Chapter 52

Gastritis and Gastroduodenal Ulceration

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"If your stomach disputes you, lie down and pacify it with cool thoughts."
Satchel Paige

CLINICAL IMPORTANCE

Gastritis is one of the most common causes of vomiting in dogs and cats (Van der Gaag, 1988). Acute gastroenteritis is covered in Chapter 56. The prevalence of gastritis in the pet population is unknown, but is thought to be high because many different insults can result in gastric mucosal inflammation (**Table 52-1**). In one survey, 9% of research beagles had histologic evidence of gastritis in the absence of clinical signs (Hottendorf and Hirth, 1974). Gastritis has been diagnosed in 35% of dogs presented for chronic vomiting and has been identified in 26 to 48% of asymptomatic dogs. The prevalence in cats is unknown (Simpson, 2006). The National Companion Animal Study was developed in the early 1990s to determine the most common disorders affecting dogs and cats examined at private veterinary practices in the United States. In 1995, 31,484 dogs and 15,226 cats were examined at 52 private veterinary clinics in 31 states. In this study, the prevalence of vomiting was 2.1% for dogs and 2.2% for cats (Lund et al, 1999). Acute gastritis often accompanies acute enteritis and is called acute gastroenteritis.

Previously, the prevalence of gastroduodenal ulcers in dogs and cats was thought to be low compared with the prevalence reported in people. In many cases, the historical infrequent diagnosis of gastroduodenal ulceration was possibly due to the absence of obvious clinical signs. For example, in experimental studies involving dogs, extensive gastroduodenal ulceration was present, whereas only mild clinical signs were evident (Dow et al, 1990). However, gastroduodenal ulceration is now diagnosed more frequently in veterinary patients. While advances in diagnostics (endoscopy) have provided improved capability to identify gastroduodenal ulcers, the apparent increase in prevalence has been associated with the use of nonsteroidal antiinflammatory drugs (NSAIDs) for pain management and treatment of inflammatory conditions. The actual incidence of gastroduodenal ulceration related to NSAID use in dogs is unknown (Hinton et al, 2002; Lascelles et al, 2005; Dowers et al, 2006).

PATIENT ASSESSMENT

History and Physical Examination

Although some patients are asymptomatic, vomiting is the most common presenting complaint for patients with acute or chronic gastritis. Typically, owners report intermittent vomiting of food or bile-stained fluid. Fresh or digested blood appearing as "coffee grounds" may be present in the vomitus. Associated signs may include diarrhea, abdominal pain and melena. Anorexia is the presenting sign in many patients with gastritis. The clinician should obtain details regarding frequency, duration and progression of the vomiting episodes. In addition, the vomitus should be characterized (e.g., color, contents). It is important to differentiate vomiting from regurgitation (Simpson, 2005; Willard, 2005). Some owners may report that their dog

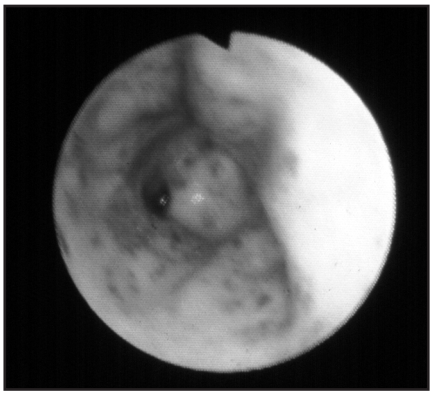

Figure 52-1. Endoscopic appearance of antral gastritis in a dog with chronic vomiting. Note the multiple hemorrhagic erosive lesions of the gastric mucosa. (Courtesy Dr. Michael Leib, Virginia-Maryland Regional College of Veterinary Medicine, Blacksburg, VA.)

assumes a "praying posture," which is considered a manifestation of upper abdominal pain.

Patient history often is adequate to provide a presumptive diagnosis of gastritis. Owners should be questioned closely about potential for toxin exposure (e.g., lead, arsenic) and foreign body ingestion (e.g., bones, coins, garbage) by the patient. A history of NSAID administration provides a presumptive diagnosis of drug-induced gastroduodenal erosions or ulcerations. The veterinarian should question the owner specifically about the use of over-the-counter agents (e.g., aspirin, ibuprofen) in addition to prescription NSAIDs.

Physical examination is often unremarkable in dogs and cats with gastritis or gastroduodenal ulcerations. Reduced skin turgor and tacky mucous membranes indicate dehydration. Abdominal pain may be recognized, particularly in those patients that develop peritonitis as a consequence of a perforated ulcer. In chronic cases, weight loss and poor body condition may be noted. Pallor and weakness may be present in patients with significant gastrointestinal (GI) blood loss. Other findings may reflect the underlying cause of gastritis (e.g., cutaneous masses or hepatosplenomegaly associated with mastocytosis).

