

# The Canine Gastrointestinal Tract: Stomach



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## KEY POINTS

- **Dietary rest and rehydration for 24 hours are indicated in acute gastritis and gastroenteritis to allow gastrointestinal “rest” and relief from vomiting; this should be followed for several days with feeding a highly digestible, “bland” diet before gradually introducing the patient’s long-term diet.**
- **A very short-term restriction of dietary protein may be helpful in cases of gastritis but should not be continued long term as it inhibits gastric healing and immune responses.**
- **In chronic gastritis, starvation is not indicated and feeding a novel, “hypoallergenic” protein is indicated, particularly in lymphocytic-plasmacytic or eosinophilic gastritis, where there may be an underlying immune-mediated process.**
- **Generally, a low-fat, low-fiber diet is indicated in chronic gastric disease, as fat and soluble fiber slow gastric emptying and increase acid secretion.**
- **Feeding little and often overcomes the reduced gastric compliance that is common in gastric disease and may thus reduce vomiting.**
- **Feeding liquid foods little and often may help speed gastric emptying and is indicated in most gastric diseases; feeding a liquid low-fat diet little and often may indeed be the only manipulation necessary to control functional delayed gastric emptying (pylorospasm).**
- **Feeding a more solid diet higher in fat and fiber is indicated in cases where delayed gastric emptying is beneficial (i.e., with “gastric dumping” syndrome and bilious vomiting associated with gastroduodenal reflux).**

Gastric disease in dogs often (but not always) causes vomiting. However, many nongastric diseases will also cause vomiting, and it is important to rule out such diseases and to differentiate carefully between vomiting and regurgitation before implicating the stomach. Some diseases are confined to the stomach, but many diseases also involve other parts of the gastrointestinal (GI) tract, particularly the small intestine, and thus treatment is often aimed at several areas. Nonspecific, self-resolving acute gastritis or

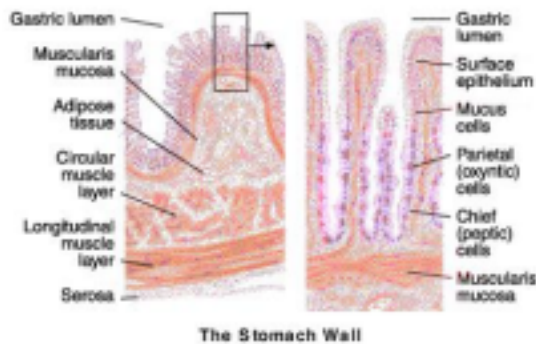
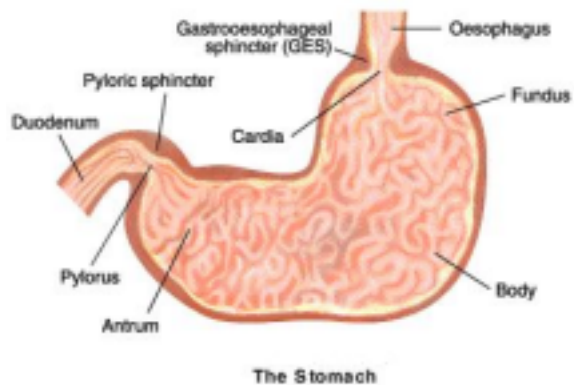
gastroenteritis is the most common GI disease seen in small animal practice and can often be controlled by dietary management alone. Drugs and/or surgery are usually necessary in the treatment of more chronic gastric disease, but dietary manipulations also play a pivotal role, as the composition and consistency of the diet has a profound effect on gastric function and healing ability.

## ANATOMY

The stomach can be divided into cardia, fundus, body, antrum, and pylorus. Gastric glands provide all the secretions.

The stomach wall is made up of different layers, which are (from lumen outwards):

- The mucosa
- The submucosa
- Two layers of muscularis, with an outer longitudinal and an inner circular layer
- The serosa



The gastric mucosa forms an important protective barrier against autodigestion. First, the layer of bicarbonate ion-containing mucus protects against the stomach acid. Additionally, rapid epithelial cell turnover and a good mucosal blood supply may also limit any damage.

## FUNCTION

### Secretions

Gastric secretions are produced in response to food intake. The parietal cells that secrete HCl are receptive to both neural (acetylcholine) and hormonal (histamine) signals; histamine is released in response to stimulation from the locally secreted hormone gastrin.

Gastrin itself is released from the G cells in the gastric antrum and duodenum, and release is stimulated by both the presence of protein in the stomach and amino acids in the small intestine. Also, stomach distension and stimulation by the vagus nerve (e.g., in sham feeding) stimulate gastrin release.

