

# GI INTERVENTION

## Approach to Diagnosis and Therapy of the VOMITING PATIENT

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Vomiting is a very common clinical complaint in both dogs and cats. It is also a clinical sign seen in diseases of many body systems. Clinicians must avoid assuming vomiting is synonymous with gastrointestinal (GI) disease.

In a large national survey, GI signs accounted for 4% of visits at primary care clinics.<sup>1</sup>

### DEFINITION

Vomiting is the active expulsion of gastric, sometimes duodenal, contents and is typically preceded by apparent nausea and retching. However, it can often be confused with:

- **Regurgitation** associated with esophageal disorders
- **Gagging/coughing** associated with respiratory disease
- **Oropharyngeal dysphagia.**

### CLINICAL SIGNS

Key elements of vomiting are:

- Forceful abdominal contractions (one of the most reliable ways to confirm vomiting)
- Retching and presence of bile.

A thorough history will usually confirm whether the pet is vomiting. If doubt remains, attempt to visualize the behavior by asking the owner to video it or provocatively feeding the patient in the hospital.

Be cautious in overinterpreting the timing of the event in relation to consumption of meals. In some cases, regurgitating animals can passively expel esophageal or gastric contents hours after ingestion of a meal.

### PATHOPHYSIOLOGY

Vomiting is a complex, protective reflex that occurs in carnivores but is not well developed in all species. Although several afferent pathways may be responsible for initiating emesis, it is coordinated by the emetic (or vomiting) center in the medulla (**Table**).

An important concept of vomiting is that it occurs through activation of the:

- Chemoreceptor trigger zone (CRTZ) by blood-borne substances (humoral pathway)
- Emetic center by vagosympathetic, vestibular, or cerebrocortical neurons (neural pathway).

Many spontaneous vomiting disorders of cats and dogs, particularly those due to primary GI disease, are believed to result from activation of the neural pathway.

- Visceral afferent input to the emetic center arises from receptors located throughout the body.
- Most are distributed in the abdominal viscera, with the largest number in the duodenum.
- Visceral receptors are sensitive to chemical irritation, inflammation, distention, and changes in osmolality.

Several neurotransmitters and their respective receptors stimulate the emetic center; these form the basis for antiemetic classification.

### CHRONIC VOMITING

Chronic vomiting is commonly defined as persistent vomiting of variable frequency and, typically, duration of 3 weeks or longer.

In cases of chronic vomiting, relatively extensive diagnostic evaluation is almost always warranted in order to determine a cause rather than solely providing supportive and symptomatic care. See **Determining Reasons for Vomiting & Appropriate Diagnostics**, page 21, for further information. The remainder of this overview will focus on acute vomiting.

### ACUTE VOMITING: DIAGNOSTICS

Acute vomiting is commonly defined as vomiting of variable frequency over a period of less than 7 days, although, in practical terms, acute vomiting is usually of 1 to 3 days' duration since owners will commonly seek medical attention within this interval.

From the signalment, history, and physical examination, the clinician should be able to:

1. Categorize the patient as:
  - Stable, with no criteria for further immediate assessment or treatment
  - Unstable, with 1 or more criteria for intervention.
2. Establish a differential list.
3. Identify appropriate diagnostic interventions and therapy.

Table. Four Main Stimulating Pathways of the Vomiting Center

#### 1. Peripheral Sensory Receptors

- Intra-abdominal
  - » Stomach, intestines, pancreas, liver, gallbladder, peritoneum, kidneys, ureter, urinary bladder
  - » Visceral afferent fibres in sympathetic and vagal nerves
- Heart and large vessels via vagus nerve
- Pharynx via glossopharyngeal nerve

#### 2. Stimulation of the Chemoreceptor Trigger Zone

- Uremia
- Electrolyte imbalances
- Toxins
- Drugs

#### 3. Vestibular Input

- Inflammatory disorders
- Motion sickness via acoustic nerve

#### 4. Higher Central Nervous System Centers

- Psychogenic (eg, fear, stress, excitement) via catecholamine release
- Inflammatory CNS lesions

*Courtesy Susan Little, DVM, Diplomate ABVP (Feline Practice); modified with permission*

### Mild Acute Vomiting

Pets with a history of mild, acute vomiting (with or without concurrent diarrhea) that have a normal physical examination and no other concurrent signs usually have self-limiting signs and can be treated symptomatically or simply monitored. In such cases, signs resolve after 1 to 2 days, with or without supportive therapy.<sup>2</sup>

The suggested minimum diagnostic evaluation of a healthy vomiting animal includes:

- Packed cell volume and total protein (provides a crude assessment of hydration status)
- Fecal flotation.

