Improve Veterinary Education

Diagnostics and Treatment Planning for Vomiting in Dogs and Cats

Introductory summary

This handout presents a structured, stepwise approach to dogs and cats with vomiting, integrating history, examination, minimum database, targeted diagnostics, triage and stabilisation, fluid and dietary therapy, and rational use of antiemetics, antiacids, prokinetics and appetite stimulants.

Structured clinical reasoning and the risk of skipping steps

Effective management of vomiting relies on integrating history, physical examination, a minimum database, and then focused diagnostics, in that order. A consistent routine reduces missed diagnoses and prevents delays created by having to revisit earlier steps. Point-of-care ultrasound (POCUS) can be valuable, but it should not replace fundamental clinical assessment. A classic reminder is the “blocked cat”, which is often more reliably identified from an appropriate history and careful abdominal palpation than by immediately reaching for an ultrasound probe.

History taking and physical examination as the foundation

History and physical examination remain central to decision-making, including distinguishing vomiting from regurgitation and contextualising the differential diagnoses. The physical examination should assess hydration status, identify signs of systemic disease, evaluate pain, and determine whether an abdominal mass is palpable. In chronic vomiting cases, dietary history requires particular attention, as these presentations are commonly frustrating for both clinicians and owners. A detailed diet history should document all diets previously used, including protein source, fat content, and whether fibre was present, and should record whether any dietary change resulted in improvement or deterioration. Nutritional assessment questionnaires and “cheat sheets” produced by the World Small Animal Veterinary Association (WSAVA) can facilitate accurate data collection and are often best completed by owners before the appointment, as assembling this information can be time-consuming.

Diagnostic strategy in acute vomiting

In acute vomiting, the initial objective is to categorise the patient according to illness severity and determine whether outpatient or inpatient management is most appropriate. Mild signs are often self-limiting and may justify minimal or no diagnostics. In contrast, severe or life-threatening signs warrant immediate investigation with bloodwork and imaging.

The key practical question in acute presentations is whether the patient can be managed as an outpatient or requires hospitalisation. Stable, hydrated patients that can retain some water and some food, that have no abdominal pain, no toxin exposure, and no history suggesting foreign body ingestion are often suitable for outpatient care. In these patients, supportive therapy such as fluids and anti-nausea medication may be sufficient to achieve improvement. Unstable patients that vomit frequently and cannot retain anything orally are more likely to require inpatient management.

Red flags prompting escalation include significant dehydration, hypovolaemic shock, haematemesis, abdominal pain, fever, polyuria and polydipsia, lethargy, and jaundice, all of which should trigger a more extensive diagnostic approach. When the history and examination do not provide a clear explanation for vomiting, a minimum database is advisable. This may include a complete blood count, or a packed cell volume with total solids, alongside a chemistry profile including electrolytes. A “minimal chemistry” typically includes glucose, urea nitrogen (BUN), creatinine, and electrolytes, and many analysers also report liver parameters. Based on clinical context, additional first-line tests may include parvovirus testing in unvaccinated dogs, urinalysis and/or urine specific gravity when renal disease is a concern, and abdominal imaging with radiography or POCUS.

Diagnostic strategy in chronic vomiting

Chronic vomiting requires a broader diagnostic work-up than acute, mild disease. Baseline testing commonly includes complete bloodwork, urinalysis, and appropriate endocrine screening. In cats, total T4 should be measured routinely. In dogs, total T4 testing should be reserved for cases in which hypothyroidism is a genuine concern, because low total T4 in a chronically unwell vomiting dog is frequently attributable to euthyroid sick syndrome rather than primary hypothyroidism. Imaging is often needed, and abdominal ultrasonography is frequently more informative than radiography in chronic vomiting, although radiographs remain a useful first-line option and may be more widely available. Ultrasonography is operator-dependent; diagnostic confidence is increased when performed by an experienced ultrasonographer or radiologist.

In chronic vomiting patients, gastrointestinal panels are often used to evaluate whether vomiting reflects broader gastrointestinal involvement. These panels commonly include cobalamin, folate, and pancreatic markers. Dietary modification is frequently the first-line therapeutic step once appropriate baseline investigations have been completed, and its success depends on a precise dietary history that records which diets have already been trialled and how the patient responded. Diets may be conceptualised according to protein type, including

novel protein or hydrolysed protein, and by fat content, including high-fat or low-fat formulations. Some chronic vomiting patients ultimately require endoscopy, necessitating referral to a facility able to perform this procedure.