During meals, gastric pH is about 1 to 1.5. Unlike in humans, the secretion of gastric acid is intermittent in cats and dogs, and during low-fasting acid secretion between meals, gastric pH can rise as high as 3 to 6.5. Therefore, blood in vomit may appear either as hematemesis (digested, “ground coffee” appearance) or as fresh blood.

### Motility

As the stomach stretches during a meal, vagally mediated inhibition allows gastric smooth muscle to relax, and the stomach size increases without an increase in pressure. Gastric inflammation and neoplasia easily disrupt this “compliance,” resulting in vomiting on eating.

Gastric contractions mix and break up the food against a closed pylorus, letting out particles of 1 to 2 mm in size, except between meals when giant contractions (migrating motility complexes) push out larger items (e.g., foreign bodies). Under normal conditions, the pylorus contracts 2 to 3 times per minute and the stomach normally empties by 10 to 12 hours after a meal; however, this may vary with the type and size of food.

### Vomiting Reflex

Nausea increases salivation and swallowing, with or without tachycardia. The next step is antiperistalsis of duodenum and jejunum and reduction in gastric tone. The animal will then start to retch with a spasm of the diaphragm and the intercostal and abdominal muscles. This allows distal esophageal sphincter pressure to be overcome. During the act

of vomiting, the distal esophageal sphincter relaxes and food is pushed out of the relaxed stomach by action of abdominal and respiratory muscles.

### ***Chemoreceptor Trigger Zone***

There are two “classical” pathways for stimulating vomiting, the humoral pathway and the neural pathway. The neural pathway is from the vomiting center in the lateral reticular formation of the medulla. Afferents pass to the vomiting center in the Xth cranial nerve (vagus) and sympathetic nerves (from stomach and abdominal organs), IX (from pharynx), VIII (from vestibular apparatus), and in nerves from the chemoreceptor trigger zone (CRTZ) and higher centers.

The humoral pathway is via stimulation of the CRTZ by blood-borne substances. The CRTZ is positioned in the dorsal medulla on the floor of the fourth ventricle and, importantly, outside the blood-brain barrier, so that it can be stimulated by substances in the plasma, which would not otherwise have been able to cross into the brain. Substances that stimulate the CRTZ are much better understood than the vomiting center, and there are also important species differences affecting the toxicity of drugs and usefulness of centrally acting antiemetic drugs.

There are receptors in both the vomiting center and the CRTZ for a number of different neurotransmitters. Centrally acting antiemetics are antagonists of neurotransmitters in the CRTZ and the vomiting center. There are some important species differences in the central control of vomiting that result in variations in the way vomiting is triggered and the drugs used for treatment. Motion sickness, for example, is mediated via the CRTZ in dogs but via the vomiting center in cats. Also, dopamine is an important transmitter in the CRTZ of dogs but not cats. Apomorphine, a dopamine agonist, therefore induces vomiting in dogs but not cats. Likewise, dopamine antagonists, such as metoclopramide, are less effective antiemetics in cats than in dogs.

## **CLINICAL DISORDERS**

Acute gastroenteritis is the most common gastric disorder seen in the dog and is often due to scavenging or a change of diet. Gastric foreign bodies, gastric motility disorders (pylorospasm and gastroduodenal reflux), and gastric ulceration are also relatively common. Congenital or acquired pyloric stenosis typically causes projectile vomiting. Certain toxins and infectious organisms also cause gastric disease, usually with concurrent enteritis. Chronic inflammatory gastritis is recognized and usually classified by histologic appearance, although the etiology is often unclear. Gastric neoplasia may also be seen but is uncommon.

## **Clinical and Physical Findings in Gastric Disease**

### ***General Physical Examination***

Patients may be dehydrated and show signs of electrolyte imbalance, such as hypokalemia, with chronic vomiting. Bleeding from ulcers can lead to marked anemia. If the intestine is also involved, the dog may have concurrent diarrhea.

Patients may further show signs of concurrent systemic disease or peritonitis. Severe and prolonged cases may show weight loss and cachexia.

### ***Abdominal Palpation***

Careful abdominal palpation is important and may reveal pain in the gastric region with severe disease or peritonitis. Thorough palpation of the total GI tract may also help to diagnose foreign bodies; these can be found at any part of the GI tract from the mouth (e.g., string under the patient's tongue) to the esophagus, stomach, and intestine.

### ***Vomiting***

Vomiting may reveal hematemesis or fresh blood; significant gastric bleeding will be accompanied by melena.