Even with more extensive diagnostic evaluation, a diagnosis may not be reached unless the history suggests a likely cause, such as dietary indiscretion.

Cats appear to be less likely than dogs to present with acute, self-limiting vomiting (“acute gastritis”) and are relatively more likely than dogs to require diagnostic investigation and treatment.<sup>3</sup> Feline acute hemorrhagic vomiting syndrome has been reported in the UK.<sup>3</sup> This self-limiting syndrome occurs in cats in rescue shelters and catteries; etiology has yet to be determined.

### Severe Acute Vomiting

Some characteristics of acute vomiting may indicate serious, even potentially life-threatening, diseases and

warrant immediate diagnostic investigation and treatment. This category includes patients with:

- Hematemesis (vomiting blood) or melena
- Frequent vomiting (8–10 times in 1 day)
- Concurrent signs (such as anorexia; lethargy; fever; apparent abdominal pain; or pale, “muddy,” congested, or jaundiced mucous membranes).

Diagnostic evaluation is mandatory to attempt to determine the underlying cause and guide therapy, and includes:

- Survey abdominal radiographs
- CBC, serum biochemical profile, urinalysis
- SNAP Parvo Test (idexx.com) (puppies or kittens), regardless of vaccination history.

Additional diagnostic studies may include further laboratory testing, such as:

- Canine or feline pancreatic lipase (Spec cPL or fPL Tests, idexx.com)
- Resting cortisol and/or adrenocorticotropic hormone (ACTH) stimulation testing
- Abdominal ultrasonography
- Upper GI endoscopy and/or barium contrast series
- Surgical exploration of abdomen.

### ACUTE VOMITING: MEDICAL THERAPY

The goals of symptomatic or supportive therapy for acute vomiting are:

- Treating or removing the underlying cause
- Controlling the vomiting episodes
- Addressing abdominal pain, if present
- Correcting the fluid, electrolyte, and acid–base abnormalities that may occur as a consequence of frequent vomiting.

### Antiemetic Therapy

Antiemetic therapy is warranted when:

1. Vomiting is frequent or severe, making the animal uncomfortable
2. Persistent vomiting puts the animal at risk for aspiration pneumonia or acid–base and electrolyte disturbances
3. The animal is *not* suffering from GI obstruction or toxicity.

Antiemetics control emesis by either central or peripheral blockade of neurotransmission at receptor sites (see **Medications for Vomiting: Dogs & Cats**, page 26).

- **In the emetic center**, neurokinin (NK)1 receptors and alpha-2 adrenergic receptors are the most clinically important. Selective NK1 receptor antagonists (ie, maropitant) act by blocking the binding of substance P within the emetic center and CRTZ; therefore, they uniquely inhibit vomiting through both the humoral and neural pathways.

### Maropitant: A Multimodal Antiemetic

Maropitant citrate (Cerenia, zoetis.com), a potent selective NK1 receptor antagonist, plays an important role in managing vomiting, mediated via both the vomiting center and CRTZ (ie, humoral and neural pathways).<sup>3–5</sup> The drug is effective for:

- Prevention of motion sickness in dogs
- Chemotherapy-induced nausea and vomiting
- Management of vomiting due to other causes.

