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ANTI-EMETICS IN DOGS

Anti-Emetics in Dogs: Which to Pick When (PDF)

For a vomiting dog of unknown cause, maropitant 1 mg/kg SC once daily is the default — it is the only FDA-approved canine antiemetic and it blocks both central and peripheral input. Inside: Two labelled figures and five reference tables.

Contents

The 12 sections of this guide, in order.

IN SHORT

For a vomiting dog of unknown cause, **maropitant 1 mg/kg SC once daily** is the default — it is the only FDA-approved canine antiemetic and it blocks both central and peripheral input. But the best antiemetic *changes with the cause*: **ondansetron** for chemotherapy, where a head-to-head trial found **metoclopramide did nothing at all**; **maropitant at 8 mg/kg** for motion sickness, where 5-HT_3 drugs simply do not work; **metoclopramide** when you also want prokinesis; and in **parvovirus** all three perform the same. Before any of it, exclude obstruction and stabilise — suppressing vomiting in an obstructed patient removes the sign you are monitoring. Full canine doses from Papich and Plumb's below, plus a free one-page dose chart.

KEY POINTS

1. **There is no single best antiemetic.** The winner changes with the scenario, and the published head-to-head trials disagree with each other for exactly that reason.
2. **Maropitant is the only FDA-approved canine antiemetic** and the sensible default for undifferentiated acute vomiting — one injection covers 24 hours. (Licensing differs by country: metoclopramide holds a veterinary authorisation in Europe as Emeprid.)
3. **Chemotherapy is the exception that matters.** Ondansetron reduced nausea by 90% against maropitant's 25%, and metoclopramide had *no effect*.
4. **Motion sickness needs the 8 mg/kg maropitant dose**, not the vomiting dose — and 5-HT_3 antagonists do not work on the vestibular pathway at all.
5. **Ondansetron tablets are a weak substitute for the injection** — oral absorption in dogs is under 10%.
6. **Metoclopramide is the only prokinetic of the three**, and the only one outright contraindicated if obstruction is possible.
7. **Exclude obstruction and stabilise first.** An antiemetic treats a sign, not a cause.

About the doses

Every dose on this page is transcribed from the two standard references — *Papich Handbook of Veterinary Drugs*, 5th ed. and *Plumb's Veterinary Drug Handbook*, 10th ed. — and marked where use is extra-label. Doses are for **dogs**. Always confirm against the current label and your own formulary before prescribing.

Ask ten clinicians which antiemetic they reach for in a vomiting dog and most will say maropitant. They are usually right — and in one common setting they are measurably wrong. The published head-to-head trials do not agree with one another, and that is not a flaw in the evidence: it is the answer. **Antiemetics for dogs** work at different receptors on different limbs of the same reflex, so the drug that wins depends entirely on which limb is driving the vomiting.

This guide is built to be used with a patient in front of you. It opens with the answer, explains the one piece of neuroanatomy that makes the answer make sense, then gives the scenario table, the trial data behind it, and the canine doses in full.

01 The 30-second answer

If you read nothing else, read this table. Everything after it is the reasoning and the dosing detail.

CLINICAL PICTURE	FIRST CHOICE	WHY
Acute vomiting, cause not yet known	**Maropitant** 1 mg/kg SC q24h	The only antiemetic with an FDA approval in dogs, it works on both central and peripheral input, and one injection covers 24 hours
Chemotherapy	**Ondansetron** 0.3–0.5 mg/kg slow IV, 30 min before	Serotonin release from damaged gut drives this vomiting. In a head-to-head trial metoclopramide did nothing here
Motion sickness	**Maropitant** 8 mg/kg PO	A higher dose than for vomiting, and separately approved for it. 5-HT_3 antagonists are ineffective for motion sickness
Before an opioid / anaesthesia (anesthesia)	**Maropitant**, given before the opioid	Beat ondansetron in a randomised trial in dogs premedicated with hydromorphone
Parvoviral enteritis	**Any of the three**	Maropitant, ondansetron and metoclopramide were equally effective. Choose on route, cost and whether you also want prokinesis
Ileus or delayed gastric emptying	**Metoclopramide** CRI	The only one of the three that is also a prokinetic — but it is contraindicated if there is any chance of obstruction
Vestibular disease	**Maropitant**, or an antihistamine	Vestibular input runs through H_1 and M_1 receptors, which the 5-HT_3 drugs do not touch
Suspected obstruction	**None yet**	Image and stabilise first. An antiemetic here masks the sign that tells you the patient is deteriorating

> **The one rule that outranks the table**Exclude obstruction before you suppress vomiting. In an obstructed patient the vomiting *is* the monitor, and metoclopramide in particular is contraindicated. Our canine abdominal anatomy guide covers the surgical geography if imaging takes you that way.

