PART THREE ▪ Digestive System Disorders

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CHAPTER 26

Clinical Manifestations of Gastrointestinal Disorders

DYSPHAGIA, HALITOSIS, AND DROOLING

Dysphagia, halitosis, and drooling often coexists in animals with oral disease. Dysphagia (i.e., difficulty in eating) usually results from oral pain, masses, foreign objects, trauma, neuromuscular dysfunction, or a combination of these (Box 26.1). Halitosis typically signifies an abnormal bacterial proliferation secondary to tissue necrosis, tartar, periodontitis, or oral/esophageal retention of food (Box 26.2). Drooling occurs because animals are unable or unwilling to swallow (i.e., pseudoptyalism). Excessive salivation is often due to nausea; animals that are not nauseated rarely produce excessive saliva (Box 26.3). Although any disease causing dysphagia may have an acute onset, clinicians usually should first consider foreign objects or trauma as the cause in acutely dysphagic patients. The environmental and vaccination history should always be assessed to determine whether rabies is a reasonable possibility.

The next step is a thorough oral, laryngeal, and cranial examination. This examination is often the most important diagnostic step because most problems producing oral pain can be partially or completely defined at physical examination. Ideally, this is done without chemical restraint to facilitate detection of pain. However, many animals must be anesthetized for an adequate oral examination to search for anatomic abnormalities, inflammatory lesions, pain, and discomfort. If pain is found, the question is whether it occurs when the mouth is opened (e.g., retrobulbar inflammation), is associated with extraoral structures (e.g., muscles of mastication), or originates from the oral cavity. The clinician must search for fractures, lacerations, crepitus, masses, enlarged lymph nodes, inflamed or ulcerated areas, draining tracts, loose teeth, excessive temporal muscle atrophy, inability to open the mouth while the animal is under anesthesia, and ocular problems (e.g., proptosis of the eye, inflammation, or strabismus suggestive of retrobulbar disease). If oral pain is apparent but cannot be localized, retrobulbar lesions, temporomandibular joint disease, and posterior pharyngeal lesions should be considered. A concurrent clinicopathologic evaluation may be useful, especially if oral examination findings indicate systemic disease (e.g., lingual necrosis resulting from uremia, chronic infection secondary to hyperadrenocorticism).

Mucosal lesions (e.g., masses, inflamed or ulcerated areas) and painful muscles of mastication should be biopsied. Masses that do not disrupt the mucosa, especially those on the midline and dorsal to the larynx, can be difficult to discern and are often best found by digital palpation. Fine-needle aspiration and cytologic evaluation are reasonable first steps for diagnosing masses. Remember that fine-needle aspirates can only find disease; they do not exclude disease (i.e., they are not sensitive tests). Subtle masses or those dorsal to the larynx are often best aspirated with ultrasonographic guidance. Multiple aspirations are usually done before a wedge or punch biopsy is performed.

Incisional biopsy specimens must include generous amounts of submucosal tissues. Many oral tumors cannot be diagnosed with superficial biopsy specimens because normal oral flora cause superficial necrosis and inflammation obscuring the lesion. Clinicians are often afraid to biopsy aggressively because these lesions bleed profusely and are hard to suture. The clinician should avoid major vessels (e.g., the palatine artery) and use silver nitrate to stop hemorrhage. It is better to have difficulty stopping hemorrhage after obtaining an adequate biopsy specimen than to have less difficulty stopping hemorrhage after obtaining a nondiagnostic specimen. If diffuse oral mucosal lesions are noted, search carefully for vesicles (e.g., pemphigus); if found, remove them intact for histopathologic and immunofluorescent studies. If vesicles are not found, then at least two or three tissue samples representing a spectrum of new and old lesions should be obtained.

BOX 26.1

Causes of Dysphagia

Oral Pain

Fractured bones or teeth
Trauma
Periodontitis or caries (especially cats)
Mandibular or maxillary osteomyelitis
Other causes
  Retrobulbar abscess/inflammation
  Various other abscesses or granulomas of the oral cavity
  Temporal-masseter myositis
Stomatitis, glossitis, pharyngitis, gingivitis, tonsillitis, or sialoadenitis
  Immune-mediated disease
  Feline viral rhinotracheitis, calicivirus, leukemia virus, or immunodeficiency virus
  Lingual foreign objects, other foreign objects, or granulomas
  Tooth root abscess
  Uremia
  Electrical cord burn
  Miscellaneous causes
  - Thallium
  - Caustics
Pain associated with swallowing: esophageal stricture or esophagitis

Oral Mass

Tumor (malignant or benign)
Eosinophilic granuloma
Foreign object (oral, pharyngeal, or laryngeal)
Retropharyngeal lymphadenomegaly
Inflammatory polyp of middle ear (primarily cats)
Sialocele

Oral Trauma

Fractured bones (e.g., mandible, maxilla)
Soft tissue laceration
Hematoma

Neuromuscular Disease

Localized myasthenia
Temporal-masseter myositis
Temporomandibular joint disease
Oral, pharyngeal, or cricopharyngeal dysfunction
  Cricopharyngeal achalasia
Tick paralysis
Rabies
Tetanus
Botulism
Various cranial nerve dysfunctions/central nervous system disease

BOX 26.2

Causes of Halitosis

Bacterial Causes

Food retained in the mouth
  Anatomic defect allowing retention (exposed tooth roots, tumor, large ulcer)
  Neuromuscular defect allowing retention (pharyngeal dysphagia)
Food retained in the esophagus
Tartar or periodontitis
Damaged oral tissue
  Neoplasia/granuloma of mouth or esophagus
  Severe stomatitis/glossitis

Eating Noxious Substances

Necrotic or odoriferous food
Feces

BOX 26.3

Major Causes of Drooling

Ptyalism

Nausea
Hepatic encephalopathy (especially feline)
Seizure activity
Chemical or toxic stimulation of salivation (organophosphates, caustics, bitter drugs [e.g., atropine, metronidazole])
Behavior
Hyperthermia
Salivary gland hypersecretion

Pseudoptyalism

Oral pain, especially stomatitis, glossitis, gingivitis, pharyngitis, tonsillitis, or sialoadenitis (see Box 26.1)
Oral or pharyngeal dysphagia (see Box 26.1)
Facial nerve paralysis

If oral examination findings are not helpful, plain oral and laryngeal radiographs are usually the best next steps. Oral cultures are rarely helpful because normal oral flora makes interpretation of results difficult. Even animals with severe halitosis or stomatitis secondary to bacterial infection rarely benefit from bacterial culture, unless there is a draining tract or abscess.

Halitosis often accompanies dysphagia, in which case it is usually more productive to determine the cause of the dysphagia. If halitosis occurs without dysphagia, the clinician should first be sure that the odor is abnormal and then check for the ingestion of odoriferous substances (e.g., feces). A thorough oral examination is still the most important test. Halitosis not attributable to an oropharyngeal lesion may be

originating from the esophagus. Radiographs (plain and/or contrast) or esophagoscopy may reveal a tumor or retained food secondary to stricture or weakness. If the history and oral examination are unrevealing except for mild to moderate tartar accumulation, the teeth should be cleaned to try to resolve the problem.

Drooling is usually caused by nausea, oral pain, or dysphagia. The approach to the diagnosis of oral pain and dysphagia is described under the appropriate headings. Nausea is considered in the section on vomiting.

Dysphagic animals without demonstrable lesions or pain may have neuromuscular disease. Dysphagia of muscular origin usually results from atrophic myositis (see Chapter 67). Finding swollen, painful temporal muscles suggests acute myositis. Finding severe temporal-masseter muscle atrophy plus difficulty opening the mouth (even when the animal is anesthetized) suggests chronic temporal-masseter myositis. Biopsy of affected muscles is indicated. It is critical that muscle tissue be retrieved; it is easy to obtain only fibrous scar tissue. A positive test for antibodies to type 2M muscle fibers is consistent with masticatory muscle myositis but not polymyopathy.

Neurogenic dysphagia is caused by disorders in the oral (i.e., also called *prehensile*), pharyngeal, or cricopharyngeal phases of swallowing (disorders of the latter two phases are discussed in the section on regurgitation). Rabies should always be considered, despite its relative rarity. After rabies is presumptively ruled out, cranial nerve deficits (especially deficits of cranial nerves V, VII, IX, XII) should be considered. Because clinical signs vary depending on the nerve (or nerves) affected, a careful neurologic examination is indicated.

Inability to pick up food or having food drop from the mouth while eating usually indicates a prehensile disorder. Dysphagia may be noticeable in dogs and cats with pharyngeal and cricopharyngeal dysfunction, but regurgitation is often more prominent. Dynamic contrast-enhanced cinefluoroscopy or fluoroscopy is best for detecting and defining neuromuscular dysphagia. Localized myasthenia is an important cause of pharyngeal dysphagia and should be ruled out with serology. If neuromuscular problems are seemingly ruled out by imaging and serology, then anatomic lesions and occult causes of pain (e.g., soft tissue inflammation or infection) must be reconsidered.

DISTINGUISHING REGURGITATION FROM VOMITING FROM EXPECTORATION

Regurgitation is expulsion of material (i.e., food, water, saliva) from the mouth, pharynx, or esophagus and must be differentiated from vomiting (expulsion of material from the stomach and/or intestines) and expectoration (expulsion of material from the respiratory tract). Historical and physical examination findings sometimes allow differentiation (Table 26.1). Expectoration is generally associated with coughing at the time of the event. However, because dogs that cough and gag excessively may stimulate themselves to vomit as well, careful history taking is important. Animals that regurgitate and some that vomit may cough due to aspiration.

TABLE 26.1

Aids to Differentiate Regurgitation From Vomiting*

SIGN	REGURGITATION	VOMITING
Prodromal nausea†	No	Usually
Retching‡	No	Usually
Material produced		
Food	±	±
Bile	No	±
Blood	± (undigested)	± (digested or undigested)
Amount of material	Any amount	Any amount
Time relative to eating	Anytime	Anytime
Distention of cervical esophagus	Rare	No
Dipstick analysis of material		
pH	≥7	≤5 or ≥8
Bile	No	±

*These are *guidelines* that often help distinguish vomiting from regurgitation. However, occasional animals will require plain and/or contrast-enhanced radiographs to distinguish between the two. In particular, animals that are vomiting may appear to be regurgitating. The reverse is less common.
†May include salivation, licking lips, pacing, and an anxious expression. The owner may simply state that the animal is aware that it will soon "vomit."
‡These are usually forceful, vigorous abdominal contractions or dry heaves. This is not to be confused with gagging, which is common in regurgitation.

The criteria in Table 26.1 are guidelines. Some animals that appear to be regurgitating are vomiting and vice versa. In particularly severe cases of esophageal weakness, one may see the cervical esophagus balloon in and out during respiration (Video 26.1). If the clinician cannot distinguish between two based on history and physical examination findings, a urine dipstick may be used to determine pH and whether there is bilirubin in *freshly* "vomited" material. If the pH is 5 or less, the material is probably gastric in origin, resulting from vomiting. A pH of 7 or greater without evidence of bilirubin is most consistent with regurgitation. Finding bilirubin means the material is duodenal in origin (i.e., vomiting). Finding blood in the urine dipstick test is not helpful.

If vomiting and regurgitation still cannot be distinguished, plain thoracic radiographs ± a barium-contrast esophagram will detect most esophageal lesions. However, some esophageal disorders (e.g., hiatal hernia, partial stricture, segmental motility defects) are easily missed unless a careful radiographic technique and/or fluoroscopy is used. Endoscopy is sometimes required to detect esophageal lesions missed by imaging (e.g., esophagitis).

REGURGITATION

If regurgitation is confirmed, the disease should be localized to the oropharynx or esophagus (Fig. 26.1). History or observing the pet eating should allow the clinician to detect dysphagia (e.g., undue stretching or flexing of the neck during swallowing, repeated efforts at swallowing, food falling from the mouth during swallowing). Some animals with dysphagia associated with neuromuscular disorders have more difficulty swallowing liquids than solid foods, probably because it is easier to aspirate liquids. Oropharyngeal dysphagic animals in particular often cough when swallowing water.

If a regurgitating animal is dysphagic, then oral, pharyngeal, and cricopharyngeal dysfunctions must be considered; the latter two clinically mimic each other. Fluoroscopic evaluation of swallowing a barium meal is necessary to differentiate pharyngeal from cricopharyngeal dysfunction. If they are not accurately differentiated, inappropriate therapy may cause morbidity or mortality.

