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Vomiting Cat Cases: You Can Figure Them Out

Vomiting, one of the most common reasons for cats to be presented for evaluation, is often considered to be “normal.” There is some truth to the idea that cats vomit more readily from eating too much or too fast; eating foods that are unusual, especially food that contains toxins; or grooming (vomiting hair). However, such vomiting should not be routine. If it is, there is often an underlying cause that needs to be addressed. Adult and senior cats have different causes of vomiting than kittens do, but there are similarities in the approach to diagnosis of vomiting in cats of any age.

To simplify the process, it is sometimes helpful to separate the multitude of causes of vomiting into two more distinct groups: vomiting caused by diseases or disorders of the gastrointestinal (GI) tract itself or vomiting due to systemic or non-GI diseases and disorders that trigger either peripheral or central neural pathways (**Table 1**). Vomiting caused by primary GI diseases includes such differentials as infectious, inflammatory, parasitic, anatomic

(obstructive, trichobezoars), neoplastic (alimentary lymphoma), and drug-related or food-related (hypersensitivity, intolerance disorders).¹⁻³ Cats that are vomiting due to extra-GI diseases may have a myriad of different systemic problems, but endocrinopathies (eg, hyperthyroidism), metabolic diseases (eg, renal or liver failure), inflammatory diseases of the liver or pancreas, cardiovascular diseases (eg, heartworm disease), central nervous system (CNS) disorders (eg, vestibular or inflammatory CNS diseases), and neoplasia (eg, mast cell tumors, other cancers affecting visceral organs outside the GI tract) are the most common.³⁻⁶

This wide spectrum of potential causes of vomiting in cats increases the difficulty of making a definitive diagnosis. Nevertheless, it is important to carefully consider each of the potential differentials to prevent the problem from progressing to create further problems. This article provides an overview of the process of making a diagnosis of some of the more common causes of vomiting in cats and discusses the best approaches to their treatment. Because it is an important factor, the role of diet in both diagnosis and treatment of vomiting is also discussed.

Primary Gastrointestinal Disease

The GI tract should always be carefully evaluated in a cat that is vomiting; however, vomiting is not pathognomonic for gastric or intestinal disease. Furthermore, while it is rare for laboratory evaluation (ie, hemogram, serum biochemistry panel, urinalysis) to provide the definitive diagnosis for primary GI disease, initial evaluation of the vomiting cat should include a minimum database of routine blood analysis and, if appropriate, thyroid testing, GI function testing, and viral serology—particularly in adult or senior cats with chronic (>3 weeks) vomiting. It is important to note that these tests may not reveal the primary problem but are necessary to determine basic physiologic sta-

TABLE 1
Approach to Diagnosis of Vomiting

| Primary GI Causes | Extra-GI Causes |
|---|--|
| <ul style="list-style-type: none"> • Gastric parasitic or infectious disease • Gastric or intestinal neoplasia • Gastric ulcers or erosions • Gastric motility disturbances • Gastric outflow obstruction • IBD • Dietary hypersensitivity (allergy or intolerance) • Intestinal dysbiosis • Mechanical or obstructive disease | <ul style="list-style-type: none"> • Pancreatic disease (pancreatitis or exocrine pancreatic insufficiency) • Cholangitis or hepatic disease (hepatic lipidosis, intrahepatic cholestasis, cholesterolemia, infection) • Endocrinopathies (eg, hyperthyroidism, diabetic ketoacidosis) • Systemic infectious disease (eg, toxoplasmosis, feline infectious peritonitis, fungal) • Neoplasia (eg, lymphosarcoma, especially in abdomen or brain, mast cell tumor) • Heartworm/parasitic lung disease • Acute or chronic renal disease • Diseases of CNS, causing nausea |

CNS = central nervous system, GI = gastrointestinal, IBD = inflammatory bowel disease

tus (ie, electrolyte, acid–base, fluid needs) and rule out other systemic diseases.

GI function testing includes that of feline pancreatic lipase immunoreactivity, feline trypsin-like immunoreactivity, cobalamin, and folate. These tests are important for assessing pancreatic function but also give an indication of small intestinal health, as cobalamin and folate are important indicators of intestinal dysbiosis or disease.