02 Why the reflex decides the drug

Vomiting in the dog is a reflex with **four separate afferent inputs** converging on one emetic centre in the medulla. Each input runs on its own receptors — and each antiemetic blocks only the receptors it was built for. That single fact explains every apparent contradiction in the trial literature.

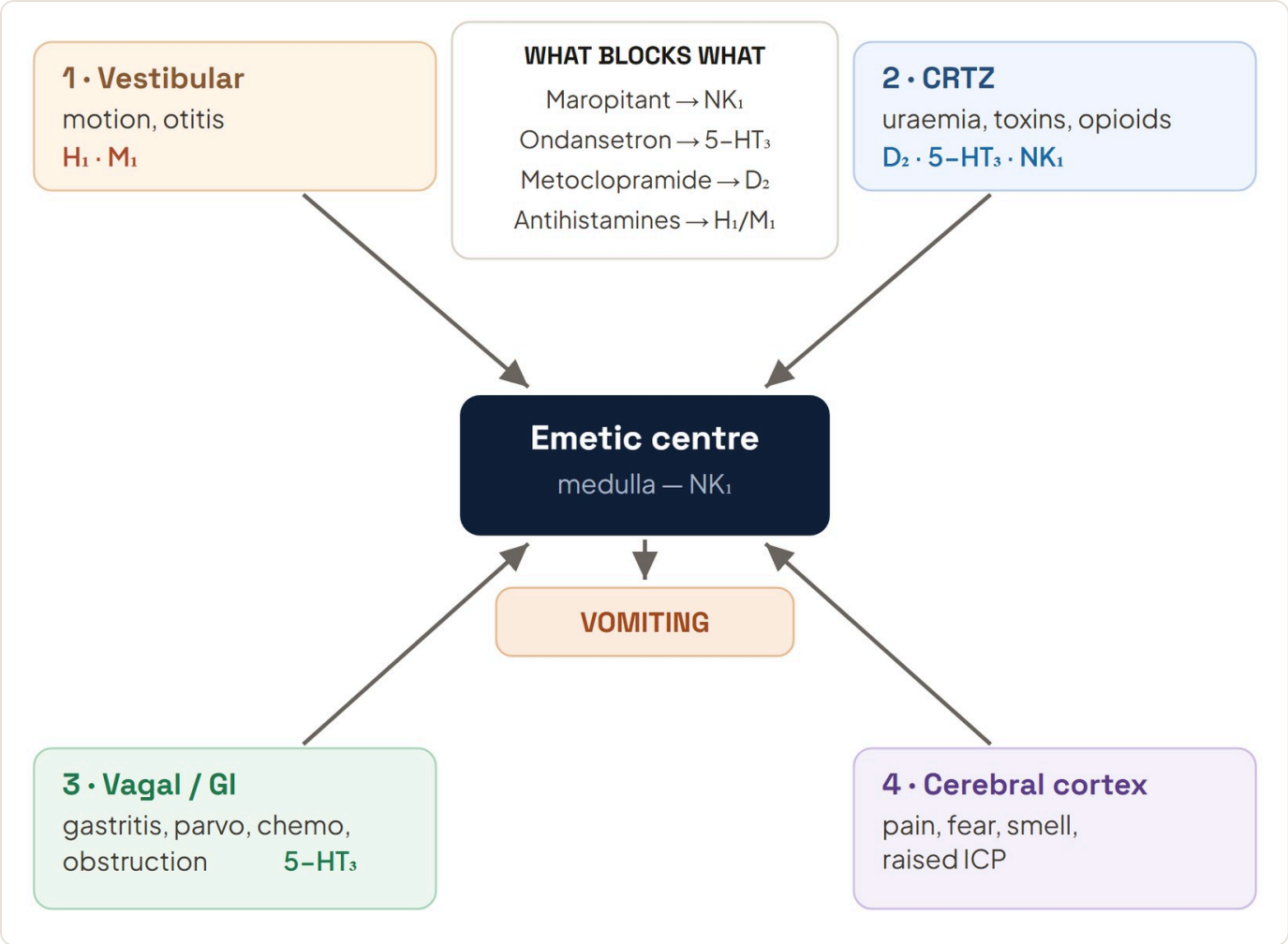

Fig 1 — The emetic reflex in the dog. Four inputs converge on the emetic centre, each carried by its own receptor family. A drug can only work on the limb whose receptors it blocks — which is why a 5-HT_3 antagonist is excellent for chemotherapy and useless for motion sickness.

