, J.R., Brodbelt, D.C., and Fuentes, V.L. Cardiomyopathy prevalence in 780 apparently healthy cats in rehoming
We can all relate to the emotional dread we experience when we hear the unique yowl of the feline in severe distress. That yowl could easily be a urinary obstruction (UO), but it could just as easily be a feline aortic thromboembolism (FATE) or “saddle thrombus”. While both UOs and FATEs can be professionally stimulating, FATE is less likely to result in a satisfying patient outcome. While there have been innovations in predicting and diagnosing this challenging condition, there have been few, if any, clear innovations in treating these cats and improving both short and long-term survival.

### Risk Factors

No discussion of thromboembolic disease is complete without at least a passing mention of Virchow’s triad: hypercoagulability, blood stasis, and endothelial dysfunction. In the feline patient, heart disease is the leading cause of thromboembolic disease, with clots suspected to originate within the left atrium or left auricle. Both blood stasis within a dilated cardiac chamber, endothelial injury of the endocardial surface, and increased platelet activation can occur in cats, completing Virchow’s criteria for thromboembolism (TE). It’s thought that intracardiac clots start out as activated platelets and mature to incorporate fibrin in layers. These layers are less stable, and portions of the original clot can break off, or the entire clot can migrate out into circulation. Sadly, there is no way to reliably assess the risk of TE disease, but the literature reveals that 12-17% of cats with cardiomyopathy will develop FATE. Males are at increased risk of FATE, which likely represents the increased occurrence of heart disease in male cats. Cats with FATE typically have a larger left atrium, larger end-systolic left ventricular diameter, and decreased fractional shortening\(^1\). There is also thought that the presence of a cardiac “gallop,” spontaneous echogenic contrast (smoke), or a visible clot on echocardiography puts patients at increased risk of FATE\(^2\). Thromboprophylaxis is likely indicated in patients with cardiomyopathy displaying these characteristics.

### Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>FATE</td>
<td>Feline aortic thromboembolism</td>
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<tr>
<td>UO</td>
<td>Urinary obstruction</td>
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<tr>
<td>TE</td>
<td>Thromboembolism/thromboembolic</td>
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<tr>
<td>AKI</td>
<td>Acute kidney injury</td>
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<tr>
<td>tPA</td>
<td>Tissue plasminogen activator</td>
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<tr>
<td>UFH</td>
<td>Unfractionated heparin</td>
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<td>LMWH</td>
<td>Low molecular weight heparin</td>
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### Clinical Signs

While most feline TE disease manifests as FATE, it is also possible to have migration to a forelimb, kidney, brain, mesentery, or spleen. Forelimb TEs occur in approximately 10% of TE patients, with the right forelimb more commonly affected than the left because of the branching of the brachial arteries. Patients with forelimb TE can have subtle to severe sings, and patients that present for FATE may have a recent history of “forelimb lameness” that was actually a previous TE event. FATE can result in partial or
complete occlusion of the aortic and collateral flow to one or both hindlimbs. Up to 75% of FATE cats are affected in both pelvic limbs. Affected limbs are often painful, display paresis or paralysis, absent segmental reflexes, muscle discomfort and contraction, diminished or absent arterial pulses, cyanotic nailbeds, and hypothermia. Severe cases of bilateral FATE can also result in a decreased rectal temperature due to decreased perfusion as a result of cardiac failure or TE arterial occlusion. Clinical signs often develop acutely and can be concomitant with a new or breakthrough cardiac failure event. Most (90%) cats with FATE have underlying cardiac disease, but only 10% have been diagnosed prior to the FATE event. It can be difficult to determine if respiratory effort in FATE patients is due to pain, stress, or cardiac disease.

**Diagnosis**

Diagnosis of a FATE event is often pretty straightforward, with an acute onset of pain, hindlimb paralysis or paresis, and the absence of arterial pulses. Patients with unilateral disease or incomplete vascular occlusion (weak but present limb pulses) can potentially pose a challenge. There are both simple and advanced diagnostic options available to clinicians. The most basic of which is to locate and measure the arterial pulse pressure (or lack thereof) in the affected limb with a Doppler crystal. A small blood sample obtained from an affected limb can be used to compare glucose and lactate measurements and compare to a central (jugular) blood sample. The glucose in an affected limb is expected to be significantly lower than peripheral, and lactate is expected to be significantly higher than peripheral blood. Ultrasonographic evaluation of the TE can reveal renal artery occlusion and can be used to serially evaluate blood flow around and through the occlusion. Advanced imaging options such as thrombus contrast-enhancing MRI are also potentially available, but rarely necessary or employed in veterinary medicine.

Blood testing performed on cats with FATE may reveal evidence of acute kidney injury (AKI), electrolyte derangement, and elevated indicators of muscle damage (CK, AST, & ALT). Hyperkalemia is not common in the acute phase of disease, but it can be severe and is a common sequela of reperfusion injury and/or AKI when

**Treatment**

Currently, no “magic bullet” treatment has been described for FATE. Current recommendations are based on the principles of supportive care: pain management, preventing thrombus propagation (growth), managing underlying cardiomyopathy, preventing mutilation, and preventing TE recurrence. The ischemic injury that occurs in affected limbs is almost certainly painful and should be managed with pure mu agonist opioids initially. Cardiomyopathy and congestive disease should be managed as needed to maintain optimum cardiovascular stability and peripheral perfusion. Limiting thrombus propagation and prevention of recurrence are both accomplished with antiplatelet medications and/or anticoagulants. Physical clot removal has been reported by several modalities, but, is not currently recommended. Treatment with “clot buster” thrombolytic medications (e.g. tPA- tissue plasminogen activator) has shown efficacy in breaking down occlusive TEs, but complication rates are high, outcomes are not improved, and survival is similar to conservative therapy. It’s hard to know whether or not to recommend tPA for cats severely affected with FATE, as they have the gravest prognosis and potentially the most to gain
from thrombolysis, but they also have the highest risk of sudden death, AKI, and life-threatening reperfusion injury.

