Gastric disease is usually the result of inflammation, ulceration, neoplasia, or obstruction and manifests clinically as vomiting, hematemesis, melena, retching, burping, hypersalivation, abdominal distension, abdominal pain, or weight loss. The clinical approach is simplified by considering gastric diseases as a group of clinical syndromes based on the combination of etiology, pathology, and clinical presentation:

<table>
<thead>
<tr>
<th>Clinical syndrome</th>
<th>Predominant features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute gastritis</td>
<td>Vomiting of sudden onset</td>
</tr>
<tr>
<td>Ulceration or erosion</td>
<td>Vomiting, hematemesis, melena, ± anemia</td>
</tr>
<tr>
<td>Gastric dilatation/ volvulus</td>
<td>Non-productive retching, abdominal distention, tachycardia</td>
</tr>
<tr>
<td>Chronic gastritis</td>
<td>Chronic vomiting of food or bile</td>
</tr>
<tr>
<td>Delayed gastric emptying</td>
<td>Acute to chronic vomiting more than 8 to 10 hr after feeding</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>Chronic vomiting, weight loss, ± anemia</td>
</tr>
</tbody>
</table>

Because a large and varied group of non-gastric disorders can cause similar clinical signs, a systematic approach is essential to determine if gastric disease is the cause. The diagnostic approach initially focuses on historical and physical findings, with clinicopathologic testing and diagnostic imaging employed in patients with systemic involvement or chronic signs.

Signalment, history, and physical examination
The age and breed of the patient are helpful in diagnosis of certain gastric disorders. Young dogs are more likely to ingest foreign bodies or to suffer from outflow obstruction caused by Pythium insidiosum, whereas gastric cancer is typically encountered in much older dogs and cats. Gastric dilatation and volvulus are typically encountered in giant breed or dogs with deep chests such as Great Danes and Irish Setters. There are also breed predispositions to hypertrophic gastropathy (Drentse patrijshond, basenji, and small breed brachycephalic dogs, such as shih tzu), atrophic gastritis (Lundehund) and gastric cancer (Belgian shepherd, rough Collie, Staffordshire bull terrier, beagle, Lundehund).

Vomiting is the principal clinical sign of gastric disease, and a major objective of the history is to distinguish vomiting from regurgitation (active abdominal effort, presence of bile), and to obtain a clear picture of the vomiting episodes (duration, frequency, contents, color, progression, relation to eating). Where vomiting cannot be adequately distinguished from regurgitation it is important to observe “vomiting episodes” and the animal eating. Where regurgitation is still a possibility, thoracic radiographs help to detect esophageal dilatation or obstruction.

A thorough review of the environment (indoor, outdoor, single or multi-animal household), access to foreign bodies, toxins or medications, vaccination status, body systems (attitude, mentation, presence of polyuria, polydipsia, weight loss, diarrhea, coughing, sneezing, exercise tolerance), past medical history, and physical examination helps to discriminate many non-gastric from gastric causes of vomiting.

Physical examination is frequently normal in patients with primary gastric disease. Abdominal distension may be detected in those with GD/GDV or delayed gastric emptying. Abnormal perfusion, hydration status, temperature, respiratory rate, mucosal pallor, and abdominal pain often accompany diseases such as GD/GDV, gastric outflow obstruction, ulceration, and perforation.

Historical and physical findings are integrated to determine if the patient is systemically well or unwell, and clinical signs are acute, chronic, mild, or severe. Non-productive vomiting, retching, and abdominal distension in deep-chested large breed dogs are frequently associated with gastric dilatation/gastric dilatation and volvulus (GDV), which requires rapid diagnosis and treatment.

The presence of fresh or digested blood (“coffee grounds”), in vomitus, with or without melena raises the possibility of gastric ulcers or erosions.

Vomiting of food greater than 8 to 10 hours after ingestion suggests delayed gastric emptying and requires investigation to distinguish gastric outflow obstruction from defective gastric propulsion.

Weight loss is infrequently associated with gastric disease but can accompany cancer, fungal infections, outflow obstruction, and gastropathies that are part of a more generalized disease process, such as basenji and Lundehund gastroenteropathy.

If vomiting is acute and the animal is systemically well, with no historical or physical “red flags,” further diagnostic testing is often postponed in favor of symptomatic therapy. If the animal is systemically unwell or has significant historical or physical abnormalities the emphasis is on efficiently identifying conditions that require surgical intervention, such as gastric dilatation and septic peritonitis.
and ruling out non-gastrointestinal causes of vomiting, before proceeding to more specialized or invasive diagnostic procedures aimed at detecting primary gastric and intestinal disorder.