Triage and stabilisation priorities

Triage directly affects outcomes, and stabilisation must not be delayed in high-risk presentations. Priorities include patients with haematemesis and severe dehydration, those in hypovolaemic shock, and those with gastric dilatation–volvulus (GDV) or marked abdominal distension. Foreign body ingestion should prompt urgent assessment, first because emesis induction may be possible if the object remains in the stomach, and second because established obstruction is associated with rapid deterioration. Paediatric patients, including puppies and kittens, warrant prompt triage because they become hypoglycaemic quickly, and early intervention is essential.

Fluid therapy principles in vomiting patients

Fluid therapy is a foundational treatment for severe vomiting because ongoing losses commonly cause depletion of water and electrolytes. Subcutaneous fluids are appropriate in mild dehydration, typically under 5%, using an isotonic crystalloid such as lactated Ringer's solution (LRS) or 0.9% sodium chloride. A practical dosing guide is 10–20 mL/kg per site, such that a 5 kg cat receives approximately 100 mL and a 10 kg dog approximately 200 mL. Caution is required in patients with a cardiac murmur; heart failure should be excluded and lower volumes may be advisable, while ensuring the dose remains aligned with the estimated dehydration. Subcutaneous fluids are generally absorbed within 6–8 hours, and rapid "disappearance" of subcutaneous fluid soon after administration may indicate more severe dehydration than initially appreciated.

Intravenous fluids are first-line in moderate to severe dehydration, commonly over 7%, and in unstable patients. Isotonic crystalloids remain the mainstay, including LRS, Plasma-Lyte, and Normosol-R, with 0.9% sodium chloride as an alternative. 0.9% sodium chloride may be preferred when metabolic alkalosis or significant chloride loss is suspected. In many cases, however, the priority is prompt restoration of circulating volume, and the clinical differences between isotonic crystalloids may be less important than delivering appropriate volumes in a timely fashion. After any necessary bolus therapy, patients should be placed on a rehydration plan.

Electrolyte monitoring is integral to fluid therapy, particularly potassium. Vomiting and gastroenteritis commonly cause total body potassium depletion, and hypokalaemia contributes

to gastrointestinal hypomotility and can predispose to ileus. Potassium should therefore be measured and corrected as needed. Potassium chloride (KCl) supplementation is commonly added to fluids, often in the range of 20–40 mmol/L, administered at an appropriate rate, while remaining well below maximum safe infusion thresholds. Fluid plans should be calculated by determining maintenance requirements and estimating the dehydration deficit. In acute dehydration, more rapid correction over approximately 6 hours may be appropriate, whereas more chronic deficits may be corrected over approximately 12 hours, with the final plan reflecting both calculations and the patient's clinical status.

Dietary management and safe reintroduction of feeding

Dietary management typically begins with a short period of nil per os (NPO; nothing per os). With active vomiting, withholding food for at least 6 hours is commonly recommended to reduce the risk of aspiration pneumonia and to allow anti-nausea medications time to take effect, thereby limiting further fluid and electrolyte losses. Once vomiting or regurgitation has ceased, food should be reintroduced using a highly digestible diet, ideally a low-fat gastrointestinal formulation, or a home-cooked option that is similarly low in fat. Fat restriction is generally more important in dogs than in cats. Feeding should begin with small, frequent meals, followed by transition back to the regular diet over the next few days. In acute vomiting, slow escalation of total daily calories is usually unnecessary, but caution is warranted when refeeding patients that have been anorexic for prolonged periods, particularly those requiring feeding tube support, in which case reintroduction should proceed gradually to allow physiological adaptation.

Pharmacological therapy: antiemetics, antiacids, prokinetics, and appetite stimulants

Antiemetic therapy is the most important pharmacological component for many vomiting patients. The most potent antiemetic and anti-nausea agents commonly used in dogs and cats include maropitant and the 5-HT_3 antagonists ondansetron or dolasetron. These drugs are available in injectable and oral formulations, allowing flexible use during stabilisation and outpatient continuation.

Maropitant is a selective neurokinin-1 (NK_1) receptor antagonist and is widely recognised under the trade name Cerenia, with generic formulations also available. In dogs, it is commonly used at approximately 1 mg/kg subcutaneously once daily, or 2 mg/kg orally once daily for acute vomiting. In cats, dosing is commonly approximately 1 mg/kg subcutaneously or orally once daily, with oral use considered off-label in many regions. Although some labelling restricts duration of use in certain jurisdictions, maropitant is frequently used off-label for longer periods

in chronic vomiting associated with conditions such as renal disease or chronic pancreatitis when clinically indicated and appropriately monitored.