Laboratory and Other Clinical Information

Routine hematology, serum biochemistry profiles and urinalyses help rule out metabolic causes of gastritis. These tests readily identify renal disease, hepatopathies and hypoadrenocorticism. The hematocrit and hemogram are useful in assessing severity and chronicity of gastric disease. Inflammatory leukograms may be identified in animals with neoplasia, perforated GI ulcers, inflammatory bowel disease (IBD) and pythiosis. Eosinophilia may indicate parasitism or eosinophilic gastritis. In cats, extreme eosinophilia is suggestive of hypereosinophilic syndrome or systemic mastocytosis. Identification of circulating mast cells is generally diagnostic for mast cell tumors, which are associated with GI ulcer disease due to hyperhistaminemia.

Fecal examinations for parasites and occult blood are important screening tests. Parasites are an unlikely cause of gastritis, but should be considered. Gastric parasites, such *Ollulanus tricuspis* or *Physaloptera* spp., are identified more readily in vomitus or gastric juice or on endoscopic visualization. The accuracy of fecal occult blood testing has been confirmed in dogs consuming dry foods (Dow et al, 1990; Gilson et al, 1990). Moist meat-based foods often yield false-positive results. Both the modified guaiac and orthotoluidine tests are sensitive and specific for detecting occult blood in feces (Gilson et al, 1990).

Imaging modalities (e.g., survey and contrast radiography and ultrasonography) are noninvasive diagnostic techniques for evaluating pets with gastritis or GI ulceration. Abdominal radiographs frequently are normal in patients with gastritis (Simpson, 2005). Survey radiography may be useful in the diagnosis of radiopaque foreign bodies. Abnormalities in renal size or shape may suggest renal insufficiency as the cause of gastritis. Hepatosplenomegaly in cats suggests systemic mastocytosis or alimentary lymphosarcoma. Free air in the abdomen is diagnostic for viscus rupture associated with a perforated GI ulcer and indicates the need for immediate exploratory surgery.

Contrast radiographic examinations may be useful. Iodinated contrast agents[a] should be used if GI perforation is suspected. Otherwise, barium sulfate is the contrast agent of choice for GI studies because of its superior ability to coat the GI mucosa. More complete descriptions of radiographic findings in gastric disease are available (Moon and Myer, 1986).

Endoscopic examination is the most sensitive test for detection of gastritis and gastroduodenal ulcerative disease. Gastric fluid can be collected for parasitic and microbiologic examination. Endoscopic evaluation allows for the identification of mucosal and submucosal hemorrhages, erosions and ulcers, tumors and foreign bodies (**Figure 52-1**). Gastric and duodenal biopsy specimens for histopathologic examination and brush cytology samples can be collected endoscopically (Jergens et al, 2000). *Helicobacter* spp. can be identified in impression smears prepared from such samples (Simpson, 2005). Gastric biopsy specimens can be evaluated for *Helicobacter* spp. using the rapid urease test[b] (Leib and Duncan, 2005).

Risk Factors

Dogs with liver or kidney disease, hypoadrenocorticism, spinal cord disease, shock, stress, neoplasia, mastocytosis and systemic disease are at increased risk for gastroduodenal ulceration (Lascelles et al, 2005; Simpson, 2005; Henderson and Webster, 2006).

Older pets are more likely to be suffering from metabolic or neoplastic causes of gastritis. Dogs of any age receiving NSAIDs, corticosteroids, or both, for management of osteoarthritis are at risk for gastritis and gastroduodenal ulceration.

Younger dogs and cats and unsupervised pets are more likely to suffer from gastritis secondary to foreign bodies or dietary indiscretion. Several breed-associated causes of gastritis have been recognized (Table 51-1). Dachshunds, miniature schnauzers, toy poodles and other small- and toy-breed dogs are most commonly affected with hemorrhagic gastroenteritis (Guilford and Strombeck, 1996). Several breeds are at risk for chronic

gastritis including the basenji, Norwegian lundenhund and Drentse patrijshond (Slappendel et al, 1997; Hart, 2004; Simpson, 2005, 2006; Berghoff et al, 2007). Racing sled dogs in competition are at increased risk for gastroduodenal erosions and ulceration although dogs in training are not (Davis et al, 2006). Nearly 50% of dogs completing the Iditarod race were found to have gastric lesions (Davis et al, 2003, 2003a).