### Nausea

Nausea cannot be reliably assessed in animals, but signs interpreted as nausea include salivation, increased frequency of or exaggerated swallowing motions, and licking of lips. A recent study evaluating maropitant as an antiemetic for dogs premedicated with hydromorphone found that maropitant effectively prevented vomiting, retching, and nausea associated with hydromorphone administration.<sup>6</sup>

### Analgesia

Two recent studies indicate that maropitant also provides visceral analgesia in dogs and cats.<sup>7,8</sup> During visceral ovarian and ovarian ligament stimulation, maropitant decreased anesthetic requirements. This analgesic property makes maropitant especially suitable for managing vomiting caused by painful intra-abdominal conditions, such as pancreatitis and cholangitis, and painful gastric or intestinal disorders. *Note: At this time, this use of maropitant should only be considered adjunctive to other methods of pain control.*

### Administration

Common doses for maropitant are given in **Medications for Vomiting: Dogs & Cats**, page 26. Maropitant is commonly administered off label in both dogs and cats. Hickman and colleagues reported on the pharmacokinetics of PO, SC, and IV use in cats.<sup>5</sup> Because maropitant is metabolized by the liver, a lower dosage of 0.5 mg/kg IV is sometimes used for treatment or prevention of vomiting in both species, if there is concern about liver function.

The label states that using Cerenia for treatment or prevention of acute emesis should not last longer than 5 consecutive days.

- Maropitant has nonlinear pharmacokinetics in dogs. Pharmacokinetic studies conducted since the approval of Cerenia have shown that a steady state is reached in dogs in 4 days (at 2 mg/kg daily). A steady state is reached in cats in 7 days.
- Another reason for this concern is that, if vomiting persists longer than 5 days, the underlying cause needs to be thoroughly reinvestigated.

In dogs, the injectable solution and tablets may be used interchangeably for once daily dosing to prevent acute vomiting.

### Safety

Cerenia has been tested for safety in both dogs and cats at 1×, 3×, and 5× the label dose for 15 days (3× the duration of treatment recommended on the label) as required by the FDA.

## Determining Reasons for Vomiting & Appropriate Diagnostics

Vomiting can be caused by a wide variety of GI, intra-abdominal, metabolic, systemic, and neurologic diseases. An efficient clinical approach is to determine whether vomiting results from a:

- Primary GI problem
- Metabolic problem secondarily causing GI signs.

### CLINICAL APPROACH

Primary disease is most likely when:

- An abnormality is palpable in the gut (eg, foreign body, intussusception).
- Vomiting is associated with significant diarrhea.
- If the animal is otherwise historically and clinically normal.

In the case of an animal with acute vomiting, it is important to rule out obstructions or other disorders that might require emergency surgical intervention.

#### Primary GI Disease Diagnostics

- Survey and contrast radiographs
- Abdominal ultrasonography
- Endoscopy
- Exploratory laparotomy

In chronic vomiting, emergency surgical procedures are usually not needed. In that case, it is less invasive, less expensive and usually faster to first investigate whether a metabolic problem is causing secondary GI signs with appropriate laboratory tests; then investigate primary GI disease if clinical pathology results are normal.

### CAUSES & DIAGNOSTICS

The many causes of vomiting pose a challenge when determining the degree of diagnostic evaluation warranted. This clinical decision is largely based on:

- Chronicity and frequency of vomiting
- Presence or absence of other historical and/or physical examination abnormalities.

#### Dogs

**Rosé & colleagues.** In a recent publication, 213 dogs that had vomiting as the main, or one of the main, signs were evaluated at a referral institution to determine which diagnostic tests were of greatest value.<sup>10</sup> A diagnosis was reached in 203 dogs (95.3%). See **Tables 1 and 2** for study results. Overall, there was a high incidence of nongastrointestinal diseases, especially renal, which emphasizes the need to perform a urinalysis in association with a serum biochemical profile in most animals with vomiting as the major complaint.