**Gastric erosion and ulceration**

Gastric erosions and ulcers are associated with a number of primary gastric and non-gastric disorders:

**Association of gastric ulceration and erosion with specific diseases**

<table>
<thead>
<tr>
<th>Metabolic/Endocrine</th>
<th>Hypoadrenocorticism, uremia, liver disease, mastocytosis, d.i.c.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hypergastrinemia and other APUDomas</td>
</tr>
<tr>
<td>Inflammatory</td>
<td>Gastritis</td>
</tr>
<tr>
<td>Neoplastic</td>
<td>Leiomyoma, adenocarcinoma, lymphosarcoma</td>
</tr>
<tr>
<td>Drug-induced</td>
<td>Nonsteroidal and steroidal anti-inflammatories</td>
</tr>
<tr>
<td>Hypotension</td>
<td>Shock, sepsis</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>Stress, spinal surgery, exercise induced (sled dogs)</td>
</tr>
</tbody>
</table>

Clinical signs range in duration and severity, from acute to chronic and mild to life threatening. The pathomechanisms underlying gastric damage can be broadly attributed to impairment of the gastric mucosal barrier (defined above) through direct injury, interference with gastroprotective prostaglandins (PGE2), mucous or bicarbonate, decreased blood flow, and hypersecretion of gastric acid.

**Treatment**

Treatment of gastric erosions and ulcers is directed at the underlying cause, which ensures adequate hydration and perfusion, including blood transfusion if needed, and restoring electrolyte and acid base disturbances. Additional support is directed at shoring up the gastric mucosal barrier by enhancing mucosal protection and cytoprotection, and decreasing gastric acid secretion. Where vomiting is persistent, antiemetics may help to reduce fluid loss, discomfort, and the risk of esophagitis.

**Fluid therapy**

The rate of fluid administration depends on the presence or absence of shock, the degree of dehydration, and the presence of diseases (e.g., cardiac or renal), which predispose to volume overload. Patients with a history of vomiting who are mildly dehydrated are usually responsive to crystalloids (e.g., LRS or 0.9% NaCl) at a rate that will provide maintenance and replace both deficits and ongoing losses over a 24-hour period. Potassium depletion is often a consequence of prolonged vomiting or anorexia, and most polyionic replacement fluids contain only small amounts of potassium. Therefore KCl is added to parenteral fluids on the basis of serum levels.

Patients with signs of shock require more aggressive support. The volume deficit can be replaced with crystalloids at an initial rate of 60 to 90 mL/kg/h, then tailored to maintain tissue perfusion and hydration. Colloid solutions can also be used to treat animals in shock to reduce the amount of crystalloid required (e.g., Hetastarch, hemaccel at 10 to 20 mL/kg IV over 4 to 6 hours). Plasma, colloids, packed cells, or whole blood is occasionally required to treat severe hypoproteinemia or anemia, which can develop in vomiting animals with severe ulceration or HGE.

Central venous pressure monitoring and evaluation of urine output are necessary in patients with severe GI disease, particularly those complicated by third space losses of fluid into the gut or peritoneum.

The effect of vomiting on acid-base balance is hard to predict and therapeutic intervention to correct acid-base imbalances should be based on blood gas determination. Where severe metabolic acidosis is present (pH <7.1, HCO3- <10 mmol/L), sodium bicarbonate (1 mmol/kg) can be given under careful supervision for the development of worsening hypokalemia, and hypocalcemia, and CSF acidosis. Further bicarbonate supplementation is based on repeated blood gas analysis. Metabolic alkalosis usually responds to replacing volume deficit, chloride, and potassium with IV 0.9%NaCl+KCl. Diagnostic investigations should initially center on ruling out upper GI obstruction. The administration of anti-secretory drugs such as H2 antagonists may help to limit Cl--efflux into gastric juice.

Reducing acid secretion and providing mucosal protection

Pharmacologic inhibition of acid secretion can be effected by blocking H2 (cimetidine, ranitidine, famotidine), gastrin (proglumide), and acetylcholine (atropine, pirenzipine) receptors, and by inhibiting adenyl cyclase (PGE analogs) and H+/K+ ATPase (e.g., omeprazole). Long-acting somatostatin analogs such as octreotide directly decrease the secretion of gastrin and gastric acid.

