Acute gastroenteritis is a term used to describe a syndrome characterized by the sudden onset of vomiting and/or diarrhea caused by gastrointestinal mucosal inflammation.

This diagnosis is seldom confirmed by histopathologic evaluation; instead, it is based on a consistent clinical presentation and exclusion of other potential causes for the patient’s clinical signs. Mucosal inflammation is assumed, but not proven to be present. Therefore, acute gastroenteropathy is perhaps a more appropriate name.

**DIAGNOSTIC EVALUATION**

Acute gastroenteritis is among many potential causes of acute vomiting and diarrhea (Table 1). However, in many cases, the cause of primary acute gastroenteritis is not determined. Rapid resolution of clinical signs often means that extensive diagnostic evaluation is unnecessary.

**Physical Examination**

No specific physical examination findings are pathognomonic for acute gastroenteritis, and some dogs do not have any significant abnormalities. Findings consistent with acute gastroenteritis include lethargy, pyralism, and abdominal discomfort.

It is particularly important to assess the patient’s hydration status and palpate the abdomen carefully, checking for physical examination findings that would warrant further diagnostic evaluation (ie, abnormalities that suggest the problem is more significant than straightforward acute gastroenteritis) (Table 2). Findings that indicate dehydration include dry oral mucous membranes, prolonged capillary refill time, and prolonged skin tent. Tachycardia, weak pulses, and cool extremities are consistent with hypovolemia.

**Laboratory Analysis**

Patients with a normal physical examination and
mild clinical signs may not require laboratory testing on initial presentation. However, laboratory testing may be indicated to rule out extra-gastrointestinal causes of acute gastrointestinal signs, such as acute kidney injury, acute hepatitis, and pancreatitis, and metabolic complications of acute gastroenteritis, such as electrolyte and acid base abnormalities.

When performed, laboratory testing should include a complete blood count, serum biochemical profile, and urinalysis. Measurement of serum canine pancreas-specific lipase concentration may also be indicated to diagnose pancreatitis, and baseline serum cortisol concentration may be measured in order to exclude hypoadrenocorticism.

Additional laboratory testing for infectious disease should be considered based on geographic location and signalment. For example, serology assists in diagnosis of Salmon poisoning disease in the Pacific Northwest. In dogs with diarrhea, fecal flotation and direct smear examination should be performed to screen for primary or concurrent parasitism (Figure 1).

In patients with clinical findings (Table 2) or laboratory results that suggest a serious underlying cause, or those that do not respond to therapy, further diagnostic evaluation is indicated. Early identification is especially important in patients requiring surgical intervention, such as those with an obstructive intestinal foreign body (Figure 2).

Imaging
Abdominal ultrasonography and/or abdominal radiography are strongly advised in patients presenting with abdominal pain to screen for diseases requiring surgical intervention. It is important to remember that pancreas-specific lipase concentrations can be increased in dogs and cats with gastrointestinal foreign bodies. Therefore, it is essential to rule out gastrointestinal foreign bodies with abdominal radiographs and, possibly, abdominal ultrasound before pancreatitis is diagnosed. If there is high suspicion for a gastrointestinal foreign body that may have been obscured by fluid or gas, diagnostic imaging should be repeated.

**THERAPEUTIC APPROACH**
When acute gastroenteritis is the primary cause of vomiting and/or diarrhea, the symptomatic treatments discussed in this article are appropriate for therapy. However, if gastroenteritis occurs

![FIGURE 1. Gastric nematode presumed to be Physaloptera rara visualized during gastroscopy. The hemorrhage observed is associated with gastric biopsy.](image1)

![FIGURE 2. Fabric gastric foreign body visualized during gastroscopy.](image2)
secondary to an underlying disease, such as hypoadrenocorticism, it is essential to treat the primary condition in addition to providing symptomatic and supportive therapy.

This article emphasizes symptomatic treatment of primary acute gastroenteritis rather than detailing specific treatment of serious underlying diseases that may cause similar clinical signs.