Two main reasons for esophageal regurgitation are obstruction and muscular weakness. Plain thoracic radiographs are the initial step for defining these problems. Barium-contrast esophagrams are often necessary because plain films do not detect many esophageal lesions. Using liquid barium sulfate can miss partial strictures, but mixing barium with canned food or kibble typically reveals these lesions. Fluoroscopy may be necessary to detect partial loss of peristalsis, segmental aperistalsis, gastroesophageal reflux, or sliding hiatal hernias. Sometimes the lower esophageal sphincter must be fluoroscopically observed for several minutes to detect frequency and severity of gastroesophageal reflux (normal animals may show occasional reflux). If the animal seems to be regurgitating but contrast-enhanced radiographs fail to reveal esophageal dysfunction, either the assessment of regurgitation is wrong or there is occult esophageal disease requiring esophagoscopy for diagnosis (e.g., esophagitis, gastroesophageal reflux).

Esophageal obstruction is principally caused by foreign objects, vascular anomalies, cicatrix, and tumors. Achalasia

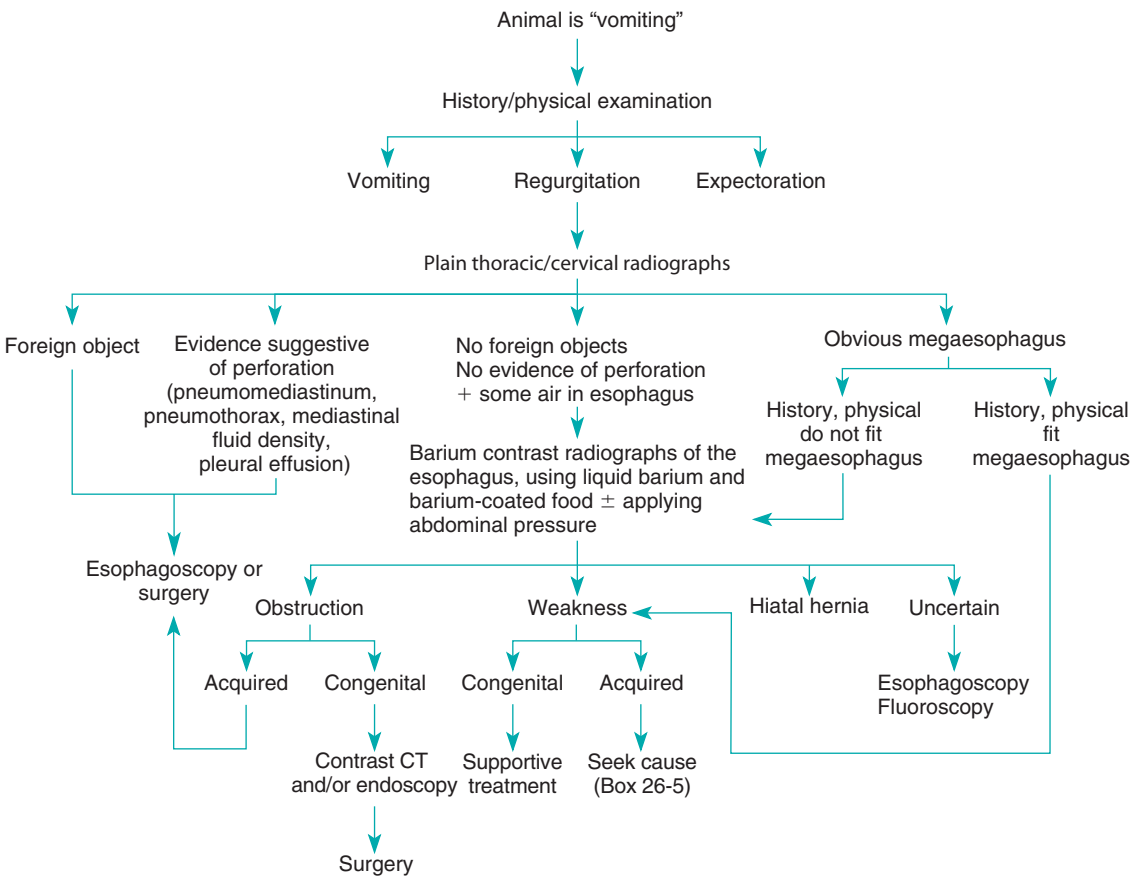

FIG 26.1
General diagnostic approach to regurgitation in the dog and cat.

of the lower esophageal sphincter may sometimes be responsible (Box 26.4). Obstruction should be characterized as congenital or acquired and as intraluminal, intramural, or extraesophageal. Congenital obstructions are usually extraesophageal vascular ring anomalies. Acquired intraluminal obstructions are usually foreign objects or cicatrix secondary to esophagitis. The clinician should always determine whether animals with esophageal foreign objects (especially small foreign objects that would have been expected to have passed) have a partial esophageal stricture that predisposed them to obstruction. Endoscopy may be both diagnostic and therapeutic in these animals; thoracotomy is seldom needed for management of cicatrix or intraluminal foreign objects.

Esophageal weakness may be congenital or acquired. Congenital weakness is typically idiopathic, and further diagnostics are unfruitful. Acquired esophageal weakness usually results from an underlying neuromuscular problem. Although an underlying cause is infrequently diagnosed, finding one may lead to a permanent cure as opposed to supportive therapy of the symptoms. A complete blood count (CBC), serum biochemistry profile, determination of serum antibody titers to acetylcholine receptors, resting serum cortisol (see Chapter 50), serum creatine kinase, and/or fecal examination for *Spirocerca lupi* ova are performed to look for causes of acquired esophageal weakness (Box 26.5). One may also consider searching for lead intoxication (nucleated red blood cells and basophilic stippling in the CBC, serum and urine lead concentrations), canine distemper (retinal lesions), and various neuropathy-myopathies (electromyography, nerve biopsy, muscle biopsy). Chagas disease causes esophageal disease in people but has not been reported to cause esophageal weakness in dogs. Lower esophageal achalasia-like syndrome has been identified in dogs with congenital and acquired weakness. The prevalence of this syndrome is currently uncertain, but assumed to be low. Fluoroscopic examination is necessary for diagnosis.

Esophagoscopy may detect esophagitis or small lesions (e.g., partial strictures) that contrast-enhanced esophagrams do not reveal. If esophagitis is found, the clinician should look carefully for a cause (e.g., hiatal hernia, gastric outflow obstruction). After entering the stomach, the endoscopist must retroflex the tip of the endoscope and examine the gastric side of the lower esophageal sphincter for leiomyomas or malformation (e.g., hiatal hernia). Gastroduodenoscopy is performed concurrently to look for gastric and duodenal reasons for gastroesophageal reflux or vomiting.

BOX 26.4

Causes of Esophageal Obstruction

Congenital Causes

Vascular ring anomaly
- Persistent fourth right aortic arch (most common type)
- Other vascular rings

Esophageal web (rare)

Acquired Causes

Foreign object (especially when there is a sudden onset)
Cicatrix/stricture (not common but very important)
Neoplasia
- Esophageal tumors
  - Carcinoma
  - Sarcoma caused by *Spirocerca lupi*
  - Leiomyoma of lower esophageal sphincter
- Extraesophageal tumors
  - Thyroid carcinoma
  - Pulmonary carcinoma
  - Mediastinal lymphosarcoma

Achalasia of the lower esophageal sphincter (sometimes found in dogs)
Gastroesophageal intussusception (very rare)

BOX 26.5

Causes of Esophageal Weakness

Congenital Causes

Idiopathic
Achalasia-like syndrome (not sure how common)

Acquired Causes

Myasthenia (generalized or localized) (important)
Hypoadrenocorticism (uncommon but important)
Severe esophagitis
- Gastroesophageal reflux
  - Hiatal hernia
  - Anesthesia-associated reflux (uncommon but important)
  - Spontaneous reflux
- Foreign body (uncommon but important)
- Caustic ingestion
  - Iatrogenic (e.g., doxycycline, clindamycin, ciprofloxacin, nonsteroidal anti-inflammatory drugs [NSAID])
  - Disinfectants, chemicals, etc.
- Persistent vomiting
- Excessive gastric acidity
  - Gastrinoma
  - Mast cell tumor
- Fungal organisms (e.g., pythiosis)

Myopathies (including muscular dystrophy)/neuropathies
Miscellaneous causes
- Achalasia-like syndrome
- Dysautonomia
- *Spirocerca lupi*
- Dermatomyositis (principally in Collies)
- Botulism
- Tetanus
- Lead poisoning
- Canine distemper

Idiopathic

VOMITING

Vomiting is usually caused by (1) motion sickness, (2) ingestion of emetogenic substances (e.g., drugs), (3) gastrointestinal (GI) tract obstruction, (4) abdominal (especially alimentary tract) inflammation or irritation, and (5) extragastrointestinal tract diseases that may stimulate the medullary vomiting center region or the chemoreceptor trigger zone (Box 26.6). Occasionally, central nervous system (CNS) disease, behavior, and learned reactions to specific stimuli may cause vomiting. If the cause of the vomiting is inapparent on history and physical examination, the next step depends on whether the vomiting is acute or chronic and whether there is hematemesis (Figs. 26.2 and 26.3). Remember that blood in vomitus may be fresh (i.e., red) or digested to varying degrees (i.e., "coffee grounds" or "dregs").

In animals with acute vomiting without hematemesis, clinicians should first search for obvious causes (e.g., ingestion of a foreign body, intoxication, organ failure, parvovirus) as well as for secondary fluid, electrolyte, or acid-base abnormalities or sepsis that require prompt, specific therapy. If the animal appears stable and there is no obvious cause, symptomatic treatment is often first tried for 1 to 2 days. If the animal is too sick for the clinician to take a chance on

BOX 26.6

Causes of Vomiting

- Motion Sickness (Acute) (Important)
- Diet (Important)
  - Dietary indiscretion
  - Dietary intolerance
- Emetogenic Substances (Acute)
  - Drugs: almost any drug can cause vomiting (especially drugs administered orally [PO]), but the following drugs seem especially likely to cause vomiting:
    - Digoxin
    - Chemotherapeutics (e.g., cyclophosphamide, cisplatin, dacarbazine, doxorubicin)
    - Selected antibiotics (e.g., erythromycin, tetracycline/doxycycline, amoxicillin plus clavulanic acid)
    - Penicillamine
    - Nonsteroidal antiinflammatory drugs
    - Apomorphine
    - Xylazine
  - Toxic chemicals
    - Strychnine
    - Heavy metals
- Gastrointestinal Tract Obstruction (Acute or Chronic) (Important)
  - Gastric Outflow Obstruction
    - Benign pyloric stenosis (uncommon)
    - Foreign object (common)
    - Gastric antral mucosal hypertrophy
    - Neoplasia
    - Nonneoplastic infiltrative disease (e.g., pythiosis)
    - Gastric malpositioning
      - Gastric dilation or volvulus (see nonproductive retching)
      - Partial gastric dilation/volvulus (does not always cause clinical signs)
  - Intestinal Obstruction
    - Foreign object (very common)
      - Nonlinear objects
      - Linear objects
    - Neoplasia
    - Intussusception
    - Cicatrix (rare)
    - Torsion/volvulus (very rare)
- Gastrointestinal/Abdominal Inflammation (Acute or Chronic) (Important)
  - Gastritis (common)
    - Without ulcers/erosions
    - With ulcers/erosions
    - Non-obstructing foreign body
    - Parasitic (i.e., *Physaloptera, Ollulanus*)
  - Enteritis (acute)
    - Parvovirus (common)
    - Acute hemorrhagic diarrheal syndrome (common)
    - Parasites (acute or chronic)
    - Inflammatory bowel disease (IBD) (more common in cats)
  - Pancreatitis (common and very important in dogs)
  - Peritonitis (acute or chronic; septic or nonseptic)
  - Colitis (acute or chronic)
  - Splenitis
- Extraalimentary Tract Diseases (Acute or Chronic) (Important)
  - Uremia (common)
  - Adrenal insufficiency (uncommon but important)
  - Hypercalcemia
  - Hepatic insufficiency or disease (Important)
  - Cholecystitis
  - Diabetic ketoacidosis
  - Pyometra
  - Endotoxemia/septicemia
- Miscellaneous Causes (Acute or Chronic)
  - Dysautonomia
  - Feline hyperthyroidism (Important)
  - Postoperative nausea/ileus (uncommon)
  - Overeating
  - Idiopathic hypomotility
  - Central nervous system disease
    - Tumor
    - Meningitis
    - Increased intracranial pressure
  - Sialoadenitis/sialoadenosis*
  - Behavior
  - Physiologic (epimeletic in female dogs)

*It is important to determine whether this is the cause of vomiting or an effect of vomiting.