If primary GI disease is considered likely based on physical examination, history, or normal laboratory results, then imaging (eg, radiographs, abdominal ultrasound) is indicated either to make a definitive diagnosis or identify abnormalities that require further diagnostic steps. Radiographs and abdominal ultrasound have different purposes based on the likely differentials. If a foreign body is suspected (eg, young cat/kitten), a radiograph is reasonable. On the other hand, if diseases of the bowel wall or abdominal organs requiring ability to measure layers or size are suspected (eg, inflammatory bowel disease [adult cat]), then abdominal ultrasonography is the best approach.

If primary GI disease is considered likely, then imaging is indicated either to make a definitive diagnosis or identify abnormalities that require further diagnostic steps.

The diagnosis of both food hypersensitivity and intolerance is based on removing the offending substance from the diet.

In some cats, more invasive tests (eg, gastroduodenoscopy, exploratory laparotomy) may be required to obtain biopsy material or remove the problem (obstruction). The decision to pursue endoscopy versus exploratory surgery depends on availability of necessary equipment and expertise as well as the likelihood that endoscopy can be a useful diagnostic or treatment tool (eg, an endoscope will not reach the mid or distal jejunum).

Gastric Disease

Among gastric diseases to consider as causes of vomiting are parasitic infestation (eg, with *Physaloptera* or *Ollanus* spp), bacterial infections (eg, with *Helicobacter* spp), neoplastic diseases (eg, lymphoma, adenocarcinoma, leiomyosarcoma), inflammatory diseases (eg, ulcers, inflammatory bowel disease [IBD]), obstructive disorders (eg, hairballs, foreign bodies, masses), and diet-related causes (ie, intolerance, hypersensitivity). Specific diagnosis of individual causes may require additional procedures (eg, histopathologic evidence of spiral organisms deep in gastric glands associated with gastritis) to rule them in or out.

Small Intestinal Disease

Small intestinal disease in cats is a common cause of vomiting associated with the prevalence of inflammatory disease; however, true idiopathic IBD must be distinguished from the simple presence of inflammatory infiltrates in the small bowel, as a variety of dietary, infectious, and parasitic agents can cause either inflammation in the small bowel or dysbiosis, the latter of which causes inflammation.

Dietary sensitivity and intolerance are also important causes of vomiting in cats and should trigger appropriate dietary trials to rule them out. This process may be easier said than done, as finding a commercially available food without the offending substance (intolerance) or antigen (hypersensi-

tivity) and that the cat will readily consume is a challenge. In most cases of small intestinal disease affecting the intestinal wall, with the exception of adverse reactions to food, obtaining a definitive diagnosis will require biopsy—either via endoscopy or exploratory surgery.

Adverse Reactions/Sensitivity to Food

Food intolerance, food allergy (hypersensitivity), food poisoning, food idiosyncrasy, and pharmacologic reactions to foods all fall under the category of adverse reactions to food.⁷ Discussion here is limited to food intolerance and food hypersensitivity (allergy).

Food intolerance, a nonimmunologic, abnormal physiologic response to a food, nutrient, or food additive, is the most common cause of food sensitivity in cats. Food allergy, or hypersensitivity, is characterized by adverse reactions to a food or food additive (typically protein) with a proven immunologic basis. Both allergy to and intolerance of food can result in vomiting, diarrhea, or a combination of signs, depending on the effects: food allergy is more commonly associated with vomiting and dermatologic signs, whereas intolerances of food can present with vomiting or diarrhea but do not produce dermatologic signs.

Dietary Elimination Trial

The diagnosis of both food hypersensitivity and intolerance is based on removing the offending substance from the diet. The major difference between the diagnostic processes of these two types of adverse food reactions is the length of time on the diet that is required to achieve a response and the need to identify a novel protein source. Cats with food hypersensitivity require 8 to 12 weeks on a novel antigen (eg, novel protein or hydrolyzed protein) elimination diet before an improvement will be seen. Alternatively, in cats with food intolerance, resolution of signs

usually occurs within days (7–14 days is typical) of a diet change in which the offending substance is removed, unless other factors influence the response.

