Feline pancreatitis is a very difficult disease to definitively diagnose ante-mortem (especially in chronic cases or in cats that do not have persistent signs), and treatment remains symptomatic and supportive. The problems of diagnosis and treatment include the lack of specific clinical signs in cats, the lack of a rapidly available test for diagnosis, and, in cats with chronic pancreatitis, the difficulty of the testing itself. This talk will review the salient features of both acute and chronic pancreatitis in cats and discuss the treatment of cats with pancreatitis.

Diagnosis

The clinical signs of pancreatitis in cats are quite different from those in dogs. Two forms of pancreatitis appear to occur in cats: acute necrotizing pancreatitis (ANP), a disease similar in appearance, if not cause, to canine acute pancreatitis, and lymphoplasmacytic pancreatitis (LP), which can present as an acute or chronic disease. In a recent histologic review of cats that were submitted to postmortem for any cause (not just pancreatitis), the percentage of cats with ANP was less than 15%, while 65% of the cats had LP changes. This illustrates that the feline disease is quite different from that recognized in the dog. The acute pancreatitis that is frequently encountered in obese dogs who are fed a high-fat diet is not reported in cats. Cats with ANP are more likely to be underweight, and high-fat diets do not appear to be an important predisposing factor for the disease. In LP, cats of all ages and breeds and both sexes are affected, although Siamese cats are reported to have pancreatitis more frequently than other cats. Finally the clinical signs of LP in cats are vague, with the most common being lethargy (reported in 100% of cats in one study), anorexia, dehydration, and abnormal body temperature (including either fever or hypothermia). This is especially true for cats with chronic or mild forms of LP. Vomiting and anterior abdominal pain, which are common clinical signs in dogs with acute pancreatitis, are reported to occur in only 35% and 25% of cats with LP, respectively, but are common in cats with ANP. However, cranial abdominal pain may be more common than is reported, as detection of abdominal pain may be difficult in obese cats or cats with very focal disease. Cats with the most severe forms of pancreatitis, such as ANP, may be icteric or in shock, and the prognosis for these cats is significantly more guarded than it is for cats with milder forms of pancreatitis. Other conditions that may occur concurrently with pancreatitis in cats include hepatic lipidosis, cholangiohepatitis, inflammatory bowel disease, interstitial nephritis, diabetes mellitus, or vitamin K–responsive coagulopathy. Thus, the clinical signs may be quite variable, and this must be taken into consideration with each patient. In addition, with increases in liver enzymes and bilirubin, the signs and abnormalities can easily be attributed to liver dysfunction, which further delays the diagnosis.

Routine evaluation of cats with suspected pancreatitis may include hematology, a serum biochemistry profile, urinalysis, abdominal radiography and/or ultrasound, and serum assays of pancreatic function (e.g., feline trypsin-like immunoreactivity [fTLI] or feline pancreatic lipase immunoreactivity [fPLI]). Hematologic findings in cats with pancreatitis are nonspecific but may include a nonregenerative anemia, leukocytosis, or leukopenia (less common). In a recent study, cats with pancreatitis consistently had an elevated WBC (20,300 cell/μL) and mild decreases in platelets (mean = 180,000 platelets/μL). Reported changes in the serum chemistry profile include elevated serum alanine aminotransferase (ALT), elevated serum alkaline phosphatase (ALP), hyperbilirubinemia, hyper- or hypoholesterololemia, hyperglycemia, azotemia, and hypokalemia. Liver enzyme elevations were more common in cats with mild pancreatitis (determined by surgical biopsy), and GGT, ALP, and ALT were all moderately elevated in these cats. Hypocalcemia is less commonly observed, but when present may be a poor prognostic sign seen in cats with severe pancreatitis or multiple organ dysfunction. Serum lipase may be increased early in acute pancreatitis, but in a recent study amylase and lipase were found to be of little diagnostic value in distinguishing normal cats from those with pancreatitis. There are no changes in the urinalysis consistently observed or specific for pancreatitis in cats.