Vomiting is an important diagnostic aspect, and it is important to differentiate gastric and nongastric causes of vomiting.

Patients may vomit food (e.g., in gastritis or gastric motility disorders) or vomit on an empty stomach (e.g., with gastric ulcers or bilious vomiting). Food may be vomited immediately or a long time after feeding, giving an indication about gastric emptying time.

Bile should normally be present in vomitus, as reverse peristalsis of the duodenum is part of the vomiting reflex. Its absence suggests a functional or mechanical pyloric obstruction.

### ***Nongastric Causes of Vomiting***

Nongastric causes of vomiting in the dog include:

- **Central Disorders**
  - Stimulation of the CRTZ
    - Apomorphine

- Cardiac glycosides
- Certain toxins (e.g., urea, bacterial toxins)
- Metabolic disorders (e.g., ketoacidosis)
- Stimulation of vomiting center by disease of the central nervous system
  - Neoplasia
  - Inflammation
  - Epilepsy
- Stimulation of vomiting center by higher centers (fear, stress, pain)
- **Extragastric Abdominal Disease**
  - Small and/or large intestinal infection, toxin, foreign body, obstruction, volvulus, intussusception, and/or inflammation
  - Acute pancreatitis
  - Acute hepatitis
  - Acute renal disease, urinary tract obstruction, and/or uremia
  - Pyometritis
  - Peritonitis
- **Metabolic/Endocrine Disease**
  - Hypoadrenocorticism (“Addison’s disease”)
  - Hypokalemia
  - Hypercalcemia (e.g., malignancy, hyperparathyroidism)
  - Diabetic ketoacidosis

### ***Laboratory Tests in Gastric Disease***

Blood samples for hematology and biochemistry screens (including electrolytes) are indicated in all cases of acute severe and chronic vomiting to rule out nongastric causes of vomiting and to assess the electrolyte, albumin, acid-base, and hydration status and anemia.

Hypokalemia is particularly common with gastric disease and causes GI hypomotility and thus may prolong the disease. Hypoalbuminemia occurs in some cases of ulceration, severe inflammatory gastritis, and gastric neoplasia. It is important in these dogs to raise blood albumin by dietary manipulation if possible, particularly if gastric biopsies are planned, as hypoalbuminemia delays mucosal healing and significantly increases the risk of postoperative wound breakdown.

B12, folate, and TLI should be measured if concurrent small intestinal/pancreatic disease is suspected.

Feces samples and/or a serologic examination should be carried out if infectious disease is suspected.

## ***Radiography***

Plain abdominal radiographs may show foreign bodies, evidence of delayed gastric emptying, peritonitis, and abnormalities of other abdominal organs. Gastric emptying can, however, also be delayed in nervous animals.

Thoracic radiographs are useful for the assessment of hypovolemia, thoracic metastases, and hiatal hernia.

Contrast examinations with double-contrast gastrography (barium and air) gives the best details of the gastric mucosa and wall thickness. For the diagnosis of gastric neoplasia and radiolucent foreign bodies, negative-contrast gastrography can be equally useful.

Barium impregnated polyethylene spheres (BIPS) may also give information about gastric emptying times with different sized particles; however, they allow no assessment of the mucosal structure.

## ***Ultrasonography***

Ultrasonography can be used to assess the thickness of the gastric wall and to identify local areas of thickening associated with severe inflammation, ulcers, or tumors. Neoplasia often results in the loss of the normal five-layered wall structure on ultrasonography.

Ultrasonographic examination may also identify tumor metastasis (e.g., in the liver).

Ultrasonography is further useful to assess local wall thickness in pyloric stenosis, the thickness and layers of the small intestine, and other abdominal organs, such as the pancreas.

## ***Endoscopy***

Endoscopy is very useful in the diagnosis of primary gastric disease as it enables the visualization of the whole stomach and the proximal duodenum and allows the acquisition of mucosal biopsies. Some foreign bodies can be removed with a special “net.”

The disadvantage is that only the mucosal surface of stomach can be visualized and assessed. Therefore, a disease process (e.g., tumor) lying entirely within the serosa and muscularis will not be seen.

## ***Exploratory Laparotomy and Biopsy in Gastric Disease***

Laparotomy allows for the assessment and biopsy of the full thickness of the stomach wall and visualization (and biopsy) of the rest of the abdomen and GI tract.

However, this is a highly invasive procedure and should only be carried if fully indicated. Care should be taken with cachectic patients as protein-energy malnutrition and hypoalbuminemia increase the risk for wound breakdown and delayed wound healing, and nutritional support may be crucial.