Diagram — Vet-eBooks.com, built for this guide. The four afferent pathways, the receptor families each carries and the antiemetic class that blocks it are from Papich Handbook of Veterinary Drugs , 5th ed. and Plumb's Veterinary Drug Handbook , 10th ed.

Read the diagram from the outside in. **Vestibular** input — motion, otitis, vestibular disease — runs on **histamine H_1** and **muscarinic M_1** receptors. The **chemoreceptor trigger zone**, which sits outside the blood-brain barrier and samples the blood directly, carries **D_2**, **5-HT_3** and **NK_1** receptors and is the route by which uraemia (uremia), toxins and opioids cause vomiting. **Vagal afferents from the gut** are dominated by **5-HT_3**: damaged enterocytes release serotonin, which is precisely what happens in chemotherapy and viral enteritis. And the **cerebral cortex** contributes pain, fear and raised intracranial pressure.

The emetic centre itself is rich in **NK_1** receptors. That is why maropitant, an NK_1 antagonist acting at the convergence point, is the closest thing to a broad-spectrum antiemetic — and why a drug acting on a single upstream limb can still beat it when that limb is the one doing the work.

03 Which to pick when

Here is the same logic as a decision path. It starts where every vomiting case should start — not with a drug.

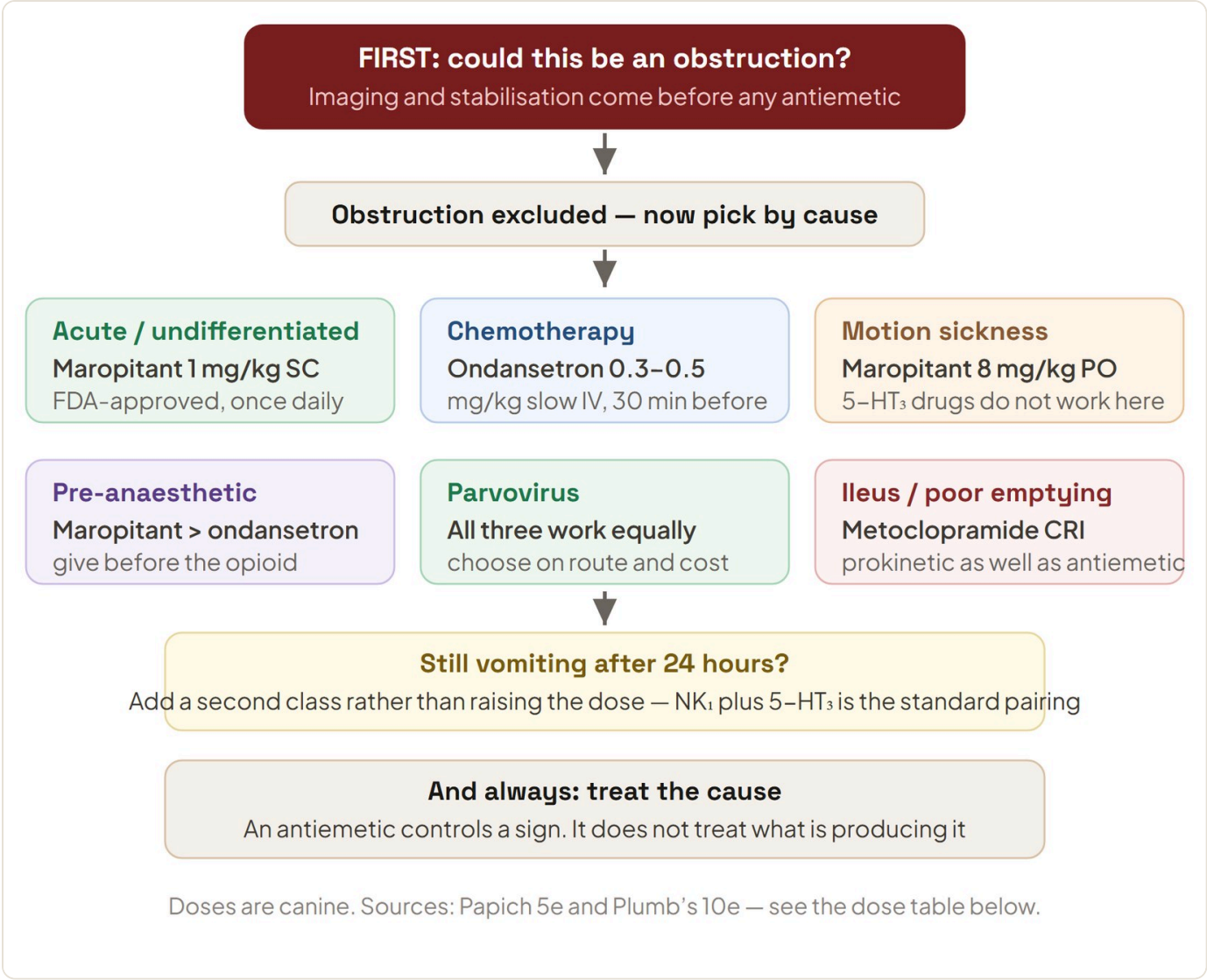

Fig 2 — Choosing an antiemetic in the dog. Obstruction is excluded first; then the cause selects the drug. If vomiting persists past 24 hours, add a second class rather than increasing the dose.