A variety of commercially available and homemade elimination formulations can be used, as can those using hydrolyzed proteins. Many different brands fall under the category of “highly digestible,” “sensitive,” and “novel,” but the key is to remember that they are not all alike. Thus, when one product from this category is not accepted by the cat, is ineffective, or seems to make the problem worse, you cannot assume that all products in this category will fail. Highly digestible products from different pet food manufacturers have a variety of formulations (**Table 2**), including different protein and carbohydrate sources, different levels of fat, and various additives designed to promote intestinal health (eg, fructooligosaccharides, maltooligosaccharides, omega-3 fatty acids, antioxidant vitamins, soluble fiber). The same is true for hydrolyzed food products.

If one type of highly digestible food has been fed for at least 2 weeks with minimal response, it is entirely reasonable to try either another comparable product from a different source or an entirely different dietary strategy (eg, high protein/low carbohydrate, novel antigen, hydrolyzed protein). Thus, a dietary trial consisting of novel meat-source proteins or hydrolyzed foods may not be adequate to remove the offending items from the diet. For example, if the problem is being created by presence or type of carbohydrate in the food, feeding a formulation high in protein (>40% metabolizable energy [ME]) and low in carbohydrates (<10% ME) that is highly digestible (>85% digestibility of protein) will resolve the problem.

In some cats, however, the only way to remove the source and confirm this problem is by feeding a homemade food that consists of a meat source (eg, cooked chicken thigh with the fat included) and a vitamin/mineral supplement but no added carbohydrate or other

TABLE 2
Dietary Protein Levels & Protein Sources in Selected Highly Digestible Diets for Cats

| Feline Food (dry) | Protein (% DM / Source) |
|--|-------------------------------|
| Purina Veterinary Diets Feline Formula EN | 56% / soy protein isolate |
| Hill's Prescription Diet i/d Feline | 40% / chicken meal |
| IAMS Veterinary Formula Intestinal Plus Low-Residue | 32% / chicken by product meal |
| Royal Canin Veterinary Diet Feline Gastrointestinal High Energy HE | 30% / chicken meal |

DM = dry matter

ingredients. This diet eliminates carbohydrates and all other commercial food additives and can be fed for up to 2 to 3 weeks, but a complete and balanced food should be formulated by a nutritionist if it must be fed longer.

The key feature that separates food intolerance from an allergy is that once the offending agent is removed from the diet, the vomiting (or other GI signs) will resolve quickly. Another key point is that in dietary intolerance the offending substance may be difficult to identify using typical commercial foods, thus a food trial using a homemade diet can be quite helpful.

The key point is that dietary management is a process of trial and error. No single diet or diet family will benefit all cats in all situations.

Inflammatory or Immune-Mediated Causes of Vomiting

IBD, a commonly diagnosed condition of adult cats, is likely due to multiple causes but ultimately culminates from a combination of genetic susceptibility, intestinal microbial dysbiosis, and persistent inflammation of the gut wall, resulting in signs of vomiting, diarrhea, weight loss, or combinations of all three.⁸ Idiopathic IBD is characterized by persistent clinical signs of GI disease occurring with histologic evidence of mucosal inflammation and structural changes of the villous epithelium without an identifiable or correctable cause (eg, food).

A dietary trial consisting of novel meat-source proteins or hydrolyzed foods may not be adequate to remove the offending items from the diet.

Studies of probiotic therapy in cats have primarily focused on the use of Fortiflora (Purina) in a shelter environment among kittens or young cats with parasitic diseases (giardiasis, cryptosporidiosis, etc) or stress-induced diarrhea.

A number of possible causes of intestinal inflammation must be considered in the diagnostic process, and all should be investigated thoroughly or therapeutic trials instituted prior to settling on the diagnosis of idiopathic IBD—a disease requiring long-term therapy with immunosuppressive drugs. In particular, appropriate food trials are an extremely important component of both diagnosis and therapy of cats with suspected IBD (or GI disease in general). In addition, the diagnostic plan for a cat with chronic vomiting should include assessment of thyroid and feline leukemia virus/feline immunodeficiency virus (FeLV/FIV) status as well as intestinal vitamin (cobalamin/folate) status.