The antiplatelet agents aspirin and clopidogrel (Plavix®) are the most-studied class of thromboprophylaxis in the treatment of FATE. Anticoagulant agents such as unfractionated heparin (UFH), low-molecular-weight heparin (LMWH), and rivaroxaban (Xarelto®) are also legitimate options for use in these patients. Monitoring of thromboprophylaxis is of limited value and can realistically only be performed with any timeliness in patients treated with UFH. For patients *at risk* for TE disease, it is probably appropriate to select one avenue of thromboprophylaxis (e.g. clopidogrel), but patients with confirmed TE disease should likely be treated with multimodal the addition of a second anticoagulation agent (e.g. clopidogrel + LMWH or clopidogrel + rivaroxaban) for both treatment and indefinite thromboprophylaxis.

**Aspirin** - inhibits the production of thromboxane A2 within platelets. Has a narrow therapeutic window in the cat and can be associated with GI side effects. Typical dosing is 81mg PO every 48 – 72h. In cats with a previous FATE event, recurrence of FATE occurs in 17 - 75% of cats (64% within the first year), and median survival times vary from 117 – 192 days. While this option is inexpensive, it has recently fallen out of favor due to its inferior performance when compared with clopidogrel⁵.

**Clopidogrel (Plavix®)** – irreversibly inhibits ADP on the platelet surface to reduce platelet reactivity, granule secretion, and fibrinogen binding. Clopidogrel requires hepatic transformation into an active metabolite via the cytochrome p450 system, which may affect the bioactivity of other medications. A loading dose of 75mg PO on initiation of treatment or a standard daily dose of 18.75mg PO q24h may be prescribed depending on clinician preference. In cats receiving clopidogrel after a previous TE event, 49% of cats had TE recurrence (36% within the first year), and they had a median survival time of 443 days. The recent expiration of the patent on this medication has made it an inexpensive treatment option.

**Low molecular weight heparins** – are short-chain heparin molecules that only inhibit factor Xa. They have a relatively low risk of bleeding complications, and monitoring is not currently recommended. Enoxaparin (Lovenox®) and dalteparin (Fragmin®) are both used in the treatment of FATE. Both of these medications are given subcutaneously and require dosing every 12 hours. Despite common usage, no data is currently available on the efficacy of LMWH in the treatment or prevention of FATE. Enoxaparin is administered as 1.0-1.5 mg/kg q12h and dalteparin is administered at 100-200 IU/kg every 12h. Both of these medications are quite expensive but potentially manageable due to the small amount administered to feline patients.

**Rivaroxaban (Xarelto®)** - factor Xa inhibitor available in an oral formulation. No published data on the use of rivaroxaban for FATE is currently available in cats, but studies are underway to evaluate the use of this medication as a sole-therapy for TE prevention in cats with previous TE events. Current evidence suggests that 2.5 – 5mg/kg q24h is well tolerated in cats. This is a relatively expensive medication that is reasonable for many clients because of the small dose required in feline patients.
Outcome

Overall survival for cats with FATE are reportedly between 27 – 45%. Hypothermia, loss of motor function, bilateral arterial occlusion, and bradycardia are all negative prognostic indicators. Cats with only one affected limb and/or intact motor function can do significantly better, with a 68 – 93% survival rate, while cats with bilateral occlusion are reported to survive only 15 – 36% of the time. Up to 40% of cats will die spontaneously despite treatment, and up to 35% of cats are euthanized during hospitalization. With these dismal numbers, it’s easy to understand clinician bias and see how up to 90% of cats with FATE are euthanized shortly after diagnosis. Patients are most likely to die or acutely worsen within the first 72 hours. Patients that survive for 72 hours are more likely to survive to discharge, which can have variable endpoints. Some patients will improve rapidly and regain motor function and blood flow in the affected limb(s). Other patients will require 6-8 weeks to maximize clot dissolution and recanalization. Regained limb function may remain absent or incomplete in some patients and predicting survival and outcome goals are impossible on presentation. Surviving cats may require physical therapy, supportive nursing care, lifelong thromboprophylaxis, and management of underlying cardiomyopathy. Even with thromboprophylaxis, cats with TE disease remain at lifelong risk of TE recurrence, which is often life-limiting. The uncertainties of treatment, cost, quality of life, and recurrence of TE disease are understandably significant obstacles for many owners and clinicians.

Veterinary clinical literature commonly focuses on heartworm (HW) infections in dogs, but often feline infections with *Dirofilaria immitis* are more challenging to diagnose and manage. How common are HW infections in cats? Companion Animal Parasite Council (CAPC) data show that the seroprevalence is somewhere around 1.0% nationally, based on antibody detection. Compiled data from several large scale studies show a national antigen positive prevalence ranges from 0.4% to 1.0%, while Texas values are approximately 2% antigen positive (Levy et al., 2017; CAPC Prevalence Maps). These numbers are influenced by the population of cats tested and the type of test used, but it is unsurprising that feline HW infection in Texas exceeds the national averages (Levy et al., 2017; CAPC Prevalence Maps).