## **Acute Gastric Disease**

The most common cause of acute gastroenteritis in dogs is dietary indiscretion, and the management of this remains predominantly dietary. Other causes of acute gastric (and often concurrent small intestinal) disease include toxins and drugs, such as lead and nonsteroidal antiinflammatory drugs (NSAIDs), and infectious organisms, such as canine parvovirus, *Salmonella*, and roundworms. Gastric dilatation - volvulus (GDV) is an acute gastric disease of particular importance in some breeds. Other nongastric causes of acute vomiting such as pancreatitis, electrolyte disturbances, toxemia, and acute renal failure must be carefully ruled out before gastric disease is investigated further.

## ***Management of Acute Gastric Disease***

The most common cause of acute gastritis/gastroenteritis is dietary indiscretion, such as scavenging, and dietary management plays a crucial role in acute gastric disorders. Often, a definite diagnosis is not determined because the symptomatic treatment is effective in reversing the clinical signs. Along with any specific medical therapy, acute diarrhea is typically managed by providing dietary rest and rehydration therapy followed by the introduction of a “bland” diet.

A “bland” diet can be defined as a high-quality, highly digestible, and nonspicy diet containing components that pose a low risk of adverse reactions. This ensures that the enterocytes are presented with minimal digestive challenge and minimizes the likelihood of acquired food allergies or intolerance.

## Gastric Dilatation - Volvulus

Gastric dilatation - volvulus often presents as a medical and surgical emergency. Once the patient is stabilized, however, life-long dietary therapy plays an important role. GDV is predominantly seen in large, deep-chested dogs. Epidemiologic studies have shown that German Shepherds, Great Danes, and Boxers account for the majority of all cases (Dupre and Corlouer 1994).

Breed	GDV risk (Odds ratio)	Risk rank
Great Dane	41.4	1
Saint Bernard	21.8	2
Weimaraner	19.3	3
Irish Setter	14.2	4
Gordon Setter	12.3	5
Standard Poodle	8.8	6
Beagle Hound	5.3	7
Dobermann Pinscher	5.5	8
Old English Sheepdog	4.8	9
German Short-haired Pointer	4.6	10
Newfoundland	4.4	11
German Shepherd Dog	4.2	12

Risk of GDV in Different Breeds  
(Source Dupre and Corlouer 1994)

The following factors have been suggested to be associated with an increased risk of GDV:

- High thoracic depth:width ratio
- Once daily feeding, although the type of food does not seem to matter
- Rapid eating
- Nervous temperament
- Stress
- Dogs being male and underweight
- Accumulation of gas in the stomach through aerophagy and bacterial fermentation
- Previous episodes of dilatation

The primary cause of GDV is poorly understood. A number of studies have investigated the role of diet as a cause of GDV, but no particular dietary regimen has found to increase or reduce the incidence of disease. It has been suggested that feeding one large meal a day encourages gastric hypertrophy and dilatation; however, an increased risk needs to be demonstrated (Dupre and Corlouer 1994). An underlying gastric motility disorder and an association between GDV and megaesophagus has also been implicated.

Stretched or dilated gastric ligaments have been suggested to play a role in the etiology of GDV; however, a normal stomach can easily be put in the position of volvulus during laparotomy. It seems that the initial dilatation causes a displacement of the pylorus and the body of the stomach, which then leads to further torsion.

## ***Surgical Management of Gastric Dilatation - Volvulus***

Patients typically present with shock and unproductive retching and gastric tympany. They are often in cardiovascular depression and may develop complicating coagulation disturbances. The initial emergency treatment encompasses preoperative stabilization of the patient, surgical correction, and postoperative patient monitoring.

Medical stabilization is crucial in dogs with GDV. A 30% mortality rate has been shown, with dogs dying of circulatory shock due to obstruction of venous return and reperfusion injury. This also includes the “myocardial depressant factor,” which may cause death from cardiac arrhythmias up to 72 hours after surgery; indeed, 35% of fatalities occur 2 to 4 days postoperatively.

