

# Vomiting

## References:

DeNovo RC, Chapter 5, "Diseases of the Stomach," in Todd R Tams *Small Animal Gastroenterology*, 2<sup>nd</sup> Edition.

## I. Etiopathogenesis:

### A. Anatomy and physiology.

#### 1. Stomach.

##### a. Four functional and anatomic regions:

- 1) Cardia - The gastric inlet.
- 2) Fundus. - Dilates during gastric filling.
- 3) Body - Stores ingesta; Secretes HCl, pepsin and lipase.
- 4) Antrum/Pylorus.
  - i. Antrum grinds food into smaller particles.
  - ii. Pylorus limits the size of food particles that leave the stomach.

##### b. Three layers:

- 1) Mucosa.
  - i. Epithelium – columnar cells secrete mucus and bicarbonate.
  - ii. Glandular lamina propria.
    - i. Glands in cardia secrete mucus and pepsinogen.
    - ii. Glands in the fundus and body have parietal cells and secrete HCl, and chief cells that secrete pepsinogens.
    - iii. Glands in the antrum secrete mucus and pepsinogens; and G cells that secrete gastrin.
- 2) Muscularis – smooth muscle.
  - i. Inner circular layer.
  - ii. Thin oblique middle layer.
  - iii. Outer longitudinal layer.
- 3) Serosa.

#### 2. Small intestine.

##### a. Three sections.

- 1) Duodenum.
- 2) Jejunum.
- 3) Ileum.

##### b. Four layers:

- 1) Mucosa.
- 2) Submucosa.
- 3) Muscularis.
- 4) Serosa.

**B. Etiologies.** Vomiting is the most common sign of gastric disease. But not all vomiting dogs have gastric disease, and not all dogs with gastric disease vomit.

1. Causes of acute vomiting.

a. Stomach.

- 1) Infectious – parasites.
  - i. Ascarids – puppies.
  - ii. *Physaloptera spp.*
  - iii. *Ollulanus spp.*
  - iv. *Giardia spp.*
  - v. *Neorickettsia spp.* (salmon poisoning).
- 2) Nutritional
  - i. Eating spoiled food.
  - ii. Abrupt dietary change, when diet is uniform.
  - iii. Gastric foreign body/material.
  - iv. Dietary intolerance.
- 3) Inflammatory – Gastric ulcer.
- 4) Toxic
  - i. Drugs – Antibiotics, NSAIDs, immunosuppressives, cardiac glycosides, anticholinergics, accidental overdose..
  - ii. Ingested caustic substance (pot pourri oil, cleaning supplies, fertilizers, petroleum distillates, organophosphates, toxic plants).
  - iii. Heavy metals – lead, zinc.
  - iv. Ethylene glycol.
- 5) Traumatic.
  - i. Acute GDV.
  - ii. Diaphragmatic hernia.

b. Small intestine.

- 1) Infectious
  - i. Bacterial – acute bacterial enteritis.
  - ii. Viral
    - i. Coronavirus.
    - ii. Parvovirus.
    - iii. Canine Distemper Virus (CDV).
  - iii. Parasitic.
    - i. Ascarids (puppies).
    - ii. *Giardia spp.*
- 2) Inflammatory.
  - i. Duodenal ulcer.
  - ii. Hemorrhagic gastroenteritis (HGE).
  - iii. IBD rarely sometimes can have an apparently acute presentation
- 3) Neoplasia.
  - i. Mast cell tumor degranulation.
  - ii. Intussusception.
  - iii. Strangulation by twisted pedunculated tumor.
- 4) Nutritional.
  - i. Eating spoiled food.
  - ii. Abrupt dietary change, when diet is uniform.
  - iii. Foreign body/material.
  - iv. Dietary intolerance.
  - v. Food allergy.
- 5) Toxic.
  - i. Immunosuppressive drugs.
  - ii. See also toxins that cause acute vomiting due to stomach disease.

- 6) Traumatic
  - i. Intestinal volvulus.
  - ii. Intussusception.
  - iii. Diaphragmatic hernia.
  
- c. Large intestine.
  - 1) Inflammatory
    - i. Hemorrhagic gastroenteritis (HGE).
    - ii. Acute colitis.
  - 2) Traumatic – acute constipation.
  
- d. Pancreas.
  - 1) Acute pancreatitis.
  
- e. Biliary disease.
  - 1) Infectious – acute bacterial cholangitis/cholangiohepatitis.
  - 2) Trauma – biliary rupture.
  
- f. Abdomen
  - 1) Peritonitis.
    - i. Septic – perforated bowel or ruptured abscess.
    - ii. Chemical – leakage of bile, urine or chyle; pancreatitis .
    - iii. Viral – FIP.
  - 2) Abdominal pain
    - i. Passing a kidney stone.
    - ii. Bile duct obstruction.
    - iii. Rapidly growing mass in encapsulated organ – liver, spleen, kidney.
    - iv. Pyelonephritis.
    - v. Urinary obstruction.
    - vi. Torsed cryptorchid testicle.
    - vii. Splenic torsion.
  
- g. Systemic Disease.
  - 1) Acute renal failure.
  - 2) Acute liver failure (see section 2).
  - 3) Diabetic ketoacidosis.
  - 4) Hypoadrenal crisis.
  - 5) Sepsis .
  - 6) Acidosis.
  - 7) Hypokalemia.
  - 8) Acute hypocalcemia.
    - i. Eclampsia.
    - ii. Cholecalciferol rodenticide toxicity.
  - 9) Hypomagnesemia.
  - 10) Immune mediated disease.
  - 11) Icterus.
  - 12) Vestibular disease.
  - 13) Neoplasia affecting vomiting center or chemoreceptor trigger zone (CRTZ).
  - 14) Cerebral edema or increased CSF pressure.
  - 15) Limbic epilepsy.
  - 16) Feline heartworm disease.

- h. Environmental
  - 1) Motion sickness.
  - 2) Heat stroke.
  - 3) Pain, fear, excitement.

2. Causes of chronic vomiting (longer than 2 weeks in duration).

- a. Distal esophageal disease.
  - 1) Anomalous – hiatal hernia.
  - 2) Inflammatory.
    - i. Gastroesophageal reflux
    - ii. Distal esophagitis.
- b. Stomach.
  - 1) Anomalous.
    - i. Pyloric mucosal hypertrophy.
    - ii. Pyloric muscular hypertrophy.
  - 2) Metabolic – hypothyroidism may cause decreased motility.
  - 3) Neurologic – motility disorders.
    - i. Gastric hypomotility.
    - ii. Gastric dilatation.
    - iii. Gastric dysrhythmia – stomach motility seems normal when the stomach is empty, but is incoordinated in response to solid food.
  - 4) Neoplasia.
    - i. Lymphoma.
      - i. Most common gastric neoplasia in cats.
      - ii. 2<sup>nd</sup> most common gastric neoplasia in dogs.
    - ii. Adenocarcinoma.
      - i. Most common gastric neoplasia in dogs.
      - ii. Metastasizes to lymph nodes, liver and lungs.
      - iii. Most commonly in the pylorus.
    - iii. Scirrhous adenocarcinoma.
    - iv. Leiomyoma/sarcoma.
      - i. Leiomyomas are most common in the cardia.
      - ii. Leiomyomas are usually clinically silent, unless they cause pyloric outflow obstruction.
      - iii. These tumors are rare in cats.
    - v. Fibroma/sarcoma.
    - vi. Squamous cell carcinoma.
    - vii. Plasma cell tumor.
    - viii. Mast cell tumor.
  - 5) Nutritional
    - i. Gastric foreign body/material.
    - ii. Eating too rapidly.
    - iii. Dietary intolerance.
    - iv. Food allergy.
  - 6) Idiopathic.
    - i. Chronic Gastric Dilatation.
      - i. Overgrowth of anaerobes.
      - ii. Motility disorder.
    - ii. Acquired pyloric mucosal hypertrophy.
  - 7) Infectious.
    - i. Bacterial – *Helicobacter spp.*
      - has been associated with chronic gastritis, gastric and duodenal ulcers, gastric carcinoma and lymphoma.
      - infection rate is much higher than associated disease.