The pathophysiology of HW infection and disease in cats is well described (AHS Guidelines). Cats are more resistant to infection than dogs, so that few larvae transmitted during mosquito feeding develop to the adult stage. Therefore, cats usually have a low adult worm burden, and the lifespan of adult worms in cats is shorter than in dogs. However, the infection is clinically relevant significant because even a small number of worms frequently cause a potentially life-threatening disease syndrome known as 'heartworm-associated respiratory disease' (HARD), particularly associated with damage in the vasculature of the respiratory system. The administration of appropriate monthly doses of a macrocyclic lactone preventive to experimentally infected cats has shown that pulmonary pathology may be reduced, but parasite-associated damage continues to accumulate in pulmonary vasculature as long as adult worms are present. Additionally, adult worms are more frequently located in aberrant locations such as systemic arteries, CNS or body cavities in cats, when compared to HW infected dogs.

Routine annual HW screening is a standard recommendation for dogs, but diagnostic screening in cats is far less common in a typical clinical practice. In a recent survey of more than 1,300 veterinary clinics from across the USA collecting data on more than 26,000 animals,
28.3% of animals were tested because of current illness, while only 23.5% of the cats were tested as a part of a new pet examination protocol, and 12.6% of cats were tested at more than one time point (Levy et al, 2017). These data suggest that feline HW infections are underdiagnosed because of a lack of routine screening as part of a wellness plan for feline patients.

Diagnosis of HW infection in cats is more challenging than in dogs. Both antigen detection and antibody detection point-of-care immunodiagnostic tests are available, but not all antigen detection tests are labeled for use in cats. The correct interpretation of antibody test results, both positive and negative, requires thoughtful analysis, and additional information/tests are usually required. The antigen detection test is considered the 'gold standard' of testing in dogs, but interpretation of antigen testing is more complicated in cats. Positive antigen tests indicate active adult worm infections in the cat patient; however, false negative antigen tests occur regularly in infected cats. The sensitivity of antigen tests is lower when there are few adult worms that produce small amounts of antigen. Additionally it is now recognized that circulating worm antigen plus cat antibody immune complexes may interfere with immunodiagnostic tests giving false negative test results. A published protocol of heat treatment of serum is designed to disrupt the ag/ab immune complexes resulting in the conversion of false negative to positive tests. While this heat treatment of serum samples is not recommended as a routine screening method, it may be helpful in clarifying problematic cases where HW disease is suspected in the feline patient, but preliminary screening test results are negative. Several commercial veterinary diagnostic laboratories now offer the heat-treated HW testing on serum on a fee-for-service basis.

Infection prevention is always preferred to treatment, and no safe, labeled parasitocidal treatment is available for cats. Unfortunately, in the recent survey from more than 1300 veterinary clinics across the USA, only 12.6% of cats (n = 34,975) that received HW diagnostic testing were prescribed with a heartworm preventive product (Levy, 2017). An increasing number of safe and effective HW preventives and combination products are now commercially available for cats from multiple pharmaceutical companies. Veterinarians, especially in high areas of endemicity such as Texas, should prioritize the education of cat owning clients to the risks of feline HW infection, and should recommend routine immunodiagnostic screening tests and monthly chemoprophylaxis as a safe and effective option for cats in this region.
HELPFUL REFERENCES and RESOURCES:


Gastrointestinal parasites are typically the most common group of infectious diseases infecting companion animals. In Texas, common parasites in cats include *Toxocara cati*, *Ancylostoma tubaeformae*, *Cystoisospora* spp. *Dipylidium caninum*, *Taenia taeniaeformis* and *Giardia duodenalis*. Prevalence ranges for each of these parasites vary depending on the population of cats tested and the diagnostic method used for parasite detection. The CAPC Prevalence maps for *Toxocara*, *Ancylostoma* and *Giardia* indicate infection in <1 to approximately 4% of cats tested in 2018 in Texas (https://capcvet.org/maps/#2019/all/roundworm/cat/united-states/texas/) (accessed Jun, 2019). However, a much higher prevalence of intestinal parasitism has been reported in shelter cats in Georgia where 45.6% of 103 cats were positive for one or more intestinal parasites (Hoggard et al., 2019). Similarly, 63.9% of 846 free-roaming cats were positive for one or more intestinal parasites in a recent study in Oklahoma (Nagamori et al., 2018).

Parasitologic fecal tests are one of the most frequently performed diagnostic tests in companion animal practice. Which parasitologic tests are performed routinely in your clinical practice? Which parasitologic tests are performed at a fee-for-service lab on behalf of your patients/clients/clinic? What expertise and quality control is needed to perform these tests competently and accurately? To take a fresh look at parasitologic testing in a practice setting, let's address the question of why we conduct parasitologic tests. First, parasite screening is usually considered a part of routine wellness checks. For production animals, the general target is to manage parasite burdens below a threshold of negative economic impact. However, with companion animals, the typical approach is to diagnose any parasites that might be present and to treat all identified infections. A second reason for parasitologic testing is related to clinical disease when a parasite(s) is included in a differential diagnostic list as a possible cause of the clinical disease. Since some of these common parasites have zoonotic potential, a third reason for parasitologic screening is to protect your clients and other people who contact the cat or share the same environment.
What fecal examination techniques are used routinely in practice? The most frequently performed parasitologic assay is a fecal flotation, followed by direct smears. While simple flotation tests are most frequently performed, a centrifugal flotation method is considered the "gold standard" since it is well documented that the centrifugation step adds sensitivity to the test and detects more parasites (Dryden et al., 2005; Zajac et al., 2002). The accuracy of an in-house fecal flotation test depends on:

1) choice of test method
2) use of appropriate reagents with correct specific gravity
3) quality and quantity of fecal sample
4) accurate performance of the test using a standard protocol
5) microscopy skills of the person evaluating the test
6) appropriate record keeping regarding test results

What about less common parasites that are not routinely diagnosed using fecal flotation techniques? Remember that the Baermann technique is used to concentrate larvae from fecal samples (Zajac and Conboy, 2012). The sedimentation technique is used to concentrate dense eggs that don't readily float such as trematode, acanthocephalan and some nematode eggs (Zajac and Conboy, 2012). If those test methods are not performed in your clinic, then make sure that a commercial diagnostic lab performing those assays is identified and that protocols are in place in advance to submit the appropriate samples.

