



# Diagnosing and Treating Pancreatitis

A roundtable discussion

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## Diagnosing and treating pancreatitis

Using the latest diagnostic tests, practitioners are detecting acute pancreatitis more frequently today than ever before. Early diagnosis is critical for the successful management of this devastating disease.

**Dr. Roberta Relford:**

We've met here today to talk about pancreatitis. Let me start by asking, "What is pancreatitis?"

**Dr. Jörg Steiner:** Pancreatitis is an inflammatory disease of the pancreas. It can be associated with different types of inflammatory cells. You could also define it as an autodigestion of the pancreas followed by inflammation.

**Relford:** Which comes first—autodigestion or inflammation?

**Steiner:** If you define autodigestion as premature enzyme activation, then autodigestion would occur first, before cytokines recruit inflammatory cells.

**Relford:** Is there a bacterial component to pancreatitis?

**Steiner:** As far as we know, there isn't a bacterial component in dogs and cats, but there appears to be one in some human patients with pancreatitis. However, the bacteria, which usually originate from the intestine and are disseminated in the bloodstream, do not cause clinical problems in people until three to four weeks after the pancreatitis begins.

**Relford:** Are all pancreatitis cases the same?

**Dr. David Twedt:**

Absolutely not. In fact, pancreatitis can vary from mild to life-threatening—and anything in between. That makes it difficult for practitioners to determine whether patients have acute gastroenteritis that will improve within 24 hours or acute pancreatitis. In dogs, we see more cases of acute pancreatitis. I don't think we're good at recognizing chronic cases.

**Dr. David Williams:**

Classically, the differentiation of acute and chronic pancreatitis is a histologic one. But defining the clinical disease itself is much more complicated than that because it's usually not feasible to obtain pancreatic biopsy samples. In addition, some cases are subclinical, and even in clinical cases, lesions may not be identified without a thorough examination of multiple pancreas samples at necropsy.

**Relford:** In acute vs. chronic pancreatitis, where does fibrosis come into play?

**Williams:** The difference between acute and chronic pancreatitis is that chronic cases involve irreversible changes to the structure of the pancreas—most commonly, fibrosis associated with some loss of function. Acute pancreatitis is a fully reversible disease, and recovery is associated with return to normal function.

However, we obviously can't obtain pancreatic biopsies in most clinical cases, so strict differentiation between acute and chronic disease is often impossible.

### Prevalence

**Relford:** How prevalent is pancreatitis?

**Steiner:** The true prevalence is unknown. The classic study conducted in Germany about 20 years ago showed exocrine pancreatic lesions in 1.7% of dogs and 1.3% of cats. Two-thirds of those lesions were pancreatitis.<sup>1</sup> However, some of the animals had only one section examined. Today we know that pancreatitis can be highly localized in any species, so researchers conducting that study 20 years ago may have missed many of the pancreatitis lesions.

A recent study conducted at The Animal Medical Center in New York<sup>2</sup> involved more than 200 dogs that were necropsied for reasons unrelated to suspected pancreatitis. Investigators sampled the pancreases every two centimeters and found lymphocytic infiltration in 52.5% of all cases and neutrophilic inflammation in 31.7% of all cases. But the question is: Which ones were clinically significant? It's unlikely that 52% of all dogs have significant pancreatitis.

**Williams:** Whatever the prevalence is, pancreatitis has

been dramatically under-recognized in the past—especially in cats. I think we are recognizing it more and more in dogs.

**Relford:** I agree. The prevalence of pancreatitis probably isn't increasing—we're just detecting it more often.

### **Clinical signs**

**Relford:** What are the clinical signs of pancreatitis in animals?

**Dr. Benita von Dehn:** The classic signs in dogs include anorexia, vomiting, and abdominal pain. Vomiting is more common in dogs than cats. Additional signs may include weakness, nausea, drooling, depression, diarrhea, and, occasionally, a fever. In cats, we tend to see more prolonged anorexia, lethargy, and weight loss. The difficulty arises in the fact that these clinical signs are also often associated with other diseases.

**Williams:** Pancreatitis isn't always an isolated disease. Increasingly, we're recognizing cases where patients have not only pancreatic lesions but also intestinal, gastric, or hepatic lesions. So the signs may have one or multiple causes, especially in cats.

**Twedt:** The abdominal pain we usually associate with pancreatitis does not occur in all cases. Some studies say approximately 50% exhibit abdominal pain.<sup>3</sup>

**Steiner:** In reality, practitioners could suspect pancreatitis with any sick patient. Recently, a friend of mine consulted with me on his older miniature schnauzer. For years, my friend went

to different veterinarians, trying to figure out why the dog wouldn't play every day. There were no other clinical signs. We diagnosed chronic pancreatitis and administered treatment, and the dog plays every day now. In fact, my friend says he has a puppy again.

Practitioners may think that clinical signs in dogs are more severe and easier to spot than the more nonspecific clinical signs found in cats. But if you look at the entire spectrum of the disease, I'm sure we would find plenty of dogs with unusual and atypical clinical signs that don't suggest pancreatitis.

**Relford:** Dr. Williams, you mentioned subclinical pancreatitis cases earlier. Is there anything practitioners can look for in patients without clinical signs?

**Williams:** Some endocrinologists recommend that veterinarians screen diabetic animals for concurrent pancreatitis because this is a potential aggravating factor that makes diabetes management more difficult. That's one example where it's reasonable to screen patients without signs of pancreatitis.

Pancreatitis isn't always an isolated disease. Increasingly, we're recognizing patients with intestinal, gastric, or hepatic lesions—not just pancreatic. So the signs may have one or multiple causes, especially in cats.



**David Williams**

**Steiner:** It depends on how aggressive veterinarians want to be. Fifty years ago, animals diagnosed with renal disease were in end-stage renal failure. That's because creatinine usually elevates before a patient shows clinical signs. By the time the clinical signs appear, about 80% of the kidney is gone. Today practitioners look for markers of early kidney disease; they don't wait for clinical signs to appear before they run tests. Every chemistry profile includes creatinine and BUN analyses. When I'm on the clinic floor, we run a chemistry profile on every dog with suspected renal disease—whether it's clinical or subclinical.

Why should it be different for pancreatitis? Like renal disease, pancreatitis may start long before the clinical signs appear. In 20 or 30 years, I think we'll be screening for pancreatitis the same way we screen for renal or hepatic disease today.

**Twedt:** Fortunately, we have found ways to prolong survival in patients with renal disease. If practitioners can anticipate complications with pancreatitis, they can try to slow down the disease's progression.

**Relford:** Is it fair to say that the earlier a clinician identifies pancreatitis, the better the likelihood for the patient's survival?

**Steiner:** That's a fair statement.

### **Clinical signs of pancreatitis**

In dogs, the classic clinical signs of pancreatitis are:

- anorexia
- vomiting
- abdominal pain.

Other signs may include:

- nausea
- drooling
- depression
- diarrhea
- fever.