--treatment is associated with elimination of clinical signs,  
but the treatment also treats many other causes of  
chronic gastritis.

- ii. Fungal.
    - i. *Histoplasma spp.*
    - ii. Phycomycosis – most often in the pylorus.
  - iii. Parasitic.
    - i. *Giardia spp.*
    - ii. *Ollulanus spp.*
    - iii. *Physaloptera spp.*
- 8) Inflammatory.
- i. Gastric ulcer.
  - ii. Chronic gastritis – superficial, atrophic, hypertrophic.
  - iii. Inflammatory bowel disease.
  - iv. Gastric polyp, if pyloric obstruction.
  - v. Duodenogastric reflux.
  - vi. Chronic hypertrophic pyloric gastropathy (acquired mucosal hypertrophy).
- 9) Toxic – organophosphates.
- 10) Traumatic.
- i. Chronic GDV.
  - ii. External compression.

c. Small Intestine.

- 1) Metabolic – hypothyroidism causes decreased motility.
- 2) Neoplasia.
  - i. Lymphoma.
  - ii. Adenocarcinoma.
  - iii. Scirrhus adenocarcinoma.
  - iv. Leiomyoma/sarcoma.
  - v. Mast cell tumor.
- 3) Neurologic – reverse intestinal peristalsis .
- 4) Nutritional.
  - i. Foreign body/material.
  - ii. Dietary intolerance.
  - iii. Food allergy.
- 5) Inflammatory
  - i. Duodenal ulcer.
  - ii. Biliary vomiting syndrome.
  - iii. Inflammatory bowel disease.
    - Eosinophilic inflammation can be associated with:
      - Parasites
      - Idiopathic inflammatory bowel disease
      - Food allergy
      - Hypereosinophilic syndrome in cats
      - Hypoadrenocorticism
      - Systemic mast cell tumor
      - Heartworm disease in cats
- 6) Infectious.
  - i. Bacterial.
    - i. Small intestinal bacterial overgrowth (antibiotic responsive diarrhea).
    - ii. *Mycobacterium spp.*
  - ii. Fungal.
    - i. *Histoplasma spp.*
    - ii. Phycomycosis.

- iii. Parasitic – *Giardia spp*
    - iv. Blue-green algae – *Prototheca spp.*
  - 7) Toxic – organophosphates.
  - 8) Traumatic - intussusception.
- d. Large intestine.
  - 1) Metabolic – hypothyroidism may predispose to megacolon.
  - 2) Inflammatory
    - i. Chronic colitis.
    - ii. Irritable bowel syndrome (IBS).
  - 3) Traumatic – chronic constipation.
- e. Pancreas – chronic pancreatitis.
- f. Biliary disease.
  - 1) Metabolic – biliary sludging.
  - 2) Neoplasia.
  - 3) Infectious – chronic bacterial cholangitis/cholangiohepatitis.
  - 4) Inflammatory – chronic immune mediated cholangiohepatitis.
  - 5) Traumatic – gall stones.
- g. Abdomen – mass compressing the GI tract.
  - 1) Cyst – hepatic, choledochal, perirenal, pancreatic.
  - 2) Abscess.
  - 3) Granuloma.
  - 4) Neoplasm.
- h. Systemic Disease.
  - 1) Chronic renal failure.
  - 2) Chronic liver failure (see section 2).
  - 3) Hypoadrenocorticism.
  - 4) Acidosis.
  - 5) Hypokalemia.
  - 6) Hypercalcemia.
  - 7) Hypocalcemia.
  - 8) Paraneoplastic.
    - i. Gastrinoma.
    - ii. Hypercalcemia.
    - iii. Systemic inflammation.
  - 9) Icterus.
  - 10) Vestibular disease.
  - 11) Neoplasia affecting vomiting center or chemoreceptor trigger zone (CRTZ).
  - 12) Increased CSF pressure.
  - 13) Feline heartworm disease.
  - 14) Feline hyperthyroidism.

### 3. Causes of hematemesis.

- a. Blood that is swallowed from the sites below, and then vomited up.
  - 1) Respiratory tract.
    - i. Coagulopathy.
      - i. Factor deficiency.
      - ii. Liver failure
      - iii. Thrombocytopenia.
      - iv. Platelet function defect.
      - v. DIC.

- vi. Vasculitis.
      - vii. Anti-vitamin K rodenticide toxicity.
    - ii. Neoplasia.
    - iii. Pulmonary thromboembolism.
  - 2) Caudal nasopharynx (rostral nasal cavity bleeding usually results in epistaxis).
    - i. Coagulopathy (see above).
    - ii. Neoplasia.
  - 3) Oral cavity.
    - i. Coagulopathy (see above).
    - ii. Dental disease.
    - iii. Neoplasia.
- b. Bleeding from the stomach.
- 1) Coagulopathy (see above).
  - 2) Ulcerative/erosive gastric disease.
    - i. Liver failure.
    - ii. Kidney failure.
    - iii. Hypoadrenocorticism.
    - iv. Gastric neoplasia – see chronic vomiting.
    - v. Pancreatic neoplasia – gastrinoma.
    - vi. Toxicity – NSAIDs, glucocorticoids, lead
    - vii. Toxicity – caustic substances
      - cleaning supplies
      - pot pourri oil
    - viii. Trauma to the gut.
    - ix. Shock – anaphylaxis, hypovolemia, septic, HGE.
    - x. Anesthesia (hypovolemia).
    - xi. Spinal trauma.
- c. Bleeding from the duodenum, refluxed into the stomach.
- 1) Coagulopathy (see above).
  - 2) Ulcerative/erosive duodenal disease – see differentials for gastric ulcerative/erosive disease above.
  - 3) Parasites – Coccidia, hookworms.
  - 4) See also Melena in the Diarrhea Section.

**C. Pathogenesis.** Causes of vomiting:

- 1. Irritation of the GI tract - stimulates the afferent nerves to the vomiting center.
  - a. Acute gastritis.
    - 1) Acute liver failure.
      - i. Decreased gastric mucus, due to abnormal protein synthesis.
      - ii. Decreased gastric epithelial cell renewal due to abnormal protein synthesis.
      - iii. Decreased gastric blood flow, due to altered vasoactive factors.
    - 2) Acute kidney failure.
      - i. Injury to gastric mucosa by uremic toxins.
      - ii. Decreased renal metabolism of gastrin by the kidneys, leading to elevated gastrin levels, and increased HCl secretion in the stomach.