Increasingly, veterinary practitioners elect to send diagnostic samples to a commercial fee-for-service laboratory instead of performing parasitologic tests in-house. When using these external services, be sure to investigate the competency of diagnostic laboratory personnel with regard to parasitologic skills and knowledge and the protocols used at the facility. Suggested questions are:

1) Who routinely performs parasitologic tests, and what is their training history?
2) Who supervises the personnel completing parasitologic tests?
3) What routine standardized parasitologic protocols are in place, and what is the source of the protocols?
4) When uncommon parasitologic diagnostic methods are needed, are appropriate protocols available (such as Baermann or sedimentation tests)?
5) When unusual parasites are suspected or submitted, how are those samples handled, and what expertise is available to provide correct identification and recommendations?
The Companion Animal Parasite Council is a non-profit agency with board membership including parasitologists and practitioners. Their current guidelines for 'Parasite Testing and Protection Guided by Veterinarians' (https://capcvet.org/guidelines/general-guidelines/) (accessed Jun, 2019) include:

1) Conduct preventive physical examinations at least every 6 to 12 months.

2) Conduct annual heartworm testing in dogs: test cats prior to placing on preventative and thereafter as indicated by history and physical findings.

3) Test annually for tick-transmitted pathogens, especially in regions where pathogens are endemic or emerging.

4) Conduct fecal examinations by centrifugation at least four times during the first year of life and at least two times per year in adults, depending on patient health and lifestyle factors.

5) Prescribe control programs to local parasite prevalence and individual pet lifestyle factors.

6) Adapt prevention recommendations to address emerging parasite threats.

7) Confirm pets have been both recently tested for parasite infection and are current on broad-spectrum internal and external parasite control prior to boarding or visiting shared space animal facilities.

Optimum parasitologic diagnostic testing demands good practices in the clinic setting and accurate, reliable service from experienced personnel at fee-for-service diagnostic laboratories. It is important to pay attention to the details in order to provide quality parasitologic services for your clientele. It is good medicine for the patient and should be profitable for the practice from an economic perspective.

REFERENCES:


Vomiting is a common clinical sign observed in feline patients, and can occur secondary to a multitude of gastrointestinal and non-gastrointestinal disorders. In the workup of a vomiting patient, there are a few important things to consider when selecting the appropriate imaging test. Patient signalment, history, duration of vomiting, concomitant clinical signs, and owner preference may all contribute to the selection of diagnostic tests to be performed. When performing a clinical workup for a vomiting patient, it is helpful to understand specific benefits and limitations of the various imaging modalities available for abdominal imaging.

1. **Abdominal Radiographs:**
   a. This modality is a great screening tool to be utilized as the first step in a vomiting patient and can aid in the diagnosis of multiple overt abnormalities such as:
      i. Mechanical gastrointestinal obstruction (including a linear foreign body)
      ii. Overt renal abnormalities (acute kidney injury, chronic kidney disease, many obstructive urogenital processes).
   b. Limitations:
      i. It is often difficult to appreciate many common but more subtle causes of feline vomiting including pancreatitis, non-obstructive gastroenteropathies (inflammatory bowel disease, lymphoma), and hepatobiliary disease. An ultrasound may be needed in addition to the already performed radiographs, which can increase client cost.
      ii. Abdominal radiographs may be difficult to interpret in patients with poor serosal detail (this can be due to a thin body condition, peritoneal effusion, or young patient age).

2. **Abdominal Ultrasound:**
   a. This modality is often the most useful imaging tool that we have in the workup of vomiting cats, and is superior to abdominal radiographs in many situations.
   b. Due to the three-dimensional nature of ultrasound, a full evaluation of the abdominal organs may be performed. Additionally, ultrasound provides the ability to obtain guided cytologic samples for potential definitive diagnoses such as:
      i. Urine for urinalysis (UA) and culture
      ii. Parenchymal aspirates in the workup of a neoplastic or infectious disease process
      iii. Free peritoneal fluid sampling
   c. In general, abdominal ultrasound is often recommended when abdominal radiographs have been normal or difficult to evaluate, in chronically vomiting
patients, and in patients with multiple co-morbidities that may complicate the overall clinical picture.

d. Limitations:
   i. Ultrasound often requires at least light sedation in non-compliant patients and for guided sampling.
   ii. Ultrasound may not be readily available at your clinic.

3. **Abdominal CT (computed tomography or CAT scan):**
   a. This modality may show overt abnormalities (similar to radiography), and may also provide further information regarding large intra-abdominal masses and obstructive urogenital conditions.
   b. If a cat is severely unstable, a rapid carrier assisted CT may be performed without sedation and with limited stress to the patient.
      i. The quality of these studies is often poor, and this is typically only utilized to identify major abnormalities.
   c. Limitations:
      i. Due to the small body size of most cats, adequate spatial resolution is a major limiting factor when imaging the cat abdomen. Many subtle abnormalities may be completely missed on a CT scan.