Cats usually exhibit signs of anorexia, lethargy, and weight loss.

Diagnosing pancreatitis can be difficult because these signs are often associated with other diseases.

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**Williams:** I think it depends on the group of animals you're evaluating. There are dogs with chronic pancreatitis that are not going to die from it, but heightened awareness can help practitioners prevent acute flare-ups. If practitioners recognize severe acute pancreatitis early, they can institute aggressive supportive therapy for this potentially fatal disease sooner.

**Twedt:** Absolutely. Pancreatitis mimics so many other diseases. If practitioners can identify pancreatitis, they can direct their efforts to managing it and not looking for a liver problem, gastroenteritis, or inflammatory bowel disease.

**von Dehn:** I would add that patients might have a better prognosis if practitioners can identify and remove an inciting or incriminating agent.

**Williams:** That's a good point. Specific causes

may not be identified frequently, but when they are recognized, practitioners can manage them.

**Steiner:** But it's also important to step back and take a more general look. One could make the statement that if a specific cause cannot be identified, then outcome is really dependent on the presence of systemic complications (*e.g.*, hypoperfusion, disseminated intravascular coagulation [DIC], pulmonary failure, renal failure) and pancreatic complications (*e.g.*, pancreatic necrosis and abscesses) and not so much on inciting causes.

### **Associated factors** Breed, age, sex

**Relford:** Which breed, age, or sex characteristics are associated with pancreatitis?

**von Dehn:** In general, pancreatitis tends to occur in middle-aged, neutered dogs

with no gender preference. Dogs with pancreatitis are often overweight. Terrier and nonsporting breeds tend to be overrepresented. Cats diagnosed with pancreatitis tend to be older, neutered, domestic shorthaired breeds with no obvious gender predilection.

**Steiner:** I generally find the same tendencies. But I now believe that cats of any age can develop pancreatitis. When we studied a group of 20 cats with exocrine pancreatic insufficiency, which we assumed was caused by chronic pancreatitis, three of them were less than 6 months old. The cats must have experienced enough pancreatitis in those first six months of their lives to destroy their pancreases. That changed my mind.

**Relford:** So is it fair to say that pancreatitis can occur in animals of any age, breed, or sex, even though there's a

higher incidence in the populations Dr. von Dehn described?

**Williams:** Yes, although there is little evidence to support that statement. There is certainly anecdotal evidence of a predisposition in miniature schnauzers, and there is published evidence of a genetic component predisposing German shepherds to chronic lymphocytic pancreatitis, which can lead to pancreatic insufficiency.<sup>4</sup>

### **Drugs**

**Relford:** Are any predisposing factors drug-related?

**Williams:** Yes. Potassium bromide and many chemotherapy agents are implicated.

**Steiner:** I would say potassium bromide is the biggest factor. We've seen pancreatitis in 6.4% of patients treated with potassium bromide. That is probably higher than with L-asparaginase or vinca alkaloids—the big cancer drugs that can cause pancreatitis. I suspect the second-highest drug-related factor for pancreatitis is L-asparaginase.

Our studies with potassium bromide show that the association with pancreatitis is not dose-dependent. That also suggests that the pancreatitis is probably an idiosyncratic reaction.

**Twedt:** We occasionally see animals that we believe have pancreatitis associated with azathioprine.

**Steiner:** In human medicine, doctors follow a more systematic reporting method. The list now contains 54 drugs and drug classes suspected of causing pancreatitis. Virtually

### **Associated factors**

#### **Drugs**

Although any drug can potentially cause pancreatitis, the most common associations involve:

- potassium bromide
- chemotherapy drugs, especially L-asparaginase
- antibiotics, especially tetracycline and sulfonamides
- organophosphates.

#### **Diseases and medical conditions**

A cause-and-effect relationship has been found between pancreatitis and the following:

- feline viral diseases (*e.g.*, calicivirus infection),

toxoplasmosis, and a hepatic fluke infection

- immune-mediated disorders
- hypercalcemia.

#### **Other factors**

Animals may also develop pancreatitis from:

- high-fat and, possibly, low-protein diets
- a genetic predisposition
- surgical interference secondary to hypovolemic, hypotensive, or ischemic insults to the pancreas
- trauma.

any drug you can imagine is on that list. I would go as far as saying that any drug can potentially cause pancreatitis, just like any drug can potentially cause acute hepatic necrosis through an idiosyncratic reaction.

**Williams:** Years ago, we saw quite a few cats develop pancreatitis after being treated with tetracyclines.

**Steiner:** That association has been reported in people, too. A whole range of antibiotics is associated with pancreatitis, but tetracycline and the sulfonamides are the most common.<sup>5</sup>

There have also been reports linking organophosphates to pancreatitis in cats.<sup>6</sup>

**Relford:** Just in cats?

**Steiner:** It hasn't been reported in dogs, but I would assume that treating a dog with organophosphates would pose the same risk. In addition to cats, children in third world countries have developed pancreatitis from organophosphates. I can't see why dogs wouldn't be affected.

**Twedt:** What about corticosteroids? The literature often references them as potential causes of pancreatitis. But if you think about all the animals treated with corticosteroids, the connection with pancreatitis is minimal.

**Williams:** There is certainly evidence that corticosteroids in dogs cause serum lipase activity to increase, and while veterinarians used to think there was a pancreatic association, there are no pancreatic lesions in treated animals.<sup>7</sup> The lipase must be

another type of lipase, possibly hepatic.

**Steiner:** In human medicine, corticosteroids have been removed from the list of drugs causing pancreatitis; however, a lot of patients on steroids have diseases that predispose them to pancreatitis, so the primary disease process may be at fault, not the steroids administered to treat those diseases.

### Diseases

**Relford:** Are there any diseases in dogs that predispose them to pancreatitis?

**Williams:** In the literature, pancreatitis is often listed as a complication of hyperadrenocorticism,<sup>8</sup> but, over the years, I have not seen that association.

**von Dehn:** Infectious etiologies have been described more often in cats, unlike dogs.<sup>9</sup>

**Steiner:** One may think that bacterial infections can cause pancreatitis in cats, but, actually, the two have never been associated. Instead, feline pancreatitis is associated with certain viral diseases (including calicivirus infection), toxoplasmosis, and

I would go as far as saying that any drug can potentially cause pancreatitis, just like any drug can potentially cause acute hepatic necrosis through an idiosyncratic reaction.



infection with a hepatic fluke called *Amphimerus pseudofelineus*. Feline infectious peritonitis, feline immunodeficiency virus infection, feline leukemia virus infection, and panleukopenia have also been reported to cause pancreatitis, but a cause-and-effect relationship has not been demonstrated.

**Williams:** In many of those cases, however, pancreatitis is only part of the picture because there are often concurrent diseases, especially in cats.