- Decompress stomach with stomach tube or needle with/without lavage using warm saline
- Concurrently, start rapid intravenous fluids; two lines may be necessary
- Consider medical shock treatment
- Parenteral antibiotics may be indicated
- Surgery involves pexy and/or removal of devitalized tissue
- Postoperative administration of fluids and electrolytes; hypokalemia is common and worsens arrhythmias; administration of antibiotics
- Careful monitoring for arrhythmias (ventricular tachycardia is most common) for at least 3 days after surgery; the use of antiarrhythmic agents may be necessary
- If gastric ulcers are evident, antiulcer therapy may be required
- Careful postoperative dietary management

## ***Dietary Considerations in Gastric Dilatation - Volvulus***

The role of diet in the cause and prevention of GDV is poorly understood, and further investigations are required to fully understand this relationship. However, it has been recommended to feed predisposed dogs often and little and to postpone exercise for a couple of hours after feeding. Delayed gastric emptying may be seen after GDV, and patients with this complication may benefit from a liquid diet with a low-fat and soluble fiber content.

## Chronic Gastritis

The underlying etiology of chronic gastritis is often unknown, but an immune-mediated process is often implicated and dietary management with a “hypoallergenic” diet is an important part of treatment.

Gastritis can be classified according to the cell type and histologic picture identified in gastric biopsy.



Gastritis  
(Source K. Hurley)

### ***Lymphocytic-Plasmacytic (Superficial) Gastritis***

- Lymphocytes and plasma cells, with or without fibrosis in the mucosa and lamina propria
- The patient tends to be systemically well and show no gastric ulceration

### ***Atrophic Gastritis***

- Very rare
- Characterized by a thin mucosa with reduced glands

### ***Hypertrophic Gastritis***

- Rare
- Characterized by a grossly thickened mucosa and rugal folds
- May be diffuse or focal (e.g., around pylorus, causing acquired pyloric stenosis)
- Histologic examination reveals mucosal epithelial and/or glandular hypertrophy, with or without variable inflammatory cell infiltrate
- Patients are usually systemically affected with gastric ulcerations

## ***Eosinophilic Gastritis***

- Sometimes but inconsistently associated peripheral eosinophilia
- Diffuse eosinophilic infiltrate of the mucosa or full thickness focal eosinophilic granulomas are often seen and may look grossly neoplastic
- Patients tend to be systemically ill with gastric ulceration

## ***Lymphocytic-Plasmacytic and Eosinophilic Gastritis***

- May affect just the stomach or both the stomach and the small and large intestines
- An immune-mediated/allergic component and/or a relation to chronic duodenal reflux may be present
- “Endoscopic patch testing” indicates food allergy in some cases
- Inflammation in eosinophilic forms may be severe enough to result in hypoproteinemia, including albumin and globulins
- In some cases, eosinophilic gastritis or gastroenteritis is associated with an endoparasitic infection; dogs infected with *Toxocara* can develop focal eosinophilic gastritis, even with no larvae in the stomach

## ***Helicobacter spp.***

*Helicobacter* are gram-negative, microaerophilic spiral bacteria. In humans, *Helicobacter pylori* is recognized as an important cause of chronic gastritis, gastric ulceration, and malignant transformation.

Spiral bacteria are often found on gastric biopsies from dogs, and a prevalence as high as 67-86% of clinically healthy dogs has been documented (Simpson 1998). It is tempting to implicate these microorganisms in canine gastritis, but many recent studies suggest that they are unlikely to play the same role as in human gastritis.

*H. pylori* is not found in dogs except experimentally. Dogs are naturally infected with *Helicobacter canis*, *Helicobacter heilmanni* (causes 1% of *Helicobacter*-associated gastritis in humans), and others.

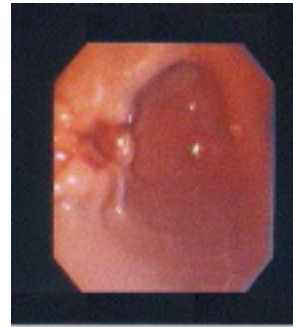
*Helicobacter* spp. may, however, play an important role due to their zoonotic potential. Recent case reports suggest the transmission of these microorganisms from pet to human (Simpson 1998), and treatment should therefore be considered even in mild manifestations in dogs.

Current treatment protocols are based on those effective in the treatment of humans with *Helicobacter* spp. infections and include the administration of antibiotics and an H2 antagonist (Simpson 1998).

## Gastric Ulceration

Gastric ulcerations are characterized by a break in the gastric mucosal barrier, which may be seen in conjunction in a number of different conditions, including:

- Hypertrophic and eosinophilic *gastritis*
- Gastric *neoplasia*
- *Uremia* in renal disease
- *Liver disease*
- *Gastric ischemia*
- The application of *NSAIDs*
- The application of *steroids* and concurrent stress/medication
- *Stress*
- *Histamine-secreting mast cell tumors*
- *Gastrinoma*



Gastric Ulcer  
(Source K. Hurley)

Dogs usually develop multiple ulcers, although in some cases there may be a single, large ulcer with neoplasia, similar to what is usually seen in humans. Ulcers may also involve the duodenum, especially with liver disease.