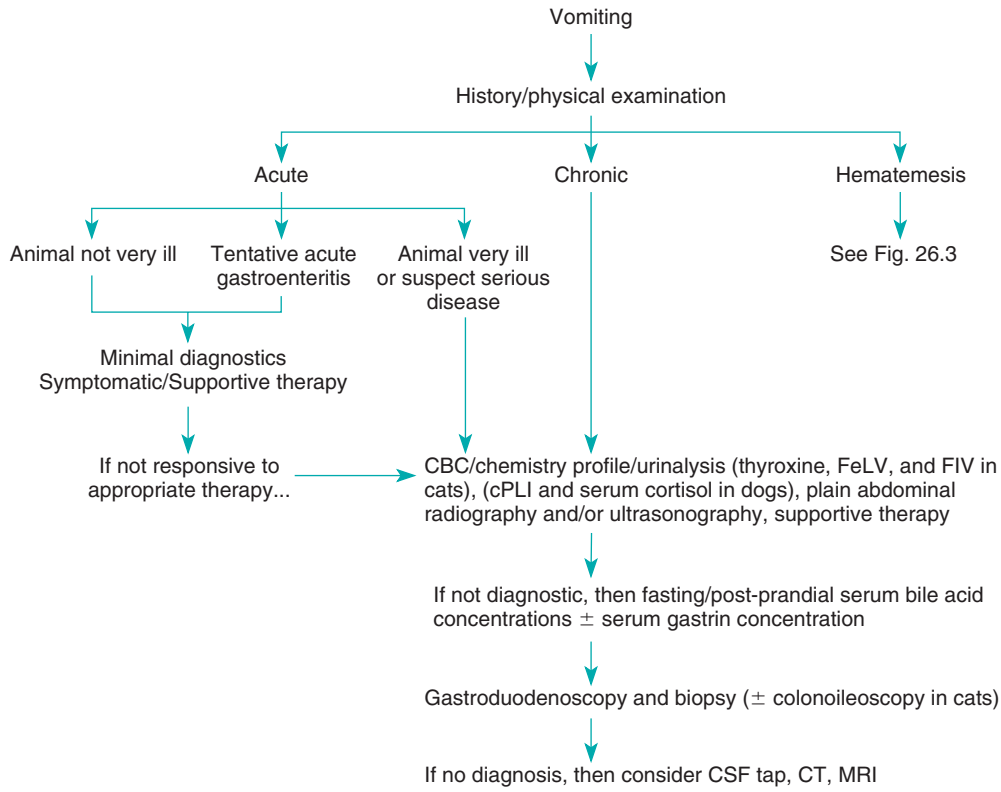

FIG 26.2
General diagnostic approach to vomiting in the dog and cat. *CBC,* Complete blood count; *cPLI,* canine pancreatic lipase immunoreactivity; *CSF,* cerebrospinal fluid; *FeLV,* feline leukemia virus; *FIV,* feline immunodeficiency virus; *MRI,* magnetic resonance imaging.

guessing wrong, if the vomiting persists for 2 to 4 days after the start of symptomatic therapy, or if the condition worsens during this initial time, then more aggressive diagnostic testing is usually appropriate.

The clinician should search for historical evidence of ingested foreign objects, toxins, inappropriate food, or drugs. Physical examination is used to look for abdominal abnormalities (e.g., masses, pain) and evidence of extraabdominal disease (e.g., uremic glossitis, a thyroid nodule indicative of hyperthyroidism). Clinicians should always look for linear foreign bodies in vomiting cats and carefully examine the base of the tongue; chemical restraint (e.g., ketamine HCl, 2.2 mg/kg of body weight given intravenously) may be necessary to examine this area properly. The abdomen is palpated to search for masses or pain, but even careful palpation may miss short ileocolic intussusceptions in the craniodorsal abdomen. It is reasonable to perform fecal examination for parasites because they can cause vomiting. If a cause cannot be found and the animal is not unduly ill, the clinician may prescribe a therapeutic trial (e.g., pyrantel and a dietary trial; see Table 28.7). Therapeutic trials should be designed so that failure of the therapy allows the clinician to exclude at least one disease.

If acute vomiting does not respond to symptomatic therapy or if the animal is so sick that the clinician cannot take a chance on symptomatic therapy being ineffective, aggressive diagnostic testing is appropriate. Animals with acute or chronic vomiting without hematemesis should undergo abdominal radiography and/or ultrasonography to look for intestinal obstruction, foreign objects, masses, pancreatitis, peritonitis, poor serosal contrast, alimentary tract ileus, free abdominal fluid, or free abdominal gas. Abdominal ultrasonography can be more revealing than plain radiographs, but radiographs may be more sensitive in revealing free air and some foreign bodies. A CBC, serum biochemistry profile, and urinalysis are also indicated. Cats should be tested for feline leukemia virus, feline immunodeficiency virus, and hyperthyroidism. It may be necessary to measure serum bile acid concentrations (or blood ammonia concentrations) or resting serum cortisol concentrations to screen for hepatic or adrenal insufficiency, respectively, that can be unsuspected based on serum biochemistry profiles. Immunoreactive pancreatic lipase activity can be helpful in diagnosing otherwise occult canine pancreatitis, but it must be remembered that the test cannot be used as a simple litmus test for clinically important pancreatitis.

If results of extensive clinical pathology testing and abdominal imaging are not diagnostic in a patient with chronic vomiting, the next diagnostic step is usually upper GI endoscopy. If endoscopy is not available, then exploratory laparotomy may be substituted, but surgery can miss gastric lesions that will be picked up endoscopically. Contrast gastrograms

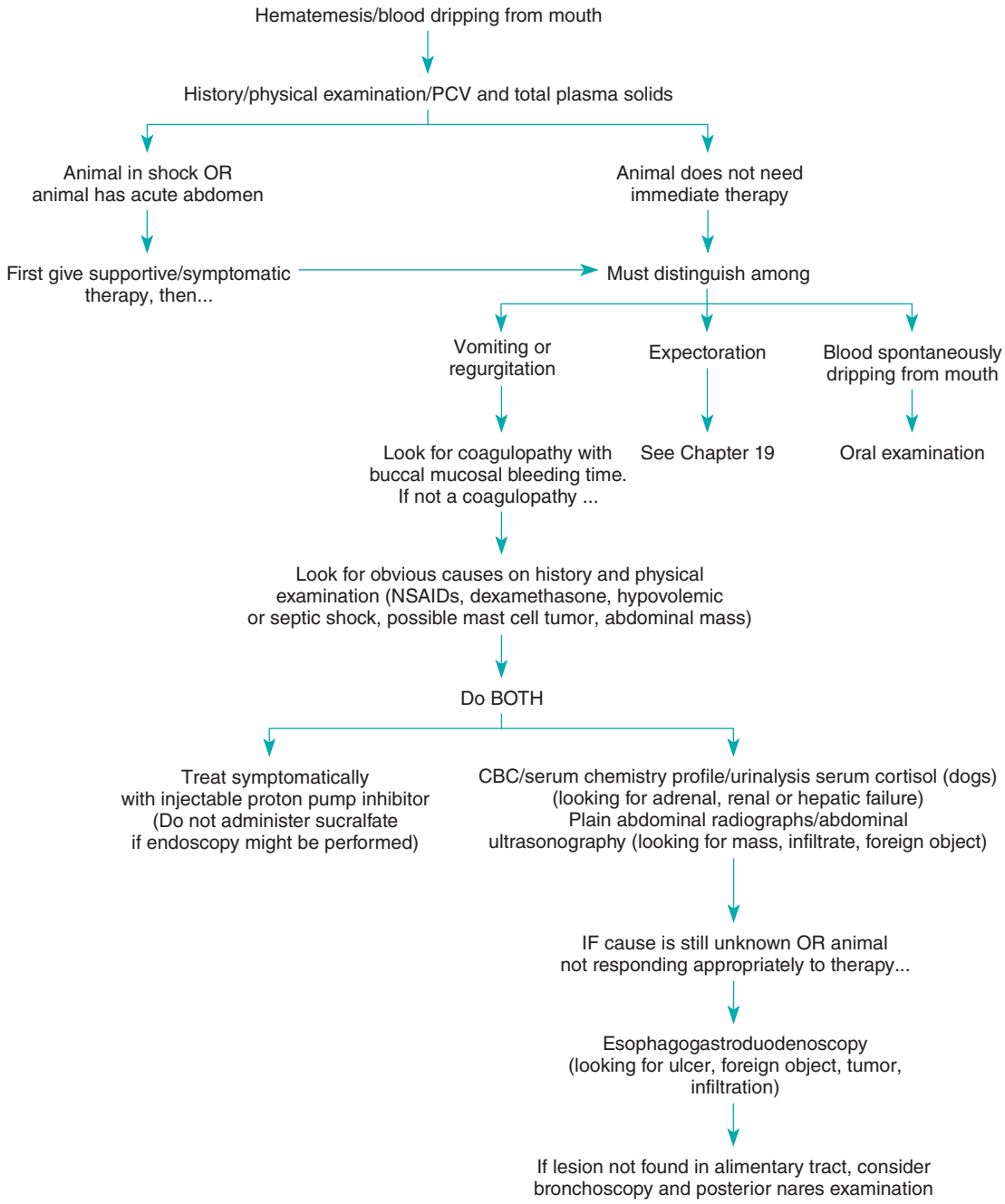

FIG 26.3
General diagnostic approach to hematemesis in the dog and cat. *CBC,* Complete blood count; *PCV,* packed cell volume.

are occasionally helpful; however, endoscopy is usually more cost-effective. During endoscopy, the clinician should biopsy the stomach and duodenum, regardless of normal gross mucosal appearance. In cats, endoscopic biopsy of the ileum and ascending colon may occasionally be required to find the cause of vomiting. If laparotomy is chosen over endoscopy, the entire abdomen should be examined. The stomach, duodenum, jejunum, ileum, mesenteric lymph nodes, liver, and, in cats, the pancreas should typically be biopsied.

If the cause of vomiting is undiagnosed after biopsy, the basis for previously excluding the different diseases should be reviewed. Diseases may be inappropriately ruled out (or diagnosed) because the clinician does not understand the limitations of certain tests. For example, dogs with hypoadrenocorticism may have normal electrolyte concentrations; inflammatory gastric and bowel disease may be localized to one area of the stomach or intestine and rarely causes significant changes in the white blood cell count; hyperthyroid cats may have normal serum thyroxine concentrations; dogs and cats with hepatic failure may have normal serum bilirubin concentrations as well as normal serum alanine aminotransferase and alkaline phosphatase activities; dogs

and cats with pancreatitis may have normal immunoreactive pancreatic lipase activities and normal abdominal ultrasound examinations; and *Physaloptera* infections are almost never diagnosed by fecal examination. Finally, the clinician may have to consider uncommon diseases that are more difficult to diagnose (e.g., idiopathic gastric hypomotility, occult CNS disease).

HEMATEMESIS

Clinicians must often use history and physical examination to help identify hematemesis as well as distinguish it from other problems. Hematemesis is expulsion of digested (i.e., "coffee grounds") or fresh blood. Animals with oral lesions that have blood dripping from their lips do not have hematemesis. Likewise, hemoptysis (i.e., coughing up blood) is not hematemesis.