- 3) NSAIDs.
  - i. Direct damage to the gastric mucosa.
  - ii. Inhibition of gastroprotective prostaglandins.
  - iii. Piroxicam, ibuprofen and naproxen undergo more complete enterophepatic circulation, and thus have prolonged half life in the dog and cat.
  - iv. COX2 selective are not as GI toxic as COX non-selective, but both can cause problems.
  
- b. Chronic gastritis.
  - 1) Chronic kidney failure – see acute gastritis.
  - 2) NSAIDs – see acute gastritis.
  - 3) Glucocorticoids:
    - i. Decreased mucosal cell growth and mucus production.
    - ii. Increased gastric acid secretion.
    - iii. Not usually acutely GI toxic, unless really high dose.
    - iv. Can cause chronic GI toxicity when used for many weeks at immunosuppressive dose (>1 mg/lb/day).
    - v. Cause chronic toxicity usually when combined with other risk factors: NSAIDs, hypotension, bile acid reflux, spinal cord disease, liver disease, renal disease, Addison’s disease, mast cell tumor degranulation, gastrinoma.
  
- c. Elevated gastrin levels.
  - 1) Gastrin produced by gastric mucosa and pancreas.
  - 2) Gastrin stimulates the gastric mucosa to make HCl.
  - 3) Excessive gastrin levels can result in:
    - i. Gastroesophageal reflux.
    - ii. Distal esophagitis.
    - iii. Esophageal, gastric and duodenal ulceration.
    - iv. Chronic gastritis and/or duodenitis.
  
2. Chemical stimulation of the chemoreceptor trigger zone.
  
3. Altered GI motility.
  - a. Acute vomiting due to acute decrease in GI motility.
    - 1) Inflammatory disease of the stomach and/or small intestine.
      - i. Inflammation of the abdomen or proximal organs – peritonitis, pancreatitis, etc.
      - ii. Inflammation/infection of the gut – bacterial, viral, etc.
    - 2) Ulcerative disease of the stomach and/or small intestine.
    - 3) Metabolic disease.
      - i. Hypoadrenocorticism– acute episode.
      - ii. Diabetic ketoacidosis .
      - iii. Uremia – acute renal failure .
      - iv. Hypocalcemia – eclampsia.
    - 4) Spinal injury.
    - 5) Post-abdominal surgery.
    - 6) Drugs.
      - i. Anticholinergics – Centrine (aminopentamide) is NOT indicated for vomiting or diarrhea due to ileus.
      - ii. Beta agonists (bronchodilators such as terbutaline).
      - iii. Opiates.
    - 7) Toxicity – hypercalcemia caused by acute cholecalciferol toxicity.



- b. Chronic vomiting due to chronic decrease in GI motility.
  - 1) Degenerative disease – dysautonomia.
  - 2) Infiltrative disease of the stomach and/or small intestine.
  - 3) Infectious – mycobacterial, fungal, blue-green algae.
  - 4) Inflammatory.
    - i. IBD - lymphoplasmacytic, eosinophilic, pyogranulomatous.
    - ii. Chronic gastritis/enteritis.
    - iii. Gastric/duodenal ulceration.
    - iv.
  - 5) Metabolic.
    - i. Chronic liver failure.
    - ii. Hypothyroidism.
    - iii. Diabetes mellitus.
    - iv. Hypoadrenocorticism.
    - v. Hyperadrenocorticism.
    - vi. Hypocalcemia - see diagnostics section for more details.
    - vii. Hypercalcemia – see diagnostics section for more details.
    - viii. Hypergastrinemia – see diagnostics section for more details.
    - ix. Uremia – chronic renal failure.
  - 6) Neoplastic – gastric or enteric.
  - 7) Neurologic.
    - i. Constipation.
    - ii. Gastric or enteric dysrhythmia.
    - iii. Tachygastria – reverse waves.
    - iv. Bradygastria.
  - 8) Trauma – post-GDV, or post chronic gastric dilatation.
- 4. Frequent exposure to things that cause acute gastritis can result in chronic gastritis.

## II. Epidemiology/Signalment

### A. Breed.

- 1. Deep chested dogs – GDV.
- 2. Toy breeds and all size poodles – hypoadrenocorticism.
- 3. Brachycephalic breeds – hiatal hernia, pyloric mucosal hypertrophy.
  - i. Boxer.
  - ii. Pug.
  - iii. Lhasa.
  - iv. Shih Tzu.
  - v. Boston Terrier.
  - vi. Pekingese.
- 4. Bacterial overgrowth – German shepherds.

### B. Age.

- 1. Young adult
  - i. Hiatal hernia.
  - ii. Pyloric mucosal hypertrophy.
  - iii. Pyloric muscular hypertrophy.
- 2. Geriatric – neoplasia (except lymphoma).

### C. Sex.

- 1. Female – hypoadrenocorticism.

### III. History - vomiting.

- A. Distinguish from regurgitation, and from productive cough/gagging up phlegm (see Megaesophagus - III).
- B. Vomiting can occasionally lead to secondary diarrhea.
- C. Weight loss.
  - 1. Nausea is resulting in anorexia.
  - 2. Vomiting results in decreased food passing through the GI tract.
  - 3. Increased caloric demand due to poor digestion, infection, inflammation or neoplasia.
- D. Gastric neoplasia.
  - 1. Anorexia is the most common clinical sign.
  - 2. Followed by weight loss.
  - 3. Then vomiting with time.

### IV. Physical Exam.

- A. Fever.
  - 1. Infection – fungal, bacterial.
  - 2. Neoplasia.
  - 3. Heat stroke.
- B. Assess hydration status.
- C. Shock if:
  - 1. Sepsis.
  - 2. Acute hypoadrenal crisis.
  - 3. Heat stroke.
  - 4. Organ or gut torsion, or other acute abdomen.
  - 5. Mast cell tumor degranulation.
  - 6. Anaphylaxis.
- D. Repeated swallowing should direct you to evaluate for dysphagia/megaesophagus.
- E. Pallor if anemic, hypovolemic, or shocky.
- F. Icterus – prehepatic, hepatic, post-hepatic.
- G. Petechiae if coagulopathy (see section on hematemesis).
- H. Oral hyperemia followed by ulceration if ingestion of caustic substances.
- I. Uremic halitosis if renal failure.
- J. Endocrine alopecia.
  - 1. Hypothyroidism.
  - 2. Hyperadrenocorticism.
  - 3. Diabetes mellitus.
- K. Lung and heart sounds may be muffled if diaphragmatic hernia (may be asymmetric).
- L. Abdominal pain on palpation – gut distension/ileus, abdominal mass, peritonitis, ureteral calculi, pancreatitis, pyelonephritis.
- M. Abdominal distension.
  - 1. Gas – in gut or abdominal – GDV will be tympanic.
  - 2. Fluid – in gut or abdominal.
  - 3. Tissue - mass/organomegaly – neoplasia, infection, inflammation, cyst.
- N. Enlarged colon on abdominal palpation if constipated.
- O. Neurologic exam
  - 1. Various diffuse abnormalities if hepatic encephalopathy.
  - 2. Cranial nerve abnormalities of CNS disease affecting the vomiting center or CRTZ.
  - 3. Normal if limbic epilepsy.
  - 4. Twitching/seizure activity if acute hypocalcemia.
- P. Rectal exam.
  - 1. Bloody stools – see sections on melena and hematemesis.

