Kirk and Bistner’s Handbook of

VETERINARY PROCEDURES
AND EMERGENCY TREATMENT
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AND
EMERGENCY TREATMENT

NINTH EDITION

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Dr. Robert W. Kirk
May 20, 1922 – January 20, 2011

Clinician, educator, dedicated teacher.
A man whose commitment and contributions to companion animal medicine have been global in reach and legendary in scope.
The 9th edition of *Kirk & Bistner’s Handbook of Veterinary Procedures and Emergency Treatment* exemplifies the pace of change occurring in veterinary medicine today. The veterinary profession and the patients we serve continue to benefit from impressive technological advances in emergency and critical care medicine, diagnostic testing, and therapy. As the editors of this edition, we have made significant effort to include current diagnostic techniques, procedures, and management recommendations consistent with the standards of care in companion animal medicine.

To facilitate quick and easy access to information, the text is divided into six distinct sections with special emphasis placed on **Section 1**, Emergency Diagnostic and Therapeutic Treatment. This section is organized to facilitate rapid access to diagnostic and treatment recommendations for emergency and critical care patients. Included are major subsections on Prehospital Management, Initial Emergency Triage and Management, Emergency Procedures, Pain Assessment and Management, and Emergency Management of Specific Conditions.

Sections 2 through 5 focus on diagnostic strategies including patient evaluation, problem identification, routine and advanced procedures, and laboratory testing/interpretation. Each of these four sections addresses specific aspects of the patient’s clinical presentation.

**Section 2**, Patient Evaluation and Organ System Examination, focuses on the initial patient assessment and includes templates for medical record entries and plans for advanced diagnostics.

**Section 3**, Clinical Signs, is a problem-based approach to differential diagnoses and is redesigned such that the patient’s problem is represented from the client’s perspective—the same way problems are presented in clinical practice.

**Section 4** addresses both routine and advanced diagnostic, as well as therapeutic, procedures. Advanced procedures are now presented in an organ-system format to enhance access to current diagnostic procedures that may be needed when evaluating complex cases.

**Section 5**, Laboratory Diagnosis and Test Protocols, is a succinct, highly structured reference for performing routine and advanced diagnostic testing in cats and dogs. Each test represented includes information on patient preparation, the test protocol, type of sample to collect versus type of sample to submit, interpretation of test results, and more.

**Section 6** is a compilation of clinically pertinent tables and charts that have been extensively reviewed and updated. Some of the tables included provide information on Annualized Vaccination Protocol for Cats and Dogs, Common Drug Indications and Dosages, and Emergency Hotlines.

It was Dr. Robert W. Kirk who, in 1969, published the first edition of this text. It is Dr. Kirk who can be credited with being among the first academicians to recognize a unique role for emergency care in veterinary medicine. It was his vision that has ultimately led to the development and growth of specialty practices in emergency and critical care medicine. We are all indebted to Dr. Kirk for his commitment and dedication to veterinary medicine.
Regretfully, Dr. Kirk passed away earlier this year. His numerous contributions, however, will continue to serve the profession for years to come. We are honored to dedicate this edition of the *Emergency Handbook* to Dr. Kirk.

*Richard B. Ford, DVM, MS*
*Elisa Mazzaferro, MS, DVM, PhD*
Veterinary technicians and Veterinary Technician Specialists (VTS) serve a vital role in assisting the veterinarian in diagnosis, prognosis, and prescribing therapy for their patients. A thorough knowledge of the clinical manifestations of common diseases as well as methods for diagnosing and treating those diseases is essential to understanding the significance of test results. The technician is involved in monitoring the patient and performing and recording observations of patients. The rationale used by the veterinarian in choosing specific diagnostic tests and treatment protocols improves the veterinary technician’s ability to assess and monitor patients.

The expanded and updated 9th edition of this essential reference text is organized into six sections to provide rapid access to relevant information on clinical signs of disease, patient evaluation, emergency care, diagnostic and therapeutic procedures, and laboratory diagnostics, as well as charts of normal values, vaccination protocols, and a drug formulary. The sections on Diagnostic and Therapeutic Procedures and Laboratory Diagnosis and Test Protocols are particularly applicable to Veterinary Technicians, as these skills are primarily performed by the veterinary technician under the direction of the veterinarian in a progressive small animal practice. These sections as well as those on Emergency Care contain practical information related to skills performed on a daily basis by many veterinary technician specialists.

Veterinary technician students will find this text a useful adjunct to their studies. The book provides a ready reference that allows the student to quickly review clinical applications of basic concepts while studying foundation courses such as anatomy and physiology, in addition to more advanced clinical pathology, radiology, and pharmacology courses.

I hope that every veterinary technician, veterinary technician specialist, and veterinary technician student includes this valuable resource in their personal library.

Margi Sirois, EdD, MS, RVT
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PREHOSPITAL MANAGEMENT OF THE INJURED ANIMAL

SURVEY OF THE SCENE

1. *Call for help!* At the accident scene, it usually takes more than one person to assist the animal and prevent injury to the animal and human bystanders.

2. If an accident has occurred in a traffic zone, alert oncoming traffic regarding the injured animal in the road. Make sure you have a piece of clothing or other object to alert oncoming traffic. Do not become injured yourself because oncoming traffic cannot see or identify you!

3. If the animal is conscious, prevent yourself from becoming injured while moving the animal to a safe location. Use a belt, rope, or piece of long cloth to make a muzzle to secure around the animal’s mouth and head. If this is not possible, cover the animal’s head with a towel, blanket, or coat before moving it, to prevent the animal from biting you.

4. If the animal is unconscious or is unconscious and immobile, move it to a safe location with a back support device that can be made from a box, door, flat board, blanket, or sheet.

INITIAL EXAMINATION

1. Is there a patent airway? If airway noises are present or the animal is stuporous, gently and carefully extend the head and neck. If possible, extend the tongue. Wipe mucus, blood, or vomitus from the mouth. In unconscious animals, maintain head and neck stability.

2. Look for signs of breathing. If there is no evidence of breathing or the gum color is blue, begin mouth-to-nose breathing. Encircle the muzzle area with your hands to pinch down on the gums, and blow into the nose 15 to 20 times per minute.

3. Is there evidence of cardiac function? Check for a palpable pulse on the hind legs or for an apex beat over the sternum. If no signs of cardiac function are found, begin external cardiac compressions at 80 to 120 times per minute.

4. Is there any hemorrhage? Use a clean cloth, towel, paper towel, or disposable diaper or feminine hygiene product to cover the wound. Apply firm pressure to slow hemorrhage and prevent further blood loss. Do not use a tourniquet, because this can cause further damage. Apply pressure, and as blood seeps through the first layer of bandage material, place a second layer over the top.

5. Cover any external wounds. Use a clean bandage material soaked in warm water, and transport the animal to the nearest veterinary emergency facility. Address penetrating wounds to the abdomen and thorax immediately.

6. Are there any obvious fractures present? Immobilize fractures with homemade splints made of newspaper, broom handles, or sticks. Muzzle the awake animal before attempting to place any splints. If a splint cannot be attached safely, place the animal on a towel or blanket and transport the animal to the nearest veterinary emergency facility.
7. Are there any burns? Place wet, cool towels over the burned area and remove as the 
compress warms to body temperature.
8. Wrap the patient to conserve heat. If the animal is shivering or in shock, wrap it in a 
blanket, towel, or coat and transport it to the nearest veterinary emergency facility.
9. Is the animal experiencing heat-induced illness (heat stroke)? Cool the animal with 
room-temperature wet towels (not cold) and transport it to the nearest veterinary 
emergency facility.

**PREPARATION FOR TRANSPORT**

1. Call ahead! Let the facility know that you are coming. Be prepared by having emergency 
numbers and locations available. The police or sheriff’s department may be able to aid in 
locating the nearest veterinary emergency facility.
2. Line upholstery with plastic bags or sheeting to prevent soilage, when possible.
3. Move the injured patient carefully. Use the same approach as moving the animal from 
the pavement.
4. Drive safely. Do not turn one accident into two. Ideally, have a bystander or friend or 
family member drive while another person stays in the backseat with the animal.

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**INITIAL EMERGENCY EXAMINATION, MANAGEMENT, AND TRIAGE**

Examination of the acutely injured animal that is unconscious, in shock, or demonstrating 
acute hemorrhage or respiratory distress must proceed simultaneously with immediate 
aggressive lifesaving treatment. Because there often is no time for detailed history taking, 
diagnosis is largely based on the physical examination findings and simple diagnostic tests. 
Triage is the art and practice of being able to assess patients rapidly and sort them according 
to the urgency of treatment required. Immediate recognition and prompt treatment 
potentially can be lifesaving.

**PRIMARY SURVEY AND EMERGENCY RESUSCITATION MEASURES**

Perform a brief but thorough systematic examination of the animal, noting the most 
important ABCs of any emergent patient.

**ABCs**

**A = Airway**

Is the airway patent? Pull the patient’s tongue forward and remove any debris obstructing 
the airway. Suction and a laryngoscope may be necessary. Intubate, or place a transtracheal 
oxygen source, if necessary. An emergency tracheostomy may be necessary if upper 
airway obstruction is present and cannot be resolved immediately with the foregoing 
measures.

**B = Breathing**

Is the animal breathing? If the animal is not breathing, immediately intubate the animal and 
start artificial ventilations with a supplemental oxygen source (see Cardiac Arrest and 
Cardiopulmonary Cerebral Resuscitation).

If the animal is breathing, what are the respiratory rate and pattern? Is the respiratory rate 
normal, increased, or decreased? Is the respiratory pattern normal, or is the breathing rapid 
and shallow, or slow and deep with inspiratory distress? Are the respiratory noises normal, 
or is there a high-pitched stridor on inspiration characteristic of an upper airway obstruc 
tion? Does the animal have its head extended and elbows abducted away from the body with 
orthopnea? Do the commissures of the mouth move with inhalation and exhalation? Is there 
evidence of expiratory distress with an abdominal push on exhalation? Note the lateral chest 
wall. Do the ribs move out and in with inhalation and exhalation, or is there paradoxical chest
wall motion in an area that moves in during inhalation and out during exhalation, suggestive of a flail chest? Is there any subcutaneous emphysema that suggests airway injury?

Auscultate the thorax bilaterally. Are the breath sounds normal? Do they sound harsh with crackles because of pneumonia, pulmonary edema, or pulmonary contusions? Are the lung sounds muffled because of pleural effusion or pneumothorax? Are there inspiratory wheezes in a cat with bronchitis (asthma)? What is the mucous membrane color? Are the mucous membranes pink and normal, or pale or cyanotic? Palpate the neck, lateral thorax, and dorsal cervical region to check for tracheal displacement, subcutaneous emphysema, and rib fractures.

C = Circulation
What is the circulatory status? What is the status of the patient’s heart rate and rhythm? Can you hear the heart, or is it muffled because of hypovolemia, pleural or pericardial effusion, pneumothorax, or diaphragmatic hernia? Palpate the pulses. Is the pulse quality strong and regular and synchronous with each heartbeat, or are there thready, dropped pulses? What are the patient’s electrocardiogram (ECG) rhythm and blood pressure (BP)?

Is there arterial hemorrhage? Note whether there is any bleeding present. Use caution if there is any blood on the fur. Wear gloves. The blood may be from the patient, and gloves will help prevent further contamination of any wounds; or the blood may be from a good Samaritan bystander. If external wounds are present, note their character and condition. Place a pressure bandage on any arterial bleeding or external wounds to prevent further hemorrhage or contamination with nosocomial organisms.

Establish large-bore vascular or intraosseous access (see Vascular Access Techniques). If hypovolemic or hemorrhagic shock is present, institute immediate fluid resuscitation measures. Start with one fourth of a calculated shock dose of crystalloid fluids (0.25 × [90 mL/kg] for dogs; 0.25 × [44 mL/kg] for cats), and reassess perfusion parameters of heart rate, capillary refill time, and BP. If pulmonary contusions are suspected, use of a colloid such as hydroxyethyl starch at 5 mL/kg in incremental boluses can improve perfusion with a smaller volume of fluid. In cases of head trauma, hypertonic (7%) sodium chloride (saline) can be administered (4 mL/kg intravenous bolus) with hydroxyethyl starch. Acute abdominal hemorrhage caused by trauma can be tamponaded with an abdominal compression bandage.

After the immediate ABCs, proceed with the rest of the physical examination and treatment by using the mnemonic A CRASH PLAN.

A CRASH PLAN
A = Airway
C and R = Cardiovascular and Respiratory
A = Abdomen
Palpate the patient’s abdomen. Is there any pain or are there any penetrating injuries present? Look at the patient’s umbilicus. Reddening around the umbilicus can suggest intraabdominal hemorrhage. Is there a fluid wave or mass palpable? Examine the inguinal, caudal, thoracic, and paralumbar regions. Clip the fur to examine the patient for bruising or penetrating wounds. Percuss and auscultate the abdomen for borborygmi.

S = Spine
Palpate the animal’s spine for symmetry. Is any pain or obvious swelling or fracture present? Perform a neurologic examination from C1 to the last caudal vertebra.

H = Head
Examine the eyes, ears, mouth, teeth, nose, and all cranial nerves. Stain the eyes with fluorescence dye to examine for corneal ulcers in any case of head trauma. Is anisocoria or Horner syndrome present?
P = Pelvis
Perform a rectal examination. Palpate for fractures or hemorrhage. Examine the perineal and rectal areas. Examine the external genitalia.

L = Limbs
Examine the pectoral and pelvic extremities. Are there any obvious open or closed fractures? Quickly splint the limbs to prevent further damage and help control pain. Examine the skin, muscles, and tendons.

A = Arteries
Palpate the peripheral arteries for pulses. Use a Doppler piezoelectric crystal to aid in finding a pulse if thromboembolic disease is present. Measure the patient’s BP.

N = Nerves
From afar, note the level of consciousness, behavior, and posture. Note respiratory rate, pattern, and effort. Is the patient conscious, or is the patient obtunded or comatose? Are the pupils symmetric and responsive to light, or is there anisocoria present? Does the patient display any abnormal postures such as Schiff-Sherrington posture (extended rigid forelimbs, flaccid paralysis of the hindlimbs) that may signify severe spinal shock or a severed spinal cord? Examine the peripheral nerves for motor and sensory input and output to the limbs and tail.

ANCILLARY DIAGNOSTIC EVALUATION

HEMODYNAMIC TECHNIQUES
Perform electrocardiography, direct or indirect BP monitoring, and pulse oximetry in any critically ill traumatized patient.

IMAGING TECHNIQUES
Obtain radiographs of the thorax and abdomen in any animal that has sustained a traumatic injury once the patient’s condition is more stable and the animal can tolerate positioning for the procedures. Survey radiographs may reveal pneumothorax, pulmonary contusions, diaphragmatic hernia, pleural or abdominal effusion, or pneumoperitoneum.

AFAST AND TFAST
Focused assessments of the abdomen and thorax after trauma (AFAST and TFAST) have been described to evaluate the abdomen for fluid and to evaluate the thorax for fluid, free air, and pericardial effusion. During these examinations, ultrasound is used to evaluate four quadrants of the abdomen: (1) the diaphragm or hepatic view, on ventral midline just caudal to the sternum, (2) the spleno-renal view in the left lateral quadrant, (3) the cysto-colic view on ventral midline over the urinary bladder, and (4) the hepato-renal view on right lateral, the most dependent area of the abdomen with the patient in right lateral recumbency. For evaluation of the thorax, the patient is positioned in lateral recumbency and the ultrasound probe is directed in a horizontal plane at the dorsal aspect of the ninth intercostal space, in the transverse and longitudinal planes caudal to the point of the elbow, over the heart to evaluate for pericardial and pleural effusion. The examinations take only small amounts of time and can reveal whether ongoing fluid loss is occurring. As with other ultrasonographic techniques, AFAST and TFAST results are sometimes operator-dependent.
**LABORATORY TESTING**
Immediate diagnostic testing should include hematocrit, total solids, glucose, blood urea nitrogen (BUN) or Azostix, and urine specific gravity. Ancillary diagnostic tests that can be performed soon thereafter include a complete blood count and peripheral blood smear to evaluate platelet count and red and white blood cell (WBC) morphology. Also consider arterial blood gas and electrolytes, coagulation parameters (activated clotting time [ACT], prothrombin time [PT], activated partial thromboplastin time [APTT]), serum biochemistry profile, serum lactate, and urinalysis.

**INVASIVE TESTING**
Invasive diagnostic techniques that may need to be performed include thoracocentesis, abdominal paracentesis, and diagnostic peritoneal lavage (DPL).

**SUMMARY OF PATIENT STATUS**
After completing the initial physical examination, answer the following questions: What supportive care is required at this time? Are additional diagnostic procedures needed? If so, which procedures, and is the patient stable enough to tolerate those procedures without further stress? Should an additional period of observation be instituted before further definitive treatment plans are undertaken? Is immediate surgical intervention necessary? Is additional supportive care required before surgery? What anesthetic risks are evident?

**THE RAPIDLY DECOMPENSATING PATIENT**
Animals that do not respond to initial resuscitation usually have severe ongoing or preexisting physiologic disturbances that contribute to severe cardiovascular and metabolic instability. A patient that does not respond to or responds to and then stops responding to initial resuscitation efforts should alert the clinician that decompensation is occurring (Boxes 1-1 and 1-2).

### BOX 1-1 CLINICAL SIGNS OF DECOMPENSATION

| Weak or poor peripheral pulse quality | Depression |
| Cool peripheral extremities          | Tachycardia or bradycardia |
| Cyanosis or muddy-colored (gray) mucous membranes | Declining hematocrit |
| Pale mucous membranes                | Distended, painful abdomen |
| Prolonged capillary refill time      | Cardiac dysrhythmia |
| Increased or decreased body temperature | Abnormal respiratory pattern |
| Decreased renal output in a euvoletic patient | Respiratory difficulty or distress |
| Inappropriate mentation or confusion | Gastrointestinal blood loss via hematemesis or in feces |

### BOX 1-2 CAUSES OF ACUTE DECOMPENSATION

| Acute renal failure | Internal hemorrhage |
| Acute respiratory distress syndrome | Multiple organ dysfunction syndrome |
| Bowel and gastric rupture | Pneumothorax |
| Cardiac dysrhythmia | Pulmonary contusions |
| Central nervous system edema and hemorrhage, and brainstem herniation | Pulmonary thromboembolism |
| Coagulopathies including disseminated intravascular coagulation | Sepsis or septic shock |
|                         | Systemic inflammatory response syndrome |
|                         | Urinary bladder rupture |
Additional Reading

ABDOMINAL PARACENTESIS AND DIAGNOSTIC PERITONEAL LAVAGE

Abdominocentesis (abdominal paracentesis) refers to puncture into the peritoneal cavity for the purpose of fluid collection. Abdominal paracentesis is a somewhat sensitive technique for fluid collection as long as more than 6 mL/kg of fluid are present within the abdominal cavity. In the event that you suspect peritonitis and have a negative tap with abdominal paracentesis, DPL can be performed.

To perform abdominal paracentesis, follow this procedure:
1. Place the patient in left lateral recumbency and clip a 4- to 6-inch square with the umbilicus in the center.
2. Aseptically scrub the clipped area with antimicrobial scrub solution.
3. Wearing gloves, insert a 22- or 20-gauge needle or over-the-needle catheter in four quadrants: cranial and to the right, cranial and to the left, caudal and to the right, and caudal and to the left of the umbilicus. As you insert the needle or catheter, gently twist the needle to push any abdominal organs away from the tip of the needle. Local anesthesia typically is not required for this procedure, although a light sedative or analgesic may be necessary if severe abdominal pain is present. In some cases, fluid will flow freely from one or more of the needles. If not, gently aspirate with a 3- to 6-mL syringe or aspirate with the patient in a standing position. Avoid changing positions with needles in place because iatrogenic puncture of intraabdominal organs may occur.
4. Save any fluid collected in sterile red- and lavender-topped tubes for cytologic and biochemical analyses and bacterial culture. Monitor hemorrhagic fluid carefully for the presence of clots. Normally, hemorrhagic effusions rapidly become defibrinated and do not clot. Clot formation can occur in the presence of ongoing active hemorrhage or may be caused by the iatrogenic puncture of organs such as the spleen or liver.

If abdominal paracentesis is negative, DPL can be performed. Although peritoneal dialysis kits are commercially available, they are fairly expensive and often impractical for the general practitioner.

To perform DPL, follow this procedure:
1. Clip and aseptically scrub the ventral abdomen as described previously.
2. Wearing sterile gloves, cut multiple side ports in a 16- or 18-gauge over-the-needle catheter. Use care to not cut more than 50% of the circumference of the catheter, or else the catheter will become weakened and potentially can break off in the patient's abdomen.
3. Insert the catheter into the peritoneal cavity caudal and to the right of the umbilicus, directing the catheter dorsally and caudally.
4. Infuse 10 to 20 mL of sterile lactated Ringer’s solution or 0.9% saline solution that has been warmed to the patient’s body temperature. During the instillation of fluid into the peritoneal cavity, watch closely for signs of respiratory distress because an increase in intraabdominal pressure can impair diaphragmatic excursions and respiratory function.

5. Remove the catheter.

6. In ambulatory patients, walk the patient around while massaging the abdomen to distribute the fluid throughout the abdominal cavity. In nonambulatory patients, gently roll the patient from side to side.

7. Next, aseptically scrub the patient’s ventral abdomen again, and perform abdominal paracentesis as described previously. Save collected fluid for culture and cytologic analyses; however, biochemical analysis findings may be artifactual decreased because of dilution. Remember that you likely will retrieve only a small portion of the fluid that was instilled.

**Additional Reading**


**BANDAGING AND SPLINTING TECHNIQUES**

In general, bandages can be applied to open or closed wounds. Bandaging is used for six general wound types: open contaminated or infected wounds, open wounds in the repair stage of healing, closed wounds, wounds in need of a pressure bandage, wounds in need of pressure relief, and wounds in need of immobilization. Box 1-3 lists various functions of bandages.

The materials and methods of bandaging depend on the type of injury, the need for pressure and immobilization, the need to prevent pressure, and the stage of healing. In general, bandage material has three component layers. If pressure relief or immobilization is required, splint material also may be incorporated into the bandage. The contact layer is the layer of bandage material that actually is adjacent to the wound itself. The secondary or intermediary layer is placed over the contact (primary) layer. Finally, the outer tertiary layer covers the bandage and is exposed to the outside.

**Open Contaminated and Infected Wounds**

Open contaminated or infected wounds often have large amounts of necrotic tissue and foreign debris and emit copious quantities of exudate. The contact layer used in an open contaminated or infected wound should be wide-mesh gauze sponges with no

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**BOX 1-3  FUNCTIONS OF BANDAGES AND SPLINTS**

<table>
<thead>
<tr>
<th>Function</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exert pressure</td>
<td>Protect a wound from environmental bacteria</td>
</tr>
<tr>
<td>Obliterate dead space</td>
<td>Protect the environment from wound blood, exudate, and bacteria</td>
</tr>
<tr>
<td>Reduce edema</td>
<td>Immobilize a wound and support underlying osseous structures</td>
</tr>
<tr>
<td>Minimize hemorrhage</td>
<td>Minimize patient discomfort</td>
</tr>
<tr>
<td>Prevent pressure on a wound</td>
<td>Serve as a vehicle for antiseptics and antibiotics</td>
</tr>
<tr>
<td>Prevent decubitus ulcers</td>
<td>Serve as an indicator of wound secretions</td>
</tr>
<tr>
<td>Pack wounds</td>
<td>Provide an esthetic appearance</td>
</tr>
<tr>
<td>Wet-to-dry bandages—treat deep shearing injuries</td>
<td></td>
</tr>
<tr>
<td>Absorb exudate and debride wounds</td>
<td></td>
</tr>
</tbody>
</table>
cotton filling. The sponges can be left dry if the wound has minimal exudate but should be moistened with sterile 0.9% saline or lactated Ringer’s solution if the wound has high-viscosity exudate. Topical ointments may be applied (silver sulfadiazine, chlorhexidine ointment) if necessary. The intermediate layer should be thick absorbent wrapping material, covered by an outer layer of porous tape such as Elastikon (Johnson & Johnson Medical, Arlington, Texas), or Vetrap (3M, St Paul, Minnesota). Change the bandages at least once daily or more frequently if strike-through of exudate occurs through the bandage.

To place a wet-to-dry bandage over a wound, first place the contact layer over the wound. Next, apply strips of adhesive tape to the patient’s paw on either side, if possible. The strips (stirrups) will be used to hold the bandage in place and prevent it from slipping down the limb. Wrap the intermediate layer over the contact layer. Turn the adhesive strips around so that the adhesive layer can be secured to the intermediary layer in place. Wrap the final, or tertiary, layer over the bandage.

The function of a wet-to-dry bandage is to help debride a wound. The moistened gauze dries and is pulled off the wound at each bandage change. Dry necrotic tissue and debris that adhere to the gauze are pulled off with it. In addition, the moistened material dilutes the wound exudate and enhances its absorption into the gauze contact layer. If large amounts of exudate come from the wound, the contact layer and intermediate layer absorb the exudate, wicking the material away from the wound. Finally, delivery of medications into the wound can occur to promote the development of healthy granulation tissue.

**Open Wound in Repair Stage of Healing**

**Early Repair**

During the early stage of repair, granulation tissue, some exudate, and minor epithelialization are observed. Place a nonadherent bandage with some antibacterial properties (petroleum- or nitrofurazone-impregnated gauze) or absorbent material (foam sponge, hydrogel, or hydrocolloid dressing) in direct contact with the wound to minimize disruption of the granulation tissue bed. Next, place an absorbent intermediate layer, followed by a porous outer layer, as previously described. Granulation tissue can grow through gauze mesh or adhere to foam sponges and can be ripped away at the time of bandage removal. Hemorrhage and disruption of the granulation tissue bed can occur.

**Late Repair**

Later in the repair process, granulation tissue can exude sanguineous drainage and have some epithelialization. A late nonadherent bandage is required. The contact layer should be some form of nonadherent dressing, foam sponge, hydrogel, or hydrocolloid substance. The intermediate layer and outer layers should be absorbent material and porous tape, respectively. With nonadherent dressings, wounds with viscous exudates may not be absorbed well. This may be advantageous and enhance epithelialization, provided that complications do not occur. Infection, exuberant granulation tissue, or adherence of absorbent materials to the wound may occur and delay the healing process.

**Moist Healing**

Moist healing is a newer concept of wound management in which wound exudates are allowed to stay in contact with the wound. In the absence of infection a moist wound heals faster and has enzymatic activity as a result of macrophage and polymorphonuclear cell breakdown. Enzymatic degradation or “autolytic debridement” of the wound occurs. Moist wounds tend to promote neutrophil and macrophage chemotaxis and bacterial phagocytosis better than use of wet-to-dry bandages. A potential complication and disadvantage of moist healing, however, is the development of bacterial colonization, folliculitis, and trauma to wound edges that can occur because of the continuously moist environment.
Use surfactant-type solutions (Constant-Clens, Kendall, Mansfield, Massachusetts) for initial wound cleansing and debridement. Use occlusive dressings for rapid enzymatic debridement with bactericidal properties to aid in wound healing. Bandage wet necrotic wounds with a dressing premoistened with hypertonic saline (Curasalt [Kendall], 20% saline) to clean and debride the wounds. Hypertonic saline functions to desiccate necrotic tissue and bacteria to debride the infected wound. Remove and replace the hypertonic saline bandage every 24 to 48 hours. Next, place gauze impregnated with antibacterial agents (Kerlix AMD [Kendall]) over the wound in the bandage layer to act as a barrier to bacterial colonization.

If the wound is initially dry or has minimal exudate and is not obviously contaminated or infected, place amorphous gels of water, glycerin, and a polymer (Curafil [Kendall]) over the wound to promote moisture and proteolytic healing. Discontinue moisture gels such as Curafil once the dry wound has become moist.

Finally, the final stage of moist healing helps to promote the development of a healthy granulation tissue bed. Use calcium alginate dressings (Curasorb or Curasorb Zn with zinc [Kendall]) in noninfected wounds with a moderate amount of drainage. Alginate gels promote rapid development of a granulation tissue bed and epithelialization.