**Steiner:** I also think we should mention immune-mediated pancreatitis. Twenty years ago, the disease was unknown in people, and now it's considered fairly common. If you look at the progression in people, you can almost predict the same progression in animals. I would bet that 20 years from now, veterinarians will be saying, "Yes, a lot of these idiopathic cases—past and present—are actually immune-mediated disorders." However, it will take a lot of studies to define the disease in dogs and cats.

**Williams:** At this point, it has been shown that immune-

mediated lymphocytic pancreatitis predisposes German shepherds to pancreatic atrophy.<sup>4</sup>

**Steiner:** One may think this differentiation of idiopathic and immune-mediated pancreatitis is merely academic; however, it is really huge: Idiopathic pancreatitis can't be treated, but immune-mediated pancreatitis can be treated with corticosteroids.

### Medical conditions

**Relford:** Are any other medical conditions considered to be predisposing factors?

**Steiner:** Hypercalcemia causes pancreatitis. I see this quite frequently in the clinic. Dogs that present with a serum calcium concentration of 14 mg/dl or higher may have concurrent pancreatitis. Of course, lymphoma or an anal sac adenocarcinoma is usually the underlying cause of the hypercalcemia.

**Williams:** We test numerous blood samples from diabetic animals, and some of them show evidence of chronic pancreatitis or acute pancreatitis flare-ups, which may predispose the animals to periods of insulin resistance.

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Roberta Relford

In chronic cases, serum chemistry profiles can be totally normal. Practitioners need to watch for occasional vomiting, loss of appetite, weight loss, or unusual behaviors.

**von Dehn:** Wouldn't everyone agree that evidence suggests that obese dogs on low-protein, high-fat diets are potentially predisposed to pancreatitis?<sup>10,11</sup>

**Williams:** Some animals with normal body weights develop pancreatitis, but there is evidence from experimental studies that obese dogs will get more severe pancreatitis than underweight dogs, and obesity is certainly a potential risk factor for complications of pancreatitis.

**Relford:** So does diet play a role?

**Williams:** Hyperlipidemia, which clients and veterinarians can modify with diet, almost certainly does. We believe that it's associated with pancreatitis in schnauzers, but I don't know if it's been proved that hyperlipidemia predisposes animals to pancreatitis.

**Relford:** So what about those animals that are fed bacon in the morning and presented to the veterinarian in the afternoon with pancreatitis?

**Williams:** Some of them have pancreatitis, but many of them probably have non-specific gastroenteritis.

**Steiner:** There are two sides to the fat issue. Of course there is a clinical impression that most veterinarians share: High-fat meals cause pancreatitis. However, there is little scientific evidence for a cause-and-effect relationship. Only one study has been done that evaluates the relationship between a high-fat diet and pancreatitis.<sup>11</sup> In this study, a group of healthy dogs were fed pork lard exclusively for six weeks. These dogs developed pancreatitis, but they died of hepatic necrosis. Unfortunately, I don't think the study is clinically relevant to the patients practitioners see every day.

However, I do believe that diet plays a role because dietary management in many of these dogs is quite successful, whether it's for acute or chronic pancreatitis.

**Williams:** Diets extremely low in protein have been shown to predispose animals to pancreatitis, although it's rare.<sup>11</sup> Dr. Steiner hit on the bigger point: Patients with chronic pancreatitis often respond to low-fat diets.

**Relford:** So I'm hearing that diet plays an important role in managing pancreatitis, but it rarely causes pancreatitis. If a happy, healthy dog runs around wagging its tail one day and the next day gets pancreatitis, what flipped the switch?

**Williams:** Those cases are mostly idiopathic. There's one study that says obesity is a predisposing factor,<sup>12</sup> but the control group might have been underweight. The pancreatitis population was heavier than the nonpancreatitis population, but I'm not sure they were really obese. That's why I'm hesitant to agree with the study's assertion. However, there are many other reasons to try to control obesity.

### Other factors

**Relford:** Are there any other predisposing factors we haven't mentioned?

**von Dehn:** In addition to high-fat diets and maybe low-protein diets, practitioners should consider hereditary causes in dogs, as they have been implicated in the human literature.<sup>10</sup>

Pancreatitis may also be induced by surgical interference secondary to hypovolemic, hypotensive, or ischemic

### Serum chemistry findings associated with pancreatitis

- Variable changes in amylase and lipase activity (elevated lipase may be present in about 50% of active pancreatitis cases)
- Liver enzyme elevation (may reflect underlying endotoxemia, dehydration, or both)
- Elevated BUN and creatinine levels (most often secondary to hypovolemia, dehydration, or acute renal failure)
- Hypocalcemia (usually secondary to concurrent hypoalbuminemia)
- Hyperglycemia (may be due to hyperglucagonemia, stress, or destruction of islet cells by ongoing pancreatitis)
- Hyperbilirubinemia (secondary to severe hepatocellular damage, intrahepatic or extrahepatic obstruction of bile flow, or both)
- Hypoalbuminemia (usually due to vascular and peritoneal leakage)
- Hypercholesterolemia
- Hypertriglyceridemia

insults to the pancreas. Even a trauma, like getting hit by a car, could predispose an animal to pancreatitis by causing pancreatic hypoxia.

**Williams:** That's true. Cats that have fallen out of high buildings have developed pancreatitis, which is probably related to subsequent shock and hypovolemia.

**Twedt:** It's also a widespread misconception that touching the pancreas causes pancreatitis. In fact, it's unusual to see pancreatitis develop from either a laparoscopic or surgical biopsy of the pancreas. If it happens, it's probably because of vascular changes, hypovolemia, or cytokine release.

**Steiner:** Doctors in human medicine used to believe the same thing. Now they know they're seeing hypoperfusion of the pancreas during anesthesia. As it turns out, there is an increased rate of pancreatitis in human patients who have undergone abdominal surgery, open-heart surgery, and leg amputation. Of course, animals can develop pancreatitis if the surgeon traumatizes the pancreas, but gentle handling of the pancreas is unlikely to cause pancreatitis.

## **Diagnostics** Blood tests

**Relford:** If an animal presents with anorexia, abdominal pain, vomiting, or other gastrointestinal signs, what tests should practitioners perform first?

**von Dehn:** I don't assume it's pancreatitis, but it's definitely on the differential list. Every dog receives a complete

physical examination and a minimum database evaluation, including a complete blood count (CBC), serum chemistry profile, and urinalysis. A pancreatic lipase immunoreactivity (PLI) test may be included. I use the CBC and serum chemistry profile to check for systemic complications that might indicate more severe underlying problems, such as thrombocytopenia or hypocalcemia. I also recommend radiographs of the chest and abdomen to rule out other causes of anorexia and vomiting, such as foreign bodies, and to check for pleural

serum chemistry findings are associated with pancreatitis (see the sidebar on page 6).

**Steiner:** But pancreatitis isn't like kidney failure, where you can rule it out just by finding a normal serum creatinine concentration. With pancreatitis, it's more complicated; the findings aren't always cut and dried. If practitioners don't find the signs Dr. von Dehn just mentioned, it only makes pancreatitis less likely.