## *Uremia in Renal Failure*

- Ammonia is toxic to the gastric mucosa
- There is a reduced metabolism of gastrin protecting the mucosa and a reduced gastric blood flow

## *Liver Disease*

- Portal hypertension causes ischemia leading to reduced epithelial cell turnover and reduced mucus excretion

## ***Nonsteroidal Antiinflammatory Drugs***

- Acidic NSAIDs, such as aspirin, can cause direct mucosal injury. This is because they are less ionized in the acid environment of the stomach but more ionized in the higher pH of the gastric epithelial cells; nonionized NSAIDs cross cell membranes more easily, which results in “trapping” of high concentrations of NSAIDs within the gastric epithelial cells
- Inhibition of prostaglandin synthesis can reduce gastric mucus and bicarbonate secretion, increase acid secretion, and may reduce mucosal cell turnover

## ***Steroids***

- Steroids are rarely ulcerogenic alone but may reduce mucosal cell renewal and prostaglandin synthesis and therefore reduce mucus secretion and increase acid secretion

## ***Gastric Motility Disorders***

Disorders of gastric motility in the dog may occur secondary to a physical obstruction, such as hiatal hernia or GDV, or a pyloric foreign body. Alternatively, there may be a primary functional disorder, such as delayed gastric emptying or pylorospasm and gastroduodenal reflux (bilious vomiting syndrome).

These motility disorders generally result in delayed gastric emptying and vomiting of food long after feeding. An exception is the “bilious vomiting syndrome,” which results in vomiting bile on an empty stomach. A small number of dogs exhibit “gastric dumping,” which can be idiopathic or occur after certain surgical interventions such as colectomy, which disrupts the normal reflex slowing of gastric emptying when undigested nutrients reach the colon. “Gastric dumping” of large volumes of ingesta in the small intestine can cause maldigestion and/or malabsorption and diarrhea. Dietary management is usually of primary importance in the treatment of functional gastric motility disorders.

## ***Functional Delayed Gastric Emptying***

This term describes a group of disorders with a variety of causes and resulting in abnormalities of gastric myenteric nerve or smooth muscle function. Furthermore, the coordination between pylorus and duodenum may be impaired. The end result is a delay in stomach emptying, and food may be vomited a long time (more than 10-12 hours) after feeding.

This may result from an obvious gastric disease (e.g., infection, inflammation, ulcers, or recovery from GDV). Alternatively, it may be due to secondary conditions, such as metabolic and electrolyte disturbance, the administration of certain drugs (e.g., anticholinergics, adrenergic agonists, opiates), acute abdominal inflammation (e.g., pancreatitis, peritonitis), or acute stress.

This disease complex also includes the classic “pylorospasm” seen particularly in nervous dogs of miniature and toy breeds.

### ***Gastroduodenal Reflux***

Gastroduodenal reflux or bilious vomiting syndrome has a typical history, with otherwise healthy dogs vomiting bile only in the mornings. The condition is quite common, particularly in nervous dogs. It is normal for some bile to reflux back in to the stomach, but it should be rapidly cleared. It has been suggested that a motility disorder in these patients results in prolonged contact between bile and gastric mucosa. As bile is a detergent, this can further result in mucosal damage and focal antral gastritis.

The vomiting of bile occurs in the early morning because the stomach is empty and the bile is not diluted by food.

A definitive diagnosis is reached by ruling out other causes and demonstrating a response to treatment. Endoscopic examination may reveal antral gastritis with or without excessive bile retention. <sup>99</sup>Tc-labeled bile may be used in specialized referral centers.

### ***Pyloric Stenosis, Foreign Bodies, and Neoplasia***

Gastric foreign bodies, such as swallowed toys or stones, are the most common cause of pyloric outflow obstructions in dogs. Clinical signs usually include vomiting (immediately or a long time after eating) and can be intermittent as the foreign body moves in and out of the pyloric antrum.



Pyloric Foreign Body - Spring  
(Source: K. Hurley)



Pyloric Stenosis  
(Source K. Hurley)

Congenital pyloric stenosis caused by congenital muscular hypertrophy also leads to pyloric outflow obstruction. Congenital cases are predominantly seen in brachycephalic dog breeds, such as Boxers and Boston Terriers.

