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Abdominal enlargement with ascites

Definition

Ascites is the abnormal accumulation of fluid in the peritoneal cavity sufficient to cause observable enlargement to the appearance of the abdomen. In this case, abdominal enlargement is observable to the owner. However, especially in the early, formative stages, ascites may not be associated with abdominal enlargement. Fluid accumulation can result from a variety of inflammatory, infectious, metabolic, degenerative, and neoplastic disorders. Ascites must be distinguished from abdominal enlargement not associated with fluid accumulation (pregnancy, organomegaly, or advanced hyperadrenocorticism).

Associated signs

The clinical history may include increased water consumption and urination, diarrhea, vomiting, increased or decreased appetite, pain, apparent or real weight gain, and loss of muscle mass. Examine the patient for the presence of a heart murmur and palpable arrhythmia. If fluid is not present, determine the presence or absence of a mass within the abdominal cavity. When feasible, analyze the fluid character by physical appearance, biochemical composition, and cytology.

Differential diagnosis (Figure 3-1)

Diagnostic plans

1. Physical examination, to establish or rule out cardiopulmonary disease. Evaluate skin and hair coat for signs supporting endocrine disease (especially hyperadrenocorticism).
2. Verify ascites or abdominal enlargement by ballottement, abdominal radiography, abdominal ultrasound, or abdominocentesis.
3. If fluid is present, abdominocentesis, fluid analysis, and, if available, abdominal ultrasound. A laboratory database also is recommended.

Abdominal enlargement without ascites

Definition

Abdominal enlargement not associated with ascites refers to any condition in a dog or cat that causes a real or apparent enlargement of the abdominal cavity as observed during the physical examination. Real abdominal enlargement can be physiologic or normal (such as postprandial enlargement in a puppy or kitten, pregnancy) or abnormal (such as that associated with organomegaly or obesity).
Figure 3-1: Diagnostic algorithm for the patient with ascites. SG, specific gravity; TP, total protein; TG, triglycerides.
ASSOCIATED SIGNS

Regardless of the underlying cause, abdominal enlargement is most likely to be associated with increased respiratory effort, usually characterized as tachypnea (increased respiratory rate). Dogs are more likely than cats to vocalize during expiration (grunt). Increased heart rate, lethargy, diminished appetite, and orthopnea (positional breathing) are variably observed.

DIFFERENTIAL DIAGNOSIS

| Differential Diagnosis of Abdominal Enlargement |
|-----------------------------------------------|
| **Physiologic Enlargement**                   |
| Postprandial                                  |
| Pregnancy                                    |
| **Without Fluid Accumulation**                |
| Organomegaly                                  |
| Neoplasia                                    |
| Obstipation                                   |
| Gastric dilation                              |
| Hyperadrenocorticism                          |
| Ruptured prepubic tendon                      |
| Bladder distension                            |
| Pneumoperitoneum                              |
| **With Fluid Accumulation**                   |
| High–protein-content fluid: >2.5 g/dL         |
| Hepatic failure                              |
| Right-sided congestive heart failure         |
| Inflammatory-infectious (e.g., feline infectious peritonitis [FIP]) |
| Chemical or drug peritonitis                  |
| Trauma                                        |
| Neoplasia                                     |
| Hepatic vein thrombosis or vascular anomaly   |
| Chyloabdomen                                  |
| Low protein: <2.5 g/dL                        |
| Hypoproteinemia (renal, hepatic, or gastrointestinal cause) |
| Portal hypertension subsequent to primary liver disease |
| Neoplasia                                     |

DIAGNOSTIC PLANS

1. History. Establish duration and progression of abdominal enlargement; in females, establish whether or not pregnancy is possible.
2. Abdominal palpation. *Note:* Preferably accomplished with the patient in right lateral recumbency. Examination is carried out using two hands simultaneously.
3. Abdominal ballottement. Manipulate the abdominal wall in an attempt to determine whether or not an accumulation of fluid exists within the abdomen.
4. Imaging. Abdominal radiograph or abdominal ultrasound.
5. Laboratory profile. Generally conducted to assess patient overall health status.
6. Fine needle aspiration and cytology. Aspiration of solid organs or masses may be indicated.
7. Exploratory surgery. Laparoscopy may be a necessary alternative.

AGGRESSION

DEFINITION

Aggression is a condition (either normal or abnormal) in the dog or cat characterized by threatening, destructive, or attacking behavior. Furthermore, aggression can be categorized as offensive or defensive. Specific knowledge of the pattern and type of aggression is critical if effective intervention is to be accomplished. For the criteria of this definition to be met, it is assumed that organic causes of aggression (e.g., pain or intracranial mass) have been ruled out.
ASSOCIATED SIGNS

Aggression as a presenting problem may be the result of organic disease, particularly disorders affecting the brain. In these patients the onset of aggressive behavior is usually acute and may be associated with other neurologic signs suggesting cerebral dysfunction (e.g., seizures and circling). However, animals with pain may also manifest aggressive behavior, an apparent secondary response to discomfort. Animals with unilateral or bilateral blindness or deafness may bite or manifest aggressive behavior when approached and touched from the blind or deaf side. This behavior is probably the result of the animal's being startled and is far less likely to be representative of abnormal behavior.

DIFFERENTIAL DIAGNOSIS

| AGGRESSIVE BEHAVIOR IN THE DOG: DIFFERENTIAL DIAGNOSIS ACCORDING TO ORIGIN |
|---------------------------------------------------------------|
| **Pathophysiologically Based Aggressive Behavior**            |
| Rabies                                                        |
| Intracranial neoplasia                                       |
| Cerebral hypoxia                                              |
| Seizure activity                                              |
| Neuroendocrine disturbances                                  |
| **Species-Typical Aggressive Behavior**                      |
| Dominance aggression                                         |
| Possessive aggression                                         |
| Protective aggression (food, toys, bedding)                   |
| Predatory aggression                                          |
| Fear-induced aggression                                       |
| Intermale and interfemale aggression                          |
| Pain-induced, punishment-induced, and irritable aggression    |
| Maternal aggression                                           |
| Redirected aggression                                         |

From Young MS: Aggressive behavior. In Ford RB, editor: Clinical signs and diagnosis in small animal practice, New York, 1988, Churchill Livingstone.

*These behavior patterns are not pathologic states. They are typical patterns of the species and are therefore normal. Familiarity with the normal, species-typical aggressive pattern of the dog enables differentiation of species-typical patterns from pathophysiologically based aggression. As with many animal behavior problems, species typicality does not lessen the problem’s disruptiveness or danger.

DIAGNOSTIC PLANS

1. Laboratory profile and neurologic examination to assess the presence of pain or underlying organic disease (intracranial disease).
2. Note: Administration of a psychotropic drug as empiric therapy for aggression is not recommended before determining a possible cause and attempting to modify behavior through training.

| AGGRESSIVE BEHAVIOR IN THE CAT: DIFFERENTIAL DIAGNOSIS ACCORDING TO ORIGIN |
|---------------------------------------------------------------------------|
| **Pathophysiologically Based Aggressive Behavior**                        |
| Rabies                                                                    |
| Intracranial neoplasia and lesions                                        |
| **Species-Typical Aggressive Behavior**                                   |
| Intermale aggression                                                      |
| Predatory aggression                                                      |
| Play aggression                                                           |
| Territorial aggression                                                    |
| Fear-induced aggression                                                   |
| Pain-induced aggression                                                   |
| Maternal aggression                                                       |
| Redirected aggression                                                     |

*These behavior patterns are not necessarily caused by pathologic states. They are characteristic behavior patterns of the species and therefore can be normal. Familiarity with the normal, species-typical aggressive patterns of the cat enables differentiation of species-typical patterns from pathophysiologically based aggression. As with many animal behavior problems, species typicality does not lessen the problem’s disruptiveness or danger.
ALOPECIA See Hair Loss: Alopecia.

ATAXIA See Incoordination: Ataxia.

BLINDNESS See Vision Loss: Total Blindness.

BLOOD IN URINE: HEMATURIA, HEMOGLOBINURIA, MYOGLOBINURIA

DEFINITION
Hematuria is the presence of blood in the urine; the presence of trace amounts of blood in the urine will not be obvious on gross appearance of a urine sample. Therefore any noticeable change in the color of urine observed by the owner is likely to be interpreted as “blood in the urine.” Further evaluation of the patient is necessary to determine whether or not the discoloration is associated with small blood clots in recently voided urine, blood-tinged urine, or brown or red urine. The presence of blood in the urine, whether gross or occult, is most often indicative of upper or lower urinary tract bleeding, although systemic coagulopathies and reproductive tract disorders may also cause hematuria. The presence of hemoglobin in urine (hemoglobinuria) is not necessarily a reflection of urinary tract disease. Systemic disorders (e.g., those leading to intravascular hemolysis) can be associated with significant hemoglobinuria in the presence of a normal urinary system.Owners are likely to interpret this clinical sign to be “blood in the urine.” In true hemoglobinuria, without hematuria, microscopic examination will reveal the absence of red blood cells (RBCs).

Distinguishing hemoglobinuria from hematuria is an important diagnostic consideration. Conventional urine test strips (dipsticks) do not differentiate between the two; therefore microscopic examination of urine sediment for the presence of significant numbers of RBCs is critical.

Myoglobinuria is characterized by brown to dark-red urine, the absence of RBCs in the urine sediment, and a positive finding on testing for occult blood. Bilirubinuria can also cause dark-brown to dark-orange urine but alone will not produce a test result positive for occult blood. Myoglobinuria is a serious sign and denotes generalized muscle disease.

ASSOCIATED SIGNS
Hematuria associated with the urinary tract may not be associated with any other clinical signs. In patients with significant bleeding of renal origin, evidence of systemic illness may be present but is unlikely to localize the source of hematuria. Hematuria originating from the bladder is more likely to be associated with clinical signs, particularly pollakiuria and dysuria. Reproductive tract disorders (e.g., prostatitis and vaginitis) can also cause significant hematuria. Patients with hematuria or hemoglobinuria should be examined carefully for evidence of systemic bleeding, coagulopathies, and neoplasia.

DIFFERENTIAL DIAGNOSIS

DIAGNOSTIC PLANS
1. Thorough history and physical examination, with emphasis on examination of the genitalia, palpation of the prostate, and caudal abdominal palpation.
2. If practical, assessment of urethral patency and the patient’s ability to urinate. Attempt to pass a urethral catheter if significant dysuria and evidence of lower urinary tract obstructions are present.
### Causes of Apparent or Actual Hematuria in Dogs and Cats Classified by Anatomic Site of Origin

| Site                          | Diseases                                                                 |
|-------------------------------|--------------------------------------------------------------------------|
| **Kidney**                    | Pylonephritis                                                            |
|                               | Glomerulonephropathy or glomerulonephritis                               |
|                               | Neoplasia                                                                |
|                               | Calculi                                                                   |
|                               | Renal cysts                                                               |
|                               | Renal infarction                                                          |
|                               | Renal trauma                                                              |
|                               | Benign renal bleeding                                                     |
|                               | Hematuria of Welsh Corgis                                                |
|                               | *Dioctophyma renale* infection                                            |
|                               | Microfilaria of *Dirofilaria immitis*                                     |
|                               | Chronic passive congestion                                               |
| **Bladder, ureter, urethra**  | Infection, inflammation, cystitis, LUTD                                   |
|                               | Cystic calculi                                                            |
|                               | Neoplasia                                                                |
|                               | Trauma                                                                    |
|                               | Thrombocytopenia                                                          |
|                               | *Capillaria plica* infection                                              |
|                               | *Cyclophiliasis*                                                          |
|                               | Transmissible venereal tumor (TVT)                                         |
| **Any site**                  | Coagulopathy                                                             |
|                               | Heat stroke                                                               |
|                               | DIC                                                                       |
| **Extraurinary sources**      | Prostate                                                                  |
| (genital tract or spurious    | Neoplasia                                                                |
| hematuria)                    | Infection                                                                 |
|                               | Hypertrophy                                                              |
|                               | Uterus, pyometra                                                          |
|                               | Estrus                                                                    |
|                               | Subinvolution                                                             |
|                               | Infection                                                                 |
|                               | Neoplasia (including TVT)                                                |
|                               | Vagina                                                                    |
|                               | Trauma                                                                    |
|                               | Penis                                                                     |
|                               | TVT                                                                       |

DIC, Disseminated intravascular coagulation; LUTD, lower urinary tract disease; TVT, transmissible venereal tumor.
3. Complete urinalysis. Using a fresh sample, include assessment of gross appearance, specific gravity, biochemical reagent strips (dipsticks), and microscopic examination of urine sediment. Ideally, two samples should be collected: a voided urine sample followed by a urine sample collected by cystocentesis.

4. Culture and sensitivity, if bacteria are present.

5. Routine laboratory profile, to include hematology and biochemistry panel.

6. Coagulation profile, if hemoglobinuria is present.

7. Abdominal radiographs, for evidence of calculi, prostatic enlargement, and soft tissue masses.

8. Contrast radiography of the upper and lower urinary tracts.

9. Ultrasound examination of the prostate, urinary bladder, and kidneys.

10. Exploratory laparotomy (if coagulation profile is normal).

**COMA: LOSS OF CONSCIOUSNESS**

**DEFINITION**

Coma is a state of complete reversible or irreversible unconsciousness that can result from neurologic as well as nonneurologic disease (drug overdose, especially in dogs). Coma can be a consequence of diffuse or multifocal lesions of the cerebrum or a lesion affecting the rostral brainstem and ascending reticular activating system. A variety of organic central nervous system (CNS) diseases leading to metabolic or toxic encephalopathy can also produce coma.

**ASSOCIATED SIGNS**

Despite the fact that the comatose patient is unconscious, a complete neuroophthalmologic examination should be completed. Altered pupil size and pupillary light responses usually indicate brainstem disease. Emergency cardiac assessment of the unconscious patient justifies an electrocardiogram (ECG) and thoracic radiographs. Laboratory assessment of the comatose patient includes hepatic enzymes and, when feasible, hepatic function, electrolytes, and glucose level.

**DIFFERENTIAL DIAGNOSIS**

| Differential Diagnosis of Coma | Neurogenic | Nonneurogenic |
|-------------------------------|------------|---------------|
| Acute, nonprogressive         | Intracranial hemorrhage | — |
| Acute, progressive            | Metastatic lesions | Hypoglycemia |
| Acute, progressive            | Epidural, subdural hemorrhage | Diabetic coma (hyperosmotic) |
| Acute, progressive            | Meningoencephalitis | Heat stroke |
| Acute, progressive            | Cerebral edema | Hepatic or uremic encephalopathy |
| Chronic, progressive          | Hemorrhage (rare) | Infection |
| Chronic, progressive          | Storage diseases | Hypoxia |
| Chronic, progressive          | Hydrocephalus | Thiamine deficiency (cat) |
| Chronic, progressive          | Encephalitis | Heavy metal and drug toxicity |
| Chronic, progressive          | | Carbon monoxide poisoning |

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| Acute, nonprogressive         | Intracranial hemorrhage | — |
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| Chronic, progressive          | Storage diseases | Hypoxia |
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| Chronic, progressive          | Encephalitis | Heavy metal and drug toxicity |
| Chronic, progressive          | | Carbon monoxide poisoning |
CONSTIPATION (OBSTIPATION)

DIAGNOSTIC PLANS

1. Critical: Assessment of vital signs to evaluate airway, breathing, and circulation (pulse, heartbeat, and ECG). Take thoracic radiographs if indicated. If cerebral edema is suspected, administer ventilation support, intravenous hyperosmotic agents (e.g., mannitol 20%, 1 to 2 g/kg of body weight q6h), and glucocorticoids.
2. Conduct careful neurologic examination directed toward evaluation of brainstem function, including motor function, pupillary light responses (or lack thereof), and eye movement.
3. Comprehensive laboratory profile, to include hematology, biochemical profile, and urinalysis.
4. Special diagnostic tests as appropriate:
   a. Metabolic coma: Serum ammonia, bile acids, glucose, blood and urine lead levels
   b. Neurologic coma: Skull radiographs, cerebral spinal fluid analysis, electroencephalography
   c. Assessment of response to intravenous mannitol.

CONSTIPATION (OBSTIPATION)

See also Straining to Defecate: Dyschezia.

DEFINITION

Constipation is the infrequent or difficult passage of feces. Obstipation is intractable constipation resulting in fecal impaction through the rectum and possibly the colon. The act of straining to defecate or painful defecation, the likely manifestation of constipation or obstipation, typically represents the reason for which a constipated dog or cat is presented (see also Straining to Defecate: Dyschezia).

There is no strict definition of bowel regularity; therefore there is no “normal” number of daily or weekly bowel movements, deviations from which constitutes constipation. Practically, constipation can be considered to exist when a significant delay in frequency of passing formed stools has been noted or when the stool is observed to be of unusually hard or dry consistency. Constipation is categorized under one of the following headings: neurogenic; mechanical (physical); muscular (smooth muscle); or iatrogenic (drug-induced).

The owner who perceives a pet as straining to defecate may, in fact, be observing a pet that is straining to urinate. This is particularly true in cats with disorders of the lower urinary tract, such as feline urologic syndrome (FUS). In the context of this discussion, dyschezia is discussed only insofar as it is associated with constipation and obstipation (Figure 3-2).

ASSOCIATED SIGNS

Assessment of the patient presented because of constipation or obstipation can represent a significant medical challenge because of the complex and varied pathogenic mechanisms involved. Animals with neurogenic causes of constipation may have significant perianal or rectal pain associated with focal lesions. Other patients may have nonpainful neurologic disease or long-term complications stemming from previous pelvic or spinal trauma.

Mechanical causes are either extraluminal or intraluminal. Abdominal and rectal palpation is indicated in both male and female dogs and cats. Narrow or blood-tinged feces may signal the presence of an intraluminal lesion, whereas in patients with extraluminal lesions, associated clinical signs may not be present.

Muscular causes are the least common and are generally the result of extreme metabolic aberrations. Idiopathic colonic atony is reported, but constipation may also result from severe catabolic states. Laboratory evidence of endocrine disease and electrolyte abnormalities should be assessed.