Foam dressings also can be applied to exudative wounds after a healthy granulation bed has formed. Change foam dressings at least once every 4 to 7 days.

**Sugar Bandages**

The use of granulated sugar has become popular in recent years to treat open wounds that are contaminated and/or infected. Sugar possesses antibacterial properties and helps promote wound healing and development of a granulation bed. Sugar bandages are an excellent choice for shearing or degloving injuries, burns, and decubital ulcers, particularly ones that are infected with *Pseudomonas* species, *Escherichia coli*, or streptococcal species.

The placement of a sugar bandage is similar to that of a wet-to-dry bandage, in that first the wound must be thoroughly lavaged with either tap water or sterile saline. Next, devitalized tissue must be debrided. Next, pour a thick layer (approximately 1 cm thick) of granulated sugar over the wound bed. Next, wrap the wound with sterile gauze squares, cotton bandage material, and an outer layer. Change the wound dressing at least once to twice daily initially, then once daily to every other day as the granulation bed becomes healthier. The sugar can be eliminated from the bandaging process once a healthy granulation bed is present.

**Closed Wounds**

**Wounds with No Drainage**

For closed wounds without any drainage, such as a laceration that has been repaired surgically, a simple bandage with a nonadherent contact layer (e.g., Telfa pad [Kendall]), an intermediate layer of absorbent material, and an outer porous layer (Elastikon, Vetrap) can be placed to prevent wound contamination during healing. The nonadherent pad will not stick to the wound and cause patient discomfort. Because there usually is minimal drainage from the wound, the function of the intermediate layer is more protective than absorptive. Any small amount will be absorbed into the intermediate layer of the bandage. It is important with any bandage to place the tape strips or “stirrups” on the patient’s limb and then overlap in the bandage, to prevent the bandage from slipping. Place the intermediate and tertiary layers loosely around the limb, starting distally and working proximally, with some overlap with each consecutive layer. This method prevents excessive pressure and potential impairment of venous drainage. Leave the toenails of the third and fourth digits exposed, whenever possible, to allow daily examination of the bandage to determine whether the bandage is impairing venous drainage. If the bandage is too tight and constricting or impeding vascular flow, the toes will become swollen and spread apart. When placed and maintained properly (e.g., the bandage does not get wet), there usually are relatively few complications observed with this type of bandage.
**Open Wounds**

**Wounds with Drainage**

In some cases it is necessary to cover a wound in which a Penrose drain has been placed to allow drainage. In many cases there is a considerable amount of drainage from the drain and underlying soft tissues. The function of the bandage is to help obliterate dead space created by the wound itself, absorb the fluid that drains from the wound and that will contaminate the environment, and prevent external wicking of material from the external environment into the wound. When the bandage is removed, the clinician can examine the amount and type of material that has drained from the wound in order to determine when the drain should be removed.

When a bandage is placed over a draining wound, the contact layer should be a commercially available nonadherent dressing and several layers of absorbent wide-mesh gauze placed directly over the drain at the distal end of the incision. Overlay the layers of gauze with a thick layer of absorbent intermediate dressing to absorb fluid that drains from the wound. If the gauze and intermediate layers are not thick or absorbent enough, there is a potential for the drainage fluid to reach the outer layer of the bandage and provide a source of wicking of bacteria from the external environment into the wound, leading to infection.

**Wounds in Need of a Pressure Bandage**

**Minor Hemorrhage**

Some wounds such as lacerations have minor bleeding or hemorrhage that require an immediate bandage until definitive care can be provided. To create a pressure bandage, place a nonadherent dressing immediately in contact with the wound, followed by a thick layer of absorbent material, topped by a layer of elastic bandage material such as Elastikon or Vetrap. Unlike the bandage for a closed wound, the top tertiary outer layer should be wrapped with some tension and even pressure around the limb, starting from the distal extremity (toes) and working proximally. The pressure bandage serves to control hemorrhage but should not be left on for long periods. Pressure bandages that have been left on for too long can impair nerve function and lead to tissue necrosis and slough. Therefore pressure bandages should be used in the hospital only, so that the patient can be observed closely. If hemorrhage through the bandage occurs, place another bandage over the first until the wound can be repaired definitively. Removal of the first bandage will only disrupt any clot that has formed and cause additional hemorrhage to occur.

**Initial Fracture Immobilization**

Fractures require immediate immobilization to prevent additional patient discomfort and further trauma to the soft tissues of the affected limb. As with all bandages, a contact layer, an intermediate layer, and an outer layer should be used. Place the contact layer in accordance with any type of wound present. The intermediate layer should be thick absorbent material, followed by a top layer of elastic bandage material. An example is to place a Telfa pad over a wound in an open distal radius-ulna fracture, followed by a thick layer of cotton gauze cast padding, followed by an elastic layer of Kling (Johnson & Johnson Medical, Arlington, Texas), pulling each layer tightly over the previous layer with some overlap until the resultant bandage can be “thumped” with the clinician’s thumb and forefinger and sound like a ripe watermelon. The bandage should be smooth with consecutive layers of even pressure on the limb, starting distally and working proximally. Leave the toenails of the third and fourth digits exposed to allow monitoring for impaired venous drainage that would suggest that the bandage is too tight and needs to be replaced. Finally, place a top layer of Vetrap or Elastikon over the intermediary layer to protect it from becoming contaminated. If the bandage is used with a compound or open fracture, drainage may be impaired and actually lead to enhanced risk of wound infection. Bandages placed for initial fracture immobilization are temporary until definitive fracture repair can be performed once the patient’s cardiovascular and respiratory status is stable.
Exuberant Granulation Tissue
Wounds with exuberant granulation tissue must be handled carefully so as to not disrupt the healing process but to keep an overabundance of tissue from forming that will impair epithelialization. To bandage a wound with exuberant granulation tissue, place a corticosteroid-containing ointment on the wound, followed by a nonadherent contact layer. The corticosteroid will help control the exuberant growth of granulation tissue. Next, carefully wrap an absorbent material over the contact layer, then carefully place an overlay of elastic bandage material to apply some pressure on the wound. Leave the toenails of the third and fourth digits exposed so that circulation can be monitored several times daily. Bandages that are too tight must be removed immediately to prevent damage to neuronal tissue and impaired vascularization, tissue necrosis, and slough. Because wound drainage may be impaired, there is a risk of infection.

Obliteration of Dead Space
Gapping wounds or those that have undermined the areas between layers of subcutaneous tissue and fascia should be bandaged with a pressure bandage to help obliterate dead space and prevent seroma formation. An example of a wound that may require this type of bandage is the wound resulting from removal of an infiltrative lipoma on the lateral or ventral thorax. Use caution when placing pressure bandages around the thorax or cervical region because bandages placed too tightly may impair adequate ventilation. To place a pressure bandage and obliterate dead space, place a nonadherent contact layer over the wound. Usually a drain is placed in the wound, so place a large amount of wide-mesh gauze at the distal end of the drain to absorb any wound exudate or drainage. Place several layers of absorbent material over the site to further absorb any drainage. Place a layer of elastic cotton such as Kling carefully but firmly over the dead space to cause enough pressure to control drainage. Place at least two fingers between the animal’s thorax and the bandage to ensure that the bandage is not too tight. In many cases the bandage should be placed once the animal has recovered from surgery and is able to stand. If the bandage is placed while the animal is still anesthetized and recumbent, there is a tendency for the bandage to be too tight. Finally, the tertiary layer should be an elastic material such as Elastikon or Vetrap.

Wounds in Need of Pressure Relief
Many wounds require a pressure relief bandage to prevent contact with the external environment. Wounds that may require pressure relief for healing include decubitus ulcers, pressure bandage or cast ulcers, impending ulcer areas (such as the ileum or ischium of recumbent or cachexic patients), and surgical repair sites of ulcerated areas. Pressure relief bandages can be of two basic varieties: modified doughnut bandage and doughnut-shaped bandage.

Modified Doughnut Bandage
A modified doughnut bandage should be placed over bony prominences on the limbs when there are early signs of pressure such as hyperemia, to prevent further injury. To place a modified doughnut bandage, cast padding material, thick wrapping material, and porous adhesive or loose elastic tape are required. Because this type of bandage becomes compressed after two or three bandage changes, it must be replaced frequently.

To place a modified doughnut bandage, follow this procedure (Figure 1-1):
1. Make several layers of cast padding, and fold them over together, making a 3- × 3-inch pad.
2. Fold the pad over on itself, and cut a slit in the center. Form this slit into a hole.
3. Place the hole in the cast padding over the bony prominence.
4. Wrap bandaging material over the pad.
5. Place tapes over the wrapping material, with overlap of the tape strips, to secure it in place.

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6. Alternatively, place several loose stay sutures percutaneously in the skin surrounding the bony prominence, and secure the doughnut in place with umbilical tape woven through the stay sutures and over the doughnut.

**Doughnut-Shaped Bandage**

Like the modified doughnut bandage, a doughnut-shaped bandage is used over bony prominences to help prevent excessive pressure over the area. The bandage commonly is used over bony prominences on the distal limbs, such as the lateral malleolus, when more padding is indicated than is provided with a modified doughnut bandage. To make a doughnut-shaped bandage, use a hand towel or length of stockinet bandage material, tape, cotton gauze, elastic bandage material, or suture with umbilical tape. As the bandage becomes compressed or soiled, change it to prevent further damage to the underlying tissues.

To create a doughnut-shaped bandage, follow this procedure (Figures 1-2 and 1-3):

1. Roll a hand towel tightly and wrap tape around it to create a circle with a hole in the center. Alternatively, take a length of stockinet bandage material and roll it as you would a sock, creating a padded circle with a hole in the center. Make sure that the hole in the center is large enough to fit around the surgical repair site or ulcer.
2. Place the hole in the center over the ulcer or surgical repair site.
3. Secure the roll in place with strips of tape and cotton and then elastic bandage material. Alternatively, place loose loops of suture through the skin adjacent to and around the wound. Secure the doughnut in place with umbilical tape secured through the suture loops and over the bandage. The wound in the center can be observed and treated through the hole in the center, if necessary.
External Pin Splints

An external pin splint is required when fractures or luxations are associated with open wounds. In some cases it may be difficult to bandage under the bars of the pin splint in such a way that the bandage is in contact with the wound. To create padding around the pins, fit foam rubber sponges to lie securely under and around the pins. Place bandages around the external fixator apparatus in layers to decrease contamination of the wound from the external environment and to absorb fluid that drains from the wound (Figures 1-4 to 1-6).

Cup or Clamshell Splints

A cup splint is indicated when bandaging pad wounds to decrease pressure on the footpad and prevent spreading of the footpads when the dog or cat places the paw down. If the toes spread, spreading of the footpad can delay or impair wound healing. The splint functions to place the paw in a more vertical direction so that the patient bears weight on the toe tips and not directly on the pads during the healing process.

Figure 1-2: Doughnut-shaped bandage created from stockinet bandage material over the olecranon.

Figure 1-3: The tarsus.
To create a cup or clamshell splint, follow this procedure (Figures 1-7 to 1-11):

1. Place a nonadherent contact layer directly over the wound.
2. Place stirrups of tape in contact with the skin of the dog, to be placed over the intermediate layer and prevent the bandage from slipping.
3. Place a fairly thick layer of absorbent intermediate bandage material over the contact layer such that the bandage is well padded. Pull the tape stirrups and secure them to the intermediate layer.
4. Place a length of cast material that has been rolled to the appropriate length, such that the cast material is cupped around the patient’s paw, and lies adjacent to the caudal aspect of the limb to the level of the carpus or tarsus. In the case of a clamshell splint, place a layer of cast material on the cranial and caudal aspect of the paw and conform it in place.

Figure 1-4: Foam rubber pads are placed under and around the pins of the external fixator, adjacent to the wound.

Figure 1-5: Cotton cast padding is placed around the external fixator to keep the foam rubber and contact layer securely in place.
Figure 1-6: Vetrap is placed over the intermediate layer to prevent contamination from the external environment.

Figure 1-7: Tape stirrups in place.

Figure 1-8: Layer of absorbent roll cotton.
5. Take the length of cast padding and soak it in warm water after it has been rolled to the appropriate length. Wring out the pad, and secure and conform it to the caudal (or cranial and caudal, in the case of a clamshell splint) aspect of the distal limb and paw.
6. Secure the cast material in place with a layer of elastic cotton gauze (Kling).
7. Secure the bandage in place with a snug layer of Elastikon or Vetrap.

**Lateral or Caudal Splints**

Short or long splints made of cast material can be incorporated into a soft padded bandage to provide extra support of a limb above and below a fracture site. For a caudal or lateral splint to be effective, it must be incorporated for at least one joint above any fracture site to prevent a fulcrum effect and further disruption or damage to underlying soft tissue structures. A short lateral or caudal splint is used for fractures and luxations of the distal metacarpus, metatarsus, carpus, and tarsus.
To place a short lateral or caudal splint, follow this procedure:
1. Secure a contact layer as determined by the presence or absence of any wound in the area.
2. Place tape stirrups on the distal extremity, to be secured later to the intermediate bandage layer and to prevent slipping of the bandage distally.
3. Place layers of roll cotton from the toes to the level of the mid tibia and fibula or mid radius and ulna. Place the layers with even tension, with some overlap of each consecutive layer, moving distally to proximally on the limb.
4. Secure the short caudal or lateral splint and conform it to the distal extremity to the level of the toes and proximally to the level of the mid tibia and fibula or mid radius and ulna.
5. Secure the lateral or caudal splint to the limb with another outer layer of elastic cotton (Kling).
6. Cover the entire bandage, and splint with an outer tertiary layer of Vetrap or Elastikon. Make sure that the toenails of the third and fourth digits remain visible to allow daily evaluation of circulation.

Long lateral or caudal splints are used to immobilize fractures of the tibia or fibula and radius or ulna. The splints are fashioned as directed for short splints but extend proximally to the level of the axilla and inguinal regions to immobilize above the fracture site.

**Spica Splint**

A spica splint is used to immobilize the humerus and elbow and shoulder joints in the case of luxation or fracture. To place a spica splint, follow this procedure:
1. Place a contact layer if any wounds are present.
2. Apply tape stirrups to the distal limb to attach to the intermediate layer and prevent slipping of the bandage distally.
3. Place layers of conforming cotton gauze circumferentially and overlapping, moving up the limb from distal to proximal.
4. Incorporate the leg bandage into a layer of cotton bandage that is secured over the thorax.
5. Secure the cotton in place with a layer of snug elastic cotton material such as Kling. Make sure that the bandage is not so tight that breathing is impaired.
6. Place splint material on the lateral aspect of the limb, extending the material from the level of the toes proximally over the entire limb and extending proximally to over the scapula and dorsal midline.
7. After rolling the splint to an appropriate length and width, moisten the splint material in warm water to allow it to set and harden.
8. Replace the splint and conform it to the bandage over the patient’s body.
9. Secure the splint in place with another layer of cotton Kling.
10. Wrap the entire bandage in place with a layer of tertiary bandage material such as Elastikon or Vetrap.

**Additional Reading**

**BLOOD COMPONENT THERAPY**

**Collection and Administration**
Blood component therapy involves the separation of blood into its cellular and fluid components and infusing the components specific for each patient’s needs. Blood component therapy is the mainstay of initial and ongoing management of hematologic emergencies and can provide support of the critically ill patient until the underlying disease process has been controlled. The separation of blood into red blood cells (RBC), plasma, cryoprecipitate, and platelet-rich products allows for more specific replacement of the animal’s deficit(s), decreases the risks of transfusion reactions, and allows for more efficient use of donor blood. Box 1-4 lists indications for transfusion of RBCs, platelet-rich plasma, fresh frozen or fresh plasma, and cryoprecipitate.

**Blood Types and Antigenicity**
Cell membrane receptors on the surface of RBCs serve the purpose of self-recognition versus non–self-recognition during states of health. The presence or absence of various glycoprotein and glycolipid moieties on the RBC surface helps to define blood groups or “types” within a species. In dogs, six major cell surface dog erythrocyte antigens (DEAs, 1.1, 1.2, 3, 4, 5, and 7) have been identified. Dogs that are negative for DEAs 1.1, 1.2, and 7 but

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**BOX 1-4 APPROACH TO BLOOD COMPONENT THERAPY**

**Red Blood Cell Support**
- Packed cell volume that drops rapidly to less than 20% in the dog and less than 12% to 15% in the cat
- Acute loss of more than 30% of blood volume (30 mL/kg in dog, 20 mL/kg in cat)
- Clinical signs of lethargy, collapse, hypotension, tachycardia, tachypnea (acute or chronic blood loss)
- Ongoing hemorrhage
- Poor response to crystalloid and colloid infusion

**Platelet Support**
- Life-threatening hemorrhage caused by thrombocytopenia or thrombocytopenia
- Surgical intervention necessary in a patient with severe thrombocytopenia or thrombo-cytopenia

**Plasma Support**
- Life-threatening hemorrhage with decreased coagulation factor activity
- Severe inflammation (pancreatitis, systemic inflammatory response syndrome)
- Replenish antithrombin (disseminated intravascular coagulation, protein-losing enteropathy or nephropathy)
- Surgery necessary in a patient with decreased coagulation factor activity
- Severe hypoproteinemia—to partially replenish albumin, globulin, and clotting factors
positive for DEA 1.4 are known as “universal donors” and have DEA 1.1–negative blood. DEAs 1.1 and 1.2 are the most immunogenic RBC antigens known in canine transfusion medicine. Transfusion of DEA 1.1– or 1.2–positive blood to a DEA 1.1– and 1.2–negative dog can result in immediate hemolysis or a delayed-type hypersensitivity reaction. In addition, viability of DEA 1.1– and 1.2–positive cells in a DEA 1.1– and 1.2–negative recipient is short-lived, ultimately defeating the long-term goal of increasing oxygen delivery in the recipient.

As with dogs, feline blood groups are defined by specific carbohydrate moieties attached to lipids (glycolipids) and proteins (glycoproteins) on the RBC surface. Three blood types (A, B, and AB) have been identified in cats. Type A is the most common blood type in cats. Type B is relatively uncommon and occurs in Abyssinian, Persian, Devon Rex, and British Shorthair cats but can be found in domestic shorthair and longhair cats as well. Type A is completely dominant over type B by simple mendelian genetics. Type AB is a rare blood type that has been identified infrequently in domestic shorthair cats; Birman, Abyssinian, Somali, British Shorthair, and Scottish Fold cats; and Norwegian Forest Cats. Unlike dogs, cats possess naturally occurring antibodies against other feline blood types. The presence of naturally occurring autoantibodies is of paramount importance, necessitating blood typing with or without crossmatch before any feline transfusion, because hemolytic transfusion reactions potentially can be fatal, even with no prior sensitization or blood transfusion. Type B cats possess large quantities of anti-A antibodies, primarily of the immunoglobulin M (IgM) subclass. Type A blood infused into a type B cat will be destroyed within minutes to hours, and as little as 1 mL of incompatible blood can cause a life-threatening reaction. Type A cats typically possess weak anti-B antibodies of IgG and IgM subtypes. Transfusion of type B blood into a type A cat will result in milder clinical signs of reaction and a markedly decreased survival half-life of the infused RBCs to just 2 days. Because type AB cats possess both moieties on the cell surface, they lack naturally occurring alloantibodies; transfusion of type A blood into a type AB cat can be performed safely if a type AB donor is not available. The life span of an RBC from a type-specific transfusion into a cat is approximately 33 days.

Blood Donor Programs

Each clinic must weigh the cost-benefit ratio, the need for blood products, and the overall quantity of blood products in the practice when deciding which option works best for the staff, the clientele, and patient needs. Busy hospitals requiring large quantities of blood products at regular intervals may elect to keep an in-house colony of donor dogs and cats. Maintenance of a closed donor colony may be impractical because of the economics of feeding and housing the animals and using cage space that can be used for other patients. In addition, care of the animals—including frequent health examinations, blood testing (complete blood count, biochemistry panels, heartworm tests), and daily care—are labor intensive for veterinarian and support staff alike. Other options include using staff- or client-owned animals as donors. This practice eliminates the expense of housing donors within the clinic and the labor required for daily care. Donor animals can be used as needed or can have scheduled collections to replenish the stock of blood products. The final option, which may be more practical for clinics with an infrequent need for blood products, is to purchase blood components from a commercial blood bank (Table 1-1).

Blood donors should receive annual physical examinations and general health screenings, including a complete blood count, serum biochemistry panel, and occult heartworm antigen test. Canine donors also should be screened initially for Lyme disease, Babesia, Rocky Mountain spotted fever (Rickettsia rickettsii), Ehrlichia, and Brucella. The prevalence of Babesia species in the racing Greyhound industry in Florida, Arizona, and Colorado is high (estimated to be 30% to 50%). Dogs ideally should weigh more than 50 lb (27 kg), be 1 to 8 years of age, have a packed cell volume (PCV) of at least 40%, and have never received a transfusion. A healthy donor safely can donate 10 to 20 mL/kg of whole blood every 3 to 4 weeks if necessary.

Feline blood donors ideally should weigh more than 8 lb, be 1 to 8 years of age, and have never received a transfusion. In addition, donor cats should be screened for feline
leukemia virus (FeLV), feline immunodeficiency virus (FIV), Mycoplasma haemofelis, and feline infectious peritonitis (FIP) before donations and should have a minimally acceptable PCV of 30%, although 35% to 40% is preferred.

**Blood Collection and Handling**

Any blood collection should be performed in a manner that is the least stressful for the donor animal. Physical examination and determination of PCV and total solids should be performed before any donation. Blood can be obtained from a jugular vein or femoral artery. However, because of the risk of lacerating the femoral artery, with subsequent hemorrhage or development of compartmental syndrome, I strongly advocate using the jugular vein as the primary site of blood collection in dogs and cats. Carefully clip the fur over the jugular vein, avoiding skin abrasions. Place dogs in lateral recumbency; however, sternal recumbency or sitting on the floor are also acceptable methods. Aseptically scrub the area over the clipped jugular vein with iodine or chlorhexidine wipes, alternating with alcohol or sterile saline. Blood can be collected from an open or closed system. Closed-system collection is preferred because it decreases the potential for contamination of the blood product and facilitates processing of blood components. Alternatively, an open system can be used if the blood is going to be transfused within 24 hours. Gently insert a 16-gauge needle into the jugular vein. Place the collection system on a scale on the floor and zero the scale. Then remove the hemostat placed on the collection tubing and allow the blood to flow by gravity. Canine units should be approximately 450 mL, which translates to 450 g on the tared scale, because 1 mL weighs approximately 1 g. Although a volume of 450 mL can be obtained every 21 days, if necessary, from a healthy canine donor, less frequent donation of every 3 months is preferred.

Alternatively, feline blood collection often requires the use of sedation, unless a multiaccess port has been implanted surgically. All donor cats should undergo physical examination and determination of PCV and total solids before sedation and subsequent blood donation. Clip the fur over the jugular vein and aseptically prepare the site as described previously. Insert a 19-gauge butterfly catheter into the jugular vein and aspirate blood with gentle pressure to avoid venous collapse. The butterfly catheter is attached to a three-way stopcock and 60-mL syringe into which 7 mL of citrate phosphate dextrose adenine anticoagulant have been placed. In most cases a total volume of 53 mL of blood is

<table>
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<th>Name and Address</th>
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<th>Website</th>
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<tr>
<td>Animal Blood Resources International (West Coast)</td>
<td>800-243-5759 (800-2HELPK9)</td>
<td><a href="http://www.animalbloodbank.com">www.animalbloodbank.com</a></td>
</tr>
<tr>
<td>P.O. Box 1118 Dixon, CA 95620</td>
<td></td>
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<tr>
<td>Eastern Veterinary Blood Bank 844 Ritchie Highway Suite 204 Severna Park, MD 21146</td>
<td>800-949-EVBB (800-949-3822)</td>
<td><a href="http://www.evbb.com">www.evbb.com</a></td>
</tr>
<tr>
<td>Hemopet Blood Bank Office 11330 Markon Drive Garden Grove, CA 92841</td>
<td>714-891-2022</td>
<td><a href="http://www.itsfortheanimals.com/HEMOPET.HTM">www.itsfortheanimals.com/HEMOPET.HTM</a></td>
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<tr>
<td>Animal Blood Resources International (Midwest) 4983 Bird Drive Stockbridge, MI 49285</td>
<td>877-517-6227 (877-517-MABS)</td>
<td><a href="http://www.midwestabs.com">www.midwestabs.com</a></td>
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</tbody>
</table>

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obtained. The blood can be transfused immediately or placed into a small sterile collection bag that contains 0.14 mL of citrate phosphate dextrose adenine anticoagulant per milliliter of whole blood. No more than 11 to 15 mL/kg should be obtained at any given time from a feline donor (Box 1-5).

**Blood Component Processing and Storage**

Production of blood component therapy has become more commonplace in human and veterinary medicine. Component therapy involves the separation of whole blood into its cellular and plasma components and then administration of specific components to a recipient based on each patient’s individual needs. Preparation of fresh frozen plasma, frozen plasma, cryoprecipitate, and cryopoor plasma requires the use of a refrigerated centrifuge. Floor and tabletop models are currently available for purchase. In many cases, purchase of a refrigerated centrifuge is impractical because of the expense and the space required for its storage. A veterinary community potentially can pool resources for the cost of the equipment and house the unit at a centrally located facility, such as a local emergency hospital. Alternatively, human hospitals or blood banks may provide separation services for a nominal fee. Investigation of guidelines in your area may provide a means of creating blood components from your donors for use in your practice.

Once obtained, blood should be stripped from the collection tubing, and the line sealed using a thermal seal or aluminum clips. The bag should be labeled clearly with donor name, donor blood type, date of collection, PCV of donor at time of collection, and date of expiration. If the blood is not going to be used immediately or prepared for platelet-rich plasma, the unit should be refrigerated. The unit then can be spun at 4000 to 5000 times gravity for 5 minutes to separate the RBCs from plasma components. Use of a plasma extractor will facilitate flow of plasma into designated satellite bags for further storage.

Fresh frozen plasma, cryoprecipitate, and cryopoor plasma should be frozen within 8 hours of collection to ensure preservation of labile clotting factors, including factors V and VIII and von Willebrand factor (vWF). Fresh frozen plasma has a shelf life of 1 year past the date of collection. Before freezing, place an elastic band around the bag to crimp the bag during the freezing process. The elastic band is removed once the unit is frozen. In case of an inadvertent or unobserved power failure, the crimp in the unit provides a quality control measure that inadvertent thawing has not occurred. Partial thawing and differential centrifugation of fresh frozen plasma allows preparation of cryoprecipitate and cryopoor plasma. After 1 year, or if a unit of plasma has been prepared after 8 hours of collection, frozen plasma results. Frozen plasma contains all of the vitamin K–dependent coagulation factors (II, VII, IX, X), immunoglobulins, and albumin but is relatively devoid of the labile clotting factors. Frozen plasma has a shelf life of 5 years after the original date of collection or 4 years after expiration of a unit of fresh frozen plasma. Packed RBCs should be stored at 1° to 6° C immediately after collection and processing. Packed RBCs and frozen plasma also can be prepared in the absence of a refrigerated centrifuge by storing the unit of whole blood upright in a refrigerator at 1° to 6° C for 12 to 24 hours until the RBCs have separated out. The plasma can be drawn off into a second storage bag and frozen as frozen plasma. Because of the delay in processing, the resultant plasma does not contain the labile clotting factors.
factors. Fresh frozen plasma, frozen plasma, cryoprecipitate, and cryopoor plasma should be stored at 20° C until use (Table 1-2). The products should be thawed in tepid water until no crystals are observed. No plasma product should be heated to greater than 37° C, because protein denaturation can occur.

**Crossmatch Procedure**

Before administering blood products, find out the donor’s and recipient’s blood types and perform a crossmatch procedure as time allows. At minimum, a blood type should be performed before administration. Rapid blood typing cards are available for use in dogs and cats (RapidVet-H; DMS Laboratories, Flemington, New Jersey), although these cards are problematic and cannot be used in animals with autoagglutination. A simpler and often more accurate method of blood typing has also been developed that can be used in animals with autoagglutination (Plasvacc USA, Templeton, California); I prefer this method.