## V. Diagnosis.

General plan for working up vomiting:

**Step 1 – Minimum database** – to screen for systemic causes of vomiting

- CBC
- General health profile (NOT mini-panel)
- Electrolytes and venous blood gases
- Urinalysis
- Fecal flotation
- T4 and free T4 in cats older than 5 years old
- Coagulation panel if hematemesis and/or seriously ill

You don't have to do ALL of Step 1 on the first visit – start with the tests which will diagnose the diseases highest on the differential list. If more than a few weeks go by, or if the patient's condition worsens, don't hesitate to repeat any or all of these tests.

If all tests in Step 1 are normal, suspect that your patient has primary GI disease, neurologic disease, or neoplasia.

**Step 2 – Imaging.**

- Abdominal radiographs
- Abdominal ultrasound
- Thoracic radiographs – 3 views if looking for metastasis (right lateral, left lateral, VD)
- Consider barium series if any evidence of GI mural disease, malpositioning of GI tract, or motility disorder.
- Consider fluoroscopy if evidence of motility disorder.

If all tests in Step 2 are normal, suspect that your patient has either primary GI disease, neurologic disease, or diffuse neoplasia.

**Step 3 – Flexible endoscopy.**

- Upper and lower GI, with biopsies.
- Consider blood tests such as ACTH stim or gastrin level first, if hematemesis.

If endoscopic exam and biopsies provide no diagnoses, consider that your patient may have disease of the external layers of the gut or abdominal organs, or neurologic disease.

**Step 4 – Exploratory surgery, with biopsies.**

**Step 5 – Empirical treatment** – if all of the above are normal.

- Phenobarbital for limbic epilepsy.
- Cisapride for motility disorder.

**Abnormalities in any test result might lead you directly to other diagnostic tests rather than to the next step.**

A. CBC – often normal in vomiting pets.

1. Anemia.

- a. Always request reticulocyte count if PCV less than 30%.
- b. Regenerative anemia.
  - 1) Signs of regenerative anemia.
    - i. Absolute reticulocyte count (count only aggregates in cats)
      - i. Mild regenerative response.  
--100-300,000 in dogs  
--50-100,000 in cats
      - ii. Moderate regenerative response.  
--300-500,000 in dogs  
--100-200,000 in cats
      - iii. Marked regenerative response.  
-->500,000 in dogs  
-->200,000 in cats
    - ii. Anisocytosis.
    - iii. Polychromasia.
  - 2) Rule out hemolysis, and blood loss from sites other than the GI tract.
  - 3) Consider GI blood loss, especially if both albumin and globulin are low.
  - 4) Do rectal exam and even fecal cytology to look for melena.
- c. Non-regenerative anemia – absolute reticulocyte count 60,000 or less.
  - 1) Anemia of chronic inflammatory disease.
  - 2) Chronic blood loss can eventually become non-regenerative, as iron deficiency anemia develops.
    - i. Microcytosis.
    - ii. Hypochromasia.
  - 3) Acute blood loss can be non-regenerative initially (the first 3-7 days), until the bone marrow has a chance to respond.

2. Polycythemia.

- a. Dehydration/volume contraction – albumin usually high.
- b. HGE – albumin usually normal, due to loss of protein from the gut.
- c. Paraneoplastic (production of EPO like hormone) – globulins often high.

3. Leukopenia – usually neutropenia.

- a. Sepsis – with left shift (greater than 3% bands); toxic neutrophils.
- b. Parvoviral enteritis.
- c. Bone marrow disease.
  - 1) *Histoplasma spp.*
  - 2) Lymphosarcoma or other neoplasia.

4. Leukocytosis.

- a. Neutrophilia.
  - 1) Acute pancreatitis
  - 2) Bacterial enterocolitis, cholangiohepatitis.
  - 3) Systemic bacterial or fungal infection.
  - 4) Neoplasia.
  - 5) Rarely inflammatory bowel disease.
- b. Eosinophilia/basophilia.
  - 1) Parasites (GI and heartworms in cats).
  - 2) Mast cell tumor.
  - 3) Hypereosinophilic syndrome in cats.
  - 4) Hypoadrenocorticism.
- c. Lymphocytosis.

- 1) Chronic infection or inflammation – look for elevated globulins and reactive lymphocytes.
- 2) Viremia.
- 3) Lymphoma – especially if atypical lymphocytes
- 4) Hypoadrenocorticism.
- 5) All pets with lymphocytosis > 10,000 should have blood smear cytology.

**B. Serology:**

1. Serum proteins.
  - a. Low albumin and globulin.
    - 1) Consider protein losing enteropathy (PLE) if HCT normal.
    - 2) Consider blood loss if HCT low.
  - b. low albumin and normal globulin
    - 1) liver disease.
    - 2) PLE.
    - 3) Protein losing nephropathy (PLN).
    - 4) Vasculitis.
  - c. low albumin and high globulin – chronic inflammation/infection.
  - d. High albumin – dehydration or volume contraction.
2. Azotemia.
  - a. High BUN with normal creatinine and phosphorus.
    - 1) Melena or carnivore diet.
    - 2) Early pre -renal azotemia – think hypoadrenocorticism.
    - 3) Early renal disease.
    - 4) Pyelonephritis.
  - b. High BUN, creat, phosphorus.
    - 1) Check urine specific gravity.
      - i. Renal azotemia if isosthenuric.
      - ii. Likely pre -renal if concentrated.
    - 2) Check urine sediment and culture.
3. Liver enzymes.
  - a. Pattern recognition for liver disease:
    - 1) High liver enzymes.
    - 2) High bilirubin with normal PCV.
    - 3) Low albumin.
    - 4) Low glucose.
    - 5) Abnormal cholesterol/triglycerides.
  - b. Any combination of the above, along with liver abnormalities on imaging, indicate fasting and 2 hour post-prandial bile acids,
4. Calcium abnormalities.
  - a. Hypocalcemia.
    - 1) First, correct for hypoalbuminemia.
 

$\text{Corrected calcium} = (3.5 - \text{albumin}) + \text{calcium}$
    - 2) Pancreatitis – consider PLI and abdominal US.
    - 3) Renal disease – consider urine sediment, urine culture and abdominal US.
    - 4) Low vitamin D diet.
    - 5) Ethylene glycol toxicity – consider abdominal US and EG blood test.
    - 6) Intestinal malabsorption – likely will require intestinal biopsy for diagnosis.
    - 7) Primary hypoparathyroidism – consider parathyroid hormone assay.

- b. Hypercalcemia.
  - 1) Growing puppies and kittens can have physiologic hypercalcemia.
  - 2) Lipemia - If sample is lipemic, repeat assay after 12 hour fast. If still lipemic, work up for hyperlipidemia.
  - 3) Renal disease - check serum panel for azotemia.
  - 4) Neoplasia.
    - i. After thorough physical exam, imaging is indicated, followed by endoscopy and/or exploratory surgery.
    - ii. The most common cause of hypercalcemia in the dog and cat is neoplasm.
    - iii. The most common neoplasm in dogs is LSA.
    - iv. Hypercalcemia is most commonly caused by LSA and SCC in the cat.
  - 5) Primary hyperparathyroidism - if imaging and minimum database are negative, consider ionized calcium and parathyroid hormone assay.
  - 6) Hypoadrenocorticism - ACTH stim.
  - 7) Osteolysis - careful orthopedic exam and review of radiographs for bony pain/lysis.
  - 8) Vitamin D overdose, if the owner gives multiple food supplements.
  - 9) Granulomatous disease – fungal infection, mycobacterial infection, inflammatory granulomatous disease. Imaging is indicated.
  - 10) Idiopathic hypercalcemia in the cat – consider if all above test results are normal.
  - 11) No matter the cause of hypercalcemia, check the urinary tract for calcium oxalate stones.