Diagram — Vet-eBooks.com, built for this guide. The choices it encodes are the ones set out in this article, from the comparative trials cited in the sources below and the canine doses in Papich Handbook of Veterinary Drugs , 5th ed.

Two things in that path are worth stating plainly. First, **the default and the best choice are not always the same drug**. Maropitant is the right default precisely because it is approved for the indication, broad and once-daily — but if you already know the cause is chemotherapy or motion sickness, the default is not the best available answer. Second, **escalation means adding a class, not raising a dose**. Blocking a receptor harder does nothing if the vomiting is being driven through a receptor you have not touched.

04 What the head-to-head trials show

These are the studies that ought to shape prescribing, and they are the reason this article exists in the shape it does. Note that they disagree — and that the disagreement is informative rather than confusing once you have the receptor map in mind.

SETTING	WHAT THE TRIAL FOUND	PRACTICAL READING
Parvoviral enteritis Yalcin 2017	Metoclopramide, ondansetron and maropitant were **equally effective** at reducing the frequency and severity of vomiting	No drug earns a premium here. Pick on route, cost, and whether you also want prokinesis
Chemotherapy Kenward 2017	Ondansetron cut the nausea-behaviour AUC by **90%** and maropitant by **25%**. **Metoclopramide had no effect** on vomiting or nausea	This is the one setting where the choice clearly matters. Reach for ondansetron

SETTING	WHAT THE TRIAL FOUND	PRACTICAL READING
Pre-anaesthetic (anesthetic) JAVMA 2022	In dogs premedicated with hydromorphone, acepromazine and glycopyrrolate, oral **maropitant beat ondansetron** at preventing vomiting and nausea	Give maropitant, and give it *before* the opioid rather than after the dog vomits
Motion sickness label and Merck	Maropitant is approved at the higher **8 mg/kg** oral dose. **5-HT$_3$ antagonists are not effective** for motion sickness	Do not substitute ondansetron for a travel-sick dog — wrong receptor, wrong pathway

Nausea and vomiting are not the same endpoint The chemotherapy trial scored *nausea behaviour*, not just the act of vomiting — which is why its ranking differs from trials that counted emetic events. A dog can be fully protected from vomiting and still be profoundly nauseated, inappetent and miserable. When you judge whether an antiemetic is working, judge appetite and demeanour, not only whether the bowl stays clean.

Put together, the picture is coherent. Where vomiting is driven by **serotonin released from damaged gut** — chemotherapy above all — the 5-HT$_3$ antagonist wins outright and the dopamine antagonist fails completely. Where the driver is **vestibular**, the 5-HT$_3$ drugs have nothing to block and maropitant or an antihistamine is required. Where several limbs are firing at once, as in **parvovirus**, blocking any one of them helps about equally. The receptor map predicts every one of those results.

For the chemotherapy setting in particular, our guide to how cancer develops in animals gives the background to why cytotoxic drugs injure the gut epithelium in the first place.

05 Antiemetic doses in dogs

All doses are canine. Maropitant is the only agent here with an **FDA approval in dogs**; in the United States everything else is extra-label use, carrying the usual obligations around consent and record-keeping. **Licensing is not the same everywhere** — metoclopramide holds a veterinary authorisation in Europe (Emeprid), so check your own national position before calling a use off-label.