Etiopathogenesis

Acute Gastritis and Gastroduodenal Ulceration

Acute gastritis is characterized by sudden-onset vomiting, resulting from gastric mucosal insult or inflammation. Hematemesis usually indicates that gastroduodenal erosions or ulcerations are present (Willard, 2005). Gastroduodenal ulceration occurs following disruption of the gastric mucosal barrier. The gastric mucosal barrier is a group of physical and chemical defense mechanisms designed to protect the gastric mucosa from insults leading to erosions or ulcers. Disruption of the gastric mucosal barrier may involve direct injury, decreased mucosal blood flow, alterations in protective prostaglandins (prostaglandin E_2 [PGE_2]) or hypersecretion of gastric acid (e.g., gastrinoma) (Simpson, 2005; Henderson and Webster, 2006).

Several metabolic disorders are associated with acute gastritis and gastroduodenal ulceration (**Table 52-1**). Uremia may result in diffuse GI tract hemorrhage. GI erosions and ulcers are thought to result from effects of uremic toxins on the gut mucosa. Additionally, increased circulating concentrations of gastrin have been identified in patients with uremia. The kidneys normally excrete up to 40% of circulating gastrin. Clearance of gastrin is decreased with chronic kidney disease. The resulting hypergastrinemia leads to increased acid production. Studies suggest that hypersecretion of gastrin may contribute to gastric ulceration in chronic kidney disease (Thornhill, 1983; Peters et al, 2005; Polzin et al, 2005; Henderson and Webster, 2006).

GI signs and histopathologic changes are seen in dogs with chronic kidney disease. A retrospective study was done to determine the prevalence of gastric histopathology in necropsy samples from 28 dogs with chronic kidney disease and to characterize the histopathologic changes. All dogs presented with GI signs, including anorexia and vomiting. Twenty-two (79%) of the 28 dogs had gastric pathology. The most common pathology included edema, vasculopathy, glandular atrophy and mineralization. No evidence of ulceration was seen histopathologically and only one dog had ulceration noted on gross necropsy. Dogs with higher serum biochemistry scores (i.e., blood urea nitrogen, creatinine and calcium-phosphorus product) were more likely to have gastric pathology. The authors concluded that gastric ulceration may be uncommon in dogs with chronic kidney disease (Peters et al, 2005).

Liver disease is a common cause of GI ulcerations, which may be manifested as hematemesis. Liver disease was one of the two most common risk factors (the other being treatment with NSAIDs) in a retrospective study of 43 dogs with gastroduodenal ulceration (Henderson and Webster, 2006). The pathogenesis of mucosal ulceration associated with hepatopathies is multifactorial and associated coagulopathies may worsen clinical manifestations. Potential mechanisms include altered gastric blood flow due to portal hypertension, delayed epithelial turnover, gastric hyperacidity and hypergastrinemia. Experimental evidence suggests that hypergastrinemia is a less important mechanism than previously suspected (Booth, 1990; Henderson and Webster, 2006).

A variety of adverse drug reactions have been reported following the use of NSAIDs in dogs. These include GI bleeding, ulceration or both and hepatotoxicity and nephrotoxicity (Sennello and Leib, 2006). The adverse GI effects occur because some NSAIDs have a topical irritant effect on the gastric mucosa and can inhibit protective prostaglandins (McCarthy, 1999; Enberg et al, 2006). Experimentally induced and spontaneous gastritis and gastroduodenal ulcerations have been reported to occur in dogs in conjunction with the use of NSAIDs, including aspirin, indomethacin, naproxen, ibuprofen, phenylbutazone, flunixin meglumine, piroxicam, sulindac and meclofenamic acid (Dow et al, 1990; Lipowitz et al, 1986; Wallace et al, 1990; Davenport, 1992). The ulcerogenicity of NSAIDs is attributed to inhibition of the enzyme cyclooxygenase (COX) in the prostaglandin synthesis pathway, resulting in the loss of the gastric protective effects of prostacyclin and prostaglandin E (Davenport, 1992).

Table 52-1. Potential causes of gastritis and/or gastroduodenal ulceration.

Adverse reactions to food
Food allergy (hypersensitivity)
Food intolerance
Dietary indiscretion
Chemicals
Foreign bodies
Garbage toxicosis
Gluttony
Heavy metal toxicosis
Plants
Drug administration
Corticosteroids
Nonsteroidal antiinflammatory agents
Idiopathic gastritis
Infectious agents
Fungi
Parasites
Spiral bacteria
Inflammatory bowel disease
Neoplasia
Gastrinoma
Mastocytosis
Primary gastric neoplasia
Reduced gastric blood flow
Disseminated intravascular coagulopathy
Neurologic disorders
Sepsis
Shock
Reflux gastritis
Systemic disease
Hypoadrenocorticism
Kidney disease
Liver disease
Pancreatitis

Table 52-2. Key nutritional factors for dogs and cats with gastritis and/or gastroduodenal ulceration.*