Ondansetron is a 5-HT_3 (serotonin) receptor antagonist that is particularly effective for nausea and also treats vomiting. Its activity includes effects on vagal afferents in the gastrointestinal tract and on the chemoreceptor trigger zone, and it has been evaluated for chemotherapy-induced, uraemic, and visceral nausea, as well as in parvoviral disease. A commonly used dose range is 0.5–1 mg/kg orally or intravenously every 12 hours. In refractory nausea or vomiting, maropitant and ondansetron may be combined because their mechanisms are complementary, with maropitant inhibiting substance P signalling at NK_1 receptors and ondansetron blocking serotonin signalling at 5-HT_3 receptors. Oral ondansetron may have variable bioavailability, and some patients respond better to the injectable formulation. Both maropitant and ondansetron can be uncomfortable when administered subcutaneously, and this should be anticipated when selecting the route and counselling owners.

Antacid therapy is frequently overused, prompting the American College of Veterinary Internal Medicine (ACVIM) to publish guidance on rational prescribing. Antacids are indicated in a limited set of circumstances, including confirmed or strongly suspected gastroduodenal ulceration, such as that associated with NSAID toxicosis, in which an antacid may be combined with a gastric protectant. They are also used for oesophagitis, where acid suppression is typically paired with sucralfate as a mucosal protectant, and for management of reflux oesophagitis in high-risk settings, such as brachycephalic patients, including Pugs, undergoing anaesthesia, particularly when there is a history of hiatal hernia and regurgitation. Gastrointestinal bleeding, including melena or haematemesis, and mucosal bleeding associated with coagulopathies, such as immune-mediated thrombocytopenia, may also justify antacid therapy. Finally, critically ill or shocked patients may develop mucosal injury, and acid suppression may be appropriate in this context. Outside these scenarios, antacids are generally not recommended, including for routine management of renal failure. When acid suppression is indicated, proton pump inhibitors such as omeprazole or pantoprazole are typically the most efficacious agents. Sucralfate remains an important gastroprotectant and is particularly useful in oesophagitis.

Prokinetic medications must be used cautiously. They should not be used when gastrointestinal obstruction is suspected, nor in GDV, nor in megaoesophagus. Their role is primarily in paralytic ileus and related functional hypomotility, such as ileus associated with pancreatitis, where the stomach may be distended without evidence of mechanical obstruction, and in selected cases of regurgitation secondary to paralytic ileus. Cisapride is an orally administered prokinetic used in both dogs and cats. In dogs, dosing commonly falls within approximately 0.1–0.5 mg/kg orally

every 8–12 hours, ideally given around 30 minutes prior to feeding, while in cats a commonly used dose is approximately 1 mg/kg orally every 8 hours. Metoclopramide is often more effective when administered intravenously, and oral dosing may not reliably produce the desired peak effect. Continuous rate infusion can be used intravenously at approximately 1–2 mg/kg/day.

Appetite stimulants may be helpful in selected cases but should never replace assisted feeding. Patients that require a feeding tube do not benefit from prolonged reliance on appetite stimulants, and delayed escalation to assisted feeding risks malnutrition and avoidable deterioration, particularly in chronic gastrointestinal disease.

In cats, mirtazapine is commonly considered the best-studied appetite stimulant and also has antiemetic properties via 5-HT_3 receptor effects. A typical dose is 1.88 mg per cat once daily, with every-other-day dosing in renal failure because drug accumulation can occur. In dogs, the evidence base is less clear, but mirtazapine is used clinically, and a commonly cited dose is approximately 0.6 mg/kg orally every 12 hours, reflecting the shorter half-life compared with cats. Oral mirtazapine in cats often produces a noticeable effect within 30–60 minutes followed by decline thereafter. A transdermal formulation is labelled for use in cats and provides more stable absorption with fewer adverse effects such as hyperexcitability. When using the transdermal product, the application site should alternate between ears to reduce local irritation.

Capromorelin is a ghrelin receptor agonist used as an appetite stimulant, with dosing commonly around 3 mg/kg orally once daily in dogs. In a 30 kg dog, where a 30 mg/mL solution is available, this corresponds to 3 mL. A feline product exists, and a commonly used dose is approximately 2 mg/kg orally once daily, although hypersalivation may occur and the evidence base in cats is smaller than for mirtazapine. Appetite stimulants may be useful even in relatively acute conditions, such as acute gastritis, when patients have not eaten for a day or two and encouraging intake supports gastrointestinal recovery.

Conclusion and transition to case-based application

This lecture segment has focused on practical principles for deciding which vomiting patients can be managed as outpatients and which require inpatient care, and has reviewed the diagnostic and therapeutic steps that support consistent, efficient clinical reasoning. The subsequent lecture is intended to be shorter and case-based, applying these principles to real clinical scenarios to demonstrate management in practice.