DRUG	CLASS / RECEPTOR	CANINE DOSE	NOTES
Maropitant Cerenia · FDA-approved	NK$_1$ antagonist	**1 mg/kg SC q24h** (IV from 4 months, over 1–2 min) **2 mg/kg PO q24h** **8 mg/kg PO** — motion sickness **1 mg/kg SC/IV** once, 45–60 min before an emetogenic or chemotherapy drug	Up to 5 consecutive days in dogs 2–7 months; until vomiting stops in older dogs. Injection from 2 months, IV and motion-sickness tablets from 4 months
Ondansetron Zofran · extra-label	5-HT$_3$ antagonist	**0.5–1 mg/kg slow IV q8–12h** Chemotherapy: **0.3–0.5 mg/kg slow IV** 30 min before Parvovirus: **0.5 mg/kg slow IV q8h** Vestibular nausea: **0.5 mg/kg slow IV** once	Papich notes **oral absorption under 10%** in dogs, so the tablet is a poor substitute for the injection
Metoclopramide Reglan, Emeprid · extra-label	D$_2$ antagonist; 5-HT$_3$ antagonist; 5-HT$_4$ agonist (prokinetic)	**0.5 mg/kg q8h IV, IM or PO** CRI: **0.04–0.09 mg/kg/h** (1–2 mg/kg/day) Before morphine: **0.2–0.5 mg/kg SC** 30–45 min prior	As a prokinetic, give 30–60 min before a meal. Contraindicated with obstruction, perforation, haemorrhage (hemorrhage), seizures or phaeochromocytoma
Dolasetron	5-HT$_3$ antagonist	Prevention: **0.6 mg/kg q12–24h** IV, SC or PO, or 1.2 mg/kg once Treatment: **1 mg/kg q24h** IV or PO	Same class as ondansetron. Oral absorption is low and may be insufficient for a clinical effect
Chlorpromazine	Phenothiazine — D$_2$, H$_1$, M$_1$, α-adrenergic	**0.5 mg/kg q6–8h IM or SC**	Broad receptor coverage, but sedating and it causes **vasodilation and hypotension** — a poor choice in a dehydrated vomiting patient
Dimenhydrinate	H$_1$ antihistamine	**4–8 mg/kg q8–12h PO** (IM or IV for the injectable)	Vestibular and motion-related vomiting only. Sedating
Diphenhydramine	H$_1$ antihistamine	**2.2 mg/kg q8–12h PO, IM or SC**	As above. In large dogs this works out at 25 or 50 mg per dog orally

DRUG	CLASS / RECEPTOR	CANINE DOSE	NOTES
Mirtazapine	Tetracyclic — 5-HT_3 and H_1 blockade	**0.6–1 mg/kg q24h PO** (once daily — a 20 kg dog: 12–20 mg once daily)	Chiefly an appetite stimulant with antiemetic activity — useful in the inappetent chronic patient, not in the acute vomiter

> **Reading the table**Where a drug has several entries, they are genuinely different doses for different jobs — not a range to pick from. Maropitant at 8 mg/kg is a motion-sickness dose, not a bigger antiemetic dose, and ondansetron before chemotherapy is timed to the infusion rather than to the clock.

06 Maropitant in practice

Maropitant blocks substance P at the **NK_1** receptor in the emetic centre — the convergence point rather than one of the inputs — which is why it works against both centrally and peripherally driven vomiting. Peak concentration comes about **45 minutes** after injection and the effect lasts **24 hours**.

The label is more specific than most people use it. There are **minimum ages attached to each route**: the subcutaneous injection is approved from **2 months**, intravenous administration and the motion-sickness tablets from **4 months**. Duration is capped at **5 consecutive days** in dogs aged 2–7 months, but in dogs over 7 months treatment simply continues until vomiting stops.

> **The refrigeration trick**Maropitant injection stings, and the reason is chemical rather than inevitable. Papich notes that the pain comes from alteration of the formulation at room temperature — the cyclodextrin complex is more stable cold. **Store the injectable in the fridge and inject it immediately** — a study found dogs had less injection-site pain when the product was refrigerated and given straight away, so do not let the syringe warm on the prep bench. (Plumb's also notes a benzyl-alcohol-preserved formulation was less painful than the metacresol-containing product currently sold in the US.) It is the single easiest welfare improvement in this article.

Two limits worth knowing. There is **no evidence supporting its use for gastro-oesophageal reflux**, despite how often it is reached for. And while NK_1 blockade has attracted interest as a visceral analgesic, that remains experimental — there are no clinical studies in dogs demonstrating an analgesic effect, so it should not be counted as part of a pain plan.

07 Ondansetron in practice

Ondansetron blocks **5-HT_3** receptors on vagal afferents and in the CRTZ. Its niche is precise: vomiting driven by **serotonin release from injured gut**, which means chemotherapy, viral enteritis, pancreatitis and inflammatory bowel disease.

The practical catch is bioavailability. Papich records **oral absorption below 10% in dogs**, which means the tablet is not a like-for-like substitute for the injection — a point that rarely survives into discharge instructions. Plumb's lists oral dosing as commonly used while noting that supporting studies are limited. If you need reliable effect, use it **slow IV**.