Decreasing gastric acid secretion with an H2 receptor antagonist has been shown to promote mucosal healing in dogs with a variety of experimentally induced ulcers and erosions. Famotidine is an attractive choice as it does not inhibit P450 enzymes and can be given once daily. The additional prokinetic activity of ranitidine or nizatidine (mediated by anticholinesterase activity) may make them good choices in the face of delayed gastric emptying associated with defective propulsion. In patients with severe or persistent gastric ulceration that is refractory to H2 antagonists, more complete inhibition of gastric acid secretion can be achieved with a
In sled dogs with exercise-associated gastric hemorrhage treatment with omeprazole significantly reduced mean gastric severity score compared to placebo but also was associated with increased frequency of diarrhea (omeprazole 54%, placebo 21%). The authors recommended further investigation of diarrhea associated with omeprazole treatment before omeprazole can be recommended for routine prophylactic treatment in these athletes.37

The combination of omeprazole and the long acting somatostatin analog Octreotide effectively reduced vomiting in a dog with gastrinoma (Octreotide 2 to 20 µg/kg SC TID). Octreotide can also be employed to rapidly decrease gastric acid secretion in patients discovered to have large ulcers at endoscopy and has been used to control gastric bleeding in people.

**Mucosal protectants**

The PGE2 analog, misoprostol, protects against NSAID-induced erosions in dogs at doses that do not inhibit acid secretion (3 to 5 mg/kg PO TID in dogs) and may be given to dogs receiving chronic NSAIDs for arthritis. The main side effect of misoprostol is diarrhea and it should not be given to pregnant animals.

The mucosal protectant polyaluminum sucrose sulfate (sucralfate) binds to areas denuded of mucosal epithelium regardless of the underlying cause and is useful for treating gastric erosions and ulcers and esophagitis. Sucralfate can be given to patients receiving injectable antacids, but it may compromise absorption of other oral medications and is probably best separated from these by 2 hours or so.

In contrast to the efficacy of misoprostol and H2 antagonists in preventing NSAID-induced erosions, the prophylactic administration of various combinations of misoprostol, cimetidine, and omeprazole has not been shown to prevent gastric erosions in dogs with or without intervertebral disk disease receiving high-dose glucocorticoids. However, these drugs may speed healing of gastric lesions in these patients. Sucralfate is probably the drug of choice for treating GI ulceration in patients receiving high doses of corticosteroids because it is not dependent on the premise that acid is causing or delaying healing.

Mast cell tumors are also worth considering separately as gastric ulceration is a frequent and severe complication. Mast cell tumors are thought to cause vomiting via the central effects of histamine on the CRTZ and the peripheral effects of histamine on gastric acid secretion (with resultant hyperacidity and ulceration). Treatment of mastocytosis with H1 and H2 histamine antagonists (e.g., diphenhydramine and famotidine) should reduce the central and peripheral effects of histamine. Corticosteroids are used to decrease tumor burden. Where acid hypersecretion is present, or is suspected, it is likely best managed with proton pump inhibitors (e.g., omeprazole 1 mg/kg BID). Somatostatin analogs may also be useful for controlling refractory gastric acid hypersecretion (Octreotide 2 to 20 µg/kg SC TID).

**Antiemetics**

Antiemetics can be used where vomiting is severe or compromising fluid and electrolyte balance, or causing discomfort. Antiemetics commonly used in dogs include metoclopramide, which antagonizes D2-dopaminergic and 5HT3-serotonergic receptors and has cholinergic effects on smooth muscle (1 mg/kg/24h CRI IV), phenothiazine derivatives such as chlorpromazine and prochlorperazine, which are antagonists of a1 and a2-adrenergic, H1- and H2-histaminergic, and D2-dopaminergic receptors in the vomiting center and CRTZ, ondansetron (0.5mg/kg IV), which antagonizes peripheral 5HT3 receptors, and maropitant (1mg/kg IV SID, or 2mg/kg PO SID not more than 5 days) which antagonizes NK-1 receptors. Comparison of these antiemetics indicates greater efficacy of maropitant and ondansetron for controlling peripheral vomiting induced by ipecac than metoclopramide or chlorpromazine, and similar efficacy of maropitant, chlorpromazine and metoclopramide for controlling centrally mediated vomiting induced by apomorphine. Maropitant should not be used in puppies <16wks of age, as in puppies younger than 11 weeks of age, histological evidence of bone marrow hypoplasia was seen at higher frequency and greater severity in puppies treated with maropitant than in control puppies. Nonselective cholinergic receptor antagonists such as atropine, scopolamine, aminopentamide, and isopropamide are generally avoided as they may cause ileus, delayed gastric emptying, and dry mouth.