**Table 1. Diagnoses by Category<sup>10</sup>**

1. Gastrointestinal (43.7%)
2. Systemic (27.7%)
3. Nongastrointestinal abdominal (16.4%)
4. Miscellaneous (6.1%)
5. Neurological (1.4%)

**Table 2. Value of Diagnostic Tests<sup>10</sup>**

	Enabled Diagnosis	Assisted Diagnosis
<b>Blood analysis</b>	12.2%	26.8%
<b>Cytology</b>	3.3%	4.2%
<b>Fecal analysis</b>	6.6%	1.4%
<b>Radiographs</b>	1.9%	8.5%
<b>Ultrasound</b>	5.2%	17%
<b>Urinalysis</b>	2.3%	9.9%

**Leib & colleagues.** In another study, the diagnostic utility of abdominal ultrasound was evaluated in 89 dogs with chronic vomiting.<sup>11</sup> Ultrasound examination was considered to be vital or beneficial to diagnosis in 22.5% of dogs. Increasing age and a final diagnosis of gastric adenocarcinoma or GI lymphoma were associated with increased diagnostic utility.

#### Cats

**Batchelor & colleagues.** A recent evidence-based review of mechanisms, causes, diagnostic investigation, and management of vomiting in cats evaluated the most common causes (**Table 3**).<sup>2</sup>

Most notable is the fact that vomiting in cats might be associated with a wide range of diseases originating outside of the GI tract, such as neoplasia, splenic disease, infectious disorders, chronic nasal disease, pyothorax, aortic thromboembolism, and bronchial disease. However, the authors noted that further exploration was needed and vomiting may have been incidental.

**Table 3. Common Causes of Vomiting in Cats<sup>2</sup>**

Vomiting (Overall)
<ul style="list-style-type: none"> <li>• Adverse reactions to food</li> <li>• Infectious agents, such as panleukopenia and feline infectious peritonitis</li> <li>• Acute self-limiting emesis of undetermined cause (so-called “acute gastritis”)</li> </ul>
Chronic Vomiting
<ul style="list-style-type: none"> <li>• Inflammatory bowel disease</li> <li>• Adverse reactions to food</li> <li>• Liver disease</li> <li>• Uremia</li> <li>• Hyperthyroidism</li> </ul>

**Additional causes.** Cats frequently vomit trichobezoars (hairballs) and also vomit after administration of alpha-2 adrenergic drugs, such as xylazine and dexmedetomidine, reflecting the importance of these receptors in the brainstem areas that control vomiting.<sup>12,13</sup>

- **In the CRTZ in dogs**, dopamine and histamine are significant neurotransmitters, making dopaminergic and histaminergic receptor antagonists important antiemetic classes.
- **In the CRTZ in cats**, alpha-2 adrenergic and 5-HT<sub>3</sub> serotonergic receptors are the significant neurotransmitters.

**Specific Notes.** An antiemetic is commonly administered concurrently with a prokinetic agent. In addition, antiemetics with different modes of action may be combined in patients with refractory vomiting.

- Maropitant, ondansetron, and dolasetron are very effective antiemetics for cats.
- In dogs with uremia, the central component of vomiting can be treated with antiemetics; the peripheral component is best treated with gastroprotectants.
- Chemotherapy drugs induce vomiting by stimulating 5-HT<sub>3</sub> serotonergic receptors; effective antiemetics include dolasetron, maropitant, and ondansetron. Metoclopramide is less effective but less expensive; if administered to dogs, it should be used at high doses (1 mg/kg).
- Metoclopramide is considered a weak prokinetic agent. Higher doses (up to 4 mg/kg/day CRI or 1 mg/kg PO Q 8 H) are occasionally used in dogs but patients must be carefully monitored for extrapyramidal side effects.

**Side Effects.** The main side effects of antiemetics include:

- **Systemic hypotension:** Chlorpromazine and prochlorperazine
- **Sedation:** Phenothiazines (chlorpromazine and prochlorperazine), antihistamines, and yohimbine
- **Behavioral changes** (eg, dose-related excitation): Metoclopramide

Only administer chlorpromazine or prochlorperazine if the patient is normotensive or is receiving adequate IV fluid support. These drugs were thought to reduce the seizure threshold but clinical experience suggests they can be used in patients with seizure disorder histories.

Patients with GI obstruction should not receive prokinetic agents, including metoclopramide. However, many experienced clinicians report that serious adverse effects have not been seen when these agents have been inadvertently given to such patients, with the exception of those with linear foreign bodies.