**Relford:** So if the results of a minimum database are not consistent with acute

**Relford:** In chronic cases, serum chemistry profile results can be totally normal. Practitioners need to watch for clinical signs, which we mentioned earlier, including loss of appetite, occasional vomiting, weight loss, or unusual behaviors.

**Williams:** In patients with suspected chronic pancreatitis or chronic small intestinal disease, I would combine an evaluation of the pancreas with serum cobalamin and folate tests, which are good screening tests for chronic small intestinal disease.

I get fooled all the time by patients with high bilirubin, abnormal liver enzymes, and vomiting. I assume it's liver disease, but additional diagnostics reveal pancreatitis as the primary cause.



**David Twedt**

effusion or other thoracic abnormalities. After reviewing the radiographs, I usually pursue ultrasonography, if indicated.

**Relford:** What should practitioners look for in a CBC and serum chemistry panel?

**von Dehn:** Inflammatory or infectious leukograms are a common laboratory change in patients with severe acute pancreatitis. Mild elevations in the packed cell volume are likely a result of dehydration and will often decrease with fluid therapy.

In addition, numerous

pancreatitis, that doesn't mean pancreatitis isn't present.

**Twedt:** Absolutely. I get fooled all the time by cases where the bilirubin is high, the liver enzymes are abnormal, and the animal is vomiting. I assume it's liver disease. But additional diagnostics reveal pancreatitis as the primary cause; the liver problems are secondary to the pancreatitis. Practitioners have to rule out the other diseases first because a minimum database or radiograph won't necessarily indicate pancreatitis, and pancreatitis mimics many other diseases.

**Steiner:** Let's face it, most of the abnormalities in a serum chemistry profile are not caused by pancreatitis; they are changes associated with the complications from pancreatitis. Practitioners won't find those changes unless the pancreatitis is severe enough to cause those complications.

**Relford:** What about lipase and amylase activities? Animals with pancreatitis may or may not have elevated levels.

**Twedt:** We still run amylase and lipase at our hospital. If all other clinical parameters and laboratory findings suggest

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pancreatitis, it may help support that diagnosis. It's just another piece of the puzzle.

**Williams:** Elevated amylase and lipase activities mean something is wrong with the animal, but it's most likely gastrointestinal, hepatic, or renal disease. Unless activities are severely increased, pancreatitis is possible but not probable.

**Steiner:** I wouldn't limit it to those three diseases. The list of conditions that could trigger false elevations of serum lipase activity in small animals is endless—hepatic disease, renal disease, sepsis, cancers, pulmonary failure, and muscle activity are just a few. Nevertheless, veterinarians can use it as a guideline, but in my mind, it isn't acceptable for a definitive diagnosis.

**von Dehn:** If the CBC and serum chemistry profile don't help with the diagnosis of pancreatitis and I am unsure about serum amylase and lipase activity, I may run a serum PLI test. This test is more sensitive and specific than any other test, including trypsin-like immunoreactivity (TLI).

**Relford:** Dr. Williams, could you comment on using TLI to diagnose pancreatitis?

**Williams:** We've been using TLI for more than 20 years to diagnose pancreatic insufficiency, and it remains an excellent test. When the test became available, I was hoping we could use it to diagnose acute pancreatitis. It didn't take long for us to realize its limitations. Currently, it identifies pancreatitis in 33% to 66% of affected animals. In

cats, it's much better than testing for amylase and lipase activities, but it falls far short of what practitioners would like to see. But it's excellent for diagnosing pancreatic insufficiency in dogs and cats.

**Relford:** Amylase and lipase tests can produce false positives and negatives. Are TLI elevations usually consistent with pancreatitis?

**Williams:** TLI elevations can occur in animals with severe renal failure because trypsinogen is filtered by the kidneys. Before increases in serum TLI become apparent in patients with renal failure, creatinine must increase to at least 3.5 mg/dl in cats and 4.5 mg/dl in dogs. In most patients, TLI values remain normal in the face of renal failure. If practitioners obtain high TLI levels, they need to look at renal function and follow up with a PLI test to confirm or eliminate possible pancreatitis. Perhaps most importantly, normal TLI levels do not rule out pancreatitis.

There are certain things we don't know about PLI either. But if practitioners find a high PLI, there's a good chance the animal has pancreatitis because concentrations do not increase with renal failure.

**Steiner:** Veterinarians must remember that there's a difference between lipases measured by a PLI test and those measured by a serum chemistry profile. There are many different lipases in the body. The PLI test is based on an immunoassay and evaluates the molecular concentration; it is not based on enzymatic function. A serum chemistry profile may pick up any lipase circulating in the blood,

whether it's from the liver, stomach, or pancreas.

**Twedt:** I use PLI tests in cases where I suspect pancreatitis and want to confirm it. For example, I use it in acute pancreatitis cases that I'm treating symptomatically, subtle chronic cases where I'm not sure what's going on (especially in cats), chronic gastrointestinal disease patients, diabetic patients that aren't doing well or don't respond to therapy, patients that may have underlying pancreatitis, or patients with acute ketoacidosis.

**Relford:** Should we be testing for pancreatitis more often to rule it out or to see if it's occurring with other diseases?

**Steiner:** It's sort of a standing joke at Texas A&M that, whenever I see a nontypical patient, I run a PLI on it. When those patients with suspected pancreatitis have normal PLI tests, we look further and often find a different disease process. One time, we did a feeding trial using veterinary students' pets. To be sure the six normal control dogs were healthy, we ran a CBC, serum chemistry profile, and the newly released PLI. Two of the PLI tests were abnormal, and one showed a marked elevation. We asked the student if the dog was exhibiting any clinical signs of pancreatitis. She said the dog was happy and healthy. An ultrasound confirmed pancreatitis without a doubt. After two weeks on a new diet, the student realized that her dog hadn't always eaten breakfast before but was now eating it every day. If a veterinary student can't detect clinical signs, how do we think our clients can? I truly believe that practi-

tioners will find many cases of subclinical pancreatitis. And any case of subclinical pancreatitis can lead to diabetes, pancreatic insufficiency, or a potentially fatal acute episode.

**Relford:** So the PLI test is helpful for diagnosing clinical or subclinical pancreatitis that may or may not produce ultrasonographic findings. This could help practitioners improve the outcome for pancreatitis patients or rule out pancreatitis and look for other diseases.

**Twedt:** Absolutely. If you don't look for the disease, you can't diagnose it. A practitioner might identify low-grade pancreatitis in an animal with nonspecific gastrointestinal signs. It may not change the overall management of the case, but it alerts the practitioner about what to expect long-term.

**Williams:** Yes. It may not require immediate action, but it makes the practitioner and owner aware that more severe signs of pancreatitis may surface sooner or later.

**Relford:** Dr. Williams, you've worked closely with IDEXX Laboratories during the last few years on the Spec cPL (canine pancreas-specific lipase) test, a modification of the canine PLI assay. Will the increased availability of this assay alter the way practitioners handle pancreatitis cases?