Factors	Recommended levels
Potassium	0.8 to 1.1%
Chloride	0.5 to 1.3%
Sodium	0.3 to 0.5%
Protein	***Highly digestible food approach:*** ≤30% for dogs and ≤40% for cats ***Elimination food approach:*** Limit dietary protein to one or two sources Use protein sources that the patient has not been exposed to previously or feed a protein hydrolysate (Chapter 31) 16 to 26% for dogs 30 to 40% for cats
Fat	<15% for dogs <25% for cats
Fiber	≤5% crude fiber; avoid foods with gel-forming fiber sources such as pectins and gums (e.g., gum arabic, guar gum, carrageenan, psyllium gum, xanthan gum, carob gum, gum ghatti and gum tragacanth)
Food form and temperature	Moist foods are best; warm foods to between 70 to 100°F (21 to 38°C)

*Nutrients expressed on a dry matter basis.

Isoforms of COX have been identified. COX-1 is a constitutive form that is found in many tissues (e.g., gastric mucosa), where it is involved in the production of protective prostaglandins. COX-2 is primarily an inducible enzyme that is involved in the production of inflammatory mediators, including proinflammatory prostaglandins. Newer NSAIDs have been developed to minimize the effects on COX-1 and thereby, to decrease the adverse effects on gastric mucosa. The newer NSAIDs are selective inhibitors of COX-2 and generally are considered to be "gastric sparing." However, despite the selective inhibition of COX-2, these newer NSAIDs still carry risk of GI ulceration and perforation. Newer veterinary-approved selective COX inhibitors include flunixin meloxicam, carprofen, etodolac, ketoprofen, tepoxalin, previcox and deracoxib (McCarthy, 1999; Enberg et al, 2006; Dowers et al, 2006; Sennello and Leib, 2006). The use of NSAIDs in patients with underlying renal or hepatic insufficiency may increase the risk of GI ulcerative disease. Concurrent NSAID and corticosteroid use should also be avoided due to the risk of gastric injury.

GI ulcers are recognized complications of critical illnesses (e.g., hypotension, coagulopathy, sepsis) in people. They are thought to develop as a response to the stress of the critical illness and are termed "stress ulcers" (Henderson and Webster, 2006). Stress ulcerations are poorly defined entities in veterinary patients. However, gastroduodenal ulcerations have been noted in companion animals in conjunction with severe burns, heat stroke, multiple trauma, head injuries and spinal cord disorders. In addition, hypovolemic shock and sepsis may be complicated by development of GI ulcers. Experimentally, endotoxin in septic dogs decreases gastric blood flow resulting in mucosal ischemia. Histamine release stimulated by catecholamines worsened the mucosal damage (Henderson and Webster, 2006).

Gastrin-producing pancreatic tumors, histamine-producing tumors (e.g., mast cell tumors, basophilic leukemia) and a polypeptide-producing pancreatic tumor have been associated with gastric or duodenal ulceration in dogs and cats. Persistent gastric hyperacidity stimulated by gastrin, histamine or pancreatic polypeptide was thought to induce ulcers in these patients.

Helicobacter pylori has a recognized association with gastritis, gastroduodenal ulcers and gastric neoplasia in people. The role of *Helicobacter* spp. in GI disease in dogs is unclear although the prevalence is high. These spiral bacteria have been found in 67 to 100% of clinically healthy dogs and 74 to 90% of vomiting dogs. Gastric inflammation has been present in some, but not all, infected dogs. No significant relation has been demonstrated between *Helicobacter* spp. infection and clinical signs or GI ulceration in dogs. *Helicobacter* spp. have been identified in 40 to 100% of healthy and sick cats (Simpson, 2005; Henderson and Webster, 2006; Happonen et al, 2001; Rohrer et al, 1999; Simpson et al, 1999; Peters et al, 2005; Lecoindre et al, 2000).

Chronic Gastritis

Chronic gastritis generally is defined as intermittent vomiting that occurs for more than one to two weeks' duration (Hart, 2004) (**Box 52-1**). Vomiting of food or bile is the primary clinical manifestation of chronic gastritis. Other signs include decreased appetite, weight loss, hematemesis or melena (Simpson, 2005, 2006). Chronic gastritis is diagnosed based on histopathologic examination of gastric biopsy specimens. The histopathology (e.g., cellular infiltrate, architectural abnormalities and severity) and etiology, if identified, determine the type of chronic gastritis affecting the patient (Simpson, 2006).

The etiopathogenesis of chronic gastritis in dogs and cats is not fully understood. In some cases, an underlying etiology, such as parasitism or a metabolic disorder (e.g., uremia, liver disease), can be identified. In most cases, however, an immune-mediated response is hypothesized to be responsible for inflammatory infiltrates within the gastric mucosa (Simpson, 2005, 2006). Experimentally, chronic gastritis can be produced in dogs via mucosal irritants, systemic administration of gastric juices or prenatal thymectomy (Smith et al, 1958; Hennes et al, 1962; Krohn and Finlayson, 1973; Fukuma et al, 1988). Each of these treatments disturbs oral tolerance to antigens.

