# Medical and Nutritional Management of Acute Gastroenteritis

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Vomiting (and diarrhea) can be caused by a wide variety of GI, intra-abdominal, metabolic, systemic and neurologic diseases. An efficient clinical approach to is to first decide whether the cause of the vomiting is likely due to a primary GI problem or to a metabolic problem secondarily causing GI signs (Primary GI vs. Secondary GI disease). The latter will require more extensive evaluation and will not be covered in this discussion.

**Dietary management of acute vomiting and diarrhea**

Dogs or cats presenting with recent-onset vomiting are commonly held NPO (*nothing per os*) for about 12 hours until the vomiting ceases. The need for such a period of NPO has not been shown in an evidence-based manner and can often be considerably shortened with the appropriate use of an antiemetic. Pets with diarrhea often benefit from withholding food for 6-12 hours. After vomiting has ceased for 2-3 hours or diarrhea frequency is reduced, a small volume of a highly digestible (intestinal formula) food should be fed. A diet of modest fat content is often selected in dogs, but dietary fat content appears to play a lesser role in gastric emptying in cats. Commercial diets or homemade diets based on boiled rice with lean chicken or low fat cottage cheese are useful choices. Feeding small meals frequently (3-6 times per day initially) will minimize gastric distention, gastric acid secretion and provide high digestibility to improve diarrhea. A gradual transition to the pet's usual diet is made over 2-3 days providing that signs have resolved.

For pets with signs of acute colitis (large bowel diarrhea), a high fiber diet is often used instead of a highly digestible diet as fiber may be beneficial in reducing tenesmus and facilitating colonic epithelial repair. Commercial products with increased mixed (soluble and insoluble) fiber are ideal. Soluble fiber (psyllium mucilloid 1 tsp/10 kg body weight) may be added to a GI diet.

**Fluid therapy**

Dehydration is a sign that pets with acute gastroenteritis require further diagnostic evaluation in addition to supportive therapy. Milder cases may benefit from subcutaneous fluids administration to ensure adequate hydration is maintained, especially during a short period NPO. Isotonic fluids should be used and no more than 10 to 20 ml/kg should be given at each injection site. The rate of subcutaneous fluid flow usually is governed by patient comfort. Acetated polyionic solutions such as Normosol-R and Plasmalyte should not be given SQ due to the discomfort associated with administration. Generally, all subcutaneous fluids are resorbed within 6 to 8 hours. If fluids are still noted subcutaneously after this time, the use of intravenous fluids to reestablish peripheral perfusion should be considered. Fluids should be administered intravenously to animals that are assessed to be $\geq$ 7% dehydrated**.**

**Antiemetic therapy**

Antiemetic therapy is warranted: (1) when the vomiting is frequent or severe enough to make the animal feel uncomfortable; (2) persistent vomiting places the animal at risk for aspiration pneumonia or acid-base and electrolyte disturbances; and (3) the animal is not suffering from gastrointestinal obstruction or toxicity. There are four main pathways that stimulate the emetic center and provides a useful framework in which to place the many causes of vomiting and think about antiemetic therapy. An important concept is that vomiting occurs either through activation of the chemoreceptor triggering zone (CRTZ) by blood-borne substances (humoral pathway), or through activation of the emetic center by vago-sympathetic, CRTZ, vestibular, or cerebrocortical neurons (neural pathway). Many of the spontaneous vomiting disorders,particularly those due to primary GI disease, are believed to result from activation of the neural pathway. Visceral afferent input to the vomiting center arises from receptors located throughout the body; most are distributed in the abdominal viscera, with the largest number in the duodenum. These visceral receptors are sensitive to chemical irritation, inflammation, distention and changes in osmolality. Several neurotransmitters and their respective receptors stimulate the CRTZ; these form the basis for classification of antiemetics. Antiemetics control emesis by either a central or peripheral blockade of neurotransmission at receptor sites (Table). In the emetic center, NK-1 and $\alpha_2$-adrenergic receptors are the most clinically important. In the CRTZ in dogs, dopamine and histamine are significant neurotransmitters, making dopaminergic and histaminergic receptor antagonists important antiemetic classes. In contrast, metoclopramide (a $D_2$-dopaminergic antagonist) is of questionable efficacy for central control of vomiting in cats. The most effective antiemetics for cats appear to be those that work via $NK_1$ (eg, maropitant) or serotonin ($5HT_3$) (eg, ondansetron) receptors.