**Williams:** I'm sure that it will. It's a new test using a relatively new technology, but the evidence to date clearly shows that it surpasses the other available options (data on file, IDEXX Laboratories). It will be a useful tool for expanding our



knowledge of pancreatitis and refining patient management in the future (Table 1).

**Relford:** So practitioners using this new test shouldn't be startled if they start finding more

animals with elevations in pancreatic lipase concentration.

**Williams:** I'm sure the test will help practitioners find more pancreatitis cases and gain an increasing apprecia-

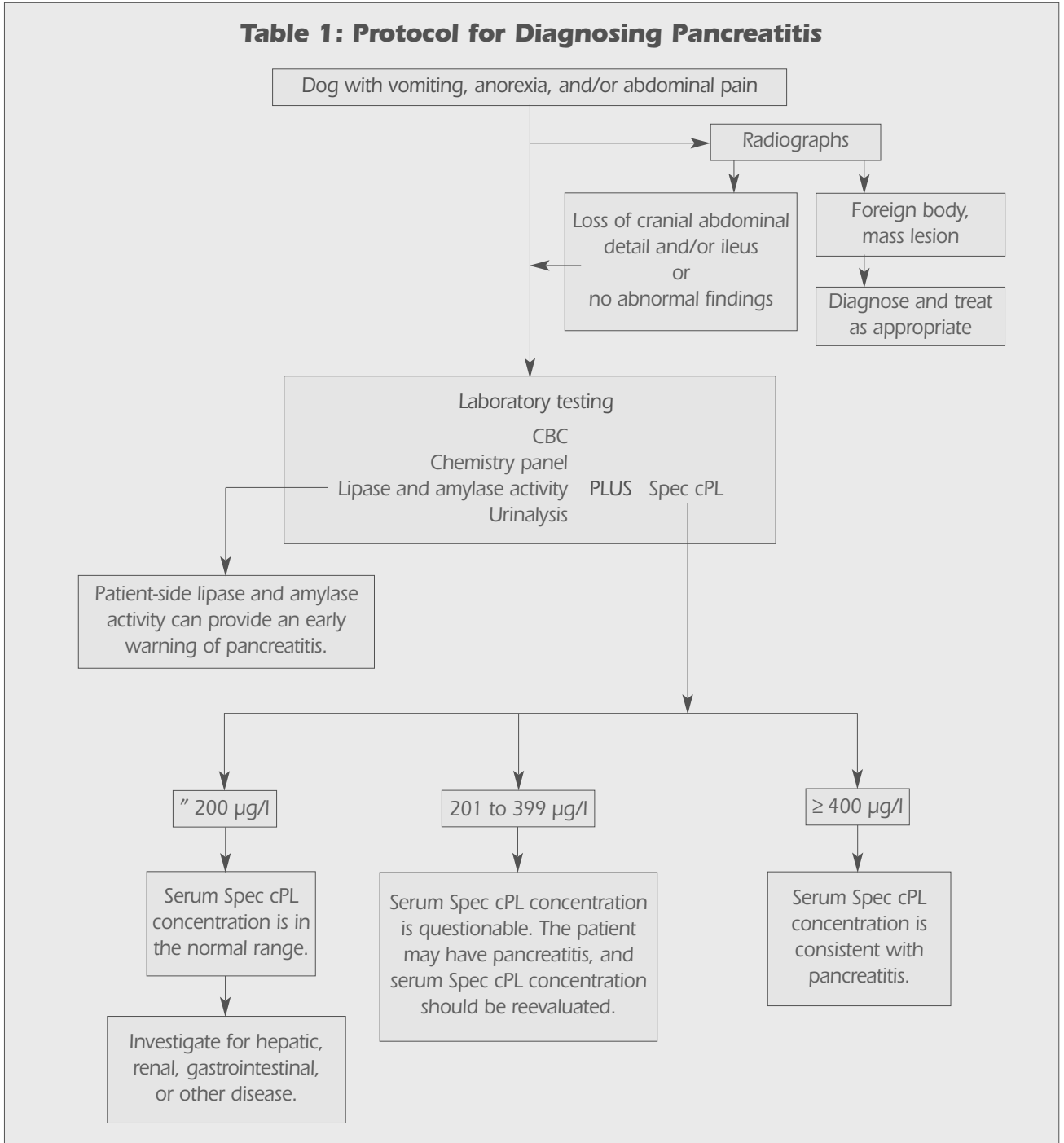
tion for those patients that have atypical clinical signs.

**Relford:** Can the PLI test be used for cats, and is it available?

**Steiner:** The PLI test for

cats is available only through the gastrointestinal laboratory at Texas A&M University ([www.cvm.tamu.edu/gilab](http://www.cvm.tamu.edu/gilab); 979-862-2861). The test for cats is completely different from the test for dogs.

**Table 1: Protocol for Diagnosing Pancreatitis**



When performing ultrasounds, practitioners don't see the normal pancreas in 100% of the animals. This can skew their reference point of a normal pancreas.



**Relford:** What about the Spec cPL test?

**von Dehn:** Currently, the Spec cPL test is available through IDEXX Reference Laboratories. The turnaround time is 12 to 24 hours.

### Imaging

**Relford:** When practitioners turn to radiographs and ultrasound for diagnosing pancreatitis, what should they look for?

**Saunders:** Practitioners should take abdominal and thoracic radiographs to rule out a radiopaque foreign body, signs of gastrointestinal obstruction, peritonitis, other causes of an acute abdomen, or a pleural effusion secondary to pancreatitis.

However, I rarely rely on abdominal radiographs to diagnose pancreatitis. I just don't find the classic radiographic signs of pancreatitis: widening of the gastroduodenal angle, loss of right cranial quadrant abdominal detail, loss of peritoneal contrast and detail, lateral displacement of the descending duodenum, gas in the descending duodenum, and gas in the transverse colon at the hepatic flexure. I use radiographs

mainly to rule out other possible disease processes.

On the other hand, ultrasound works well on dogs with severe acute pancreatitis that includes hemorrhage and necrosis. In those cases, the ultrasound will show a severely hypoechoic, almost mass-like lesion in the right cranial quadrant. The pancreas will be irregular and no longer visible as a distinct structure. There is substantial peripancreatic inflammation demonstrated by hyperechoic mesentery. There may or may not be a small volume of peritoneal effusion, either around the pancreas or throughout the peritoneal cavity. Finally, the duodenum may appear plicated and irritated, or it may appear static, without active contractions.

I also think it's important to look for extrahepatic biliary obstruction and portal vein thrombosis. This can be difficult—especially in animals that have a lot of pain from mesenteric inflammation. I have a hard time finding the portal vein and doing color flow Doppler on painful patients.

The cases without acute pancreatic hemorrhage and necrosis are less severe-looking. There may be duo-

denal motility and an absence of effusion. The pancreas is a hypoechoic structure surrounded by hyperechoic mesentery; it doesn't have that mass effect in the right cranial quadrant. Beyond that, I have found that ultrasound isn't useful for detecting low-grade, chronic, fibrosing pancreatitis.