5. Pancreatic assays.

- a. PLI more effective than TLI for testing for acute or chronic pancreatitis.
- b. Make sure you send assay to lab that does species specific test.
  - 1) Antech.
  - 2) GI lab at Texas A&M Small Animal Clinic.
  - 3) Pattern recognition for pancreatitis.
    - i. Abnormal glucose.
    - ii. Low albumin.
    - iii. Low calcium.
    - iv. High amylase and/or lipase.
    - v. High triglycerides.
    - vi. High TLI.
    - vii. High fPLI or cPLI.

6. Hypokalemia.

- a. Increased loss.
  - 1) GI loss – vomiting and diarrhea.
  - 2) Urinary loss – CRF, osmotic diuresis of DKA.
  - 3) Failure to supplement fluid therapy with potassium.
  - 4) Drugs.
    - i. Diuretics.
    - ii. Amphotericin B.
    - iii. Penicillins (rare).
  - 5) mineralocorticoids excess (rare)
    - i. Hyperadrenocorticism.
    - ii. Hyperaldosteronism.

- b. Translocation from extracellular to intracellular fluid.
    - 1) Glucose containing IV fluids.
    - 2) Insulin therapy.
    - 3) Alkalosis, or bicarbonate administration to treat acidosis.
    - 4) Total parenteral nutrition.
    - 5) Hypokalemic periodic paralysis in Burmese cats (rare)
    - 6) Catecholamine release (rare)
  - c. Decreased intake – usually significant only when pet is anorectic, and either increased loss or translocation is occurring.
7. Hyperkalemia.
- a. Hypoadrenocorticism – ACTH stimulation test.
  - b. Oliguric/anuric renal failure – check urine sediment for specific gravity.
  - c. Pseudohyperkalemia.
    - 1) Whipworm infection – check fecal flotation and worm empirically with Panacur 50 mg/kg PO SID x 3 days.
    - 2) Repeatedly drained abdominal effusion.
    - 3) Severe bacterial enterocolitis.
8. Hypochloridemia – if protracted vomiting.
9. Acid-base status.
- a. Acidosis.
    - 1) Lactic acidosis.
      - i. Dehydration.
      - ii. Volume contraction.
      - iii. Sepsis.
    - 2) Uremic acidosis.
    - 3) Loss of bicarbonate from duodenal vomiting.
  - b. Alkalosis.
    - 1) Loss of HCl from vomiting (low chloride also).
    - 2) Gastric outflow obstruction.
  - c. Normal pH.
    - 1) Mild illness.
    - 2) Loss of HCl and bicarbonate can balance each other.

**C. Thyroid hormone assays.**

- 1. T4 and freeT4 in the cat.
  - a. freeT4 alone will give false positives, as freeT4 can be elevated in sick cats.
  - b. Total T4 can be falsely low in hyperthyroid cats who are systemically ill.
  - c. Thyroid scan is the definitive diagnosis for hyperthyroidism.
- 2. TSH, T4 and freeT4 in the dog who has motility disorders – screen for hypothyroidism.
  - a. freeT4 can be falsely elevated by thyroid hormone antibodies, when tested by radioimmunoassay.
  - b. freeT4 by equilibrium dialysis removes the autoantibodies.

**D. Gastrin level – to check for gastrinoma.**

- 1. If evidence of hyperacidity (ulceration, gastroesophageal reflux, etc), especially if unresponsive to H2 blockers and proton pump blockers.
- 2. If pancreatic tumor found on ultrasound.

3. Causes of high gastrin levels:
  - a. Gastrinoma (usually pancreatic tumor).
  - b. Renal failure.
  - c. Chronic gastric distension.
  - d. Chronic administration of proton pump blockers – no negative feedback of HCl produced by gastric mucosa.

#### **E. Urinalysis.**

1. Isosthenuria with azotemia usually indicates renal failure.
2. Bacteriuria indicates UTI and perhaps pyelonephritis.
3. Ketones with glucosuria and hyperglycemia indicate DKA.
4. Ketones can also be caused by prolonged fasting, or starvation.

#### **F. Fecal analysis.**

1. fecal flotation – to look for parasites.
2. fecal wet mount – to look for swimming parasites.
3. fecal cytology – to look for fungal organisms, inflammation, melena.
4. fecal parvo ELISA – if puppy with bloody diarrhea and/or leukopenia.
5. fecal electron microscopy (EM) – for definitive diagnosis if viral enteritis.

#### **G. Toxicology.**

1. blood lead if lead toxicity suspected.
2. organophosphate (OP) toxicology screen if OP toxicity suspected.

#### **H. Radiographs.**

1. Survey abdominal radiographs.
  - a. Radiopaque foreign body.
  - b. Obstruction (loops of dilated bowel).
  - c. GDV – both right and left laterals can be helpful, to locate the pylorus.
  - d. Gastric dilation.
  - e. Pneumoperitoneum.
    - 1) Gut perforation.
    - 2) Peritonitis due to gas producing bacteria.
    - 3) Perforation of vagina or uterus.
    - 4) Abdominal wall perforation.
  - f. Abdominal fluid.
    - 1) Transudate from end stage liver or portal hypertension.
    - 2) Neoplastic effusion – modified transudate.
    - 3) Exudate due to peritonitis.
    - 4) Bleeding.
    - 5) Pancreatitis.
    - 6) Ruptured biliary tract.
    - 7) Chyloabdomen.
    - 8) Uroabdomen.
2. Barium studies.
  - a. Procedure.
    - 1) Avoid drugs that will affect GI motility.
      - i. Acepromazine has been reported to affect GI motility, but Todd Tams recommends its use for barium study.
      - ii. Anticholinergics – atropine, aminopentamide (Centrine).
      - iii. Beta agonists – bronchodilators (terbutaline).
      - iv. Opiates.



- 2) Shoot scout films.
    - i. 2 views – lateral and VD/DV.
    - ii. Evaluate for obvious abnormalities.
    - iii. Get exposure right before barium is administered, and time is of the essence for the first 3 films.
  - 3) Administer barium PO.
    - i. 4-6 ml/lb for small dogs and cats.
    - ii. 2-4 ml/lb for large dogs.
    - iii. May need to pass an orogastric tube.
  - 4) 5 minutes
    - i. right lateral
    - ii. left lateral
    - iii. VD or DV.
  - 5) 30 minutes – same 3 films as 5 minutes.
  - 6) Every hour until bariums is completely gone from the stomach.
    - i. Lateral
    - ii. VD or DV.
- b. Things that can be better seen on barium series than survey films.
- 1) Filling defect if radiolucent foreign body is present.
  - 2) Filling defect if mural mass.
  - 3) Outward extension of barium if deep ulcer.
  - 4) Barium will adhere and stay behind in a thin layer if erosive/ulcerative disease. LOOK HERE when you ultrasound, scope or do surgery.
  - 5) Can better assess gut wall thickness.
- c. Assessment of motility.
- 1) Liquid barium should be in the duodenum within 20 minutes.
  - 2) If there is no barium in the duodenum within 30 minutes:
    - i. Look for pyloric outflow obstruction.
    - ii. Consider motility disorder.
  - 3) Stomach should be nearly empty of liquid barium within 3-4 hours.
  - 4) Barium coated food can remain in the stomach as long as 12-15 hours in normal dogs and cats. However, most of the food should be gone within 8-10 hours.
3. BIPs – barium impregnated polyethylene spheres.
- a. Commercially available barium markers given PO.
    - 1) Large spheres – 5mm – detect GI obstruction.
    - 2) Small spheres – 1.5mm - detect transit times.
  - b. Procedure.
    - 1) Capsules given with a canned meal which is 25% of the daily intake.
    - 2) Take rads every 4 hours for 12-24 hours.
    - 3) Manufacturer provides normal reference ranges.
4. Thoracic radiographs – three views, if looking for metastatic neoplasia.
- a. Right lateral.
  - b. Left lateral.
  - c. DV or VD.