**ANTIEMETIC DRUGS**

For acute gastroenteritis, antiemetic therapy is often used for the initial 24 to 48 hours when vomiting is a prominent clinical sign (Table 3). Benefits include:

- Improved patient comfort
- Decreased ongoing fluid and electrolyte losses
- Earlier reintroduction of enteral nutrition
- Reduced risk of esophagitis and esophageal stricture formation.

Take care not to mask ongoing disease with prolonged (ie, greater than 3 days) antiemetic therapy. In addition, to reduce the risk of gastrointestinal perforation by masking clinical signs of intestinal obstruction, do not administer antiemetic or prokinetic drug therapy when a foreign body is suspected or confirmed.

Several classes of antiemetic drugs are used in small animal medicine. Occasionally, refractory cases require the use of more than one of these drugs at the same time.

**Table 3. Medical Therapy for Vomiting Due to Acute Gastroenteritis**

<table>
<thead>
<tr>
<th>DRUG</th>
<th>DOGS</th>
<th>CATS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Antiemetics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ondansetron</td>
<td>0.1–1 mg/kg PO Q 12–24 H</td>
<td>0.1–1 mg/kg PO or IV Q 12–24 H</td>
</tr>
<tr>
<td>Dolasetron</td>
<td>0.5–1 mg/kg IV Q 12 H</td>
<td>0.6 mg/kg IV Q 12 H</td>
</tr>
<tr>
<td>Maropitant</td>
<td>1 mg/kg SC Q 24 H</td>
<td>1 mg/kg SC Q 24 H</td>
</tr>
<tr>
<td></td>
<td>2 mg/kg PO Q 24 H</td>
<td>2 mg/kg PO Q 24 H</td>
</tr>
<tr>
<td></td>
<td>1 mg/kg IV Q 24 H</td>
<td>1 mg/kg IV Q 24 H</td>
</tr>
<tr>
<td>Metoclopramide</td>
<td>0.2 mg/kg SC or PO Q 8 H</td>
<td>0.2–0.4 mg/kg PO or SC Q 6–8 H</td>
</tr>
<tr>
<td></td>
<td>1–2 mg/kg/H IV CRI</td>
<td>1–2 mg/kg/H IV CRI</td>
</tr>
<tr>
<td><strong>Gastroprotectants</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sucralfate</td>
<td>0.5–1 g PO Q 8–12 H (tablet or slurry)a</td>
<td>0.5 g PO Q 8–12 H (tablet or slurry)b</td>
</tr>
<tr>
<td>Famotidine</td>
<td>1 mg/kg PO or IV Q 12 H</td>
<td>1 mg/kg PO or IV Q 12 H</td>
</tr>
<tr>
<td>Omeprazole</td>
<td>1 mg/kg PO Q 12 H</td>
<td>1 mg/kg PO Q 12 H</td>
</tr>
<tr>
<td>Pantoprazole</td>
<td>1 mg/kg IV Q 24 H</td>
<td>1 mg/kg IV Q 24 H</td>
</tr>
<tr>
<td>Misoprostol</td>
<td>2–5 mcg/kg PO Q 8–12 H</td>
<td>Not applicable</td>
</tr>
</tbody>
</table>

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**Ondansetron & Dolasetron**

Ondansetron and dolasetron are serotonin (5-HT₃) antagonists with potent antiemetic activity that are commonly used off-label to control nausea in dogs and cats. This class of drug blocks the chemoreceptor trigger zone and vagal afferent pathways involved in emesis. In our experience, these drugs are very effective for control of vomiting in dogs and cats.

**Maropitant**

Substance P is a neurotransmitter that binds to neurokinin-1 (NK-1) receptors and can result in vomiting. Therefore, NK-1 receptor antagonists are powerful antiemetics effective at treating both peripheral and central causes of vomiting.