DIFFERENTIAL DIAGNOSIS

DIAGNOSTIC PLANS See Figure 3-2.
Figure 3-2: Clinical algorithm for constipation in the dog or cat. EMG, Electromyogram.
**COUGH**

**DEFINITION**
A cough is a sudden, forceful expiratory response to irritating stimuli (e.g., secretions) situated in the tracheobronchial tree. Cough is the most frequent clinical presentation (followed by dyspnea and hemoptysis) that is referable to the lower respiratory tract. At presentation, cough should be characterized as "acute-onset" (duration of only a few days) or "chronic" (duration longer than 2 weeks). Attempting to characterize cough as productive or nonproductive is difficult in animals and therefore has little value in the overall diagnostic plan.

**ASSOCIATED SIGNS**
Although cough is a principal sign of lower respiratory tract disease, particularly lower airway (tracheal and bronchial) disease, it may also occur in animals with nonpulmonary disease, particularly cardiac and intrathoracic diseases. Associated signs, therefore, may include a wide spectrum of findings; there may also be no associated signs. Particular attention should be given to determining the character of the cough: it can be paroxysmal and severe, which usually indicates the need for immediate intervention, or mild but persistent. Animals in need of immediate attention are those with cough associated with syncope, dyspnea, or hemoptysis. Orthopnea, the inability to breathe without assuming a particular (usually upright) position, is a serious sign that suggests compromised respiratory function and also warrants immediate attention. Nasal discharge, tachypnea, and hyperpnea are less commonly associated with cough. Cough can be misinterpreted by the owner as vomiting, particularly in dogs with infectious airway disease.

---

**DIFFERENTIAL DIAGNOSIS OF CONSTIPATION**

| NEUROGENIC CAUSES | MUSCULAR CAUSES | MECHANICAL CAUSES | DRUG-INDUCED CAUSES |
|-------------------|----------------|-------------------|---------------------|
| Cortical (pain-induced constipation) | Colonic atony | Extraluminal | Anesthetics |
| Perianal neoplasia | Severe malnutrition and cachexia | Prostate (neoplasia or hyperplasia) | Anticholinergics (e.g., atropine) |
| Anal sac disease | Hypothyroidism | Large intraabdominal tumors | Anticonvulsants |
| Perianal fistulas | Hypercalcemia | Pregnancy (?) | Barium sulfate |
| Myiasis | Hyperkalemia | Pelvic fracture | Diuretics |
| Central nervous system disease | Hyperparathyroidism | Rectal stricture (e.g., adenocarcinoma) | Prolonged laxative therapy |
| Spinal trauma | Segmental dilation subsequent to surgery | Colonic stricture | Monoamine oxidase inhibitors |
| Spinal neoplasia | | Granulomas (e.g., histoplasmosis) | Heavy metal toxicity (e.g., lead) |
| Degenerative myelopathy | | Benign colorectal tumors | Behavioral factors |
| Peripheral nerve disease (e.g., complication after pelvic trauma) | | Fecalith | Soiled or odiferous litter |
| | | Rectal-colonic prolapse | No litter available |
| | | Intussusception | |
DIFFERENTIAL DIAGNOSIS

DIFFERENTIAL DIAGNOSIS OF COUGH

PRIMARY RESPIRATORY TRACT DISEASE
Canine infectious respiratory disease (CIRD; formerly “kennel cough”); multiple viruses and bacteria may be involved
Tonsillitis and pharyngitis
Tonsillar neoplasm
Pharyngeal polyp (cat)
Laryngeal cyst
Laryngeal neoplasm
Laryngeal paralysis
Tracheal hypoplasia (usually with secondary tracheitis)
Segmental tracheal stenosis
Tracheal collapse—acquired and congenital
Tracheal neoplasia
Tracheal osteochondral dysplasia
Foreign body
Bronchiectasis
Bronchial collapse
Immotile cilia syndrome
Aspiration
Respiratory parasites (e.g., Capillaria aerophila in cats; Filaroides osleri in dogs)

PULMONARY VASCULAR DISEASE
Pulmonary edema (multiple causes)

PULMONARY PARENCHYMAL DISEASE
Pulmonary hypertension, especially heartworm disease

PRIMARY RESPIRATORY TRACT DISEASE
Bacterial pneumonia
Systemic mycoses (e.g., histoplasmosis)
Pulmonary neoplasia
Pulmonary abscess
Protozoan pneumonia (e.g., feline toxoplasmosis)
Viral pneumonia
Allergic pneumonitis (e.g., feline asthma)
Metabolic and endocrine disease (e.g., hyperadrenocorticism)

CARDIOVASCULAR DISEASE
Left-sided heart disease
Left-sided heart failure (cardiogenic pulmonary edema)

INTRATHORACIC DISEASE
Mediastinal abscess
Mediastinal neoplasia

DIAGNOSTIC PLANS
1. History and physical examination. Focus on recent exposure risk (boarding) and heartworm preventative in dogs. Physical examination is particularly valuable in determining the extent of respiratory tract involvement and characterizing the type of cough present, particularly when the cough can be elicited by manipulation of the cervical trachea.
2. Careful thoracic auscultation to determine the presence or absence of heart murmur or abnormal lung or airway sounds.
3. Thoracic radiographs using lateral and ventrodorsal projections are critical, particularly when the patient has associated signs compatible with respiratory distress. Oxygen should be available to the dyspneic patient throughout the radiographic procedure. Radiographs should be carefully reviewed for changes in vascular, cardiac, and airway patterns. Patients suspected of having thoracic neoplasia should have left and right lateral thoracic radiographs assessed.
4. A laboratory profile, to include hematology, biochemistry panel, fecal flotation, urinalysis, heartworm test, and feline leukemia virus and feline immunodeficiency virus (FeLV/FIV) test in the cat.
5. Special diagnostics:
   a. Primary respiratory disease: transtracheal aspiration, bronchial lavage, bronchoscopy, contrast bronchography, fluoroscopy, and radionuclide assessment of mucociliary transport
   b. Primary pulmonary disease: fine-needle lung aspiration, arterial blood gases, fungal serology, nuclear studies (perfusion-ventilation), lung biopsy
   c. Primary cardiac disease: ECG, echocardiogram (M-mode and two-dimensional), and nonselective angiography
COUGHING BLOOD: HEMOPTYSIS

See also Difficulty Breathing.

DEFINITION

Hemoptysis is the expectoration, during cough, of blood. Seldom is the volume of blood loss sufficient to cause anemia; however, once confirmed, hemoptysis is a severe clinical finding indicative of bleeding into or from the lower airways. Hemoptysis can be attributed to direct injury of the pulmonary or, less commonly, the tracheobronchial blood vessels; pulmonary hypertension; or coagulopathy. Although an uncommon presenting sign, hemoptysis is more prevalent in dogs than in cats.

Because vomiting can be mistaken by the owner for coughing, it becomes essential to differentiate between hemoptysis and hematemesis during the initial examination. Hemoptysis is regarded as an emergency presentation.

ASSOCIATED SIGNS

The most common, and least significant, sign associated with hemoptysis is melena, or dark-red or black discoloration of stool that occurs subsequent to swallowing expectorated blood. More serious associated signs include coughing, hyperpnea, orthopnea, and cyanosis. Apparent episodic weakness and collapse may also be reported.

DIFFERENTIAL DIAGNOSIS

| CARDIOVASCULAR HEMOPTYSIS | INFLAMMATION-INDUCED HEMOPTYSIS |
|---------------------------|---------------------------------|
| Thromboembolic disease    | Chronic bronchitis               |
| Heartworm disease (in the dog and cat) | Pneumonia                  |
| Hyperadrenocorticism      | Mycotic lung infection          |
| Cardiomyopathy            | Lung abscess                    |
| Renal amyloidosis (in the dog) |                             |
| Idiopathic hemoptysis     | Neoplasm                        |
| Acute pulmonary edema     | Either primary or metastatic neoplasia |
| Arteriovenous fistula     | Neoplasia                       |

| PARASITIC HEMOPTYSIS |
|----------------------|
| Lung flukes (e.g., Paragonimus species) |
| Lungworms (e.g., Aelurostrongylus species) |

DIAGNOSTIC PLANS

1. Thorough history and physical examination. In addition, an attempt should be made to determine that the sign for which the patient was presented is, in fact, expectoration of blood during coughing and not bloody vomitus.
2. Routine laboratory profile, to assess the patient’s overall health status. Emphasis should be placed on the fecal examination and heartworm tests. Multiple attempts to locate parasite ova in the stool should be made, because lung parasites may be few in number and ova shed intermittently.
3. Thoracic radiographs (especially for evidence of advanced heartworm disease in dogs).
4. Coagulation profile, particularly in those animals with significant bleeding from other sites.
5. Transtracheal aspiration with cytologic studies or bacterial culture and sensitivity tests, or both.
6. Special procedures, including ultrasonography of the lung, particularly when discrete masses are seen on radiographs; echocardiography; blood gas analysis; bronchoscopy; bronchography; and angiography.
7. Radionuclide scans. Although availability is limited, studies may detect areas of pulmonary embolization.

**DEAFNESS OR HEARING LOSS**

**DEFINITION**

Deafness is the detectable lack or loss (complete or partial) of the sense of hearing. Deafness can result from abnormalities at any one of several levels from the ear to the brain. Peripheral deafness is categorized as either conduction deafness, involving abnormalities of the transduction apparatus (external ear canal, tympanic membrane, auditory ossicles in the middle ear), or nerve deafness, involving the hearing receptors in the cochlea or the auditory branch of the eighth cranial nerve. Congenital deafness is usually nerve deafness and is the result of abnormal development of the middle and/or inner ear. Central hearing loss (intracranial cause) is uncommon.

Loss of hearing, either complete or partial, in one or both ears does occur in both dogs and cats but is particularly difficult to confirm. Partial loss of hearing occurs most commonly in older animals and is noted by owners as decreased response to voice or noise (e.g., thunder).

**ASSOCIATED SIGNS**

Although rare, invasive lesions or panencephalitis could conceivably cause central hearing loss. However, the associated neurologic signs would be extensive, and hearing loss becomes a secondary or insignificant clinical issue.

Animals with peripheral hearing loss caused by acquired unilateral lesions (severe otitis externa) may manifest a variety of signs referable to the inner ear, particularly head tilt and, less often, circling. Pain or increased sensitivity may be associated with infectious lesions affecting hearing in either ear. Physical evidence of otitis externa is readily detected during routine examinations. Severe swelling associated with a chronic inflammation, a ruptured or damaged tympanic membrane, and infections of the middle ear may effectively decrease hearing acuity. Hypothyroidism may also be associated with degeneration of the cochlea and subsequent decrease in hearing acuity. The clinical history is important and should include any prior exposure to drugs known to be toxic to the cochlear nerve and organ of Corti (e.g., aminoglycoside therapy).

Congenital (hereditary) deafness is associated with a white or merle hair coat in both dogs and cats. In dogs, the highest incidence occurs in the Dalmatian. However, several breeds are reported to be affected.

**DIFFERENTIAL DIAGNOSIS**

| Differential Diagnosis of Deafness |
|-----------------------------------|
| **ACQUIRED HEARING LOSS**          |
| Degenerative causes               |
| Neurogenic deafness in the geriatric dog and cat |
| Hearing loss occurring subsequent to chronic inflammatory disease (middle and inner ear structures) |
| Metabolic (endocrine) cause        |
| Hypothyroidism                     |
| Neoplastic cause                   |
| Invasive tumors of the pharynx and retropharyngeal tissue |
| Infectious-inflammatory causes     |
Decreased Urine Production: Oliguria and Anuria

Definition
Oliguria is a reduced amount of urine production and output in relation to fluid intake. Patients in which urine production ceases have anuria and are considered to be anuric. In contrast to polyuric states, neither oliguria nor anuria is likely to be the primary problem for which a dog or cat is presented. The metabolic consequences of decreased urine production are severe and generally represent significant compromises in renal blood flow or in the functional status of a critical nephron mass. The daily urine volume at which oliguria begins is a function of solute load and renal concentrating ability. In general, oliguria exists when daily urine production is reduced by 75% or more. Production of 0.5 to 1.0 mL of urine per kilogram per hour indicates adequate renal perfusion in the dog. Anuria begins or terminates with oliguria; therefore early detection and treatment of the underlying cause are critical to the overall prognosis.

Associated Signs
The problem(s) for which an oliguric or anuric patient is presented will likely be related to the metabolic consequences of compromised renal function. Uremia, characterized by vomiting, hematemesis, diarrhea, lethargy, or anorexia, predominates. Any one or a combination of signs may present at the time of initial examination. Some patients may be presented in a comatose or semiconscious state, in which case it is essential that renal function and urinary output be established immediately.

Because acute renal failure (ARF) is the principal differential diagnosis in oliguria and anuria, once it has been established the clinician must obtain a thorough clinical history and laboratory profile, including urinalysis if possible, in an attempt to determine the cause of renal failure and to institute corrective therapy.

Diagnostic Plans
1. Assessment of response to noise while the animal is relaxed or asleep.
2. Thorough physical examination, particularly of the external ear canal and tympanic membrane.
3. Otoscopic or videoscopic examination in the anesthetized patient.
4. Neurologic examination.
5. Assessment of thyroid hormone levels.
6. Radiography or computed tomography (CT) of the head, with particular emphasis on the tympanic bullae, for evidence of otitis media.
7. Electrophysiologic studies, including electroencephalography, tympanometry, and brainstem auditory evoked potentials (BAER test).

Otitis (externa, media, and/or interna)
Canine distemper virus infection
Protothecosis (in the dog)
Toxic cause
Aminoglycoside antimicrobials, especially gentamicin, streptomycin, and neomycin
Traumatic hearing loss
Idiopathic hearing loss

Congenital Hearing Loss—Breed Predisposition
White, blue-eyed cats (may be unilateral or bilateral)
Several breeds affected, particularly those with a white or merle hair coat
DIFFERENTIAL DIAGNOSIS

DIFFERENTIAL DIAGNOSIS OF ACUTE RENAL FAILURE

| INFLAMMATORY-INFECTIOUS CAUSES | Extreme dehydration |
|-------------------------------|---------------------|
| Leptospirosis                 | Hemorrhage          |
| Pyelonephritis                | Trauma              |
| Immune complex glomerulonephropathy | Sepsis    |
| Systemic lupus erythematousus | Surgery            |
| Heartworm disease             | Thromboembolic diseases |
| Pyometra                      |                      |
| Endocarditis                  |                      |
| Feline leukemia virus infection|                      |
| Lyme borreliosis (uncommon outside of endemic areas) | |

Viral causes

- Canine distemper virus infection
- Infectious canine hepatitis infection (rare in the United States)
- Canine herpesvirus infection (rare)

PRIMARY RENAL CAUSES (NEPHROSSES)

- Hypoperfusion (ischemia)

DIAGNOSTIC PLANS

1. Initiation of fluid therapy and placement of an indwelling urinary catheter, to establish the rate of urine production.
2. History, to address any possible exposure to toxins, particularly antifreeze, as well as recent drug treatment.
3. Radiographs of the abdomen. These may reveal enlarged kidneys, thereby supporting a diagnosis of ARF. Do not rule out the diagnosis of ARF if kidney size appears normal. Ultrasound imaging of kidneys is also helpful in establishing diagnosis.
4. Complete blood count (CBC). The biochemical profile should include electrolytes as well as blood urea nitrogen (BUN) and creatinine levels. Urinalysis (must include urine specific gravity) with microscopic examination of sediment for evidence of crystalluria, RBCs, white blood cells (WBCs), and casts is essential even if only a small volume of urine can be obtained.
5. Blood gases, to assess for metabolic acidosis, which may be severe in ARF.
6. Urine protein-creatinine ratio, to assess proteinuria.
7. If possible, determinations of serum osmolality and serum osmolar gap.
8. Special diagnostics: intravenous pyelogram (IVP), renal biopsy, and determinations of lead and other heavy metals in the blood as indicated.

DIARRHEA, ACUTE-ONSET

DEFINITION

A definition of acute-onset diarrhea is probably unnecessary; when it happens, you know it! The condition involves a sudden change in bowel pattern, characterized as increased fluidity, frequency, or volume, that is sustained despite empiric or supportive therapy (see also Diarrhea, Chronic). Fundamentally, diarrhea occurs when the amount of water and other intestinal contents reaching the colon exceed the ability of the colon to store the feces and adequately remove the excess water. The pathogenesis of acute diarrhea may be classified as osmotic diarrhea, abnormal gut permeability, secretory diarrhea, or abnormal bowel motility.
In the patient with acute diarrhea, it is conceivable that only one of these mechanisms is involved. However, the longer the underlying cause of the diarrhea persists, the more likely that homeostatic and compensatory mechanisms will be overwhelmed. The pathogenesis of the patient’s diarrhea is then related to a combination of events.

**ASSOCIATED SIGNS**

Acute diarrhea is a common presenting sign for which multitudes of diagnostic possibilities exist. The list of associated signs can be, in the clinical setting, extensive. Among the most common signs encountered in an animal presented with acute diarrhea are vomiting, dehydration, slight weight loss, and hematochezia. Abdominal pain, halitosis, flatulence, and borborygmus are other gut-associated signs. However, not all patients with acute diarrhea have primary intestinal disease, such as those with renal or hepatic failure or hypoadrenocorticism; icterus, oral ulcers, muscle weakness, and so on may also be encountered. Fever, anorexia, and lethargy may also accompany acute diarrhea in the dog and cat.