### Gastroprotective or Cytoprotective Agents

Peripheral pathways are mediated through irritation and inflammation of the GI mucosa. Therefore, another common approach to therapy is administration of gastroprotective agents, such as drugs that:

- Inhibit gastric acid production: H<sub>2</sub> histaminergic receptor antagonists and proton pump inhibitors
- Act locally on the gastric mucosa: Sucralfate.

### Histamine H<sub>2</sub> Receptor Antagonists

Histamine H<sub>2</sub>-receptor antagonists are the most commonly used drugs to manage gastric ulceration or severe gastritis.

These agents competitively block the H<sub>2</sub> receptor on the parietal cell, reducing gastric acid secretion.

- **Cimetidine** is the least potent of the H<sub>2</sub> receptor antagonists and also inhibits the cytochrome P-450 enzyme system, potentially altering metabolism of co-administered drugs that are metabolized by the same enzyme system.
- **Ranitidine** also inhibits the cytochrome P-450 enzyme system, but much less so than cimetidine.
- **Famotidine** and **nizatidine** are more potent than cimetidine and famotidine and do not inhibit the cytochrome P-450 enzyme system. In addition, they might stimulate gastric emptying in the cat and dog by inhibiting acetylcholinesterase activity.

### Proton Pump Inhibitors

Proton pump inhibitors (PPIs) are currently the most potent inhibitors of gastric acid secretion. They irreversibly block the gastric proton pump (hydrogen-potassium ATPase), causing a marked decrease in gastric acid secretion.

PPIs are recommended for use in small animals diagnosed with severe reflux esophagitis or gastric ulceration.

- **Omeprazole (0.7 mg/kg PO Q 24 H, dogs and cats)** is now available over the counter, markedly reducing its cost and increasing its availability and usage in small animals. It has come into common use (perhaps overuse) in vomiting animals without hematemesis.
- **Pantoprazole (0.7–1 mg/kg PO or IV PO Q 24 H, dogs and cats)** is a newer PPI available for oral or IV use.

### Sucralfate

Sucralfate (0.25–1 g PO Q 8–12 H, dogs and cats) is a basic aluminum salt of a sulfated disaccharide that selectively binds to proteins at sites of ulceration.

- This drug has a sustained local protective effect against acid, pepsin, and bile at the ulcer site, forming a protective barrier.
- It also increases the luminal concentration of prostaglandin E<sub>2</sub>, which protects against ulcerogenic factors.
- Because sucralfate is not absorbed from the GI tract, it has virtually no systemic toxicity.

Constipation is a rare side effect that occurs because of the aluminum moiety. Sucralfate may also inhibit the absorption of other drugs, including doxycycline and, potentially, H<sub>2</sub> receptor antagonists.

### Prokinetic Agents

Agents that enhance gastrointestinal motility may be indicated for:

- Vomiting associated with delayed gastric emptying
- Vomiting caused by gastritis, metabolic derangements, and postoperative gastric dilatation volvulus
- Dogs that vomit bile in the morning prior to eating (bilious vomiting syndrome). Therapeutic choices for prokinetics include:
  - 5-HT<sub>4</sub> serotonergic agonists: Cisapride, metoclopramide

## MOTION SICKNESS: HELPING PETS & THEIR OWNERS

With warmer weather quickly approaching, many pet owners will be eager to head outside—and, for many, back on the road—with their pets. However, motion sickness in pets creates an unpleasant situation that often results in the pet being left out of the fun. It may even deter owners from bringing their pets to the clinic for veterinary care.

Natalie Marks, DVM, of Blum Animal Hospital in Chicago, has worked with families that have pets with motion sickness. “As veterinarians, we want to do all we can to enhance the human–animal bond for our clients,” Marks says. “For many pet owners, companionship—both off and on the road—is central to the relationships with their pets. Having a pet that doesn’t enjoy those experiences can leave the owner and pet’s bond unfulfilled.”