Acquired pyloric stenosis may be the result of external compression of the pylorus, caused, for example, by a pancreatic or hepatic tumor. Alternatively, such gastric lesions as gastric tumors, gastric polyps, or mucosal hypertrophy may block the pyloric antrum.

There is also an uncommon syndrome of acquired antral pyloric mucosal hypertrophy in middle-aged to old, small-breed dogs. This results in marked hypertrophy and folding of the mucosa, with the gastric muscle remaining normal. The exact cause is unclear; however, because it particularly affects small, excitable breeds, it may be related to chronic stress and pylorospasm.

In dogs, benign tumors are more common than malignant ones, with malignant gastric tumors only accounting for 1% of all tumors in the dog. Affected dogs are usually middle-aged to elderly.

Adenocarcinomas are the most common types found in dogs, whereas lymphosarcomas, fibrosarcomas, and leiomyosarcomas and gastric metastases are much less common.

Tumors can cause altered gastric motility, with reduced compliance and either rapid emptying or pyloric outflow obstruction, and are often ulcerated.



Gastric Neoplasia  
(Source K. Hurley)

Patients often present with vomiting, with or without hematemesis and melena, and often develop weight loss and protein-losing gastropathy. However, other animals present primarily with anorexia and very little vomiting.

## MANAGEMENT

Different gastric disorders may demand very different medical and surgical management. Dietary management is vitally important in all gastric diseases in order to allow gastric healing and resolution of clinical signs. Dietary management may be used alone or in combination with medical or surgical management depending on the disease involved.

### Dietary Management

Dietary manipulations are important as primary or supportive treatments in most gastric diseases. Except for short-term control of vomiting, dietary rest is usually inappropriate in most chronic gastric diseases. Dietary manipulations play a crucial role in the overall treatment because changes in the nutrient composition of the diet can have profound effects on healing of the gastric mucosa and gastric function in general.

### *Consistency of Diets and Feeding Methods*

Diets of different consistency vary significantly in gastric emptying time - even when their nutritional compositions are the same - because the pylorus only allows small particles in to the duodenum, except during meals. Liquid food empties most rapidly, tinned food more slowly, and dried food most slowly of all.

Delayed gastric emptying often occurs secondary to inflammatory gastritis, gastric neoplasia, pyloric stenosis, and GDV. It is also the primary problem in pylorospasm. Liquid or mushy foods are therefore indicated to speed gastric emptying in dogs with these problems.

Feeding a low-fat liquid food, little and often, may be the only therapy required in some cases of pylorospasm. Feeding the food little and often helps overcome the reduced gastric compliance associated with gastritis.

In a few cases, such as gastroduodenal reflux and gastric dumping, the aim is actually to slow gastric emptying; in these circumstances, a dry diet is indicated. Feeding the diet frequently, and particularly late in the evening, ensures there is food in the stomach overnight, which may be curative alone in gastroduodenal reflux.

Recurrent vomiting due to inflammation may counteract efforts to feed animals with chronic gastric disease. Judicious use of antiemetics and feeding an appropriate diet little and often may resolve the vomiting.

If the latter is severe and persistent in spite of therapy, there may be a role for enterostomy feeding tubes or total parenteral nutrition, although this is rarely necessary. Food should be given enterally wherever possible, as this provides more effective nutrition to both the patient as well as the GI tract itself. Also, food should be introduced as proximal as possible, as this ensured a more physiologically normal process of digestion and absorption.

## ***Energy***

Chronic gastric diseases associated with chronic vomiting and/or protein-losing gastropathy will predispose to the development of protein-calorie malnutrition. It is therefore important to feed a high-energy diet to enable sufficient calorie intake to maintain lean body mass and allow gastric healing. In gastric disease, however, these calories should typically be provided mainly in the form of carbohydrate and protein, as a high dietary fat density may be contraindicated in many cases.

## ***Protein***

Protein induces gastric acid secretion by inducing the release of the hormone gastrin from the gastric antrum and duodenum. Reducing dietary protein in the short term may therefore help dogs with gastritis and gastric ulceration, as it will reduce gastric acid secretion. It is, however, important not to restrict dietary protein long term in gastric disease, as protein restriction reduces epithelial cell turnover, the production of small intestinal brush border enzymes, and GI immunity.

Furthermore, protein is very important for palatability and provides an additional source of calories when feeding a fat-restricted diet. In acute gastritis, a highly digestible, high-quality protein should be fed, preferably from a single source, to reduce the occurrence and significance of acquired dietary hypersensitivity. In chronic gastritis, the long-term use of a single novel protein source in a “hypoallergenic” diet is more specifically indicated in lymphocytic-plasmacytic and eosinophilic gastritis because dietary allergy may be important in the underlying pathogenesis.