A crossmatch procedure simulates in vitro the response of a recipient to donor plasma and RBC antigens. The crossmatch procedure is performed to decrease the risk of transfusion reactions in patients that have been sensitized previously, in patients that have naturally occurring alloantibodies, or in situations of neonatal isoerythrolysis. Other indications for crossmatching include decreasing the risk of sensitizing a patient if more than one transfusion is anticipated. Crossmatching can be divided into major and minor categories. The major crossmatch mixes the donor’s RBCs with recipient’s plasma, thus testing whether the recipient has antibodies against donor RBCs. A minor crossmatch mixes donor plasma with recipient RBCs, testing for the unlikely occurrence that the donor serum contains antibodies directed against recipient RBCs. Box 1-6 gives a complete step-by-step description of major and minor crossmatch procedures. The crossmatch procedures do not check for other sources of immediate hypersensitivity transfusion reactions, including WBCs and platelets.

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**Table 1-2: Storage of Blood Components**

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<thead>
<tr>
<th>Component</th>
<th>Anticoagulant</th>
<th>Shelf Life</th>
<th>Comments</th>
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<tbody>
<tr>
<td>WB</td>
<td>Heparin 625 international units per 250 mL WB</td>
<td>37 days, 4° C, 24 hours, 4° C, 21 days, 4° C, 35 days, 4° C</td>
<td>No preservation, coagulation factor inhibition, ACD rarely used Will maintain 75% PTV</td>
</tr>
<tr>
<td>RBCs</td>
<td>CPDA-1</td>
<td>20 days, 4° C, 37 days, 4° C</td>
<td>Will maintain 75% PTV</td>
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<td>Platelet-rich plasma</td>
<td>CPDA-1</td>
<td>3-5 days, 23° C, 2 hours, 4° C</td>
<td>Needs constant agitation</td>
</tr>
<tr>
<td>Fresh frozen plasma</td>
<td>CPDA-1</td>
<td>1 year, −30° C, 3 months, −18° C</td>
<td>Frozen &lt;6 hours after collection of blood; all coagulation factors present</td>
</tr>
<tr>
<td>Plasma, cryopoor plasma</td>
<td>CPDA-1</td>
<td>5 years, −30° C</td>
<td>Does not contain factors V and VIII</td>
</tr>
<tr>
<td>Cryoprecipitate</td>
<td>CPDA-1</td>
<td>1 year, −30° C</td>
<td>High concentration of vWF, factor VIII, and fibrinogen</td>
</tr>
</tbody>
</table>

_ACD_, Acid citrate dextrose; _AS-1_, additive solution; _CPDA-1_, citrate phosphate dextrose adenine; _PTV_, posttransfusion viability; _RBCs_, red blood cells; _vWF_, von Willebrand factor; _WB_, whole blood.
There are many indications for administering transfusions of whole blood and component blood products. Take a stepwise approach for every patient that may require a transfusion. If a patient is at risk for blood loss or is anemic, consider a transfusion. Make a decision regarding the type of transfusion therapy appropriate for each particular patient. Once a decision has been made about which components need to be administered, consider the following:

**Supplies Needed**
- 0.9% Physiologic saline in wash bottle
- 3-mL test tubes
- Pasteur pipettes
- Centrifuge
- Agglutination viewer lamp

**Steps**
1. Label test tubes as follows:

   - RC: Recipient control
   - RR: Recipient RBCs
   - RP: Recipient plasma
   - DB: Donor whole blood*
   - DC: Donor control*
   - DR: Donor whole blood*
   - DP: Donor plasma*
   - Ma: Major crossmatch*
   - Mi: Minor crossmatch*

2. Obtain a crossmatch segment from blood bank refrigerator for each donor to be crossmatched, or use an EDTA tube of donor’s blood. Make sure tubes are labeled properly.
3. Collect 2 mL of blood from recipient and place in an EDTA tube. Centrifuge blood for 5 minutes.
4. Extract blood from donor tubing. Centrifuge blood for 5 minutes. Use a separate pipette for each transfer because cross-contamination can occur.
5. Pipette plasma off of donor and recipient cells and place in tubes labeled DP and RP, respectively.
6. Place 125 mL of donor and recipient cells in tubes labeled DR and RR, respectively.
7. Add 2.5 mL of 0.9% sodium chloride solution from wash bottle to each red blood cell tube, using some force to cause cells to mix.
8. Centrifuge RBC suspension for 2 minutes.
9. Discard supernatant and resuspend RBCs with 0.9% sodium chloride from wash bottle.
10. Repeat steps 8 and 9 for a total of three washes.
11. Place two drops of donor RBC suspension and two drops of recipient plasma in tube labeled Ma (this is the major crossmatch).
12. Place two drops of donor plasma and two drops of recipient RBC suspension in tube labeled Mi (this is the minor crossmatch).
13. Prepare control tubes by placing two drops of donor plasma with two drops of donor RBC suspension (this is the donor control); and place two drops of recipient plasma with two drops of recipient RBC suspension (this is the recipient control).
14. Incubate major and minor crossmatches and control tubes at room temperature for 15 minutes.
15. Centrifuge all tubes for 1 minute.
16. Read tubes using an agglutination viewer.
17. Check for agglutination and/or hemolysis.
18. Score agglutination with the following scoring scale:

   - 4+: One solid clump of cells
   - 3+: Several large clumps of cells
   - 2+: Medium-sized clumps of cells with a clear background
   - 1+: Hemolysis, no clumping of cells
   - NEG: Negative for hemolysis; negative for clumping of red blood cells

*Indicates that this must be done for each donor being tested.

**EDTA**, Ethylenediaminetetraacetic acid; **RBC**, red blood cell.

**Indications for Transfusion Therapy**

There are many indications for administering transfusions of whole blood and component blood products. Take a stepwise approach for every patient that may require a transfusion. If a patient is at risk for blood loss or is anemic, consider a transfusion. Make a decision regarding the type of transfusion therapy appropriate for each particular patient. Once a decision has been made about which components need to be administered, consider the following:

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administered, calculate a volume to be delivered. Exercise caution when administering larger volumes to small patients or those with cardiac insufficiency, because volume overload potentially can occur. If RBC products are to be administered, at minimum a blood typing procedure should be performed before type-specific blood is given. The gold standard is to perform a crossmatch for each unit administered to decrease the risk of a transfusion reaction or sensitization of the patient to foreign RBC antigens. In patients with severe hemorrhage when there is not enough time even for performing a blood typing procedure, universal blood (DEA 1.1–, 1.2–, and 1.7–negative) can be administered.

A common misconception is that administration of whole blood or packed RBCs should occur when patient PCV decreases to a certain number. In fact, no absolute “transfusion trigger” number actually exists. Administer a transfusion whenever a patient demonstrates clinical signs of anemia, including lethargy, anorexia, weakness, tachycardia, and/or tachypnea (Table 1-3). Indications for fresh whole blood transfusion include disorders of hemostasis and coagulopathies including disseminated intravascular coagulation (DIC), von Willebrand disease, and hemophilia. Fresh whole blood and platelet-rich plasma also can be administered in cases of severe thrombocytopenia and thrombocytopenia. Stored whole blood and packed RBCs can be administered in patients with anemia. If PCV drops to below 10% or if rapid hemorrhage causes the PCV to drop below 20% in the dog or below 12% to 15% in the cat, a transfusion is advocated. Consider

<table>
<thead>
<tr>
<th>Blood Products</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fresh whole blood</td>
<td>Coagulopathy with active hemorrhage (DIC, thrombocytopenia; massive acute hemorrhage; no stored blood available)</td>
</tr>
<tr>
<td>Stored whole blood</td>
<td>Massive acute or ongoing hemorrhage; hypovolemic shock caused by hemorrhage that is unresponsive to conventional crystalloid and colloid fluid therapy; unavailability of equipment required to prepare blood components</td>
</tr>
<tr>
<td>Packed red blood cells</td>
<td>Nonregenerative anemia, immune-mediated hemolytic anemia, correction of anemia before surgery, acute or chronic blood loss</td>
</tr>
<tr>
<td>Fresh frozen plasma</td>
<td>Factor depletion associated with active hemorrhage (congenital—von Willebrand factor, hemophilia A, hemophilia B; acquired—vitamin K antagonist, rodenticide intoxication, DIC); acute or chronic hypoproteinemia (burns, wound exudates, body cavity effusion; hepatic, renal, or gastrointestinal loss); colostrum replacement in neonates</td>
</tr>
<tr>
<td>Frozen plasma (contains stable clotting factors)</td>
<td>Acute plasma or protein loss; chronic hypoproteinemia; colostrum replacement in neonates; hemophilia B and selected clotting factor deficiencies</td>
</tr>
<tr>
<td>Platelet-rich plasma*</td>
<td>Thrombocytopenia with active hemorrhage (immune-mediated thrombocytopenia, DIC); platelet function abnormality (congenital—thrombasthenia in Bassett Hounds; acquired—NSAIDs, other drugs)</td>
</tr>
<tr>
<td>Cryoprecipitate (concentration of factor VIII, von Willebrand factor, and fibrinogen)</td>
<td>Congenital factor deficiencies (routine or before surgery): hemophilia A, hemophilia B, von Willebrand disease, hypofibrinogenemia; acquired factor deficiencies</td>
</tr>
</tbody>
</table>

DIC, Disseminated intravascular coagulation; NSAIDS, nonsteroidal antiinflammatory drugs. *Must be purchased because logistically one cannot obtain enough blood simultaneously to provide a significant amount of platelets; platelets infused have a very short (<2 hours) half-life.
fresh frozen plasma or cryoprecipitate administration in cases of coagulopathy, including von Willebrand disease, rodenticide intoxication with depletion of activated vitamin K–dependent coagulation factors, and hemophilia or in cases of severe hypoproteinemia with albumin concentrations less than 2.0 g/dL. Frozen plasma also will suffice in cases of severe hypoproteinemia, warfarin-like compound intoxication, and factor IX deficiency (hemophilia B).

**Considerations for Administration of Blood Component Therapy**

When considering the type of blood component product required for transfusion, one should answer a number of questions to decrease the risk of a transfusion reaction and to decrease the risk for rejection or destruction of the component that has been infused. First, knowledge of a patient's blood type is essential. Whenever possible, type-specific RBCs should be administered. If an animal has received prior transfusion(s), the risk of a transfusion reaction or rejection is increased because of the development of antibodies directed against glycoprotein moieties on the surface of RBCs. If a prior transfusion has taken place, the patient's blood (RBCs and plasma) must be crossmatched with the donor blood (RBCs and plasma) to make sure that no incompatibility exists. In dogs, if neither a blood typing nor a crossmatch procedure is available or if the emergent situation requires that a transfusion be administered before blood typing or crossmatch can be performed, blood from a universal donor (e.g., DEA 1.1–, 1.2–, and 1.7–negative) should be administered whenever possible. Because there is no universal donor in the cat and because cats possess naturally occurring alloantibodies, all cat blood should be typed and crossmatched before any transfusion.

**Transfusion of Blood Component Products**

Table 1-4 indicates blood component dose and administration rates.

**Red Blood Cell Component Therapy**

Blood products should be warmed slowly to 37°C before they are administered to the patient. Blood warmer units are available for use in veterinary medicine to facilitate rapid transfusion without decreasing patient body temperature. RBC and plasma products should be administered in a blood administration set containing a 170-µm in-line filter. Smaller in-line filters (20 µm) also can be used in cases in which extremely small volumes are to be administered. Blood products should be administered over a period of 4 hours, whenever possible, according to guidelines set by the American Association of Blood Banks.

The volume of blood components required to achieve a specific increment in the patient’s PCV depends largely on whether whole blood or packed RBCs are transfused and whether there is ongoing hemorrhage or RBC destruction. Because the PCV of packed RBCs is unusually high (80% for Greyhound blood), a smaller total volume than with whole blood is required to achieve a comparable increase in the patient’s PCV. In general, 10 mL of packed RBCs per kilogram or 20 mL of whole blood per kilogram will raise the recipient's PCV by 10%. The “Rule of Ones” states that 1 mL per 1 lb of whole blood will raise the PCV by 1%. If the patient’s PCV does not raise by the amount anticipated by the foregoing calculation(s), causes of ongoing hemorrhage or destruction should be considered. The goal of red blood component therapy is to raise the PCV to 25% to 30% in dogs and 15% to 20% in cats.

If an animal is hypovolemic and whole blood is administered, the fluid is redistributed into the extravascular compartment within 24 hours of transfusion. This will result in a secondary rise in the PCV 24 hours after the transfusion in addition to the initial rise 1 to 2 hours after the RBC transfusion is complete.

**Use of Fresh Frozen Plasma**

The volume of plasma transfused depends largely on the patient’s need. In general, plasma transfusion should not exceed 22 mL/kg during a 24-hour period for normovolemic animals. Thaw plasma at room temperature, or place it in a Ziploc freezer bag and run
under cool (not warm) water until thawed. Then administer the plasma through a blood administration set that contains an in-line blood filter or through a standard drip-type administration set with a detachable in-line blood administration filter. The average rate of plasma infusion in a normovolemic patient should not exceed 22 mL/kg/hr. In acute need situations, plasma can be delivered at rates up to 5 to 6 mL/kg/min. For patients with cardiac insufficiency or other circulatory problems, plasma infusion rates should not exceed 5 mL/kg/hr. Plasma or other blood products should not be mixed with or used in the same infusion line as calcium-containing fluids, including lactated Ringer’s solution, calcium chloride, or calcium gluconate. The safest fluid to mix with any blood product is 0.9% sodium chloride.

Administer fresh frozen plasma, frozen plasma, and cryoprecipitate at a volume of 10 mL/kg until bleeding is controlled or the source of ongoing albumin loss is eliminated. The goal of plasma transfusion therapy is to raise the albumin to a minimum of 2.0 g/dL or until bleeding stops as in the case of coagulopathies. Monitor the patient to ensure that bleeding has stopped, coagulation profiles (ACT, APTT, and PT) have normalized, hypovolemia has stabilized, and/or total protein is normalizing, which are indications for discontinuing ongoing transfusion therapy.
Use of Cryoprecipitate
Plasma cryoprecipitate can be purchased or manufactured through the partial thawing and then centrifugation of fresh frozen plasma. Cryoprecipitate contains concentrated quantities of vWF, factor VIII, and fibrinogen and is indicated in severe forms of von Willebrand disease and hemophilia A (factor VIII deficiency).

Platelet-Rich Plasma
Platelet-rich plasma must be purchased from a commercial source. One unit of fresh whole blood contains 2000 to 5000 platelets. The viability of the platelets contained in the fresh whole blood is short-lived, just 1 to 2 hours after transfusion into the recipient. Because platelet-rich plasma is difficult to obtain, animals with severe thrombocytopenia or thrombocytopenia should be treated with immunomodulating therapies and the administration of fresh frozen plasma.

Transfusion in Dogs
In dogs, blood and plasma transfusions can be administered intravenously (IV) or intraosseously. The cephalic, lateral saphenous, medial saphenous, and jugular veins are used most commonly. Fill the recipient set so that the blood in the drip chamber covers the filter (normal 170-μm filter). With small amounts of blood (50 mL) or critically ill patients, use a 40-μm filter. Avoid latex filters for plasma and cryoprecipitate administration. Blood can be administered at variable rates, but the routine figure of 4 to 5 mL/min often is used. Normovolemic animals can receive blood at 22 mL/kg/day. Dogs in heart failure should receive infusions at no more than 4 mL/kg/hr. Volume is given as needed. To calculate the approximate volume of blood needed to raise hematocrit levels, use the following formula for the dog:

\[
\text{Anticoagulated blood volume (mL)} = \text{Body mass (kg)} \times 90 \times \frac{\text{PCV desired} - \text{PCV of recipient}}{\text{PCV of donor in anticoagulant}}
\]

An alternative formula is the following:

\[
2.2 \times \text{Recipient body mass (kg)} \times 30 \times \frac{\text{PCV desired} - \text{PCV of recipient}}{\text{PCV of donor in anticoagulant}}
\]

Surgical emergencies and shock may require several times this volume within a short period. If more than 25% of the patient’s blood volume is lost, supplementation with colloids, crystalloids, and blood products is indicated for fluid replacement. One volume of whole blood achieves the same increase in plasma as two to three volumes of plasma. If the patient’s blood type is unknown and DEA 1.1–negative whole blood is not available, any dog blood can be administered to a dog in acute need if the dog has never had a transfusion before. If mismatched blood is given, the patient will become sensitized, and after 5 days, destruction of the donor RBCs will begin. In addition, any subsequent mismatched transfusions may cause an immediate reaction (usually mild) and rapid destruction of the transfused RBCs.

The clinical signs of a transfusion reaction typically are seen only when DEA 1.1 blood is administered to a DEA 1.1–negative recipient that was sensitized previously. Incompatible blood transfusions to breeding females can result in isoimmunization and in hemolytic disease in the puppies. The DEA 1.1–negative bitch that receives a transfusion with DEA 1.1–positive blood and that produces a litter from a DEA 1.1–positive stud can potentially have puppies with neonatal isoerythrolysis.

Transfusion in Cats
Cats with severe acute anemia in need of a blood transfusion are typically extremely depressed, lethargic, and anorexic. The stress of restraint and handling can push these critically ill patients over the edge and cause them to die. Extreme gentleness and care are mandatory in restraint.
and handling. The critically ill cat should be cradled in a towel or blanket. Supplemental flow- 
by or mask oxygen should be administered, whenever possible, although it may not be clini-
cally helpful until oxygen-carrying capacity is replenished with infusion of RBCs.

Blood can be administered by way of the cephalic, medial saphenous, or jugular vein. In 
tramedullary infusion is also possible, if vascular access cannot be accomplished. The 
average 2- to 4-kg cat can accept 40 to 60 mL of whole blood injected IV over a period of 30 
to 60 minutes. Administer filtered blood at a rate of 5 to 10 mL/kg/hr. The following formula 
can be used to estimate the volume of blood required for transfusion in a cat:

\[
\text{Anticoagulated blood volume (mL)} = \text{Body mass (kg)} \times 70 \times \frac{\text{PCV desired} - \text{PCV of recipient}}{\text{PCV of donor in anticoagulant}}
\]

**Transfusion Reactions**

The exact overall incidence and clinical significance of transfusion reactions in veterinary 
medicine are unknown. Several studies have been performed that document the incidence 
of transfusion reactions in dogs and cats. Overall, the incidence of transfusion reactions in 
dogs and cats is 2.5% and 2%, respectively. Transfusion reactions can be immune-mediated 
and non–immune-mediated and can happen immediately or can be delayed until after a 
transfusion. Acute reactions usually occur within minutes to hours of the onset of the 
transfusion but may occur up to 48 hours after the transfusion has been stopped. Acute 
immunologic reactions include hemolysis and acute hypersensitivity including RBCs, 
platelets, and leukocytes. Signs of a delayed immunologic reaction include hemolysis, pur-
pura, immunosuppression, and neonatal isoerythrolysis. Acute nonimmunologic reactions 
include donor cell hemolysis before onset of transfusion, circulatory volume overload, 
bacterial contamination, citrate toxicity with clinical signs of hypocalcemia, coagulopathies, 
hyperammonemia, hypothermia, air embolism, acidosis, and pulmonary microembolism. 
Delayed nonimmunologic reactions include the transmission and development of infectious 
diseases and hemosiderosis. Clinical signs of a transfusion reaction typically depend on the 
amount of blood transfused, the type and amount of antibody involved in the reaction, and 
whether the recipient has had previous sensitization.

Monitoring the patient carefully during the transfusion period is essential for recogniz-
ing early signs of a transfusion reaction, including those that may become life-threatening. 
A general guideline for patient monitoring is first to start the transfusion slowly during the 
first 15 minutes. Monitor temperature, pulse, and respiration every 15 minutes for the first 
hour, 1 hour after the end of the transfusion, and every 12 hours minimally thereafter. Also 
obtain a PCV immediately before the transfusion, 1 hour after the transfusion has been 
stopped, and every 12 hours thereafter. Monitor coagulation parameters such as ACT and 
platelet count at least daily in patients requiring transfusion therapy.

The most common documented clinical signs of a transfusion reaction include pyrexia, 
urticaria, salivation or ptyalism, nausea, chills, and vomiting. Other clinical signs of a trans-
fusion reaction may include tachycardia, tremors, collapse, dyspnea, weakness, hypotension, 
collapse, and seizures. Severe intravascular hemolytic reactions may occur within minutes 
of the start of the transfusion, causing hemoglobinemia, hemoglobinuria, DIC, and clinical 
signs of shock. Extravascular hemolytic reactions typically occur later and will result in 
hyperbilirubinemia and bilirubinuria.

Pretreatment of patients to help decrease the risk of a transfusion reaction remains 
controversial, and in most cases pretreatment with glucocorticoids and antihistamines is 
ineffective at preventing intravascular hemolysis and other reactions. The most important 
component of preventing a transfusion reaction is to screen each recipient carefully and pro-
cess the donor component therapy carefully before the administration of any blood products. 
Treatment of a transfusion reaction depends on its severity. In all cases, stop the transfusion 
immediately when clinical signs of a reaction occur. In most cases discontinuation of the 
transfusion and administration of drugs to stop the hypersensitivity reaction will be suffi-
cient. Once the medications have taken effect, restart the transfusion slowly and monitor the patient carefully for further signs of reaction. In more severe cases in which a patient’s cardiovascular or respiratory system becomes compromised and hypotension, tachycardia, or tachypnea occurs, immediately discontinue the transfusion and administer diphenhydramine (1 mg/kg intramuscularly [IM]), dexamethasone-sodium phosphate (0.25 to 0.5 mg/kg IV), and epinephrine to the patient. The patient should have a urinary catheter and central venous catheter placed for measurement of urine output and central venous pressure (CVP). Aggressive fluid therapy may be necessary to avoid renal insufficiency or renal damage associated with severe intravascular hemolysis. Overhydration with subsequent pulmonary edema generally can be managed with supplemental oxygen administration and intravenous or intramuscular administration of furosemide (2 to 4 mg/kg). Plasma products with or without heparin can be administered for DIC.

**Additional Reading**


**CENTRAL VENOUS PRESSURE MEASUREMENT**

*Central venous pressure* refers to the hydrostatic pressure in the anterior vena cava and is influenced by vascular fluid volume, vascular tone, function of the right side of the heart, and changes in intrathoracic pressure during the respiratory cycle. The CVP is not a true measure of blood volume but is used to gauge fluid therapy as a method of determining how effectively the heart can pump the fluid that is being delivered to it. Thus the CVP reflects the interaction of the vascular fluid volume, vascular tone, and cardiac function. Measure CVP in any patient with acute circulatory failure, large-volume fluid diuresis (e.g., because of a toxin or oliguric or anuric renal failure), fluid in-and-out monitoring, and cardiac dysfunction. The placement of central venous catheters and thus CVP measurements is contraindicated in patients with known coagulopathies including hypercoagulable states.

To perform CVP monitoring, place a central venous catheter in the right or left jugular vein. In cats and small dogs, however, a long catheter placed in the lateral or medial saphenous vein can be used for trends in CVP monitoring. First, assemble the equipment necessary for jugular catheter (see Vascular Access Techniques for how to place a jugular or saphenous long catheter) and CVP monitoring (*Box 1-7*). After placing the jugular catheter, take a lateral thoracic radiograph to ensure that the tip of the catheter sits just outside the right atrium for proper CVP measurements (see Figure 1-13 on p. 57).

To establish an intravenous catheter for CVP, follow this procedure:

1. Assemble the CVP setup such that the male end of a length of sterile intravenous catheter extension tubing is inserted into the T port of the jugular or medial or lateral saphenous catheter. Make sure to flush the length of tubing with sterile saline before connecting it to the patient to avoid iatrogenic air embolism.
2. Next, insert the male end of a three-way stopcock into the female end of the extension tubing.
3. Attach a 20-mL syringe filled with heparinized sterile 0.9% saline to one of the female ports of the three-way stopcock and either a manometer or a second length of intravenous extension tubing attached to a metric ruler.
4. Lay the patient in lateral or sternal recumbency.
5. Turn the stopcock OFF to the manometer or ruler and ON to the patient. Infuse a small amount of heparinized saline through the catheter to flush the catheter.
6. Next, turn the stopcock OFF to the patient and ON to the manometer. Gently flush the manometer or length of extension tubing with heparinized saline from the syringe. Use care not to agitate the fluid and create air bubbles within the line or manometer, which will artifactually change the CVP measured.
7. Next, lower the 0 cm point on the manometer or ruler to the level of the patient’s manubrium (if the patient is in lateral recumbency) or the point of the elbow (if the patient is in sternal recumbency).
8. Turn the stopcock OFF to the syringe, and allow the fluid column to equilibrate with the patient’s intravascular volume. Once the fluid column stops falling and the level rises and falls with the patient’s heartbeat, measure the number adjacent to the bottom of the meniscus of the fluid column. This is the CVP in centimeters of water (see Figure 1-4).
9. Repeat the measurement several times with the patient in the same position to make sure that none of the values has been increased or decreased artifactually in error. Alternately, attach the central catheter to a pressure transducer and perform electronic monitoring of CVP.

There is no absolute value for normal CVP. The normal CVP for small animal patients is 0 to 5 cm H₂O. Values less than 0 cm H₂O are associated with absolute or relative hypovolemia. Values of 5 to 10 cm H₂O represent borderline hypervolemia, and values greater than 10 cm H₂O suggest intravascular volume overload. Values greater than 15 cm H₂O may be correlated with congestive heart failure (CHF) and the development of pulmonary edema. In individual patients, the trend in change in CVP is more important than absolute values. As a rule of thumb, when using CVP measurements to gauge fluid therapy and avoid vascular and pulmonary overload, the CVP should not increase by more than 5 cm H₂O in any 24-hour period. If an abrupt increase in CVP is found, repeat the measurement to make sure that the elevated value was not obtained in error. If the value truly has increased dramatically, temporarily discontinue fluid therapy and consider administration of a diuretic.

Additional Reading
FLUID THERAPY

The diagnosis of intracellular fluid deficit is difficult and is based more on the presence of hypernatremia or hyperosmolality than on clinical signs. An intracellular fluid deficit is expected when free water loss from insensible losses and vomiting, diarrhea, or urine is not matched by free water intake. Consideration of the location of the patient’s fluid deficit, the degree and type of acid-base and electrolyte disorders, and the presence of any ongoing fluid losses should dictate and help guide each patient’s individualized fluid therapy plan (Table 1-5).

ACID-BASE PHYSIOLOGY

Normal pH in dogs and cats ranges from 7.30 to 7.45. The three major mechanisms that maintain blood pH within a normal physiologic range include buffering systems, respiratory mechanisms that alter carbon dioxide, and renal (metabolic) mechanisms that retain or excrete hydrogen and bicarbonate ions. The metabolic contribution to acid-base balance can be estimated by measuring total carbon dioxide and pH or by calculating the bicarbonate or base deficit or excess values. Hydrogen and bicarbonate ions have an important influence on normal structure and function of cellular proteins. Treat acidemia if the bicarbonate is less than 12 mEq/L, if the pH is less than 7.2, or if the base deficit is less than −10 mEq/L. Normal bicarbonate concentration is 18 to 26 mEq/L in dogs and 17 to 23 mEq/L in cats (Boxes 1-8 and 1-9).

The respiratory system further contributes to acid-base status by changes in the elimination of carbon dioxide. Hyperventilation decreases the blood P\textsubscript{a}\textsubscript{CO\textsubscript{2}} and causes respiratory alkalosis. Hypoventilation increases the blood P\textsubscript{a}\textsubscript{CO\textsubscript{2}} and causes respiratory acidosis. Depending on the altitude, the P\textsubscript{a}\textsubscript{CO\textsubscript{2}} in dogs can range from 32 to 44 mm Hg. In cats, the normal value is 28 to 32 mm Hg. Venous P\textsubscript{a}\textsubscript{CO\textsubscript{2}} values are 33 to 50 mm Hg in dogs and 33 to 45 mm Hg in cats.