In this lecture, we will cover some of the most common and readily identifiable causes of vomiting in the feline patient, and the appearance of these disease processes on the above-discussed imaging modalities. These are listed below. We will focus heavily on abdominal radiographic and ultrasonographic findings, with computed tomography discussed less frequently. In the laboratory associated with this lecture, we will take this topic one step further by evaluating images in a case-based discussion regarding the clinical picture, imaging findings, and proceeding diagnostic steps.

1. **Mechanical gastrointestinal obstruction (mechanical ileus):** One of the most common causes of vomiting in young, playful cats who may be prone to foreign body ingestion. Foreign bodies in cats frequently have a linear component, which can result in plication of small intestinal loops. Imaging findings may include:
   a. Two sizes/populations of bowel with portions of bowel being dilated and other portions being normal in size.
   b. Material identified within the gastric lumen or small intestinal loops that persists for > 12-24 hours.
   c. **Linear foreign body:** Intestinal plication with multiple small, irregularly shaped gas bubbles and intestinal clumping. Don’t forget to check under the tongue in all linear foreign body cases!
2. **Gastrointestinal mural abnormalities:** This includes inflammatory bowel disease (IBD) and lymphoma, and is one of the most common causes of chronic vomiting in middle aged to older cats. Ultrasound is necessary to visualize the walls of the GI tract, and radiographic findings often do not aid in this diagnosis. Imaging findings may include:
   a. Diffuse or multifocal thickening of the gastrointestinal walls, or thickening of the muscularis layer of the gastrointestinal wall.
   b. Loss of delineation between wall layers.
   c. Enlarged, hypoechoic, and rounded intra-abdominal lymph nodes (more severe in lymphoma cases).
   d. **Note:** It can be quite difficult to differentiate lymphoma and inflammatory bowel disease, and additional testing (including scope procedures, bloodwork, or cytologic sample collection and evaluation) may be necessary.

3. **Pancreatitis:** This may represent the sole cause of a patient’s vomiting, or may occur as a sequela to other disease processes. Radiographic evaluation of the pancreas is difficult, and ultrasound is often utilized for diagnosis. Imaging findings may include:
   a. Loss of detail in the expected region of the left limb of the pancreas on radiographs.
   b. A hypoechoic (dark) and enlarged pancreas with surrounding hyperechoic (bright) fat and mesentery on ultrasound.
   c. Small volume peritoneal effusion surrounding the pancreas.

4. **Renal disease:** This is a large category that can include acute kidney injury, chronic renal degeneration, and obstructive uropathies. In general, chronic renal degeneration is the most common finding identified both radiographically and ultrasonographically, especially in older cats with chronic clinical signs. The findings for all three of these disease processes can vary greatly. Many of these diagnoses may be strongly suspected from radiographs; however, a combination of ultrasound, bloodwork, and urinalysis results are often necessary to fully evaluate these patients. Imaging findings may include:
   a. **Chronic renal degeneration:** Bilaterally small, irregularly marginated kidneys that may be hyperechoic (bright) on ultrasound with other degenerative changes including renal cortical cysts and mild pyelectasia (renal pelvis dilation).
   b. **Acute kidney injury:** Bilaterally enlarged, smoothly marginated kidneys that are significantly hyperechoic (bright) on ultrasound and may be surrounded by perinephric effusion.
   c. **Obstructive uropathy:** Unilaterally enlarged, smoothly marginated kidney with significant pyelectasia. A mineral opaque urolith may be identified in the plane of the urogenital tract; however, many other underlying obstructive processes (mucous plugs, strictures, or masses) can only be identified via ultrasound or computed tomography.

*References will be made available in the accompanying powerpoint presentation to this lecture.*
Special needs of cats

The natural diet of cats in the wild is meat-based (e.g. rodents, birds, insects, small mammals), and, as such, they are metabolically adapted to utilize protein and fat preferentially as energy sources without the need for or ability to utilize effectively dietary starches. More importantly, when protein is limited in the diet, cats will immediately use muscle tissue from their body to meet their protein and amino acids needs. Recently, Laflamme and coworkers reported that adult cats that did not consume at least 5.2 g protein/kg body weight per day (e.g.12 g protein/100 kcal or approximately 40-45% protein ME) lost lean body mass. Thus, cats literally start to use their own muscle to meet their body protein needs when the amount of protein in their diet does not exceed 40%ME. This finding is important, not just as an indicator of optimal protein for maintenance of health, but because lean body mass (muscle) is a key driver of metabolism and is important in maintenance of health during states of illness (including cancer). Muscle mass is critical to normal energy metabolism, normal insulin function, and skeletal/bone mass maintenance, as well as many other essential metabolic functions. Typical feline diets, including over-the-counter (OTC) and many therapeutic dry cat foods are 30-38% ME. Thus, one of the first and most important changes to recommendations in providing healthy nutrition for cats, especially those with cancer is to feed diets with at least 12g/100 kcal or greater than 40% ME of a high-quality animal based protein.

In addition to the needs for a larger amount of protein in the diet of adult cats for maintenance of muscle mass and the optimal health that goes with it, recent studies in cats over 12 years of age show that aging cats actually have an increased need for protein, fatty acids and certain vitamins (especially B vitamins). In a lifetime study of geriatric cats, researchers found that with each year of age beyond twelve, healthy geriatric cats have a naturally occurring, but progressive, loss of their ability to digest and absorb protein and fatty acids – so much so that by the ages of 15 or beyond, they have nearly a 25% reduction in their digestive efficiency.