**DIFFERENTIAL DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS FOR ACUTE-ONSET DIARRHEA**

| INFECTIOUS CAUSES | TOXIC CAUSES |
|-------------------|--------------|
| Intestinal parasites: Nematodes (e.g., ascarids, hookworms, whipworms, Strongyloides, Trichinella); protozoa (e.g., Coccidia, Giardia, Cryptosporidium, Pentatrichomonas) | Antimicrobials or antibiotics, parasiticides, antineoplastic agents, heavy metals, insecticides, organophosphate-containing compounds, antiinflammatory drugs |
| Bacterial: Escherichia coli, Salmonella, Pseudomonas, Clostridium, Campylobacter, Yersinia enterocolitica, Staphylococcus, Helicobacter (?) | |
| Viral: Paramyxovirus (canine distemper), parvovirus (feline and canine), adenovirus 1; corona-virus and reovirus—minor or insignificant) | |
| Rickettsial: Salmon poisoning | |

| DIETARY CAUSES | BOWEL OBSTRUCTION | EXTRaintestinal CAUSES* |
|---------------|------------------|------------------------|
| Dietary indiscretion, engorgement, food hypersensitivity, sudden change in diet | Foreign body, intussusception, volvulus, neoplasia | Renal failure, hepatic disease, hypoadrenocorticism (Addison disease), pancreatitis (acute and chronic) |

| IDIOPATHIC CAUSES |
|-------------------|
| *Although characteristically associated with chronic disease, the onset of diarrhea may be acute. |

**DIAGNOSTIC PLANS**

1. History and physical examination, including abdominal palpation. Establish possible exposure to infectious agents and associated signs.
2. Intravenous fluids containing NaCl may be a critical part of the early evaluation (signs associated with hypoadrenocorticism or Addison disease may resolve within minutes to hours) in severely dehydrated patients presented with acute diarrhea.
3. Laboratory profile (to include routine hematology), biochemistry profile (to include amylase or lipase and sodium and potassium), urinalysis, examination of feces (direct and flotation). Perform several examinations before ruling out parasitic disease. Cats should be tested for FeLV and FIV. Dogs should be tested for parvovirus antigen in stool.

4. Abdominal radiographs.

5. Special diagnostic tests as indicated: abdominal ultrasound; endoscopy and mucosal biopsy; stool culture for viruses or bacteria; serologic studies for rickettsial, viral, and fungal disease; and abdominal laparotomy.

### DIARRHEA, CHRONIC

**DEFINITION**

Chronic diarrhea is a persistent or gradual change in bowel pattern, characterized by increased fluidity, frequency, or volume of stool, that is sustained for more than 1 to 2 weeks despite empiric or supportive therapy (see also Diarrhea, Acute-Onset). In the clinical setting, the clinical history and associated signs should be used to further characterize chronic diarrhea as large-bowel or small-bowel diarrhea.

**ASSOCIATED SIGNS**

Clinical differentiation of small-bowel and large-bowel diarrhea is fundamentally important for the diagnosis and treatment of chronic diarrhea (Table 3-1).

Less specific signs associated with chronic diarrheal diseases include dehydration, poor-quality hair coat, and fever. On abdominal palpation, discrete masses, thickened bowel loops, pain, or gas may occasionally be detected. Edema, ascites, and pleural effusion in patients with chronic diarrhea suggest substantial protein losses through the bowel. The patient with pallor should be assessed for intestinal bleeding, as well as for an anemia of chronic inflammatory disease.

Hematologic signs of greatest significance include eosinophilia (allergic or inflammatory) and significant lymphopenia (lymphangiectasia). Hypoproteinemia is associated with extreme malnutrition, protein-losing enteropathies, and enteric blood loss. Hyperglobulinemia is associated with Basenji enteropathy and feline infectious peritonitis (FIP).

**Table 3-1 Clinical Differentiation of Diarrhea of the Small Bowel and Large Bowel**

| Clinical Signs                  | Small-Bowel Diarrhea                                      | Large-Bowel Diarrhea                                    |
|--------------------------------|-----------------------------------------------------------|---------------------------------------------------------|
| Fecal volume                   | Markedly increased daily output (large quantity of bulky | Normal or slightly increased daily output (small quantities with each defecation) |
|                                | or watery feces with each defecation)                     |                                                          |
| Frequency of defecation        | Normal or slightly increased                             | Very frequent: 4-10 times per day                       |
| Urgency of tenesmus            | Rare                                                      | Common                                                  |
| Mucus in feces                 | Rare                                                      | Common                                                  |
| Blood in feces                 | Dark black (digested)                                     | Red (fresh)                                             |
| Steatorrhea (malassimilation)   | May be present                                            | Absent                                                  |
| Weight loss and emaciation     | Usual                                                     | Rare                                                    |
| Flatulence                     | May be present                                            | Absent                                                  |
| Vomiting                       | Occasional                                                | Occasional                                              |
### DIAGNOSIS OF SPECIFIC CHRONIC DIARRHEAL DISORDERS

| Diarrhea                                             | Diagnostic Test or Procedure                                                                 |
|------------------------------------------------------|---------------------------------------------------------------------------------------------|
| **SMALL-BOWEL TYPE**                                 |                                                                                             |
| Exocrine, pancreatic insufficiency                    | Serum trypsin-like immunoreactivity (TLI)                                                   |
| Chronic inflammatory small bowel disease              |                                                                                             |
| Eosinophilic enteritis                                | Eosinophilia, biopsy                                                                        |
| Lymphocytic-plasmacytic enteritis                     | Biopsy                                                                                      |
| Immunoproliferative enteropathy of Basenjis           | Serum protein electrophoresis                                                               |
| Granulomatous enteritis                               | Radiography, biopsy                                                                         |
| Lymphangiectasia                                      | Lymphopenia, intestinal biopsy, and total protein and lymphocyte count                      |
| Villous atrophy                                       |                                                                                             |
| Gluten enteropathy                                    | Response to gluten-free diet                                                                |
| Idiopathic                                            | Biopsy                                                                                      |
| Histoplasmosis                                        | Serology, cytology, biopsy                                                                  |
| Lymphosarcoma                                         | Biopsy and cytology                                                                         |
| Small intestinal bacterial overgrowth (SIBO)          | Culture of intestinal aspirate, folate, response to antibiotics                             |
| Giardiasis                                            | Fecal examinations, response to parasiticides                                               |
| Lactase deficiency                                    | Response to lactose-free diet                                                                |
| **LARGE-BOWEL TYPE**                                  |                                                                                             |
| Chronic colitis                                       | Colonoscopy, colon biopsy (multiple samples are required)                                   |
| Idiopathic                                            |                                                                                             |
| Histiocytic                                           |                                                                                             |
| Eosinophilic                                          |                                                                                             |
| Whipworm colitis                                      | Fecal flotation, colonoscopy, response to fenbendazole                                       |
| Protozoan colitis                                     | Saline fecal smears                                                                         |
| Amebiasis                                             |                                                                                             |
| Balantidiasis                                         |                                                                                             |
| Trichomoniasis                                        |                                                                                             |
| *Histoplasma* colitis                                 | Fecal cytology, colon biopsy, serology, culture                                              |
| *Salmonella* colitis                                  | Culture                                                                                     |
| *Campylobacter* colitis                               | Culture                                                                                     |
| *Prototheca* colitis                                  | Colon biopsy                                                                                |
| Tritrichomonads                                       |                                                                                             |
| Rectocolonic polyps                                   | Digital palpation, barium enema                                                             |
| Colonic adenocarcinoma                                | Colonoscopy, barium enema, possibly abdominal ultrasound                                    |
| Colonic lymphosarcoma                                 | Barium enema, colonoscopy                                                                   |
| Functional diarrhea (irritable colon)                 | History, diagnostic workup excludes all other diseases                                       |
Diagnostic Plans

1. Clinical history and physical examination findings, to classify the diarrhea as small bowel or large bowel. Routine patient screening should include hematologic studies, biochemical profile, fecal flotation and direct examination, and urinalysis.

2. Diagnosis of intestinal parasites. Perform a visual examination of the feces and anus for proglottids, a zinc sulfate flotation test for Giardia and Coccidia cysts, a saline suspension for protozoan trophozoites, and a sedimentation or Baermann determination for Strongyloides larvae. Adult whipworms can be seen in the colon on colonoscopy.

3. Additional fecal studies. Beyond routine fecal flotation and direct examination, several other fecal tests are indicated, including microscopic examinations for fat (Sudan preparation), starch (iodine preparation), and cytologic staining (Gram stain and Wright stain) to assess for presence of leukocytes and infectious agents. Malassimilation can be assessed through quantitative fecal fat analysis and fecal weight (daily output), although in clinical practice these tests are seldom performed. Several special biochemical and physical tests can also be carried out on feces: fecal water content, nitrogen content (for azotorrhea and malassimilation), electrolytes, pH, osmolality, fecal occult blood, and cultures for both fungi and bacteria.

4. Tests of absorptive and digestive function, such as trypsin-like immunoreactivity (TLI), serum folate, and vitamin B₁₂ assay.

5. Gastrointestinal (GI) radiography and ultrasonography.

6. GI endoscopy (gastroscopy, duodenoscopy, and colonoscopy), with biopsy of intestinal mucosa. Duodenal intubation and aspiration can be performed to obtain specimens for cytologic examination and culture.

7. Exploratory laparotomy and intestinal biopsy.

8. Response to empiric treatment: Enzyme replacement or treatment of occult parasite infections.

Difficulty Breathing or Respiratory Distress: Cyanosis

Definition

Cyanosis is a bluish discoloration of the skin and mucous membranes resulting from excessive concentration (> 5 g/dL) of reduced hemoglobin in the blood. In dogs and cats, cyanosis may develop acutely in hypoxic states or may be chronic. Although cyanosis can develop during hypoxia, the terms are not synonymous.

Note: The increased concentration of reduced hemoglobin in blood is the result of either an increase in the quantity of venous blood in the cutaneous tissues (passive venous congestion) or a decrease in oxygen saturation in capillary blood. It is the absolute, rather than the relative, amount of reduced hemoglobin that actually causes the cyanosis to develop. If the concentration of hemoglobin is also reduced, the absolute concentration of reduced hemoglobin is also decreased. Therefore even in severe anemia, cyanosis is not evident. On the other hand, patients with an elevated RBC mass, or polycythemia, tend to be cyanotic at higher levels of arterial oxygen saturation than patients with a normal RBC mass. Cyanosis also occurs when functional abnormalities of hemoglobin (e.g., methemoglobinemia [dark-brown blood]) exist. In the dog and cat, disorders affecting the oxygen-carrying capacity of hemoglobin are usually drug- or chemical-induced. As little as 1.5 g of methemoglobin per deciliter or 0.5 g of sulfhemoglobin per deciliter will produce cyanosis.
ASSOCIATED SIGNS

Cyanosis can result from disorders affecting the cardiovascular system, ventilation, or oxygen-carrying capacity of RBCs. Several cardiovascular diseases, particularly those that compromise cardiac output or are associated with right-to-left vascular shunts, predispose to cyanosis. Therefore animals with both acquired and congenital cardiac disease are susceptible. Associated signs include cough, respiratory distress, and syncope. The most common congenital heart defects associated with right-to-left shunts are (1) pulmonary valve stenosis as seen in tetralogy of Fallot, stenosis, and ventricular septal defect (VSD) and (2) pulmonary hypertension as seen in patent ductus arteriosus (PDA) and VSD.

Respiratory disorders affecting ventilation predispose to cyanosis. Severe infiltrative lung disease (e.g., neoplasia, pulmonary edema, or generalized pneumonia) can produce cyanosis associated with increased respiratory effort.

Animals with cyanosis not associated with clinical signs other than increased respiratory rate may have abnormal hemoglobin levels, which, if present in sufficient concentration, will cause cyanosis. Associated signs include methemoglobinuria and methemoglobinemia.

Central cyanosis is defined as compromised oxygen saturation or abnormal hemoglobin; peripheral cyanosis is compromised blood flow.

DIFFERENTIAL DIAGNOSIS

| CARDIOVASCULAR CAUSES |
|-----------------------|
| Right-to-left shunting congenital heart defect (e.g., right-to-left shunting patent ductus arteriosus) |
| Pulmonary embolism |
| Decreased cardiac output |
| Arterial obstruction |

| PULMONARY CAUSES |
|------------------|
| Airway collapse or obstruction (multiple causes) |
| Hypoxia |
| Pulmonary edema |
| Oxygen diffusion–alveolar ventilation abnormalities |
| Pulmonary arterial-venous shunts or fistulas |
| Restrictive lung disease (e.g., hydrothorax, diaphragmatic hernia) |

| TOXIC OR DRUG-RELATED CAUSES |
|-----------------------------|
| Paraquat poisoning |
| Acetaminophen (cats) |

DIAGNOSTIC PLANS

1. Provide 100% oxygen, particularly in patients with respiratory distress. Reassess color of the mucous membranes at 2- or 3-minute intervals. Auscultate the heart and lungs.
2. Thoracic radiographs. Oxygen should be available at all times.
3. Hematology, with particular emphasis on RBC morphology (Heinz bodies in the cat and hematocrit values), biochemical profile, and urinalysis.
4. Special diagnostics: Arterial blood gases (with and without 100% oxygen), ECG, echocardiogram, and nonselective angiogram.
DIFFICULTY BREATHING OR RESPIRATORY DISTRESS: DYSPNEA

DEFINITION

Dyspnea is pathologic breathlessness and labored breathing most commonly associated with cardiac or pulmonary disease. What actually is and is not true breathlessness in veterinary medicine can be difficult to define in clinical practice. Serious respiratory distress associated with substantive respiratory compromise may appear, to the owner at least, as only a minor problem. Physical examination and patient assessment are critical to the recognition and interpretation of this clinical sign.

Dyspnea may result from (1) the need for oxygen, (2) metabolic aberrations leading to acidosis (a compensatory mechanism), (3) high environmental temperatures (heat stroke), (4) CNS disease, (5) disorders affecting motor innervation to the muscles of respiration, and (6) pain. In any event, once confirmed, diagnostic evaluation of the patient presented in respiratory distress should not be delayed.

ASSOCIATED SIGNS

The most common respiratory signs that characterize distress or dyspnea include (1) tachypnea (increased respiratory rate), (2) hyperpnea (increased respiratory rate and depth), (3) orthopnea, and (4) cough. In obstructive upper airway diseases, stridor (laryngeal) and stertor (pharyngeal) abnormalities may be present (patient must be examined under anesthesia).

Fluid accumulation in the thoracic cavity may be accompanied by ascites and hepatomegaly. Physical evidence of hyperadrenocorticism supports thromboembolic pulmonary disease. Cyanosis, pallor, evidence of physical trauma, shock, and coma are serious signs often associated with respiratory distress.

DIFFERENTIAL DIAGNOSIS

DIAGNOSTIC PLANS

1. Physical examination is a priority to establish need for supplemental oxygen administration. This is justified even before a comprehensive history is obtained. Patient stabilization, as required, must be accomplished.
2. History. Historical information relevant to duration, progression, past illnesses, and exposure to noxious substances or trauma is indicated. Knowledge of all current medications, including heartworm preventative, must be established.
3. Laboratory profile, to include a CBC, biochemistry panel, urinalysis, heartworm test (in dogs), and FeLV and FIV tests (in cats). Cytologic, bacteriologic, and biochemical assessments of body cavity effusions are indicated.
4. Thoracic and cervical radiographs. Presence of a heart murmur, cardiac arrhythmia, or both should be further evaluated by electrocardiography and echocardiography.
5. Examination of the upper respiratory tract in the anesthetized patient and endoscopy (lower respiratory tract) when signs of tracheal and bronchial disease exist.

DIFFICULTY SWALLOWING: DYSPHAGIA

DEFINITION

Dysphagia is painful or difficult swallowing. Clinically, dysphagic animals characteristically are presented for making frequent and forced attempts to swallow with or without regurgitation. Signs are most apparent immediately after prehension of food or water.
### Upper Airway
- Stenotic nares
- Rhinitis or sinusitis
- Laryngeal diseases
- Nasopharyngeal tumor or foreign body
- Necrotic laryngitis
- Edema
- Paralysis of vocal folds
- Everted saccules
- Laryngeal collapse
- Neoplasia
- Intraluminal tracheal or bronchial foreign body or mass
- Extraluminal tracheal or bronchial obstruction
- Mediastinal mass
- Tracheal or bronchial collapse
- Hilar lymphadenopathy

### Lower Airway
- Bronchial diseases
- Emphysema (rare)
- Chronic airway disease
- Allergic bronchitis (asthma, PIE)
- Lungworms
- Pneumonia
- Pulmonary edema
- Left-sided heart failure
- Hypoalbuminemia
- Others
- Pulmonary thromboembolism
- Heartworm disease
- Hyperadrenocorticism
- Others
- Pulmonary contusions (trauma)
- Pulmonary fibrosis
- Pulmonary granulomatosis
- Deep mycosis

### Restrictive
- Pneumothorax
- Pleural effusion
- Right-sided heart failure
- Neoplasia
- Hypoalbuminemia
- Hemotorax
- Chylothorax
- Pyothorax
- Feline infectious peritonitis
- Pericardial effusion
- Diaphragmatic hernia
- Intrathoracic neoplastic mass
- Thoracic wall trauma
- Hail chest
- Extreme obesity
- Severe hepatomegaly
- Marked ascites
- Large intraabdominal mass
- Severe gastric distension (gastric volvulus)

### Miscellaneous
- Anemia
- Methemoglobinemia
- Compensation for metabolic acidosis
- Heat stroke
- Damage to respiratory center
- Head trauma
- Encephalitis
- Neoplasia
- Neuromuscular weakness
- Polyradiculoneuritis (Coonhound paralysis)
- Diaphragmatic paralysis
- Others
- Pain
- Fractured ribs or vertebrae
- Pleuritis
- Others
- Paraquat poisoning

PIE, pulmonary infiltrates with eosinophils.
Swallowing is a complex reflex requiring coordination of multiple muscular and neurologic reactions involving the tongue, palate, pharynx, larynx, esophagus, and gastroesophageal junction. Dysphagia may occur as a result of disorders affecting any one of the three swallowing phases: oropharyngeal, esophageal, and gastroesophageal. Disorders affecting the oropharyngeal phase of swallowing are responsible for causing pronounced dysphagia, whereas disorders affecting the esophageal and gastroesophageal phases of swallowing are associated with regurgitation.

**ASSOCIATED SIGNS**

Dysphagia is observed in young animals, particularly in association with congenital esophageal motility disorders and as an acquired condition in older animals. This is more common as a presenting sign in dogs than in cats.