Use a systematic approach whenever attempting to interpret a patient’s acid-base status. Ideally, obtain an arterial blood sample so that you can monitor the patient’s oxygenation and ventilation. Once an arterial blood sample has been obtained, follow these steps:

1. Determine whether the blood sample is arterial or venous by looking at the oxygen saturation (Sa\textsubscript{O\textsubscript{2}}). The Sa\textsubscript{O\textsubscript{2}} should be greater than 90% if the sample is truly arterial, although it can be as low as 80% if a patient has severe hypoxemia.
2. Consider the patient’s pH. If the pH is outside of the normal range, an acid-base disturbance is present. If the pH is within the normal range, an acid-base disturbance may or may not be present. If the pH is low, the patient is acidotic. If the pH is high, the patient is alkalotic.

<table>
<thead>
<tr>
<th>TABLE 1-5 Correlation of Clinical Signs with Estimated Percent Dehydration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical Signs of Interstitial Dehydration</td>
</tr>
<tr>
<td>History of vomiting and diarrhea, no visible clinical signs of deficit</td>
</tr>
<tr>
<td>Dry mucous membranes, mild skin tenting</td>
</tr>
<tr>
<td>Increased skin tenting, dry mucous membranes, mild tachycardia, normal pulse*</td>
</tr>
<tr>
<td>Increased skin tenting, dry mucous membranes, tachycardia, weak pulse pressure</td>
</tr>
<tr>
<td>Increased skin tenting, dry corneas, dry mucous membranes, elevated or decreased heart rate, poor pulse quality, altered level of consciousness*</td>
</tr>
</tbody>
</table>

*Note: These measures are largely subjective because patients with severe weight loss and loss of subcutaneous fat and very young and very old animals may have increased skin tenting or sunken eyes even in the absence of dehydration.
3. Next, look at the base excess or deficit. If the base excess is increased, the patient has higher than normal bicarbonate. If there is a base deficit, the patient may have a low bicarbonate or increase in unmeasured anions (e.g., lactic acid or ketoacids).

4. Next, look at the bicarbonate. If the pH is low and the bicarbonate level is low, the patient has a metabolic acidosis. If the pH is high and the bicarbonate level is elevated, the patient has a metabolic alkalosis.

Modified from DiBartola SP: Fluid, electrolyte, and acid-base disorders in small animal practice, St Louis, 2005, Elsevier.
5. Next, look at the PaCO₂. If the patient’s pH is low and the PaCO₂ is elevated, the patient has respiratory acidosis. If the patient’s pH is high and the PaCO₂ is low, the patient has respiratory alkalosis.

6. Finally, if you are interested in the patient’s oxygenation, look at the Pao₂. Normal Pao₂ is greater than 80 mm Hg.

7. Next, you must determine whether the disorders present are primary disorders or an expected compensation for disorders in the opposing system. For example, is the patient retaining bicarbonate (metabolic alkalosis) because of carbon dioxide retention (respiratory acidosis)? Use the chart in Table 1-6 to evaluate whether the appropriate degree of compensation is occurring. If the adaptive response falls within the expected range, a simple acid-base disorder is present. If the response falls outside the expected range, a mixed acid-base disorder is likely present.

8. Finally, you must determine whether the patient’s acid-base disturbance is compatible with the history and physical examination findings. If the acid-base disturbance does not fit with the patient’s history and physical examination abnormalities, question the results of the blood gas analyses and possibly repeat them.

The most desirable method of assessing the acid-base status of an animal is with a blood gas analyzer. Arterial samples are preferred over venous samples, with heparin used as an anticoagulant (Table 1-7).

| Table 1-6 Compensatory Renal and Respiratory Responses in Patients with Primary Acid-Base Disorders |
|---|---|---|
| Disorder | Primary Change | Compensatory Response |
| Metabolic acidosis | ↓ HCO₃⁻ | 0.7 mm Hg decrement in Pco₂ for each 1 mEq/L decrement in HCO₃⁻ |
| Metabolic alkalosis | ↑ HCO₃⁻ | 0.7 mm Hg increment in Pco₂ for each 1 mEq/L increment in HCO₃⁻ |
| Acute respiratory acidosis | ↑ Pco₂ | 1.5 mEq/L increment in HCO₃⁻ for each 10 mm Hg increment in Pco₂ |
| Chronic respiratory acidosis | ↑ Pco₂ | 3.5 mEq/L increment in HCO₃⁻ for each 10 mm Hg increment in Pco₂ |
| Acute respiratory alkalosis | ↓ Pco₂ | 2.5 mEq/L decrement in HCO₃⁻ for each 10 mm Hg decrement in Pco₂ |
| Chronic respiratory alkalosis | ↓ Pco₂ | 5.5 mEq/L decrement in HCO₃⁻ for each 10 mm Hg decrement in Pco₂ |


| Table 1-7 Acid-Base Values in Acute Uncompensated Disturbances |
|---|---|---|
| Disturbance | pH | PaCO₂ | Sodium bicarbonate |
| Metabolic acidosis | ↓ | — | ↓ |
| Metabolic alkalosis | ↑ | — | ↑ |
| Respiratory acidosis | ↓ | ↓ | — |
| Respiratory alkalosis | ↑ | ↓ | — |
**Electrolyte Maintenance and Abnormalities**

**Potassium**

Potassium primarily is located in the intracellular fluid compartment. Serum potassium is regulated by the actions of the sodium-potassium–adenosine triphosphatase pump on cellular membranes, including those of the renal tubular epithelium. Inorganic metabolic acidosis artifactually can raise serum potassium levels because of redistribution of extracellular potassium in exchange for intracellular hydrogen ion movement in an attempt to correct serum pH.

Potassium is one of the major players in the maintenance of resting membrane potentials of excitable tissue, including neurons and cardiac myocytes. Changes in serum potassium can affect cardiac conduction adversely. Hyperkalemia lowers the resting membrane potential and makes cardiac cells, particularly those of the atria, more susceptible to depolarization. Characteristic signs of severe hyperkalemia that can be observed on an ECG rhythm strip include an absence of P waves, widened QRS complexes, and tall tented or spiked T waves. Further increases in serum potassium can be associated with bradycardia, ventricular fibrillation, and cardiac asystole (death). Treatment of hyperkalemia consists of administration of insulin (0.25 to 0.5 units/kg, intravenous regular insulin) and dextrose (1 g dextrose per unit of insulin administered, followed by 2.5% dextrose IV via constant rate infusion (CRI) to prevent hypoglycemia), calcium (2 to 10 mL of 10% calcium gluconate administered IV slowly to effect), or sodium bicarbonate (1 mEq/kg IV slowly). Insulin plus dextrose and bicarbonate therapy help drive the potassium intracellularly, whereas calcium antagonizes the effect of hyperkalemia on the myocardial cells. All of the treatments work within minutes, although the effects are relatively short-lived (20 minutes to 1 hour) unless the cause of the hyperkalemia is identified and treated appropriately (Box 1-10). Dilution of serum potassium also results from restoring intravascular fluid volume and correcting metabolic acidosis, in most cases. Treatment with a fluid that does not contain potassium (preferably 0.9% sodium chloride) is recommended.

Hypokalemia elevates the resting membrane potential and results in cellular hyperpolarization. Hypokalemia may be associated with ventricular dysrhythmias, but the ECG changes are not as characteristic as those observed with hyperkalemia. Causes of hypokalemia include renal losses, anorexia, gastrointestinal loss (vomiting, diarrhea), intravenous fluid diuresis, loop diuretics, and postobstructive diuresis (Box 1-11). If the serum potassium concentration is known, potassium supplementation in the form of potassium chloride or potassium phosphate can be added to the patient’s intravenous fluids. Correct serum potassium levels less than 3.0 mEq/L or greater than 6.0 mEq/L. Potassium rates should not exceed 0.5 mEq/kg/hr (Table 1-8).

**Bicarbonate Concentration**

Metabolic acidosis from bicarbonate depletion often corrects itself with volume restoration in most small animal patients. Patients with moderate to severe metabolic acidosis may benefit from bicarbonate supplementation therapy. The metabolic contribution to acid-base balance is identified by measuring the total carbon dioxide concentration or calculating the bicarbonate concentration. If these measurements are not available, the degree of expected metabolic acidosis can be estimated subjectively by the severity of underlying disease that often contributes to metabolic acidosis: hypovolemic or traumatic shock, septic shock, diabetic ketoacidosis (DKA), or oliguric or anuric renal failure. If the metabolic acidosis is estimated to be mild, moderate, or severe, add sodium bicarbonate at 1, 3, and 5 mEq/kg body mass, respectively. Bicarbonate concentration also can be artifactually decreased in patients with severe DKA. Patients with DKA may not require bicarbonate administration once volume replacement has been accomplished and perfusion has been restored, and the ketoadids are metabolized to bicarbonate. If the bicarbonate measurement of base deficit is known, the following formula can be used as a gauge for bicarbonate supplementation:

\[
\text{Base deficit} \times 0.3 = \text{Body mass (kg)} = \text{mEq Bicarbonate to administer}
\]
Osmolality is measured by freezing point depression or a vapor pressure osmometer, or it may be calculated by the following formula:

\[
\text{mOsm/ kg} = 2[(\text{Na}^+) + (\text{K}^+)] + \text{BUN} / 2.8 + \text{Glucose} / 18
\]

where sodium and potassium are measured in milliequivalents, and BUN and glucose are measured in milligrams per deciliter. Osmalalities less than 260 mOsm/kg or greater than 360 mOsm/kg are serious enough to warrant therapy. The difference between the measured...
**DIFFERENTIAL DIAGNOSES FOR HYPOKALEMIA**

**DECREASED INTAKE**
Alone unlikely to cause hypokalemia unless diet is aberrant
Administration of potassium-free fluids (e.g., 0.9% sodium chloride or 5% dextrose in water) or potassium-deficient fluids (e.g., lactated Ringer’s solution over several days)
Bentonite clay ingestion (e.g., cat litter)

**TRANSLOCATION (EXTRACELLULAR FLUID TRANSFER TO INTRACELLULAR FLUID)**
Alkalemia
Insulin- or glucose-containing fluids
Catecholamines
Hypothermia
Hypokalemic periodic paralysis (Burmese cats)
Albuterol overdosage

**INCREASED LOSS**
Gastrointestinal (FEK less than 4% to 6%)
Vomiting of stomach contents
Diarrhea
Urinary (FEK greater than 4% to 6%)
Chronic renal failure in cats
Diet-induced hypokalemic nephropathy in cats
Distal (type I) renal tubular acidosis
Proximal (type II) renal tubular acidosis after sodium bicarbonate treatment
Postobstructive diuresis
Dialysis
Mineralocorticoid excess
Hyperadrenocorticism
Primary hyperaldosteronism (adenoma, adenocarcinoma, hyperplasia)

**DRUGS**
Loop diuretics (e.g., furosemide and ethacrynic acid)
Thiazide diuretics (e.g., chlorothiazide and hydrochlorothiazide)
Amphotericin B
Penicillins
Unknown mechanism
Rattlesnake envenomation

FEK, Fractional excretion of potassium.

** Guidelines for Routine Intravenous Potassium Supplementation in Dogs and Cats**

<table>
<thead>
<tr>
<th>Serum Potassium Chloride to Add</th>
<th>250 mL of Fluid (mEq)</th>
<th>Potassium Chloride to Add</th>
<th>1 L of Fluid (mEq)</th>
<th>Maximal Potassium Fluid Rate (mEq/L)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;2.0†</td>
<td>20</td>
<td>80</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>2.1-2.5</td>
<td>15</td>
<td>60</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>2.6-3.0</td>
<td>10</td>
<td>40</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>3.1-3.5</td>
<td>7</td>
<td>28</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td>3.6-5.0</td>
<td>5</td>
<td>20</td>
<td>25</td>
<td></td>
</tr>
</tbody>
</table>

*Maximal rate of potassium supplementation should not exceed 0.5 mEq/kg/hr.
†If refractory hypokalemia is present, supplement magnesium at 0.75 mEq/kg/day for 24 hours.
osmolality and the calculated osmolality (the osmolal gap) should be less than 10 mOsm/kg. If the osmolar gap is greater than 20 mOsm/kg, consider the presence of unmeasured anions such as ethylene glycol metabolites.

**Sodium**

The volume of extracellular fluid is determined by the total body sodium content, whereas the osmolality and sodium concentration are determined by water balance. Serum sodium concentration is an indication of the amount of sodium relative to water in the extracellular fluid and provides no direct information about the total body sodium content. Patients with hyponatremia or hypernatremia may have decreased, normal, or increased total body sodium content (Boxes 1-12 and 1-13). An increased serum sodium concentration implies hyperosmolality, whereas a decrease in serum sodium concentration usually, but not always, implies hypoosmolality. The severity of clinical signs of hypernatremia and hyponatremia is related primarily to the rapidity of the onset of the change rather than to the magnitude of the associated plasma hyperosmolality or hypoosmolality. Clinical signs of neurologic disturbances include disorientation, ataxia, and seizures, and coma may occur at serum sodium concentrations less than 120 mEq/L or greater than 170 mEq/L in dogs.

Therapy for hypernatremia or hyponatremia with fluid containing low or higher concentrations of sodium should proceed with caution, because rapid changes (decreases or increases) in serum sodium and osmolality can cause rapid changes in the intracellular and extracellular fluid flux, leading to intracellular dehydration or edema, even though the serum sodium has not been returned to normal. A rule of thumb is to not raise or lower the serum sodium by more than 15 mEq/L during any one 24-hour period. Restoration of the serum sodium concentration over a period of 48 to 72 hours is better. In almost all circumstances, an animal will correct its sodium balance with simple fluid restoration. If severe hypernatremia exists that suggests a free water deficit, however, the free water deficit should be calculated from the following formula:

$$\text{Free water deficit} = 0.4 \times \text{Body mass (kg)} \times \left[ \frac{(\text{Plasma sodium} / 140) - 1}{1} \right]$$

**BOX 1-12  DIFFERENTIAL DIAGNOSES FOR HYPO Natremia**

<table>
<thead>
<tr>
<th>WITH NORMAL PLASMA OSMOLALITY</th>
<th>Antidiuretic drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperlipemia</td>
<td>Myxedema coma of hypothyroidism</td>
</tr>
<tr>
<td>Hyperproteinaemia</td>
<td>Hypotonic fluid infusion</td>
</tr>
<tr>
<td>WITH HIGH PLASMA OSMOLALITY</td>
<td>And Hypovolemia</td>
</tr>
<tr>
<td>Hyperglycemia</td>
<td>Gastrointestinal loss</td>
</tr>
<tr>
<td>Mannitol infusion</td>
<td>Vomiting</td>
</tr>
<tr>
<td>WITH LOW PLASMA OSMOLALITY</td>
<td>Diarrhea</td>
</tr>
<tr>
<td>And Hypervolemia</td>
<td>Third-space loss</td>
</tr>
<tr>
<td>Severe liver disease</td>
<td>Pancreatitis</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>Peritonitis</td>
</tr>
<tr>
<td>Nephrotic syndrome</td>
<td>Uroabdomen</td>
</tr>
<tr>
<td>Advanced renal failure</td>
<td>Pleural effusion (e.g., chylothorax)</td>
</tr>
<tr>
<td>And Normovolemia</td>
<td>Peritoneal effusion</td>
</tr>
<tr>
<td>Psychogenic polydipsia</td>
<td>Cutaneous loss</td>
</tr>
<tr>
<td>Syndrome of inappropriate antidiuretic hormone secretion</td>
<td>Burns</td>
</tr>
<tr>
<td></td>
<td>Hypoadrenocorticism</td>
</tr>
<tr>
<td></td>
<td>Diuretic administration</td>
</tr>
</tbody>
</table>

Hypernatremia can be corrected slowly with 0.45% sodium chloride plus 2.5% dextrose, 5% dextrose in water, or lactated Ringer’s solution (sodium content 130 mEq/L). Correct hyponatremia initially with 0.9% sodium chloride.

**Anion Gap**

Sodium is balanced predominantly by chloride and bicarbonate. The difference between these concentrations, \( \text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-) \) has been called the anion gap. The normal anion gap is 12 to 25 mEq/L. When the anion gap exceeds 25, consider the possibility of an accumulation of unmeasured anions (e.g., lactate, ketoacids, phosphate, sulfate, ethylene glycol metabolites, and salicylate). Abnormalities in the anion gap may be helpful in determining the cause of metabolic acidosis (Boxes 1-14 and 1-15).

**Oncotic Pressure**

The colloid oncotic pressure of blood is associated primarily with large–molecular-weight colloidal substances in circulation. The major player in maintaining intravascular and interstitial oncotic pressure, the water-retaining property of each fluid compartment, is albumin. Albumin contributes roughly 80% to the colloid oncotic pressure of blood. The majority of albumin is located within the interstitial space. Hypoalbuminemia can result from increased loss in the form of protein-losing enteropathy or nephropathy and wound exudates, or it may result from lack of hepatic albumin synthesis. Serum albumin pools are in a constant flux with interstitial albumin. Once interstitial albumin pools become depleted...
from replenishing serum albumin, serum albumin levels can continue to decrease, which can lead to a decrease in colloidal oncotic pressure. Serum albumin less than 2.0 g/dL has been associated with inadequate intravascular fluid retention and the development of peripheral edema and third spacing of fluid. Oncotic pressure can be restored with the use of artificial or synthetic colloids or natural colloids (see Colloids).

**Maintenance Fluid Requirements**

Maintenance fluid requirements have been extrapolated from the formulas used to calculate a patient’s daily metabolic energy requirements because it takes 1 mL of water to metabolize 1 Kcal of energy (Table 1-9). The patient’s daily metabolic water (fluid) requirements can be calculated by the following formula:

\[
\text{Fluid (mL)} = [30 \times \text{Body mass (kg)}] + 70
\]

Administration of an isotonic crystalloid fluid for maintenance requirements often can produce iatrogenic hypokalemia. In most cases, supplemental potassium must be added to prevent hypokalemia resulting from inappetence, kaliuresis, and supplementation with isotonic crystalloid fluids.

**Calculation of Fluid Deficits and Ongoing Losses**

The most reliable method for determining the degree of fluid deficit is by weighing the animal and calculating acute weight loss. Acute weight loss in a patient with volume loss in the form of vomiting, feces, wound exudates, and urine is a result of fluid loss and not loss of muscle or fat. Lean body mass normally is not gained or lost rapidly enough to cause major changes in body weight. One milliliter of water weighs approximately 1 g. This fact allows calculation of the patient’s fluid deficit, if ongoing losses can be measured. When a patient is first presented, however, the body weight before a fluid deficit has occurred rarely is known. Instead, one must rely on subjective measures of dehydration to estimate the patient’s percent dehydration and to calculate the volume of fluid required to rehydrate the patient over the next 24 hours. To calculate the volume deficit, use the following formula:

\[
\text{Body mass (kg)} \times (\% \text{ dehydration}) \times 1000 = \text{Fluid deficit (mL)}
\]

The patient’s fluid deficit must be added to the daily maintenance fluid requirements and administered over a 24-hour period. Ongoing losses can be determined by measuring urine output, weighing the patient at least two or three times a day, and measuring the volume or weight of vomitus or diarrhea.

**Crystalloid and Colloid Fluids**

**Crystalloid Fluids**

A crystalloid fluid contains crystals of salts with a composition similar to that of the extracellular fluid space and can be used to maintain daily fluid requirements and replace fluid deficits or ongoing fluid losses (Table 1-10). Metabolic, acid-base, and electrolyte imbalances...
<table>
<thead>
<tr>
<th>Body Mass (kg)</th>
<th>Total Energy (Kcal) per Day</th>
<th>Total Water (mL) per Hour</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>100</td>
<td>4.2</td>
</tr>
<tr>
<td>2</td>
<td>130</td>
<td>5.4</td>
</tr>
<tr>
<td>3</td>
<td>160</td>
<td>6.7</td>
</tr>
<tr>
<td>4</td>
<td>190</td>
<td>7.9</td>
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<td>7</td>
<td>280</td>
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<tr>
<td>90</td>
<td>2770</td>
<td>115.4</td>
</tr>
<tr>
<td>95</td>
<td>2920</td>
<td>121.7</td>
</tr>
<tr>
<td>100</td>
<td>3070</td>
<td>127.9</td>
</tr>
</tbody>
</table>

*30 \times \text{Body weight (kg)} + 70 = \text{mL/day}. Note: This formula will slightly underestimate the requirements for patients that weigh less than 2 kg and will slightly overestimate the requirements for patients that weigh more than 70 kg.
also can be treated with isotonic fluids with or without supplemental electrolytes and buffers. Depending on the patient’s clinical condition, choose the specific isotonic crystalloid fluid to replace and maintain the patient’s acid-base and electrolyte status (Table 1-11). Crystalloid fluids are readily available, are relatively inexpensive, and can be administered safely in large volumes to patients with no preexisting cardiac or renal disease or cerebral edema. After infusion, approximately 80% of the volume of a crystalloid fluid infused will redistribute to the interstitial fluid compartment. Therefore crystalloid fluids alone are ineffective for ongoing intravascular volume depletion when given as a bolus. The crystalloid fluid bolus must be followed by a CRI, taking into consideration the patient’s daily maintenance fluid requirements and ongoing fluid losses. Administration of a large volume of crystalloid fluids can cause dilutional anemia and coagulopathies. Obtain the patient’s hematocrit before fluid infusion and regularly during the course of fluid therapy, particularly in patients with preexisting anemia or hypoproteinemia.

**Colloids**

A colloid is a large–molecular-weight particle that acts as an effective volume expander by drawing fluid from the interstitial fluid compartment into the intravascular space. When administered with a crystalloid, a colloid serves to hold or retain the crystalloid fluid within the vascular space for a longer time than if the crystalloid fluid were administered alone.
Because of this property, colloids can promote better tissue perfusion at lower infusion volumes and equivalent colloid oncotic pressures and mean BPs than crystalloids. Administer the synthetic colloids in incremental boluses of 5 to 10 mL/kg over 5 to 15 minutes during the treatment of hypotension. When synthetic colloids are administered for maintenance of colloidal oncotic pressure in hypoalbuminemic or hypoproteinemic patients, the recommended dose is 20 to 30 mL/kg/day as a CRI. Because colloids retain fluid in the vascular space, the volume of crystalloid fluid infused (maintenance + deficit + ongoing losses) should be decreased by 25% to 50% to avoid vascular volume overload.

Two major classes of colloids exist: natural and synthetic. Natural colloids (whole blood, packed RBCs, plasma) are discussed elsewhere in this text. Concentrated human albumin and canine specific purified concentrated albumin are natural purified colloids that recently have become more popular in the treatment of advanced hypoalbuminemia and hypoproteinemia and will be discussed here. Synthetic colloids are starch polymers and include hydroxyethyl starch and pentastarch.

Concentrated human albumin is available as a 5% or 25% solution. The 5% solution has an osmolality similar to that of serum (308 mOsm/L), whereas the 25% solution is hyperoncotic (1500 mOsm/L). A 25% albumin solution draws fluid from the interstitial space into the intravascular space. Concentrated albumin solutions often are used to restore circulating volume when synthetic colloids are not available. Albumin not only is important at maintaining the colloidal oncotic pressure of blood but also serves as a valuable free-radical scavenger and carrier of drugs and hormones necessary for normal tissue function and healing. Albumin levels less than 2.0 g/dL have been associated with increased morbidity and mortality. Concentrated human albumin solutions can be administered as an effective method of restoring interstitial and serum albumin concentrations in situations of acute and chronic hypoalbuminemia. Albumin (25%) is available in 50- and 100-mL vials and is more cost-efficient as an albumin replacement than fresh frozen plasma when procurement and administration are considered. Recommended albumin infusion rates are 2 to 5 mL/kg over 4 hours, after pretreatment with diphenhydramine. Although concentrated human albumin is structurally similar to canine albumin, closely monitor the patient for signs of allergic reaction during and after the infusion. Experimentally, concentrated infusion of human albumin into normoalbuminemic healthy dogs has resulted in the development of antialbumin antibodies, urticaria, fever, and acute anaphylaxis leading to death. Extreme reactions, however, have not been reported in clinically hypoalbuminemic patients into which albumin was infused. Polyarthritis and urticaria have also been documented within 3 weeks of albumin infusion. For these reasons, albumin therapy may be beneficial, but it is not innocuous. The perceived benefit must outweigh the potential risk of acute and chronic reactions in canine patients.

Hydroxyethyl starch solutions contain a large–molecular-weight amylopectin polymer that has molecules with a molecular weight that exceeds 100,000 D and has an average half-life of 24 to 36 hours in circulation. Hetastarch can bind with vWF and cause prolongation of the ACT and APTT; however, it does not cause coagulopathy. Recommended rates of hetastarch infusion are 5- to 10-mL incremental boluses for the treatment of hypotension and 20 to 30 mL/kg/day as a CRI for maintenance of colloidal oncotic pressure.

**Implementing the Fluid Therapy Plan**

Many are the acceptable ways to administer the fluids prescribed for each patient based on the degree of dehydration, estimation of ongoing losses, ability to tolerate oral fluid, and metabolic, acid-base, and electrolyte derangements. Administer the fluids in a manner that is best for the patient and most appropriate for the practice.

To determine the rate of intravenous fluid infusion, take the total volume of fluids that have been prescribed and divide the total volume by the total number of hours in a day that intravenous fluids can be delivered safely and monitored. The safest and most accurate way to deliver intravenous fluids, particularly in extremely small animals or those with CHF, is through an intravenous fluid pump. Fluid should not be administered IV if the patient cannot be monitored to make sure that the fluids are being delivered at a safe rate and that the fluid line has not become disconnected.
Supplement fluids over as many hours as possible to allow the patient as much time as possible to redistribute and fully use the fluids administered. Fluids administered too quickly can cause diuresis to occur, such that the majority of the fluids administered will be excreted in the urine. If time is limited or if extra time is needed for safe administration of fluids, consider using a combination of IV and subcutaneously (SQ) administered fluids. Intravenous is the preferred route of administration of fluids in any patient with dehydration and hypovolemia. As intravascular volume depletion occurs, reflex peripheral vasoconstriction occurs to restore core perfusion. The subcutaneous tissues are not perfused well, and therefore fluids administered SQ will not be absorbed well into the interstitial and intravascular spaces. SQ administered fluids can be absorbed slowly and delivered effectively in the management of mild interstitial dehydration and in the treatment of renal insufficiency. SQ administered fluids should never take the place of IV administered fluids in a hypovolemic patient or one with severe interstitial dehydration.

Intramedullary (intraosseous) infusion works well in small patients in which vascular access cannot be established. Shock doses of fluids and other substances, including blood products, can be administered under pressure through an intraosseous cannula. Because of the inherent discomfort and risk of osteomyelitis with intraosseous infusion, establish vascular access as soon as possible.

Rates of Administration
The safest and most efficient method of intravenous fluid infusion is through a fluid pump. In cases in which a fluid pump is unavailable, infusion by gravity feed is the next option. Infusion sets from various manufacturers have calibrated drip chambers such that a specific number of drops will equal 1 mL of fluid. Fluid rates can be calculated based on the number of drops that fall into the drip chamber per minute:

\[
\text{Fluid volume to be infused (mL) } \div \text{Number of hours available} = \text{mL / hour}
\]

Many pediatric drip sets deliver 60 drops/mL, such that milliliters per hour equals drops per minute. Carefully record fluid orders so that the volume to be administered is recorded as milliliters per hour, milliliters per day, and drops per minute. This will allow personnel to detect major discrepancies and calculation errors more readily. The volume actually delivered should be recorded in the record by nursing personnel. All additives should be listed clearly on the bottle on a piece of adhesive tape or a special label manufactured for this purpose. A strip of adhesive tape also can be attached to the bottle and marked appropriately to provide a quick visualization of the estimate of volume delivered.

Additional Reading
Orogastric lavage is indicated for gastric decontamination of most types of toxins, for elimination of food during food bloat, and for gastric decompression during surgery for gastric dilatation-volvulus syndrome (GDV). Equipment needed to perform orogastric lavage includes a large-bore flexible orogastric lavage tube, permanent marker or white tape, lubricating jelly, warm water, two large buckets, a roll of 2-inch white tape, and a manual lavage pump.