Chronic idiopathic gastritis is probably a subset of the IBD syndrome or may arise as an adverse reaction to food antigens. Chronic idiopathic gastritis may be localized or can occur with more diffuse IBD of the small or large bowel. Chapters 31 and 57 discuss adverse food reactions and IBD, respectively. Once present, inflammation interferes with gastric motility and reservoir function leading to vomiting. Nutrients including proteins are lost through the inflamed mucosal surface.

Key Nutritional Factors

Key nutritional factors for patients with gastritis and gastroduodenal ulceration are listed in **Table 52-2** and discussed in detail below.

Box 52-1. Hairballs.

Hairballs occur commonly in cats because of their normal grooming behavior and sharp barbs on the tongue that enhance hair ingestion. Cats with longer, thicker coats and those with fastidious grooming behavior usually have more problems with hairballs. Swallowed hair initially accumulates as loose aggregates or more compacted, soft aggregates mixed with mucus. Hairballs are regurgitated periodically from the oropharynx or esophagus or vomited from the stomach, or they pass into the intestinal tract, where they are voided in the feces. Owners observe periodic gagging, retching and regurgitation or vomiting of hair and mucus (usually not containing food or bile). Hairballs are often tubular.

Trichobezoars are harder concretions within the stomach or intestines formed of hair, mucus and other material. Trichobezoars probably begin as simple aggregates of hair, but progress to larger and harder concretions. They are less common in cats than typical hairballs, but are more likely to cause severe clinical signs. Trichobezoars are a common cause of anorexia in pet rabbits (Chapter 70). Large trichobezoars may obstruct pyloric outflow or the intestines and must be removed by surgery or endoscopy.

How cats eliminate aggregates of hair is probably similar to how they eliminate the pelts of small mammals that are ingested as part of a natural diet. Cats that hunt frequently may be seen vomiting the pelts of voles, mice, small rabbits and other mammals. This may be a protective mechanism for eliminating less digestible portions of prey.

Although hairballs do not usually cause significant clinical disease, their associated clinical signs are considered to be a nuisance by many cat owners. Hairballs generally can be controlled. Various laxatives, lubricants, treats and foods are available for routine management of these problems. Several commercial foods are available to help reduce the frequency with which cats vomit hairballs. Most of these foods have increased amounts of dietary fiber. Insoluble fiber, specifically cellulose, increases fecal hair content as compared to other fibers when incorporated in complete foods. Kibble size is another important feature of foods designed to reduce vomiting associated with hairballs. Radiographic gastrointestinal transit studies indicate that a larger kibble size is associated with an increased tendency for hairballs to exit the stomach and be eliminated in the feces, thereby reducing the frequency of vomiting. There is little or no evidence to support the use of lubricants (e.g., petroleum jelly) or papain for the treatment of hairballs in cats. If used, laxatives and lubricants should be given intermittently because large daily doses may interfere with normal digestion and nutrient absorption.

Frequent regurgitation or vomiting of hairballs (i.e., every day) with or without diarrhea, weight loss, anorexia or abdominal pain usually indicates an underlying problem (e.g., gastric motility defect or lymphoplasmacytic enteritis). Cats with severe or frequent clinical signs should be evaluated more extensively with diagnostics including hematology, serum biochemistry profiles, radiography and upper gastrointestinal endoscopy.

The Bibliography for **Box 52-1** can be found at www.markmorris.org.

Water

Water is the most important nutrient for patients with acute vomiting because of the potential for life-threatening dehydration due to excessive fluid loss and inability of the patient to replace those losses. Patients with persistent nausea and vomiting should be supported with subcutaneous or intravenous rather than oral fluids. Moderate to severe dehydration should also be corrected with appropriate parenteral fluid therapy.

Electrolytes

Gastric and intestinal secretions differ from extracellular fluids in electrolyte composition, so their loss can result in systemic electrolyte abnormalities. Dogs and cats with vomiting and diarrhea may have low, normal or high serum potassium, chloride and sodium concentrations. The derangement that predominates in a particular animal depends on several factors, such as the severity of the disease, nutritional status of the patient and site of the disease process. Serum electrolyte concentrations are helpful in tailoring appropriate fluid therapy and nutritional management of these patients. Mild hypokalemia, hypochloremia and either hypernatremia or hyponatremia are the electrolyte abnormalities most commonly associated with acute vomiting (and diarrhea).