Serum cobalamin levels in cats commonly decrease with chronic pancreatitis or severe bowel disease; in cats with hypocobalaminemia, inappetence or vomiting will not resolve until replacement therapy has been instituted.⁹ Cobalamin therapy (250 g/cat SC weekly for 6 weeks, then once every other week) in some cats may be lifelong, while in others once the clinical disease resolves the supplementation can be discontinued.

In addition, radiography and ultrasonography are important in detecting the presence of infiltrative diseases, such as feline infectious peritonitis, granulomas, histoplasmosis, or lymphosarcoma. Ultrasonography has been particularly helpful in identifying intestinal wall layer changes and mesenteric lymphadenopathy—two findings that support intestinal inflammation or disease but do not differentiate type. Ultimately, intestinal biopsies, obtained either endoscopically or at exploratory surgery, are essential for both diagnosing IBD and ruling out other specific causes of GI clinical signs.

Treating IBD

At this time, therapy of IBD in cats continues to include inflammatory suppression and antibiotic therapy, and while evidence to support a specific role for probiotic therapy is

lacking, its use to help control dysbiosis or IBD seems to have merit. The most effective therapies for IBD include steroids (prednisolone or methylprednisolone, 1–2 mg/kg q12h PO) or other drugs that interrupt the proinflammatory pathways active in the gut. In cats intolerant to steroids or those in which steroids are no longer effective, immunosuppressive therapy may be necessary. Currently, either chlorambucil or cyclosporine is most frequently chosen.

Metronidazole (5–10 mg/kg q12h PO) or tylosin (5–15 mg/kg q12h PO) has been effective for control of bacteria-associated disease and continues to be recommended for initial therapy of IBD. Whether this action can be attributed to the antibiotic effects of these drugs and their influence on the intestinal microflora or their immune-modulating activities is unknown. Nevertheless, such therapy is often helpful. Caution is advised in using either drug on a continuous or long-term basis but especially metronidazole due to its potential for genotoxicity. If needed, they should be used intermittently, not continuously.

Studies of probiotic therapy in cats have primarily focused on the use of Fortiflora (Purina) in a shelter environment among kittens or young cats with parasitic diseases (giardiasis, cryptosporidiosis, etc) or stress-induced diarrhea. Under these circumstances, probiotic therapy resulted in a faster resolution of diarrhea and more rapid resolution of infection. However, placebo-controlled clinical trials using probiotics in cats with IBD are only in their early stages, so specific recommendations await their findings.

Finally, general agreement exists among gastroenterologists that foods with fewer carbohydrates (a source of intolerance and maldigestion) and based on highly digestible protein sources (primarily meat) are beneficial in cats with IBD, reducing the bacterial changes that can occur when undigested foods remain in the GI tract. These formulations may include so-called *hypoallergenic*

diets but do not necessarily require hypoallergenicity. A scoring system that can be used in monitoring response to treatment of IBD as well as to assist in diagnosis in cats in which IBD is suspected has been published by Albert Jergens et al.⁸

Extraintestinal Causes of Vomiting: Feline Pancreatitis & Cholangitis

Feline pancreatitis is difficult to diagnose definitively ante-mortem, especially in its more common lymphoplasmacytic form, and is associated with vomiting only occasionally or intermittently.^{4,10} This difficulty is partly attributable to lack of both specific clinical signs in cats and a highly sensitive test for diagnosis of the disease.