Prehension of food in animals presented with dysphagia is characteristically normal. Hypersalivation may occasionally be reported, particularly in animals with nasal discharge associated with regurgitation.

Regurgitation is an inconsistent sign associated with dysphagia that does not necessarily correlate with the severity of the underlying disorder. Generally, regurgitation is a consequence of abnormalities of the esophageal and gastroesophageal phases of swallowing. Although most dysphagic patients have a normal to increased appetite (polyphagia), anorexia, weight loss, and coughing may be associated with severe or chronic obstructive esophageal disease or esophageal ulceration.

*Caution:* Assessment of affected patients for evidence of neurologic signs is of paramount importance, because dysphagia is a principal neurologic complication associated with rabies virus infection.

**DIFFERENTIAL DIAGNOSIS**

| DIFFERENTIAL DIAGNOSIS OF DYSPHAGIA |
|-------------------------------------|
| **CARDIOVASCULAR** |
| Megaesophagus secondary to congenital persistent fourth aortic arch |
| Lymphatic and immune causes |
| Mandibular, retropharyngeal, and less commonly bronchial lymphadenopathy associated with lymphosarcoma, thymic neoplasia in FeLV-positive cats, and systemic mycoses (histoplasmosis or blastomycosis) |
| Epidermolysis bullosa–induced esophagitis (rare) |
| **GASTROINTESTINAL** |
| Esophageal obstruction from foreign body, parasitic granuloma (*Spirocerca lupi*), stricture, esophageal neoplasia |
| Cricopharyngeal achalasia (young dogs) |
| Megaesophagus secondary to pyloric obstruction in cats |
| Esophageal diverticula |
| Traumatic esophageal rupture |
| Reflux esophagitis |
| Doxycycline-induced esophagitis |
| Feline herpesvirus–induced esophagitis (rare) |
| **NEUROLOGIC** |
| Congenital and acquired megaesophagus |
| Myasthenia gravis in dogs |
| Rabies virus infection |

*FeLV*, Feline leukemia virus.
DIAGNOSTIC PLANS

1. Observation of the patient’s attempt to swallow food and water.
2. CBC, a biochemistry profile, and urinalysis. Findings are usually of little diagnostic value but are important in assessing overall patient status. A fecal flotation test for parasite ova can be diagnostic for *Spirocerca lupi*.
3. Special laboratory tests, including antinuclear antibody (ANA) titer and lupus erythematosus cell results, to assess for the presence of immune-mediated disease. Serum thyroxine ($T_4$) and thyroid-stimulating hormone (TSH) tests are indicated to rule out peripheral neuropathy caused by primary hypothyroidism.
4. Noncontrast thoracic and cervical radiographs.
5. Positive contrast esophagram, both thoracic and cervical.
6. Esophagoscopy, which may be therapeutic if an esophageal foreign body can be retrieved. Esophageal endoscopy is not a reliable means for diagnosing megaesophagus.
7. Fluoroscopic evaluation of esophageal motility.
8. Visual examination of the oropharynx in the anesthetized patient. (Findings are of low diagnostic value.)

HAIR LOSS: ALOPECIA

DEFINITION

Alopecia is the loss or absence of hair coat in any amounts and any distribution. Physiologic loss of hair (e.g., normal shedding or hereditary hair loss such as in the Rex cat breed) is excluded from this definition. In clinical practice, hair loss, with and without pruritus, is among the most common reasons a cat or dog is presented. In most cases the loss of hair is secondary to some underlying disorder rather than being a primary event. The distribution of hair loss is important in that it can be characteristic of the underlying cause.

Alopecia can be classified on the basis of distribution as (1) diffuse, (2) regional, (3) multifocal, and (4) focal. The causes for hair loss are varied and often complex. Abnormalities of follicular structure may be inherited, ranging from complete absence of hair follicles to selective absence of follicles that produce hair of a specific color. Inflammatory skin diseases that incorporate the hair follicle may disrupt hair growth and maintenance. Bacterial folliculitis, demodectic mange, and follicular hyperkeratosis are examples.

Disorders that disrupt the normal follicular cycles can interrupt hair growth without loss or injury to the hair follicle. The cycle is as follows: anagen (growth phase), catagen (transitional phase), and telogen (resting phase).

ASSOCIATED SIGNS

The complex pathogenesis of alopecia supports a multitude of associated clinical signs in any animal presented with hair loss. Pruritus is an important associated sign if present. Allergic, inflammatory, and parasitic skin diseases are likely to cause pruritus. Secondary traumatic excoriation of the skin may further provoke cutaneous injury, thereby intensifying the pruritus. Alopecia caused by endocrine, genetic, and metabolic factors is less likely to be associated with pruritus, although pruritus may become a factor if the exposed skin becomes particularly dry or sunburned. Immune-mediated diseases leading to alopecia are variably pruritic, depending on the distribution and type of skin injury. Nutritional alopecia is rarely confirmed but can be a source of dermatitis and associated pruritus.
Alopecia without pruritus may be associated with dramatic physical signs resulting from endocrine or metabolic disorders. Dermatologic signs include thickened skin, hyperpigmentation, and dry and brittle hair coat (hypothyroidism). On the other hand, skin may appear thin and lack elasticity (canine Cushing syndrome, Sertoli cell tumor). Gynecomastia, skin softness, calcinosis cutis, and pigmented macules are other dermatologic signs associated with alopecia.

**DIFFERENTIAL DIAGNOSIS**

Virtually all dogs and cats with primary skin disease manifest some degree of alopecia. The pattern of hair loss is typically asymmetric, and primary skin disease can appear to be symmetric (e.g., parasitic dermatosis). In pursuing the diagnosis in dogs or cats presented with hair loss, thorough systemic and skin examinations are indicated. The clinician may find it helpful to characterize a patient's hair loss according to various etiologic categories, as follows.

### PRIMARY CUTANEOUS CAUSES OF HAIR LOSS

- Infection
- Bacteria
- Ectoparasites
- Dermatophytoses
- Dermatomycoses
- Neoplasia
- Keratinization

### SECONDARY CAUSES OF HAIR LOSS

- Genetic causes
- Nutrition
- Endocrine conditions (e.g., hypothyroidism, hypoadrenocorticism)
- Keratinization
- Atopic (allergic) or contact hypersensitivity
- Drug therapy (especially corticosteroids and chemotherapeutic agents)
- Environmental factors
- Neoplasia
- Psychogenic causes

### DIFFERENTIAL DIAGNOSIS OF GENETIC DISORDERS CAUSING ALOPECIA

- Hairless breeds (e.g., African Sand Dog, Abyssinian Dog, Chinese Crested, Xoloitzcuintli, Turkish Naked Dog; Sphinx Cat, Rex Cat [seasonal alopecia])
- Ectodermal and follicular dysplasias (e.g., Miniature Poodles)
- Hypotrichosis
- Black hair follicular dysplasia
- Color-mutant alopecia
- Pattern baldness
- Feline alopecia universalis
- Demodicosis

### DIAGNOSTIC PLANS

1. History and physical examination, to determine the nature and extent of primary and secondary skin lesions. Distribution, pattern of alopecia, and associated cutaneous lesions should be characterized. Use the physical examination to determine whether or not evidence of systemic disease is present. Time of onset or the seasonal nature of alopecia may be significant, particularly when accompanied by pruritus.
2. Examination (macroscopic and microscopic) of affected and unaffected hair.
3. Skin scraping (multiple), fungal cultures, and bacterial cultures (particularly of pustules).
   a. Fine-needle aspiration of discrete intracutaneous masses.
   b. Skin biopsy, to include normal and affected skin.
4. Laboratory database, to include hematology, biochemical profile, urinalysis, and fecal flotation. In addition, cats should be tested for FeLV and FIV.
5. Special diagnostics:
   a. Allergic skin disease: Intradermal antigen inoculation
   b. Endocrine alopecia: T4 before and after TSH stimulation, adrenocorticotropic hormone (ACTH) stimulation or dexamethasone suppression test (high dose versus low dose), serum testosterone
6. Implementation of an elimination diet trial (minimum 6 weeks’ duration).
7. Environmental allergen or irritant.

**HEMORRHAGE** See Spontaneous Bleeding: Hemorrhage.

**ICTERUS** See Yellow Skin or Mucous Membranes: Icterus (or Jaundice).

**INCOORDINATION: ATAXIA**

**DEFINITION**
Ataxia is the loss of coordination without spasticity, paresis, or involuntary movement. In practice, however, it is possible for ataxia to be accompanied by additional neurologic signs. Ataxia is the result of disorders of the conscious or unconscious proprioceptive system, disorders of the cerebellum, or disorders of the vestibular system.

**ASSOCIATED SIGNS**
In the spectrum of disorders causing ataxia, lesions of the vestibular system predominate. However, vestibular signs may result from other brain disorders and spinal cord syndromes. Associated signs include head tilt, nystagmus, circling, and hemiparesis. Patients with cerebellar lesions typically have symmetric signs: hypermetria, abnormally long range of movement (goose-stepping gait); hypometria, abnormally short range of movement; or tremor, particularly of the head.

**DIFFERENTIAL DIAGNOSIS**

| DIFFERENTIAL DIAGNOSIS OF ATAXIA |
|----------------------------------|
| **CONGENITAL (SIGNS PRESENT BEFORE 3 MONTHS OF AGE)** |
| Reported in Siamese and Burmese cats and several dog breeds. Multiple congenital disorders are present with multiple neurologic signs, including ataxia. Bilateral congenital vestibular disorders have been observed in Doberman Pinschers, Beagles, and Akitas. |
| **INFLAMMATORY** |
| Otitis interna, as an extension of otitis externa and media |
| Neuritis of the eighth cranial nerve (recrudescent feline herpesvirus-1 ?) |
| Infections |
| **TOXIC** |
| Drug-induced aminoglycoside therapy |
| **NUTRITIONAL** |
| Thiamine deficiency (cat only—rare) |
| **METABOLIC** |
| Central nervous system signs secondary to other diseases (e.g., hepatic, renal) |

*Continued*
**Clinical Signs**

**Diagnostic Plans**

1. Physical examination, with particular attention to the external ear and tympanic membrane.
2. Neurologic examination, to include assessment of the cranial nerves with the intent of localizing the lesion.
3. Laboratory profile, to assess metabolic or infectious causes.
4. Skull radiographs, to include the tympanic bullae.
5. Collection and examination of cerebrospinal fluid (CSF).
6. Special diagnostics, depending on availability (e.g., electroencephalogram [EEG], CT, or magnetic resonance imaging [MRI]).

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**Increased Urination and Water Consumption: Polyuria and Polydipsia**

**Definition**

In practice, polyuria (PU) and polydipsia (PD), also abbreviated PU/PD, are loosely interpreted to mean an increase in urination and water consumption, respectively. True PU, however, is an abnormal increase in urine production, usually of low specific gravity. Although PD is an abnormal or absolute increase in water consumption usually associated with increased thirst, water intake is seldom quantitated. Use of the terms polyuria and polydipsia is usually justified when a client presents a dog or cat with subjective increases in urination frequency and water intake as the primary problem. When clear evidence of increased urination and increased thirst is not present, actual documentation of 24-hour urinary output and water intake may be necessary.

PD is a compensatory sign that develops subsequent to PU. Primary PD with compensatory PU is uncommon. Primary PD subsequent to increased thirst can cause secondary PU but is an uncommon clinical finding. Compulsive water drinking (pseudopsychogenic PD) is probably the most important type of primary PD, although the underlying cause is not known. Hypothalamic lesions, hypercalcemia, and increased levels of plasma renin are less common causes of primary PD.

**Associated Signs**

Signs associated with PU or PD are varied and dependent on the underlying disease. Generalized signs include weakness, decreased appetite, weight loss, diarrhea, and fever. Polyphagia with weight loss occurs in animals with diabetes mellitus and in cats with...
hyperthyroidism. Paraneoplastic syndromes, particularly hypercalcemia, may develop in conjunction with PU/PD. A comprehensive physical examination and a laboratory assessment are justified in all patients presented with PU/PD as the primary complaint.

**DIFFERENTIAL DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS OF POLYURIA AND POLYDIPSIA**

| POLYURIA OF RENAL ORIGIN | POLYURIA OF NONRENAL CAUSES |
|---------------------------|----------------------------|
| Renal failure             | Diabetes insipidus (nephrogenic) |
| Glomerulonephritis        |                             |
| Tubular dysfunction       |                             |
| Renal medullary dysfunction |                           |
| Postobstructive diuresis (e.g., feline urologic syndrome) | |
| Diabetes insipidus (nephrogenic) |                        |
| Hypercalcemic nephropathy |                             |
| Fanconi syndrome          |                             |
| Medullary washout         |                             |
| Diabetes mellitus         |                             |
| Hyperadrenocorticism      |                             |
| Liver disease (nonspecific) |                           |
| Pyometra                  |                             |
| Pseudopsychogenic polydipsia |                         |

**DRUG-INDUCED POLYURIA**

- Glucocorticoids (especially in dogs)
- Mannitol, intravenous
- Dextrose, concentrations > 50 mg/dL (5.0%)
- Alcohol
- Diuretic therapy (e.g., furosemide)
- Phenytoin
- Vitamin D intoxication

**DIAGNOSTIC PLANS** *(Figure 3-3)*

1. History and physical examination, to facilitate verification of the problem in addition to determining the duration of the problem and associated signs. Of particular importance is knowledge of the recent administration of medication.
2. Laboratory database. The primary focus of the diagnostic plan is interpreting results from a laboratory database, including a CBC, biochemistry profile, urinalysis, fecal culture, heartworm test (in dogs), FeLV and FIV tests (in cats), and urine culture.
3. Collecting urine and measuring water intake over a 24-hour period, to document the problem, if necessary.
4. Abdominal radiographs, if indicated.
5. Special diagnostic tests, if indicated, based on results from a laboratory database:
   a. Water deprivation and modified water deprivation tests (contraindicated in the presence of azotemia, dehydration, or hypercalcemia)
   b. Antidiuretic hormone (ADH, vasopressin) response test
   c. Glucose tolerance test
   d. ACTH stimulation or dexamethasone suppression test
   e. Serum $T_4$
   f. Liver function studies (e.g., serum ammonia, bile acids)
   g. Abdominal ultrasound
   h. Tissue biopsy (e.g., renal and hepatic)
   i. Exploratory laparotomy

**ITCHING OR SCRATCHING: PRURITUS**

See also Hair Loss: Alopecia.

**DEFINITION**

Pruritus is an unpleasant, sometimes intense, epidermal stimulation that causes abnormally frequent scratching or biting. Histamine, endopeptidases, and other polypeptides liberated from skin cells serve as mediators of pruritus. Histamine is the primary mediator of itch...
History of polydipsia/polyuria

Rule out iatrogenic causes

Normal physical exam

Verify by measurement at home if necessary

CBC, biochemical profile, urinalysis

Negative

Nondehydrated

Water deprivation test

Positive APP

Intermediate Partial CDI APP + MSW RI

Negative CDI NDI APP + MSW

Exogenous ADH test

Positive CDI

Intermediate APP + MSW

Negative NDI APP + MSW

Partial water deprivation or Hickey-Hare test

Positive APP

Negative NDI

Rule out (confirm with specific tests)

Hyperthyroidism
Renal failure
Diabetes mellitus
Renal tubular glucosuria
Postobstructive diuresis
Pyometra
Hypoadrenocorticism
Hyperadrenocorticism
Hepatic failure
Polycythemia
Hypercalcemia
Hypokalemia

Positive

Dehydrated

Creatinine clearance test

Normal CDI NDI APP

Decreased RI

Rehydrate

Key:

APP = apparent psychogenic polydipsia
CDI = central diabetes insipidus
NDI = nephrogenic diabetes insipidus
MSW = medullary solute washout
RI = renal insufficiency with solute diuresis

Figure 3-3: Clinical approach to the patient with polydipsia and polyuria. ADH, Antidiuretic hormone; CBC, complete blood count. (From Fenner WR: Quick reference to veterinary medicine, ed 2, Philadelphia, 1991, Lippincott.)
associated with wheal-and-flare reaction. Histamine-mediated itching cannot be completely inhibited by either H₁- or H₂-receptor antagonists (blockers). The close association between itching and inflammation of the skin is attributed to the fact that many of the endogenous mediators and potentiators are released in situ during inflammatory events.

Itching, although a protective response, can become more harmful than helpful. As a feature of dermatitis, itch mediators cannot be removed by the patient. In fact, scratching and biting eventually promote more inflammation and subsequently perpetuate the itching.

**ASSOCIATED SIGNS**

Skin lesions are commonly associated with pruritus; however, it becomes important to characterize the lesions and to distinguish those that are primary from those that are secondary to scratching or biting. Papules and pustules are characteristic primary lesions that may ultimately develop into secondary lesions, such as crusts, ulcers, scale in collarettes, and pigmented macules. Vesicles and bullae, plaques, and urticaria (wheels) can also occur as primary skin lesions. Linear crusts, irregular ulceration, lichenification, diffuse scaling and pigmentation, and patchy alopecia are characteristic lesions that develop secondary to excoriation.

Pruritus can also occur without primary lesions (i.e., “essential” pruritus). This type of itching is a manifestation of systemic disease, although mediation may be central or cutaneous. Causes include atopy, dry skin, and neurogenic and psychogenic disorders. A spectrum of renal, hepatic, hematopoietic, allergic, and endocrine diseases are associated with essential pruritus.