Clinicians should distinguish vomiting that produces specks of blood from vomiting in which there is substantial blood present. The former may be caused by gastric mucosal trauma secondary to vigorous vomiting from any cause, and animals with such "hematemesis" should generally be treated as described in the previous section on vomiting. Patients that produce more substantial amounts of blood generally should be approached differently. Although hematemesis is usually caused by gastroduodenal ulceration and erosion (GUE), clinicians should not make this assumption and automatically treat for such. The clinician should first check the hematocrit and plasma total protein concentration to determine whether a blood transfusion is necessary (see Fig. 26.3). The clinician should next try to determine whether there is a coagulopathy (uncommon but important), ingestion of blood from another site (including the respiratory tract), or a gastrointestinal tract (GIT) lesion (e.g., GUE) (Box 26.7). Platelet counts and a measure of clotting capability (e.g., buccal mucosal bleeding time) are strongly recommended. The clinician should next look for obvious causes of GIT hemorrhage (e.g., acute gastritis, acute hemorrhagic diarrheal syndrome [used to be called *hemorrhagic gastroenteritis*], or GUE due to ulcerogenic drugs [e.g., nonsteroidal

BOX 26.7

Causes of Hematemesis

Coagulopathy (Uncommon)

Thrombocytopenia/platelet dysfunction
Clotting factor deficiency
Disseminated intravascular coagulation

Alimentary Tract Lesion

Gastrointestinal tract ulceration/erosion (common and important)
- Infiltrative disease (important)
  - Neoplasia
    - Leiomyoma
    - Carcinomas
    - Lymphomas
  - Pythiosis (especially younger dogs in the southeastern United States)
  - Inflammatory bowel disease (uncommon)
- "Stress" ulceration
  - Hypovolemic shock
  - Septic shock (i.e., systemic inflammatory response syndrome)
  - After gastric dilation or volvulus
  - Neurogenic "shock"
  - Extreme or sustained exertion (common in select working animals)
- Hyperacidity
  - Mast cell tumor
  - Gastrinoma (rare)
- Iatrogenic causes
  - Nonsteroidal antiinflammatory drug (common and important)
  - Glucocorticoids (especially dexamethasone) (important)
- Other causes
  - Hepatic disease (common and important)
  - Hypoadrenocorticism (uncommon but important)
  - Pancreatitis (common disease but uncommonly causes ulceration or hematemesis)
  - Renal disease (common disease but uncommonly causes ulceration or hematemesis)
  - Inflammatory diseases
- Foreign objects (rarely a primary cause of hematemesis, but will worsen preexisting ulceration or erosion)

Gastritis
- Acute gastritis (common)
- Acute hemorrhagic diarrheal syndrome (common)
- Chronic gastritis (uncommon in dogs)
- *Helicobacter*-associated disease (doubtful association with hematemesis in dogs and cats)

Gastric mucosal trauma from vigorous vomiting*
Gastric polyps
Esophageal disease (uncommon)
- Tumor
- Severe esophagitis
- Trauma

Bleeding oral lesion
Gallbladder disease (especially tumors) (rare)

Extraalimentary Tract Lesion (Blood Is Swallowed and Then Vomited) (Rare)

Respiratory tract disorders
- Lung lobe torsion
- Pulmonary tumor
- Posterior nares lesion

Dietary indiscretion

*Hematemesis caused by vigorous vomiting usually consists of specks of blood as opposed to larger quantities.

antiinflammatory drugs, dexamethasone] or recent severe hypovolemic shock or systemic inflammatory response syndrome or abdominal masses that may involve the gastric mucosa or cutaneous mast cell tumors). Remember: mast cell tumors can grossly mimic almost any other benign or malignant neoplasm, especially lipomas.

If acute gastritis, acute hemorrhagic diarrheal syndrome, nonsteroidal antiinflammatory drug or dexamethasone–induced GUE, or GUE resulting from shock is strongly suspected, the clinician may elect a limited diagnostic workup (e.g., CBC, serum biochemistry panel) to define the degree of blood loss and look for evidence of renal, hepatic, or adrenal failure. Then the animal can be treated symptomatically for 3 to 5 days (see Chapter 28, pp. 437-440) to see what effect this has in controlling clinical signs. Endoscopy is not necessary or helpful in many of these cases because it cannot reliably distinguish between ulcers that will heal with medical therapy and those that will require surgical resection. However, if the cause of hematemesis or GUE is unknown, more aggressive diagnostic tests (e.g., abdominal ultrasound and gastroduodenoscopy) should be considered (see Fig. 26.3). The stomach and duodenum should be imaged, preferably by ultrasonography, to look for alimentary tract infiltrations, foreign objects, and masses. Endoscopy is the most sensitive and specific means of finding and evaluating GUE. The principal indications for endoscopy in animals with upper GI blood loss include (a) distinguishing potentially resectable ulcers from widespread, unresectable erosions in patients with life-threatening GI bleeding; (b) localizing ulcers when considering surgical resection; and (c) determining the cause of GUE in patients with upper GI blood loss of unknown cause. Abdominal exploratory surgery may be performed instead of endoscopy, but it is easy to miss bleeding mucosal lesions when examining the serosal surface of the GIT. Intraoperative endoscopy (i.e., endoscopic examination of the mucosal surface of the stomach and duodenum while the abdomen is opened) may sometimes be useful in finding lesions that the surgeon cannot discern from the serosal surface.

If the source of bleeding cannot be found using gastroduodenoscopy, the clinician should consider possible bleeding sites beyond the reach of the endoscope; blood being swallowed from a lesion in the mouth, posterior nares, trachea, or lungs; hemorrhage from the gallbladder; or an intermittently bleeding gastric or duodenal lesion. Capsule endoscopy has recently become available for dogs and can also be used to look for bleeding lesions throughout the GI tract. It can be used when endoscopy is not available or when the bleeding lesion is suspected to be beyond the reach of the endoscope. Endoscopy of the trachea and choana can be diagnostic in some cases of occult respiratory hemorrhage.

DIARRHEA

Diarrhea is nothing more than excessive fecal water. This explains why many animals with severe small bowel disease do not have diarrhea. When diarrhea is present, one should first distinguish acute from chronic problems.

Acute diarrhea is usually caused by diet, parasites, or infectious diseases (Box 26.8). Dietary problems are often detected by history; parasites by fecal examination; and infectious diseases by history (i.e., evidence of contagion or exposure), CBC, fecal enzyme–linked immunosorbent assay for canine parvoviral antigen, and exclusion of other causes.

BOX 26.8

Causes of Acute Diarrhea

Diet (Common and Important)

Intolerance/allergy
Poor-quality food
Rapid dietary change (especially in puppies and kittens)
Bacterial food poisoning
Dietary indiscretion

Parasites (Common and Important)

Helminths
Protozoa
- *Giardia*
- *Tritrichomonas* (feline)
- *Coccidia*

Infectious Causes

Viral causes
- Parvovirus (canine, feline) (dogs: common and important)
- Coronavirus (canine, feline) (infrequent, not important)
- Feline leukemia virus (including infections secondary to it)
- Feline immunodeficiency virus (specifically infections secondary to it)
- Various other viruses (e.g., rotavirus, canine distemper virus)

Bacterial causes
- *Salmonella* spp. (uncommon)
- *Clostridium perfringens* (common and important in large bowel diarrheas)
- Verotoxin-producing *Escherichia coli*
- *Campylobacter jejuni* (uncommon)
- *Yersinia enterocolitica* (questionable)
- Various other bacteria

Rickettsial infection
- Salmon poisoning (regionally important)

Other Causes

Acute hemorrhagic diarrheal syndrome
Intussusception
Ingestion of "toxins"
- "Garbage can" intoxication (spoiled foods)
- Chemicals
- Heavy metals
- Various drugs (antibiotics, antineoplastics, anthelmintics, antiinflammatories, digitalis, lactulose)

Acute pancreatitis (diarrhea is usually a modest component of clinical signs but it can be major)
Hypoadrenocorticism

If acute diarrhea becomes unduly severe or persistent (i.e., chronic), then additional diagnostic tests are recommended. Animals with chronic diarrhea should be examined for parasites. Typically multiple fecal examinations looking for nematodes, *Giardia,* and *Tritrichomonas* are indicated. The in-house enzyme-linked immunosorbent assay (ELISA) "snap" tests for Giardia are more sensitive than fecal examination and have excellent negative predictive values. The indirect fluorescent antibody test (IFA) test for *Giardia* is considered the "gold standard" test, but requires feces to be sent off to a laboratory. Next, the clinician should determine whether the diarrhea originates from the small or large intestine. History is often the best tool (Table 26.2). Failure to lose weight or body condition despite chronic diarrhea almost always indicates large bowel disease. Weight loss usually indicates small bowel disease although severe large bowel diseases (e.g., pythiosis, histoplasmosis, protothecosis, malignancy) may cause weight loss; however, animals with large bowel disease causing weight loss usually have obvious signs of colonic involvement (e.g., fecal mucus, tenesmus, hematochezia). Whenever tenesmus is present, the clinician should ascertain whether or not it was present when the disease began. If tenesmus did not begin until late in the course of the diarrhea, it may be due simply to perineal scalding or anal soreness resulting from chronic irritation.

Chronic small intestinal diarrhea can be categorized as maldigestion, nonprotein-losing malabsorptive disease, and protein-losing enteropathy. Maldigestion is principally caused by exocrine pancreatic insufficiency (EPI) and infrequently causes marked hypoalbuminemia (i.e., < 2.0 g/dL with a normal range 2.5-4.4 g/dL). Film digestion tests for fecal trypsin activity, Sudan staining of feces for undigested fats, and fat absorption tests yield many false-negative and false-positive results. The most sensitive and specific test for EPI is the serum trypsin-like immunoreactivity (TLI; see Chapter 27, p. 423), which is indicated in patients with chronic small intestinal diarrhea.

Attempting to diagnose EPI by supplementing pancreatic enzymes is strongly discouraged. If a dog's diarrhea ameliorates in response to pancreatic enzyme supplementation, it might be due to EPI or antibiotic-responsive enteropathy (ARE; i.e., dysbiosis) or it might just be a fortuitous, temporal effect. A false-positive diagnosis of EPI results in the unnecessary supplementation of expensive enzymes. Up to 15% of dogs with EPI do not respond when enzymes are added to their diet; therefore if EPI is incorrectly ruled out in such a case, unnecessary endoscopies or operations often result. Therefore the clinician should definitively diagnose or rule out EPI before proceeding with other diagnostic tests or treatments.

Malabsorptive intestinal disease may be protein-losing enteropathy (PLE) or nonprotein-losing (Fig. 26.4). Diarrhea occurs only if the absorptive capacity of the colon is exceeded. Therefore a dog or cat can be losing weight and/or albumin because of small intestinal disease and not have diarrhea (see the section on Weight Loss). The serum albumin concentration can be mildly or markedly decreased (i.e., 2.0 g/dL or less [normal range 2.5-4.4 g/dL]) in patients with PLE. If an animal has marked hypoalbuminemia not caused by protein-losing nephropathy, hepatic insufficiency, or skin lesions, then PLE is diagnosed by process of exclusion. If the serum albumin is mildly decreased (i.e., 2.1-2.4 g/dL), then other causes (e.g., sequestration in third space, nutrition) should also be considered. Hypoglobulinemia sometimes develops in patients with PLE, but many patients with PLE do not have panhypoproteinemia. In general, dogs with PLE are preferably approached with aggressive diagnostics.

In patients with nonprotein-losing malabsorptive disease, the clinician may perform additional diagnostic tests (e.g., intestinal biopsy) or perform therapeutic trials. If the patient

TABLE 26.2

Differentiation of Chronic Small Intestinal From Large Intestinal Diarrheas

SIGN	SMALL INTESTINAL DIARRHEA	LARGE INTESTINAL DIARRHEA
Weight loss*	Expected	Uncommon*
Polyphagia	Sometimes	Rare to absent
Frequency of bowel movements	Often near normal	Sometimes very increased but often normal
Volume of feces	Often increased, but can be normal volume	Sometimes decreased (because of the increased frequency) but can be normal
Blood in feces	Melena (rare)	Hematochezia (sometimes†)
Mucus in feces	Uncommon	Sometimes
Tenesmus	Uncommon (but may occur later in chronic cases)	Sometimes
Vomiting	May be seen	May be seen

*Failure to lose weight or condition is the most reliable indication that an animal has large bowel disease. However, animals with colonic histoplasmosis, pythiosis, lymphoma, protothecosis, or similar severe infiltrative diseases may have weight loss due to large bowel disease.
†Hematochezia becomes much more important as a differentiating feature in animals that are losing weight. Its presence in such animals confirms the presence of large bowel involvement (either by itself or in combination with small bowel disease) despite weight loss.