**I. Fluoroscopy.**

1. Can see real time motility with liquid barium, to measure
  - a. Transit times.
  - b. Gastric arrhythmia.
    - 1) Bradygastria (liquid barium).
    - 2) Tachygastria (liquid barium).
    - 3) Gastric dysrhythmia (barium coated meal).
  - c. Retrograde transit.
    - 1) Nasopharyngeal reflux.
    - 2) Gastroesophageal reflux.
    - 3) Delayed gastric emptying.
    - 4) Duodenogastric reflux.
2. Especially helpful for chronic bloating, and chronic vomiting.

**J. Nuclear Scintigraphy** – can also be used to assess transit times.

**K. Abdominal Ultrasound.**

1. Can see:
  - a. Increased gut wall thickness.
  - b. Obliteration of gut wall layers (neoplasia, fungal infiltration.
  - c. GI foreign body (if not surrounded by air.
  - d. May see GI ulcer.
  - e. Organ disease causing vomiting.
    - 1) Neoplasia.
    - 2) Pancreatitis.
    - 3) Liver disease.
    - 4) Kidney disease,
  - f. Limited ability to image the stomach due to air.

**L. Flexible Endoscopy.**

1. Preparation.
  - a. Withhold food, barium and sucralfate for 24 hours prior to upper GI endoscopy.
  - b. Withhold the same for 48 hours for lower GI endoscopy.
  - c. Do not withhold water, until the morning of endoscopy.
  - d. Give bisacodyl 5 mg PO 24 hours prior to lower GI endoscopy.
  - e. Enemas 24, 12, and 1-2 hours prior to lower GI endoscopy.
  - f. Some use GoLytely or another similar colon evacuant prior to lower GI endoscopy.
2. Visualizes mucosal surface of the esophagus, stomach, duodenum, ileum and colon.
3. Can not visualize pathology beneath the submucosa (neoplasia).
4. Allows biopsies of the GI tract.
  - a. Take biopsies even if gut appears normal.
  - b. Pathology which can appear normal on the surface.
    - 1) Inflammatory bowel disease.
    - 2) Lymphosarcoma.
    - 3) Histoplasmosis.
    - 4) Maybe mast cell tumor.
  - c. Make sure biopsies are flattened out on a sponge or tongue depressor prior to putting them in the formalin, to keep them from rolling up.
  - d. If gastritis, putting a biopsy into urease agar can give you diagnosis of *Helicobacter* within 14 hours (CLO test).
  - e. Keep at least 1 biopsy from each area for cytology (new methylene blue for *Helicobacter spp.*).
  - f. Request Warthin-Starry silver stain for *Helicobacter spp.*
5. Allows removal of some foreign bodies.

6. Neoplasia often appears as:
  - a. Discolored, thickened mucosa.
  - b. Raised plaques.
  - c. Ulceration with thick margins – take biopsy at edge.
  - d. Raised masses.
  - e. Bleeding.
  - f. Scirrhous adenocarcinoma can be hard to detect – may appear as diffuse thickening of the wall.

#### **M. Exploratory Laparotomy.**

1. Test coagulation status first – at least BMBT.
2. Incision from the manubrium to the umbilicus.
3. Visualize every organ.
  - a. Liver, gallbladder.
  - b. Stomach.
  - c. Pancreas.
  - d. Spleen.
  - e. Duodenum, jejunum, ileum, colon.
  - f. Mesenteric and sublumbar lymph nodes.
  - g. Both kidneys (look at cranial pole for enlarged adrenals).
  - h. Ureters.
  - i. Urinary bladder.
  - j. Reproductive organs if present (including cryptorchid testicles).
4. Run the entire gut between your fingers, looking for evidence of obstruction or pathology.
5. Take Biopsies.
  - a. Always for vomiting patients:
    - 1) Biopsy:
      - i. Liver.
      - ii. Stomach, duodenum, jejunum, ileum (full thickness biopsies are an advantage).
      - iii. Mesenteric lymph node.
      - iv. Pancreas (cats).
    - 2) Aspiration cytology of spleen.
  - b. If abnormal:
    - 1) Aspirate contents from gall bladder for culture.
    - 2) Biopsy any grossly abnormal organ.
    - 3) Biopsy spleen or do splenectomy if cytology abnormal.
    - 4) Biopsy of adrenal gland can cause severe bleeding.
6. Electrogastrogram can be done intraoperatively, to assess motility.

## **VI. Treatment**

### **A. Empiric/Symptomatic/Supportive therapy.**

1. Nothing Per Os (NPO).
  - a. Until 12-24 hours with no vomiting.
  - b. Then water only for 12-24 hours.
  - c. Then offer food (see Nutritional Therapy below).
2. Fluid therapy.
  - a. Treat and prevent dehydration.
  - b. Support mucosal perfusion.

3. Dewormers.
  - a. Physaloptera - Pyrantel pamoate (Strongid T) 1 cc per 10 pounds PO SID x 1-5 days; repeat in 2-3 weeks.
  - b. Ollulanus - Fenbendazole (Panacur) 50 mg/kg PO SID x 3-5 days.
4. A few doses of antiemetics (see below).

**B. Treat the underlying cause.**

1. Renal disease.
2. Liver disease.
3. Endocrine disease.
4. Neoplasia.
5. Infectious Disease.
6. Surgical disease.
7. *Helicobacter* gastritis.
  - a. Treatment with a single antibiotic is seldom effective, because the bacteria rapidly develops resistance.
  - b. 14 days of treatment is usually sufficient.
  - c. Combination therapy is more often effective (many choices).
    - 1) Dual therapy - Omeprazole and an antibiotic is a favorite choice.
      - a) Metronidazole 5 mg/lb PO BID-TID.
      - b) Amoxicillin 10 mg/lb PO BID-TID.
      - c) Tetracycline 10 mg/lb PO TID.
      - d) Azithromycin 2.5 mg/lb PO SID for cats and 5 mg/lb PO SID for dogs, for 7 days rather than 14 (continue omeprazole for 14).
      - e) See proton pump blockers above for information on omeprazole.
    - 2) Triple Therapy - Bismuth subsalicylate, an antacid and an antibiotic.
      - a) Bismuth has antimicrobial activity against *Helicobacter spp.*
    - 3) Some prefer to use 2 antibiotics simultaneously, but I have not found it necessary.
8. Hemorrhagic gastroenteritis.
  - a. IV fluids (shock bolus, followed by 2x maintenance, unless there is heart disease).
  - b. Antibiotics are controversial, but I use them – these dogs seem septic.
    - 1) I use IV ampicillin (10 mg/lb IV TID-QID) for 1-2 days, then PO (10 mg/lb PO BID) for 14 days.
    - 2) Some like metronidazole or Tylan.
  - c. Sucralfate 0.5g/15 lbs PO BID-TID if hematemesis/melena is severe.
  - d. Antiemetics as needed.
  - e. Treat sequelae of sepsis/shock – renal failure, DIC, skin slough, etc.