**Antibiotics and analgesia**

Prophylactic antibiotic cover (e.g., cephalosporins, ampicillin) may be warranted in animals with shock and major GI barrier dysfunction. Leukopenia, neutrophilia, fever, and bloody stools are additional indications for prophylactic antibiotics in animals with vomiting or diarrhea. Initial choices in these situations include ampicillin or cephalosporin (effective against gram-positive and some gram-negative and anaerobic bacteria), which can be combined with an aminoglycoside (effective against gram-negative aerobes) when sepsis is present and hydration status is adequate. Enrofloxacin is a suitable alternative to an aminoglycoside in skeletally mature patients at risk of nephrotoxicity from an aminoglycoside.

Analgesia can be provided using opioids like buprenorphine (0.0075 to 0.01 mg/kg IM).

Surgery may be required when the cause of ulceration is unclear or to resect large non-healing ulcers or those about to perforate.
Gastritis
Gastritis is a common finding in dogs, with 35% of dogs investigated for chronic vomiting and 26-48% of asymptomatic dogs affected. The prevalence in cats has not been determined. The diagnosis of chronic gastritis is based on the histological examination of gastric biopsies and it is usually sub-classified according to histopathological changes and aetiology.

Treatment
Treatment of gastritis initially centers on the detection and treatment of underlying metabolic disorders and the removal of drugs, toxins, foreign bodies, parasites, and fungal infections.

Parasitic gastritis
Ollulanus tricuspis is a microscopic worm (0.7 to 1 mm long, 0.04 mm wide) that infects the feline stomach. Its predominant cat-to-cat transmission is through ingestion of vomitus. It can also undergo internal autoinfection with worm burdens reaching up to 11,000 per stomach. Mucosal abnormalities range from none, to rugal hyperplasia, and nodular (2 to 3 mm) gastritis.

Histologic findings include lymphoplasmacytic infiltrates, lymphoid follicular hyperplasia, fibrosis, and up to 100/hpf globular leukocytes. Ollulanus spp. are not detected by fecal examination and require evaluation of gastric juice, vomitus, or histologic sections for larvae or worms. Gastric lavage and xylazine-induced emesis have been described to aid diagnosis. Treatment with fenbendazole 10 mg/kg PO SID 2d may be effective.

Physalloptera spp. are about 2 to 6 cm long worms that are sporadically detected in the stomachs of dogs and cats. Physalloptera rara are most commonly described and appear to be primarily a parasite of coyotes. Diagnosis is difficult as worm burden is often low and the eggs are transparent and difficult to see in sugar floatation. Treatment with pyrantel pamoate (5 mg/kg PO: dogs single dose; cats two doses 14 days apart) may be effective. Control of infection may be difficult due to the ingestion of intermediate hosts, such as cockroaches and beetles, and paratenic hosts, such as lizards and hedgehogs.

Given the difficult diagnosis of Ollulanus and Physalloptera spp., empirical therapy with an anthelminthic such as fenbendazole may be warranted in dogs and cats with unexplained gastritis.

Gastric infection with Gnathostoma spp. (cats), Spirocerca spp. (dogs), and Aonchotheca spp. (cats) has been associated with gastric nodules that have been treated by surgical resection of affected gastric tissue.

Helicobacter-associated gastritis
The general lack of knowledge of the pathogenicity of gastric Helicobacter spp. has meant that veterinarians are faced with the dilemma of either treating or ignoring spiral bacteria observed in biopsies from patients with chronic vomiting and gastritis. In light of their pathogenicity in man, ferrets, cheetahs, and mice, it would seem prudent that eradication of gastric Helicobacter spp. is attempted prior to initiating treatment with immunosuppressive agents to control gastritis. However, this must be decided on an individual basis. For example, in the patient with a lymphoplasmacytic infiltrate of the stomach and small intestine with a concomitant gastric Helicobacter spp. infection, should one treat for inflammatory bowel disease, Helicobacter, or both?