The main reasons healthy geriatric cats lose body weight and muscle as they age is due to a combination of 1) the general use of lower protein/lower fat adult or senior diets, and many therapeutic diets, 2) reduced activity due to osteoarthritis or indoor lifestyle which contributes to loss of muscle, and 3) the phenomenon of sarcopenia (e.g. muscle loss due to the combination of aging and chronic inflammation) (Freeman 2011). There is little debate that in aging animals, many diseases and disorders that cause or are associated with inflammation are common, but recent evidence in people over the age of 50 suggests that diets high in simple sugars and highly digestible CHO are believed to contribute to increased pro-inflammatory states in elderly humans – a connection not yet studied in cats but likely to be a factor in the loss of muscle. In addition to their increased protein needs, diets for senior cats need to have more energy from fat (e.g. > 20% fat), with increased omega-3 fatty acids and B vitamins (especially cobalamin) (Sparkes 2011, Laflamme & Gunn-Moore 2014).

In summary, to provide optimal nutrition for adult/senior cats it is ideal to feed a complete and balanced diet. This can be a commercial or a homemade diet depending on the
preference of the owner for whole versus processed food sources and their willingness to actively participate in providing an appropriate diet for their cats. The ideal feline diet should contain >40% ME of an animal based, highly digestible protein with less than 10% ME of CHO.

Feline Cancer Patients

Oncology is one of the areas of medicine where recent advances and progress can improve outcomes for patients. However, the frequent presence of malnutrition in cancer patients can limit their response to even the best therapies if nutritional issues are not appropriately managed. Malnutrition and a loss of muscle mass are frequent in cancer patients and have a negative effect on clinical outcome. They may be driven by inadequate food intake, decreased physical activity and catabolic metabolic derangements. It is important to screen for, prevent, assess in detail, monitor and treat malnutrition in cancer patients. And, nutritional care should always be accompanied by exercise training or increased activity to the best of pets ability. To counter malnutrition in patients with advanced cancer there are few pharmacological agents (e.g. appetite stimulants) and pharmaconutrients (e.g. supplements) but these have only limited effects. This paper discusses some of key features of nutrition in pets with cancer.

Major derangements leading to malnutrition and nutritional invention/support:

Inadequate nutritional intake is observed frequently in patients with cancer and is associated with weight loss, which may be severe. The causes for impaired intake are complex and multifactorial. Reduced food intake is caused by primary anorexia (i.e. central nervous system level) and may be compounded by secondary impairments to oral intake, some of which are reversible with suitable medical management. Key secondary causes of reduced intake include oral ulceration, xerostomia, poor dentition, intestinal obstruction, malabsorption, constipation, diarrhea, nausea, vomiting, reduced intestinal motility, chemosensory alteration, uncontrolled pain, and side effects of drugs. Total inability to eat due to factors such as bowel failure or complete obstruction cannot be tolerated and requires timely implementation of artificial nutrition (unless there are specific contraindications) to avoid starvation. Partial reduction in food intake also results in large caloric deficits over time and, in this instance, consideration should be given to the percent daily deficit (e.g. >25%, >50%, or >75% of energy requirements), the expected duration, as well as the degree of depletion of body reserves.

Muscle protein depletion is a hallmark of cancer cachexia, severely impinging quality of life and negatively impacting physical function and treatment tolerance. This can be particularly profound in cats due to their increased need for protein in their diet for use as energy under normal healthy conditions. Studies of the body composition of human patients with cancer reveal that it is specifically the loss of skeletal muscle with or without loss of fat, which is the main aspect of cancer-associated malnutrition that predicts risk of physical impairment, post-operative complications, chemotherapy toxicity, and mortality. There are fewer studies in dogs and cats assessing these complications of malnutrition, but cachexia is increasingly recognized as a major contributor to morbidity in both dogs and cats, and particularly in senior pets.

A systemic inflammation syndrome is frequently activated in patients with cancer. This can vary in degree but impacts all relevant metabolic pathways including:

- Protein metabolism: systemic inflammation is associated with altered protein turnover, a loss of fat and muscle mass and an increase in the production of acute phase proteins.
- Carbohydrate metabolism: systemic inflammation is frequently associated with insulin resistance and impaired glucose tolerance.
- Lipid metabolism: The capacity for lipid oxidation is maintained or even increased in cancer patients and especially so in the presence of weight loss.

Other considerations:
- Feed normal Maintainence Energy requirements (or more if needed to maintain weight) – the idea that calories and nutrients feed the cancer is debunked – animals with cancer need to eat as near to normal RER or MER to maintain their body weight
- Addition of glutamine to protect the GI tract from chemotherapy or provide nutrients to help the GI tract is not proven to be beneficial, but is not harmful.
- Protein nutrition is critical in cats – muscle loss cannot be prevented or overcome without adequate dietary protein – the amount of protein recommended has not been studied in cancer patients, but it will be at least as much as needed in a normal cat, which is greater than 40%.
- Omega 3 FAs are anti-inflammatory and while controlled studies showing benefit in all cancer patients is lacking, consensus in human cancer nutrition is supportive of use.
- Increasing activity / exercise has been shown to be extremely important in human cancer patients to improving appetite and metabolism, and is also expected to be beneficial to feline patients.