Maropitant, a NK-1 receptor antagonist, is currently the only licensed antiemetic for use in dogs and cats, and, in our opinion, is very effective. This drug may also have an analgesic effect and, thus, is widely used in patients with vomiting and abdominal pain, such as those with pancreatitis. The efficacy of maropitant for the control of presumed nausea is controversial as some studies have shown a benefit while others have not documented a benefit.

While maropitant is not licensed for IV use, we and other clinicians have administered it by this route—at a dose of 1 mg/kg Q 24 H—without apparent adverse effects. The manufacturer recommends that after 5 days of continuous therapy.
administration the drug should be discontinued for 24 hours to avoid drug accumulation; then treatment can be restarted, if necessary.

**Metoclopramide**
Antidopaminergic agents, such as metoclopramide, have both central and peripheral antiemetic effects and are used off-label in dogs and cats. Metoclopramide also stimulates the release of acetylcholine from postganglionic nerves in the peripheral nervous system, which leads to increased gastric contractions and increased gastroesophageal sphincter tone. This promotility effect may also contribute to its antiemetic properties.

Metoclopramide can cause neurologic side effects, including excitement and restlessness. The short half-life of this drug necessitates frequent dosing. In our experience, it is most effective when delivered via an IV constant rate infusion but is not as effective as maropitant, ondansetron, or dolasetron. Metoclopramide may not have a direct central antiemetic effect and is a less effective antiemetic agent in cats than in dogs. For this reason, we seldom use it for this purpose in the former unless a prokinetic agent is also indicated.

**Phenothiazines**
Phenothiazines, such as chlorpromazine and prochlorperazine, are potent centrally acting antidopaminergics that inhibit the vomiting center and chemoreceptor trigger zone. This class of drug also has antihistaminergic and anticholinergic properties. They are not recommended in hypovolemic patients due to potential for hypotension, and these drugs are not licensed for use in dogs and cats. Phenothiazines can cause mild sedation and are no longer commonly used due to the availability of other effective antiemetic drugs.

**GASTROPROTECTANTS**
**Sucralfate**
Sucralfate is a sulfated disaccharide that binds to the acidic moieties of exposed collagen in damaged mucosa, creating a protective barrier against further acid damage. It also stimulates prostaglandin release and cellular proliferation at sites of ulceration and increases mucus production and bicarbonate secretion. Sucralfate is recommended only in cases of suspected gastrointestinal erosion or ulceration, such as those that present with hematemesis or melena. It can interfere with absorption of other drugs, and is typically given at least 2 hours before or after other medications.

**Famotidine & Ranitidine**
Famotidine and ranitidine are histamine-2 (H₂)-receptor antagonists that competitively inhibit histamine-induced acid secretion by the gastric parietal cells.

Famotidine is more effective at increasing canine gastric pH compared with ranitidine, but ranitidine also has anticholinesterase activity. This activity may result in some prokinetic action but in studies was only as effective as a saline placebo at increasing gastric pH in dogs and cats; thus, it is not recommended for its acid-suppressing effects. 

H₂-receptor antagonists are less efficacious than proton pump inhibitors (PPIs) in vivo, but the degree of gastric acid inhibition necessary for therapeutic effect is unknown. H₂-receptor antagonists may also have cytoprotective properties. 

**Omeprazole & Pantoprazole**
Omeprazole and pantoprazole are PPIs that irreversibly inhibit acid production by gastric parietal cells. This class of drug is more efficacious and has a longer duration of activity than H₂-receptor antagonists; it may also exert a cytoprotective effect by enhancing prostaglandin synthesis.

In dogs and cats, twice-daily dosing of omeprazole is more effective at reducing gastric acid secretion than once-daily administration. Coadministration of famotidine and pantoprazole does not seem to be any more effective than therapy with pantoprazole alone and, thus, there is no benefit in using a H₂-receptor antagonist for the first 24 hours of therapy. Because of this, PPIs are the preferred treatment for dogs and cats known or suspected to have esophageal or gastroduodenal ulceration. 