The author recommends treating only symptomatic patients that have biopsy-confirmed Helicobacter spp. infection and gastritis. Current treatment protocols are based on those found to be effective in humans infected with H. pylori. An uncontrolled treatment trial of dogs and cats with gastritis and Helicobacter spp. infection showed that clinical signs in 90% of 63 dogs and cats responded to treatment with a combination of metronidazole, amoxicillin, and famotidine, and that 74% of 19 animals re-endoscoped had no evidence of Helicobacter spp. in gastric biopsies. A recent controlled trial of amoxicillin (15mg/kg PO BID x 14d), metronidazole (10mg/kg PO BID 14d) and bismuth ± famotidine in 24 dogs found a similar decrease in vomiting (86.4%) and reduced gastritis scores in dogs that were Helicobacter negative at 6mo. The use of famotidine did not improve resolution of clinical signs or eradication of Helicobacter. Unfortunately, only 43% of dogs were free from Helicobacter at 6 months, which echoes the results of controlled studies in asymptomatic Helicobacter-infected dogs and cats. Treatment combinations evaluated in these asymptomatic animals include (1) amoxicillin (20 mg/kg PO BID 14d), metronidazole (20 mg/kg PO BID 14d), and famotidine (0.5 mg/kg PO BID 14d) in dogs; (2) clarithromycin (30 mg PO BID 4d), metronidazole (30 mg PO BID 4d), ranitidine (10 mg PO BID 4d), and bismuth (20 mg PO BID 4d) (CMRB) in H. heilmannii infected cats and (3) azithromycin (30 mg PO SID 4d), tinidazole (100 mg PO SID 4d), ranitidine (20 mg PO SID 4d) and bismuth (40 mg PO PO SID 4d)(ATRB) in H. heliominii-infected cats. Re-evaluation of infection status at 3 days (dogs) or 10 days (cats) after treatment revealed six of eight dogs and 11 of 11 CMRB and four of six ATRB-treated cats to be Helicobacter spp. free on the basis of histology and urease testing (dogs) or 13C-urea breath test (dogs and cats). However, at 28 days (dogs) or 42 days (cats) after completing antimicrobial therapy, eight of eight dogs and four of eleven cats that received CMRB, five of six cats that received ATRB were found to be re-infected. A transient effect of combination therapy (amoxicillin 20 mg/kg PO TID 21d, metronidazole 20 mg/kg PO TID 21d, and omeprazole 0.7 mg PO SID 21d) on bacterial colonization has also been observed in six cats with H. pylori infection.

Further analysis of gastric biopsies from infected dogs and H. pylori infected cats using PCR and Helicobacter-specific primers revealed persistence of Helicobacter DNA in gastric biopsies that appeared negative on histology and urease testing. These studies suggest that antibiotic regimens that are effective against H. pylori in people may only cause transient suppression, rather than eradication, of gastric Helicobacter spp. in dogs and cats.
A recent study which gave metronidazole (11-15mg/kg PO BID), amoxicillin (22mg/kg PO BID) and bismuth subsalicylate (0.22ml/kg PO Q-TID) to 5 Helicobacter infected animals (3 dogs and 2 cats) for 21d documented resolution of vomiting and long term eradication of Helicobacter (9-38months) in all animals. The author has employed the combination of amoxicillin (20 mg/kg PO BID), clarithromycin (7.5 mg/kg PO BID) and metronidazole (10 mg/kg PO BID) for 14 days to successfully eradicate Helicobacter pylori infection in cats. These studies suggest that a longer duration of treatment (21d) or the use of antibiotics that can eradicate intracellular Helicobacter (clarithromycin) improve eradication, but further studies are required before clear guidelines regarding the treatment of gastric Helicobacter spp. in dogs and cats can be made.

Chronic gastritis of unknown cause
Lymphocytic plasmacytic gastritis of unknown cause is common in dogs and cats. It may be associated with similar infiltrates in the intestines, particularly in cats (who should also be evaluated for the presence of pancreatic and biliary disease). The cellular infiltrate varies widely in severity and it may be accompanied by mucosal atrophy or fibrosis, and less commonly hyperplasia.

Patients with mild lymphoplasmacytic gastritis are initially treated with diet. The diet is usually restricted in antigens to which the patient has been previously exposed, such as a lamb-based diet if the patient has previously been fed chicken and beef, or contains hydrolyzed proteins (usually chicken or soy) that may be less allergenic than intact proteins. Many of these diets are also high in carbohydrate and restricted in fat, which facilitates gastric emptying, and may contain other substances such as menhaden fish oil or antioxidants that may alter inflammation.