To perform orogastric lavage, follow this procedure:

1. Place all animals under general anesthesia with a cuffed endotracheal tube in place to protect the airway and prevent aspiration of gastric contents into the lungs.
2. Place a roll of 2-inch white tape into the animal’s mouth, and secure the tape around the muzzle. You will insert the tube through the hole in the center of the roll of tape.
3. Next, place the distal end of the tube at the level of the last rib, directly adjacent to the animal’s thorax and abdomen. Measure the length of the tube from the most distal end to the point where it comes out of the mouth, and label this location on the tube with a permanent marker or piece of white tape.
4. Lubricate the distal portion of the tube, and gently insert it through the roll of tape in the animal’s mouth.
5. Gently push the tube down the esophagus. Palpate the tube within the esophagus. Two tubes should be palpable: the orogastric tube and the patient’s trachea. Push the tube down into the stomach. You can verify location by blowing into the proximal end of the tube and simultaneously auscultating the stomach for borborygmi.
6. Insert the manual pump to the proximal end of the tube, and instill the warm water. Alternate instilling water with removal of fluid and gastric debris by gravity. Repeat the process until the efflux fluid is clear of any debris.
7. Save the gastric efflux fluid for toxicologic analyses.

Additional Reading


OXYGEN SUPPLEMENTATION

Hypoxia, or inadequate tissue oxygenation, is the primary reason for supplemental oxygen therapy. Major causes of hypoxia include hypoventilation, ventilation-perfusion mismatch, physiologic or right-to-left cardiac shunt, diffusion impairment, and decreased fraction of inspired oxygen (Table 1-12). Inadequate tissue perfusion caused by low cardiac output or vascular obstruction also can result in circulatory hypoxia. Finally, histiocytic hypoxia results from inability of cells to use oxygen that is delivered to them. This form of hypoxia can be observed with various toxin ingestions (bromethalin, cyanide) and in septic shock.

A patient’s oxygenation status can be monitored invasively by drawing arterial blood gas samples or noninvasively through pulse oximetry, in most cases (see Acid-Base Physiology and Pulse Oximetry sections). Inspired air at sea level has a Po2 of 150 mm Hg. As the air travels through the upper respiratory system to the level of the alveolus, the Po2 drops to 100 mm Hg. Tissue oxygen saturation in a normal healthy animal is 95 mm Hg. After oxygen has been delivered to the tissues, the oxygen left in the venous system (Pvo2) is approximately 40 mm Hg.
Normally, oxygen diffuses across the alveolar capillary membrane and binds reversibly with hemoglobin in RBCs. A small amount of oxygen is carried in an unbound diffusible form in the plasma. When an animal has an adequate amount of hemoglobin and hemoglobin becomes fully saturated while the animal is breathing room air, supplemental oxygen administration will only increase the $\text{SaO}_2$ a small amount. The unbound form of oxygen dissolved in plasma will increase. If, however, inadequate hemoglobin saturation is obtained by breathing room air, as in a case of pneumonia or pulmonary edema, for example, breathing a higher fraction of inspired oxygen ($\text{FiO}_2$) will improve bound and unbound hemoglobin levels. The formula for calculating oxygen content of arterial blood is as follows:

$$\text{CaO}_2 = (1.34 \times \text{Hb} \times \text{SaO}_2) + (0.003 \times \text{PaO}_2)$$

where $\text{CaO}_2$ is the arterial oxygen content, 1.34 is the amount of oxygen that can be carried by hemoglobin (Hb), $\text{SaO}_2$ is the hemoglobin saturation, and 0.003 $\times \text{PaO}_2$ is the amount of oxygen dissolved (unbound) in plasma.

Dissolved oxygen actually contributes little to the total amount of oxygen carried in the arterial blood, and the majority depends on the amount or availability of hemoglobin and the ability of the body (pH and respiratory status) to saturate the hemoglobin at the level of the alveoli.

**Indications for Oxygen Therapy**

Oxygen therapy is indicated whenever hypoxia is present. The underlying cause of the hypoxia also must be identified and treated, for chronic, lifelong oxygen therapy is rarely feasible in veterinary patients. If hemoglobin levels are low because of anemia, oxygen supplementation must occur along with RBC transfusions to increase hemoglobin mass. Whenever possible, use arterial blood gas analyses or pulse oximetry to gauge a patient’s response to oxygen therapy and to determine when an animal can be weaned from supplemental oxygen.

The goal of oxygen therapy is to increase the amount of oxygen bound to hemoglobin in arterial blood. Oxygen supplementation can be by hood, oxygen cage or tent, nasal or nasopharyngeal catheter, or tracheal tube. In rare cases, administration of oxygen with mechanical ventilation may be indicated.

Administration of supplemental oxygen to patients with chronic hypoxia is sometimes necessary but is also dangerous. With chronic hypoxia the patient develops a chronic respiratory acidosis (elevated $\text{Paco}_2$) and depends almost entirely on the hypoxic ventilatory drive to

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**TABLE 1-12 Types of Hypoxia and Response to Oxygen Supplementation**

<table>
<thead>
<tr>
<th>Type of Hypoxia</th>
<th>Cause</th>
<th>Response to Oxygen</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hypoxic hypoxia</strong></td>
<td>Central nervous system disease, drugs, rib fractures, thoracic cage damage, pneumothorax, pleural effusion</td>
<td>Responsive</td>
</tr>
<tr>
<td>Alveolar hypoventilation</td>
<td>Pneumonia, atelectasis</td>
<td>Partially responsive</td>
</tr>
<tr>
<td>Arteriovenous (physiologic)</td>
<td>Pneumonia, pulmonary edema, fibrosis, emphysema</td>
<td>Responsive</td>
</tr>
<tr>
<td>shunt</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diffusion impairment</td>
<td>Smoke inhalation, altitude</td>
<td>Responsive</td>
</tr>
<tr>
<td>Decreased $\text{FiO}_2$</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Histiocytic hypoxia</strong></td>
<td>Septic shock, toxins</td>
<td>Not very responsive</td>
</tr>
<tr>
<td><strong>Circulatory hypoxia</strong></td>
<td>Low cardiac output, vascular obstruction</td>
<td>Responsive</td>
</tr>
</tbody>
</table>

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breathe. Administration of supplemental oxygen increases \( Pao_2 \) and may inhibit the central respiratory drive, leading to hypoventilation and possibly respiratory arrest. Therefore, closely monitor animals with chronic hypoxia that are treated with supplemental oxygen.

**Oxygen Hood**

Oxygen hoods can be purchased from commercial sources or can be manufactured in the hospital using a rigid Elizabethan collar, tape, and plastic wrap. To make an oxygen hood, place several lengths of plastic wrap over the front of the Elizabethan collar and tape them in place. Leave the ventral third of the collar open to allow moisture and heat to dissipate and carbon dioxide to be eliminated. Place a length of flexible oxygen tubing under the patient’s collar into the front of the hood, and run humidified oxygen at a rate of 50 to 100 mL/kg/min. Animals may become overheated with an oxygen hood in place. Carefully monitor the patient’s temperature so that iatrogenic hyperthermia does not occur.

**Oxygen Cage**

Commercially available Plexiglas oxygen cages can be purchased from a variety of manufacturers. The best units include a mechanical thermostatically controlled compressor cooling unit, a circulatory fan, nebulizers or humidifiers to moisten the air, and a carbon dioxide absorber. Alternately, a pediatric (infant) incubator can be purchased from hospital supply sources, and humidified oxygen can be run into the cage at 2 to 10 L/min (depending on the size of the cage). High flow rates may be required to eliminate nitrogen and carbon dioxide from the cage. In most cases the \( FiO_2 \) inside the cage reaches 40% to 50% using this technique. Disadvantages of using an oxygen cage are high consumption or use of oxygen, rapid decrease in the \( FiO_2 \) within the cage whenever the cage must be opened for patient treatments, lack of immediate access to the patient, and potential for iatrogenic hyperthermia.

**Nasal or Nasopharyngeal Oxygen**

One of the most common methods for oxygen supplementation in dogs is the use of nasal or nasopharyngeal oxygen catheters. Nasal oxygen catheters tend to be irritating to the nasal mucosa and are not as well tolerated by patients as nasopharyngeal oxygen is.

1. To place a nasal or nasopharyngeal catheter, obtain a red rubber catheter (8F to 12F, depending on the size of the patient).
   a. For nasal oxygen supplementation, measure the distal tip of the catheter from the medial canthus of the eye to the tip of the nose.
   b. For nasopharyngeal oxygen supplementation, measure the catheter from the ramus of the mandible to the tip of the nose.
2. Mark the tube length at the tip of the nose with a permanent marker.
3. Instill topical anesthetic such as proparacaine (0.5%) or lidocaine (2%) into the nostril before placement.
4. Place a stay suture adjacent to (lateral aspect) the nostril while the topical anesthetic is taking effect.
5. Lubricate the tip of the tube with sterile lubricant.
6. Gently insert the tube into the ventral medial aspect of the nostril to the level made with the permanent marker. If you are inserting the tube into the nasopharynx, push the nasal meatus dorsally while simultaneously pushing the lateral aspect of the nostril medially to direct the tube into the ventral nasal meatus and avoid the cribiform plate.
7. Once the tube has been inserted to the appropriate length, hold the tube in place with your fingers adjacent to the nostril, and suture the tube to the stay suture. If the tube is removed, you can cut the suture around the tube and leave the stay suture in place for later use, if necessary.
8. Suture or staple the rest of the tube dorsally over the nose and between the eyes to the top of the head, or laterally along the zygomatic arch.
9. Attach the tube to a length of flexible oxygen tubing, and provide humidified oxygen at 50 to 100 mL/kg/min.
10. Secure an Elizabethan collar around the patient’s head to prevent the patient from scratching at the tube and removing it.

**Mechanical Ventilation**

The Rule of 60s states that if a patient’s Pao$_2$ is less than 60 mm Hg, or if the Paco$_2$ is 60 mm Hg, mechanical ventilation should be considered. For mechanical ventilation, anesthetize the patient and intubate the patient with an endotracheal tube. Alternately, a temporary tracheostomy can be performed and the patient can be maintained on a plane of light to heavy sedation and ventilated through the tracheostomy site. This method, although technically more invasive initially, allows the patient to be awake despite requiring mechanical ventilation. Mechanical ventilation is required in the most critically ill animals. If mechanical ventilation is required, 24-hour one-on-one veterinary care is necessary.

**Additional Reading**


**Pulse Oximetry**

A noninvasive means of determining oxygenation is through the use of pulse oximetry. A pulse oximeter uses different wavelengths of light to distinguish characteristic differences in the properties of the different molecules in a fluid or gas mixture—in this case, oxygenated (oxyhemoglobin) and deoxygenated hemoglobin (deoxyhemoglobin) in pulsatile blood. The process is termed pulse oximetry.

Oxyhemoglobin and deoxyhemoglobin are different molecules that absorb and reflect different wavelengths of light. Oxyhemoglobin absorbs light in the infrared spectrum, allowing wavelengths of light in the red spectrum to transmit through it. Conversely, deoxyhemoglobin absorbs wavelengths of the red spectrum and allows wavelengths in the infrared spectrum to transmit through the molecule. The spectrophotometer in the pulse oximeter transmits light in the red (660 nm) and infrared (920 nm) spectra. The different wavelengths of light are transmitted across a pulsatile vascular bed and are detected by a photodetector on the other side. The photodetector processes the amount of light of varying wavelengths that reaches it, then transmits an electrical current to a processor that calculates the difference in the amount of light originally transmitted and the amount of light of similar wavelength that actually reaches the photodetector. The difference in each reflects the amount of light absorbed in the pulsatile blood and can be used to calculate the amount or ratio of oxyhemoglobin to deoxyhemoglobin in circulation, or the functional hemoglobin saturation, by the following formula:

\[
\text{Sao}_2 = \frac{Hbo_2}{Hbo_2 + Hb}
\]

where Hbo$_2$ is oxygenated hemoglobin, and Hb is deoxygenated hemoglobin. Four molecules of oxygen reversibly bind to hemoglobin for transport to the tissues. Carbon monoxide similarly binds to hemoglobin and forms carboxyhemoglobin, a molecule that is detected similarly to oxygenated hemoglobin. Thus Sao$_2$ as detected by a pulse oximeter is not reliable if carboxyhemoglobin is present.
In most cases, pulse oximetry or \( \text{Sa}_2 \) corresponds reliably to the oxyhemoglobin dissociation curve. Oxygen saturation greater than 90% corresponds to a \( \text{Pa}_2 \) greater than 60 mm Hg. Above this value, large changes in \( \text{Pa}_2 \) are reflected in relatively small changes in \( \text{Sa}_2 \), making pulse oximetry a relatively insensitive method of determining oxygenation status when \( \text{Pa}_2 \) is normal.

Because pulse oximetry measures oxygenated versus nonoxygenated hemoglobin in pulsatile blood flow, it is fairly unreliable when severe vasoconstriction, hypothermia, shivering or trembling, or excessive patient movement are present. In addition, increased ambient lighting and the presence of methemoglobin or carboxyhemoglobin also can cause artifactual changes in the \( \text{Sa}_2 \), and therefore the measurement is not reliable or accurate. Most pulse oximeters also display a waveform and the patient’s heart rate. If the photodetector does not detect a good-quality signal, the waveform will not be normal, and the heart rate displayed on the monitor will not correlate with the patient’s actual heart rate.

**CAPNOMETRY (END-TIDAL CARBON DIOXIDE MONITORING)**

The efficiency of ventilation is evaluated using the \( \text{Paco}_2 \) value on an arterial blood gas sample. Alternatively, a noninvasive method to determine end-tidal carbon dioxide is through use of a capnograph. The science of capnometry uses a spectrophotometer to measure carbon dioxide levels in exhaled gas. The capnometer is placed in the expiratory limb of an anesthetic circuit. A sample of exhaled gas is aliquoted from the breath, and an infrared light source is passed across the sample. A photodetector on the other side of the sample flow measures the amount or concentration of carbon dioxide in the sample of expired gas. The calculated value is displayed as end-tidal carbon dioxide. This value also can be displayed as a waveform.

When placed in graphic form, a waveform known as a *capnogram* is displayed throughout the ventilatory cycle. Normally, at the onset of exhalation the gas exhaled into the expiratory limb of the tubing comes from the upper airway or physiologic dead space and contains relatively little carbon dioxide. As exhalation continues, a steep uphill slope occurs as more carbon dioxide is exhaled from the bronchial tree. Near the end of exhalation, the capnogram reaches a plateau, which most accurately reflects the carbon dioxide level at the level of the alveolus. Because carbon dioxide diffuses across the alveolar basement membrane so rapidly, this reflects arterial carbon dioxide levels. If a plateau is not reached and notching of the waveform occurs, check the system for leaks. If the baseline waveform does not reach zero, the patient may be rebreathing carbon dioxide or may be tachypneic, causing physiologic positive end-expiratory pressure. The Sodasorb in the system should be replaced if it has expired. Conversely, low end-tidal carbon dioxide may be associated with a decrease in perfusion or blood flow. Decreased perfusion can be associated with low end-tidal carbon dioxide values, particularly during cardiopulmonary cerebral resuscitation (CPCR). End-tidal carbon dioxide levels are one of the most accurate predictors of the efficacy of CPCR and patient outcome. In addition, the difference between arterial carbon dioxide levels (\( \text{Paco}_2 \)) and end-tidal carbon dioxide can be used to calculate dead-space ventilation. Increases in the difference also occur with poor lung perfusion and pulmonary diffusion impairment.

**Additional Reading**


THORACOCENTESIS

Thoracocentesis refers to the aspiration of fluid or air from within the pleural space. Thoracocentesis may be diagnostic to determine whether air or fluid is present and to characterize the nature of the fluid obtained. Thoracocentesis also can be therapeutic when large volumes of air or fluid are being removed to allow pulmonary reexpansion and correction of hypoxemia and orthopnea.

To perform thoracocentesis, follow this procedure:
1. First, assemble the equipment necessary (Box 1-16).
2. Next, clip a 10-cm square in the center of the patient’s thorax on both sides.
3. Aseptically scrub the clipped area.
4. Ideally, thoracocentesis should be performed within the seventh to ninth intercostal space. Rather than count rib spaces in an emergent situation, visualize the thoracic cage as a box, and the clipped area as a box within the box. You will insert your needle or catheter in the center of the box and then direct the bevel of the needle dorsally or ventrally to penetrate pockets of fluid or air present.
5. Attach the needle or catheter hub to the length of intravenous extension tubing. Attach the female port of the intravenous extension tubing to the male port of the three-way stopcock. Attach the male port of the 60-mL syringe to one of the female ports of the three-way stopcock. The apparatus is now assembled for use.
6. Insert the needle through the intercostal space such that the bevel of the needle initially is directed downward.
7. Next, push down on the hub of the needle such that the needle becomes parallel with the thoracic wall. Through movement of the hub of the needle in a clockwise or counterclockwise manner, the bevel of the needle will move within the thoracic cavity to penetrate pockets of air or fluid. In general, air is located dorsally and fluid is located more ventrally, although this is not always true.
8. Aspirate air or fluid. Save any fluid obtained for cytologic and biochemical analyses and bacterial culture and susceptibility testing. In cases of pneumothorax, if thoracocentesis needs to be repeated more than three times, consider using a thoracostomy tube.

THORACOSTOMY TUBE

Place a thoracostomy tube in cases of pneumothorax whenever negative suction cannot be obtained or repeated accumulation of air requires multiple thoracocentesis procedures. Thoracostomy tubes also can be placed to drain rapidly accumulating pleural effusion and for the medical management of pyothorax. Before attempting thoracostomy tube placement, make sure that all necessary supplies are assembled (Box 1-17; Table 1-13).

To place a thoracostomy tube, follow this procedure:
1. Lay the patient in lateral recumbency.
2. Clip the patient’s entire lateral thorax.
3. Aseptically scrub the lateral thorax.
4. Palpate the tenth intercostal space.

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**Box 1-16 Equipment Required for Thoracocentesis**

- 22- to 20-inch over-the-needle catheters or hypodermic needles
- 60-mL catheter-tipped syringe
- Intravenous extension tubing
- Three-way stopcock
- Clippers
- Antimicrobial scrub
- Latex gloves
5. Have an assistant pull the patient’s skin cranially and ventrally toward the point of the elbow. This will facilitate creating a subcutaneous tunnel around the thoracostomy tube.

6. Draw up 2 mg of 2% lidocaine per kilogram (1 mg/kg for cats) along with a small amount of sodium bicarbonate to take away some of the sting.

7. Insert the needle at the dorsal aspect of the tenth intercostal space and to the seventh intercostal space. Inject the lidocaine into the seventh intercostal space at the point where the trocarized thoracic drainage catheter will penetrate into the thoracic cavity. Slowly infuse the lidocaine as you withdraw the needle to create an anesthetized tunnel through which to insert the catheter.

8. While the local anesthetic is taking effect, remove the trocar from the catheter and cut the proximal end of the catheter with Mayo scissors to facilitate adaptation with the Christmas tree adapter.

9. Attach the Christmas tree adapter to the three-way stopcock and the three-way stopcock to a length of intravenous extension tubing and the 60-mL syringe so that the apparatus can be attached immediately to the thoracostomy tube after placement.

10. Aseptically scrub the lateral thorax a second time and then drape it with sterile huck towels secured with towel clamps.

11. Wearing sterile gloves, make a small stab incision at the dorsal aspect of the tenth intercostal space.

12. Insert the trocar back into the thoracostomy drainage tube. Insert the trocar and tube into the incision. Tunnel the tube cranially for approximately three intercostal spaces while an assistant simultaneously pulls the skin cranially and ventrally toward the point of the elbow.

13. At the seventh intercostal space, direct the trocar and catheter perpendicular to the thorax. Grasp the catheter apparatus at the base adjacent to the thorax to prevent the trocar from going too far into the thorax.

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**TABLE 1-13  Size of Dog or Cat and Appropriate Chest Catheter Size**

<table>
<thead>
<tr>
<th>Dog or Cat size</th>
<th>Catheter</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;7 kg</td>
<td>14F-16F</td>
</tr>
<tr>
<td>7-15 kg</td>
<td>18F-22F</td>
</tr>
<tr>
<td>16-30 kg</td>
<td>22F-28F</td>
</tr>
<tr>
<td>&gt;30 kg</td>
<td>28F-36F</td>
</tr>
</tbody>
</table>

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**BOX 1-17  SUPPLIES REQUIRED FOR PLACEMENT OF A THORACOSTOMY TUBE**

- Argyle trocar thoracic drainage catheter
- Three-way stopcock
- 22-gauge orthopedic wire
- No. 10 scalp blade
- Needle holder (sterile)
- Sterile huck towels
- Thumb forceps (sterile)
- 2-0 to 0 nonabsorbable suture
- Gauze 4- × 4-inch squares (sterile)
- Clippers
- Clear adhesive antimicrobial barrier drape
- Christmas tree adapter
- Intravenous extension tubing
- Mayo scissors (sterile)
- Scalpel handle (sterile)
- 2% Lidocaine
- Towel clamps
- 25-gauge hypodermic needle
- 3- to 6-mL syringe
- Cotton roll gauze
- Elastikon
- Sterile gloves

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14. Place the palm of your dominant hand over the end of the trocar, and push the trocar and catheter into the thoracic cavity, throwing your weight into the placement in a swift motion, not by banging the butt of your hand on the end of the stylette. For small individuals, standing on a stool or kneeling over the patient on the triage table can create leverage and make this process easier. The tube will enter the thorax with a pop.

15. Gently push the catheter off of the stylette and remove the stylette.

16. Immediately attach the Christmas tree adapter and have an assistant start to withdraw air or fluid while you secure the tube in place.

17. First, place a horizontal mattress suture around the tube to cinch the skin securely to the tube. Use care to not penetrate the tube with your needle and suture.

18. Next, place a purse-string suture around the tube at the tube entrance site. Leave the ends of the suture long, so that you can create a finger-trap suture to the tube, holding the tube in place.

19. Place a large square of antimicrobial-impregnated adhesive tape over the tube for further security and sterility.

20. If antimicrobial adhesive is not available, place a gauze pad 4 × 4 inches square over the tube, and then wrap the tube to the thorax with cotton roll gauze and Elastikon adhesive tape.

21. Draw the location of the tube on the bandage to prevent cutting it with subsequent bandage changes.

An alternate technique to use if a trocar thoracic drainage catheter is not available is the following:

1. Prepare the lateral thorax and infuse local lidocaine anesthetic as listed before.

2. Make a small stab incision with a No. 10 scalpel blade as listed before.

3. Obtain the appropriately sized red rubber catheter and cut multiple side ports in the distal end of the catheter, taking care to not cut more than 50% of the circumference of the diameter of the tube.

4. Insert a rigid, long urinary catheter into the red rubber catheter to make the catheter more rigid during insertion into the pleural space.

5. Grasp the distal end of the catheter(s) in the teeth of large Carmalt forceps. Tunnel Metzenbaum scissors under the skin to the seventh intercostal space and make a puncture through the intercostal space.

6. Remove the Metzenbaum scissors, and then tunnel the Carmalt forceps and red rubber tube under the skin to the hole created in the seventh intercostal space with the Metzenbaum scissors.

7. Insert the tips of the Carmalt forceps and the red rubber catheter through the hole, and then open the teeth of the Carmalt forceps.

8. Push the red rubber catheter cranially into the pleural cavity.

9. Remove the Carmalt forceps and the rigid urinary catheter, and immediately attach the suction apparatus. Secure the red rubber catheter in place as listed before.

Additional Reading


TRACHEOSTOMY

Placement of a temporary tracheostomy can be lifesaving; it can be used to relieve upper respiratory tract obstruction, to facilitate removal of airway secretions, to decrease dead space ventilation, to provide a route of inhalant anesthesia during maxillofacial surgery, and to facilitate mechanical ventilation.

In an emergent situation in which asphyxiation is imminent and endotracheal intubation is not possible, any cutting instrument placed into the trachea distal to the point of obstruction can be used. To perform a slash tracheostomy, quickly clip the fur and scrub the skin over the third tracheal ring. Make a small cut in the trachea with a No. 11 scalpel blade, and insert a firm tube, such as a syringe casing. Alternately, insertion of a 22-gauge needle attached to intravenous extension tubing and adapted with a 1-mL syringe case to attach to a humidified oxygen source also temporarily can relieve obstruction until a temporary tracheostomy can be performed.

In less emergent situations, place the patient under general anesthesia and intubate the patient. Assemble all the equipment necessary before starting the temporary tracheostomy procedure (Box 1-18).

To perform a tracheostomy, follow this procedure:

1. Place the patient in dorsal recumbency.
2. Clip the ventral cervical region from the level of the ramus of the mandible caudally to the thoracic inlet and dorsally to midline.
3. Aseptically scrub the clipped area, and then drape with sterile huck towels secured with towel clamps.
4. Make a 3-cm ventral midline skin incision over the third to sixth tracheal rings, perpendicular to the trachea.
5. Bluntly dissect through the sternohyoid muscles to the level of the trachea.
6. Carefully pick up the fascia overlying the trachea and cut it away with Metzenbaum scissors.
7. Place two stay sutures through or around adjacent tracheal rings.
8. Incise between trachea rings with a No. 11 scalpel blade. Take care to not cut more than 50% of the circumference of the trachea.
9. Using the stay sutures, pull the edges of the tracheal incision apart, and insert the tracheostomy tube. The Shiley tube contains an internal obturator to facilitate placement into the tracheal lumen. Remove the obturator, and then insert the inner cannula, which can be removed for cleaning as needed.
10. Once the tube is in place, secure the tube around the neck with a length of sterile umbilical tape.

TRACHEOSTOMY TUBE CARE

Postoperative care of the tracheostomy tube is as important as the procedure itself. Because the tracheostomy tube essentially bypasses the protective effects of the upper respiratory system, one of the most important aspects of tracheostomy tube care and maintenance is to maintain sterility at all times. Any oxygen source should be humidi-

**BOX 1-18  SUPPLIES REQUIRED FOR A TRACHEOSTOMY**

- Sterile huck towels
- Towel clamps
- Antimicrobial scrub
- No. 10 scalpel blade
- Curved mosquito hemostats
- Metzenbaum scissors
- Thumb forceps
- 3-0 to 2-0 nonabsorbable suture material
- Needle holders
- Shiley low-pressure cuff tracheostomy tube or endotracheal tube that has been cut and adapted to create a tracheostomy tube
- Umbilical tape
fied with sterile water or saline to prevent drying of the respiratory mucosa. If supplemental oxygen is not required, instill 2 to 3 mL of sterile saline every 1 to 2 hours to moisten the mucosa. Wearing sterile gloves, remove the internal tube and place it in a sterile bowl filled with sterile hydrogen peroxide, to be cleaned every 4 hours (or more frequently as necessary). If a Shiley tube is not available, apply suction to the internal lumen of the tracheostomy tube every 1 to 2 hours (or more frequently as needed) with a sterile 12F red rubber catheter attached to a vacuum pump to remove any mucus or other debris that potentially could plug the tube. Unless the patient demonstrates clinical signs of fever or infection, the prophylactic use of antibiotics is discouraged because of the risk of causing a resistant infection. After the temporary tracheostomy is no longer necessary, remove the tube and suture, and leave the wound to heal by second intention. Primary closure of the wounds could predispose the patient to subcutaneous emphysema and infection.

Additional Reading

UROHYDROPULSION
Urohydropulsion is a therapeutic procedure for removal of uroliths from the urethra of the male dog. The technique works best if the animal is heavily sedated or is placed under general anesthesia (Figure 1-12).

To perform urohydropulsion, follow this procedure:
1. Place the animal in lateral recumbency.
2. Clip the fur from the distal portion of the prepuce.
3. Aseptically scrub the prepuce and flush the prepuce with 12 to 20 mL of antimicrobial flush solution.
4. Have an assistant who is wearing gloves retract the penis from the prepuce.
5. While wearing sterile gloves, lubricate the tip of a rigid urinary catheter as for urethral catheterization.
6. Gently insert the tip of the catheter into the urethra until you meet the resistance of the obstruction.
7. Pinch the tip of the penis around the catheter.
8. Have an assistant insert a gloved lubricated finger into the patient’s rectum and press ventrally on the floor of the rectum to obstruct the pelvic urethra.
9. Attach a 60-mL syringe filled with sterile saline into proximal tip of the catheter.
10. Quickly inject fluid into the catheter and alternate compression and relaxation on the pelvic urethra such that the urethra dilates and suddenly releases the pressure, causing dislodgement of the stone. Small stones may be ejected from the tip of the urethra, whereas larger stones may be retropulsed back into the urinary bladder to be removed surgically at a later time.