Anecdotally, some dogs and cats with acute gastroenteritis, but no other findings that indicate gastroduodenal ulceration, such as hematemesis or melena, appear to respond favorably to acid-suppressing drugs. However, use of these drugs has not proven beneficial in any studies in dogs and cats with uncomplicated gastroenteritis; thus, they are not routinely recommended for brief episodes.

In humans, long-term use of PPIs has been associated with such side effects as cobalamin deficiency, iron deficiency, hypomagnesemia, increased susceptibility to pneumonia, enteric infections, fractures, hypergastrinemia, and cancer. To our knowledge, other than hypergastrinemia, the side effects described above have not been reported in dogs and cats receiving long-term treatment with PPIs.
**Misoprostol**

Misoprostol is a synthetic prostaglandin E1 that acts on parietal cells to inhibit secretion of gastric acid. Additionally, it has a cytoprotective effect by increasing secretion of mucus by gastric goblet cells, increasing gastric mucosal blood flow and increasing turnover of gastric mucosal cells.

PPIs and H2-receptor antagonists are thought to be more effective for treatment of gastrointestinal ulcers, and misoprostol may cause vomiting, diarrhea, and abdominal pain. Use of misoprostol is, therefore, not advised in dogs with acute gastroenteritis unless gastroduodenal ulceration associated with nonsteroidal anti-inflammatory drug (NSAID) use is thought to be the cause.

**Antidiarrheal Therapy**

Most cases of uncomplicated acute gastroenteritis that present with either small or large bowel diarrhea resolve without therapeutic intervention. Cases that present with diarrhea should have a fecal examination and consider empirical deworming with a broad spectrum anthelmintic. However, there are a few options for symptomatic treatment of diarrhea.

**Loperamide**

Loperamide, an opioid antimotility drug, has been used off-label in dogs with diarrhea. It decreases intestinal motility and reduces mucosal secretions. Doses used to treat diarrhea can cause neurologic toxicity in dogs with the \( ABCB1 \) (formerly \( MDR1 \)) mutation; therefore, avoid this drug in all dogs carrying this allele and at-risk dog breeds (ie, Australian shepherd, Shetland sheepdog, long-haired whippet, collie, English shepherd, German shepherd) that have an unknown status. We do not recommend use of this drug for treating dogs or cats with acute gastroenteritis due to this potential toxicity and because the diarrhea associated with gastroenteritis is usually self-limiting.

**Probiotics**

Probiotics are live microorganisms that confer a health benefit on the host.\(^6\) These health effects are exerted by direct inhibition of colonization by pathogenic microorganisms, or by immune-enhancing effects on gut-associated lymphoid tissue.\(^7\)-\(^9\)

Probiotics (Table 4) are sometimes used to treat dogs and cats with acute diarrhea. Each probiotic has a different formulation of bacteria, and it is unknown which, if any, are most useful for treatment of acute gastroenteritis with resultant diarrhea. Therefore, further study of these products is needed before definitive recommendations can be made.

The efficacy of some probiotics for treatment of chronic diarrhea in dogs and cats has been evaluated but, to our knowledge, there have only been 2 studies evaluating the efficacy of probiotics in dogs with acute diarrhea; both found that probiotics decreased the duration of diarrhea in dogs with acute idiopathic diarrhea.\(^20\)-\(^23\)

When selecting a probiotic, it is important to choose a product that has been subjected to adequate quality control during the manufacturing process, such as the ones listed in Table 4.

**Antimicrobial Therapy**

Antimicrobial therapy with metronidazole or tylosin is sometimes used empirically in dogs and cats with idiopathic acute gastroenteritis that present with either small or large bowel diarrhea. Both antibiotics are used to potentially treat specific bacteria that may cause acute gastroenteritis (eg, \( Clostridium perfringens \)).