One interaction deserves attention: ondansetron is serotonergic, so **combining it with other serotonergic drugs** — trazodone, fluoxetine, tramadol among them — carries a serotonin syndrome risk. That is an easy combination to assemble by accident in an anxious oncology patient.

08 Metoclopramide in practice

Metoclopramide is the odd one of the three because it does two jobs. Centrally it is a **D_2 antagonist** at the CRTZ (with 5-HT_3 antagonism at higher doses); peripherally it is a **5-HT_4 agonist** and therefore a **prokinetic**, increasing gastric and proximal small-intestinal motility. Plumb's is blunt that in dogs it is **more potent as an antiemetic than as a prokinetic**, and that it may work best as an **IV constant-rate infusion** rather than intermittently.

Use it when you want motility as well as antiemesis — ileus and delayed gastric emptying. As a prokinetic it should be given **30–60 minutes before a meal**. What it should not be used for, on the evidence above, is chemotherapy-induced nausea.

Do not reach for it to stop reflux The theory is attractive — raise lower oesophageal sphincter tone, reduce reflux — but the canine evidence does not support it. Plumb's records a study showing **no significant change in LES pressure** in dogs, no reduction in **gastro-oesophageal reflux** under acepromazine/propofol/isoflurane anaesthesia, and no reduction in **aspiration pneumonia** after arytenoid lateralisation for laryngeal paralysis. The one positive signal is a metoclopramide-plus-famotidine protocol in brachycephalic airway surgery, where postoperative regurgitation fell. Note too that the effect on distal oesophageal peristalsis is **species-specific and weaker in dogs** than in humans or cats.

Another argument for the CRI The extrapyramidal reactions reported in dogs — head tremors, dystonia of the back and thoracic muscles — followed an **IV bolus**. Giving it as a constant-rate infusion avoids the peak that seems to provoke them, and Plumb's already notes the drug works best that way. Watch particularly for tremors in dogs with **renal insufficiency**.

Contraindications you must not skipGI **haemorrhage, obstruction or perforation**; **seizure disorders** (relatively, and by some accounts absolutely); and **phaeochromocytoma**, where IV administration can release catecholamines and trigger a hypertensive crisis. Efficacy is also reduced by concurrent atropine-like drugs.

09 Phenothiazines, antihistamines and mirtazapine

The older drugs still have places, but they are narrower than they once were.

Phenothiazines — chlorpromazine, prochlorperazine — block D_2, H_1, M_1 and α-adrenergic receptors, which makes them genuinely broad-spectrum. The problem is the last of those: α-blockade produces **vasodilation and hypotension**, and sedation on top. In a dehydrated, vomiting patient that is the wrong pharmacology, which is why these have largely been displaced. Chlorpromazine remains reasonable for vomiting from toxins or drugs in a *well-perfused* dog.

Antihistamines — dimenhydrinate, diphenhydramine — block H_1 and are therefore aimed squarely at the **vestibular** limb. They are for motion and vestibular disease, and they will do very little for gastroenteritis. Expect sedation.

Mirtazapine combines 5-HT_3 and H_1 blockade with appetite stimulation. Its role is the **inappetent chronic patient** — the nauseated dog with kidney disease that will not eat — rather than the acute vomiter in front of you today.

10 Combining antiemetics

Combination is not a last resort; it is the logical consequence of a reflex with four inputs. Papich states directly that other antiemetics — ondansetron, metoclopramide, dopamine antagonists — **can safely be added** to maropitant.

The standard pairing is **NK_1 plus 5-HT_3** — maropitant with ondansetron — which covers the convergence point and the serotonergic limb together. In human oncology the equivalent combination adds dexamethasone for highly emetogenic protocols. If a patient is still vomiting after 24 hours on an appropriate single agent, **add a second class rather than increasing the dose**: a receptor you have already saturated will not yield more by being blocked harder.

What combination cannot fix is a missed diagnosis. Persistent vomiting on appropriate multi-class therapy should send you back to imaging, not further up the dose.

11 Adverse effects and what to tell the owner

Choosing a drug is only half the decision; the other half is knowing what it will do to the patient and what the owner should be told to expect. None of these agents is dangerous in the way an NSAID can be, but each has a characteristic nuisance profile, and two of them have effects that get mistaken for the disease.