## ***Fat***

High-fat foods delay gastric emptying and thus increase acid secretion via an indirect increase in gastrin secretion. Fat also reduces gastroesophageal sphincter tone and therefore encourages gastroesophageal reflux.

Low-fat foods are therefore generally indicated in gastric disease, as they help:

- Reduce acid secretion in gastritis and gastric ulceration
- Hasten gastric emptying in functional delayed gastric emptying (pylorospasm)
- After surgery to correct GDV

A low-fat diet is also indicated in dogs with concurrent enteritis or pancreatic disease because fat maldigestion and malabsorption is common in these conditions.

## ***Carbohydrate and Fiber***

Carbohydrate provides the main source of calories in most gastric diseases in which a low-fat diet is indicated. Carbohydrates should therefore be highly digestible and readily available.

Fiber has varying effects on gastric emptying depending on the type:

- ***Soluble fiber*** delays gastric emptying, thus increasing acid secretion; it is therefore contraindicated in gastritis and most gastric diseases.
- ***Insoluble fiber*** may, however, actually speed gastric emptying and have a helpful buffering action while in the stomach; however, more work is necessary to assess its benefits before its use can be generally recommended.

It is generally recommended to avoid fiber in gastritis; in rare conditions, however, where the stomach empties too rapidly (“gastric dumping”), increased soluble fiber may be helpful.

## ***Vitamins and Minerals***

There are no vitamin requirements specific to gastric disease, but adequate amounts and a good balance of essential vitamins should be included in long-term diets for gastric disease. This is particularly important when hypoallergenic diets are used. Homemade

diets are often used short term for diagnosis but are best replaced in the long term with a suitable manufactured diet to avoid possible nutrient deficiencies and imbalances.

The most important mineral to consider in gastric disease is potassium, as chronic vomiting often results in hypokalemia. This is particularly seen with concurrent anorexia. Dietary supplementation with potassium may therefore be necessary after checking the patient's potassium status.

## **Surgical Management**

Surgical management is indicated in patients with gastric foreign bodies that cannot be retrieved endoscopically, congenital and acquired pyloric stenosis, and some types of gastric neoplasia, although most malignant gastric neoplasms are too extensive to resect. Surgery is particularly important in GDV.

## **Medical Management**

Medical management of gastric disease involves the use of antiemetics to control vomiting through central antiemetic and peripheral promotility effects or central actions, as long as there is no contraindication to their use (e.g., pyloric outflow). Gastric acid secretory inhibitors should be used in all cases of chronic gastritis, even if there is no clinically obvious ulceration, because mucosal damage in gastritis increases back diffusion of acid into the stomach wall.

In cases of gastric ulceration, acid secretory inhibitors can be used in conjunction with drugs lining the ulcer. In some cases of ulceration, it may also be possible to use antacids, which are particularly useful in uremic gastric problems because they also bind phosphate and reduce its absorption. Furthermore, synthetic prostaglandins (e.g., PGE<sub>1</sub>) can be used to prevent and treat drug-induced (steroids and NSAIDs) ulceration. New and emerging ulcer treatments in humans include the use of synthetic somatostatin, which reduces acid secretion, as well as agents to generate nitric oxide and encourage mucosal vasodilation.

Effective endoparasitic control should be ensured, particularly in cases of eosinophilic gastritis. The use of steroids may be indicated for lymphocytic-plasmacytic gastritis (antiinflammatory doses) and eosinophilic enteritis (immunosuppressive doses). However, their use must be balanced against their ulcerogenic potential. Promotility drugs have a role in bilious vomiting syndrome and pylorospasm.

## SUMMARY

The dietary management of gastric disease varies, depending on the disease involved. The three main considerations when recommending a diet for gastric disease are to assess:

- Whether the condition is acute or chronic
- If reduced gastric acid secretion would be beneficial
- If delayed or hastened gastric emptying would be beneficial

The ideal diet can then be chosen on the basis of these answers.

In acute gastritis, 24-hour dietary rest and rehydration are followed by feeding a highly digestible, “bland” diet, whereas dietary rest is not usually indicated in chronic cases.

When aiming for reduced gastric acid secretion, feeding a liquid diet low in fat and soluble fiber is recommended. This is equally true if hastened gastric emptying is desired. To delay gastric emptying, however, the opposite is suggested.

The aim in all cases is to complement and support medical and surgical management with appropriate dietary manipulations; in some cases, dietary changes alone may be curative.

## References

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