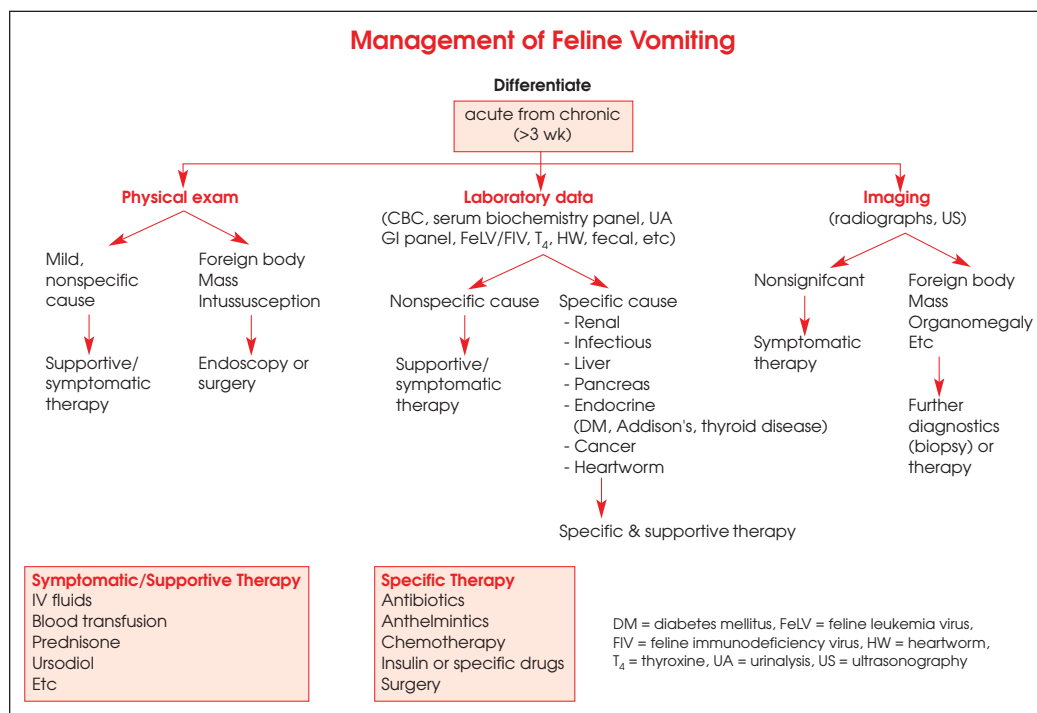
The clinical signs of pancreatitis in cats can be quite different from those in dogs. Acute necrotizing pancreatitis is frequently encountered in obese dogs fed a high-fat diet, while cats are more likely to be underweight and high-fat diets do not appear to be an important predisposing factor. Cats of all ages, sexes, and breeds are affected, although Siamese cats reportedly have the more acute, necrotizing form of pancreatitis more frequently.

The most common form in cats, lymphoplasmacytic pancreatitis, is more insidious, and the clinical signs are vague, with the most common being lethargy (100% of cats in one study), anorexia, and dehydration.¹¹ Vomiting and anterior abdominal pain, which are common clinical signs in dogs with acute pancreatitis, occur in only 35% and 25% of cats, respectively. However, there is strong belief among feline practitioners that pancreatic pain or discomfort may be underreported due to the tendency

of cats to hide overt signs and the apparent response of cats given pain relief medication. Thus, clinical signs may be quite variable, and this must be taken into consideration with each patient.

Routine evaluation of vomiting cats with suspected pancreatitis or other extra-GI causes of vomiting is similar to that mentioned above: a minimum database, GI function testing, and retroviral testing are always appropriate. Tests for hyperthyroidism, liver function, or other specific tests may be indicated in some cats.

Hematologic findings in cats with pancreatitis are nonspecific but may include nonregenerative anemia, leukocytosis, or leukopenia (less common). In a recent study, cats with pancreatitis consistently had elevated white blood cells (20,300/L) and mild decreases in platelets (mean, 180,000/L). Neutrophils were not degenerate or toxic. Reported changes in the serum biochemistry profile include elevated serum alanine aminotransferase (ALT), elevated serum alkaline phosphatase (ALP), hyperbilirubinemia, hyper- or hypocholesterolemia, hyperglycemia, azotemia, and



In cats with chronic pancreatitis, it is still necessary to evaluate the combined historical, physical examination, and laboratory data as well as imaging information, along with the fPLI results, when making a diagnosis.

hypokalemia. Common abnormalities in cats with severe pancreatitis were hyperglycemia (180 mg/dL), hyperbilirubinemia (2.5 mg/dL), hypocholesterolemia (130 mg/dL), and hypoalbuminemia (1.8 g/dL).