DRUG	WHAT TO EXPECT	WHAT TO WARN THE OWNER ABOUT
Maropitant	Well tolerated in dogs and cats. Allergic reactions are possible but **rare**, and typically resolve within 48 hours of stopping the drug	**Pain or swelling at the injection site** is the common complaint. At the higher **motion-sickness dose the two most frequent effects are pretravel vomiting and hypersalivation** — warn owners, or the drug looks like it caused the problem it was given to prevent. Depression, lethargy, anorexia and diarrhoea (diarrhea) are also reported; anaphylactoid reactions with facial swelling are rare

DRUG	WHAT TO EXPECT	WHAT TO WARN THE OWNER ABOUT
Ondansetron	Appears well tolerated; Papich notes adverse effects have **not been reported in animals**	From human data (each under 10%): constipation or diarrhoea, sedation, extrapyramidal signs such as **head shaking**, raised liver enzymes, arrhythmias and hypotension
Metoclopramide	The **most common effects in dogs are changes in mentation and behaviour** — motor restlessness, involuntary spasms, aggression, vocalisation, hyperactivity, drowsiness or depression. Infrequent, but the reason to watch	**Extrapyramidal signs** — head tremors and dystonia of the back and thoracic muscles — have followed an **IV bolus**. **Tremors are more likely in renal insufficiency.** It also increases detrusor contractility and reduces bladder capacity, and can cause constipation
Chlorpromazine	Sedation is the usual effect, with α-adrenergic blockade and vasodilation	Expect a flat, sleepy dog, and expect blood pressure to fall — which is why it is a poor choice in the patient who has been vomiting
Antihistamines	Sedation	Drowsiness is the point of complaint; it is otherwise a well-tolerated group

Two are worth repeating because they are so easily misread. A dog given maropitant for **motion sickness** may vomit and salivate *before* the journey — that is a recognised effect of the high oral dose, not a treatment failure. And a dog on **metoclopramide** that becomes restless, vocal or oddly aggressive is not in pain and has not become unwell: it is showing central dopaminergic blockade, and the drug should be stopped rather than the dose increased.

12 When NOT to give one

This is the section that matters most, because the errors here are the ones that harm patients.

SITUATION	WHY AN ANTIEMETIC IS THE WRONG MOVE
Possible GI obstruction	Vomiting is the sign that tells you the patient is obstructing. Suppress it and you remove your own monitor while the bowel keeps deteriorating. Image first. Metoclopramide is **explicitly contraindicated** here
Not yet stabilised	A dog that has been vomiting is hypovolaemic and often hypokalaemic. Fluids and electrolytes come first — an antiemetic does nothing for perfusion
Seizure disorder (metoclopramide)	Relatively, and by some accounts absolutely, contraindicated. Use a different class
Phaeochromocytoma (metoclopramide)	IV injection can release catecholamines and precipitate a **hypertensive crisis**
Hypotension or dehydration (phenothiazines)	Chlorpromazine causes α-adrenergic blockade and vasodilation. It is the wrong drug in exactly the patient who is vomiting
Toxin ingestion where emesis is wanted	If you still need the stomach emptied, an antiemetic works directly against you

The general principle is worth stating once, plainly: **an antiemetic controls a clinical sign.** It buys comfort, it protects fluid and electrolyte balance, and it makes the patient easier to manage — but it does nothing whatever about the reason the dog is vomiting. The vomiting patient still needs fluid resuscitation and electrolyte correction; our guide to fluid therapy physiology covers why that comes first.

One more overlap worth flagging: if the vomiting patient is on an **NSAID**, stop it. GI ulceration is a common and entirely iatrogenic cause of vomiting, and continuing the drug while suppressing the sign is the worst of both worlds — see our NSAIDs in dogs and cats guide for the washout and toxicity detail.

13 Antiemetics for dogs — frequently asked questions

What is the best antiemetic for dogs?

For acute vomiting of unknown cause, **maropitant (Cerenia)** at 1 mg/kg SC once daily — it is the only FDA-approved canine antiemetic, it acts at the convergence point of the vomiting reflex, and one dose lasts 24 hours. But "best" is scenario-dependent: **ondansetron** is clearly better for chemotherapy-induced nausea, and only maropitant works for motion sickness.

Can I give a dog maropitant and ondansetron together?

Yes. Papich states that ondansetron, metoclopramide and dopamine antagonists can safely be added to maropitant. NK_1 plus 5-HT_3 is the standard pairing when a single agent is not holding, because the two drugs block different limbs of the same reflex.

How long does maropitant take to work in a dog?

Peak plasma concentration is reached about **45 minutes** after subcutaneous injection, and the effect lasts around **24 hours** — which is why it is a once-daily drug.

Why does the Cerenia injection sting, and can I stop it?

Largely, yes. The pain comes from alteration of the formulation when it is stored at room temperature; the cyclodextrin complex is more stable refrigerated. **Keep the injectable in the fridge and administer it cold** and the injection is noticeably less painful.

Does ondansetron work orally in dogs?