Additional Reading
Osborne CA, Finco DR: Canine and feline nephrology and urology, Baltimore, 1995, Williams & Wilkins.
The type of catheter that you choose for vascular access depends largely on the size and species of the patient, the fragility of the vessels to be catheterized, the proposed length of time that the catheter will be in place, the type and viscosity of the fluid or drug to be administered, the rate of fluid flow desired, and whether multiple repeated blood samples will be required (Table 1-14).

A variety of over-the-needle, through-the-needle, and over-the-wire catheters are available for placement in a variety of vessels, including the jugular, cephalic, accessory cephalic, medial saphenous, lateral saphenous, dorsal pedal artery, and femoral arteries.

<table>
<thead>
<tr>
<th>Table 1-14 Catheter Sizes for Vascular Access</th>
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<tbody>
<tr>
<td><strong>Cephalic or Tarsal Vein</strong> (Catheter Gauge)</td>
</tr>
<tr>
<td>Cat or small dog</td>
</tr>
<tr>
<td>Medium-sized dog</td>
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<tr>
<td>Large dog</td>
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</table>

**Figure 1-12:** Removal of urethrolith in a male dog by urohydropropulsion. 1, Urethrolith originating from the urinary bladder has lodged behind the os penis. 2, Dilation of the urethral lumen is achieved by injecting fluid with pressure. Digital pressure applied to the external urethral orifice and the pelvic urethra has created a closed system. 3, Sudden release of digital pressure at the external urethral orifice and subsequent movement of fluid and urethrolith toward the external urethral orifice. 4, Sudden release of digital pressure at the pelvic urethra and subsequent movement of fluid and urethrolith toward the urinary bladder. (From Osborne CA, Finco DR: Canine and feline nephrology and urology, Baltimore, 1995, Williams & Wilkins.)
One of the most important aspects of proper catheter placement and maintenance is to maintain cleanliness at all times. The patient’s urine, feces, saliva, and vomit are common sources of contamination of the catheter site. Before placing a peripheral or central catheter in any patient, consider the patient’s physical status including the presence of vomiting, diarrhea, excessive urination, or seizures. In a patient that has an oral mass and is drooling excessively or a patient that is vomiting, peripheral cephalic catheterization may not be the most appropriate, to prevent contamination. Conversely, in a patient with excessive urination or diarrhea, a lateral or medial saphenous catheter is likely to become contaminated quickly.

Whenever one places or handles a catheter or intravenous infusion line, the person should wash the hands carefully and wear gloves to prevent contamination of the intravenous catheter and fluid lines. One of the most common sources of catheter contamination in veterinary hospitals is caretakers’ hands. In emergent situations, placement of a catheter may be necessary under less than ideal circumstances. Remove those catheters as soon as the patient’s condition is more stable, and place a second catheter using aseptic techniques.

In general, once the location of the catheter has been decided, set up all equipment necessary for catheter placement before starting to handle and restrain the patient. Box 1-19 lists the equipment needed for most types of catheter placement.

After setting up all of the supplies needed, clip the fur over the site of catheter placement. Make sure to clip all excess fur and long feathers away from the catheter site to prevent contamination. For catheter placement in limbs, clip the fur circumferentially around the site of catheter placement to facilitate adherence of the tape to the limb and to facilitate catheter removal with minimal discomfort at a later date. Next, aseptically scrub the catheter site with an antimicrobial scrub solution such as Hibiclens. The site is now ready for catheter insertion.

Central Venous Catheters
Consider using a central venous catheter whenever multiple repeated blood samples will need to be collected from a patient during the hospital stay. Central venous catheters also can be used for CVP measurement, administration of hyperoncotic solutions such as parenteral nutrition, and administration of crystalloid and colloid fluids, anesthesia, and other injectable drugs (Figures 1-13 and 1-14).

Through-the-needle or over-the-wire catheters are available from a variety of veterinary and human manufacturers and distributors. Over-the-wire central venous catheters can be placed by the Seldinger technique. Sterility must be maintained at all times, regardless of the type of catheter placed.

Percutaneous Over-the-Wire Jugular Catheter Placement (Seldinger Technique)
Central catheters also can be placed via the Seldinger or over-the-wire technique. A number of companies manufacture kits that contain the supplies necessary for over-the-wire catheter placement. Each kit minimally should contain an over-the-needle catheter to

<table>
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<tr>
<th>BOX 1-19  EQUIPMENT NECESSARY FOR INTRAVENOUS CATHETER PLACEMENT</th>
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<tbody>
<tr>
<td>• Antimicrobial scrub</td>
</tr>
<tr>
<td>• Cotton ball</td>
</tr>
<tr>
<td>• Electric clippers and No. 40 blade</td>
</tr>
<tr>
<td>• Gauze, 4 × 4-inch squares</td>
</tr>
<tr>
<td>• Heparinized saline flush</td>
</tr>
<tr>
<td>• Intravenous catheter</td>
</tr>
<tr>
<td>• ½- and 1-inch white adhesive tape</td>
</tr>
<tr>
<td>• Male adapter or T port flushed with heparinized saline</td>
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Contributed by A. Looney, B. Hansen, and E. Hardie.
place into the vessel, a long wire to insert through the original catheter placed, a vascular
dilator to dilate the hole in the vessel created by the first catheter, and a long catheter to
place into the vessel over the wire. Additional accessories can include a paper drape, sterile
gauze, a scalpel blade, local anesthetic, 22-gauge needles, and 3- or 6-mL syringes.

To place a jugular central venous catheter, place the patient in lateral recumbency
and extend the head and neck such that the jugular furrow is straight. Clip the fur
from the ramus of the mandible caudally to the thoracic inlet and dorsally and ven-
trally to midline. Wipe the clipped area with 4- × 4-inch gauze squares to remove any
loose fur and other debris. Aseptically scrub the clipped area with an antimicrobial
cleanser.

Figure 1-13: Lateral thoracic radiograph of a central venous catheter. Note that the tip of the
catheter is inserted in its proper location, just outside the right atrium.

Figure 1-14: Measuring the patient’s central venous pressure (CVP). Note that the 0 marker
on the manometer is at the patient’s manubrium.
Wearing sterile gloves, drape the site of catheter placement with sterile drapes, and occlude the jugular vein at the level of the thoracic inlet.

Pick up the skin over the site of catheter placement, and insert a small bleb of local anesthetic through the skin. The local anesthetic should not be injected into the underlying vessel (Figure 1-15). Make a small nick into the skin through the local anesthetic with a No. 10 or No. 11 scalpel blade. Use care to avoid lacerating the underlying vessel. Next, occlude the jugular vein as previously described, and insert the over-the-needle catheter into the vessel. Watch for a flash of blood in the catheter hub. Remove the stylette from the catheter. Next, insert the long wire into the catheter and into the vessel (Figures 1-16 and 1-17).

Figure 1-15: Infusion of local anesthetic. Before making a nick incision through the skin, insert a bleb of lidocaine over the proposed site of catheter insertion. Pick up the skin to avoid intravenous injection of the anesthetic.

Figure 1-16: The J-wire is curved at its tip to prevent iatrogenic trauma to the vessel and the heart. Pull the J-wire back so that the curve straightens out, and then insert the J-wire into the vessel.
Never let go of the wire. Remove the catheter, and place the vascular dilator over the wire and into the vessel (Figure 1-18). Gently twist to place the dilator into the vessel a short distance, creating a larger hole in the vessel. The vessel will bleed more after creation of a larger hole. Remove the vascular dilator, and leave the wire in place within the vessel. Insert the long catheter over the wire into the vessel (Figure 1-19). Push the catheter into the vessel to the catheter hub (Figure 1-20). Slowly thread the wire through a proximal port in the catheter. Once the catheter is in place, remove the wire, and suture the catheter in place to the skin with nonabsorbable suture.

Gently wrap layers of cotton roll gauze, Kling, and Elastikon or Vetrap over the catheter. Secure a male adapter or T port that has been flushed with heparinized saline,
and then label the catheter with the size and length of catheter, date of catheter placement, and initials of the person who placed the catheter. The catheter is ready for use. Monitor the catheter site daily for erythema, drainage, vessel thickening, or pain on infusion. If any of these signs occur, or if the patient develops a fever of unknown origin, remove the catheter, culture the catheter tip aseptically, and replace the catheter in a different location. As long as the catheter is functional without complications, the catheter can remain in place.
PERIPHERAL ARTERIAL AND VEINOUS CATHETER PLACEMENT

Cephalic Catheterization

Place the patient in sternal recumbency as for cephalic venipuncture. Clip the antebrachium circumferentially, and wipe the area clean of any loose fur and debris (Figure 1-21). Aseptically scrub the clipped area, and have an assistant occlude the cephalic vein at the crook of the elbow. The person placing the catheter should grasp the distal carpus with the nondominant hand and insert the over-the-needle catheter into the vessel at a 15- to 30-degree angle (Figure 1-22). Watch for a flash of blood in the catheter hub, and then gently push the catheter off of the stylette (Figure 1-23). Have the assistant occlude the vessel over the catheter to prevent backflow. Flush the catheter with heparinized saline solution.

Figure 1-21: Clip the patient’s antebrachium circumferentially to allow proper placement of the cephalic intravenous catheter.

Figure 1-22: Insert the catheter through the skin into the vessel, watching for a flash of blood in the catheter hub.
Make sure that the skin and catheter hub are clean and dry to ensure that the tape adheres to the catheter hub and skin. Secure a length of ⅛-inch white tape tightly around the catheter and then around the limb. Make sure that the catheter hub does not "spin" in the tape, or the catheter will fall out. Next, secure a second length of 1-inch adhesive tape under the catheter and around the limb and catheter hub (Figure 1-24). This piece of tape helps to stabilize the catheter in place. Finally, place a flushed T port or male adapter in the catheter hub and secure to the limb with white tape. Make sure that the tape is adhered to the skin securely, but not so tightly as to impede venous outflow (Figure 1-25). The catheter site can be covered with a cotton ball impregnated with antimicrobial ointment and layers of bandage material. Label all catheters with the date of placement, the type and gauge of catheter inserted, and the initials of the person who placed the catheter.
Percutaneous Femoral Artery Catheterization

The femoral artery can be catheterized for placement of an indwelling arterial catheter. Indwelling arterial catheters can be used for continuous invasive arterial BP monitoring and for procurement of arterial blood samples. Place the patient in lateral recumbency, and tape the down leg in an extended position. Clip the fur over the femoral artery and aseptically scrub the clipped area. Palpate the femoral artery as it courses distally on the medial surface of the femur and anterior to the pectineus muscle. Make a small nick incision over the proposed site of catheter placement using the bevel of an 18-gauge needle. Place a long over-the-needle catheter through the nick in the skin and direct it toward the palpable pulse. Place the tip of the catheter so that the needle tip rests in the subcutaneous tissue between the artery and the palpating index finger. Advance the needle steeply at a 30-degree angle to secure the superficial wall of the vessel and then the deep wall of the vessel. The spontaneous flow of blood in the catheter hub ensures that the catheter is situated in the lumen of the artery. Feed the catheter off of the stylette, and cover the hub with a catheter cap. Flush the catheter with sterile heparinized saline solution, and then secure it in place. Some persons simply tape the catheter in place with pieces of \( \frac{1}{2} \) - and 1-inch adhesive tape. Others use a “butterfly” piece of tape around the catheter hub and suture or glue the tape to the adjacent skin for added security.

Percutaneous Dorsal Pedal Artery Catheterization

The dorsal pedal artery commonly is used for catheter placement. To place a dorsal pedal arterial catheter, place the patient in lateral recumbency. Clip the fur over the dorsal pedal artery, and then aseptically scrub the clipped area. Tape the distal limb so that the leg is twisted slightly medially for better exposure of the vessel, or the person placing the arterial catheter can manipulate the limb into the appropriate position. Palpate the dorsal pedal pulse as it courses dorsally over the tarsus. Place an over-the-needle catheter percutaneously at a 15- to 30-degree angle, threading the tip of the needle carefully toward the pulse. Advance the needle in short, blunt movements, and watch the catheter hub closely for a flash of pulsating blood that signifies penetration into the lumen of the artery. Then thread the catheter off of the stylette, and cover the catheter hub with a catheter cap. Secure the catheter in place with lengths of \( \frac{1}{2} \) - and 1-inch adhesive tape as with any other intravenous catheter, and then flush it with heparinized saline solution every 2 to 4 hours.
Surgical Cutdown for Arterial and Venous Catheter Placement

Any vessel that can be catheterized percutaneously also can be catheterized with surgical cutdown. Restrain the patient and clip and aseptically scrub the limb or jugular vein as for a percutaneous catheterization procedure. Block the area for catheter placement with a local anesthetic before cutting the skin over the vessel with a No. 11 scalpel blade. While wearing sterile gloves, pick up the skin and incise the skin over the vessel. Direct the sharp edge of the blade upward to avoid lacerating the underlying vessel. Using blunt dissection, push the underlying subcutaneous fat and perivascular fascia away from the vessel with a mosquito hemostat. Make sure that all tissue is removed from the vessel. Using the mosquito hemostat, place two stay sutures of absorbable suture under the vessel. Elevate the vessel until it is parallel with the incision, and gently insert the catheter and stylette into the vessel. Secure the stay sutures loosely around the catheter. Suture the skin over the catheter site with non-absorbable suture, and then tape and bandage the catheter in place as for percutaneous placement. Remove catheters placed surgically as soon as possible and exchange them for a percutaneously placed catheter to prevent infection and thrombophlebitis.

Maintenance of Indwelling Arterial and Venous Catheters

The most important aspect of catheter maintenance is to maintain cleanliness and sterility at all times. An indwelling catheter can remain in place for as long as it is functional and no complications occur. Change the bandage whenever it becomes wet or soiled to prevent wicking of bacteria and debris from the environment into the vessel. Check the bandages and catheter sites at least once a day for signs of thrombophlebitis: erythema, vessel hardening or ropiness, pain on injection or infusion, and discharge. Also closely examine the tissue around and proximal and distal to the catheter. Swelling of the paw can signify that the catheter tape and bandage are too tight and are occluding venous outflow. Swelling above the catheter site is characteristic of perivascular leakage of fluid and may signify that the catheter is no longer within the lumen of the vessel.

Remove the catheter if it is no longer functional, if there is pain or resistance on infusion, if there is unexplained fever or leukocytosis, or if there is evidence of cellulitis, thrombophlebitis, or catheter-related bacteremia or septicemia. Aseptically culture the tip of the indwelling catheter for bacteria. Animals should wear Elizabethan collars or other forms of restraint if they lick or chew at the catheter or bandage.

Catheter patency may be maintained with constant fluid infusion or by intermittent flushing with heparinized saline (1000 units of unfractionated heparin per 250 to 500 mL of saline) every 6 hours. Flush arterial catheters more frequently (every 2 hours). Disconnect intravenous connections only when absolutely necessary. Wear gloves whenever handling the catheter or connections. Label all fluid lines and elevate them off of the floor to prevent contamination. Date each fluid line and replace it every 24 to 36 hours.

Intraosseous Catheter Placement

If an intravenous catheter cannot be placed because of small patient size, hypovolemia, hypothermia, or severe hypotension, needles can be placed into the marrow cavity of the femur, humerus, and tibia for intraosseous infusion of fluids, drugs, and blood products. This technique is particularly useful in small kittens and puppies and in exotic species. Contraindications to intraosseous infusion include avian species (which have pneumatic bones), fractures, and sepsis, because osteomyelitis can develop. An intraosseous catheter is relatively easy to place and maintain but can cause patient discomfort and so should be changed to an intravenous catheter as soon as vascular access becomes possible.

To place an intraosseous catheter, clip and aseptically scrub the fur over the proposed site of catheter placement. The easiest place for intraosseous placement is in the intertrochanteric fossa of the femur. Inject a small amount of a local anesthetic through the skin and into the periosteum where the trocar or needle will be inserted. Place the patient in lateral recumbency, and grasp the leg between your fingers, with the stifle braced against the palm of your hand. Push the stifle toward the abdomen (medially) to abduct the proximal femur.
away from the body. This will shift the sciatic nerve out of the way of catheter placement. Insert the tip of the needle through the skin and into the intertrochanteric fossa. Gently push with a simultaneous twisting motion, pushing the needle parallel with the shaft of the femur, toward your palm. You may feel a pop or decreased resistance as the needle enters the marrow cavity. Gently flush the needle with heparinized saline. If the needle is plugged with bone debris, remove the needle and replace it with a fresh needle of the same type and size in the hole that you have created. A spinal needle with an internal stylette also can be placed. The stylette will prevent the needle from becoming clogged with bone debris during insertion. Secure the hub of the needle with a butterfly length of white adhesive tape and then suture it to the skin to keep the catheter in place. The catheter is now ready for use. The patient should wear an Elizabethan collar to prevent disruption or removal of the catheter. The intraosseous catheter can be maintained as any peripheral catheter, with frequent flushing and daily evaluation of the catheter site.

Additional Reading


PAIN: ASSESSMENT, PREVENTION, AND MANAGEMENT*

The definition of pain has been debated philosophically over the ages and has changed as knowledge has increased. Pain is defined as an unpleasant sensory or emotional experience associated with actual or perceived tissue damage. Until recognition of a noxious stimulus occurs in the cerebral cortex, no response or adaptation results. Rational management of pain requires an understanding of the underlying mechanisms involved in pain and an appreciation of how analgesic agents interact to disrupt pain mechanisms.

Multiple factors and causes produce pain in human beings and domestic animal species. The causes of pain, psychological and physical, may derive from many different mechanisms within emergency medicine, among them trauma, infectious disease, neglect, environmental stress, surgery, and acute decompensation of chronic medical conditions. The two major classes of pain are acute and chronic pain. Box 1-20 gives specific categories and causes of pain.

The pain sensing and response system can be divided into the following categories: nociceptors, which detect and filter the intensity of the noxious stimuli; primary afferent nerves, which transmit impulses to the central nervous system (CNS); ascending tracts, which are part of the dorsal horn and the spinal cord that conveys stimuli to higher centers in the brain; higher centers, which are involved in pain discrimination, some memory, and motor control; and modulating or descending systems, which are a means of

*Contributed by A. Looney, B. Hansen, and E. Hardie.
processing, memorizing, and modifying incoming impulses. Current analgesic therapies may inhibit afferent nociceptive transmission within the brain and spinal cord; directly interrupt neural impulse conduction through the dorsal horn, primary afferent nerves, or dorsal root ganglion; or prevent the nociceptor sensitization that accompanies initial pain and inflammation. The physiologic aspects of pain are believed to be produced by the transmission, transduction, and integration of information from initial nerve endings, peripheral neuronal input, and ascending afferent nerves via the thalamus to the cerebral cortex. Ascending afferent nerves to the limbic system are believed to be responsible for the emotional aspects of pain.

There are several classification schemes for different types of pain. Acute pain, such as the pain that results from trauma, surgery, or infectious agents, is abrupt in onset, is relatively short in duration, and may be alleviated easily by analgesics. In contrast, chronic pain is a long-standing physical disorder or emotional distress that is slow in onset and difficult to treat. Both types of pain can be classified further based on site of origin. Somatic pain arises from superficial skin, subcutaneous tissue, body wall, or appendages. Visceral pain arises from abdominal or thoracic viscera and primarily is associated with serosal irritation. Analgesia, then, is the loss of pain without the loss of consciousness. This is in contrast to anesthesia, which is the loss of sensation in the whole body or a part of the body with the loss of consciousness or at least depression of the CNS.

**PHYSIOLOGIC IMPACT OF UNTREATED PAIN**

Untreated pain causes immediate changes in the neurohormonal axis, which in turn causes restlessness, agitation, increased heart and respiratory rates, fever, and BP fluctuations, all of which are detrimental to the healing of the animal. A catabolic state is created as a result of increased secretion of catabolic hormones and decreased secretion of anabolic hormones. The net effect the majority of neurohormonal changes produce is an increase in the secretion of catabolic hormones. Hyperglycemia is produced and may persist because of production of glucagon and relative lack of insulin. Lipolytic activity is stimulated by cortisol, catecholamines, and growth hormone. Cardiorespiratory effects of pain include increased cardiac output, vasoconstriction, hypoxemia, and hyperventilation. Protein catabolism is a common occurrence and major concern regarding healing. Pain associated with inflammation causes increase in tissue and blood levels of prostaglandins and cytokines, both of which promote protein catabolism indirectly by increasing the energy expenditure of the body.

Powerful evidence indicates that local anesthetic, sympathetic agonist, and opioid neural blockade may produce a modification of the responses to these physiologic changes. Variable reduction in plasma cortisol, growth hormone, antidiuretic hormone, β-endorphin, aldosterone, epinephrine, norepinephrine, and renin is based on the anesthetic technique and the drugs selected. Prophylactic administration of analgesics blunts the response before it occurs; analgesics administered after perception of pain are not as effective, and higher doses are generally necessary to achieve an equivalent level of analgesia.
RECOGNITION AND ASSESSMENT OF PAIN

Effective pain control can be achieved only when the signs of pain can be assessed effectively, reliably, and regularly. The experience of pain is unique to each individual, which makes pain assessment difficult, especially in traumatized and patients and those in critical condition. Most attempts to assess clinical pain use behavioral observations and interactive variables in addition to assessment of physiologic responses such as heart rate and respiratory rate, BP, and temperature. But many factors can influence the processing and outward projection of pain, including altered environments, species differences, within-species variations (age, breed, sex), and the type, severity, and chronicity of pain.

Within-species differences (age, breed, and sex) further complicate the pain assessment. Most notable is that different breeds of dogs act differently when confronted with pain or fear. Labrador Retrievers tend to be stoic, whereas Greyhounds and teacup breeds tend to react with a heightened state of arousal around even the simplest of procedures (e.g., subcutaneous injections and nail trims). The individual character and temperament of the animal further influences its response. Pediatric and neonatal animals seem to have a lower threshold for pain and anxiety than older animals. In any species, the duration and type of pain make it more (acute) or less (chronic) likely to be expressed or exhibited outwardly. Unfamiliarity with normal behaviors typical of a particular species or breed makes recognition of their pain behaviors and responses impossible.

The definition and recognition of pain in an individual animal are challenging. Because of all the differences discussed, there is no straight line from insult, albeit actual or perceived, to degree of pain experienced. Nor is there a formula for treating “X” type of pain with “Y” type of analgesic. A goal of analgesia is to treat all animals with analgesic drugs and modalities as preemptively as possible and using a multimodal approach. Use analgesic treatment as a tool for diagnosis of pain in the event that recognition of these phenomena is difficult for the patient. In other words, with countless drugs and treatment modalities available, analgesic administration should never be withheld from an animal, even if pain is questionable.

PAIN ASSESSMENT IN DOGS AND CATS

It is important to remember that no behavior or physiologic variable in and of itself is pathognomonic for pain. Interactive and unprovoked (noninteractive) behavior assessments and trending of physiologic data are useful to determine the pain in an individual animal. This is known as pain scoring. Baseline observations, especially observations from someone who has known the animal well, can be helpful to serial behavior and pain assessments. Pain scoring systems have been developed and are reviewed elsewhere; the purposes of these systems are to evaluate and to help guide diagnostic and analgesic treatments (Table 1-15). Regardless of the scale or method used to assess pain, the caregiver must recognize the limitations of the scale. If in doubt regarding whether pain is present or not, analgesic therapy should be used as a diagnostic tool.

BEHAVIORAL SIGNS OF ACUTE PAIN

Classic behaviors associated with pain in dogs and cats include abnormal postures, gaits, movements, and behaviors (Boxes 1-21 and 1-22). Stoicism is apparent apathy and indifference in the presence of pain and is perhaps the “number one” sign of ineffective pain relief or persistent pain in many animals, because so many display apathy and classically normal physiologic parameters even in the face of severe distress, overt suffering, or blatant trauma and illness. The absence of normal behaviors is also a clinical sign of pain, even when abnormal behaviors are not observed.

SIGNS OF CHRONIC PAIN IN CATS AND DOGS

Acute pain results in many of the aforementioned behavioral and physiologic signs, but chronic pain in small animals is an entirely different and distinct entity. Chronic pain is often present in the absence of obvious tissue pathology and changes in physical demeanor.
Again, the severity of the pain may not correlate with the severity of any pathologic condition that may or may not be present. Chronic pain, especially if insidious in onset (cancer, dental, or degenerative pain), may well go unnoticed in dogs and cats, even by family members or intermittent caregivers. Inappetence, lack of activity, panting in a species classically designed to be nose breathers, decreased interest in surroundings, different activity patterns, and abnormal postures are just a few signs of chronic pain in cats and dogs. Cats are a species that in particular are exemplary in their abilities to hide chronic pain. They will exhibit marked familial withdrawal, finding secluded areas where they may remain for days to weeks when they experience acute and chronic pain.

**ACUTE PAIN MANAGEMENT FOR EMERGENT, CRITICAL OR INTENSIVE CARE, AND TRAUMA PATIENTS**

When deciding on a pain management protocol for a patient, always perform a thorough physical examination and include a pain score assessment before injury and pain have occurred, whenever possible. Form a problem list to guide your choice of anesthesia and
analgesia. For example, using a nonsteroidal antiinflammatory drug (NSAID) in an animal with renal failure would not be wise. Remember to account for current medications that the patient may be taking that may augment or interfere with the analgesic or anesthetic drugs. Use multimodal techniques and regional therapy and drugs to target pain at different sites before it occurs. Once a strategy has been decided on, frequently reassess the patient and tailor the protocol to meet each patient’s response and needs.

**Methods to Reduce Pain**

Drug therapy (in particular, opioids with or without α₂-agonists) is a cornerstone for acute pain treatment and surgical preemptive pain prevention. However, local anesthetics delivered epidurally, via perineural or plexus injection or intraarticular or trigger point injection, are also effective analgesics for acute and chronic forms of pain and inflammation. The NSAIDs that classically have been reserved for treatment of more chronic or persistent pain states now are being used regularly for treatment of acute and perioperative pain once BP, coagulation, and gastrointestinal parameters have been normalized.

**Pharmacologic Means to Analgesia: Major Analgesics**

**Opioids**

An opioid is any natural or synthetic drug that is derived from the poppy, which interacts with opiate receptors identified on cell membranes. The drugs from this class constitute the most effective means of controlling acute, perioperative, and chronic pain in human and veterinary medicine (Table 1-16). Their physiologic effects result from the interaction with one or more of at least five endogenous opioid receptors (μ, σ, δ, ε, and κ). μ-Receptor agonists are noted for their ability to produce profound analgesia with mild sedation. These drugs diminish “windup,” the hyperexcitable state resulting from an afferent volley of nociceptive impulses. They elevate the pain threshold and are used preemptively to prevent acute pain.

As a class, opioids cause CNS depression with their intense analgesia. Dose-related respiratory depression reflects diminished response to carbon dioxide levels. Cardiac depression is secondary only to bradycardia and is more likely with certain opioids such as morphine and oxymorphone. Narcotics produce few if any clinically significant cardiovascular effects in dogs and cats; they are considered cardiac soothing or sparing. Because opioids increase intracranial and intraocular pressure, use them more cautiously in patients with severe cranial trauma and or ocular lesions. Opioids directly stimulate the chemoreceptor trigger zone and may cause nausea and vomiting. Most opioids depress the cough reflex via a central mechanism; this may be helpful in patients recovering from endotracheal intubation irritation. A key characteristic of opioids that makes them desirable for use in emergency and critical care situations is their reversibility. Antagonists block or reverse the effect of agonists by combining with receptors and producing minimal or no effects. Administer all reversal agents, such as naloxone and naltrexone, slowly if given intravenously and to effect.

**α₂-Agonists**

As a class of drugs, α₂-agonists warrant special attention because most members of the group possess potent analgesic power at doses that are capable of causing sedation, CNS depression, cardiovascular depression, and even general anesthetic states. Originally developed for antihypertensive use, α₂-agonists quickly have attained sedative analgesic status in veterinary medicine (Table 1-17). Like the opioids, α₂-agonists produce their effects by aggravating α-adrenergic receptors in the CNS and periphery.