The test diet is fed exclusively for a period of about 2 weeks while vomiting episodes are recorded. If vomiting is improved a challenge with the original diet is required to confirm a diagnosis of food intolerance. The introduction of a specific dietary component to the test diet, such as beef, is required to confirm dietary sensitivity. If vomiting is unresponsive the patient may be placed on a different diet for another 2 weeks, usually the limit of client tolerance, or started on prednisolone (1 to 2 mg/kg/day PO, tapered to every other day at the lowest dose that maintains remission over 8 to 12 weeks).

Patients with moderate to severe lymphoplasmacytic gastritis are usually started on a combination of a test diet and prednisolone. If the patient goes into remission they are maintained on the test diet while prednisolone is tapered and potentially discontinued. Antacids and mucosal protectants are added to the therapeutic regimen if ulcers or erosion are detected at endoscopy or if hematemesis or melena is noted.

If gastritis is unresponsive to diet, prednisolone, and antacids, additional immunosuppression may be indicated. Gastric biopsies should be carefully re-evaluated for evidence of lymphoma. In dogs immunosuppression is usually increased with azathioprine (PO 2 mg/kg SID for 5d then EOD, on alternating days with prednisolone). Chlorambucil is a safer alternative to azathioprine in cats (PO) and has been successfully employed in the management of inflammatory bowel disease and small cell lymphoma (see below).

If gastritis is unresponsive to diet, prednisolone, and antacids, additional immunosuppression may be indicated. Gastric biopsies should be carefully re-evaluated for evidence of lymphoma. In dogs immunosuppression is usually increased with azathioprine (PO 2 mg/kg SID for 5d then EOD, on alternating days with prednisolone). Chlorambucil is a safer alternative to azathioprine in cats (PO) and has been successfully employed in the management of inflammatory bowel disease and small cell lymphoma (see below).

Prokinetic agents such as metoclopramide, cisapride, and erythromycin can be used as an adjunct where delayed gastric emptying is present. These are discussed below.

Diffuse eosinophilic gastritis of undefined etiology is usually approached in a similar fashion to lymphoplasmacytic gastritis. The presence of eosinophilia, dermatologic changes, and eosinophilic infiltrates may be even more suggestive of dietary sensitivity. In cats it should be determined if it is part of a hypereosinophilic syndrome. Treatment for occult parasites, dietary trials, and immunosuppression can be carried out as described above. Focal eosinophilic granulomas can be associated with parasites or fungal infection that should be excluded prior to immunosuppression with corticosteroids.

Hypertrophic gastritis
Hypertrophy in the fundic mucosa is uncommon and is often part of the breed-specific gastropathies or gastroenteropathies mentioned above. Concurrent hypergastrinemia should prompt consideration of underlying hepatic or renal disease, achlorhydria, or gastrin-producing tumors, which should be pursued appropriately. Basenji gastroenteropathy is variably associated with fasting hypergastrinemia and exaggerated secretin stimulated gastrin, and anecdotal reports suggest that affected basenjis may respond to antimicrobial therapy. Antral hypertrophy of brachycephalic dogs causes outflow obstruction and is treated with surgery.

Delayed gastric emptying and motility disorders
Disorders of gastric motility can disrupt the storage and mixing of food and its expulsion into the duodenum. Normal gastric motility is the result of the organized interaction of smooth muscle with neural and hormonal stimuli. Delayed gastric emptying is the most commonly recognized manifestation of gastric motility disorders. Rapid gastric emptying and motility disorders associated with retrograde transit of bile or ingesta are less well defined.

Delayed gastric emptying is caused by outflow obstruction or defective propulsion and is usually suspected by the vomiting of food at least 8 and often 10 to16 hours after a meal.
Causes of delayed gastric emptying