Poorly. Papich records **oral absorption below 10%** in dogs, so tablets may not achieve a clinically useful concentration. Where effect matters, give it **slowly intravenously**.

Which antiemetic is best for parvovirus?

On the available head-to-head evidence, it does not matter much: metoclopramide, ondansetron and maropitant were **equally effective** at reducing vomiting frequency and severity in parvoviral enteritis. Choose on route, cost, and whether prokinesis is also wanted. Plumb's lists ondansetron at 0.5 mg/kg slow IV every 8 hours for this indication.

What can I give a dog for motion sickness?

Maropitant at 8 mg/kg orally — note that this is much higher than the 2 mg/kg vomiting dose — given on an empty stomach or with a small amount of food, for up to two consecutive days. Do not feed a full meal before travel. Warn the owner that at this dose the commonest adverse effects are **pretravel vomiting and hypersalivation**, so a dog may be sick *before* the journey and the drug can be blamed for the very problem it prevents. Antihistamines are an alternative; ondansetron is not, because 5-HT_3 antagonists do not act on the vestibular pathway.

When should you NOT give a dog an antiemetic?

When **obstruction has not been excluded** — vomiting is the sign you are monitoring, and metoclopramide is specifically contraindicated. Before the patient is stabilised. When emesis is still wanted after toxin ingestion. And avoid metoclopramide with seizure disorders or phaeochromocytoma, and phenothiazines in a hypotensive or dehydrated dog.

Can you give maropitant to a puppy or kitten, and from what age?

Age genuinely matters here, and for a specific reason: **bone marrow**. Plumb's states plainly that maropitant should **not be given to puppies younger than 8 weeks**, because bone marrow problems can occur. It also advises caution with the *higher* motion-sickness dose in **puppies younger than 11 weeks**, where a greater frequency and severity of histological **bone marrow hypoplasia** was seen compared with controls; in puppies of **16 weeks or older** that hypocellularity was not observed. There is also a pharmacokinetic difference worth knowing — puppies at 10 weeks and kittens at 16 weeks clear maropitant *faster* than adults do. Check the label for your formulation and jurisdiction, because the licensed minimum age differs between the tablet and the injection and between countries.

Does maropitant need a dose reduction in liver disease?

Yes. Maropitant is hepatically metabolised, and Plumb's advises caution in hepatic dysfunction with the dose **reduced by 50%** in those patients. It is an easy one to overlook, because the patients most likely to be vomiting persistently are often exactly the ones whose liver function is compromised.

Is metoclopramide a good antiemetic for dogs?

It is a reasonable one, and Plumb's notes it is more potent as an antiemetic than as a prokinetic in dogs. But it **failed completely against chemotherapy-induced nausea** in a controlled trial, so it should not be used there. Its distinctive value is that it is the only one of the three that is also a prokinetic.

When to refer

- **Default** — maropitant 1 mg/kg SC q24h. The only FDA-approved option.
- **Chemotherapy** — ondansetron 0.3–0.5 mg/kg slow IV, 30 min before. Not metoclopramide.
- **Motion sickness** — maropitant 8 mg/kg PO. Not a 5-HT_3 antagonist.
- **Pre-anaesthetic** — maropitant, before the opioid.
- **Parvovirus** — all three equal; choose on route and cost.
- **Ileus or delayed emptying** — metoclopramide CRI, 0.04–0.09 mg/kg/h. Not for reflux — the canine evidence is negative.
- **Vestibular** — maropitant or an antihistamine.
- **Ondansetron orally** — under 10% absorbed. Use IV.
- **Still vomiting at 24 h** — add a class, do not raise the dose.
- **Watch for** — behaviour change on metoclopramide; pretravel vomiting on high-dose maropitant.
- **Never** — before excluding obstruction, or before stabilising.

Sources

1. Papich MG. *Papich Handbook of Veterinary Drugs*, 5th ed. Elsevier — primary source for the canine doses of maropitant, ondansetron, metoclopramide, dolasetron, chlorpromazine, dimenhydrinate, diphenhydramine and mirtazapine, the ondansetron oral-absorption figure, and the statement that other antiemetics can safely be added to maropitant, plus the adverse-effect profiles of metoclopramide, ondansetron and dolasetron.
2. *Plumb's Veterinary Drug Handbook*, 10th ed. — the FDA-approved maropitant dosage grid with minimum ages by route, scenario-specific ondansetron and metoclopramide dosing, the metoclopramide contraindications, and the dog-specific adverse-effect profiles — including the refrigerated-injection study, the pretravel vomiting and hypersalivation seen at the motion-sickness dose, and the extrapyramidal signs reported after an IV metoclopramide bolus.
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