The fTLI was developed years ago as the definitive test for diagnosis of exocrine pancreatic insufficiency (EPI), and the data and follow-up have confirmed its utility for this condition. In recent years, others have evaluated the fTLI as a diagnostic test for acute pancreatitis, working on the premise that an elevation in serum concentrations were
consistent with pancreatic leakage or inflammation. While an increase in fTLI can be found in cats with acute pancreatitis, a normal fTLI does not rule out pancreatitis. This is because the leakage of enzymes tends to decrease or is controlled by the body’s peptidases (macroglobulin, etc.) within 12 to 24 hours following an acute insult. Further, in chronic or low-grade pancreatitis, the leakage is not great enough to be detected by this assay. While an increase in fTLI is specific for pancreatic enzyme leakage, it is not sensitive enough to be a definitive test for pancreatitis. Nevertheless, the fTLI is an important test of pancreatic function in cats, because cats with chronic low-grade pancreatitis are at risk of losing exocrine (and endocrine) function, and thus may become an EPI patient. However, in cats with EPI, the signs are much more subtle, with weight loss being the most common sign (not diarrhea). Therefore, measurement of TLI at that same time of the PLI (to test for inflammation) is important in cats.

The fPLI was developed by the GI lab at Texas A&M University. The assay is species specific, has been used to detect elevations in pancreatic lipase in clinical cases, and has appeared to be more specific and sensitive than the fTLI for diagnosis of pancreatitis in cats. The sensitivity in mild pancreatitis was found to be 65% to 80%, while the specificity in healthy cats was 75%. However, in severe pancreatitis (determined by pancreatic biopsy), the sensitivity and specificity were both 100%. These findings underscore the utility of this test in cats with acute pancreatitis; however, there still is a problem with detection of low-grade or chronic pancreatic inflammation in cats with this assay. This is especially a problem in cats with chronic pancreatitis that have pancreatic atrophy and a reduction in enzyme production or release. Further, there appears to be an influence on this enzyme by other local factors, such as inflammatory bowel disease (IBD), that may affect the diagnostic interpretation. In cats with chronic pancreatitis, enzyme levels can be quite variable, and thus it will still be necessary to evaluate the combined historical, physical exam, lab data, and imaging information along with the fPLI when making a diagnosis. Recently, a more widely available test has been developed for testing PLI in cats (specFPL). This test has the same limitations that the original test had for detection of low-grade inflammation; however, because it is more readily available and results are prompt, it provides a useful adjunct for early evaluation of cats with suspected pancreatitis.

Imaging studies are frequently used to help identify cats with acute pancreatitis; however, the changes are not consistent and can be particularly subject to interpretation and operator expertise. The most common radiographic abnormalities include a generalized or focal (upper right quadrant) loss of peritoneal detail (suggesting peritonitis or peritoneal effusion), presence of a mass in the area of the pancreas, hepatomegaly, dilated intestinal loops, or a fluid-filled duodenum. However, these findings are not specific for pancreatitis, and the sensitivity of radiography for diagnosing pancreatitis is low in cats. Ultrasonography may reveal a hypoechoic pancreas, hyperechoic mesentery, a mass effect, a dilated common bile duct, or a normal pancreas. In previous studies, the sensitivity of ultrasound for diagnosis of pancreatitis was reported to be 24%. In a recent study, mild pancreatitis was still shown to be difficult to diagnose via abdominal ultrasound. However, in that same study, ultrasound had an 80% sensitivity, and an 88% specificity in cats with moderate to severe pancreatitis. In humans, the “gold standard” for a noninvasive diagnosis of pancreatitis is CT, but in this study, only 2 of the 10 cats showed evidence consistent with pancreatitis, and there was large variability in the ability of this imaging technique to assess pancreatic size. Thus, the cost and availability of CT and the difficulties in imaging the normal feline pancreas using this method make it less attractive for use in the diagnosis of feline pancreatitis.

The most reliable method for making an accurate diagnosis of pancreatic disease remains confirmation of inflammation on histopathology. However, this method can be expensive and can increase the risk of complications (anesthesia/surgery); in addition, in cases with focal lesions, the lesions may be missed on visual or histopathologic inspection. Thus, although biopsy is an important tool, it cannot be used in all cases, and if the biopsy reveals a normal pancreas, focal or chronic segmental pancreatitis cannot be ruled out. The are several primary histopathologic differences between acute pancreatitis and chronic pancreatitis in cats. Acute pancreatitis is characterized by neutrophilic or lymphoplasmacytic inflammation, with edema and fat necrosis. Chronic pancreatitis is characterized by the absence of inflammation, fibrosis of pancreatic tissue, and cystic degeneration with zymogen depletion. The lesions of chronic pancreatitis were more prominent in the left limb of cats with concurrent GI
disease. The chronic form of pancreatitis in cats resembles the chronic form in humans, where pain management using opioids and stent placement are key aspects of therapy.