Total body depletion of potassium is a predictable consequence of severe or chronic GI disease because the potassium concentration of gastric and intestinal secretions is high. Hypokalemia in association with GI disease will be particularly profound if losses are not matched by sufficient intake of potassium.

Electrolyte disorders should be corrected initially with appropriate parenteral fluid and electrolyte therapy. Foods for patients with acute gastroenteritis should contain levels of potassium, chloride and sodium above the minimum allowances for normal dogs and cats. Recommended levels of these nutrients are 0.8 to 1.1% potassium (dry matter [DM]), 0.5 to 1.3% DM chloride and 0.3 to 0.5% DM sodium.

Protein

Foods for patients with acute gastritis and/or gastroduodenal ulcers should probably not provide excess protein (no more than 30% for dogs and 40% for cats). Products of protein digestion (peptides, amino acids and amines) increase gastrin and gastric acid secretion (Feldman and Grossman, 1980; Delvalle and Yamada, 1990).

Some authors recommend "hypoallergenic" or elimination foods for patients with chronic idiopathic gastritis because dietary antigens are suspected to play a role in the etiopathogenesis (Guilford, 1997). In some cases, elimination foods may be used successfully without pharmacologic intervention because mild to moderate chronic gastritis may respond to dietary management alone. Ideal elimination foods should: 1) avoid protein excess (16 to 26% for dogs; 30 to 40% for cats), 2) have

Table 52-3. Key nutritional factors in selected commercial veterinary therapeutic foods compared to recommended levels for dogs with gastritis and/or gastroduodenal ulceration.*

Moist foods**	Potassium (%)	Chloride (%)	Sodium (%)	Protein (%)***	Fat (%)	Crude fiber (%)
Recommended levels	**0.8-1.1**	**0.5-1.3**	**0.3-0.5**	**≤30**	**<15**	**≤5**
Hill's Prescription Diet i/d Canine	0.95	1.22	0.44	25.0	14.9	1.0
Iams Veterinary Formula						
Intestinal Low-Residue	0.84	0.84	0.53	35.9	13.2	3.9
Medi-Cal Gastro Formula	0.6	na	0.6	22.1	11.7	1.0
Purina Veterinary Diets						
EN GastroENteric Formula	0.61	0.78	0.37	30.5	13.8	0.9
Royal Canin Veterinary Diet						
Digestive Low Fat LF	0.74	1.06	0.39	31.9	6.9	3.0
Royal Canin Veterinary Diet Intestinal HE	0.80	0.92	0.57	23.1	11.8	1.4
Dry foods	**Potassium (%)**	**Chloride (%)**	**Sodium (%)**	**Protein (%)*** **	**Fat (%)**	**Crude fiber (%)**
Recommended levels	**0.8-1.1**	**0.5-1.3**	**0.3-0.5**	**≤30**	**<15**	**≤5**
Hill's Prescription Diet i/d Canine	0.92	1.04	0.45	26.2	14.1	2.7
Iams Veterinary Formula						
Intestinal Low-Residue	0.90	0.66	0.35	24.6	10.7	2.1
Medi-Cal Gastro Formula	0.8	na	0.5	22.9	13.9	1.9
Purina Veterinary Diets						
EN GastroENteric Formula	0.66	0.85	0.60	27.0	12.6	1.5
Royal Canin Veterinary Diet						
Digestive Low Fat LF 20	0.88	1.10	0.49	24.2	6.6	2.3
Royal Canin Veterinary Diet Intestinal HE 28	0.88	0.99	0.55	33.0	22.0	1.6

Key: na = information not available from manufacturer.
*From manufacturers' published information or calculated from manufacturers' published as fed values; all values are on a dry matter basis unless otherwise stated.
**Moist foods are best and ideally they should be offered at temperatures between 70 to 100°F (21 to 38°C).
***Dietary protein may need to be limited to one or two sources that the patient has not been exposed to previously. Table 31-5 contains foods with these characteristics.

high protein digestibility (≥87%) and 3) contain a limited number of novel protein sources to which the patient has never been exposed. Alternatively a food containing a protein hydrolysate may be fed (Chapter 31).

Fat

Solids and liquids higher in fat are emptied more slowly from the stomach than similar foods with less fat. Fat in the duodenum stimulates the release of cholecystokinin, which delays gastric emptying. Foods with less than 15% DM fat for dogs and less than 25% DM fat for cats are appropriate for dietary management of gastritis and gastroduodenal ulcers.