Cats with Chronic Kidney Disease

That brings us to the complicated issue of protein in cats with CKD. The most important clinical goals for cats with IRIS stage I (non-azotemic) or stage II (mildly azotemic) CKD are 1) maintenance of hydration (e.g. maintain renal blood flow), 2) maintenance of normal phosphorus levels. (Protein deprivation at this stage is not only not indicated, it is detrimental to the cat.) (Kidder & Chew 2009), and 3) controlling other risk factors (e.g. hypertension, hypokalemia) (Scherk & Laflamme 2016). Traditional renal diets have 23-27% ME protein. Vastly lowering protein levels in the diet will lower blood urea nitrogen (BUN) (low dietary protein) and creatinine (due to muscle mass loss), but these reductions can give the clinician a false sense of security that the disease is being well managed. In fact, it is maintenance of renal blood flow and glomerular filtration rate (GFR) that will maintain renal function and prolong life, all the while preserving the quality of life. Maintenance of renal blood and function is best accomplished by controlling hypertension, maintaining hydration, and preventing hyperphosphatemia and the onset of renal secondary hyperparathyroidism, and will not be achieved by simply changing their diet. Furthermore, in most cats with advanced renal disease, e.g. stage 3 or 4, one of the greatest issues in maintaining their quality of life is food intake. There is clear evidence that cats prefer high protein diets over diets high in CHO (Salaun 2016), and because food intake is an important factor in both feline well being and their owners perception of quality of life, there is further
reasoning for not restricting their dietary protein to the level found in “renal diets”. In cats with stage 4 disease have signs of uremia or hyperphosphatemia that can no longer be obtained with other means, and then, and only then, would a mild restriction of protein (30-38%ME) be included in the plan – and only if the cat will eat enough of the diet to meet its nutritional and caloric needs. The key point is that there is a fine line between feeding a diet that is ideal for a disease process and feeding a diet that is better for the overall health of the animal, and this is clearly a situation in which the veterinarian must balance the two.

Cats with GI Disease

The use of diet to assist in the management of GI disease is not a new concept. Nevertheless, the type of diet used to help manage the problem has become an increasingly complex issue – particularly in older cats. In many, if not most cats with intestinal disease, especially those cases without a definitive diagnosis or without significant weight loss or other morbidity, the best approach is to feed a highly digestible diet or change the diet to one with fewer additives, flavorings, or other substances than may be associated with food intolerance. This term is not defined in a regulatory sense, but generally indicates a product with protein digestibility of > 87% (typical diets are 78-81%), and the digestibility of fat and CHO should be greater than 90% (typical diets are 77-85% and 69-79%, respectively). These types of diets are designed to provide food that is easy to digest (moderate to low fat, moderate to increased in protein, moderate to decreased carbohydrate), may have additives to improve intestinal health (soluble fibers, omega 3 fatty acids, increased anti-oxidant vitamins, etc), and contain no gluten, lactose, food coloring, preservatives, etc. There are many different brands available that fall under the category “highly digestible”, but, the key is to remember that they are not all alike. In particular, the protein digestibility of the diet chosen is one of the key factors they may determine the success of the diet. This information can be difficult to access, but in general, meat source proteins are more digestible than plant source (e.g. wheat gluten or other plant protein sources added to foods), animal proteins are more digestible than meat by products. Meat meals are a good source of protein. Also, to increase digestibility of foods in cats, decrease the number and amount of carbohydrates in the food – a single source carbohydrate food is better than foods with many different sources, highly digestible carb sources are better than complex plant source carbs. Thus, when one diet from this category not accepted or is ineffective, or seems to make the diarrhea worse, you cannot assume that all diets in this category will be ineffective. The highly digestible diets from different pet food manufacturers have a wide variety of different formulations: different protein and carbohydrate sources, different levels of fat, and a variety of additives designed to promote intestinal health (FOS, MOS, omega 3 fatty acids, antioxidant vitamins, soluble fiber, etc). If one type of highly digestible diet has been fed for at least 2 weeks with minimal response, then is it entirely reasonable to either try another diet from a different source, or try an entirely different dietary strategy (e.g. high protein/low carb, novel antigen, hydrolyzed, etc). Another consideration is that the diarrhea may be due to carbohydrate intolerance or bacterial changes resulting from diet changes. Finally, there are a number of cats with chronic diarrhea that will respond to a homemade diet (high protein, no carb, no additives) – the best diet to try (after you have exhausted trials with appropriately chosen commercial diets) is boiled or microwaved cooked chicken thighs – with the fat/skin attached and the bones removed. You may need to shop this into a fine mince or blenderize it to
get some cats to eat it (especially if they are not wet food eating cats or do not get table food). Adding a vitamin/mineral supplement is important to prevent calcium/phosphorus imbalances and other micronutrient deficiencies. If an intolerance is the cause of the diarrhea, it will stop in this time frame, if not, an intolerance is not the cause of the diarrhea and other solutions must be sought. You must not feed this diet for longer than 2-3 weeks without adding a vitamin/mineral supplement mix and appropriate calcium to balance the diet. The best approach is to use Balanceit.com as an on-line approach or to consult with a nutrition specialist to get the appropriate information to properly balance the diet. Thus, addition of probiotics or prebiotics to help influence the microflora are also reasonable therapeutic options. Many cats that improve on a homemade diet can eventually go back to a commercial food, as long as the offending substance is not present in the diet – this may be carbohydrates, additives or any of a number of other things – so careful, slow reintroduction of foods is needed.