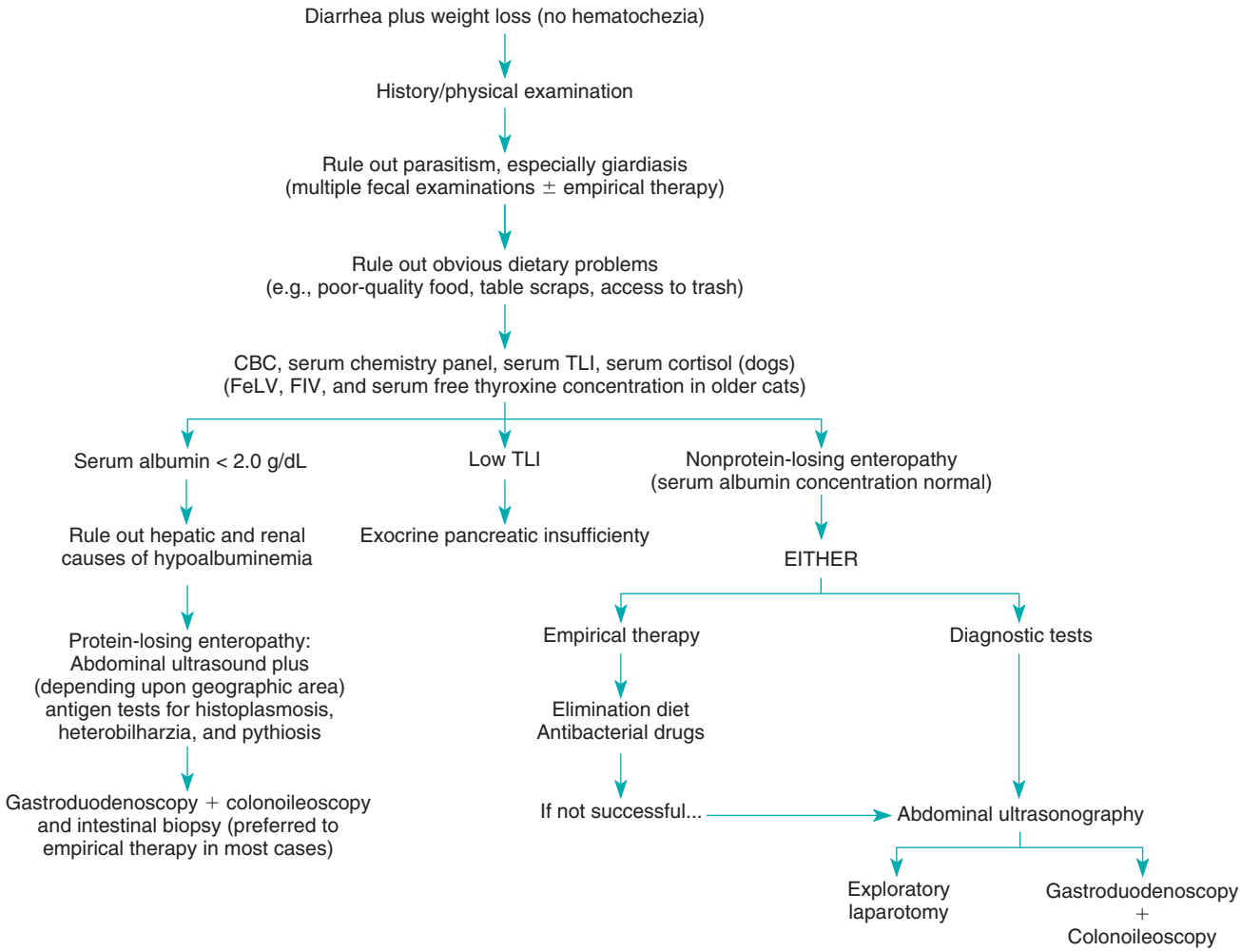

FIG 26.4
General diagnostic approach to small intestinal diarrhea in the dog and cat. *CBC,* Complete blood count; *FeLV,* feline leukemia virus; *FIV,* feline immunodeficiency virus, *TLI,* trypsin-like immunoreactivity.

is not emaciated or rapidly losing weight and is not hypoalbuminemic, then biopsy is seldom the best next step. Screening for atypical hypoadrenocorticism with resting serum cortisol concentrations is appropriate. Testing for histoplasmosis, pythiosis, and heterobilharziasis can be appropriate, depending upon geographical location. Therapeutic trials are the best way to diagnose dietary-responsive diarrhea and ARE (also called *antibiotic-responsive diarrhea* and/or *dysbiosis*), which are two of the most common causes of chronic small bowel diarrhea in dogs. ARE cannot be diagnosed by measuring serum cobalamin and folate concentrations. If a therapeutic trial is performed, the clinician must be sure that it is done properly (e.g., long enough, correct dose) so that it has a high likelihood of succeeding if the animal has the suspected disease. If the patient seems particularly ill (e.g., severe or rapid weight loss), or is hypoalbuminemic, or has failed well-implemented clinical trials and is negative for infectious causes (i.e., histoplasmosis, heterobilharziasis, pythiosis) and atypical hypoadrenocorticism, then abdominal ultrasonography is the next step. Ultrasonography should always precede biopsy because it may be diagnostic if it shows dilated lymphatics in the intestinal mucosa (i.e., lymphangiectasia) or lymphadenopathy or intestinal infiltrates that can be aspirated percutaneously. If ultrasonography is not diagnostic, then biopsy is the next step (Boxes 26.9 and 26.10). Either laparotomy or endoscopy can be performed to biopsy the bowel. If ultrasonography reveals a localized lesion that cannot be reached with an endoscope, then laparotomy is indicated. Otherwise, endoscopy is quicker and safer than laparotomy and may allow the clinician to biopsy lesions not discernable from the serosal surface. Endoscopic biopsy specimens can easily be nondiagnostic if the endoscopist has not been carefully trained in biopsy techniques. If laparotomy is performed in hypoalbuminemic animals, it may be prudent to use nonabsorbable suture material and/or perform intestinal serosal patch grafting. Distended intestinal lymphatics or lipogranulomas in the intestinal wall are suggestive of lymphangiectasia. If intestinal biopsy specimens are not helpful, the main possibilities are that the tissue specimens were inadequate (e.g., not deep enough, from

BOX 26.9

Major Causes of Malabsorptive Disease

Dog

Parasitism: giardiasis, nematodes (common and important)
Dietary responsive (food intolerance or allergy; common and important)
Antibiotic-responsive enteropathy (also called "dysbiosis") (common and important)
Inflammatory bowel disease
Neoplastic bowel disease (especially lymphoma; important but not common)
Fungal infections (regionally important)
  Pythiosis
  Histoplasmosis

Cat

Dietary responsive (food intolerance or allergy; common and important)
Parasitism: giardiasis
Inflammatory bowel disease: lymphocytic-plasmacytic enteritis (common and important)
Neoplastic bowel disease (especially lymphoma; common and important)

BOX 26.10

Major Causes of Protein-Losing Enteropathy*

Dog

Intestinal lymphangiectasia (common and important)
Alimentary tract lymphoma (important)
Severe inflammatory bowel disease
Alimentary tract fungal infections
  Histoplasmosis (regionally important)
  Pythiosis (regionally important)
Chronic intussusception (especially young dogs)
Alimentary tract hemorrhage (e.g., ulceration or erosion, neoplasia, parasites)
Unusual enteropathies (e.g., chronic purulent enteropathy, severe ectasia of mucosal crypts)
Massive hookworm or whipworm infestation (regionally important)

Cat

Alimentary tract lymphoma (important)
Severe inflammatory bowel disease (common and important)
Alimentary hemorrhage (e.g., neoplasia, duodenal polyps, idiopathic ulceration)

*Any gastrointestinal disease can cause protein-losing enteropathy, but these are the most common causes. Except for lymphangiectasia, these diseases do not consistently produce protein-losing enteropathy.

BOX 26.11

Major Causes of Chronic Large Intestinal Diarrhea

Dog

Dietary responsive (intolerance or allergy; important and common)
Fiber-responsive (important and common)
Parasitism
  Whipworms (regionally important and common)
  Heterobilharzia (regionally important)
Bacterial diseases
  "Clostridial" colitis (important and common)
  Histiocytic ulcerative colitis (usually Boxers and French Bulldogs)
Fungal infections (regionally important and common)
  Histoplasmosis
  Pythiosis
Inflammatory bowel disease (uncommon in dogs)
Neoplasia
  Lymphoma
  Adenocarcinoma

Cat

Dietary responsive (intolerance or allergy; important and common)
Fiber-responsive (important and common)
Inflammatory bowel disease (important)
Tritrichomonas (especially important in exotic cats and in catteries)
Feline leukemia virus infection (including infections secondary to it)
Feline immunodeficiency virus infection (specifically infections secondary to it)

the wrong place, too much artifact), the pathologist did not recognize the lesion, the animal has occult giardiasis or ARE or dietary intolerance, or there is disease (e.g., lymphangiectasia, neoplasia, inflammation) localized to where the clinician did not biopsy.

There are some differences in cats with chronic small bowel disease compared with dogs. PLE is less common in cats, and when present typically indicates severe infiltrative disease (usually not lymphangiectasia) and the need for biopsy. Nematode infections causing chronic diarrhea are much less common in cats than in dogs.

Dogs with chronic large intestinal diarrhea (Box 26.11) should first undergo a digital rectal examination to search for mucosal thickening or proliferation. The rectum is the most common site of canine colonic neoplasia, and finding obvious mucosal lesions indicates the need for biopsy. If the rectal mucosa seems normal, the animal has not lost weight,

and the serum albumin is well within the normal range (a serum albumin at the lower end of the reference range may not be "normal" enough), then it is often most appropriate to try therapeutic trials after a few basic diagnostic tests. Fecal examination to detect whipworms, fecal ELISA to detect *Giardia* (a small bowel problem that can mimic large bowel disease), and fecal PCR to detect *Tritrichomonas* (cats) are indicated. Therapeutic trials usually consist of high-fiber diets, elimination diets, antibacterials to control "clostridial" colitis, and/or treatment for parasites.

Additional diagnostic tests that may be done instead of therapeutic trials principally include biopsying colonic mucosa by colonoscopy, and fecal assays for toxins (e.g., clostridial toxin) and/or specific organisms (e.g., *Campylobacter, Salmonella*). Fecal cultures and antigen tests for specific pathogens should be considered if history indicates a strong likelihood of a contagious disorder. Fecal ELISA and PCR assays should be done before performing enemas or administering lavage solutions. Unless there is a good epidemiologic reason to strongly suspect infectious bacteria, fecal cultures and antigen/DNA tests tend to be very low-yield procedures that are difficult to interpret. Colonoscopy/biopsy is principally useful to diagnose histoplasmosis, histiocytic ulcerative colitis, protothecosis, Heterobilharzia, or neoplasia in dogs, whereas cats are often diagnosed with colonic inflammatory bowel disease.

If the results of these tests are not diagnostic, the clinician must consider three main possibilities. First, the biopsy specimens may not be representative of the entire colonic mucosa. For example, if the disease is localized to the region of the ileocolic valve, it will be necessary to use a flexible endoscope to reach the area. Second, the pathologist may not have recognized the lesions. Third, there may be no mucosal lesions. This typically occurs in animals with a dietary intolerance or allergy, "clostridial" colitis, or fiber-responsive diarrhea, all common problems in dogs.

HEMATOCHEZIA

If the patient has hematochezia (fresh blood in the feces) and diarrhea, the problem should usually be approached in the same manner as for animals with large bowel diarrhea (see Chapter 26, pp. 401-402). The patient with normal stools plus hematochezia is approached slightly differently. Streaks of blood on the outside of otherwise normal feces usually indicates a distal colonic or rectal lesion, whereas blood mixed into feces suggests that bleeding is occurring higher in the colon. Coagulopathies rarely cause bleeding from the rectum only. Focal bleeding lesions in the distal colon, rectum, or perineal region (Box 26.12) are especially important. Acute hematochezia may also result from trauma (e.g., passing a foreign body).

A thorough digital rectal examination is the best initial step (even if anesthesia is necessary). The clinician should express each anal sac repeatedly and examine the contents. If the problem is chronic and digital rectal examination is not helpful, then colonoscopy and biopsy are indicated. Barium enema is not recommended. Biopsy specimens of masses must include submucosa or many neoplastic lesions will be missed. Hematochezia is rarely severe enough to cause anemia; however, a CBC can be performed to look for and characterize anemias.