These 3 steps outline a therapeutic approach to motion sickness in pets:

### 1 Start the Motion Sickness Conversation

“Many times, motion sickness is brought up to us as veterinarians. Pet owners are generally very in tune with their pets and, unfortunately, may see the problem immediately in their cars,” Marks explains. However, while owners of severely stressed pets are well aware when their pets exhibit the main sign of motion sickness—vomiting—others may not recognize the less obvious signs, such as drooling, panting, licking lips, or yawning.

Veterinarians can begin a pet’s appointment by asking the owner about the ride to the clinic. This simple question may lead to discovery of motion sickness in the pet.

### 2 Make Travel a Positive Experience

Marks says that many cases of motion sickness can be addressed through simple training methods and adjustments to the travel process, such as making sure the pet does not eat 30 minutes prior to any trip.

“In these cases, I encourage pet owners to start slowly and remove any fear the pet may have of the car itself. This may begin by (1) showing the pet the car without going anywhere, (2) letting the pet take in the sights and smells, and (3) rewarding the pet with a treat. This process can evolve to a short trip to the post office, again offering a reward after completing the trip,” Marks explains.

She also encourages pet owners to make sure the car is welcoming to the pet by setting a lower temperature, cracking open a window for air circulation and, of course, making sure the pet is properly restrained facing forward in either a seat harness or carrier.

### 3 Consider Treatment Options

If motion sickness is chronic or cannot be resolved with behavioral methods, Marks recommends a prescribed treatment program to pet owners. “I like to discuss all the options with pet owners to find treatments that best fit their pets’ needs and the family’s lifestyle.”

Marks says. “There are excellent options available. For example, veterinarians recognize that antiemetics are excellent for GI cases, but I’m not sure everyone realizes that some are FDA-approved, and very effective, for motion sickness.”

Marks sums up the motion sickness discussion with, “The most important element of our work is building trust with our clients and letting them know we are there for them completely—not just for wellness, illness, or injury—but for their overall lifestyle experiences with their pets. Helping manage motion sickness can be an important part of ensuring the human–animal bond is developed to the fullest.”



### BILIOUS VOMITING SYNDROME

Dogs that suffer from this syndrome can be treated with prokinetic agents. Other therapeutic approaches, alone or in combination, include:

- Dividing the total daily food amount into an extra meal that can be given late in the evening
- Using an acid inhibitor (H<sub>2</sub> receptor antagonist) once daily in the evening
- Administering a calcium-containing antacid (such as Tums [gsk.com]) late in the evening.

- D<sub>2</sub> dopaminergic antagonist/5-HT<sub>3</sub> serotonergic antagonist: Metoclopramide
- Cholinesterase inhibitors: Ranitidine, nizatidine
- Motilin agonists: Low-dose erythromycin (dogs only).

**Cisapride** is superior to metoclopramide for treating gastric emptying disorders in cats and dogs. Cisapride stimulates GI motility from the lower esophageal sphincter to the colon (through stimulation of 5-HT<sub>4</sub> serotonergic receptors), with minimal direct antiemetic effects.

**Metoclopramide** is used to increase gastroesophageal sphincter tone, and as a prokinetic for treating gastric emptying disorders and enhancing the coordination of antropyloroduodenal contractions. The prokinetic effects of metoclopramide are not readily or exclusively explained by dopamine receptor antagonism. However, metoclopramide has other pharmacologic properties, including stimulation of 5-HT<sub>4</sub> receptors, which may better explain some of its effects on the GI tract.

**Erythromycin** stimulates phase III migrating myoelectric complex activity in the dog, but the physiologic regulation of migrating spike complex activity in the cat is different; therefore, erythromycin is not used as a prokinetic in cats.

When these drugs are used for delayed gastric emptying, they should be administered 30 minutes prior to feeding. Metoclopramide has a short half-life (60–90 min) in dogs, and is best given as a CRI for maximal effect.

### Antibiotics

Antibiotics are not routinely used for empirical therapy in acute vomiting unless the patient is febrile or has an abnormal CBC that suggests systemic infection.

When indicated, broad-spectrum antibiotics, such as amoxicillin combined with enrofloxacin, provide excellent coverage against most bacteria associated with infection following breakdown of the GI mucosal barrier.