<table>
<thead>
<tr>
<th>Outflow Obstruction</th>
<th>Defective Propulsion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital stenosis</td>
<td>Gastric disorders</td>
</tr>
<tr>
<td>Foreign bodies</td>
<td>Gastritis</td>
</tr>
<tr>
<td>Hypertrophy of pyloric mucosa</td>
<td>Ulcers</td>
</tr>
<tr>
<td>Granuloma</td>
<td>Neoplasia</td>
</tr>
<tr>
<td>Polyps</td>
<td>Gastroenteritis</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>Peritonitis</td>
</tr>
<tr>
<td>Extragastric masses</td>
<td>Pancreatitis</td>
</tr>
<tr>
<td></td>
<td>Metabolic (hypokalemia, hypocalcemia, hypoadrenocorticism)</td>
</tr>
<tr>
<td></td>
<td>Nervous inhibition (trauma, pain, stress?)</td>
</tr>
<tr>
<td></td>
<td>Dysautonomia</td>
</tr>
<tr>
<td></td>
<td>GDV</td>
</tr>
<tr>
<td></td>
<td>Surgery</td>
</tr>
<tr>
<td></td>
<td>Drugs (e.g., anticholinergics, narcotics)</td>
</tr>
<tr>
<td></td>
<td>Idiopathic</td>
</tr>
</tbody>
</table>

Diagnosis
Vomiting of food >8-10hrs after ingestion is the most common sign. Vomiting may be projectile with pyloric stenosis. Abdominal distension, weight loss, melena, abdominal discomfort, distention, bloating, and anorexia are more variably present.

Treatment
Treatment of gastric emptying disorders is directed at the underlying cause. Gastric ulcers, erosions, and inflammation should be investigated and managed medically as described above. Foreign bodies are removed either endoscopically or surgically. Pyloric stenosis, polyps, and hypertrophic gastro-pathy that is not associated with hypergastrinemia are managed surgically. When hypertrophic gastropathy, ulcers or erosions, or excessive gastric juice is encountered at endoscopy, intravenous H2-antagonists can be given during the endoscopic procedure to try to prevent postoperative perforation or esophagitis. Neoplasia, polyps, and granulomas may require extensive gastric resection and Billroth procedures.

Dietary modification to facilitate gastric emptying may be beneficial irrespective of cause.

Small amounts of semi-liquid, protein- and fat-restricted diets fed at frequent intervals may facilitate emptying, such as an “intestinal disease diet” blended with water and mixed with an equal volume of boiled rice.

In nonobstructive situations gastric emptying can be enhanced and duodenogastric reflux inhibited by prokinetic agents such as metoclopramide, cisapride, erythromycin, or ranitidine. The choice of prokinetic depends if a central antiemetic effect is required (e.g., metoclopramide), if a combined antacid prokinetic is indicated (e.g., ranitidine), or if treatment with one agent has been ineffective or caused adverse effects (e.g., behavioral changes with metoclopramide). Metoclopramide (0.2 to 0.5 mg/kg PO SC TID) has central antiemetic properties in addition to its prokinetic activity in the stomach and upper GI tract and is frequently an initial choice in patients with underlying metabolic diseases associated with vomiting and delayed gastric emptying.

However, metoclopramide may only facilitate the emptying of liquids and is less effective in promoting organized gastroduodenal and intestinal motilility than cisapride. Cisapride (0.1 to 0.5 mg/kg PO TID) has no central antiemetic effects but is generally more potent in promotion of the gastric emptying of solids than metoclopramide, but it does have more drug inter- actions and its availability is limited. Erythromycin (dog: 0.5 to 1.0 mg/kg PO TID, between meals) releases motilin and acts at motilin receptors and mimics phase III of the interdigestive migrating myoelectric complex (MMC) promoting the emptying of solids. Niaztidine and ranitidine (0.25 to 0.5 mg/lb PO TID) have prokinetic activity attributed to an organophosphate-like effect.

No controlled trials in dogs and cats have evaluated the efficacy of different prokinetics in different disease states, and treatment is usually based on a best guess/least harmful basis. Where true prokinetic activity is required, cisapride and erythromycin appear to be the most efficacious. Treatment trials with prokinetics should probably be structured to last between 5 and 10 days to determine benefit. A diary of clinical signs and the objective assessment of gastric emptying using the tests described above, before and after therapy helps to optimize treatment. Combination therapy, such as erythromycin and cisapride, is not recommended due to the potential for adverse drug interactions. The prognosis for patients with delayed gastric emptying depends on the cause.

A suspected motility disorder characterized by duodenogastric reflux is thought to account for a syndrome known as the bilious vomiting syndrome. Affected dogs usually vomit early in the morning. Remission may be achieved by feeding the animal late at night. Prokinetic agents may also be employed.
Further reading
Simpson KW Textbook of Veterinary Internal Medicine , Ettinger and Feldman 7th. Chapter 269 – Diseases of the Stomach.