**Useful antiemetic drugs for dogs and cats**

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| NK-1 Receptor Antagonists | |
| *Maropitant (Cerenia®) | 1 mg/kg sid SQ or IV (dogs and cats);<br>administer the SQ injection cold to reduce injection<br>pain. 2 mg/kg PO (dogs) and 1 mg/kg PO (cats)<br>and 8 mg/kg PO for motion sickness (dogs) |
| 5-$HT_3$-Serotonergic Antagonists | |
| Ondansetron (Zofran®) | 0.5-1 mg/kg bid PO, or 30 min prior to<br>chemotherapy, or 0.1-0.5 mg/kg bid IV |
| Dolasetron (Anzemet®) | 0.5-1 mg/kg bid PO, or 30 min prior to<br>chemotherapy, or 0.1-0.5 mg/kg bid IV |
| Metoclopramide (Reglan®) | 0.2-1 mg/kg qid PO, SQ, IM;<br>CRI @ 2-4 mg/kg/day. Use at 1 mg/kg<br>for chemotherapy-induced nausea in dogs |
| α2-Adrenergic Antagonists | |
| Prochlorperazine (Compazine®) | 0.5 mg/kg tid, SQ, IM, Rectal Suppository |
| Chlorpromazine (Thorazine®) | 0.2-0.4 mg/kg tid SQ, IM |

* Maropitant also provides visceral analgesia in dogs and cats. This analgesic property of maropitant makes it especially suitable for use to treat vomiting caused by painful intra-abdominal conditions such as pancreatitis.

The classic contraindication to prokinetic agents, including metoclopramide, is GI obstruction, although many experienced clinicians report that serious adverse effects have not been seen when these agents are inadvertently given to patients with GI obstruction with the exception of animals with linear foreign bodies. Drugs such as chlorpromazine and prochlorperazine can cause systemic hypotension and should only be given if the patient is not hypotensive or if there is IV fluid support. These drugs can cause unwanted sedation and were thought to reduce the seizure threshold but clinical experience suggests they can be used in patients with seizure disorder histories. Behavioral changes may be seen with metoclopramide, especially dose-related excitation.

**Gastroprotectants**

Peripheral pathways are mediated through irritation and inflammation of the gastrointestinal mucosa. Therefore another approach to therapy that is commonly used is administration of gastroprotective agents such as drugs that inhibit gastric acid production ($H_2$-histaminergic receptor antagonists and proton pump inhibitors such as omeprazole and pantoprazole) and drugs that act locally on the gastric mucosa (such as sucralfate).

Histamine $H_2$-receptor antagonists are the most commonly used drugs to manage gastric ulceration or severe gastritis. These agents competitively block $H_2$ receptors on gastric parietal cells, reducing gastric acid secretion. The $H_2$ receptor antagonists currently in wide use are ranitidine and famotidine. Cimetidine is the least potent of the H2-receptor antagonists and inhibits the cytochrome P-450 enzyme system so is no longer used. In addition to its effect on $H_2$ receptors, ranitidine stimulates gastric emptying in the cat and dog.

Proton pump inhibitors (PPIs) are currently the most potent inhibitors of gastric acid secretion available. They irreversibly block the gastric proton pump (hydrogen-potassium ATPase), causing a marked decrease in gastric acid secretion. They are recommended for use in small animals diagnosed with severe reflux esophagitis or gastric ulceration**.** Omeprazole (eg, Prilosec) is now available OTC, markedly reducing its cost and increasing its availability and usage in small animals. It is dosed at 0.7 mg/kg *sid* PO in dogs and cats. Pantoprazole (eg, Protonix) is a newer PPI available for oral or IV use. It is dosed at 0.7-1 mg/kg *sid* PO or IV.

Sucralfate (Carafate) selectively binds to proteins at sites of ulceration forming a protective barrier. In addition, it increases the luminal concentration of $PGE_2$, which is also protective against ulcerogenic factors. Sucralfate is dosed at 0.25–1.0 gram q 8–12 h PO in the dog and cat.

**Management of acute diarrhea**

Dogs and cats frequently develop diarrhea that starts abruptly and lasts for less than 7 days. Regardless of cause, most cases are likely associated with changes to the intestinal microbiota. Most cases are mild and self-limiting, but patient comfort and owner convenience may be aided by prescribing non-specific therapies. Nutritional management, therapeutic deworming, and probiotic therapy, sometimes in conjunction with an anti-diarrheal agent, should be considered in mild cases in which there is no indication for antimicrobial therapy. In cases with more severe signs, a thorough diagnostic plan must be followed to obtain an accurate diagnosis and guide appropriate therapy.