In cats with mild or lymphoplasmacytic pancreatitis, liver enzyme elevations were more common, with γ -glutamyl transferase, ALP, and ALT being moderately elevated. 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 feline trypsin-like immunoreactivity (fTLI) test is the definitive test for diagnosis of exocrine pancreatic insufficiency. While an increase in fTLI can be found in cats with pancreatitis, a normal value does not rule out pancreatitis, as the leakage of enzymes tends to decrease rapidly following an event and the enzymes are inactivated and scavenged by the body's peptidases (eg, macroglobulin) within 12 to 24 hours following an acute insult. This test is very useful in cats with chronic pancreatitis, however, as they may sustain a loss of pancreatic function, indicated by a decreased fTLI. In fact, in a recent unpublished study by the author's group of 150 cats with exocrine pancreatic insufficiency tested at the Texas A&M GI laboratory, the most common clinical sign was weight loss (85%) not diarrhea (45%) or vomiting. Thus, measurement of fTLI is an important aspect of assessment in cats with chronic low-grade inflammation of the pancreas that may not have overt signs of inflammation or illness but have lost significant pancreatic functional capacity.

The test of choice for pancreatic leakage

is the radioimmunoassay for feline pancreatic lipase (fPLI); this test has a sensitivity and specificity of nearly 100% in cats with severe pancreatitis (determined by pancreatic biopsy).¹² However, the sensitivity in moderate pancreatitis was found to be 80% and as low as 65% in mild pancreatitis, while the specificity in healthy cats was 75%. Thus, in cats with suspected chronic pancreatitis, it is still necessary to evaluate the combined historical, physical examination, and laboratory data as well as imaging information, along with the fPLI results, when making a diagnosis.

Imaging Studies

Imaging studies are frequently used to help identify cats with acute pancreatitis, but in those cats with the more common chronic form, changes on ultrasound imaging can be particularly subject to interpretation and operator expertise. The most common ultrasonographic findings are hypoechoic pancreas, hyperechoic mesentery, mass effect, dilated common bile duct, or normal appearance throughout. In a recent study, mild pancreatitis was still shown to be difficult to diagnose via abdominal ultrasound imaging, but ultrasound was 80% sensitive and 88% specific in cats with moderate to severe pancreatitis.¹³

The most reliable method for making an accurate diagnosis of pancreatic disease remains direct visualization and histopathology. This approach can be expensive and can increase the risk for complications (during anesthesia/surgery). In cases with focal involvement, which is common with chronic pancreatitis, lesions may be missed. In short, pancreatitis remains a challenging diagnosis and an even more challenging disease to treat once the diagnosis has been confirmed.

Feline Liver Diseases (Cholangitis, Idiopathic Hepatic Lipidosis)

Cats have four major types of liver disease:

hepatic lipidosis, cholangiohepatitis complex, infectious hepatitis (eg, feline infectious peritonitis, toxoplasmosis, fungal/parasitic hepatitis), and neoplastic liver disease (eg, lymphoma). As with most diseases of the liver, histopathology is an important step in determining treatment and prognosis. Nevertheless, once a diagnosis has been obtained, the goal for treatment of cats with liver disease is to provide optimal nutritional and pharmacologic support that maximizes liver function; minimizes future liver or biliary duct damage or scarring; controls concurrent clinical signs, such as vomiting; and thus promotes a high quality of life.

Inflammatory, infectious, or metabolic liver disease can be present in cats with few external clinical signs other than inappetence, vomiting, or lethargy or can cause severe illness resulting in development of ascites, icterus, hepatoencephalopathy, coagulopathy, and loss of ability to metabolize protein or carbohydrates appropriately. Thus, there is no single set of clinical signs or laboratory abnormalities that can define all liver disease patients. Nevertheless, some important clues can help guide the clinician to making a definitive diagnosis.