Probiotics or an antibiotic (metronidazole, tylosin) may be useful for controlling acute diarrhea accompanying vomiting.

### ACUTE VOMITING: ADDITIONAL THERAPY

#### Dietary Management

Dogs or cats presenting with acute vomiting are commonly held NPO (*nothing per os*) for 12 to 24 hours until the vomiting ceases. While a period of NPO has

not been evaluated in an evidence-based manner, its prevention of aspiration pneumonia, additional fluid losses, and discomfort of the patient are excellent reasons to use this approach.

After vomiting has been controlled or has ceased for several hours, a small volume of a digestible intestinal formula or elimination diet (containing a novel, single protein source or hydrolyzed peptides) should be fed.

- A highly digestible, **low-fat diet** is usually selected for dogs, but dietary fat content appears to play a smaller role in gastric emptying in cats.
- **Cats** do not need a carbohydrate source and are sometimes best managed with a **single-protein source**, such as cooked chicken breast.

Feeding small meals frequently will minimize gastric distention and gastric acid secretion. A gradual transition to the pet's usual diet is made over 2 to 3 days, providing that signs have resolved.

### Fluid Therapy

Vomiting of gastric and intestinal contents usually involves:

- Loss of fluid containing chloride, potassium, sodium, and bicarbonate
- Dehydration accompanied to a variable extent by hypochloremia, hypokalemia, and hyponatremia. Subcutaneous fluids are useful for mild dehydration.
- **Isotonic fluids** should be used, with no more than **10 to 20 mL/kg** administered at each injection site. The rate of SC fluid flow usually is governed by patient comfort.
- Acetated polyionic solutions, such as Normosol-R and Plasmalyte, should not be administered SC due to discomfort associated with administration.
- Generally, all SC fluids are absorbed within 6 to 8 hours. If pockets of SC fluid are still present after this time, use of IV fluids to reestablish peripheral perfusion should be considered. Fluids should be administered IV to animals that are moderately to severely dehydrated ( $\geq 7\%$ ).
- **Potassium** supplementation to replace that lost in vomitus is usually necessary, since whole body depletion of potassium can cause GI hypomotility.
- Metabolic acidosis is the most common acid–base alteration in dogs with GI disease and is usually corrected by appropriate fluid therapy with **lactated Ringer's solution** or **0.9% saline**.
- Foreign bodies causing GI obstruction that involves the stomach or proximal duodenum can result in metabolic alkalosis, but such patients can also have metabolic acidosis or normal acid–base status, so no presumption should be made without laboratory evaluation.<sup>9</sup>

### IN SUMMARY

There are many causes of vomiting and evaluation of the vomiting dog or cat requires consideration of the whole animal, not just the GI tract.