BOX 26.12

Major Causes of Hematochezia*

Dog

Anal-rectal disease

Anal sacculitis (important and common)
Neoplasia
- Rectal adenocarcinoma (important)
- Rectal polyp (important)
- Colorectal leiomyoma or leiomyosarcoma

Perianal fistulas (important)
Anal foreign body
Rectal prolapse
Anal-rectal trauma (e.g., foreign body, thermometer, enema tube, fecal loop, pelvic fractures)

Colonic/intestinal disease

Parasitism
- Whipworms (important and common)
- Hookworms (severe infections can involve the colon)

Dietary responsive (intolerance or allergy; common)
"Clostridial" colitis (common disease but uncommonly causes hematochezia)
Acute hemorrhagic diarrheal syndrome (important and common)
Parvoviral enteritis (important and common)
Histoplasmosis (regionally important and common)
Pythiosis (regionally important)
Intussusception (more common in young animals)
- Ileocolic
- Cecocolic

Inflammatory bowel disease
Colonic trauma
Coagulopathy
Colonic vascular ectasia

Cat

Dietary responsive (intolerance or allergy)
Inflammatory bowel disease (important)
Coccidia
Rectal tumors (uncommon)

*These diseases do not consistently produce hematochezia, but when hematochezia is present, these are the most common causes.

MELENA

Melena is caused by digested blood and is seen as "coal tar"-black (not just "dark") feces. Clinicians must distinguish melena from stools that are intensely dark green. Melena is strongly suggestive of upper alimentary tract bleeding or

ingestion of blood (Box 26.13). However, a lot of blood must enter the GI tract in a relatively short time to produce melena, which is why most animals with upper GI hemorrhage do not have melena. A CBC is indicated to look for iron deficiency anemia (i.e., microcytosis, hypochromasia). Measuring total serum iron concentration and total iron-binding capacity plus staining the bone marrow for iron are more definitive tests for iron deficiency anemia. Ultrasonography is helpful when looking for infiltrated, bleeding lesions (e.g., an intestinal tumor). Gastroduodenoscopy is the most sensitive test for GUE (which is often missed by ultrasonography). If ultrasound and gastroduodenoscopy are nonrevealing, then one must suspect small intestinal lesions beyond the reach of the endoscope. If imaging reveals a lesion beyond the reach of the endoscope, exploratory laparotomy is required. The clinician may elect to perform exploratory surgery immediately, but it is easy to miss bleeding mucosal lesions when examining the serosa or palpating the bowel. Intraoperative endoscopy (i.e., having the surgeon manually advance the tip of the endoscope while pushing the intestines onto the endoscope) may be helpful if no lesion is detected at surgery. Capsule endoscopy may be helpful in confirming that there is a bleeding lesion in the distal small intestines before laparotomy (or in finding a more orad lesion previously missed). Contrast radiographs rarely detect bleeding lesions and are not recommended

TENESMUS

Tenesmus (i.e., ineffectual or painful straining at urination or defecation) and dyschezia (i.e., painful or difficult elimination of feces from the rectum) are principally caused by obstructive or inflammatory distal colonic or urinary bladder or urethral lesions (Box 26.14). Colitis, constipation, perineal hernias, perianal fistulas, prostatic disease, and cystic/urethral disease are the most common causes of tenesmus. Most rectal masses and strictures cause hematochezia; however, some do not disrupt the colonic mucosa and cause only tenesmus.

The first goal (especially in cats) is to distinguish lower urinary tract from alimentary tract disease. In cats tenesmus secondary to a urethral obstruction is often misinterpreted as constipation. By observing the patient, the clinician may be able to determine whether the animal is attempting to urinate or defecate. Palpating the bladder is important; a distended urinary bladder often indicates an obstruction whereas a small, painful bladder often indicates cystitis. A urinalysis can also be helpful. If necessary, one can catheterize the urethra to determine if it is patent.

If the clinician suspects tenesmus resulting from alimentary tract disease, the next steps are to palpate the abdomen, perform a digital rectal examination, and visualize the anus and perineal areas even if this requires sedation/anesthesia.

BOX 26.13

Major Causes of Melena*

Dog

- Hookworms (important)
- Gastroduodenal tract ulceration/erosion (see Box 26.7) (important)
- Gastric or small intestinal tumor (important)
  - Lymphoma
  - Adenocarcinoma
  - Leiomyoma or leiomyosarcoma
  - Polyp
- Ingested blood
  - Oral lesions
  - Nasopharyngeal lesions
  - Pulmonary lesions
  - Diet
- Hypoadrenocorticism (uncommon but important)
- Coagulopathies (uncommon but important)

Cat (Rare)

- Gastrointestinal tumor
  - Lymphoma
  - Duodenal polyps
  - Other tumors (adenocarcinoma, mast cell tumor)
- Coagulopathies: vitamin K deficiency (intoxication or resulting from malabsorption)

*These diseases do not consistently produce melena, but if melena is present, these are the most common causes.

BOX 26.14

Major Causes of Tenesmus and/or Dyschezia

Dog

- Perineal inflammation or pain: anal sacculitis (common and important)
- Rectal inflammation/pain
  - Perianal fistulae (important)
  - Tumor (important)
  - Proctitis (either primary disease or secondary to diarrhea or prolapse)
  - Histoplasmosis/pythiosis
- Colonic/rectal obstruction
  - Rectal neoplasia
  - Rectal granuloma
  - Perineal hernia (important)
  - Constipation
  - Prostatomegaly (common and important)
  - Pelvic fracture
  - Other pelvic canal masses
  - Rectal foreign object

Cat

- Urethral obstruction (common and very important)
- Rectal obstruction
  - Pelvic fracture
  - Perineal hernia
- Constipation
  - Abscess near rectum

The clinician should not assume that constipation, if present, is causing the tenesmus. Severe pain (e.g., that resulting from proctitis) may make the animal refuse to defecate and cause secondary constipation. Most rectal strictures, perineal hernias, masses, enlarged prostates, pelvic fractures, and rectal tumors can be detected during a digital rectal examination. The clinician may need to use two fingers to detect partial strictures when examining large dogs. Perianal fistulae are usually visible but may be detected only as perirectal thickenings. Next, the clinician expresses the anal sacs and examines their contents. Finally, the clinician evaluates the feces to determine whether they are excessively hard or have abnormal contents (e.g., hair, trash).

A biopsy should be done of any mass, stricture, or infiltrative lesion found by rectal examination. A rectal scraping is sometimes sufficient (e.g., histoplasmosis), otherwise biopsy specimens taken with rigid biopsy forceps that include submucosa are preferred. Fine-needle aspiration should be performed on extracolonic masses because abscesses occasionally occur.

If the clinician is confused by physical examination findings, observing the animal defecate may help define the underlying process. Animals with inflammation often continue to strain after defecating, whereas a constipated animal strains before feces are produced. Tenesmus that occurs when an animal is in a squatting position often results from colitis, whereas tenesmus that occurs when an animal is in a semiwalking or partial squatting position usually results from constipation.

CONSTIPATION

Constipation (infrequent and difficult evacuation of feces) and obstipation (intractable constipation) have several causes (Box 26.15). The initial use of symptomatic therapy is often successful, but it is important to look for causes because some problems may become harder to treat if symptomatic therapy masks signs while the underlying disease progresses.

Iatrogenic, dietary, environmental, or behavioral causes should be sought on history. Feces should be examined to determine whether they contain plastic, bones, hair, popcorn, or other such material. Physical and digital rectal examinations are done to search for rectal obstruction or infiltration. Plain pelvic radiographs can help show whether the animal has anatomic abnormalities or a previously undetected

BOX 26.15

Causes of Constipation

Iatrogenic Causes

Drugs
- Opiates
- Anticholinergics
- Carafate (sucralfate)
- Barium sulfate

Behavioral/Environmental Causes

Change in household/routine (especially cats)
Soiled litter box/no litter box (especially cats)
House training
Inactivity

Refusal to Defecate

Behavioral
Pain in rectal/perineal area (see Box 26.14)
Inability to assume position to defecate
- Orthopedic problem
- Neurologic problem

Dietary Causes

Excessive fiber in dehydrated animal
Abnormal diet (especially dogs)
- Hair
- Bones
- Indigestible material (e.g., plants, plastic)

Colonic Obstruction

Pseudocoprostasis
Deviation of rectal canal: perineal hernia (important)
Intraluminal and intramural disorders
- Tumor
- Granuloma
- Cicatrix
- Rectal foreign body
- Congenital stricture

Extraluminal disorders
- Tumor
- Granuloma
- Abscess
- Healed pelvic fracture
- Prostatomegaly (common and important)
- Prostatic or paraprostatic cyst
- Sublumbar lymphadenopathy

Colonic Weakness

Systemic disease
- Hypothyroidism (important)
- Hypercalcemia
- Hypokalemia

Localized neuromuscular disease
- Spinal cord trauma
- Pelvic nerve damage
- Dysautonomia
- Chronic, massive dilation of the colon causing irreversible stretching of the colonic musculature

Miscellaneous Causes

Severe dehydration
Idiopathic megacolon (especially cats)

colonic obstruction (e.g., prostatomegaly, enlarged sublumbar lymph node). Ultrasonography is the preferred technique when looking for infiltrates. A serum biochemistry panel may reveal causes of colonic inertia (e.g., hypothyroidism, or rarely hypercalcemia or hypokalemia,).

Colonoscopy is indicated if the clinician suspects an obstruction too orad to be detected by digital examination. Ultrasound-guided fine-needle aspiration of infiltrative colonic lesions sometimes yields diagnostic findings, but colonoscopy (especially rigid) allows a more reliable biopsy specimen to be obtained. If a thorough diagnostic workup fails to identify a cause in a patient with a grossly dilated colon, idiopathic megacolon may be present.

FECAL INCONTINENCE

Fecal incontinence is typically caused by lower motor neuron disease (e.g., cauda equina syndrome, lumbosacral stenosis) or a partial rectal obstruction. Severe irritative proctitis may cause urge incontinence. Animals with rectal obstructions continually try to defecate because the anal canal is filled with feces. Proctitis is suspected on the basis of rectal examination findings and confirmed by proctoscopy and biopsy findings. Neuromuscular disease is suspected if an abnormal anal reflex is found, usually in conjunction with other neurologic defects in the anal, perineal, hindlimb, or coccygeal region. Defects in the coccygeal region are discussed in Chapter 65.

WEIGHT LOSS

Weight loss may be due to any of several categories of problems (Box 26.16). If other problems with more restrictive lists of differentials (e.g., ascites, vomiting, diarrhea, polyuria/polydipsia) are also present, they should usually be investigated first because it may be easier to find the cause. If there are no other concurrent problems that facilitate localization of the disease, then the clinician should determine what the animal's appetite was when the weight loss *began* (Fig. 26.5). Almost any disease can eventually cause anorexia/hyporexia. Weight loss despite an adequate caloric intake (or failure to gain weight despite an excessive caloric intake) usually indicates maldigestion, malabsorption, excessive utilization (e.g., hyperthyroidism, lactation) or inappropriate loss (e.g., diabetes mellitus) of calories. If the patient is hyporexic, it is important to determine if the hyporexia is severe enough that the weight loss can reasonably be attributed to the decreased caloric intake.

The animal's history should be reviewed for evidence of dietary problems, dysphagia, regurgitation, vomiting, or increased use of calories (e.g., lactation, strenuous work, extreme temperatures). Signalments suggestive of particular diseases (e.g., hyperthyroidism in older cats, hepatic failure in young Yorkshire Terriers with signs of portosystemic shunts) should be recognized. It is important to remember that diarrhea may be absent in animals with severe small intestinal disease.