**DIFFERENTIAL DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS OF PRURITUS (NOT A COMPREHENSIVE LIST)**

| Pustular dermatitis | Folliculitis (bacterial, fungal, demodectic) |
| Infectious | Parasitic (Sarcoptes, Cheyletiella, lice, fleas) |
| Puppy pyoderma | Vasculitis (Rocky Mountain spotted fever) |
| Folliculitis and furunculosis | Immune |
| Immune-mediated | Allergy (atopy) |
| Pemphigus foliaceus | Autoimmune (pemphigus foliaceus, SLE) |
| Vesicle-forming disorders (e.g., drug eruption) | Idiopathic |
| Linear immunoglobulin A (IgA) γ dermatosis | |
| **Idiopathic** | |
| Puppy “strangles” | |
| Subcorneal pustular dermatitis | |
| **VESICULAR OR BULLOUS ERUPTION** | |
| Bullous dermatosis | |
| Systemic lupus erythematosus (SLE) | |
| Toxic epidermal necrolysis | |
| Drug eruption | |
| Acute contact dermatitis | |
| **PLAQUE FORMATION** | |
| Infectious dermatitis | |
| Immune-mediated dermatitis | |
| Neoplasia (e.g., mast cell tumor) | |
| **PAPULAR ERUPTION (DOG)** | |
| Infectious | |
DIAGNOSTIC PLANS

1. History and physical examination, to characterize the skin lesion and its distribution, to determine whether or not the condition appears to be contagious, and to determine whether or not systemic disease is present.
2. Laboratory database, if evidence of systemic disease is present.
3. Skin and coat examination. Perform multiple skin scrapings, and examine skin and hair coat with Wood light.
4. Microbiologic testing for bacteria and dermatophytes.
5. Immunologic testing, to include intradermal skin testing and direct fluorescent antibody testing of skin (both normal and affected) biopsy specimens.
6. Skin biopsy with dermatohistopathology.
7. Provocative exposure to selected environmental agents, diet, and drugs.

JAUNDICE See Yellow Skin or Mucous Membranes: Icterus (or Jaundice).

JOINT SWELLING: ARTHROPATHY

DEFINITION

Joint swelling, or joint enlargement, is any abnormal increase in size, either visible or palpable, of any joint that is not directly caused by a proliferation of tissue. In practice, joint swelling is the primary presenting sign only occasionally. Pain and associated lameness are more likely causes for presentation, whereas actual enlargement of a joint is detected during physical examination. However, there is not necessarily an association between joint swelling and pain.

Joint swelling, or effusion, occurs subsequent to injury to the synovial membrane in which there is not only an increase in volume of synovial fluid produced, but quantitative biochemical and cellular changes as well. Most joint swelling is attributed to inflammation of the synovial membrane, or synovitis. Abnormal synovial fluid accumulation (effusion) may be classified as serous, fibrinous, purulent, septic, or hemorrhagic.

ASSOCIATED SIGNS

Although lameness is the most common clinical sign associated with joint swelling, it is not consistently present. Joint swelling may also be associated with, or mistaken for, hyperplasia, metaplasia, or neoplasia of the synovium, joint capsule, articular cartilage, or periarticular bone. Hemorrhagic joint effusion (hemarthrosis) may be associated with coagulopathy and spontaneous bleeding from the respiratory, GI, or urinary tract. Subluxation or fracture of a carpus, tarsus, or stifle may also be associated with detectable joint swelling. Arthritis associated with systemic disease (e.g., infectious or immune mediated) can also be accompanied by significant joint swelling.

DIFFERENTIAL DIAGNOSIS

| ARTHROPATHIES IN THE DOG AND CAT |
|----------------------------------|
| **NONINFLAMMATORY**              |
| Degenerative joint disease (osteoarthritis, osteoarthrosis) |
| Primary                          |
| Secondary                        |
| As a sequela to acquired or congenital defects of the joints and supporting structures |
| **INFLAMMATORY**                 |
| Infectious                       |
| Traumatic                        |
| Neoplastic involvement           |
| Drug-induced                     |
DIAGNOSTIC PLANS

1. History. The history generally focuses on associated signs rather than primary joint swelling and should address duration, exposure to ticks, known injury, and evidence of spontaneous bleeding. Physical examination establishes the presence of joint swelling and the number of joints involved. Evidence of inflammation, crepitus, joint laxity, abnormal range of motion, a drawer sign, luxations, or fractures should be determined.

2. Radiography of the affected joint(s) and surrounding bone.

3. Synovial fluid analysis, including biochemical, cytologic, and culture findings.

4. Coagulation profile in the presence of hemarthrosis.

5. Immune function testing: ANA titer, rheumatoid factor.

6. Contrast arthrography.

7. Joint capsule–synovial membrane biopsy.

8. Periarticular bone biopsy.

LOSS OF APPETITE: ANOREXIA

DEFINITION

Strictly speaking, anorexia is the complete lack or loss of appetite. In veterinary medicine, this term is loosely used to describe diminished interest in eating or observed reduction in daily food volume consumed. In addition, part of the difficulty in assessing the patient that is presented with loss of appetite is grounded in owner expectation of what is and what is not a normal appetite in a dog or cat. Although domesticated pets do tend to eat at regular intervals throughout the day, some do experience transient periods of sustained inappetence that may, in fact, be entirely normal and not associated with underlying disease. When assessing a dog or cat for partial loss of appetite, careful history and physical evaluation are indicated to determine whether or not underlying disease may be the cause of this vague clinical sign. In addition, the clinical history must establish the duration of the anorexia and whether the loss of appetite is complete or partial.

Note: What makes anorexia such an important clinical sign is the fact that loss of appetite (either complete or partial) is often the first outward sign an owner may notice when a pet is ill.

ASSOCIATED SIGNS

Anorexia is regarded as a low-yield clinical sign that may be associated with numerous underlying disorders. Historical evidence of a significant change in the pet’s environment (e.g., a new child in the family) or daily routine (e.g., the dog is home alone during the day...
for the first time) is important to assess. Knowledge of current drug therapy, whether the pet eats sticks or other foreign material, whether or not the pet food type recently changed or may not be fresh (moldy canned and dry food will generally not be consumed) is important.

Physical examination should determine overall body conformation, body weight, extent of weight loss (if present), and any obvious external injuries that might contribute. Age is an important factor in the assessment of anorexia. Diminished sense of smell, neoplasia, joint disease, and dental disease are common age-related disorders that may contribute to anorexia.

**DIFFERENTIAL DIAGNOSIS**

Differential diagnoses associated with anorexia are too numerous to be of assistance in resolving to a diagnosis. The clinician faced with a patient that has only anorexia is faced with a significant clinical challenge in defining the underlying disorder. Even the categories of disease that could be associated with inappetence are wide ranging and include psychologic, metabolic, orthopedic, infectious, inflammatory, and neoplastic causes.

**DIAGNOSTIC PLANS**

1. Careful observation of the patient on and off the examination table is important.
2. A methodical physical examination.
3. A standard laboratory profile to include hematology, biochemistry, and urinalysis (fecal is optional depending on the presenting signs).
4. Radiography or other imaging study is indicated if the pain can be localized to a discrete region of the body (e.g., abdominal cavity).
5. Special diagnostic tests are indicated if specific abnormalities can be detected (e.g., biopsy, aspiration and cytology, myelography).

**LYMPH NODE ENLARGEMENT: LYMPHADENOMEGALY**

**DEFINITION**

Lymphadenomegaly refers to those lymph nodes that are larger than expected with or without commensurate changes in consistency. Involved nodes may be unusually soft, firm, or painful, suggestive of inflammation, whereas enlarged, firm, nonpainful lymph nodes suggest neoplasia. Lymphadenomegaly is usually not a presenting problem, with the possible exception of generalized enlargement of all superficial lymph nodes.

Lymph nodes become enlarged as a result of inflammation (pyogenic or granulomatous), reactive lymphoid hyperplasia, or neoplasia (primary or neoplastic). In pyogenic inflammation, neutrophils dilate and engorge the sinuses, whereas in granulomatous inflammation an infiltrate or macrophages are present (e.g., systemic mycoses). Reactive lymphoid hyperplasia is associated with an increase in the number of germinal centers within the lymph node and an infiltrate of plasma cells. In neoplastic lymph nodes, tumor cells may invade the sinuses (metastatic), gradually destroying the normal node architecture, or the architecture of the lymph node is entirely replaced by malignant lymphocytes (lymphosarcoma)—that is, histologically the sinuses are obliterated and germinal centers cannot be found.

**ASSOCIATED SIGNS**

Characterize the consistency and number of affected nodes as well as their location (i.e., generalized or regional). Lymph node pain is an inconsistent finding usually associated with inflammatory disease (lymphadenitis) rather than neoplasia (lymphoma). Associated
signs are likely to be regional, as is the lymph node enlargement (i.e., tissue injury or infection). Patients with generalized lymphadenomegaly may not have associated signs, or there may be nonspecific signs, including weight loss, fever, decreased appetite, and lassitude as a result of systemic illness.

**DIFFERENTIAL DIAGNOSIS**

**DIAGNOSTIC PLANS**

1. History and physical examination, to determine the duration and type of associated signs, if any, and the duration of lymph node enlargement, if known.
2. Laboratory profile, with emphasis on CBC, including platelet count, biochemistry panel, and urinalysis.
3. Specific tests for infectious diseases, as indicated (e.g., FeLV antigen and FIV antibody).
4. Thoracic and abdominal radiographs, as indicated.
5. Fine-needle aspiration of affected lymph node(s).
6. Serum protein electrophoresis.
7. Bone marrow aspirate.
8. Lymph node biopsy and, if indicated, culture.

**PAIN**

**DEFINITION**

Pain is the perception of an unpleasant sensation; it may be generalized or localized. Although pain may be the single most common presenting complaint of humans who seek medical attention from a physician, the ability of a dog or cat to communicate pain and the ability of the owner to interpret the signs correctly make this a particularly complex clinical sign in animals. However, the inability of an animal to communicate pain must not be interpreted to mean there is an absence of pain. Animals do experience averse sensation and awareness of tissue injury. In animals, pain can be acute (e.g., trauma) or chronic (neuropathic pain associated with sustained tissue injury or disease).

**ASSOCIATED SIGNS**

The actual perception and manifestation of pain varies from one animal to another. Fundamental to the ability to interpret the presence of pain in an animal is the ability to recognize a change in behavior. Pain associated with acute injury can be relatively simple to ascertain. However, chronic pain emanating from a specific organ or tissue (e.g., liver or bone) can be difficult to characterize and localize. Other signs that may be associated with pain include sleeplessness; unusual posture; decreased activity; decreased appetite; reluctance to play, walk, or run; agitation; altered gait; and decreased grooming. Physical findings
are also highly varied and may include hypersalivation, mydriasis, tachycardia, shivering, or increased respiratory rate. Unfortunately, despite efforts to establish pain “scales,” there are still no standardized, objective pain test for animals.

**Note:** Pain management has become increasingly recognized as an essential part of clinical practice today. Section 1 (Tables 1-16 to 1-23) addresses indications of the drugs and doses most commonly employed in pain management in dogs and cats.

**DIFFERENTIAL DIAGNOSIS**

Pain can be associated with many disorders; therefore developing a list of differential diagnoses becomes impractical. Because pain is characteristically associated with inflammation or tissue trauma, every effort should be made to localize the source of the pain in order to focus the diagnostic search. Localizing acute-onset pain is generally less problematic than localizing chronic pain. Particularly in the patient with nonlocalizing, chronic pain, developing a clear diagnostic plan is essential in establishing a diagnosis.

**DIAGNOSTIC PLANS**

1. Careful observation of the patient as it moves, stands, sits, lies down, and so on is critical.
2. Acute pain: Physical examination addresses the physiologic (objective) assessment of the patient (e.g., heart rate, blood pressure, respiratory rate, pupils [dilation]). When feasible, effort should be made to localize the origin of the pain.
3. Acute pain: Physical examination should also address the behavioral (subjective) assessment of pain (e.g., attitude, mentation, posture, awareness of surroundings).
4. A standard laboratory profile to include hematology, biochemistry, and urinalysis (fecal is optional depending on the presenting signs).
5. Radiography or other imaging study is indicated if the pain can be localized to a discrete region of the body (e.g., abdominal cavity).
6. Special diagnostic tests are indicated if specific abnormalities can be detected (e.g., biopsy, aspiration and cytopathology, myelography).
7. In some patients, empiric treatment with analgesics or nonsteroidal antiinflammatory drugs may be indicated (see Table 1-18). However, using this method for managing pain requires the ability to provide follow-up care to that patient.

**PAINFUL URINATION: DYSURIA** See Straining to Urinate: Dysuria.

**PAINFUL DEFECATION: DYSCHEZIA** See Straining to Defecate: Dyschezia.

**RECTAL AND ANAL PAIN** See Straining to Defecate: Dyschezia.

**REGURGITATION**

See also Difficulty Swallowing: Dysphagia, and Vomiting.

**DEFINITION**

Regurgitation is retrograde esophageal transport of ingesta subsequent to a mechanical, neurogenic, or myogenic swallowing disorder. Owners most often describe regurgitation as “vomiting.” Both regurgitation and vomiting imply a backward flowing of ingesta through the esophagus; however, regurgitation is a relatively effortless act in contrast to the retching and abdominal pressure characteristic of vomiting. Regurgitation localizes the problem to...
the esophagus. Both acquired (e.g., foreign body) and congenital (e.g., familial megaesophagus) forms and esophageal disease can lead to regurgitation. Many esophageal problems remain undiagnosed if regurgitation is not present.

ASSOCIATED SIGNS

Physical signs accompanying regurgitation that are recognized by owners of dogs or cats include dysphagia characterized by difficulty swallowing food, frequent attempts to swallow food, and hypersalivation. Belching may also be reported subsequent to the entrapment of air in the esophagus. Inappetence and weight loss subsequently develop. Esophageal dilatation may be observed at the level of the lower cervical esophagus or thoracic inlet.

Owners may report expulsion of blood-tinged saliva subsequent to esophageal mucosal injury. Paroxysms of coughing and retching, particularly when eating, may be present, along with difficult breathing in animals with significant pneumonia. Nasal discharge may consist of mucoid to mucopurulent exudates or of food and liquid recently consumed.

Rarely, affected animals have swollen joints, lameness, and severe weakness associated with hypertrophic osteodystrophy subsequent to an intrathoracic lesion. Atypical signs include dyspnea (aspiration pneumonia or foreign body penetration through the intrathoracic esophagus), regurgitation unrelated to eating, and recurrent gastric bloating associated with aerophagia.

DIFFERENTIAL DIAGNOSIS

| Differential Diagnosis of Regurgitation |
|----------------------------------------|
| **FUNCTIONAL MEGAESOPHAGUS**            |
| Primary (or congenital)                 |
| Secondary (or acquired)                 |
| Foreign body                           |
| Esophageal stricture                   |
| Esophageal diverticula                 |
| Neurogenic (e.g., myasthenia gravis, rabies) |
| Myopathy, smooth muscle                |
| Extraesophageal compressive lesion (e.g., neoplasia) |
| Vascular anomaly                       |
| **ESOPHAGITIS**                        |
| Gastric reflux                        |
| Neoplastic                             |
| **RESTRICTIVE LESION WITHOUT MEGAESOPHAGUS** |
| Foreign body obstruction               |
| Intrathoracic mass                     |
| Vascular ring anomaly                  |
| Esophageal stricture                   |

*The most prevalent cause.

DIAGNOSTIC PLANS

1. History and physical examination, to characterize the nature of the problem, to distinguish between vomiting and regurgitation, and to establish the character of the regurgitated material.
2. Laboratory database, to assess patient status, particularly if secondary complications are present.
3. Survey thoracic and cervical radiography, to assess presence of megaesophagus, radiopaque intraesophageal lesion, or both.
4. Contrast esophagram, to confirm any interference with normal bolus transport at the point of obstruction, changes in mucosal integrity or luminal displacement, and the presence of extraluminal gas. (Oral suspension of barium sulfate is recommended over other contrast materials.) Note: Contrast medium retention in the esophagus is the hallmark of a motor disorder and often localizes the site of dysmotility.
5. Endoscopy and, as indicated, biopsy, to determine the cause of megaesophagus rather than to diagnose megaesophagus. In some instances, especially foreign body obstruction, endoscopy may be therapeutic.
6. Special procedures, to include contrast esophagram during fluoroscopy, CT, and exploratory laparotomy.
SEIZURES (CONVULSIONS OR EPILEPSY)

DEFINITION
The terms seizure, convulsion, epilepsy, epileptic attack, and “fit” all describe a clinical sign that is characterized by paroxysmal involuntary contraction of a series of voluntary muscles with a generally short duration, typically followed by a change in behavior. Epileptic seizure, the most common form of seizure disorder in dogs, has been described as having four component phases: (1) prodromal phase, the period of time immediately before a seizure; (2) aura, behavior suggesting that the patient is aware that seizure activity is impending; (3) ictal phase, characterized by actual seizure activity, and (4) postictal phase, the period after cessation of seizure activity, often manifesting as increased anxiety, water or food consumption, and/or transient blindness. Seizure activity may be self-limiting (one or two seizures per 24-hour period) or continuous (status epilepticus), which can be life-threatening and warrants immediate therapeutic intervention. In addition, seizures are classified as focal (facial twitching or bizarre behavioral manifestations) or generalized; generalized seizures are further characterized as tonic-clonic, clonic, myoclonic, atonic, or absence types.

Seizures result from disorders of the brain that cause spontaneous depolarizations and excitation of cerebral neurons. As a presenting problem, seizures are much more common in the dog than in the cat. Such disorders may originate from extracranial causes, metabolic or toxic diseases, and intracranial causes (e.g., organic brain disease). When seizures occur in the absence of detectable organic or metabolic CNS abnormalities, the seizures are described as idiopathic. Idiopathic epilepsy is the most common type of seizure reported in companion animal species.

ASSOCIATED SIGNS
Generalized motor seizures are the most prevalent type of seizure encountered in veterinary medicine. Most cases are diagnosed as idiopathic epilepsy on the basis that organic causes of seizure activity cannot be identified. The time between seizures (interictal period) in animals with a history of generalized motor seizures is characteristically described by owners as normal. The immediate postictal period, regardless of the cause of the seizure activity, is often associated with transient disorientation, blindness, stumbling, PD, or polyphagia.

The spectrum of possible clinical signs associated with seizure activity is extensive. Before a diagnosis of idiopathic epilepsy is reached, it is important that the patient be evaluated for cardiovascular disease, trauma, toxicity, infectious disease, parasites, neoplasia, and metabolic disorders, particularly those affecting the kidney, liver, and endocrine pancreas.