**Nonsteroidal Antiinflammatory Drugs**

NSAIDs, which classically have been used to treat chronic pain and inflammation, as well as cardiovascular diseases, have taken on a new role in the treatment of perioperative and acute pain. Recently developed potent oral and parenteral forms of these drugs have compared favorably with and sometimes superiorly to opioids for treatment of acute pain.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Agonist Effects</th>
<th>Dose</th>
<th>Cardiovascular Effects</th>
<th>Disadvantages and Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fentanyl</td>
<td>Pure μ</td>
<td>2 mcg/kg IV bolus</td>
<td>Minimal</td>
<td>Hypoventilation at high doses</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2-8 mcg/kg/hr CRI</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>10-20 mcg/kg/hr CRI (inotropic)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>Partial agonist</td>
<td>0.005-0.03 mg/kg q8h IM, IV, SQ</td>
<td>Minimal</td>
<td>Partial agonist activity, so not as potent as pure μ-agonists</td>
</tr>
<tr>
<td></td>
<td></td>
<td>can be placed topically on oral mucosa in cats</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Butorphanol</td>
<td>Agonist/antagonist</td>
<td>0.2-1.0 mg/kg q2-4h IM, IV, SQ</td>
<td>Minimal</td>
<td>Poor analgesic, adequate sedative if used in combination with an anxiolytic; extremely short duration of action; ceiling effect—more is not better</td>
</tr>
<tr>
<td>Codeine</td>
<td>Pure agonist</td>
<td>1-4 mg/kg PO q6h (dogs)</td>
<td>Minimal</td>
<td>Constipation, dysphoria</td>
</tr>
<tr>
<td>Morphine</td>
<td>Pure agonist</td>
<td>0.1-0.5 mg/kg q4-8h IM, IV, SQ</td>
<td>Minimal, can cause histamine release and hypotension if intravenous dose is high</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.05-0.1 mg/kg/hr IV CRI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oxymorphone</td>
<td>Pure agonist</td>
<td>0.02-0.1 mg/kg q4-12h IM, IV, SQ</td>
<td>Minimal</td>
<td>Noise hypersensitivity, dysphoria, panting when given IV</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>Pure agonist</td>
<td>0.02-0.2 mg/kg q4-12h IM, IV, SQ</td>
<td>Minimal</td>
<td>Panting during intravenous administration, vomiting; hyperthermia in cats</td>
</tr>
<tr>
<td>Tramadol</td>
<td>Agonist activity: μ-receptor agonist Norepinephrine and serotonin reuptake inhibition</td>
<td>1-4 mg/kg PO q6-12h</td>
<td>Cardiovascular effects: minimal</td>
<td>Side effects: agitation, anxiety, tremor, vomiting, constipation to diarrhea rarely</td>
</tr>
</tbody>
</table>

*CRI, Constant rate infusion; IM, intramuscularly; IV, intravenously; PO, orally; SQ, subcutaneously.*
Inflammation and pain (Table 1-18). Nonsteroidal drugs can be used alone, but their best use is that of providing synergistic analgesia with different classes of analgesics (narcotics) or modalities (local, regional, and epidural analgesia, physical therapy, acupuncture).

Most NSAIDS act by inhibition of cyclooxygenase (COX; also known as prostaglandin synthetase), an enzyme that catalyzes the incorporation of molecular oxygen into arachidonic acid to produce mediators of inflammation. There are at least a few forms of COX, among them COX-1, the major constitutive enzyme primarily involved in normal physiologic functions, and COX-2, the enzyme responsible for most of the hyperalgesia and pain responses experienced after tissue injury or trauma. Some NSAIDS inhibit COX and lipoxygenase activity. Most of the currently available oral and parenteral NSAIDS for small
animal medicine and surgery target the COX pathways predominantly, although one (tepoxalin) is thought to inhibit both pathways. Inhibition of COX-1 and COX-2 can inhibit the protective effects and impair platelet aggregation and lead to gastrointestinal ulceration.

There are definite contraindications and relative contraindications for the use of NSAIDs. NSAIDs should not be administered to patients with renal or hepatic insufficiency, dehydration, hypotension, or conditions that are associated with low circulating volume (CHF, unregulated anesthesia, shock), or evidence of ulcerative gastrointestinal disease. *Trauma patients* should be stabilized completely regarding vascular volume, tone, and pressure before the use of NSAIDs. Patients receiving concurrent administration of other NSAIDs or corticosteroids, or those considered to be cushingoid, should be evaluated carefully for an adequate “washout” period (time of clearance of drug from the system) before use of an NSAID or before switching NSAIDs. Patients with coagulopathies, particularly those that are caused by platelet number or function defects or those caused by factor deficiencies, and patients with severe, uncontrolled asthma or other bronchial disease are probably not the patients in which to use NSAIDs. Other advice is that NSAIDs not be administered to pregnant patients or to females attempting to become pregnant because COX-2 induction is necessary for ovulation and subsequent implantation of the embryo. The administration of NSAIDs should be considered only in the well-hydrated, normotensive dog or cat with normal renal or hepatic function, with no hemostatic abnormalities and no concurrent steroid administration.

NSAIDs can be used in many settings of acute and chronic pain and inflammation. Among these are well-stabilized musculoskeletal trauma and surgical pain, osteoarthritis management, meningitis, mastitis, animal bite and other wound healing, mammary or transitional cell carcinoma, epithelial (dental, oral, urethral) inflammation, ophthalmologic procedures, and dermatologic or otic disease. Whereas opioids seem to have an immediate analgesic effect when administered, most NSAIDS will take up to 30 minutes for their effect to be recognized. Therefore most perioperative or acute NSAID use is *part of* a balanced pain management scheme, one that uses narcotics and local anesthetic techniques. NSAIDs are devoid of many of the side effects of narcotic administration—namely, decreased gastrointestinal motility, altered sensorium, nausea and vomiting, and sedation. NSAIDs are also devoid of many of the side effects of steroid administration—namely, suppression of the pituitary adrenal axis.

**Nonsteroidal Antiinflammatory Drugs in Cats**

The toxic effects of salicylates in cats are well documented. Cats are susceptible because of slow clearance and dose-dependent elimination caused by deficient glucuronidation in this species. Because of this, the dose and the administration interval of most commonly used NSAIDs need to be altered in order for these drugs to be used. Cats that have been given canine doses of NSAIDs (twice daily or even once daily repetitively) may show hyperthermia, hemorrhagic or ulcerative gastritis, kidney and liver injury, hyperthermia, respiratory alkalosis, and metabolic acidosis. Acute and chronic toxicities of NSAIDs have been reported in cats, especially after repeat once-daily administration. Ketoprofen, flunixin, aspirin, carprofen, and meloxicam have been administered safely to cats, although like most antibiotics and other medications, they are not approved and licensed for use in cats. An important note, though, is that administration intervals ranging from 48 to 96 hours have been used, and antithrombotic effects often can be achieved at much lower doses than those required to treat fevers and inflammation. I recommend the use of no loading doses, minimum 48-hour administration intervals, and assurance of adequate circulating blood volume, BP, and renal function.

Because many of the NSAIDs are used off-label in cats, it is imperative that the clinician carefully calculate the dose, modify the administration interval, and communicate this information to the client before dispensing the drug. Even drugs that come in liquid form (meloxicam), if administered to cats via box-labeled directions used for dogs, will be given in near toxic doses. To worsen the misunderstanding about dosages for cats, drops from manufacturer’s bottles often are calibrated drops; when these same liquids are transferred into
pharmacy syringes for drop administration, the calibration, of course, is lost, and the animal potentially receives an overdose. A more accurate method of dispensing oral NSAIDs for use in cats and administering them is to calculate the dose in milligrams and determine the exact number of milliliters to administer, rather than using the drop method.

**ANALGESIA: MINOR ANALGESICS**

**Ketamine**

Ketamine classically was considered a dissociative anesthetic, but it also has potent activity as an N-methyl-D-aspartate (NMDA) receptor antagonist. This receptor, located in the CNS, mediates windup and central sensitization (a pathway from acute to chronic pain). Blockade of this receptor with microdoses of ketamine results in the ability to provide body surface, somatic, and skin analgesia with potentially lower doses of opioids and α-agonists. Loading doses of 0.5 to 2 mg/kg are used IV with CRIs of 2 to 20 mcg/kg/min. In and of itself, this drug possesses little to no analgesic ability and indeed in high doses alone often can aggravate, sensitize, or excite the animal in subacute or acute pain.

**Amantadine**

Amantadine is another NMDA blocker that has been used for its antiviral properties and stabilizing effects in Parkinson disease. Amantadine has been used for neuropathic pain in human beings but is available only in an oral form. Suggested starting doses for cats and dogs range from 3 to 5 mg/kg orally (PO) daily. When the drug is given PO or IV, patients are unlikely to develop behavioral or cardiorespiratory effects with ketamine or amantadine.

**Tramadol**

Tramadol is an analgesic that possesses weak opioid μ-agonist activity and norepinephrine and serotonin reuptake inhibition. Tramadol is useful for mild to moderate pain in small animals. Although the parent compound has very weak opioid activity, the metabolites have excellent binding affinity for the μ receptor. Tramadol has been used for perisurgical pain control when given PO in cats and dogs at a dose of 1 to 4 mg/kg PO once to four times daily. Regardless of its affinity for the opioid receptors, the true mechanism of action of tramadol in companion animals remains largely unknown.

**Gabapentin**

Gabapentin is a synthetic analog of γ-aminobutyric acid (GABA). Originally introduced as an antiepileptic drug, the mechanism of action of gabapentin remains somewhat unclear in veterinary medicine. The drug is among a number of commonly used antiepileptic medications used to treat central pain in human beings. The rationale for use is the ability of the drugs to suppress discharge in pathologically altered neurons. Gabapentin does this through calcium channel modulation without binding to glutamate receptors. Chronic, burning, neuropathic, and lancinating pain in small animals responds well to 1 to 10 mg/kg PO daily.

**ADJUNCTIVE ANALGESIC DRUGS**

Local anesthetic agents are the major class of drugs used as peripheral-acting analgesics (Table 1-19). Local anesthetics block the transmission of pain impulses at the peripheral nerve nociceptor regions. Local anesthetics may be used to block peripheral nerves or inhibit nerve “zones” through use of regional techniques. Although all local anesthetics are capable of providing pain relief, agents with a longer duration of action are preferred for pain management purposes. Bupivacaine is an example of a long-acting local anesthetic drug that is used along with lidocaine for long-acting pain relief. A single dose of bupivacaine injected at a local site will provide local anesthesia and analgesia for 6 to 10 hours.
Lidocaine administered as an intravenous CRI (50 to 75 mcg/kg/min in dogs, 1 to 10 mcg/kg/min in cats) is effective in the treatment of chronic neuropathic pain and periosteal and peritoneal pain (e.g., pancreatitis). Mexiletine, an oral sodium channel blocker, can be used as an alternative to injectable lidocaine for provision of background analgesia.

**Anxiolytics and Sedatives**

Many drugs (Table 1-20) are used in combination with opioids, $\alpha_2$-agonists, and ketamine to provide anxiolysis and sedation.

### LOCAL AND REGIONAL TECHNIQUES FOR THE EMERGENT PATIENT

Injection of local anesthetic solution into the connective tissue surrounding a particular nerve produces loss of sensation (sensory blockade) and/or paralysis (motor nerve blockade) in the region supplied by the nerve. Local anesthetics also may be administered epidurally, intrathoracically, intraperitoneally, and intraarticularly. Lidocaine and bupivacaine are the most commonly administered local anesthetics. Lidocaine provides for quick, short-acting sensory and motor impairment. Bupivacaine provides for later-onset, longer-lasting desensitization without motor impairment. Combinations of the two agents diluted with saline are used frequently to provide for quick-onset analgesia that lasts 4 to 6 hours in most patients. Adding a narcotic and/or an $\alpha_2$ agent often maximizes the analgesia and increases the pain-free interval to 8 to 18 hours. Epinephrine-free and preservative-free solutions are recommended. Precision placement of anesthetic close to nerves, roots, or plexuses is improved with the use of a stimulating nerve locator. Cats seem to be more sensitive to the effects of local anesthetics; therefore the lower ends of most dose ranges are used for blockades in this species.

<table>
<thead>
<tr>
<th>Table 1-19</th>
<th>Analgesics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug</td>
<td>Dose</td>
</tr>
<tr>
<td>Amantadine</td>
<td>3 mg/kg PO q24h (dogs and cats)</td>
</tr>
<tr>
<td>Dextromethorphan</td>
<td>1-2 mg/kg PO q6-8h (dogs and cats)</td>
</tr>
<tr>
<td>Gabapentin</td>
<td>1.25-10 mg/kg PO q24h (dogs and cats)</td>
</tr>
<tr>
<td>Tramadol</td>
<td>1-4 mg/kg PO q8-24h</td>
</tr>
</tbody>
</table>

PO, Orally.

<table>
<thead>
<tr>
<th>TABLE 1-20</th>
<th>Commonly Used Analgesic Assistance Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug</td>
<td>Dose</td>
</tr>
<tr>
<td>Acepromazine</td>
<td>0.01-0.03 mg/kg IV, IM, SQ q8-24h; 0.2-0.5 mg/kg PO q12-24h</td>
</tr>
<tr>
<td>Diazepam</td>
<td>0.5-1.0 mg/kg IV in cats and dogs, followed by 0.1-0.2 mg/kg/hr IV CRI</td>
</tr>
<tr>
<td>Midazolam</td>
<td>0.3-0.5 mg/kg IV, IM, SQ in cats and dogs, followed by 0.05 mg/kg/hr IV CRI</td>
</tr>
</tbody>
</table>

**Combination Approach**
Mix with one another and give as CRI at 10 mL/kg/hr.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>CRI Dose Provided</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphine</td>
<td>5 mg in 500 mL</td>
<td>0.1 mg/kg/hr</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>150 mg</td>
<td>3 mg/kg/hr</td>
</tr>
<tr>
<td>Ketamine</td>
<td>100 mg</td>
<td>2 mg/kg/hr</td>
</tr>
</tbody>
</table>

CRI, Constant rate infusion; IM, intramuscularly; IV, intravenously; PO, orally; SQ, subcutaneously.
Unlike most instances of general anesthesia, during which the animal is rendered unconscious and nerve transmission is decreased by virtue of CNS depression, local and regional techniques block the initiation of noxious signals, thereby effectively preventing pain from entering the CNS. This is an effective means not only of preventing initial pain but also of reducing the changes that take place in the dorsal horn of the spinal cord, spinothalamic tracts, limbic and reticular activating centers, and cortex. Frequently the neurohormonal response that is stimulated in pain and stress is blunted as well. Overall, the patient has fewer local and systemic adverse effects of pain, disease processes are minimized, chronic pain states are unlikely, and outcome is improved. Regional techniques are best used as part of an analgesic regimen that consists of their continuous administration, narcotics, \( \alpha \)-agonists, anxiolytics, and good nursing.

**Topical and Infiltrative Blockade**

Lidocaine can be added to sterile lubricant in a one-to-one concentration to provide decreased sensation for urinary catheterization, nasal catheter insertion, minor road burn analgesia, and pyotraumatic dermatitis analgesia. Procainamide is a topical anesthetic useful for corneal or scleral injuries. Local anesthetics can be used to infiltrate areas of damage or surgery through use of long-term continuous drainage catheters and small, portable infusion pumps. This is an effective means of providing days of analgesia for massive surgical or traumatic soft tissue injury. Even without the catheter, incisional or regional soft tissue blocking using a combination of 1 to 2 mg of lidocaine per kilogram and 0.5 to 2 mg of bupivacaine per kilogram diluted with equal volume of saline and 1:9 with sodium bicarbonate is effective for infiltrating large areas of injury.

**Cranial Nerve Blockade**

Administration of local anesthetic drugs around the infraorbital, maxillary, ophthalmic mental, and alveolar nerves can provide excellent analgesia for dental, orofacial, and ophthalmic trauma and surgical procedures. Each nerve may be desensitized by injecting 0.1 to 0.3 mL of a 2\% lidocaine hydrochloride solution and 0.1 to 0.3 mL of a 0.5\% bupivacaine solution using a 1.2 to 2.5-cm, 22- to 25-gauge needle. Precise placement perineurally versus intraneurally (neuroma formation common) is enhanced by using catheters in the foramen versus needle administration. Always perform aspiration before administration to rule out intravascular injection of agents.

**Intrapleural Blockade**

Intrapleural blockade is used to provide analgesia for thoracic, lower cervical, cranial abdominal, and diaphragmatic pain. After aseptic preparation, place a small through-the-needle (20 to 22-gauge) catheter in the thoracic cavity between the seventh and ninth intercostal space on the midlateral aspect of the thorax. Aseptically mix a 0.5- to 1-mg/kg lidocaine and a 0.2 to 0.5-mg/kg bupivacaine dose with volume of saline equal to the volume of bupivacaine, and slowly inject it over a period of 2 to 5 minutes after aspiration to ensure that no intravascular injection occurs. Depending on where the lesion is, position the patient to allow the intrapleural infusion to “coat” the area. Most effective is positioning the patient in dorsal recumbency for several minutes after the block to make sure local anesthetic occupies the paravertebral gutters and hence the spinal nerve roots. The block should be repeated every 3 hours in dogs and every 8 to 12 hours in cats. Secure the catheter to the skin surface for repetitive administration.

**Brachial Plexus Blockade**

Administration of local anesthetic around the brachial plexus provides excellent analgesia for forelimb surgery, particularly distal to the shoulder, and amputations. Nerve locator-guided techniques are much more accurate and successful than blind placement of local anesthetic; however, even the latter is useful.
To administer a brachial plexus blockade, follow this procedure:
1. Aseptically prepare a small area of skin over the point of the shoulder.
2. Insert a 22-gauge, 1½- to 3-inch spinal needle medial to the shoulder joint, axial to the lesser tubercle, and advance it caudally, medial to the body of the scapula, and toward the costochondral junction of the first rib. Aspirate first before injection to make sure that intravenous injection does not occur.
3. Inject one third of the volume of local anesthetic mix, and then slowly withdraw the needle and fan dorsally and ventrally while infusing the remaining fluid.
4. Local anesthetic doses are similar to those for intrapleural blockade.

**Epidural Analgesia and Analgesia**

*Epidural analgesia* refers to the injection of an opioid, a phencyclidine, an α-agonist, or an NSAID into the epidural space. *Epidural anesthesia* refers to the injection of a local anesthetic. In most patients a combination of the two is used. Epidural analgesia and anesthesia are used for acute and chronic surgical pain or traumatically induced pain in the pelvis, tail, perineum, hind limbs, abdomen, and thorax (Table 1-21). Procedures in which epidural analgesia and anesthesia are useful include forelimb and hindlimb amputation, tail or perineal procedures, cesarean sections, diaphragmatic hernia repair, pancreatitis, peritonitis, and intervertebral disk disease. Epidural blocks performed using opioids or bupivacaine will not result in hindlimb paresis or decreased urinary or anal tone (incontinence), unlike lidocaine or mepivacaine epidural blocks. Morphine is one of the most useful opioids for administration in the epidural space because of its slow systemic absorption. Epidural catheters used for the instillation of drugs through CRI or intermittent injection can be placed in dogs and cats. Routinely placed at the lumbosacral junction, these catheters are used with cocktails including preservative-free morphine, bupivacaine, dexmedetomidine, and ketamine. Extremely effective for preventing windup pain in the peritoneal cavity or caudal half of the body, the catheters may be maintained if placed aseptically for 7 to 14 days.

To provide epidural analgesia or anesthesia, follow this procedure:
1. Position the animal in lateral or sternal recumbency.
2. Clip and aseptically scrub over the lumbosacral site.
3. Palpate the craniodorsal-most extent of the wings of the ileum bilaterally and draw an imaginary line through them to envision the spine of L7, located immediately behind the imaginary line.
4. Advance a 20- to 22-gauge, 1½- to 3-inch spinal or epidural needle through the skin just caudal to the spine of L7.
5. The needle will lose resistance as it is introduced into the epidural space. Drop saline into the hub of the needle, and the saline will be pulled into the epidural space as the needle enters.

<table>
<thead>
<tr>
<th><strong>TABLE 1-21</strong> Drugs to Use for Epidural Anesthesia</th>
<th><strong>Drug</strong></th>
<th><strong>Dose</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bupivacaine 0.25%*</td>
<td>0.1-0.3 mg/kg (1 mL/5 kg epidural q4-6h (canine only, not recommended for cats)</td>
</tr>
<tr>
<td></td>
<td>Morphine (Duramorph)**†</td>
<td>0.05-0.1 mg/kg spinal q8h</td>
</tr>
</tbody>
</table>

*Preservative-free solutions should be used, with filtered needles or in-line filters if an epidural catheter with constant rate infusion is used.
†Can be diluted to a total volume of 0.1 to 0.15 mL/kg with sterile saline if advancement of the solution into the thoracic area is desired (forelimb amputation, thoracostomy, diaphragmatic hernia repair).
INTERCOSTAL NERVE BLOCKS
Discrete intercostal nerve blocks can provide effective analgesia for traumatic or postsurgical pain. Identify the area of the injury, and infiltrate three segments on either side of the injury with analgesic.

To perform an intercostal nerve block, follow this procedure:
1. Clip and aseptically scrub the dorsal and ventral third of the chest wall.
2. Palpate the intercostal space as far dorsally as possible.
3. Use a 25-gauge, \(\frac{5}{8}\) -inch needle at the caudolateral aspect of the affected rib segments and those cranial and caudal.
4. Direct the tip of the needle caudally such that the tip of the needle “drops” off of the caudal rib. (This places the needle tip in proximity to the neuromuscular bundle that contains the intercostal nerve that runs in a groove on the caudomedial surface of the rib.)
5. Aspirate to confirm that the drug will not go IV.
6. Inject while slowly withdrawing the needle. Inject 0.5 to 1.0 mL at each site, depending on the size of the animal.

Additional Reading

ACUTE CONDITION IN THE ABDOMEN
An acute condition in the abdomen is defined as the sudden onset of abdominal discomfort or pain caused by a variety of conditions involving intraabdominal organs. Many animals have the primary complaint of lethargy, anorexia, ptyalism, vomiting, retching, diarrhea, hematochezia, crying out, moaning, or abnormal postures. Abnormal postures can include generalized rigidity, walking tenderly or as if “on eggshells,” or a prayer position in which the front limbs are lowered to the ground while the hind end remains standing. In some cases it may be difficult initially to distinguish between true abdominal pain or referred pain from intervertebral disk disease. Rapid progression and decompensation of the patient’s cardiovascular status can lead to stupor, coma, and death in the most extreme cases, making rapid assessment, treatment, and definitive care extremely challenging.

Signalment and History
Often the patient’s signalment and history can increase the index of suspicion for a particular disease process. A thorough history often is overlooked or postponed in the initial stages of resuscitation of the patient with acute abdominal pain. Often, asking the same question in a variety of forms can elicit an answer from the client that may lead to the source of the problem and the reason for the acute abdominal pain. Important questions to ask the client include the following:

- What is your chief complaint or the reason you brought your animal in for emergency treatment?
- When did the signs first start, or when was your animal last normal?
- Do you think that the signs have been the same, getting better, or getting worse?
- Does your animal have any ongoing or past medical problems?
- Have similar signs occurred in the past?
- Does your animal have access to any known toxins, or does he or she run loose unattended?
Has your animal ingested any garbage, compost, or table scraps recently?
Are there any other animals in your household, and are they acting sick or normal?
Has your animal been vaccinated recently?
Has there been any change in your pet's appetite?
Have you noticed any weight loss or weight gain?
Have you noticed any increase or decrease in water consumption or urination?
Does your animal chew on bones or toys?
Have you noticed any toys, socks, underwear, or other items missing from your household?
Is there a possibility that the animal has sustained any trauma, including being hit by a car or kicked by a larger animal or person?
Have you noticed a change in your pet's defecation habits?
Have you seen any vomiting or diarrhea?
What does the vomitus or diarrhea look like?
Does the vomitus occur in relation to eating?
Is there any blood or mucus in the vomitus or diarrhea?
When was the last time your animal vomited or had diarrhea?
When your animal vomits, does it actively retch with abdominal contractions, or is it more passive like regurgitation?
What is the color of the feces? Is it black or red?
Does the vomit smell malodorous like feces?

Immediate Action

As with any other emergency, the clinician must follow the ABCs of therapy, and treat the most life-threatening problems first.

Perform a perfunctory physical examination first.

Briefly observe the patient from a distance, and consider: Are there any abnormal postures? Is there respiratory distress? Is the animal ambulatory, and if so, do you observe any gait abnormalities? Do you observe any ptyalism or attempts to vomit?

Auscult the patient's thorax for crackles, which may signify aspiration pneumonia resulting from vomiting.

Examine the patient's mucous membrane color and capillary refill time, heart rate, heart rhythm, and pulse quality. Many patients in pain have tachycardia, which may or may not be accompanied by dysrhythmias. If a patient's heart rate is inappropriately bradycardic, consider hyperkalemia, in association with hypoadrenocorticism, whipworm infestation, or urinary obstruction or trauma.

Evaluate the patient's hydration status by examining skin turgor, mucous membrane dryness, and whether the eyes appear sunken in their orbits.

Perform a brief neurologic examination, and determine whether the patient is actively having a seizure or whether mental dullness, stupor, coma, or nystagmus is present. Posture and spinal reflexes can assist in making a diagnosis of intervertebral disk disease versus abdominal pain.

Perform a rectal examination to evaluate for the presence of hematochezia or melena.

Examine the abdomen last, in case inciting a painful stimulus precludes you from evaluating other organ systems more thoroughly. Visually inspect the abdomen for the presence of external masses, bruising, or penetrating injuries. Reddish discoloration of the periumbilical area often is associated with the presence of intraabdominal hemorrhage. It may be necessary to shave the fur to inspect the skin and underlying structures visually for bruising and ecchymoses. Auscult the abdomen for the presence or absence of borborygmi to characterize gut sounds. Next, perform percussion and ballottement to evaluate for the presence of a gas-distended viscus or peritoneal effusion. Finally, perform first superficial and then deep palpation of all quadrants of the abdomen, noting abnormal enlargement or masses and whether focal pain is elicited in any one area.

Once the physical examination has been performed, implement initial therapy in the form of analgesia, fluid resuscitation, and antibiotics.
TREATMENT
Treatment for any patient with an acute condition in the abdomen and shock is to treat the underlying cause, maintain tissue oxygen delivery, and prevent end-organ damage and failure. A more complete description of shock and oxygen delivery is given in the section on shock.

ANALGESIA
The administration of analgesic agents to any patient with acute abdominal pain is one of the most important therapies in the initial stages of case management. Table 1-22 lists analgesic drugs for use in the patient with an acute condition in abdomen. Table 1-23 lists analgesic and anxiolytic drugs to avoid in the patient with an acute condition in abdomen.

FLUID RESUSCITATION
Many patients with acute abdominal pain are clinically dehydrated or are in hypovolemic shock because of hemorrhage. Careful titration of intravenous crystalloid and colloid fluids including blood products is necessary based on the patient’s perfusion parameters including heart rate, capillary refill time, BP, urine output, and PCV. Fluid therapy also should be based on the most likely differential diagnoses, with specific fluid types administered according to the primary disease process. In dogs, a shock volume of fluids is calculated based on the total blood volume of 90 mL/kg. In cats, shock fluid rate is based on plasma volume of 44 mL/kg. In most cases any crystalloid fluid can be administered at an initial volume of one fourth of a calculated shock dose and then titrated according to whether the patient’s cardiovascular status responds favorably or not. In cases of an acute condition in the abdomen from known or suspected hypoadrenocorticism, severe whipworm infestation, or urinary tract obstruction or rupture, 0.9% sodium chloride fluid without added potassium is the fluid of choice. When hemorrhage is present, the administration of whole blood or packed RBCs may be indicated if the patient has clinical signs of anemia and shows clinical signs of lethargy, tachypnea, and weakness. Fresh frozen plasma is indicated in cases of hemorrhage resulting from vitamin K antagonist rodenticide intoxication or hepatic failure or in cases of suspected DIC. A more thorough description of fluid therapy is given in the sections on shock and fluid therapy.