However, a study evaluating the efficacy of amoxicillin/clavulanic acid in dogs with acute hemorrhagic diarrhea syndrome (formerly called hemorrhagic gastroenteritis) demonstrated no benefit in treated dogs versus control dogs.\(^24\) Therefore, routine antibiotic therapy (including the use of metronidazole) is not recommended in dogs or cats with acute gastroenteritis.

Antibiotic therapy may have a role in dogs and cats suspected to have bacterial translocation through a damaged gastrointestinal mucosal barrier, and this is potentially more likely in cases of gastrointestinal bleeding. However, we reserve antimicrobial therapy for patients with:

- More definitive evidence of translocation, such as leukocytosis, elevated immature white blood cell count, and pyrexia
- Leukopenia or those that are immunosuppressed
- A specific bacterial enteropathogen (eg, campylobacteriosis)
- Chronic diarrhea (as a therapeutic trial to rule out dysbiosis).

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**TABLE 4.**

**Examples of Commercial Probiotics for Dogs & Cats**

- FortiFlora (purina.com)
- Prostora (iams.com)
- Proviable (nutramaxlabs.com)
- Sivoy (sivoy.net)
NUTRITIONAL MANAGEMENT

Fasting
The concept of complete fasting has been questioned in recent years for dogs with acute gastroenteritis. Complete restriction of food may be reasonable for a short period, but an early return to appropriate oral intake is advised unless contraindicated, such as in cases suspected to have foreign bodies.

Diet Considerations
A wide variety of commercially available diets is marketed for dogs and cats with gastroenteritis. Each is formulated with slightly different protein and carbohydrate sources and fat content; some contain other potentially beneficial constituents, such as fructooligosaccharides or omega-3 fatty acids.

Cats should receive enteral nutrition as soon as possible to avoid protein calorie malnutrition, which can lead to feline hepatic lipidosis. A commercially available, highly digestible diet is recommended. The goal of feeding a highly digestible diet is to reduce the risk for malabsorption.

Dietary Fiber
For patients with large bowel diarrhea, dietary fiber is an important component of dietary management. While the optimal amount and type of dietary fiber for treatment of dogs and cats with acute gastroenteritis are not known, there is general agreement that, in dogs and cats:

- Dietary fermentable fiber enhances normal colonic function by providing a fuel source for colonocytes
- Dietary nonfermentable fiber increases fecal bulk, which promotes normalized colonic motor function and defecation.

Potential sources include canned pumpkin, brown rice, peas, and carrots. There are no established guidelines for fiber supplementation in cases of small bowel diarrhea.

In one study, dogs with colitis that received added dietary fiber experienced significant clinical improvement compared with dogs fed diets without added fiber.

FLUID THERAPY
Acute gastroenteritis can lead to fluid losses and electrolyte imbalances that necessitate fluid therapy.

Subcutaneous Therapy
SC fluid therapy may be appropriate for mild dehydration or when outpatient therapy is elected due to financial concerns; however, it would be considered inappropriate for significant dehydration or hypovolemia. SC fluids should not be supplemented with dextrose because bacterial contamination and cellulitis can develop. Where supplementation with dextrose is needed IV administration is preferable.

Oral Therapy
Oral electrolyte solutions can be used in cases of mild dehydration. In a recent study, this route of administration was safe and effective in dogs with hemorrhagic diarrhea.

Intravenous Therapy
Goal-directed, targeted IV fluid therapy to correct an estimated percent dehydration over a specific time frame and to achieve normovolemia is recommended for moderate to severe dehydration or hypovolemia. Balanced replacement crystalloid solutions, such as lactated Ringer’s solution, are an appropriate choice in most patients. Ongoing monitoring of serum electrolyte concentrations is recommended because supplementation is sometimes required.

H₂ = histamine-2; NK-1 = neurokinin-1; NSAID = nonsteroidal anti-inflammatory drug; PPI = proton pump inhibitor

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References