The most common cause of severe liver disease or failure in the cat is idiopathic hepatic lipidosis, but the most common cause of increased liver enzymes and chronic intermittent clinical signs is cholangitis/cholangiohepatitis. In several recent studies using biopsy results to confirm diagnosis, cats with inflammation of the peribiliary structures consistent with cholangitis also had lymphoplasmacytic infiltrate in the pancreas (66%–75% in separate studies).^{13,14} Thus, there is growing evidence that these two diseases in cats may be linked: when one occurs, the other follows. At this time, there is no agreement about cause; however, there is some evidence that bacteria may be an important culprit, as bacterial DNA was found in at least 30% of livers with a neutrophilic inflammatory component.¹⁵ Con-

versely, in another article, no bacterial DNA was found, but the majority of study cats had the more chronic lymphocytic form of the disease.¹⁶ Whether this dichotomy represents two separate diseases or different stages/phases of the same disease (acute progressing to chronic) is unknown, but it suggests that further work to better control and define the origin of intestinal dysbiosis in cats is warranted.

Finally, a number of other important extra-GI causes of vomiting also need to be considered, including chronic renal disease, endocrinopathies (eg, hyperthyroidism, diabetic ketoacidosis), and other systemic diseases (eg, heartworm disease). A complete discussion of each is not possible, but readers are reminded to consider these possibilities when confronted with vomiting cats for which a definitive diagnosis has not been made.

Nonspecific Therapy of Vomiting

Several antiemetic agents are available for use in cats (**Table 3**); some are more commonly used in the hospital setting because they are injectable and may require frequent administration. The newest antiemetic drug family, neurokinin (NK) inhibitors, represented by maropitant, are clearly the most effective in cats. In addition to the excellent antinausea effects of maropitant, it also appears to be effective for controlling visceral pain, which may be an essential aspect of therapy in feline chronic pancreatitis and other visceral causes of vomiting. The feline dose is 1 to 2 mg/kg PO or SC q24h for 3 to 5 days, but it may be given longer if needed.

The 5-HT₃ antagonists are effective antiemetic agents for cats as well at doses of 0.5 to 1.0 mg/kg of ondansetron, 0.1 to 0.5 mg/kg of granisetron, or 0.5 to 1.0 mg/kg of dolasetron PO or IV q12–24h. In addition, cats may be treated with chlorpromazine, an α_2 -adrenergic antagonist, at 0.2–0.4 mg/kg q8h SC or IM. Dopaminergic antagonists such as metoclopramide are less effective in the cat and, because they antagonize

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TABLE 3
Feline Doses for Antiemetic Drugs

| Drug Class | Location of Action | Drug | Dose |
|--|--|------------------|------------------------------|
| α_2 -Adrenergic antagonists | Central (CRTZ/vomiting center) | Prochlorperazine | 0.1–0.5 mg/kg q8h SC |
| | | Chlorpromazine | 0.2–0.4 mg/kg q8h SC |
| D ₂ dopaminergic antagonists | Central (CRTZ) and peripheral (GI smooth muscle) | Metoclopramide | 0.2–0.4 mg/kg q6–8h SC |
| H ₁ histaminergic antagonists | Central (CRTZ) | Chlorpromazine | 0.2–0.4 mg/kg q8h SC |
| | | Diphenhydramine | 2–4 mg/kg q8–12h SC |
| | | Dimenhydrinate | 2–4 mg/kg q8–12h SC |
| 5-HT ₃ serotonergic antagonists | Central (CRTZ/vomiting center); peripheral (vagal afferents) | Ondansetron | 0.5–1.0 mg/kg q12–24h PO, IV |
| | | Granisetron | 0.1–0.5 mg/kg q12–24h PO, IV |
| | | Dolasetron | 0.5–1.0 mg/kg q12–24h PO, IV |
| 5-HT ₄ serotonergic antagonists | Peripheral (myenteric neurons) | Cisapride | 1.25–2.5 mg/cat q8–12h PO |
| NK ₁ neurokinin antagonists | Central (CRTZ/vomiting center) | Maropitant | 1–2 mg/kg q24h PO, SC |

CRTZ = chemoreceptor trigger zone

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dopamine, may potentially reduce pancreatic blood flow. (This effect has not been proven in cats with pancreatitis.)

While such nonspecific therapy may be indicated to control vomiting, finding the cause is more important than simply controlling the clinical sign. Thus, antiemetic therapy should be used judiciously in the clinical setting and as an adjunct to therapy for the primary problem.

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Suggested Reading

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