Ultrasonographers are reported to detect pancreatitis (confirmed histologically) in about 30% of cats<sup>13</sup> and 68% of dogs.<sup>3</sup>

**Steiner:** Do you think the 68% figure would be higher today because we're using more modern equipment?

**Saunders:** Yes. I think the current sensitivity in severe acute cases is much higher. That study was conducted 10 years ago using some relatively inexperienced ultrasonographers—including me. Our knowledge and equipment have improved since then.

**Relford:** Does sensitivity rely on individual ultrasonographers?

**Saunders:** Very much so. There's a saying about ultrasound: "You find what you look for, and you only look for what you know." So unless you're clued in to the possibili-

ty of pancreatitis, you may not attach much significance to your findings. The more pancreases practitioners visualize and follow up on, the better they'll become.

The size of the pancreas and its location also make pancreatic ultrasounds difficult to perform. Unlike the liver, the pancreas is a small organ, and it's difficult to detect the pancreas on a normal abdominal scan. Second, the pancreas is shielded by several gas-containing structures—the stomach, duodenum, and colon. The right lobe is easier to visualize in a dog, and that's why practitioners typically detect pancreatitis in the right lobe. Practitioners don't see the normal pancreas in 100% of the animals; this can skew their reference point of what is a normal pancreas. I typically don't see the normal pancreas on all the dogs I scan, but I do scan the pancreatic region for abnormalities.

**von Dehn:** In my experience, the pancreas of normal dogs and cats is often difficult to recognize as a discrete structure on abdominal ultrasound because the echogenicity is similar to the adjacent mesenteric fat.

**Steiner:** As an internist, I cannot stress enough how important it is for practitioners to rely on experienced ultrasonographers. Buying a machine and completing a two-day course is not enough. Ultrasonography isn't the same as endoscopy or a bone marrow aspiration, where you know you've been successful. It doesn't matter if you diagnose pancreatitis unless you find out if you're right.

With that said, my current concern with ultrasound re-

volves around specificity. In the UC-Davis study, three cats that had been suspected of having pancreatitis had no evidence of it.<sup>14</sup> But in two of those cats, the ultrasonographer had diagnosed pancreatitis, so if you just look at those animals, the specificity would have been 33%. That number would concern me greatly.

**Saunders:** Cats are totally different because pancreatitis is much more subtle. Few cats have the blatant-looking, necrotic, hemorrhagic pancreatitis that I see in dogs. Unfortunately, many of the ultrasonographic criteria for pancreatitis in dogs have been applied to cats. In other words, practitioners want to see a hypochoic, irregular pancreas surrounded by hyperechoic mesentery. In my experience, that finding is rare in cats, and practitioners will miss many cases of pancreatitis if they use dog criteria.

I also find that cats with pancreatitis demonstrate less pain than dogs during ultrasounds. Cats just lie there and let you press on the pancreas.

As for statistics, two retrospective studies reported a 25% to 30% sensitivity in cats for ultrasonographic detection of pancreatitis.<sup>13,15</sup> The low sensitivity is probably clinically accurate. These were real-world cases that presented with non-specific clinical signs, and pancreatitis may not have even been on the differential list.

**von Dehn:** Is pancreatic duct measurement of any relevance when performing ultrasounds in cats?

**Saunders:** It looks like pancreatic duct size increases with normal aging, but it appears unlikely that we will

be able to use duct dilation as an indicator of pancreatitis.<sup>16</sup>

**Twedt:** What do you think about the role of ultrasound-guided aspiration in suspected cases of pancreatitis?

**Saunders:** Many people feel that we need to start aspirating more pancreases, especially in cats. If aspirations and biopsies are truly warranted, I think they should be performed by specialists, not general practitioners.

**von Dehn:** How would you proceed if a pancreatic mass or cystic lesion was identified?

**Saunders:** I will aspirate a mass or cyst. A cystic structure could be either an abscess or pseudocyst—they look similar on ultrasound. You need an aspiration to tell the difference. I sometimes see cysts in the pancreas of cats, and when I do, I suspect pancreatitis.

**Williams:** Have you ever had a complication from a needle aspiration of the pancreas?

**Saunders:** No.

**Williams:** The complication rate in human medicine is incredibly low, less than 3%, and those are nearly all minor.

**Saunders:** Right. I haven't performed that many, but when I have performed aspirations, they definitely benefited the outcome of the case. It's similar to aspirating a gallbladder—everyone is reluctant to do it, but I think it's an excellent diagnostic test.

**Twedt:** I think an aspiration is definitely warranted if there's a small amount of effusion. Fine-needle aspiration is

also helpful in determining if there's a tumor or just inflammation. If you aspirate carcinoma cells, it's neoplasia.

**Relford:** Is there anything else on the ultrasound that would help practitioners determine if it's worth performing an aspiration?

**Saunders:** I don't think an aspiration or biopsy is needed in pancreatitis cases with a lot of hemorrhage and necrosis. If you stick a needle into that, you'll probably just get hemorrhage and necrosis, and it may be hard to find a more viable section of the pancreas.

I would contemplate aspiration with the cases that don't look quite normal but are not obviously acute and severe. Maybe they don't have effusion. Maybe they have slight peripancreatic inflammation, and you're undecided about the diagnosis.

**Relford:** Should veterinarians perform fine-needle or core biopsies?

**Saunders:** A core biopsy would be more diagnostic. But it's a bit more invasive to perform a core biopsy because we generally use 14- or 16-ga needles. With fine-needle aspirations, 22-ga needles can be used.

**Relford:** What about computed tomography (CT)?

**Steiner:** Two studies have been done on CT scans in cats. They both found that CT is not very useful.<sup>14,15</sup>

## Treatment

### Acute pancreatitis

**Relford:** How should practitioners manage patients with acute pancreatitis?

**von Dehn:** One of the most important treatments for dogs and cats is intravenous crystalloid support, which helps restore pancreatic perfusion, corrects dehydration and electrolyte imbalances, and compensates for ongoing losses caused by vomiting or diarrhea. Another common treatment is colloid support,

## The role of imaging in diagnosing pancreatitis

### Radiographs

Take abdominal and thoracic radiographs to rule out:

- foreign bodies
- gastrointestinal obstruction
- acute abdomen
- pleural effusion.

### Ultrasound

Best if performed on dogs with severe acute pancreatitis that includes hemorrhage and necrosis. Practitioners will see:

- a severely hypoechoic, almost mass-like lesion in the right cranial quadrant
- an irregular pancreas that is no longer visible as a distinct structure
- substantial inflammation demonstrated by hyperechoic mesentery.

## Diagnosing and treating pancreatitis

usually in the form of fresh frozen plasma. We suspect that replacing  $\alpha_2$ -macroglobulins may help with pancreatic edema and hypoalbuminemia. Obviously, removal of a possible inciting agent is also instituted.