Fiber

Many grocery brand moist foods contain gelling agents such as gums or hydrocolloids to enhance the aesthetic characteristics of the food. Foods containing gel-forming soluble fibers should be avoided in patients with gastric emptying and motility disorders because they increase the viscosity of ingesta and slow gastric emptying. Such fibers include pectins and gums (e.g., gum arabic, guar gum, carrageenan, psyllium gum, xanthan gum, carob gum, gum ghatti and gum tragacanth). However, increased levels (>8% DM crude fiber) of insoluble fiber (powdered cellulose) in dry foods fed to cats had no effect on gastric emptying (Armbrust et al, 2003). Other reports show that the ratio of slowly to rapidly fermentable fibers is important. Because of the variability of fiber types on gastric emptying, in general, the crude fiber content of foods for patients with gastritis and gastroduodenal ulcers should probably not exceed more than 5% DM.

Food Form and Temperature

Moist foods are best because they reduce gastric retention time. For the same reason, clients should warm foods to between room and body temperature (70 to 100°F [21 to 38°C]).

Other Nutritional Factors

Vitamins and Trace Minerals

Iron, copper and B vitamins may benefit patients with gastroduodenal ulceration and GI blood loss. Hematinics should be used in patients with nonregenerative, microcytic/hypochromic anemias attributable to iron deficiency. Hematinics probably are unnecessary in most animals that receive blood transfusions.

Acid Load

Alkalemia should be expected if vomiting patients lose hydrogen and chloride ions in excess of sodium and bicarbonate. Hypochloremia perpetuates the alkalosis by increasing renal bicarbonate reabsorption. Mild alkalemia is common, but profound alkalemia is more likely to occur with pyloric or upper duodenal obstruction rather than with acute gastritis.

Acidemia may occur in vomiting patients if the vomited gastric fluid is relatively low in hydrogen and chloride ion content (e.g., during fasting) or if concurrent loss of intestinal sodium and bicarbonate occurs. Severe acid-base disorders are best corrected with parenteral fluid and electrolyte therapy. Foods for

Table 52-4. Key nutritional factors in selected commercial veterinary therapeutic foods compared with recommended levels for cats with gastritis and/or gastroduodenal ulceration.*

Moist foods**	Potassium (%)	Chloride (%)	Sodium (%)	Protein (%)***	Fat (%)	Crude fiber (%)
Recommended levels	**0.8-1.1**	**0.5-1.3**	**0.3-0.5**	**≤40**	**<25**	**≤5**
Hill's Prescription Diet i/d Feline	1.06	1.18	0.33	37.6	24.1	2.4
Iams Veterinary Formula Intestinal Low-Residue	0.93	0.69	0.40	38.4	11.7	3.7
Medi-Cal Hypoallergenic/Gastro	1.1	na	0.7	35.5	35.9	1.2
Medi-Cal Sensitivity CR	1.1	na	1.1	34.5	35.1	2.5
Dry foods	**Potassium (%)**	**Chloride (%)**	**Sodium (%)**	**Protein (%)*** **	**Fat (%)**	**Crude fiber (%)**
Recommended levels	**0.8-1.1**	**0.5-1.3**	**0.3-0.5**	**≤40**	**<25**	**≤5**
Hill's Prescription Diet i/d Feline	1.07	1.11	0.37	40.3	20.2	2.8
Iams Veterinary Formula Intestinal Low-Residue	0.66	0.63	0.25	35.8	13.7	1.8
Medi-Cal Hypoallergenic/Gastro	0.8	na	0.4	29.8	11.5	3.1
Purina Veterinary Diets EN GastroENteric	0.99	0.58	0.64	56.2	18.4	1.3
Royal Canin Veterinary Diet Intestinal HE 30	0.97	0.97	0.65	34.4	23.7	5.8

Key: na = information not available from manufacturer.
*From manufacturers' published information or calculated from manufacturers' published as fed values; all values are on a dry matter basis unless otherwise stated.
**Moist foods are best and ideally they should be offered at temperatures between 70 to 100°F (21 to 38°C).
***Dietary protein may need to be limited to one or two sources that the patient has not been exposed to previously. Table 31-6 contains foods with these characteristics.

patients with acute vomiting and diarrhea should avoid excess dietary acid load. Foods that normally produce alkaline urine are less likely to be associated with acidosis.

FEEDING PLAN

The first objective in managing vomiting patients should be to correct dehydration and electrolyte and acid-base imbalances, if present. The dietary goals are to provide a food that meets the patient's nutrient requirements, allows normalization of gastric motility and function and controls vomiting. In most cases of acute vomiting, initial fasting for 24 to 48 hours, with parenteral fluid administration, reduces or resolves vomiting by simply removing the effects of undigested food and the offending agents from the stomach and duodenum. Chronic vomiting cases generally require a more detailed diagnostic and therapeutic (i.e., combined medical and nutritional) approach.