**Treatment of Acute Pancreatitis**
Acute necrotizing pancreatitis in cats can be a significant therapeutic challenge. As with the treatment of dogs, the therapy is supportive and aimed at restoring circulating blood volume while allowing the pancreas to “rest.” If an inciting cause can be identified, it should be corrected; however, more than 90% of cases are idiopathic. The mainstay of treatment is aggressive fluid therapy, and if the cat is vomiting, withholding food and water for two to three days. Colloid support can be obtained with hydroxyethyl starch (Hetastarch) or plasma if it is available. If the cat is unable to tolerate water or food after the two- to three-day period, alternative routes of nutritional support must be considered to prevent development of hepatic lipidosis or protein/calorie malnutrition and immunosuppression. If the cat is not vomiting, placement of an esophagostomy (E) or percutaneous endoscopic gastrostomy (PEG) tube are reasonable alternatives, especially in cats with known or suspected hepatic lipidosis as a concurrent problem. In vomiting cats, either parenteral IV nutrition or placement of a jejunal feeding tube is optimal. The key point is this: you cannot starve cats with pancreatitis. In cats with chronic, low-grade pancreatitis, this is an even more important aspect of long-term management.

Another important aspect of therapy that must be considered in cats with pancreatitis is pain management (whether or not they show overt pain). Careful palpation in most cats with significant pancreatic inflammation will reveal cranial quadrant pain. Pain relief can be achieved with buprenorphine (0.005–0.01 mg/kg IV, or IM q4–8 hr), meperidine (1–2 mg/kg IM q2–4 hr), or butorphanol (0.2–0.4 mg/kg IM q2–4 hr). In addition, low-dose CRI ketamine or lidocaine infusions are effective in reducing somatic pain, and lidocaine at these low doses has prokinetic activity. Morphine should be avoided, as it can cause pancreatic duct spasm. The other aspects of supportive therapy to consider are antibiotic therapy, control of vomiting, and anticoagulants (for cats in DIC). Antibiotic therapy is generally indicated in all cats with severe pancreatitis or in cats with systemic inflammatory response syndrome (SIRS), as the risk of bacterial translocation and secondary sepsis are considerable. In general, broad-spectrum antibiotics that cover intestinal aerobes and anaerobes should be chosen. Cefotaxime at a dose of 50 mg/kg administered intramuscularly every 8 hours prevents bacterial colonization of the pancreas.

**Chronic Pancreatitis**
Therapy of chronic pancreatitis is somewhat controversial because there are no evidence-based studies yet available reporting specific therapeutic approaches that are beneficial. Many have advocated the use of steroid therapy (methylprednisolone), and in some cats with chronic pancreatitis this may be reasonable, where LP inflammation is the primary problem causing clinical signs. However, in end-stage cats where fibrosis and pancreatic degeneration, not inflammation, is occurring, steroids would be expected to be counterproductive. At this time, appetite stimulation (using mirtazapine) and pain control (buprenorphine) are the most commonly recommended therapeutic approaches initially. If the cat is nauseous, antiemetic therapy (short term) with maropitant may be quite helpful. Finally, fluid therapy (SC or IV, as indicated), antioxidants (SAMe), and appropriate dietary and probiotic therapy (as for IBD) are helpful. Pancreatic biopsy is the most effective means of providing the information needed to determine the best course of therapy in these cats. Further work on the underlying causes of this disease is needed to better define therapy in the short or long term.

**Nutritional Therapy of Pancreatitis**
The diet chosen should be highly digestible and palatable, but the concept of using a low-fat diet to reduce the stimulation of pancreatic secretions is not recognized as an important aspect of therapy in cats (as it is in dogs) because of the different causes and histologic types of pancreatitis. Ultimately, the goal is to find an appropriate diet for the cat that is highly digestible, commercially available, and acceptable to the cat. An important point about feeding cats during this period is to avoid force feeding, not only because it is very difficult to achieve the appropriate level of caloric intake by this method, but also because it can induce food aversion. At this time, a high-protein, low-carb diet would also likely be beneficial to reduce the workload on the pancreatic beta cells from a
high-starch diet. Furthermore, a diet that would be appropriate for a cat with IBD is reasonable and may be helpful. Lastly, supplementation of cobalamin, if indicated by measurement of cobalamin levels, is also important.

References