BOX 26.16

Causes of Weight Loss

- **Food**
  - Not enough (especially if there are multiple animals)
  - Poor quality food or low-caloric-density food
  - Inedible
- **Anorexia** (see Box 26.17)
- **Dysphagia** (see Box 26.1)
- **Regurgitation/Vomiting** (i.e., must be losing enough calories to account for weight loss; see Boxes 26.4 to 26.6)
- **Maldigestive Disease**
  - Exocrine pancreatic insufficiency (usually but not always associated with diarrhea)
- **Malabsorptive Disease** (see Box 26.9)
  - Small intestinal disease (may be associated with normal stools)
- **Malassimilation**
  - Organ failure
    - Cardiac failure
    - Hepatic failure
    - Renal failure
    - Adrenal failure
- **Excessive Utilization of Calories**
  - Lactation
  - Increased work
  - Extremely cold environment
  - Pregnancy
  - Increased catabolism resulting from fever/inflammation
  - Hyperthyroidism
- **Increased Loss of Nutrients**
  - Diabetes mellitus
  - Protein-losing nephropathy
  - Protein-losing enteropathy
- **Neuromuscular Disease**
  - Lower motor neuron disease
- **Cancer Cachexia**

Physical examination might identify abnormalities that localize the problem to a particular body system (e.g., nasal disease preventing normal olfaction, dysphagia, arrhythmia suggestive of cardiac failure, weakness suggestive of neuromuscular disease, abnormally sized or shaped organs, abnormal fluid accumulations). Retinal examination may identify inflammatory or infiltrative diseases, especially in cats.

A CBC, serum biochemistry profile, and urinalysis should be done next to search for evidence of inflammation, organ failure, or a paraneoplastic syndrome. Cats should be tested for circulating feline leukemia virus antigen and antibodies to feline immunodeficiency virus. Serum T_4 (and sometimes fT_4) concentrations should be determined in middle-aged to older cats. If clinical pathology data are not helpful, imaging is usually the next step. Thoracic radiographs (ventrodorsal and both lateral views) are important because significant

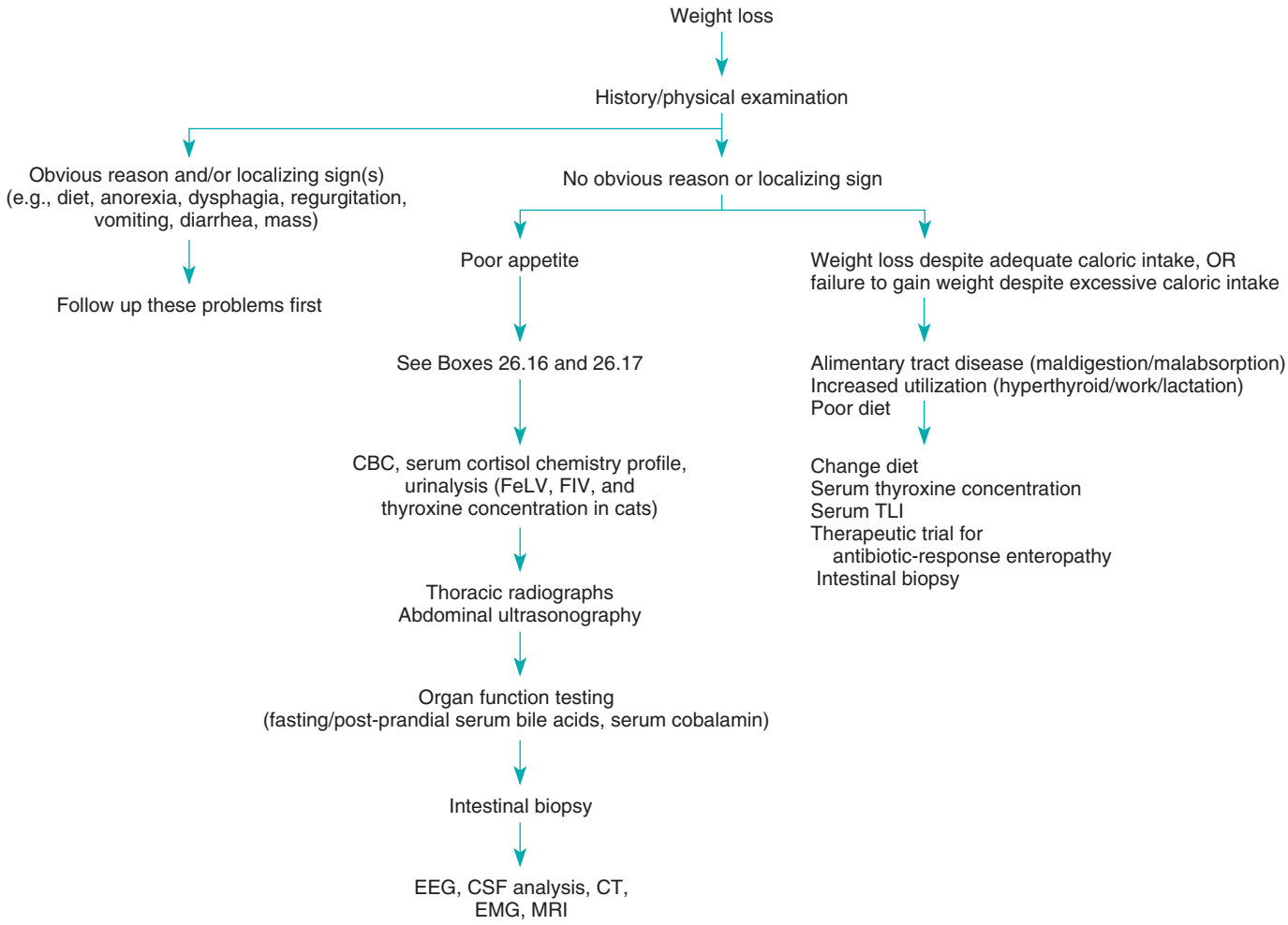

FIG 26.5
General diagnostic approach to weight loss in the dog and cat. *ACTH,* Adrenocorticotropic hormone; *CBC,* complete blood count; *CSF,* cerebrospinal fluid; *CT,* computed tomography; *EEG,* electroencephalography; *EMG,* electromyography; *FeLV,* feline leukemia virus; *FIV,* feline immunodeficiency virus; *MRI,* magnetic resonance imaging.

thoracic disease cannot be ruled out because of normal physical examination findings. Computed tomography (CT) is more sensitive for thoracic disease than plain radiographs, but three-view radiographic examinations are usually adequate. Most cats and some dogs can be palpated well enough that abdominal radiographs are not cost-effective early in the workup. However, abdominal ultrasonography may reveal infiltrative lesions that cannot be palpated or seen radiographically.

If the cause of weight loss remains unknown after these tests, additional diagnostics are necessary. Daily physical examinations can be an important means of localizing the problem. Fever of unknown origin may be noted (see Chapter 90). Organ function testing (e.g., serum bile acid concentrations, serum cortisol, serum TLI, serum cobalamin) is reasonable. Likewise, if serum T_4 concentrations are normal in a cat with suspected hyperthyroidism, the serum fT_4 concentration should be determined or other tests (e.g., nuclear scintigraphy) performed (see Chapter 48).

If the cause of weight loss still remains undiagnosed, the clinician should consider performing therapeutic trials (e.g., for ARE) or gastric/intestinal biopsy. If a laparotomy is performed instead of endoscopy, the entire abdomen should be examined and multiple biopsy samples of the alimentary tract, liver, and mesenteric lymph nodes obtained. Pancreatic biopsy should be considered in cats.

Other possible diagnostic tools include tests to evaluate the CNS (i.e., cerebrospinal fluid analysis, CT, magnetic resonance imaging). Animals that are hyporexic due to CNS disease do not always have obvious cranial nerve deficits or seizures. Peripheral nerves and muscles may be evaluated by serum creatine kinase, electromyography, and muscle/nerve biopsies. Sometimes the weakness associated with neuropathies and myopathies is mistaken for lethargy; see Chapter 59). If the cause of the weight loss still remains undiagnosed and the history and physical examination findings are still noncontributory, occult cancer becomes a major differential diagnosis. In such cases, the clinician may have to wait and

retest later with the hope that the disease will progress enough to be detected.

Causes of weight loss that can be particularly difficult to diagnose include gastric disease not causing vomiting, intestinal disease not causing vomiting or diarrhea, hepatic disease with normal serum chemistries, occult inflammatory disease, atypical hypoadrenocorticism with normal serum electrolyte concentrations, occult cancer, "dry" feline infectious peritonitis, and CNS disease without cranial nerve deficits or seizures.

ANOREXIA/HYPOREXIA

The diagnostic approach and differential diagnoses for animals with hyporexia of uncertain cause is similar to that for animals with weight loss (see Fig. 26.5) (Box 26.17). Inflammatory disease is often detected by the CBC or by finding fever. GI disease may produce hyporexia without vomiting or diarrhea. Cancer cachexia (with anorexia as the predominant sign) may stem from relatively small tumors that are not grossly detectable, although this is rare. Finally, CNS disease must be considered, especially if there is altered mentation. However, altered mentation may resemble the depression and lethargy commonly seen in animals with other diseases.

ABDOMINAL EFFUSION

Abdominal effusion is usually caused by hypoalbuminemia, portal hypertension, and/or increased vascular/lymphatic permeability (i.e., inflammation). Effusions resulting from alimentary tract disorders are primarily caused by PLE (pure low protein transudate due to severe hypoalbuminemia) or alimentary tract rupture (i.e., septic peritonitis). Some animals with PLE have normal stools, ascites being the only abnormality on history or physical examination. Malignant tumors may obstruct lymphatic flow or increase vascular permeability, causing transudates, modified transudates or nonseptic peritonitis. Modified transudates usually result from hepatic or cardiac disease or from abdominal malignancies. For further information on abdominal effusions, see Chapters 33 and 34.

ACUTE ABDOMEN

Acute abdomen refers to various abdominal disorders producing shock (hypovolemic or septic), sepsis, and/or severe pain (Box 26.18). Causes may include alimentary tract obstruction or leakage, vascular compromise (e.g., congestion, torsion, volvulus, ischemia), inflammation, neoplasia, or sepsis. The approach to this problem is determined by the severity of clinical signs (Fig. 26.6).

Shock and gastric dilation or volvulus (GDV) must be identified and treated immediately. Once these conditions are eliminated, the next major decision is whether to perform exploratory surgery or initiate medical therapy. Animals with abdominal masses, foreign objects, bunched-up loops of painful small intestine suggestive of linear foreign body, or spontaneous septic peritonitis should typically undergo surgery as soon as they are acceptable anesthetic risks. If the cause of the acute abdomen is uncertain, it can be difficult to decide whether to do surgery. Surgery is not necessarily beneficial and may be detrimental to animals with pancreatitis, parvoviral enteritis, pyelonephritis, or prostatitis. Typically, abdominal imaging (i.e., plain abdominal radiography,

BOX 26.17

Major Causes of Anorexia/Hyporexia

Inflammatory Disease (Anywhere in Body) (Common and Important)

- Bacterial infections
- Viral infections
- Fungal infections
- Rickettsial infections
- Protozoal infections
- Sterile inflammation
  - Immune-mediated disease
  - Neoplastic disease
  - Necrosis
  - Pancreatitis
- Fever of unknown origin

Dysphagia (Especially Resulting From Pain)

- Nausea (common and important)
  - Stimulation of the medullary vomiting center for any reason but especially gastric or intestinal disease, even if it is not sufficient to cause vomiting (common with gastric disease; see Box 26.6)
- Metabolic Disease
  - Organ failure (e.g., kidney, adrenal, liver, heart)
  - Hypercalcemia
  - Diabetic ketoacidosis
  - Hyperthyroidism (usually causes polyphagia, but some cats have apathetic hyperthyroidism)
- Central Nervous System Disease (often without obvious neurologic abnormalities)
- Cancer Cachexia
- Anosmia (rare)
- Psychological Causes

BOX 26.18

Major Causes of Acute Abdomen

Septic Inflammation

Septic peritonitis (common and important)
- Perforated gastric ulcer (NSAIDs, tumor) (important)
- Perforated intestines (tumor, post-op dehiscence, linear foreign body, severe inflammation) (common and important)
- Devitalized intestines (intussusception, thrombosis/infarct)
- Ruptured gallbladder (e.g., septic cholecystitis, mucocele) (uncommon but important)
- Abscess/infection
  - Splenic
  - Hepatic
  - Cholecystitis
  - Prostatic
  - Renal
- Pyometra (ruptured) (important)

Nonseptic Inflammation

Pancreatitis (common and important)
Uroabdomen (important)
Pansteatitis

Organ Distention or Obstruction

Gastric dilation or volvulus (common and important)
Intestinal obstruction resulting from many causes (common and important)
Intussusception (important, especially younger animals)
Dystocia
Mesenteric volvulus (rare)
Incarcerated obstruction (rare)

Ischemia

Torsion of spleen, liver lobe, testicle, or other organ (rare)
Thromboembolism of abdominal organ(s) (rare)

Other Causes of Abdominal Pain (see Box 26.19)

Abdominal Hemorrhage

Abdominal neoplasia (hemangiosarcoma, hepatocellular carcinoma) (common and important)
Trauma
Coagulopathy (important)

Abdominal Neoplasia

NSAIDs, Nonsteroidal antiinflammatory drugs.

ultrasonography) and clinical pathologic studies (i.e., CBC, chemistry panel) should be performed before a laparotomy is performed. Ultrasound can reveal infiltrates that radiographs cannot detect, sometimes allowing diagnosis via aspiration. However, radiographs occasionally detect lesions (e.g., small foreign bodies, free abdominal gas) that were missed ultrasonographically. Imaging may reveal spontaneous pneumoperitoneum, abdominal masses, foreign objects, alimentary tract obstruction, gastric or mesenteric torsion (these require surgical treatment), or free peritoneal fluid (this requires abdominocentesis and fluid analysis for management). Radiographic contrast series are seldom appropriate and may complicate later therapy/surgery.