## MEDICATIONS FOR ACUTE VOMITING: DOGS & CATS

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DRUG NAME (alpha order)	CLASSIFICATION	USE	DOSE (for cats and dogs unless otherwise noted)
<b>Chlorpromazine</b>	Alpha-2 adrenergic antagonist D2 dopaminergic antagonist H1 histaminergic antagonist M1 muscarinic cholinergic antagonist	Antiemetic	<b>0.2–0.4 mg/kg</b> SC or IM Q 8 H
<b>Cisapride</b>	5-HT <sub>4</sub> serotonergic agonist	Prokinetic agent	<b>0.5–1 mg/kg</b> PO Q 8 H or <b>1–1.5 mg/kg</b> PO Q 12 H or <b>Up to 3 mg/kg</b> divided into equal doses based on number of daily feedings; administered 30 min before each feeding
<b>Dimenhydrinate</b>	H1 histaminergic antagonist	Antiemetic	<b>4–8 mg/kg</b> PO Q 8 H
<b>Diphenhydramine</b>	H1 histaminergic antagonist	Antiemetic	<b>2–4 mg/kg</b> PO or IM Q 8 H
<b>Dolasetron</b>	5-HT <sub>3</sub> serotonergic antagonist	Antiemetic	<b>0.5–1 mg/kg</b> PO or IV Q 12 H or 30 min before chemotherapy
<b>Domperidone</b>	D2 dopaminergic antagonist	Antiemetic Increases gastroesophageal sphincter tone	<b>0.05–0.1 mg/kg</b> PO Q 12–24 H*
<b>Erythromycin</b>	Motilin agonist	Prokinetic agent	<b>0.5–1 mg/kg</b> PO or IV Q 8 H (dogs)
<b>Famotidine</b>	Histamine H <sub>2</sub> receptor antagonist	Gastroprotective agent	<b>0.5–1 mg/kg</b> IV or PO Q 12–24 H
<b>Maropitant</b>	NK1 receptor antagonist	Antiemetic Visceral analgesic	<b>1 mg/kg</b> SC or IV Q 24 H; administer SC injection cold to reduce pain <b>2 mg/kg</b> PO (dogs) and 1 mg/kg PO (cats) <b>8 mg/kg</b> PO for motion sickness (dogs)
<b>Metoclopramide</b>	D2 dopaminergic antagonist 5-HT <sub>3</sub> serotonergic antagonist 5-HT <sub>4</sub> serotonergic agonist	Antiemetic** Prokinetic agent Increases gastroesophageal sphincter tone	<b>0.2–0.5 mg/kg</b> PO, SC, or IM Q 8 H <b>1–2 mg/kg/day</b> CRI
<b>Nizatidine</b>	Cholinesterase inhibitor Histamine H <sub>2</sub> receptor antagonist	Gastroprotective agent Prokinetic agent	<b>2.5–5 mg/kg</b> PO Q 12 H
<b>Omeprazole</b>	Proton pump inhibitor	Gastroprotective agent	<b>0.7 mg/kg</b> PO Q 24 H
<b>Ondansetron</b>	5-HT <sub>3</sub> serotonergic antagonist	Antiemetic	<b>0.5 mg/kg</b> PO or IV Q 12–24 H or 30 min before chemotherapy <b>Doses up to 1 mg/kg</b> IV Q 12–24 H are occasionally needed
<b>Pantoprazole</b>	Proton pump inhibitor	Gastroprotective agent	<b>0.7–1 mg/kg</b> PO or IV Q 24 H
<b>Prochlorperazine</b>	Alpha-2 adrenergic antagonist D2 dopaminergic antagonist H1 histaminergic antagonist M1 muscarinic cholinergic antagonist	Antiemetic	<b>0.5 mg/kg</b> SC, IM, or suppository Q 8 H
<b>Ranitidine</b>	Histamine H <sub>2</sub> receptor antagonist	Gastroprotective agent Prokinetic agent	<b>1–2 mg/kg</b> PO Q 12 H
<b>Scopolamine or Hyoscine</b>	M1 muscarinic cholinergic antagonist	Antiemetic	<b>0.03 mg/kg</b> SC or IM Q 6 H
<b>Sucralfate</b>	Sucrose sulfate-aluminum complex	Cytoprotective agent	<b>0.25–1 g</b> PO Q 8–12 H
<b>Yohimbine</b>	Alpha-2 adrenergic antagonist	Antiemetic	<b>0.25–0.5 mg/kg</b> SC or IM Q 12 H

*Note: Mirtazapine, commonly used as an appetite stimulant, most likely also has an antiemetic effect based on data from human studies.*

\* There is scant clinical experience with this drug in dogs and cats

\*\* Useful in dogs; questionable efficacy in cats

An assessment as to whether the dog or cat has a self-limiting or potentially serious cause of vomiting is crucial and depends on a:

- Thorough history and careful physical examination
- Sound understanding of the differential diagnoses for acute vomiting
- Clinical judgment.

If in doubt, especially in cats, err on the side of caution and evaluate the animal more extensively to assess for potentially serious problems.

When treating a patient symptomatically for acute vomiting, further evaluation is indicated if:

- Signs do not resolve in 2 to 3 days
- Additional clinical signs develop. ■

ACTH = adrenocorticotropic hormone; CRTZ = chemoreceptor trigger zone; GI = gastrointestinal; PPI = proton pump inhibitor

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