AGE OF ANIMAL
Seizures in young animals (< 1 year old) are commonly caused by developmental abnormalities, hydrocephalus, lissencephaly, encephalitis (infectious), lead poisoning, severe intestinal parasitism, portacaval shunt abnormalities, and juvenile hypoglycemia. Idiopathic epilepsy usually begins when animals are 1 to 3 years of age. Animals older than 5 years of age are more likely to have CNS tumors or hypoglycemia from insulin-secreting beta cell pancreatic neoplasms.

BREED PREDISPOSITION
Some basic knowledge about breed predisposition to seizure disorders may be helpful in establishing a diagnosis. Idiopathic epilepsy has been seen in numerous dog breeds, particularly German Shepherd Dogs, Belgian Tervurens, Keeshonds, Saint Bernards, Standard and Miniature Poodles, Beagles, Irish Setters, Cocker Spaniels, Alaskan Malamutes, Siberian Huskies, and Labrador and Golden Retrievers. Juvenile hypoglycemia is most prevalent in toy breeds. Hydrocephalus is common in the toy and brachycephalic breeds. Neoplastic diseases are common in dogs of brachycephalic breeds older than 5 years of age.
Disorders of CNS metabolism include leukodystrophy (Cairn and West Highland White Terriers), lipodystrophy (German Short-Haired Pointers and English Setters), lissencephaly (Lhasa Apso), portosystemic shunts (Yorkshire Terriers), and hyperlipidemic states (Miniature Schnauzers). A unique, usually fatal, encephalitis is described in Pugs.

**Environment**

Exposure to infectious agents or other sick animals may be important, as is exposure to sources of intoxicants, such as lead in paints, linoleum, tar, batteries, or roofing material; hexachlorophene soap; ethylene glycol (antifreeze); metaldehyde snail bait; and various other insecticides, including chlorinated hydrocarbons, organophosphates, and rodenticides. Dogs and cats on the same premises with swine may be exposed to suid herpesvirus (pseudorabies, or Aujeszky disease). A high-protein diet exacerbates hepatic encephalopathy. Thiamine deficiency may result from long-term consumption of certain fish diets or from cooking pet food.

**Differential Diagnosis**

| Intracranial                        | Extracranial                  |
|-------------------------------------|-------------------------------|
| Congenital                          | Intoxication                  |
| Hydrocephalus                       | Lead                          |
| Lissencephaly                       | Organophosphates              |
| Other malformations                 | Chlorinated hydrocarbons      |
| Storage diseases                    | Strychnine                    |
| Vascular anomaly                    | Drugs                         |
| Traumatic                           | Garbage                       |
| Immediate                           | Metabolic                     |
| Postrauama                          | Hypoglycemia                  |
| Inflammatory                        | Hypocalcemia                  |
| Distemper                           | Hyperkalemia                  |
| Rabies                              | Acid-base                     |
| Feline infectious peritonitis       | Hepatic encephalopathy        |
| Feline leukemia virus               | Uremia                        |
| Toxoplasmosis                       | Hyperlipoproteinemia           |
| Mycosis                             | Nutritional                   |
| Bacteria                            | Thiamine                      |
| Reticulosis                         | Parasites?                    |
| Parasites                           | Hypoxia                       |
| Neoplasia                           | Cardiovascular disease        |
| Primary                             | Respiratory disease           |
| Metastatic                          | Birth                         |
| Vascular-cerebrovascular accident   | Anesthetic accident           |

From Russo ME: Seizures. In Ford RB, editor: Clinical signs and diagnosis in small animal practice, New York, 1988, Churchill Livingstone.

**Diagnostic Plans**

1. History, to take into consideration breed predisposition, environmental exposures, past medical illnesses, and medication. Because most seizures are of short duration and the physical (tonic-clonic) manifestations of a seizure are so dramatic, requesting the owner to describe the type and duration of seizure may elicit unreliable information.
2. Thorough physical examination, to include careful neurologic examination, with particular attention to cranial nerves, funduscopic examination, and cardiac auscultation.
3. Laboratory database, essential to rule out metabolic causes. In addition to a CBC, biochemistry profile, urinalysis, and fecal culture, any or all of the following tests are indicated: serum ammonia, bile acids, serum insulin in hypoglycemic patients, blood lead test, and serial blood blood cultures. Serum should be assessed for the presence of lipid (triglyceride).
4. Survey radiographs of the skull. These are rarely helpful, as intracranial neoplasms are not detectable on conventional skull radiographs.
5. In special circumstances, limited ultrasound examination of the brain may be possible in young dogs through a cranial fontanelle. Evidence of hydrocephalus may be seen.
6. CT or MRI (special facilities required).
7. ECG or echocardiogram, if indicated.
8. Abdominal ultrasound (portosystemic shunt).
9. Serologic studies for canine distemper, rabies, FIP, FeLV, FIV, toxoplasmosis, and systemic (deep) mycoses.
10. CSF analysis, including biochemistries, antibody titers, and cytologic parameters.
11. EEG. Although limited in availability, the EEG may be useful in detecting inflammatory brain disease and congenital intracranial abnormalities (e.g., hydrocephalus).
12. Contrast studies, requiring special equipment or facilities: radioisotope brain scan, cerebral angiography, pneumoencephalography, and CT scan.

SNEEZING AND NASAL DISCHARGE

DEFINITION

Sneezing is a protective reflex described as a sudden, involuntary, and forceful, even violent, expulsion of air from the upper respiratory tract; it may or may not be accompanied by nasal secretions. Clients easily recognize sneezing. Although sneezing is a physiologic response to irritating stimuli, increased frequency and paroxysmal sneezing episodes are readily recognized as abnormal. Like sneezing, a nasal discharge, regardless of its consistency, is a clinical sign that clients accurately interpret and reliably describe to the clinician.

Sneezing is the outward manifestation of nasal passage irritation by extraneous agents (foreign material) or endogenous agents (antigen-antibody interaction). Afferent impulses travel via the fifth cranial nerve to the medulla, where the initial reflex is triggered. Chronic nasal discharge is a clinical sign that localizes a disorder to the upper respiratory passages, particularly the nasal cavity and frontal sinuses.

ASSOCIATED SIGNS

Important associated signs suggesting systemic involvement include facial asymmetry (neoplasia or fungal infection), atrophy of the masseter and temporal muscles, difficulty prehending or masticating food, conjunctivitis, and ocular discharge. Epistaxis, which is distinguished from blood-tinged nasal discharge, is an important associated sign that further supports intranasal disease or coagulopathy. Cleft palate is a common cause of nasal discharge in neonates. Erosion and depigmentation of the planum nasale is often associated with nasal aspergillosis in dogs, whereas cats with nasal cryptococcosis may have a detectable granuloma at the rostral aspect of the nose. Occasionally, cough is associated with purulent nasal discharges and sneezing.
Differential Diagnosis

Differential Diagnosis for Sneezing and Nasal Discharge

Intranasal Causes
Serous Nasal Discharge
Acute viral upper respiratory infection (feline)
Feline chlamydiosis
Intranasal parasites
Oronasal fistula (canine tooth)
Rhinopiridiosis (canine, rare)

Purulent Nasal Discharge
Viral upper respiratory infection with secondary bacterial infection (dog and cat)
Bacterial rhinitis (especially *Bordetella bronchiseptica*)
Mycotic nasal disease
Foreign body rhinitis
Traumatic rhinitis or sinusitis
Cleft palate
Neoplasia (several types possible)
Nasopharyngeal polyps (feline, rare)
Benign nasal polyps (canine, rare)
Oronasal fistula (occasionally associated with small amounts of blood)

Mucoid to Mucopurulent Nasal Discharge
Mycotic nasal disease (e.g., aspergillosis, cryptococcosis, blastomycosis)
Neoplasia (especially adenocarcinoma); bleeding is variably observed
Epistaxis
Acute nasal trauma
Oronasal fistula

Extranasal Causes
Purulent Nasal Discharge
Bacterial pneumonia
Megaesophagus with aspiration pneumonia, congenital or acquired
Achalasia with nasal reflux of food
Acquired esophageal stricture

Epistaxis
von Willebrand disease (most common canine coagulopathy)
Factor VIII deficiency (classic hemophilia)
Other inherited factor deficiencies
Thrombocytopenia (infectious [especially *Ehrlichia canis*] or immune-mediated)
Disseminated intravascular coagulation
Hyperviscosity syndrome

Diagnostic Plans (Figure 3-4)

Spontaneous bleeding: hemorrhage

Definition
Spontaneous or prolonged bleeding is the visible, abnormal discharge of blood resulting from a failure of one or more hemostatic mechanisms. It may result from deficiencies in platelet numbers or function, in the extrinsic or intrinsic coagulation cascades, or in vascular integrity.

The hemostatic response is a complex defense system that fulfills three basic functions: ensures that blood is confined to the vascular system of the normal animal (vascular integrity), causes the arrest of bleeding at sites of vascular injury, and maintains the patency of the vascular network.

These functions are accomplished through complex interactions among blood platelets, the blood vessel wall, and a variety of plasma enzyme systems. Disorders affecting these interactions can result in spontaneous or prolonged bleeding.

The primary phase of hemostasis occurs with platelet aggregation and the formation of the relatively unstable platelet plug. The secondary phase of hemostasis, essential to complete hemostasis, reinforces the platelet plug with fibrin. Secondary hemostasis depends on adequate plasma concentration of procoagulant proteins and on their proper interaction. Coagulation can be initiated through an intrinsic pathway, which involves
Clinical signs

- Dx → Neoplasia
- Dx → Mycosis
- Dx → Foreign body
- Dx → Nasopharyngeal polyps

- Dx → Oronasal fistula
- Dx → Feline viral upper respiratory infection
- Dx → Cleft palate
- Suspect: Neoplasia
- Suspect: Mycosis
- Dx → Nasopharyngeal polyps
  (feline only)

- Additional diagnostic studies
  - Dx → Pneumonia
  - Dx → Megaesophagus

- Nasal cavity/frontal sinuses
  - nasal flush
  - Nasal biopsy
    (closed or open)

- Thoracic
  - Dx → Pneumonia
  - Dx → Megaesophagus

- Visual exam
  - Rhinoscopy
  - Pharyngoscopy

- Platelet count
  - PT
  - PTT
  - ACT
  - CT
  - Factor VIII:Ag

  | Platelet count | PT | PTT | ACT | CT | Factor VIII:Ag |
  |----------------|----|-----|-----|----|---------------|
  | ↓              | N  | N   | N   | (↑) | ↑  |
  | N              | N  | or  | N   | or  | ↑  |
  | N (↑)          | ↑  | ↑   | ↑   | ↑  | N  |
  | N              | ↑  | ↑   | ↑   | ↑  | N  |
  | ↓              | ↑  | ↑   | ↑   | ↑  | N  |

- Dx → Thrombocytopenia
- Dx → Von Willebrand disease
- Dx → Factor deficiency
- Dx → Anticoagulant toxicity
- Dx → Disseminated intravascular coagulation

- Supports systemic and extranasal causes of nasal discharge
  + Epistaxis

- Coagulation profile

- Positive findings
  - Epistaxis
  - Visual exam
- Routine laboratory data base
  - Visual exam
  - Rhinoscopy
  - Pharyngoscopy
  - Nasal flush
  - Nasal biopsy
  - Coagulation profile

- Negative findings
  - Dx → Neoplasia
  - Dx → Mycosis
  - Dx → Foreign body
  - Dx → Nasopharyngeal polyps
  - Dx → Benign nasal polyps

Positive findings

Figure 3-4  Clinical algorithm for the patient presented for sneezing, nasal discharge, or both. \( ACT \) activated clotting time; \( PT \), prothrombin time; \( PTT \), partial thromboplastin time; \( CT \), clotting time; \( Factor\ VIII:Ag \), \( Factor\ VIII-related\ antigen \); ↓, decreased (numbers); ↑, prolonged (time); \( N \), normal; \( N (↑) \), usually normal, occasionally prolonged; \( (N) \), usually prolonged, occasionally normal.
components normally found within the vasculature and which is activated by contact with a foreign surface. The extrinsic pathway is an alternative mechanism through which clotting is initiated.

Secondary hemostasis is regulated by inhibitory products that limit the extent of enzymatic reaction and prevent their dissemination: antithrombin III, a potent inhibitor of kallikrein; factors IXa, XIa, XIIa, and Xa; and thrombin. The fibrinolytic system, another plasma protein-enzyme system, removes the hemostatic plug once its function has been served.

**ASSOCIATED SIGNS**

Bleeding disorders are most apparent when bleeding develops spontaneously from one or more body orifices and is prolonged. Bleeding from the nose (epistaxis; see Figure 3-4) is perhaps the most commonly reported outward manifestation of a bleeding disorder in dogs. Bleeding into the skin or mucous membranes (e.g., petechiation) may not be immediately apparent to even the most observant owner. Excessive or prolonged bleeding into soft tissues (hematoma) or joints (hemarthrosis) may be seen as physical enlargement of the affected tissues, with pain and lameness.

There may be a history of recurrent minor bleeding episodes in some animals. The severity of clinical signs depends on such factors as type of defect, degree of clotting factor activity, and individual variation. Moderately to severely affected animals are typically young at the time of presentation. Prolonged bleeding during or after elective surgical procedures may be the first sign of a bleeding disorder.

**DIFFERENTIAL DIAGNOSIS**

| DIFFERENTIAL DIAGNOSIS OF SPONTANEOUS BLEEDING |
|-----------------------------------------------|
| **HEREDITARY DISORDERS—FACTOR DEFICIENCIES**     |
| Hypoprothrombinemia (factor II)—Boxers          |
| Hypoproconvertinemia (factor VII)—Beagles, Malamutes |
| Hemophilia A (factor VIII)—Most dog breeds and cats |
| Hemophilia B (factor IX)—Several dog breeds and British Shorthair cats |
| von Willebrand disease (vWD factor)—Most dog breeds |
| Stuart factor deficiency (factor X)—Cocker Spaniels |
| Plasma thromboplastin antecedent (PTA) deficiency (factor XI)—Springer Spaniels, Great Pyrenees, Kerry Blue Terriers |
| Hageman factor deficiency (factor XII)—cats      |
| **HEREDITARY PLATELET DISORDERS**                |
| Thrombocytopenia                                |
| Platelet dysfunction                            |
| Thrombasthenia (Glanzmann disease)*             |
| Thrombopathia (e.g., osteogenesis imperfecta, Ehlers-Danlos syndrome)* |
| **ACQUIRED CLOTTING FACTOR DISORDERS**          |
| Primary hyperfibrinolysis                       |
| Disseminated intravascular coagulation (DIC)    |
| Chemical- or drug-induced                       |
| Vitamin K deficiency                            |
| Rodenticide ingestion                           |
| Prolonged enteric antimicrobial therapy*        |
| Circulating anticoagulants                      |
| Heparin                                         |

*Continued*
DIFFERENTIAL DIAGNOSIS OF SPONTANEOUS BLEEDING—CONT’D

Warfarin
Warfarin-like chemical (e.g., diphacinone)
Plasma expander therapy*
Liver disease
Disseminated intravascular coagulopathy (DIC)
Vitamin K deficiency
Decreased factor synthesis subsequent to severe liver disease*

ACQUIRED PLATELET DISORDERS
Thrombocytopenia (relatively common)
Decreased or ineffective thrombopoiesis*
Immunologic destruction: immune-mediated, infectious, drug-induced
Consumption: DIC, vasculitis
Sequestration: splenomegaly subsequent to neoplasia*
Dilutional: Intravenous fluid administration*
Platelet dysfunction
Secondary to underlying disease: renal failure and uremia, hepatic failure, polycythemia*
Drug-induced: aspirin, phenylbutazone, estrogen, phenothiazines, plasma expanders*

* These occur rarely.

DIAGNOSTIC PLANS

1. History. Age (inherited versus acquired), sex (sex-linked versus autosomal), and breed (inherited versus acquired) of the bleeding patient must be carefully considered. Bleeding disorders in related animals should also be considered. A detailed history of recent or current drug administration and vaccination is critical.
2. Physical examination. This may be normal. However, evidence of melena, hematuria, epistaxis, and hematoma or hemarthrosis should be pursued. The skin and mucous membranes should be inspected for evidence of petechiae or ecchymoses.
3. Routine laboratory database, to include a platelet count, is indicated in all bleeding patients to assess for the presence of underlying contributory diseases, as well as the possible consequences of bleeding within major organs.
4. Antibody titers for ehrlichiosis and Rocky Mountain spotted fever.
5. Coagulation screening tests (see also Section 5):
   a. Peripheral blood smear (for the presence of platelets)
   b. Platelet count followed by buccal mucosal bleeding time (a test of platelet function) in the presence of adequate platelet numbers
   c. Assessment of clot retraction
   d. Prothrombin time (PT)
   e. Activated partial thromboplastin time (APTT)
   f. Thrombin clotting time
   g. Fibrinogen
   h. Fibrin degradation products
   i. Clot lysis
6. Specialized laboratory tests (special facilities required):
   a. Specific factor activity assays
   b. Platelet function studies (adhesion, aggregation, secretion)
   c. Antiplatelet antibody
   d. Antithrombin III
   e. Kallikrein
   f. Electron microscopic assessment of platelets
Straining to Defecate: Dyschezia

Definition
Dyschezia is painful or difficult evacuation of feces from the rectum. In the clinical setting, dyschezia may be a difficult problem to ascertain in cats and female dogs unless the owner is particularly astute and is able to distinguish effort to urinate (see Straining to Urinate: Dysuria) from effort to defecate. Therefore a concerted effort on the part of the clinician is usually necessary to differentiate disorders affecting the urinary outflow tract and micturition from disorders affecting defecation.

Rectal or perianal pain is among the most common causes for dyschezia. The origin of the pain may be mucosal, mucocutaneous (anal), or extraluminal (rectal). Rectal strictures are uncommon but may contribute to constipation and associated dyschezia. Strictures typically develop subsequent to neoplasia or deep, nonpenetrating injury to the rectum. Although uncommon, dyschezia may also occur subsequent to lesions in the lumbar spinal cord or sacrum.