**C. Antacids.**

1. H2 blockers.
  - histamine stimulates parietal cells to secrete HCl.
  - if you block H2, parietal cells become less responsive to gastrin and acetylcholine.
  - used indefinitely for gastrinomas that are not cured by surgery.
  - used for 14 days after hematemesis or melena resolves.
  - a. Cimetidine (Tagamet) 2.5-5 mg/lb PO IM IV TID-QID.
    - 1) Inhibits hepatic microsomal enzymes.
    - 2) May increase half life of drugs that are metabolized in the liver – theophylline, warfarin, phenobarbital.
    - 3) Can cause mental depression.

- b. Ranitidine (Zantac) 1 mg/lb PO SQ IM IV BID-TID.
  - 1) 5x as potent as cimetidine.
  - 2) Also a prokinetic, by inhibiting acetylcholinesterase.
    - a) Hastens gastric emptying.
    - b) Decreased gastroesophageal and Duodenogastric reflux.
  - 3) Inhibits hepatic microsomal enzymes as cimetidine, but to a lesser extent.
- c. Famotidine (Pepcid) 0.25-0.5 mg/lb PO IV SID-BID.
  - 1) Inhibits hepatic microsomal enzymes as cimetidine, but to a lesser extent.
  - 2) 20x as potent as cimetidine.
- d. Nizatidine (Axid) 1.25-2.5 mg/lb PO SID.
  - 1) 5x as potent as cimetidine.
  - 2) Also a prokinetic.

2. Proton pump blockers.

- a. Actions:
  - 1) For severe ulcerative disease, or if response to H2 blockers is inadequate.
  - 2) More effective than H2 blockers for mast cell degranulation.
  - 3) Stronger suppressors of gastric acid secretion than H2 blockers.
  - 4) Block  $H^+-K^+$ -ATPase (proton pump used to secrete HCl).
  - 5) Promotes gastric healing if gastritis.
  - 6) Diminishes proteolytic effect of pepsin.
  - 7) Maximum effect at the 5<sup>th</sup> dose (may need to use with H2 blockers for the first 3-4 days).
  - 8) Prolonged use (greater than 4 weeks) can cause reversible gastric mucosal hypertrophy.
  - 9) Rebound hypersecretion of HCl can occur if stopped abruptly (high gastrin levels due to lack of feedback).
- b. Drugs:
  - 1) Omeprazole (Prilosec).
    - i. 5 mg (1/2 capsule) PO SID, for dogs less than 11 lbs.
    - ii. 10 mg PO SID, for dogs 11-45 lbs.
    - iii. 20 mg PO SID, for dogs greater than 45 lbs.
  - 2) Lansoprazole (Prevacid).
    - i. 15 mg PO SID for small dogs
    - ii. 30 mg PO SID for large dogs.
  - 3) Esomeprazole (Nexium)
    - i. 0.7 mg/kg PO SID for dogs.
    - ii. Granules in capsule inactivated if sprinkled on food.
  - 5) Pantoprazole (Protonix) – 10-40 mg PO SID; 1 mg/kg IV SID.
  - 6) Rabeprazole (Aciphex) – 5-20 mg PO SID.

**N. Antiemetics.**

- 1. Use should be short term, for patient comfort, and to prevent fluid and electrolyte losses, while the underlying cause of the vomiting is being corrected.
- 2. Central antiemetics.
  - a. Phenothiazines.
    - 1) Act at both the CRTZ and the vomiting center.
    - 2) Use only in well hydrated patients, without low blood pressure, as they are hypotensives.
    - 3) Prochlorperazine (Compazine) 0.25 mg/lb SQ IM TID.
    - 4) Chlorpromazine (Thorazine) 0.15-0.25 mg/lb SQ TID.
  - b. Antihistamines.
    - 1) Act at the CRTZ.
    - 2) Diphenhydramine (Benadryl) 0.5-2 mg/lb PO IM or SLOWLY IV.

- 3) Dimenhydrinate (Dramamine) 2-4 mg/lb PO TID.
  - 4) Meclizine (Antivert) 12.5 mg PO SID for small dogs and cats; 25 mg PO SID for medium to large dogs.
  - c. Anticholinergics.
    - 1) Scopolamine (Hyoscine) 0.02 mg/lb SQ IM QID.
    - 2) Acts at vestibular center and CRTZ.
    - 3) Side effects ileus, dry mouth, sedation.
  - d. Yohimbine (Yobine).
    - 1) Acts at the CRTZ and the vomiting center.
    - 2) 0.15-0.25 mg/lb SQ IM BID.
3. Peripheral antiemetics.
- a. Cisapride (Propulsid).
    - 1) Antiemetic and prokinetic.
    - 2) Acts peripherally on the GI tissue – does not cross the blood brain barrier, so no associated extrapyramidal side effects.
    - 3) 0.05-0.25 mg/lb PO TID.
  - b. Anticholinergics.
    - 1) Aminopentamide (Centrine) 0.1-0.4 mg IM SQ BID-TID.
    - 2) Side effect – ileus (undesirable when there is ileus or motility disorder).
4. Antiemetics that act peripherally and centrally.
- a. Metoclopramide (Reglan).
    - 1) Antidopaminergic and antihistaminic.
    - 2) Antiemetic as well as prokinetic.
    - 3) Acts at the CRTZ to inhibit nausea and vomiting.
    - 4) 0.2-0.4 mg/kg PO, SQ, IV TID-QID.
    - 5) CRI – 0.5-1 mg/lb/day IV.
    - 6) Reduce dose by 50% in pets with RF.
    - 7) Side effects hyperactivity and constipation (extrapyramidal signs).
    - 8) Side effects more common in the cat.
    - 9) For severe metoclopramide side effects, give Benadryl.
    - 10) Because serotonin receptors dominate in the feline CRTZ rather than dopamine, metoclopramide may not work as well as an antiemetic in cats, when compared to dogs.
  - b. 5HT antagonists.
    - 1) Block vagal afferent neurons, and act at the CRTZ.
    - 2) Ondansetron (Zofran) 0.05-0.15 mg/lb PO or slowly IV SID-TID.
    - 3) Dolasetron (Anzemet) 0.4-0.6 mg IV SID-BID.
    - 4) Side effects sedation and head shaking.

#### **O. Prokinetics.**

1. Reduce gastroesophageal reflux.
2. Help control vomiting by accelerating gastric emptying.
3. Improve coordination of antrum, pylorus and duodenum.
4. Increases propagation distance of peristaltic waves.
5. Contraindicated in cases with obstruction (can precipitate perforation).
6. Metoclopramide (see antiemetics).
7. Cisapride (see antiemetics).
  - a. A more effective prokinetic than metoclopramide.
  - b. Works on entire GI tract, unlike metoclopramide that works only on the stomach and proximal small intestine.
8. Ranitidine (see H2 blocker antacids).
9. Nizatidine (see H2 blocker antacids).
10. Erythromycin 0.25-0.5 mg/lb PO TID (dose lower than for antimicrobial therapy).
11. Can usually wean prokinetics to the lowest effective dose.