Gastrointestinal disease may decrease the availability of a number of micronutrients, such as vitamins and minerals, with important consequences for the pathogenesis, diagnosis and treatment of gastrointestinal disease. The diagnostic utility of measuring the serum concentrations of cobalamin and folate in cats with suspected intestinal disease has been well established, and although the impact of deficiencies in cobalamin and folate are not completely defined, the role of cobalamin in normal function of the GI tract and in many other aspects of metabolism is well documented. Further, because cats are obligate carnivores that consume much higher amounts of protein in their diet, the importance of cobalamin and other B vitamin in maintenance of protein metabolism cannot be overstated. Thus, evaluation of all cats with any GI disease, not just cats with IBD or lymphoma, is an important part not only of the diagnostic process, but in the management of these diseases as well. While other vitamin or mineral deficiencies (e.g. vitamin E or K) may occur with long standing or severe IBD/lymphoma, they are less likely (due to storage of fat soluble vitamins and some minerals) and supplementation without documentation of a deficiency can be dangerous. Finally, in cats with hypcobalaminemia due to severe or chronic disease, the diarrhea will not resolve until replacement therapy is instituted and in some cats therapy may be lifelong. In older cats with concurrent chronic pancreatitis, decreases in production of pancreatic enzymes (detected by measuring TLI) can also result in reduced digestion of foods, development of bacterial dysbiosis, and subsequent weight loss and diarrhea. Thus, measurement of TLI in cats with chronic diarrhea is an important tool for assessment in addition to testing and replacement of cobalamin.
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Dr. August graduated with honors from the Royal Veterinary College at the University of London, and became a member of the Royal College of Veterinary Surgeons. He completed an internship, residency in small animal internal medicine, and obtained an MS at Auburn University. After coming to Texas A&M University from Virginia Tech, he was Professor and Head of the Department of Small Animal Medicine and Surgery, and then developed the feline internal medicine service after returning to the faculty. In 2016, he was appointed Dean of Faculties and Associate Provost of Texas A&M University. On March 1, 2019, he was appointed Interim Dean of the School of Public Health, Health Science Center, Texas A&M University.

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Leah Cohn specializes in small animal internal medicine with special interests in the treatment of infectious, immune-mediated, and respiratory diseases. She has a particular focus on developing better prevention and treatment of feline cytauxzoonosis, commonly known as bobcat fever, which was first documented in Missouri in the 1970s. Cohn received her bachelor’s degree and doctor of veterinary medicine from the University of Tennessee. She completed an internship and residency in small animal medicine and surgery at North Carolina State University, along with a PhD in veterinary microbiology and immunology. Cohn is a former president and chair of the Board of Regents of the American College of Veterinary Internal Medicine. When she isn’t working, she loves to spend time with her husband and children, as well her dogs, cats, chickens, goats, sheep and llamas.
Dr. Audrey Cook, DVM, DABVP, DACVIM, DECVIM – Associate Professor. VSCS.
Dr. Audrey Cook obtained her veterinary degree at the University of Edinburgh. She completed an internship in small animal medicine and surgery at North Carolina State University before starting her residency in small animal internal medicine at University of California at Davis. She is board certified through the American College of Veterinary Internal Medicine and the European College of Veterinary Internal Medicine. In addition, she is one of the few internists with additional certification in Feline Practice through the American Board of Veterinary Practitioners.

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Dr. Christine Rutter, DVM- Clinical Assistant Professor. VSCS. Dr. Rutter graduated from Mississippi State University, completed an internship in Louisville, Kentucky and a residency at Tufts University. She was in private specialty practice for 6 years. Her interests include coagulation, immune mediated blood dyscrasias, transfusion medicine, trauma management, post-operative patient care, and respiratory disease.

Dr. Jessica Vallone, DVM- Clinical Assistant Professor of Veterinary Radiology. Dr. Vallone received both her DVM and MS degrees from Mississippi State University, pursued a small animal rotating internship at VCA Veterinary Referral Associates, and spent 3 years as an both an emergency and shelter veterinarian before completing her diagnostic imaging residency at Texas A&M University. She is
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currently a Clinical Assistant Professor in the diagnostic imaging service at Texas A&M University. She absolutely loves cats, and has been fostering felines for over a decade.

Dr. Deborah Zoran, DVM, MS, PhD. DACVIM – Professor. VSCS.
Dr. Deb Zoran is a Professor in the Department of Veterinary Small Animal Clinical Sciences at Texas A&M University and founding member of the Texas A&M Veterinary Emergency Team (VET). She is a 1984 graduate of Kansas State University’s College of Veterinary Medicine. She completed a Master’s degree and became a Diplomate of the ACVIM – Small Animal Internal Medicine during her time there. In 1992, she moved to Texas A&M University and completed a PhD in Nutrition in the laboratory of Dr. Joanne Lupton. Since 1996, Dr. Zoran has been a member of the faculty in the Veterinary Medical Teaching Hospital at Texas A&M University where she is actively involved with clinical, teaching, and research activities in small animal nutrition, disaster preparedness, gastroenterology and feline medicine.

Dr. Karen F. Snowden, DVM, PhD.- Professor Karen F. Snowden is a Professor Emeritus at the College of Veterinary Medicine at Texas A&M University, where she has been a faculty member focusing on parasitology teaching and research for more than 25 years. Growing up in Alabama, she received her DVM degree from Auburn University, and spent about five years in mixed practice in western North Carolina. Returning to academia, she received her PhD from the College of Veterinary Medicine at North Carolina State University and spent five years abroad in postdoctoral and lecturer positions at the Liverpool School of Tropical Medicine in Liverpool, England, before returning to the US to join he faculty at Texas A&M. She is a founding diplomat for the parasitology specialty in American College of Veterinary Microbiology.