Associated Signs
The most common response to dyschezia is constipation, although many owners do not recognize this as a primary problem. Not uncommonly, the pain associated with rectal lesions is intense during attempts to defecate. The animal may cry or turn abruptly and lick the anus in response to the pain. Dogs may circle while assuming the position to defecate. Cats are more likely to make many attempts at defecation or may manifest inappropriate defecation in locations outside of the litter box. Unless attempting defecation, the animal is likely not to manifest pain at all.

Physical examination should include digital examination of the rectum and inspection of the perineum and each anal sac for evidence of lesions. It is important to consider shaving the perineum to assess the integrity of the skin for evidence of lesions, particularly neoplasia.

Differential Diagnosis

| Differential Diagnosis of Dyschezia |
|----------------------------------|
| **Constipation (see Box 3-5)**   |
| Idiopathic ulcerative and inflammatory lesions (e.g., rectal carcinoma, colon) |
| Colon (colitis)                  |
| Rectum (proctitis)              |
| Anal glands (pain associated with inflammation or neoplasia; usually determined at surgery) |
| Neoplasia                        |
| Mucosa (e.g., rectal carcinoma)  |
| Intestinal wall (e.g., carcinoma, sarcoma) |
| Extramural (intraabdominal prostate) |
| Anal glands                      |
| Perineum (particularly skin or mucocutaneous tissues) |
| **Direct Rectal Injury**         |
| With stricture formation         |
| Without stricture formation (e.g., linear foreign body, benign tumor) |
| **Perineal Hernia**              |

Diagnostic Plans
1. History and physical examination, to determine the ability of the patient to urinate versus defecate. Physical examination must include the following:
   a. Rectal temperature, also a means of detecting source of pain
b. Rectal examination, carefully expressing both anal glands and assessing the character of the discharge (sedation may be required)
c. Evaluation of the perianal skin (carefully shaving the perineum is recommended)
2. Abdominal radiographs or abdominal ultrasound to assess prostate size (in male dogs), presence of intraabdominal masses, or presence of fecalith formation.
3. Colonoscopy or proctoscopy, with rigid or flexible endoscope and biopsy of any obvious lesions. Recovered tissues should be examined cytologically and by histopathology. Anesthesia is rarely required for this procedure unless the integrity of the rectal mucosa is substantially compromised or pain is significant.
4. Rarely, exploratory laparotomy, to further elucidate the nature of abnormal intraabdominal findings.

STRAINING TO URINATE: DYSURIA

DEFINITION
Dysuria is painful or difficult urination. A relatively common presenting sign in both dogs and cats, dysuria should be regarded as an urgent situation worthy of immediate attention. Owner observations are not entirely reliable in describing dysuria. Therefore physical examination is usually necessary to differentiate attempts to defecate from attempts to urinate and to distinguish between incontinence and dysuria.

Dysuria generally results from disorders of the lower urinary tract (bladder or urethra), genital tract (prostate or vagina), or both that induce an impediment to urinary outflow resulting in abnormal micturition or inappropriate urination. However, a variety of neurologic lesions, particularly lesions in the caudal lumbar spine and sacrum affecting either parasympathetic or sympathetic innervation to the lower urinary tract, can result in dysuria. Neurologic dysurias are among the most difficult to characterize and to treat.

ASSOCIATED SIGNS
Clinical signs associated with dysuria can often be localized to the point of the primary lesion in the lower genitourinary tract. Dysuria is commonly associated with discolored urine (particularly hematuria), pyuria, or both, subsequent to mucosal inflammation and infection. Certain causes of urinary incontinence may also result in dysuria. The owner may also report frequent attempts at urination by the animal.

Distinguish between two additional clinical signs associated with dysuria: PU (increased volume) versus pollakiuria (increased frequency). Patients with dysuria may also manifest strangury, defined as a slow, painful discharge of urine caused by spasm of the bladder and urethra. In male dogs, dysuria caused by an enlarged prostate may also be associated with constipation.

Differential Diagnosis

| Differential Diagnosis of Dysuria |
|----------------------------------|
| **Infectious and inflammatory causes** |
| Bacterial cystitis |
| Urethritis |
| Prostatitis or benign prostatic hyperplasia (male dog) |
| Vaginitis |
| Feline lower urinary tract disease (FLUTD) |
| **Cystic and urethral calculi** |
| **Neoplasia** |
| Urinary bladder |
| Transitional cell carcinoma |
| Rhabdomyoma or fibrosarcoma |
| Prostatic carcinoma |
DIAGNOSTIC PLANS

1. Preliminary measures. The initial diagnostic plan depends on confirmation of dysuria at presentation and whether, on abdominal palpation, the urinary bladder is empty or distended (Figure 3-5).
2. Routine hematology and biochemical profile.
3. Urinalysis, with specific attention to color, specific gravity, protein, glucose, occult blood, and microscopic evaluation of urine sediment.
4. Radiography of the abdomen, including the lower urinary tract. Follow nondiagnostic studies with contrast radiography of the lower urinary tract (contrast urethrography, contrast cystography, and double-contrast cystography).

SWELLING OF THE LIMBS: PERIPHERAL EDEMA

DEFINITION

Peripheral edema is a pathologic increase in the fluid volume of the interstitium of soft tissue typically affecting the head and neck, forelimbs, or hindlimbs. The distribution pattern of peripheral edema can be characterized as generalized, regional, or focal. Peripheral edema may or may not be associated with other forms of edema, such as cerebral edema or pulmonary edema.

The distinction between normal and abnormal increases in interstitial fluid volumes is difficult to establish clinically. Moderate to severe increases (30%) in interstitial fluid volume are evident on visual examination of the patient as a result of the physical changes in the tissue caused by the fluid. Any increase in the interstitial fluid volume identified by any means (e.g., histopathology, physical examination) constitutes peripheral edema.

Albumin is the smallest plasma protein and is the primary source of plasma colloidal oncotic pressure. Edema may become clinically evident as the serum albumin concentration falls below 2 g/dL. However, other factors are also involved in the formation of edema, such as decreased plasma volume and increased extracellular space associated with decreased renal excretion of sodium.

ASSOCIATED SIGNS

Patients that are presented with peripheral edema may manifest other signs. Evidence of chronic inflammatory disease, vasculitis, ecchymoses, cardiac disease, allergy, or trauma (including burns) should be considered. Patients with peripheral edema may also have primary protein-losing (renal or GI) disorders. These patients may be presented with increased water consumption or urination or diarrhea and weight loss. Severe hepatic disease may result in diminished synthesis of albumin, thereby contributing to the formation of edema.
**Figure 3-5:** Algorithm for the differential diagnosis of dysuria. *LUTD,* Lower urinary tract disease.

*If no obstruction exists, pursue bladder detrusor or neurologic dysfunction.*
**DIFFERENTIAL DIAGNOSIS OF PERIPHERAL EDEMA**

**INCREASED CAPILLARY HYDROSTATIC PRESSURE**
- Functional or structural obstruction to blood flow
  - Congestive heart failure
  - Venous obstruction
  - Compression of a vessel by a mass lesion
- Arteriovenous fistula

**DECREASED CAPILLARY ONCOTIC PRESSURE (HYPOALBUMINEMIA)**
- Protein-losing enteropathies
- Protein-losing nephropathies
- Decreased hepatic synthesis
- Decreased dietary intake (protein malnutrition)
- Chronic hemorrhage
- Exudative lesion with large surface (e.g., burns, peritonitis)

**PERMEABILITY**
- Chronic inflammatory disease (e.g., *Ehrlichia canis*)
- Vasculitis (multiple infectious causes)
- Vascular trauma
- Toxins
- Neurogenic, physical, or other vasoactive stimuli

**DECREASED LYMPHATIC DRAINAGE (LYMPHEDEMA)**
- Congenital (primary) lymphedema—an autosomal dominant trait primarily affecting the hindlimbs by 3 to 6 months of age
- Acquired (secondary) lymphedema (focal or regional)
  - Infectious, granulomatous, neoplastic, traumatic injury, or compression of lymphatics

**INCREASED INTERSTITIAL GEL MATRIX**
- Myxedema (hypothyroidism)—rare

**DIAGNOSTIC PLANS**

1. History and physical examination, to focus on cardiac, hepatic, GI, and urinary system disease. Assess jugular vein distension or pulsations, tachycardia, and ascites.
2. Clinical pathology.
   a. Routine hematology.
   b. Biochemical profile, including electrolytes, total protein, and albumin.
   c. Urinalysis.
   d. Urine protein-creatinine ratio.
3. Special laboratory testing, as indicated:
   a. Bile acids.
   b. Quantitative urinary clearance studies.
   c. Serology—viral or rickettsial infections.
   d. ANA titer, LE cell preparation, and rheumatoid factor assay.
4. Central venous pressure (CVP).
5. Radiography:
   a. Thorax. Evaluate for pericardial effusion, pleural effusion, or cardiac disease.
   b. Abdomen. Evaluate for liver or mass lesions in particular, and peritonitis.
   c. Abdominal ultrasound.
6. Contrast radiography. Angiograms or lymphangiograms are indicated to confirm an obstructive lesion or the presence of an arteriovenous fistula.
7. Serologic studies, particularly for ehrlichiosis and Rocky Mountain spotted fever.
8. Edema fluid analysis. Collect by direct insertion of a 22-gauge needle into edematous tissue. A sample is collected into plain and ethylenediaminetetraacetic acid (EDTA)–containing tubes. Fluid is analyzed for color, consistency, and turbidity as well as protein and cellularity.

9. Postcapillary venous pressure and oxygen saturation, to confirm proximal obstruction to venous drainage or an arteriovenous fistula. (Normal postcapillary venous pressure is 13 ± 4 mm Hg.)

10. Cytology and histopathology. Studies are useful in evaluating mass lesions associated with edematous tissue.

**UNCONTROLLED URINATION: URINARY INCONTINENCE**

**DEFINITION**

Urinary incontinence is the lack of normal ability to prevent discharge of urine from the bladder. Urinary incontinence should be suspected when an animal that previously exhibited normal control of urination begins passing urine at times or in places that are inappropriate. Determining whether or not the presenting complaint of inappropriate urinary behavior is involuntary can be a formidable task in a dog or cat. Distinguishing between voluntary and involuntary urination is fundamental to the diagnostic plan.

The normal micturition reflex is a result of the complex interaction of the autonomic and somatic nervous systems. Normal control of micturition can be divided into a series of nervous pathways:

1. Sensory neurons have stretch receptors in the bladder wall that relay information through ascending spinal cord tracts to the brainstem and somesthetic cortex of the frontoparietal lobes. This pathway is the basis for the perception of a full bladder.

2. The frontoparietal motor cortex projects to the brainstem reticular formation centers for micturition, which are responsible for storage and evacuation of urine.

3. From these centers, reticulospinal tracts descend the spinal cord to influence gray matter centers responsible for the storage or evacuation of urine. For evacuation, the visceral efferent neurons in the sacral segments that innervate the detrusor muscle via the pelvic nerves are facilitated. The somatic efferent neurons in the sacral segments that innervate the striated urethralis muscle via the pudendal nerve are inhibited. Facilitation of these pudendal somatic neurons prevents urination. Urinary incontinence is the physical manifestation of any one of several disorders affecting voluntary urine retention in the bladder. Neurologic lesions involving either upper motor or lower motor neuron segments of the micturition reflex arc result in urinary incontinence. A paralytic bladder usually results in bladder overdistension and urine dribbling. Urine can be easily expressed by manual compression of the bladder in affected patients. A “cord bladder” is caused by a lesion between the brain and the spinal reflex center of micturition. There is usually temporary bladder paralysis followed by involuntary reflex micturition subsequent to manual compression.

Nonneurogenic urinary incontinence may be caused by anatomic or functional disorders (e.g., ectopic ureters) affecting the storage phase of micturition. Hormone-responsive incontinence is also a common form of nonneurogenic urinary incontinence. In these patients (usually dogs), the detrusor reflex is normal; normal urination behavior, in addition to urine dribbling, occurs.

A number of disorders of micturition are associated with excessive outlet resistance (e.g., urethral calculi, neoplasia) during voiding. Bladder overdistension and urine dribbling are frequently accompanied by dysuria and hematuria.

**ASSOCIATED SIGNS**

Evidence of urine or blood-tinged urine on the hair coat around the genitalia or on the patient’s sleeping surface is frequently the first sign of a micturition disorder that owners recognize. Patients with neurogenic urinary incontinence may show evidence of spinal
cord disease with conscious proprioceptive deficits in the hindlimbs, foot drag, and abrasions on the dorsal aspect of the hindfeet. However, lesions involving the cerebral cortex and cerebellum may also be associated with incontinence, as can behavioral disorders.

Obvious straining to urinate, particularly if associated with an enlarged abdomen, may indicate obstructive disease. Affected patients may be uremic, manifesting characteristic signs of lethargy, anorexia, and vomiting.

**DIFFERENTIAL DIAGNOSIS**

| DIFFERENTIAL DIAGNOSIS OF URINARY INCONTINENCE |
|-----------------------------------------------|
| **NEUROGENIC**                                   | Urethral incompetence |
| Cerebral lesions                            | Neoplasia             |
| Cerebellar lesions                         | Reduced bladder capacity|
| Brainstem lesions                           | Cystitis              |
| Spinal cord lesions                        |                         |
| Spinal nerve root lesions                   |                         |
| **NONNEUROGENIC WITH DISTENDED BLADDER**       |                         |
| Ectopic ureter(s)                           | Urethral obstruction, calculi, or neoplasia|
| Patent urachus                               | Detrusor-urethral dyssynergia|
| Hormone-responsive incontinence              | Overflow incontinence (associated with polyuric states) |

**DIAGNOSTIC PLANS**

1. History and physical examination. The size of the urinary bladder must also be determined.
2. Neurologic examination. A thorough neurologic examination should be performed in an attempt to establish or rule out a neurogenic cause. Particular emphasis is given to the spinal cord and sacral nerve roots. The bulbourethral and perineal reflexes should be assessed.
3. Catheterization of urinary bladder, to determine residual urine (normal < 0.2 to 0.4 mL/kg in the dog and cat). Urine collected is submitted for urinalysis and, as indicated, for culture and sensitivity.
4. Laboratory database, to evaluate patient health status.
5. Survey radiographs of the caudal abdomen and spinal cord.
6. Contrast studies, as needed, including pneumocystogram (only in the absence of hematuria), contrast urethrogram, and excretory urogram (also called *intravenous pyelogram*).
7. Cystometrogram. Special equipment is required.

**VISION LOSS: TOTAL BLINDNESS**

**DEFINITION**

Blindness is the inability to perceive visual stimuli. Because loss of visual function in animals is typically characterized by a change in behavior, the ability of pet owners to detect vision loss depends on their perception of changes in the animal’s awareness of and interaction with its surroundings. Vision loss is likely to be apparent to owners only when there is complete loss of vision. An owner is unlikely to detect visual deficits, such as partial vision loss or unilateral blindness, because of the animal’s ability to compensate.

Blindness can occur in any of four ways: (1) lesions causing opacification of clear ocular media (e.g., cornea, aqueous humor, or lens); (2) failure of the retina to process visual images; (3) failure of neurologic transmission; and (4) failure in the final image processing (i.e., cortical blindness).
DIFFERENTIAL DIAGNOSIS

When an animal is presented with acute visual loss, the owner is usually describing a bilateral ocular disease problem or the possibility of a CNS disorder. Acute unilateral visual loss problems are not often recognized except by the very astute animal owner or observer. For the veterinarian, initial assessment of the animal with acute visual loss depends initially on confirming that the ocular media are clear and allow light to pass from the anterior ocular segment and reach the photoreceptor cells (rods and cones) in the posterior ocular segment. Transillumination should be used to evaluate the ocular media. Such conditions as acute bilateral uveitis, severe corneal edema, bilateral acute keratitis, rapidly developing metabolic cataracts, or acute cyclitis with vitreous involvement may alter the ocular media to interfere with light transmission. Both direct and indirect pupillary responses should be evaluated while evaluating the anterior ocular media. Once it has been determined that light can reach the posterior ocular segment, a fundus evaluation should be done. Fundic abnormalities associated with acute visual loss may include acute choroidal hemorrhages, often associated with abnormal blood pressure in chronic renal disease; and acute optic neuritis.

Acute visual loss in the dog without accompanying fundic lesions that can be seen on ophthalmoscopic examination may be associated with a retrobulbar optic neuritis or with the syndrome of sudden acquired retinal degeneration syndrome (SARDS) in the dog. SARDS is poorly understood. The syndrome appears to involve middle-aged to old female dogs, and there is a breed predilection for the dachshund. The visual loss may first start as nyctalopia and progress over a period of weeks to complete visual loss. In some cases the visual loss is generalized and acute. Associated systemic signs of PD, PU, polyphagia, obesity, and hepatomegaly may be present. Laboratory profiles may show abnormal differentials in the WBC count, elevated liver enzymes, an abnormal response to ACTH stimulation testing, or an abnormal response to low-dose dexamethasone suppression testing. The fundus may appear absolutely normal, or early signs of retinal thinning and atrophy may be evident. Differential diagnosis with optic neuritis is based on electroretinographic (ERG) testing; the ERG response is flat in SARDS but is normal in optic neuritis. The cause of SARDS is unknown.

Acute visual loss associated with tumors of the CNS, particularly CNS tumors that involve the optic chiasm, are infrequently reported in the dog. Pituitary tumors are most likely to be the source. Pituitary tumors must become macroadenomas before invading and involving midbrain structures and the optic chiasm region. It is not uncommon for macroadenomas to be nonfunctional; thus the affected animal may not develop any clinical metabolic abnormalities. Papilledema is rarely observed with brain tumors in dogs. Although pituitary macroadenomas that produce chiasmal compression and visual loss are rare in dogs, the differential diagnosis must still be considered.