**P. Cytoprotective and mucosal protectant Agents.**

1. Especially important with ulcerative/erosive disease.
2. Cytoprotective agents:
  - a. Bind to the ulcer/erosion to create a physical protective barrier.
  - b. Inactivate pepsin.
  - c. Adsorb bile acids, which can be inflammatory.
  - d. Sucralfate (Carafate) 0.5 g/15 lb PO BID-QID.
  - e. Barium sulfate 2-6 ml/lb PO (same as for upper GI series).
  - f. Side effect constipation.
3. Mucosal protectants – prostaglandin analogs.
  - a. Increase mucosal mucus and bicarbonate production.
  - b. Decrease mucosal acid production.
  - c. Promote mucosal blood flow.
  - d. Indicated for NSAID gastritis.
  - e. Misoprostyl (Cytotec) 1-2.5 ug/lb PO TID.
  - f. Side effects include:
    - 1) Abdominal cramping.
    - 2) Vomiting, diarrhea.
    - 3) Abortion.

**Q. Nutrition.**

1. “3-5-7 Rule”:
  - a. At 3 days without food intake, you should be thinking about how you will get nutrition into the patient within the next day or two.
  - b. At 5 days without food intake, you should be supplementing nutrition enterally or parenterally.
  - c. At 7 days without food intake, you are already “behind the eight ball.”
  - d. Enteral feeding is preferred if vomiting can be managed, and the dog is not at risk for exacerbating pancreatitis.
  - e. Parenteral can be used until vomiting can be managed.
  - f. See section on Tube Feeding for more information.
2. Reintroducing food.
  - a. Support hydration with fluid therapy until water intake supports hydration.
  - b. Offer water PO after no vomiting for 12-24 hours.
  - c. Offer food after 12-24 hours of no vomiting with free choice water.
  - d. 3-4 small (1/8 of daily intake per meal) meals the first day.
  - e. Diet:
    - 1) Low fat, low fiber food will leave the stomach fastest.
    - 2) Use the dog’s regular diet if suitable.
    - 3) Hill’s I/D.
    - 4) Purina EN.
    - 5) Home made 50/50 chicken or low fat beef with white rice or pasta.
  - f. Gradually work up to full on a regular schedule feed over 3-5 days.
3. Novel antigen food.
  - a. Consider if food allergy is suspected.
  - b. Minimum STRICT trial of 8 weeks.
  - c. Diets:
    - 1) Home made – 50/50 novel meat and novel carbohydrate for dogs: 80/20 or 100% protein for cats.
    - 2) Many commercial limited antigen diets available.

- 3) Hydrolyzed diets (some with chicken allergies still don't tolerate).
  - 4) Dogs with allergies to beef often also are allergic to venison.
  - 5) Beef, Dairy, Corn and Wheat are the most common allergies.
- d. If elimination diet is successful, reintroducing new foods, no more than 1 per two weeks, and isolate the source of the problem. Make sure clinical signs are well controlled before introducing a new food.

**R. Antibiotics.**

1. for *Helicobacter* gastritis (see above).
2. If chronic gastric dilatation due to bacterial overgrowth (see section on Diarrhea).
3. If sepsis or systemic infection.

**S. Immunosuppression.**

1. For inflammatory bowel disease (see section on Diarrhea for more information).
2. There seems to be a continuum between IBD and LSA.
  - a. Some dogs and cats with biopsy IBD will not respond to therapy, and will ultimately have LSA.
  - b. Some dogs and cats with histopathologic diagnosis of LSA will go into remission for many years (5-10) with immunosuppressive therapy, seemingly having IBD.
  - c. 15% of histopathologic diagnoses in human medicine are ultimately incorrect.
3. Drugs.
  - a. Prednisone.
    - 1) Dogs:
      - i. 0.5-1 mg/lb/day for 7-14 days.
      - ii. Then wean slowly to lowest effective dose over 2-3 months.
    - 2) Cats:
      - i. 1-2 mg/lb/day for 7-14 days.
      - ii. Then wean slowly to lowest effective dose over 2-3 months.
    - 3) try dexamethasone if prednisone doesn't work or stops working – 0.1 mg/kg PO SID to QID.
  - b. Azathioprine (Imuran)
    - 1) Usually not needed for eosinophilic inflammation.
    - 2) Use if lymphoplasmacytic inflammation is particularly severe, or if there is inadequate response to pred alone.
    - 3) Do not use in dogs who have had pancreatitis (give careful consideration to administration to Schnauzers).
    - 4) Dogs:
      - i. 0.5-1 mg/lb SID x 2 weeks.
      - ii. Then QOD long term.
      - iii. Wean to lowest effective dose over 2-3 months.
    - 5) Cats - 0.1-0.2 mg/kg PO QOD.
    - 6) Monitoring:
      - i. CBC:
        - Days 0, 14, 28.
        - Then monthly for 3 months.
        - Then 3-4 times per year.
      - ii. Liver enzymes:
        - Days 0, 30.
        - Then 3-4 times per year long term.



- c. Alternating pred and Imuran QOD can sometimes work well.
- d. May be able to wean off immunosuppressives after 8 weeks of novel antigen diet, if food allergy is causing IBD..

**T. Transfusion.**

- 1. fresh whole blood if bleeding into GI tract is severe.

**U. Surgery.**

- 1. Resectable masses can be removed.
  - a. Gastrinomas can be cured if there is no metastasis.
  - b. Fibromas and leiomyomas/sarcomas are also potentially resectable.
  - c. Large eosinophilic granulomas may need to be removed.
  - d. Single LSA masses may need to be removed prior to chemo to eliminate obstruction.
  - e. Cats with splenic mast cell tumor can do potentially do quite well for long periods of time, with splenectomy and chemotherapy.
- 2. Foreign bodies removed.
- 3. Torsions corrected.
- 4. Pyloromyotomy for pyloric muscular hypertrophy.
- 5. “Y to U” Pyloroplasty and mucosal resection for pyloric mucosal hypertrophy.
- 6. Ulcer resection may be necessary if bleeding can not be controlled.
- 7. Peritonitis.
  - a. Some cases of local peritonitis are better managed with local drainage and open abdomen, rather than laparotomy.
  - b. Stirring up a walled off abscess can cause acute death.

**V. Treatment for limbic epilepsy** – phenobarbital rather than KBr, as KBr is a GI irritant.

**VII. Monitoring**

- A. For immunosuppressive therapy – see therapy section above.

**VIII. Sequella/Prognosis**

**A. Sequellae.**

- 1. HGE – DIC, renal failure, SIRS.
- 2. Chronic enteritis may lead to “leaky gut” and development of systemic allergies.
- 3. Repeated bouts of acute gastritis can lead to chronic gastritis.

**B. Prognosis.**

- 1. Excellent for acute gastritis and most causes of chronic gastritis.
- 2. Potentially good to IBD, but some severe cases become unmanageable, or could progress to LSA.

**IX. Public Health Significance.**

- A. Histoplasmosis, mycobacterial infections, and Prototheca potentially communicable to immunocompromised owners.
- B. Giardia, Trichomonas, Cryptosporidium and a number of enteric bacteria potentially transmissible from dogs and cats to people.
- C. Helicobacter has been documented to pass from people to pets in some cases, but not vice versa yet.
- D. Ascarids and hookworms can cause visceral and cutaneous larval migrans in people.