Assess and Select the Food

Bland foods often are recommended for veterinary patients with gastritis. This recommendation probably originated from physicians' orders for people recovering from GI upsets to eat bland foods. The term "bland" is poorly defined, but it is most often applied to easily digested/absorbed and nonirritating, non-spicy foods. Most pet foods fall within this category. The use of topical digests on dry foods may be construed as potentially irritating because many digests contain high concentrations of reactive amines (Guilford et al, 1994). The term "bland" is not a useful recommendation to pet owners; instead specific ingredients or nutrients to avoid should be clearly stated.

Tables 52-3 and **52-4** include the key nutritional factor content of selected commercial veterinary therapeutic foods marketed for GI diseases and compare them to the recommended levels for vomiting patients (dogs and cats, respectively). Food selection should be based on a product closely matching the key nutritional factor target levels.

Liquids are emptied from the stomach more quickly than solids due to lower digesta osmolality. Water is emptied most quickly, whereas liquids containing nutrients are emptied more slowly. High-osmolality fluids are emptied more slowly than dilute fluids. Solids are the slowest to be emptied from the stomach. Dry foods empty more slowly than moist foods in cats (Goggin et al, 1998). Thus, foods for patients with gastritis and gastroduodenal ulcers should have a liquid or semi-liquid consistency. Cold meals slow gastric emptying so food should be between room and body temperature (70 to 100°F [21 to 38°C]). Refrigerated or frozen foods should be warmed before being fed.

Assess and Determine the Feeding Method

Two feeding methods have been described for patients with acute gastric disorders. The more classic feeding method for patients with acute gastritis begins by discontinuing oral intake of food and water (i.e., nothing per os [NPO]) for 24 to 48 hours. After this period, patients should be offered small amounts of water or ice cubes every few hours. If water is well tolerated, small amounts of food can be offered several times (i.e., six to eight times) a day. In cats and probably dogs, larger meals are emptied more slowly than smaller meals (Goggin et al, 1998); thus, smaller meals promote gastric emptying. If the patient eats food without vomiting, the amount fed can be increased gradually over three to four days until the patient is receiving its estimated daily energy requirement in two to three meals per day. Food should be withdrawn and offered again after a few hours if the patient begins to vomit during this period.

In some cases, persistent vomiting may complicate refeeding. If so, metoclopramide or other antiemetic agents are rec-

ommended after GI obstruction has been ruled out (Table 51-2). Rarely, some patients may require parenteral feeding (Chapter 26).

The second approach, known as "feeding through vomiting," has been a successful alternative to NPO therapy in some vomiting patients. Pregnant women suffering hyperemesis reported feeling less nausea and preferred the placement of a nasogastric tube with slow frequent self feeding of small liquid meals to eating small regular meals or NPO therapy (MacBurney, 1993). This feeding method has also been used successfully in dogs with parvoviral enteritis (Mohr et al, 2003). A possible explanation for persistent vomiting is that the normal motility pattern throughout the length of the bowel cannot be reestablished without strong intraluminal stimulation. In fact, vomiting and mucosal atrophy probably perpetuate bowel dysfunction. Feeding restarts normal patterns of motility beginning in the esophagus and food may reestablish motility patterns as it passes down the bowel. The physical presence of food and nutrients serves as mechanical and chemical stimuli to normalize bowel motility and function.

Simply refeeding dogs (orally) and cats (via nasoesophageal tube) has stopped protracted vomiting (i.e., lasting more than seven days) successfully without using antiemetic drugs.[c] Feedings are continued although the patient may vomit. Most cases of protracted vomiting cease within 24 hours of administering liquid food. These patients then are offered small frequent meals of a highly digestible, moderate-fat food 24 hours after the last episode of vomiting (**Tables 52-3** and **52-4**).

CONCURRENT MEDICAL THERAPY

Nutritional management often is used in conjunction with other therapeutic modalities including parenteral fluids, antacids, histamine (H_2)-receptor antagonists, cytoprotective drugs, PGE_2 analogs, antibiotics and anthelmintics (Table 51-2).

REASSESSMENT

Nutritional reassessment of patients with gastritis or gastroduodenal ulcers includes monitoring changes in body weight and condition and determining the extent of vomiting. Daily food dosage should be adjusted as indicated by changes in body weight and condition.

If vomiting persists in the face of appropriate medical and nutritional therapy, further diagnostics are warranted. Additionally, different foods should be tried (**Tables 52-3** and **52-4**). If anemia was identified as a problem in pets with GI ulcers, reassessment of the hemogram is recommended to ensure adequate repletion of iron and copper. In addition, frequent monitoring of fecal occult blood loss is recommended.

ENDNOTES

a. Gastrografin. Squibb Diagnostics, New Brunswick, NJ, USA.
b. CLOtest, Ballard Medical Products, Draper, UT, USA.
c. Remillard RL. Personal observation. 1998.

REFERENCES

The references for **Chapter 52** can be found at www.markmorris.org.