The availability of CT has provided the ability to diagnose tumors of the hypothalamus that may be associated with acute visual loss. In addition, the use of the same technique has made visualization of the adrenal glands and the ability to diagnose bilateral adrenal gland hyperplasia easier. Pituitary macroadenomas are larger than 1 cm in diameter.

Optic neuritis may manifest as an acute visual loss problem. There may or may not be observable ophthalmoscopic changes of the optic nerve. Ophthalmoscopic abnormalities are characterized by edema of the disk, hemorrhages in and around the disk, edema, and inflammation of the surrounding retinal tissue. Acute optic neuritis often persists as a retrobulbar lesion without any ophthalmoscopically observable lesions. Pupils are widely dilated and nonresponsive or poorly responsive to light. In suspected acute optic neuritis, a complete physical examination, including a neurologic evaluation, peripheral blood count, and CSF analysis, should be performed, if possible. The presence of pleocytosis and increased protein content in the CSF is of significance. It may be difficult to specifically diagnose the cause of acute optic neuritis.
1. Evaluate pupillary light responses and vision by evaluating the animal’s vision in an obstacle course and in altered light conditions.
2. Perform an ophthalmic examination to evaluate the clarity of the ocular media and the ability of light to reach the photoreceptor cells. Evaluate the posterior ocular segment by performing an ophthalmoscopic examination.
3. Evaluate the general physical condition of the animal including a basic neurologic examination:
   a. If acute retinal or vitreal hemorrhage is present, determine if the bleeding involves only the eyes or if there is evidence of bleeding elsewhere in the body. Determine if blood pressure is normal and if there is evidence of chronic renal disease, hyperadrenocorticism, or hyperthyroidism.
   b. If active chorioretinitis with or without exudative retinal detachment is present, determine if the inflammation appears granulomatous; if it does, consider systemic fungal infections and consider performing vitreal or subretinal aspiration and cytologic examination to look for fungal agents. If inflammation is not granulomatous, perform a complete physical examination, CBC, and chemistry panel, and look for evidence of other systemic inflammatory diseases.
   c. If acute visual loss is unaccompanied by any fundus abnormalities, perform a complete physical examination, including a basic neurologic evaluation; if acute retrobulbar optic neuritis is suspected, a CBC and CSF examination should be considered; ERG examination may be indicated to distinguish between SARDS and acute optic neuritis.
VOMITING

See also Regurgitation.

DEFINITION

Vomiting is forceful ejection of food or fluid through the mouth from the stomach and occasionally the proximal duodenum. The term applies to those animals with overt evidence of effort associated with the expulsion of food and is characterized by vigorous abdominal pressing, arched back, gagging or retching, and hypersalivation. Projectile vomiting is the term used to describe the violent ejection of stomach contents without nausea or retching. Regurgitation, on the other hand, denotes expulsion of food or fluid from the esophagus and is a considerably more passive act than vomiting.

Note: Cough-induced gagging associated with tracheitis or tracheobronchitis is often accompanied by the expulsion of mucus from the respiratory tract and can be a forceful act. As such, productive coughs may appear to the owner to be vomiting.

Vomiting is a complex reflex that entails coordination of the GI tract, musculoskeletal system, and nervous system. Although the CNS vomiting center initiates vomiting, it must first be stimulated. Even when vomiting is drug induced, stimulation of the vomiting center is accomplished subsequent to stimulation of a medullary chemoreceptor trigger zone that forwards impulses to the vomiting center. Many sensory nerves can mediate emetic impulses. Therefore, intense pain (especially abdominal); nervous (psychogenic) stimuli; disagreeable odors, tastes, and smells; sensations from the labyrinth and pharyngeal areas; various toxins and drugs; and presumably the retention of metabolic waste products all may lead to vomiting. Numerous receptors for vomiting are located in the abdominal viscera, especially the duodenum. Afferent nerve fibers are found in the vagal and sympathetic nerves.

Vomiting can be quite debilitating. When excessive, it causes severe extracellular fluid deficits, particularly of sodium, potassium, and chloride ions and water. Loss of mainly gastric contents results in loss of hydrogen ions, a high serum bicarbonate concentration, and metabolic alkalosis. Vomited material from the proximal intestinal tract contains high concentrations of bicarbonate.

Clinically, vomiting should be addressed as a problem that originates from the GI tract (primary causes) or from causes outside the GI tract (i.e., metabolic causes [secondary]).

ASSOCIATED SIGNS

Depending on the underlying cause, vomiting may be associated with a number of significant clinical signs. Primary causes of vomiting are generally associated with other GI signs, such as diarrhea, abdominal pain, obvious foreign bodies (e.g., a linear foreign body entrapped proximally under the tongue), ingestion of known irritant materials or drugs, hematochezia, or palpable abdominal tumors. Animals with metabolic or secondary causes of vomiting may appear lethargic, anorexic, and weak, particularly when the vomiting episodes have been sustained for several days. In some animals, PU or PD, anuria, icterus, cough, and anemia are present.

DIFFERENTIAL DIAGNOSIS

| INFECTIOUS CAUSES              | Leptospirosis                  |
|--------------------------------|--------------------------------|
| Feline panleukopenia virus infection | Bacterial enteritis             |
| Canine parvovirus infection     | Parasitic enteritis            |
| Canine coronavirus infection    | Heartworm disease (cats)       |
| Infectious canine hepatitis     |                                |
**VOMITING BLOOD: HEMATEMESIS**

See also Vomiting.

**DEFINITION**

Hematemesis is the vomiting of blood. It is an uncommon presentation in the dog and particularly rare in the cat. Although the presence of blood in the vomitus is, by strict definition, hematemesis, repeated episodes of vomiting in which the vomitus is composed of large blood clots, frank, uncoagulated blood, or blood with the so-called “coffee-grounds” appearance after having been denatured by gastric acid are a serious clinical finding.

**ASSOCIATED SIGNS**

Hematemesis does not localize the diagnosis to the stomach or GI tract. Because a variety of metabolic and coagulation disorders may result in severe hematemesis, a wide spectrum of physical signs may also be present in affected animals. In addition, blood emanating...
from the upper respiratory tract may be swallowed and subsequently vomited, giving the appearance that bleeding is from the stomach.

Anorexia and vomiting are the most common associated, but nonspecific, signs. Weight loss, weakness, dark stool (melena), dehydration, and inactivity are other related signs having low diagnostic yield. Severe anemia can result from sustained gastric hemorrhage and if acute may justify exploratory laparotomy to identify the source of the bleeding.

Increased water consumption and urination may suggest underlying renal or hepatic disease. Intracutaneous or subcutaneous tumors, specifically mast cell tumors, can be associated with severe gastric ulceration and bleeding. Ulcerative lesions in the mouth may indicate recent ingestion of caustic or toxic compounds. The frenulum in the mouth should always be examined to rule out linear foreign bodies.

**Differential Diagnosis**

| Primary Gastric Disorders | Systemic Metabolic Disorders |
|---------------------------|-----------------------------|
| Gastritis                 | Acute pancreatitis          |
| Infection (e.g., parvovirus) | Adrenocortical insufficiency (Addison disease) |
| Toxins                    | Toxins (e.g., lead, ethylene glycol) |
| Bile reflux–bilious vomiting syndrome | Hepatic failure |
| Foreign body              | Chronic renal failure       |
| Gastric ulcers            | Neoplasia (mast cell tumor) |
| Drug-induced disorders (e.g., aspirin) | Coagulopathy |
| Idiopathic disorders      |                             |
| Metabolic disorders (e.g., renal failure) |                             |
| Neoplastic disorders (e.g., carcinoma) |                             |

**Diagnostic Plans**

1. Comprehensive history. This is critical and should focus on the following:
   a. Recent medications administered, both prescription and nonprescription
   b. Known and potential exposures to toxic or poisonous substances
   c. Duration of the primary and associated signs
   d. Physical appearance of the vomitus
   e. Physical status of other pets in the family, if applicable
2. Laboratory profile, including, as a minimum, hematologic values, particularly in anemic patients; biochemistry findings; urinalysis; and fecal flotation. Emphasis should be placed on renal, adrenal, and hepatic function.
3. Testing of feces for the presence of parvovirus antigen.
4. Activated coagulation time (ACT). A coagulation panel—including partial thromboplastin time (PTT), prothrombin time (PT), fibrin degradation products (FDPs), fibrinogen, and total platelet count—is indicated as appropriate.
5. Fine-needle aspiration of any intracutaneous or subcutaneous tumors.
6. Abdominal and thoracic radiographs; abdominal ultrasound.
7. Gastroscopy and esophagoscopy.
8. Exploratory laparotomy and gastrotomy.

*Note: In patients with severe hematemesis, surgery may be indicated before the results from the laboratory profile are obtained.*
WEAKNESS, LETHARGY, FATIGUE

DEFINITION

Weakness is a term commonly used by owners to describe a pet that manifests a behavior change characterized by episodic (intermittent) or continuous decline in endurance or the ability to perform routine physical tasks (walking, running, fetching). However, the cause of true weakness in a dog or cat can be difficult to characterize and even more difficult to diagnose. Most authors today consider conventional terms such as fatigue and lethargy to be interchangeable with weakness. The terms depressed and depression, although sometimes used in describing weakness, are less appropriate terms, as they are conventionally reserved for describing mood or psychomotor disorders in humans.

ASSOCIATED SIGNS

Characterizing weakness can be complicated by the fact that the patient appears normal during physical examination. The clinician must carefully discern any pertinent events that either precede or follow intermittent weakness. Age, breed, consumption of food, inappropriate water consumption, activity or exertion, conformation, underlying medical disorders, and concurrent drug therapy must be considered. Although frequently implicated in the cause of weakness, advanced age is not necessarily the primary underlying cause. Because of the extensive list of potential differential diagnoses, patients with either intermittent or continuous weakness warrant a comprehensive physical and laboratory assessment.

DIFFERENTIAL DIAGNOSIS

| DIFFERENTIAL DIAGNOSIS OF WEAKNESS |
|------------------------------------|
| **CONCURRENT DRUG THERAPY**         |
| Multiple drugs may be involved, especially anticonvulsants, corticosteroids, antitussives, cardiovascular drugs, and some antimicrobials. |
| **METABOLIC DISEASE**               |
| Renal and hepatic diseases predominate |
| Electrolyte disorders (e.g., hypokalemia, hyperkalemia, hypercalcemia, hypernatremia) |
| Acidosis or alkalosis (pulmonary and/or renal mechanisms may be involved) |
| Hypertriglyceridemia (sustained)    |
| **INFECTIOUS DISEASE**              |
| Any acute-onset or sustained infection involving any pathogenic organism, especially in young dogs and cats |
| **HEMATOLOGIC DISORDERS**           |
| Anemia predominates (acute-onset or gradual onset) |
| Bone marrow neoplasia (e.g., leukemia—multiple types, myeloma) |
| **ENDOCRINE DISORDERS (MULTIPLE DISORDERS)** |
| Hypothyroidism (especially in dogs) |
| Hyperthyroidism (feline, apathetic form) |
| Hyperadrenocorticism (Cushing syndrome) |
| Hypoadrenocorticism (Addison disease) |
| Parathyroid disorders |
| Diabetes mellitus |
| Neoplasia (insulinoma) |
1. The history and physical examination (to include blood pressure) must determine whether the patient’s weakness is episodic (intermittent) or continuous. If episodic, associated events either before or after the period of weakness should be determined.

2. A laboratory profile (hematology, biochemistry, urinalysis, fecal, heartworm and tick-borne disease [dogs], and FeLV/FIV [cats]) is essential for ruling more common conditions in or out. In patients with episodic weakness but with normal laboratory findings, obtaining hematology and biochemistry samples during a period of weakness (if feasible) may be helpful in characterizing the underlying disorder.

3. From the information obtained, establish the organ system(s) that might be involved. Advanced diagnostic testing would follow. Examples include:
   a. Thoracic radiographs and/or echocardiography (cardiorespiratory signs)
   b. Abdominal ultrasound
   c. Bile acids
   d. Comprehensive neurologic examination, including acetylcholine (ACh)-receptor antibody
   e. Endocrine testing: thyroid, adrenal, pancreas (insulin)
   f. GI testing: TLI

**DIAGNOSTIC PLANS**

**WEIGHT LOSS: EMACIATION, CACHEXIA**

**DEFINITION**

Emaciation is a serious, usually chronic and progressive condition characterized by significant (>20%) body weight loss. Cachexia is the term used to describe the end stage of emaciation. Significant weight loss, associated with emaciation or cachexia, typically results from catabolism of body fat and protein in excess of caloric intake. Increased metabolism...
(hypermetabolic), inadequate consumption or assimilation of nutrient, or excessive nutrient loss contributes to significant weight loss.

ASSOCIATED SIGNS

The clinical history should center on diet, appetite, and known health status (i.e., evidence of vomiting, diarrhea, and so on). The duration of time over which the owner perceives weight loss occurring is important. Emaciation developing within a month (e.g., with neoplasia) may carry a poorer prognosis than emaciation developing over several months. The physical examination should focus on the presence of fever, GI disease, and overt changes in size and consistency of internal organs.

DIFFERENTIAL DIAGNOSIS

A spectrum of differential diagnoses must be considered in the patient with emaciation or cachexia. Several categories of illness should be considered when emaciation or cachexia is associated with the following:

- **Malnutrition.** Quality and quantity of food, availability of food, evidence of neglect or abuse
- **Polyphagia.** Malassimilation (i.e., either maldigestion or malabsorption), hypermetabolic states (e.g., hyperthyroidism, pregnancy), excessive nutrient losses (e.g., diabetes mellitus, glomerulonephropathy)
- **Anorexia.** Infectious diseases, neoplasia, neurologic disease, toxicity (e.g., chronic lead poisoning), dental disease (pseudoanorexia)
- **GI signs.** Malassimilation (i.e., either maldigestion or malabsorption); parasitism
- **Urinary tract signs.** Excess renal loss of fluid and nutrients (polyuric states)
- **Fever.** Infectious diseases

DIAGNOSTIC PLANS (Figure 3-6)

YELLOW SKIN OR MUCOUS MEMBRANES: ICTERUS (OR JAUNDICE)

DEFINITION

Icterus, or jaundice, is a yellow discoloration of tissue (especially skin, mucous membranes, and sclera) caused by an increased serum concentration of bilirubin. It is indicative of underlying hepatocellular disease or intravascular hemolytic disease. Hyperbilirubinemia is required for icterus to develop but may not occur concurrently with icterus.

In practice, icterus is an uncommon presenting complaint, because the dense hair coat of cats and dogs precludes early detection of bile pigment in skin. Icteric tissues are most evident in the sclera and in the oral, vaginal, and preputial mucous membranes, particularly in anemic patients. Icterus can occur subsequent to the accumulation of either unconjugated (lipid-soluble) or conjugated (water-soluble) bilirubin in the blood.

Icterus can originate at any of three levels: prehepatic (hemolytic disease), hepatic (hepatocellular disease), posthepatic (obstructive or reduced bile flow).

Unconjugated hyperbilirubinemia results from rapid hemolysis (a common cause in the dog and cat), ineffective erythropoiesis, impaired hepatic uptake of conjugated bilirubin, or impaired conjugation. Conjugated (water-soluble) hyperbilirubinemia is generally the result of disorders intrinsic to the liver that affect bilirubin transport. Cholestatic disease is associated with reduced bile flow and can be characterized by significant bile acidemia and icterus.
ASSOCIATED SIGNS

Icterus can be detected in a dog or cat without overt clinical signs; however, RBC values and hepatic function should be assessed. Prehepatic icterus is characteristically associated with rapid-onset anemia and with generalized weakness, lassitude, or acute collapse (caval syndrome), and bright orange urine. Pallor can be difficult to assess in patients with marked icterus. Hepatic icterus and posthepatic icterus are generally associated with lethargy and decreased appetite and are therefore difficult to distinguish clinically. Depending on the type of hepatic injury or the level of obstruction, episodic vomiting or diarrhea, weight loss, abdominal distension, PU or PD, peripheral edema associated with hypoproteinemia, and prolonged bleeding (uncommon) may be reported.

DIFFERENTIAL DIAGNOSIS

| DIFFERENTIAL DIAGNOSIS OF ICTERUS |
|-----------------------------------|
| **Prehepatic (Hemolytic)**        | **Hepatic (Hepatocellular)**    |
| Immune-mediated hemolytic anemia  | Cholangitis or cholangiohepatitis|
| (Coombs-positive anemia)         | Chronic active liver disease     |
| Heartworm disease, especially    | Copper storage disease (Bedlington Terriers and Doberman Pinschers) |
| postcaval syndrome               | Drug-induced or vaccine-induced  |
| Hemolytic septicemia             | disease                        |
| Transfusion-induced hemolysis    | Thiacetarsamide—sporadic        |
|                                  | occurrence                     |
Diagnosis Plans

1. Thorough history. This should focus on current and previous drug therapy, including heartworm preventative, as well as duration of illness and associated signs. Physical examination confirms the presence of icterus but is unlikely to reveal the underlying cause. Abdominal palpation may reveal hepatomegaly, a discrete mass, or the presence of fluid.

   In obviously anemic patients, when practical, transfusion should be avoided until laboratory test results have been interpreted.

2. Laboratory evaluation of the icteric patient. This is essential and should initially include a CBC, biochemistry panel (to include total and direct bilirubin), fecal analysis, urinalysis, heartworm test (in dogs), serum electrophoresis (in cats), and a test for FeLV antigen and FIV antibody.

3. Anemic patient. Coombs test; ANA titer; peripheral blood smear for the presence of parasites; blood cultures, particularly if the patient is febrile; and immunofluorescent assay (IFA) on bone marrow for FeLV antigen (in cats).

4. Nonanemic patient. Abdominal radiographs, abdominocentesis with fluid analysis and cytologic study, fine-needle aspiration of liver, plasma ammonia, bile acids, serum amylase, and lipase if not included in the biochemistry panel.

5. Special diagnostic tests. Coagulation profile, followed by liver biopsy (percutaneous or at laparotomy) or exploratory laparoscopy with biopsy.

6. Abdominal ultrasound, CT, and perfusion scintigraphy (special facilities required).

Additional Reading

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