A variety of bacteria are known or suspected of being capable of causing enteritis in dogs and cats (Weese 2011). Limitations in our understanding of the incredibly complex intestinal microflora and inadequate investigation of many potential pathogens and diagnostic tests create significant challenges in establishing a diagnosis of bacterial enteritis in an individual pet. Fecal cytology is now considered to have **no** diagnostic utility as these bacteria can be present in normal animals and appearance does not differentiate between pathogenic strains and harmless commensal species. Partial analysis for enteric pathogens by fecal enteric panel (Gram-

stained fecal smear, *C. perfringens* enterotoxin ELISA, *C. difficile* toxin A/B ELISA) and fecal culture or PCR for potential bacterial pathogens (such as *Campylobacter*, *Salmonella* and *Campylobacter*) should usually be reserved for dogs and cats that have diarrhea and are systemically ill (fever, severe lethargy, leukocytosis, etc.) or for diarrheic pets in contact with an immunosuppressed person. It is very difficult to establish evidence for an association between the presence of a specific bacterium in the feces and the occurrence of diarrhea. There is mounting evidence that changes in microbial populations (dysbiosis) play an important role in the pathogenesis of acute and chronic diarrhea. (Suchodolski 2012) The observed changes in the microbiome differ between acute and chronic disease states. For example, numbers of *C. perfringens* organisms increased in acute diarrhea.

**Symptomatic treatment of acute diarrhea**

**Broad-spectrum parasiticide**

Fenbendazole, 50 mg/kg PO q24h for 3-5 days

**Antidiarrheals**

- Loperamide (Imodium$^{R}$) Dogs: 0.08-0.2 mg/kg PO q6-12h; Cats: 0.04 mg/kg PO q12-24 h; Use with caution. Avoid use in in dogs with the MDR1 mutation (primarily herding breeds).
- Diphenoxylate (Lomotil$^{lR}$) Dogs: 0.05-0.2 mg/kg PO q6-8h; Cats: 0.08-0.1 mg/kg PO q12h

**Antimicrobials for non-specific diarrhea**

- Tylosin 10-15 mg/kg PO q12-24h
- Metronidazole Dog: 10 mg/kg PO q12h; Cat 62.5 mg PO q12h

**Antimicrobials for dogs or cats at risk for bacterial translocation**

- Amoxicillin-clavulinic acid 12.5-22 mg/kg PO q12h
- Ampicillin 22 mg/kg PO q8-12h

**Probiotics (examples)**

- VSL#3$^{TM}$
- Fortiflora$^{TM}$
- Prostora$^{TM}$

**Antidiarrheals**

If diarrhea is frequent enough to interfere with the animal's (or sometimes the owner's) ability to rest, is causing apparent pain or discomfort, or occasionally when diarrhea causes large fluid losses, an opioid may be given to alter intestinal motility. These drugs are particularly effective in cases showing signs of large bowel diarrhea (colitis). Opioids prolong intestinal transit time, allowing increased fluid absorption and reduction in the frequency of diarrhea. Opioids act by increasing colonic segmentation, decreasing propulsive peristaltic contractions, increasing fluid absorption, decreasing secretion and increasing anal tone. Loperamide or diphenoxylate are very effective in reducing the frequency of diarrhea. Both are available as elixirs making dosing convenient for small dogs and cats. Loperamide has been shown to be more potent and have a faster onset and a longer duration of action than diphenoxylate. At recommended doses, both drugs are safe and have few side effects in dogs, with one major exception. At doses used to treat diarrhea, loperamide will cause neurological toxicity in dogs with the MDR1 mutation (Collies and other herding dogs. If a toxin, or possibly pathogenic bacteria are suspected as being the cause of the acute diarrhea, opioids are contraindicated as they may increase toxin absorption or allow more time for bacterial proliferation.

**Antimicrobial therapy**

The routine use of antibiotics for acute uncomplicated diarrhea is strongly discouraged. Antibiotics are very rarely indicated in vomiting patients. If an antibiotic is selected for non-specific use in diarrhea cases, metronidazole or tylosin is an appropriate choice. Markedly hemorrhagic diarrhea is often interpreted as evidence of a breach of intestinal integrity, justifying the use of antimicrobials, although no studies document this risk. Because ruling out an enteric bacterial infection is challenging and bacterial translocation is a potentially life-threatening complication, pets with acute bloody diarrhea of unknown cause are usually either treated with an antibiotic such as amoxicillin/clavulinic acid or monitored very closely if antibiotics are not administered.

**Selected readings**

Weese JS. Bacterial enteritis in dogs and cats: diagnosis, therapy, and zoonotic potential. Vet Clin Small Anim 2011; 41:287-309.
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