If optimal medical therapy is being given and the animal's condition is clearly deteriorating or does not improve after 2 to 5 days of therapy, or if the animal continues to have excruciating pain, it is often appropriate to recommend exploratory surgery. Inform the client that you may discover the animal has a disorder not surgically correctable (e.g., pancreatitis) or that nothing abnormal may be found. In the latter case, the clinician should biopsy various abdominal organs and then treat the animal's symptoms while awaiting biopsy results.

ABDOMINAL PAIN

"Abdominal" pain must first be determined to be abdominal and not extraabdominal in origin (e.g., thoracolumbar pain is often erroneously assessed as being abdominal in origin). An animal with true abdominal pain may show obvious discomfort (e.g., it paces or repeatedly assumes different positions, repeatedly looks at or licks its abdomen) and may whine, growl, or snap if the abdomen is touched. Some dogs stretch out and assume a "praying" position (i.e., the "position of relief"). Other animals have inconspicuous signs (e.g., the animal grunts or tries to walk away when palpated, the abdomen is tensed) that are easily missed. On the other hand, rough abdominal palpation technique in normal animals may elicit a guarding response that can mimic abdominal pain. Main causes of abdominal pain are listed in Box 26.19.

If the patient has abdominal pain, the goal is to determine the source. If the pain is originating from within the abdominal cavity, the diagnostic approach depends on its severity, progression of disease, and whether there are any obvious causes. The steps taken in diagnosing the cause of abdominal pain are similar to those taken in an animal with acute abdomen. Some causes of abdominal pain can be difficult to diagnose (e.g., acute pancreatitis, localized peritonitis).

ABDOMINAL DISTENTION OR ENLARGEMENT

Abdominal distention or enlargement may be associated with an acute abdomen, but these conditions are typically separate problems. It is best to believe clients who claim

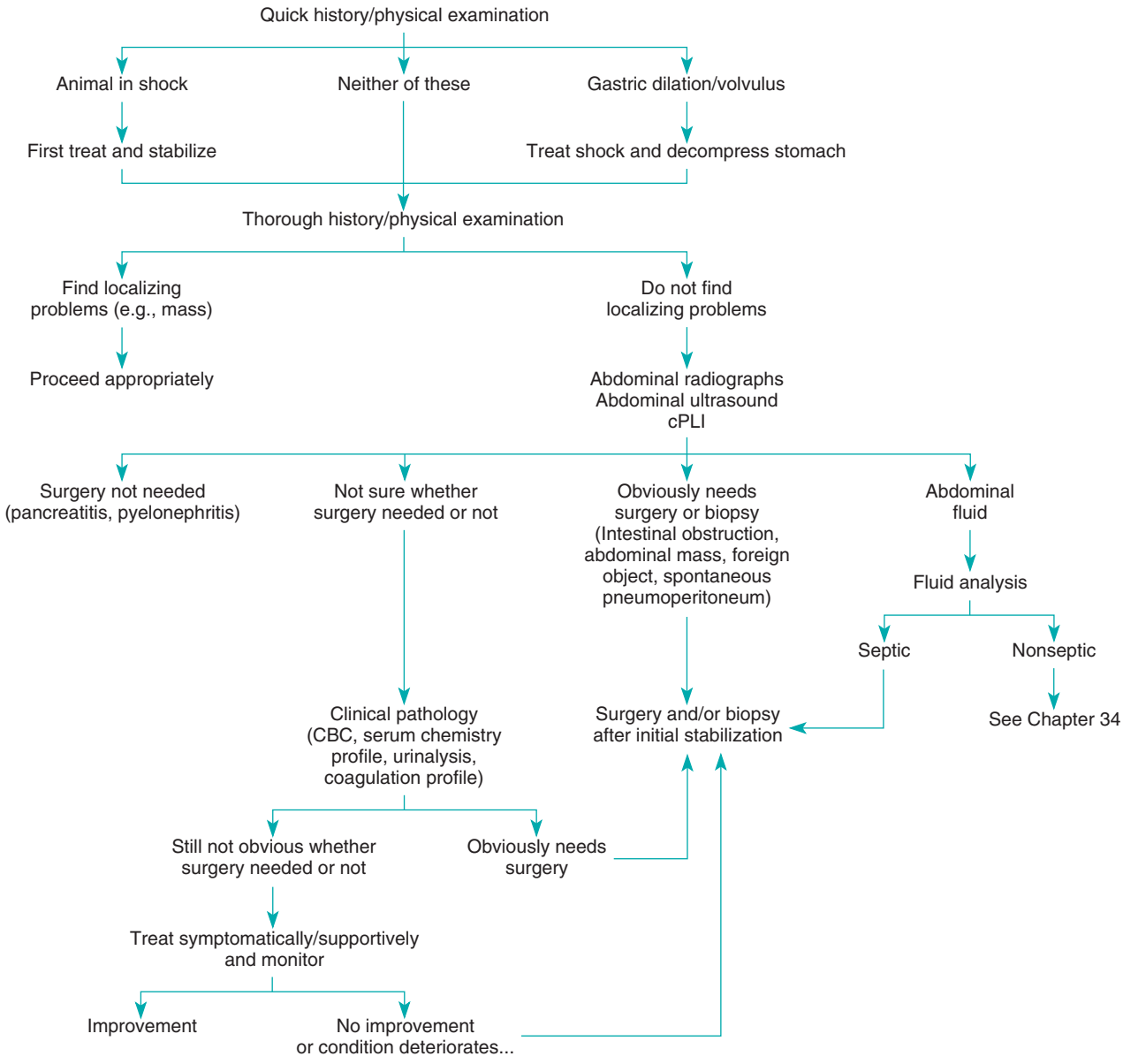

FIG 26.6
General diagnostic approach to acute abdomen in the dog and cat. *CBC,* Complete blood count; *PLI,* pancreatic lipase immunoreactivity.

there is abdominal enlargement until good cause is found to believe otherwise. There are six main causes of abdominal distention (Box 26.20).

The first concern is whether an acute abdomen is present (e.g., GDV, septic peritonitis, hemoabdomen plus shock). After an acute abdomen is ruled out, it should be possible to classify enlargement on the basis of physical examination and abdominal imaging (i.e., radiography or ultrasonography), according to the criteria in Box 26.20. Obesity and pregnancy should be obvious. Specimens of free abdominal fluid should be obtained and analyzed as described in Chapter 34. Biopsy should be performed on abdominal masses and enlarged organs, unless there is a reason not to (e.g., hepatomegaly caused by severe right-sided heart failure). Fine-needle aspiration is typically safe, although leakage of septic contents or implantation of neoplastic cells may rarely occur. Ultrasonography helps determine potential for hemorrhage or leakage (e.g., cyst, mass with ultrasonographic characteristics of hemangiosarcoma). Finding spontaneous pneumoperitoneum suggests alimentary tract rupture or septic peritonitis and is usually an indication for prompt surgical exploration. A hollow viscus dilated with gas may indicate obstruction (i.e., gastric dilation, intestinal obstruction) or physiologic ileus (see p. 420 and pp. 467-468; Figs. 27.5 and 30.3. Surgery is indicated if an obstruction seems likely. If abdominal musculature weakness is suspected, hyperadrenocorticism may be considered. Results of a CBC, serum biochemistry panel, and urinalysis are used to

BOX 26.19

Causes of Abdominal Pain

- Poor Palpation Technique ("Pseudo-pain")
- Musculoskeletal System (Mimics Abdominal Pain)
  - Fractures
  - Intervertebral disk disease (common and important)
  - Diskospondylitis (important)
  - Abscesses
- Peritoneum
  - Peritonitis
    - Septic (common and important)
    - Nonseptic (e.g., uroabdomen) (important)
  - Adhesions (rare)
- Gastrointestinal Tract
  - Gastrointestinal ulcer
  - Foreign object (especially linear)
  - Neoplasm
  - Adhesions (rare)
  - Intestinal ischemia (rare)
  - Intestinal spasm (rare)
  - See also Box 26.18, under Organ Distention or Obstruction
- Hepatobiliary Tract
  - Hepatitis
  - Cholelithiasis or cholecystitis
- Pancreas
  - Pancreatitis (common and important)
- Spleen
  - Torsion (rare)
  - Rupture
  - Neoplasm
  - Infection (rare)
- Urogenital System
  - Pyelonephritis (important)
  - Lower urinary tract infection
  - Prostatitis (important in dogs)
  - Nonseptic cystitis (common and important in cats)
  - Cystic or ureteral obstruction or rupture (common, especially after trauma)
  - Urethritis or obstruction (common)
  - Metritis
  - Uterine torsion (rare)
  - Neoplasm
  - Testicular torsion (rare)
  - Mastitis (does not cause true abdominal pain but mimics abdominal pain)
- Miscellaneous Causes
  - Postoperative pain (especially if animal has a tight suture line)
  - Iatrogenic causes
  - Drugs (e.g., misoprostol, bethanechol)
  - Adrenalitis (associated with hypoadrenocorticism) (rare)
  - Heavy metal intoxication (rare)
  - Vasculopathy (rare)
    - Rocky Mountain spotted fever vasculitis
    - Infarct

BOX 26.20

Causes of Abdominal Enlargement

- Tissue
  - Pregnancy (common and important)
  - Hepatomegaly (infiltrative or inflammatory disease, lipidosis, neoplasia)
  - Splenomegaly (infiltrative or inflammatory disease, neoplasia, hematoma)
  - Renomegaly (neoplasia, infiltrative disease, compensatory hypertrophy)
  - Miscellaneous neoplasia
  - Granuloma (e.g., pythiosis)
- Fluid
  - Contained in organ(s)
  - Congestion resulting from torsion, volvulus, or right-sided heart failure
  - Spleen
  - Liver
  - Cysts
  - Paraprostatic cyst
  - Perinephric cyst
  - Hepatic cyst
  - Hydronephrosis
  - Intestines or stomach (resulting from obstruction or ileus)
  - Pyometra
  - Free in abdomen (common and important)
    - Transudate, modified transudate, exudate, blood, chyle
- Gas
  - Contained in organ(s)
  - Stomach (gastric dilation or volvulus) (common and important)
  - Intestines (resulting from obstruction)
  - In parenchymatous organs (e.g., liver) resulting from infection with gas-producing bacteria
  - Free in abdomen
  - Iatrogenic (after laparoscopy or laparotomy)
  - Alimentary tract or female reproductive tract rupture
  - Bacterial metabolism (peritonitis)
- Fat
  - Obesity
  - Lipoma
- Weak Abdominal Muscles
  - Hyperadrenocorticism (important)
- Feces

look for specific organ involvement (e.g., hyperadrenocorticism). Contrast-enhanced alimentary or urinary tract radiographs are generally not appropriate; ultrasonography typically makes such techniques unnecessary. Sometimes abdominal CT examination will be required.

Suggested Readings

Anderson KL, Feeney DA. Radiography. In: Washabau RW, et al., eds. *Canine and feline gastroenterology*. St Louis: WB Saunders; 2013.

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