Instituting antibiotic therapy is controversial. I initiate antibiotic therapy if a potential for bacterial translocation from the gut exists. I also include analgesics in my treatment plan (e.g., butorphanol, fentanyl, or meperidine) because most of these dogs have discomfort and pain. If vomiting persists despite nothing per os, I administer antiemetics, such as metoclopramide continuous rate infusion.

**Relford:** Should practitioners feed animals with pancreatitis?

**von Dehn:** We used to withhold food and water for long periods—often longer than five days. We now realize that this may lead to gastrointestinal villus atrophy and ileus. Therefore, I now attempt enteral feeding (either by mouth, with an esophagostomy tube, or with trickle feeding through a nasogastric tube) as soon as the vomiting has subsided, which is usually by the third day.

If enteral feeding is tolerated, it has advantages over intravenous total or partial parenteral nutrition and jejunostomy tube placement, which are more costly procedures that require an experienced staff. Enteral feeding is thought to stabilize the gut barrier and improve intestinal motility, decreasing the need for antibiotics and, pos-

sibly, decreasing the length of hospitalization.

**Twedt:** I agree that withholding food is an outdated theory. We don't do it either. The pancreases in animals with severe or acute pancreatitis probably don't produce additional enzymes that make the pancreatitis worse. Withholding food can lead to substantial catabolic deficits, a higher susceptibility to bacterial intestinal translocation, decreased protein levels, and decreased immune function and neutrophil function. I think nutrition is extremely important.

**Steiner:** I agree as well. There is no evidence whatsoever that withholding food has any beneficial effect. Human doctors believed that prema-

ture enzyme activation caused pancreatitis, which led to the theory about inhibiting pancreatic function and resting the pancreas.

**Twedt:** In severe pancreatitis patients, I insert a feeding tube, usually a nasogastric, gastrostomy, or esophageal tube. If you're performing a surgical procedure, you can sometimes bypass the vomiting problem by inserting a jejunostomy feeding tube.

**Steiner:** The advent of the antiemetic drug dolasetron (0.3 to 0.6 mg/kg IV, SC, or orally once or twice daily) has dramatically changed the way I approach these patients. Before, I was always afraid of using metoclopramide because anything that antagonizes dopamine may cause splanchnic hypoperfusion. Dolasetron is an effective antiemetic, and it's affordable. Now I can stop the vomiting in a lot of these animals and feed them orally or through a stomach tube. I couldn't have done that before.

**von Dehn:** Is dolasetron within the same drug classification (5-HT<sub>3</sub> receptor antagonist) as ondansetron (Zofran)?

**Steiner:** Yes, but it's far less expensive. I don't know the exact price, but practitioners can treat a large dog for about \$10 a day vs. \$50 a day with ondansetron. That makes it inexpensive for cats.

**Relford:** Animals that present with acute vomiting often have gastritis. Maybe they got into a garbage can, but practitioners don't know if the animals have pancreatitis, and they won't get PLI test results for 12 or 24 hours.

### Treatment of pancreatitis and gastritis

#### Acute pancreatitis

- Provide intravenous crystalloid support.
- Provide colloid support (usually in the form of fresh frozen plasma).
- Administer analgesics for pain.
- Remove inciting agent, if identified.
- Administer antiemetics if vomiting persists despite nothing per os (dolasetron recommended).
- Administer antibiotics if there's a potential for bacterial translocation.
- Insert feeding tube (in severe cases) and begin enteral feeding.

#### Chronic pancreatitis

- Prescribe a low-fat diet with adequate protein content.
- Find drug alternatives for animals

taking drugs known to cause pancreatitis (see pages 4, 5, and 14).

- Check serum calcium and triglyceride concentrations routinely (prescribe gemfibrozil if diet does not help lower triglycerides).
- In cats, routinely measure serum cobalamin and correct subnormal levels.

#### Acute gastritis

After ruling out a gastrointestinal obstruction or other diseases:

- Follow a nothing per os approach for the first 24 hours. In gastritis cases, the vomiting should subside fairly quickly.
- Administer intravenous fluid therapy, if indicated.
- Administer antacids or an H<sub>2</sub> blocker for nonspecific gastritis.

How should practitioners manage that? When should they start therapy?

**Williams:** Patients with pancreatitis probably won't get better immediately. Practitioners should begin enteral feeding after a few days if the animals haven't started to eat on their own. Patients with gastritis will likely improve quickly, although they may need some fairly aggressive antiemetic therapy.

Practitioners' initial treatments also depend on the owner's resources. I think it's best to evaluate for pancreatitis initially, but if a client has limited resources, it may make more sense to treat the pet for 24 hours and then, if the patient is not recovering, look for more complicated diseases.

**Twedt:** Could some of these so-called nonspecific gastritis cases be pancreatitis flare-ups?

**Williams:** Yes, they could be.

**Steiner:** But the time frame is important, just as it is for people. If two people come into a hospital and both present with similar signs, it's difficult to determine which one needs more aggressive care. Approximately 50% of people who die from pancreatitis die within the first 72 hours.

I think dogs and cats are similar—they come in and don't appear that sick, but 24 to 72 hours later, they're dead. I recommend treating both types of cases more aggressively at the onset. If practitioners begin therapy 24 to 48 hours later, they're behind the eight ball. By then, the patients may have started to develop systemic complica-

tions of pancreatitis that may be difficult or impossible to counteract. Treatment includes pain medication and plasma with evaluation of potassium and sodium concentrations several times a day. You don't want to miss the point of no return. That's why it's so critical to diagnose pancreatitis early.

it doesn't look that bad. You don't know if the animal has pancreatitis, and you definitely don't want it to get past the point of no return.

**von Dehn:** I would use a nothing per os approach for the first 24 hours. If indicated, I would institute intravenous fluid therapy immediately

**Twedt:** I usually find that if you withhold food and water from those patients, the vomiting subsides.

**Relford:** So if it's just a dietary indiscretion or mild gastritis, the patient should only vomit a few times and then begin to improve. If it continues to vomit, then practition-

Acute pancreatitis patients will initially require more frequent evaluations and PLI monitoring. Chronic cases may only need rechecks every few weeks or months or with acute flare-ups.



**Twedt:** I think practitioners are usually presented with patients that have some sort of "garbage gut" or acute, self-limiting gastroenteritis. With symptomatic therapy, those patients usually get better within 24 to 36 hours. Again, you have to consider the severity of the disease: Is it mild? If they don't get better in a short period of time, then there's something else going on. Those are the cases I investigate further with diagnostic laboratory tests and radiography.

On the other hand, cases deemed more severe (*i.e.*, with protracted vomiting, dehydration, or abdominal pain) require immediate diagnostics and aggressive therapy.

**Relford:** So what does symptomatic therapy include? You have an animal with severe acute gastritis, but

and administer antiemetics, provided no gastrointestinal obstruction is present. I may also administer antacids or an H<sub>2</sub> blocker, such as famotidine or cimetidine, for nonspecific gastritis. Colloidal support is usually not necessary in most gastritis cases, unlike pancreatitis.

An example of a gastritis case that would warrant more aggressive therapy would be hemorrhagic gastroenteritis.

**Williams:** I would probably add radiographs to rule out other diseases.

**Steiner:** I wouldn't use antiemetics in these acute gastritis cases. I actually use vomiting as a monitoring tool to see how aggressive I need to be. But in the pancreatitis cases, my goal is to feed them early, so I want to stop the vomiting as quickly as possible.

ers need to start instituting more aggressive therapy and perform other diagnostic tests.

**Williams:** Practitioners should also remember pain management. We know that people with acute and chronic pancreatitis suffer from extreme abdominal pain. I recommend potent analgesic therapy for all dogs and cats with pancreatitis—but not in cases of nonspecific gastritis. Classical fears regarding the use of opiate analgesics (*e.g.*, morphine) in patients with pancreatitis have now been discounted.

**Steiner:** I agree. Analgesia is important—even for patients that go home.

**Twedt:** Some patients may wag their tails and don't seem to be in pain. But when you put them on pain management

## Diagnosing and treating pancreatitis

for pancreatitis, their whole demeanor changes.

**Steiner:** Practitioners can send patients home with fentanyl patches, tramadol, or oral butorphanol.

**Relford:** Once practitioners have diagnosed pancreatitis, is there anything else they might consider a potential problem?

**Williams:** Fortunately, it's relatively rare, but sometimes pancreatic adenocarcinoma causes pancreatitis. In those cases, a tumor may be causing local ischemia, which predisposes the patient to pancreatitis development.

**Twedt:** Pancreatic adenocarcinoma is a horrible and devastating disease. I think the prognosis becomes poorer for older animals or animals that develop pancreatitis from a concurrent underlying disease.

### Chronic pancreatitis

**Relford:** What are the best ways for practitioners to handle chronic pancreatitis cases?

**Twedt:** I certainly look at diet, and I look for underlying disease as well. I think it's important to keep patients' lipid and triglyceride concentrations down.

**Relford:** What does evaluating the diet entail?

**Twedt:** I want to make sure they're on relatively low-fat diets with adequate protein content.

**Steiner:** I also routinely check the serum calcium and triglyceride (18-hour fasting) concentrations. If the triglyceride concentration is more

than 500 mg/dl, I would monitor it to make sure a low-fat diet decreases it. If not, I put the patient on gemfibrozil to help lower the triglycerides.

**Twedt:** Those same precautions should be taken with high-risk dogs, including schnauzers and shelties that are prone to hyperlipidemia.

**Steiner:** It's also important to look at drugs in chronic pancreatitis cases. Veterinarians need to find drug alternatives, if absolutely needed, for potassium bromide, L-asparaginase, azathioprine, estrogen, calcium—drugs known to cause pancreatitis.

**von Dehn:** Oral pancreatic enzyme supplementation is occasionally implemented for chronic or recurrent pancreatitis. Anecdotal evidence has shown that supplementation may be beneficial in providing negative feedback to the pancreas by inhibiting pancreatic secretion.

**Twedt:** It's thought that pancreatic enzyme supplementation may decrease pancreatic pain or secretions. In veterinary medicine, we just don't know.

**Williams:** That's true. We don't know. If practitioners have done everything else, I recommend trying it for a month or two. It's a fairly benign therapy, and the clients can decide if it's helping. I often tell people to call me if they think it works, but no one has ever called me back.

**Steiner:** Physicians disagree on whether it's beneficial for people. For one thing, the patients were given high doses—not doses veterinarians would normally use.<sup>17</sup> I tell practition-

ers to try it and see what happens, but they shouldn't expect a miracle. It's not going to change the course of pancreatitis. It will only change the pain because of the feedback mechanism in the pancreas.

**von Dehn:** In patients with chronic pancreatitis, primarily cats, measuring serum cobalamin levels may be useful, as a fair number of cats have concurrent inflammatory bowel disease and hepatic lipodosis. Replacing subnormal cobalamin levels allows a more optimal therapeutic response.

**Relford:** When should practitioners expect to see some type of improvement in these chronic cases? When should patients come back?

**von Dehn:** Obviously, acute pancreatitis patients will initially require more frequent evaluations and PLI monitoring. In cases of chronic smoldering pancreatitis, practitioners may only need to schedule rechecks every few weeks or months or when acute flare-ups occur.

**Steiner:** Every case is different because the disease covers a wide spectrum. In severe acute cases, I run serum PLIs every two to three days to see if the patient is improving. I examine the patient, too, but a PLI test mirrors the disease's clinical progression quite well. For patients in intensive care for three to four weeks, it might be once a week. For the dog that occasionally doesn't eat his breakfast or receives potassium bromide, I may run it every six months. These decisions also depend on the clients and how closely they want to monitor the disease.

**Relford:** When should practitioners expect to see a change in the PLI result?

**Steiner:** If I start treating cats with chronic pancreatitis with prednisone, I check the PLI before I start the prednisone, and I check it again in 10 to 14 days because I want to see a drop in the serum PLI concentration. If I'm changing the diet, I wait three to four weeks.

**von Dehn:** How quickly will the PLI adjust?

**Steiner:** It will adjust very quickly. If veterinarians were able to stop pancreatitis on the spot, the half-life would probably be no longer than 12 hours. The problem is that pancreatitis doesn't just stop, it ebbs and flows.

**Relford:** What if practitioners want to start potassium bromide treatment or check an animal that got into the garbage? How quickly would they see the PLI increase?

**Williams:** In serum samples from dogs with mild to severe experimental pancreatitis, which were available for us to evaluate, PLI increased within hours, and concentrations were extremely high within six hours. In samples from cats with experimental mild transient pancreatitis, the increases were sustained for more than a week in all the cats.

**Steiner:** In that experimental study in dogs, PLIs were elevated in severe cases within one hour after pancreatitis was induced and within three hours for the less severe cases.

**Relford:** Is there anything practitioners or clients can do to prevent pancreatitis?

**Twedt:** Identify the risk factors (e.g., obesity, hypertriglyceridemia, drug therapy) and try to address them.

If practitioners can identify flare-ups and confirm previous pancreatitis in chronic cases, they can manage those patients much more aggressively.

**Steiner:** Perhaps it's better to ask how practitioners can prevent serious pancreatitis. Asking if we can prevent pancreatitis as a whole is too broad. It's more feasible to focus on serious pancreatitis. Practitioners can increase their awareness with miniature schnauzers, dogs on potassium bromide or L-asparaginase, and all the other predisposing factors we discussed.

**Relford:** Thank you all for participating in today's roundtable. We've come full circle, ending our discussion almost where it began—at predisposing factors. With the latest diagnostic tools and ongoing discussions, practitioners will continue to expand their knowledge of pancreatitis, learning how to better diagnose and manage this devastating, costly disease.

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