ADJUNCTIVE THERAPIES
ANTIBIOTICS
The empiric use of broad-spectrum antibiotics is warranted in cases of suspected sepsis or peritonitis as a cause of acute abdominal pain. Ampicillin sulbactam (22 mg/kg IV q6-8h) and enrofloxacin (10 mg/kg once daily) are the combination treatment of choice to cover

TABLE 1-22 Analgesic Agents for Use in Dogs and Cats with Acute Abdominal Pain

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Butorphanol</td>
<td>0.1-0.2 mg/kg IV (dogs and cats); 0.2-0.4 mg/kg SQ or IM (dogs and cats)</td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>0.005-0.02 mg/kg IV, IM, SQ q6-12h (dogs); 0.005-0.01 mg/kg IV, IM, SQ, q6-12h (cats) (Also can be placed in the mouth for buccal absorption in cats)</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>2 mcg/kg IV bolus, followed by 3-7 mcg/kg/hr CRI (dogs and cats)</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>0.1-0.2 mg/kg SQ, IM, IV (dogs and cats)</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>1-2 mg/kg IV slowly over 2-5 minutes, then 30-50 mcg/kg/min CRI</td>
</tr>
<tr>
<td>Morphine</td>
<td>0.5-1.0 mg/kg SQ, IM; 0.1 mg/kg/hr CRI (dogs); 0.25-0.5 mg/kg SQ, IM; 0.05 mg/kg/hr CRI (cats)</td>
</tr>
</tbody>
</table>

CRI, Constant rate infusion; IM, intramuscularly; IV, intravenously; SQ, subcutaneously.

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gram-negative, gram-positive, aerobic, and anaerobic infections. Alternative therapies include a second-generation cephalosporin such as cefotetan (30 mg/kg IV tid) or cefoxitin (22 mg/kg IV tid) or added anaerobic coverage with metronidazole (10 to 20 mg/kg IV tid).

**Oxygen Supplementation**

Tissue oxygen delivery depends on a number of factors, including arterial oxygen content and cardiac output. If an animal has had vomiting and subsequent aspiration pneumonitis, treatment of hypoxemia with supplemental oxygen in the form of nasal, nasopharyngeal, hood, or transtracheal oxygen administration is important (see Oxygen Supplementation under Emergency Diagnostic and Therapeutic Procedures).

**Diagnostic Procedures**

**Complete Blood Count**

Perform a complete blood count in all cases of acute abdominal pain to determine if life-threatening infection or coagulopathy including DIC is present. In cases of sepsis, infection, or severe nonseptic inflammation, the WBC count may be normal, elevated, or low. Examine a peripheral blood smear for the presence of toxic neutrophils, eosinophils, atypical lymphocytes, nucleated RBCs, platelet estimate, anisocytosis, and blood parasites. A falling PCV in the face of RBC transfusion suggests ongoing hemorrhage.

**Biochemistry Panel**

Perform a biochemistry panel to evaluate organ system function. Azotemia with elevated BUN and creatinine may be associated with prerenal dehydration, impaired renal function, or postrenal obstruction or leakage. The BUN also can be elevated when gastrointestinal

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**TABLE 1-23 Analgesic and Anxiolytic Agents That Are Contraindicated and Should Be Avoided in the Patient with Acute Abdominal Pain**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Potential Risks</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>α-ANTAGONISTS</strong></td>
<td></td>
</tr>
<tr>
<td>Acepromazine</td>
<td>α-Receptor antagonist, hypotension</td>
</tr>
<tr>
<td>Chlorpromazine</td>
<td></td>
</tr>
<tr>
<td><strong>α2-AGONISTS</strong></td>
<td></td>
</tr>
<tr>
<td>Xylazine</td>
<td>α2-Agonist, peripheral vasoconstriction, dose-dependent decrease in cardiac output, hypotension</td>
</tr>
<tr>
<td>Dexmedetomidine</td>
<td></td>
</tr>
<tr>
<td><strong>ANTIPROSTAGLANDINS</strong></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>Decreased renal and gastrointestinal perfusion, gastrointestinal ulceration</td>
</tr>
<tr>
<td>Flunixin meglumine</td>
<td></td>
</tr>
<tr>
<td>Indomethacin</td>
<td></td>
</tr>
<tr>
<td>Phenylbutazone</td>
<td></td>
</tr>
<tr>
<td>Ibuprofen</td>
<td></td>
</tr>
<tr>
<td>Carprofen</td>
<td></td>
</tr>
<tr>
<td>Ketoprofen</td>
<td></td>
</tr>
<tr>
<td>Aminopyrine</td>
<td></td>
</tr>
<tr>
<td>Fluhenamic acid</td>
<td></td>
</tr>
<tr>
<td><strong>GLUCOCORTICOIDs</strong></td>
<td></td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>Decreased renal and gastrointestinal perfusion, gastrointestinal ulceration</td>
</tr>
<tr>
<td>Dexamethasone sodium phosphate</td>
<td></td>
</tr>
<tr>
<td>Hydrocortisone sodium phosphate</td>
<td></td>
</tr>
<tr>
<td>Prednisone</td>
<td></td>
</tr>
<tr>
<td>Prednisolone sodium phosphate</td>
<td></td>
</tr>
<tr>
<td>Methylprednisolone sodium succinate</td>
<td></td>
</tr>
</tbody>
</table>

**Potential Risks**

- Decreased renal and gastrointestinal ulceration
hemorrhage is present. Serum amylase may be elevated with decreased renal function or in cases of pancreatitis. A normal serum amylase level, however, does not rule out pancreatitis as a source of abdominal pain. Serum lipase may be elevated with gastrointestinal inflammation or pancreatitis. Like amylase, a normal serum lipase level does not rule out pancreatitis. Total bilirubin, alkaline phosphatase (ALP), and alanine transaminase may be elevated with primary cholestatic or hepatocellular diseases or from extrahepatic causes including sepsis.

Urinalysis
Obtain a urinalysis via cystocentesis whenever possible, except in cases of suspected pyometra or transitional cell carcinoma. Azotemia in the presence of nonconcentrated (isosthenuric or hyposthenuric) urine suggests primary renal disease. Secondary causes of apparent renal azotemia and lack of concentrating ability also occur in cases of hypoadrenocorticism and gram-negative sepsis. Renal tubular casts may be present in cases of acute renal ischemia or toxic insult to the kidneys. Bacteriuria and pyuria may be present with infection and inflammation. When a urinalysis is obtained via free catch or urethral catheterization, the presence of bacteriuria or pyuria also may be associated with pyometra, vaginitis, or prostatitis or prostatic abscess.

Lactate
Serum lactate is a biochemical indicator of decreased organ perfusion, decreased oxygen delivery or extraction, and end-organ anaerobic glycolysis. Elevated serum lactate greater than 6 mmol/L has been associated with increased morbidity and need for gastric resection in cases of GDV and increased patient morbidity and mortality in other disease processes. A more recent study showed that initial lactate concentrations greater than 9.0 mmol/L or a lactate concentration that does not significantly decrease by 4 mmol/L or 42.5% of initial serum value after fluid resuscitation was associated with an increased risk of complications including death. A rising serum lactate level in the face of adequate fluid resuscitation is a negative prognostic sign.

Glucose
The serum glucose level can sometimes decrease in animals with sepsis, including septic peritonitis. Glucose concentrations of abdominal fluid are significantly lower in animals with septic peritonitis compared with nonseptic peritonitis. In animals with septic peritonitis, comparison of abdominal fluid glucose concentrations with peripheral serum concentrations shows that an abdominal glucose concentration 20 mg/dL or more lower than that of peripheral blood is consistent with septic peritonitis.

Abdominal Radiographs
Obtain abdominal radiographs as one of the first diagnostic tests when deciding whether to pursue medical or surgical management. The presence of GDV, linear foreign body, pneumoperitoneum, pyometra, or splenic torsion warrants immediate surgical intervention. If a loss of abdominal detail occurs because of peritoneal effusion, perform additional diagnostic tests including abdominal paracentesis (abdominocentesis) and abdominal ultrasound to determine the cause of the peritoneal effusion.

Abdominal Ultrasound
Abdominal ultrasonography is often useful in place of or in addition to abdominal radiographs. The sensitivity of abdominal ultrasonography is largely operator-dependent. Indications for immediate surgical intervention include loss of blood flow to an organ, linear bunching or placation of the intestinal tract, intussusception, pancreatic phlegmon or abscess, a fluid-filled uterus suggestive of pyometra, gastrointestinal obstruction, intraluminal gastrointestinal foreign body, dilated bile duct, gallbladder mucocele, or gas within the wall of the stomach or gallbladder (emphysematous cholecystitis). The presence of peritoneal fluid alone does not warrant immediate surgical intervention without cytologic and biochemical evaluation of the fluid present.
Abdominocentesis

See also Abdominal Paracentesis and Diagnostic Peritoneal Lavage.

Abdominal paracentesis (abdominocentesis) often is the deciding factor in whether to perform immediate surgery. Abdominocentesis is a sensitive technique for detecting peritoneal effusion in the presence of more than 6 mL/kg of fluid within the abdominal cavity. Abdominal effusion collected should be saved for bacterial culture and evaluated biochemically and cytologically based on your index of suspicion of the primary disease process. If creatinine, BUN, or potassium is elevated compared with that of serum, uroabdomen is present. Elevated abdominal fluid lipase or amylase compared with serum supports a diagnosis of pancreatitis. Elevated lactate compared with serum lactate or an abdominal fluid glucose level less than 50 mg/dL is highly sensitive and specific for bacterial or septic peritonitis. The presence of bile pigment or bacteria is supportive of bile and septic peritonitis, respectively. Free fibers in abdominal fluid along with clinical signs of abdominal pain strongly support gastrointestinal perforation, and immediate surgical exploration is required.

Diagnostic Peritoneal Lavage

In the event of negative abdominocentesis findings when peritoneal effusion or bile or gastrointestinal perforation is suspected, perform DPL. Peritoneal dialysis kits are commercially available but are often expensive and impractical (see pp. 7 and 488).

Management

Animals that have acute abdominal pain can be divided into three broad categories, depending on the primary cause of pain and the initial definitive treatment (Table 1-24). Some diseases warrant a nonsurgical, medical approach to case management. Other conditions require immediate surgery after rapid stabilization. Other conditions initially can be managed medically until the patient is hemodynamically more stable and then may or may not require surgical intervention at a later time. Specific management of each disease entity is listed under its own subheading.

Exploratory Laparotomy or Celiotomy

Box 1-23 lists specific indications for exploratory laparotomy. The best means to explore the abdominal cavity accurately and thoroughly is to open the abdomen on midline from the level of the xiphoid process caudally to the pubis for full exposure and then to evaluate all organs in every quadrant in a systematic manner. Address specific problems such as gastric or splenic torsion, enteroplication, and foreign body removal, and then copiously lavage the abdomen with warmed sterile saline solution. Suction the saline solution thoroughly from the peritoneal cavity so as to not impair macrophage function. In cases of septic peritonitis, the abdomen may be left open, or a drain may be placed for further suction and lavage. The routine use of antibiotics in irrigation solutions is contraindicated because the antibiotics can irritate the peritoneum and delay healing. When the abdominal cavity is left open, secure sterile laparotomy towels and water-impermeable dressings over the abdominal wound with umbilical tape, and then change these daily or as strike-through occurs. Open abdomen cases are often effusive and require meticulous evaluation and management of electrolyte imbalances and hypoalbuminemia. The abdomen can be closed and/or the abdominal drain removed when the volume of the effusion decreases, when bacteria are no longer present, and when the neutrophils become more healthy in appearance.

Additional Reading

# Conditions That Can Cause Clinical Signs of Acute Abdominal Pain

The following are clinical conditions, patient signalment, common history, physical examination, and characteristic findings of various diagnostic tests. A blank column next to a condition indicates no specific signalment, history, physical examination, or diagnostic test is characteristic for a particular disease process.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Signalment</th>
<th>History and Chief Complaint</th>
<th>Physical Examination Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Abdominal Wall</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hernia</td>
<td>Any</td>
<td>History of trauma, vomiting Abdominal wall swelling Pain, lethargy, anorexia</td>
<td>Abdominal wall swelling, fever, pain</td>
</tr>
<tr>
<td>Abscess</td>
<td>Any</td>
<td>Anorexia, pain, lethargy Abdominal wall swelling</td>
<td>Abdominal wall swelling, fever, pain</td>
</tr>
<tr>
<td>Blunt trauma</td>
<td>Any</td>
<td>History of trauma, lethargy, pain, inappetence</td>
<td>Pain, hematoma or ecchymosis, periumbilical redness or hemorrhage</td>
</tr>
<tr>
<td><strong>Gastrointestinal</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diaphragmatic hernia</td>
<td>Any</td>
<td>History of trauma, vomiting, lethargy, anorexia, respiratory difficulty</td>
<td>Cyanosis, respiratory difficulty, abdominal pain</td>
</tr>
<tr>
<td>Gastroenteritis, bacterial</td>
<td>Any</td>
<td>Vomiting, diarrhea, history of toxin or garbage ingestion</td>
<td>Abdominal pain, increased borborygmi, vomiting, diarrhea, hematohoezia</td>
</tr>
<tr>
<td>Parvovirus, panleukopenia</td>
<td>Young puppy, young kitten</td>
<td>Inadequate vaccination, vomiting, diarrhea, anorexia, lethargy</td>
<td>Dehydration, vomiting, diarrhea, lethargy</td>
</tr>
<tr>
<td>Parasitic</td>
<td>Any</td>
<td>Vomiting, diarrhea, history of worms in feces</td>
<td>Ileus, increased or decreased borborygmi</td>
</tr>
<tr>
<td>Metabolic, hypoadrenocorticism</td>
<td>Any, young female, specific breed predisposition</td>
<td>Waxing and waning, lethargy, vomiting, diarrhea, weakness, anorexia, weight loss, stress</td>
<td>Muscle atrophy, dehydration, melena, hematohoezia, inappropriate bradycardia</td>
</tr>
<tr>
<td>Toxin</td>
<td>Any</td>
<td>History of toxin or garbage exposure</td>
<td>Abdominal pain, lethargy</td>
</tr>
<tr>
<td>Gastric dilatation</td>
<td>Any</td>
<td>History of garlic or food exposure</td>
<td>Distended abdomen, ptyalism</td>
</tr>
<tr>
<td>Gastric dilatation-volvulus</td>
<td>Large breed or deep-chested dog; can occur in any breed</td>
<td>History of unproductive retching</td>
<td>Distended painful tympanic abdomen, cyanosis, respiratory difficulty, ptyalism, retching or unproductive vomiting</td>
</tr>
</tbody>
</table>

**Continued**
<table>
<thead>
<tr>
<th>Condition</th>
<th>Signalment</th>
<th>History and Chief Complaint</th>
<th>Physical Examination Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastric ulcer</td>
<td>Any</td>
<td>Hematemesis, coffee ground vomitus, lethargy, anorexia, melena</td>
<td>Abdominal pain, melena</td>
</tr>
<tr>
<td>Cecal inversion</td>
<td>Any</td>
<td>Vomiting, hematochezia, dyschezia, lethargy Vomiting; history of exposure to string, thread, ribbon</td>
<td>Hematochezia, abdominal pain Abdominal pain, clumped intestines on palpation, string under tongue</td>
</tr>
<tr>
<td>Colonic ulcer, perforation</td>
<td>Any</td>
<td>History of vomiting, inappetence, history of eating foreign object(s)</td>
<td>Abdominal pain, palpate abdominal mass</td>
</tr>
<tr>
<td>Linear foreign body</td>
<td>Any</td>
<td>Vomiting, anorexia, lethargy</td>
<td>Abdominal pain, fever, lethargy, dehydration, palpable mass effect</td>
</tr>
<tr>
<td>Luminal foreign body</td>
<td>Any</td>
<td>Vomiting, diarrhea, lethargy</td>
<td>Abdominal pain, fever, palpable abdominal mass (“sausage”)</td>
</tr>
<tr>
<td>Intestinal or ulcer perforation</td>
<td>Any</td>
<td>Vomiting, diarrhea, lethargy</td>
<td>Palpable mass effect, dry feces on rectal examination</td>
</tr>
<tr>
<td>Intussusception</td>
<td>Any, primarily young dogs/cats</td>
<td>Vomiting, straining to defecate, crying out in pain, anorexia</td>
<td>Abdominal pain, fever, palpable fluid wave, hematochezia with luminal tissue on rectal examination</td>
</tr>
<tr>
<td>Obstruction</td>
<td>Older</td>
<td>Vomiting, straining to defecate, crying out in pain, anorexia</td>
<td>Abdominal pain, fever, palpable fluid wave, hematochezia with luminal tissue on rectal examination</td>
</tr>
<tr>
<td>Vascular ischemia, bowel compromise</td>
<td>Any</td>
<td>Vomiting, diarrhea, hematochezia, anorexia, abdominal pain</td>
<td>Dehydration, painful abdomen, vomiting, icterus</td>
</tr>
<tr>
<td>Liver and Gallbladder</td>
<td></td>
<td></td>
<td>Dehydration, painful abdomen, vomiting, icterus</td>
</tr>
<tr>
<td>Cholangiohepatitis, hepatitis</td>
<td>Any</td>
<td>Anorexia, vomiting, pain, lethargy, icterus</td>
<td>Dehydration, painful abdomen, vomiting, icterus</td>
</tr>
<tr>
<td>Cholecystitis, emphysematous cholecystitis, gallbladder mucocele</td>
<td>Any</td>
<td>Anorexia, vomiting, pain, lethargy</td>
<td>Dehydration, painful abdomen, vomiting, icterus</td>
</tr>
<tr>
<td>Biliary rupture, bile peritonitis</td>
<td>Any</td>
<td>History of trauma, pain, lethargy</td>
<td>Dehydration, painful abdomen, peritonitis, vomiting, anorexia icterus, fever</td>
</tr>
<tr>
<td>Biliary obstruction</td>
<td>Any</td>
<td>Anorexia, vomiting, pain, lethargy</td>
<td>Dehydration, painful abdomen, vomiting, icterus</td>
</tr>
<tr>
<td>Hepatic abscess</td>
<td>Any</td>
<td>Anorexia, vomiting, pain, lethargy</td>
<td>Dehydration, painful abdomen, vomiting, icterus</td>
</tr>
<tr>
<td>Condition</td>
<td>Relevant Information</td>
<td>Signs and Symptoms</td>
<td></td>
</tr>
<tr>
<td>--------------------------------</td>
<td>--------------------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Hepatic torsion</td>
<td>Any</td>
<td>Anorexia, vomiting, pain, lethargy, vomiting, pain, vomiting, pain, lethargy</td>
<td></td>
</tr>
<tr>
<td>Hepatic neoplasia</td>
<td>Any; older animals</td>
<td>Anorexia, vomiting, pain, lethargy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dehydration, painful abdomen, fever, painful abdomen, vomiting, fever, dehydration, seizures</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Painful abdomen, vomitus, fever, dehydration, seizures</td>
<td></td>
</tr>
<tr>
<td>Pancreas</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>Any, some breed</td>
<td>Anorexia, vomiting, pain, lethargy</td>
<td></td>
</tr>
<tr>
<td>Pancreatic abscess</td>
<td>History of eating fatty meal</td>
<td>Dehydration, abdominal pain, vomitus, alopecia</td>
<td></td>
</tr>
<tr>
<td>Pancreatic pseudocyst or mucocèle</td>
<td></td>
<td>Palpable abdominal mass</td>
<td></td>
</tr>
<tr>
<td>Pancreatic neoplasia</td>
<td>Any; older animals</td>
<td>Anorexia, vomiting, pain, lethargy, weight loss, truncal alopecia</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dehydration, abdominal pain, vomitus, alopecia, palpable abdominal mass</td>
<td></td>
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<tr>
<td>Spleen</td>
<td></td>
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</tr>
<tr>
<td>Splenic torsion</td>
<td>Any</td>
<td>Acute pain, vomiting, lethargy</td>
<td></td>
</tr>
<tr>
<td>Splenic mass</td>
<td>Any; older animals</td>
<td>Acute pain, lethargy, collapse</td>
<td></td>
</tr>
<tr>
<td>Splenic infarction</td>
<td>Any</td>
<td>Acute pain, lethargy, collapse</td>
<td></td>
</tr>
<tr>
<td>Traumatic splenic laceration</td>
<td>History of trauma</td>
<td>History of trauma</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pale mucous membranes, decompensatory shock, palpable abdominal mass and splenomegaly, abdominal pain</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Abdominal pain,ballotable fluid wave, anemia, compensatory or decompensatory shock</td>
<td></td>
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<tr>
<td>Genitourinary</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Mastitis</td>
<td>Female</td>
<td>History of lactation</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Abdominal pain; fever; lethargy; anorexia; painful. swollen, sometimes abscessed mammary glands; discolored milk</td>
<td></td>
</tr>
<tr>
<td>Penis fracture</td>
<td>Male dogs</td>
<td>History of trauma, history of traumatic breeding</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Painful abdomen and penis</td>
<td></td>
</tr>
<tr>
<td>Paraphimosis</td>
<td>Male dogs</td>
<td>Persistent erection</td>
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<tr>
<td></td>
<td></td>
<td>Swollen penis outside of prepuce</td>
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<tr>
<td>Prostate</td>
<td></td>
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</tr>
<tr>
<td>Prostatitis</td>
<td>Male dogs</td>
<td>Straining to defecate</td>
<td></td>
</tr>
<tr>
<td>Prostatic abscess</td>
<td>Older male dogs</td>
<td>Straining to defecate, pain, lethargy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Painful enlarged prostate on rectal palpation, fever</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Painful enlarged prostate on rectal palpation</td>
<td></td>
</tr>
</tbody>
</table>

Continued
<table>
<thead>
<tr>
<th>Condition</th>
<th>Signalment</th>
<th>History and Chief Complaint</th>
<th>Physical Examination Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prostatic neoplasia</td>
<td>Older male dogs</td>
<td>Straining to defecate</td>
<td>Enlarged prostate on rectal examination</td>
</tr>
<tr>
<td>Renal acute nephritis</td>
<td>Any</td>
<td>Lethargy, vomiting, anorexia</td>
<td>Painful abdomen, dehydration, fever</td>
</tr>
<tr>
<td>Pyelonephritis</td>
<td>Any</td>
<td>Lethargy, polyuria and polydipsia (PU/PD), vomiting, anorexia</td>
<td>Painful abdomen, fever</td>
</tr>
<tr>
<td>Renal neoplasia</td>
<td>Any older animal</td>
<td>Anorexia, vomiting, lethargy, weight loss</td>
<td>Painful abdomen, fever, cachexia, palpable abdominal mass</td>
</tr>
<tr>
<td>Renal abscess</td>
<td>Any</td>
<td>Anorexia, vomiting, lethargy, weight loss</td>
<td>Painful abdomen, fever, cachexia, palpable abdominal mass</td>
</tr>
<tr>
<td>Renal infarct, thrombus</td>
<td>Any</td>
<td>Lethargy, PU/PD, vomiting</td>
<td>Painful abdomen, fever</td>
</tr>
<tr>
<td>Renolithiasis</td>
<td>Any</td>
<td>Lethargy, PU/PD, vomiting</td>
<td>Painful abdomen, fever</td>
</tr>
<tr>
<td>Ureteral obstruction</td>
<td>Any</td>
<td>Lethargy, PU/PD, vomiting</td>
<td>Painful abdomen, fever, dehydration</td>
</tr>
<tr>
<td>Ureteral rupture</td>
<td>Any</td>
<td>Lethargy, PU/PD, vomiting</td>
<td>Painful abdomen, fever, dehydration</td>
</tr>
<tr>
<td>Urethral obstruction</td>
<td>Any</td>
<td>Lethargy, PU/PD, vomiting, straining to urinate</td>
<td>Painful abdomen, dehydration, vomitus, painful distended nonexpressible urinary bladder</td>
</tr>
<tr>
<td>Urethral tear or rupture</td>
<td>Any</td>
<td>Lethargy, PU/PD, vomiting, history of trauma</td>
<td>Painful abdomen, dehydration, vomitus, fever</td>
</tr>
<tr>
<td>Urinary bladder neoplasia</td>
<td>Any; older animals</td>
<td>Stranguria, hematuria, weight loss, pollakiuria</td>
<td>Thickened urethra may be palpable on rectal examination</td>
</tr>
<tr>
<td>Testicles</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Testicular torsion</td>
<td>Intact male dogs</td>
<td>Pain, chewing or looking at back end</td>
<td>Swollen painful testicle, fever</td>
</tr>
<tr>
<td>Uterus and ovaries</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uterine torsion</td>
<td>Intact gravid females</td>
<td>Acute collapse, vaginal discharge, history of breeding</td>
<td>Decompensatory shock, vaginal discharge</td>
</tr>
<tr>
<td>Pyometra</td>
<td>Intact females</td>
<td>Recent heat cycle, PU/PD, vomiting, diarrhea, lethargy, vaginal discharge</td>
<td>Dehydration, soft tissue mass in caudal abdomen, vaginal discharge, fever</td>
</tr>
<tr>
<td>Uterine rupture</td>
<td>Intact gravid females</td>
<td>History of recent whelping or queening, lethargy, acute collapse</td>
<td>Abdominal pain, vaginal discharge, decompensatory shock</td>
</tr>
<tr>
<td>Discospondylitis</td>
<td>Any</td>
<td>History of pain, lethargy, anorexia</td>
<td>Painful spine, fever</td>
</tr>
<tr>
<td>Condition</td>
<td>History of possible exposure, pain, acute collapse, vomiting</td>
<td>Recumbency, muscle fasciculations, pain, vomiting, fever, collapse</td>
<td></td>
</tr>
<tr>
<td>---------------------------------------------</td>
<td>-------------------------------------------------------------</td>
<td>------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Black widow spider</td>
<td>History of possible exposure, pain, necrotizing bull's-eye ulcer formation</td>
<td>Ulcer, pain, fever, granulomatous lesion</td>
<td></td>
</tr>
<tr>
<td>Brown recluse spider</td>
<td>Acute paresis or paralysis</td>
<td>Paresis or paralysis, spinal pain</td>
<td></td>
</tr>
<tr>
<td>Intervertebral disk disease</td>
<td>Acute pain, lethargy, anorexia</td>
<td>Fever, extreme pain</td>
<td></td>
</tr>
<tr>
<td>Meningitis</td>
<td>Acute pain, lethargy, anorexia</td>
<td>Fever, extreme pain</td>
<td></td>
</tr>
<tr>
<td>Myositis</td>
<td>Acute pain, lethargy, anorexia</td>
<td>Decompensatory shock, palpable abdominal mass</td>
<td></td>
</tr>
<tr>
<td>Neoplasia</td>
<td>Vomiting, anorexia, lethargy, pain of trauma or penetrating abdominal injury</td>
<td>Pain, fever, palpable foreign object</td>
<td></td>
</tr>
<tr>
<td>Peritonitis</td>
<td>Pain, anorexia, lethargy</td>
<td>Pain, lethargy, dehydration, fever</td>
<td></td>
</tr>
</tbody>
</table>

### Diagnostic Tests

- **Lack of contiguity of body wall**
  - Surgical (immediate)
- **Lack of contiguity of body wall**
  - Surgical (immediate)
- **Hemoadenom on abdominocentesis or DPL**
  - Medical
- **Radiographic evidence of abdominal organs in thorax, may require contrast celiotomy**
  - Medical unless stomach is in thorax
- **Ileus on radiographs, WBCs in feces**
  - Medical
- **Parvovirus CITE test positive on feces, leukopenia or neutropenia**
  - Medical
- **Parasite oocysts or parasites in feces**
  - Medical
- **Atrial standstill on ECG, hyperkalemia, hyponatremia, hypocholesterolemia, hypoglycemia, hyperphosphatemia, azotemia, normal WBC count and differential, positive ethylene glycol**
  - Medical
- **Calcium oxalate dihydrate crystals, UA “halo sign” indicates hyperechoic renal cortex on US**
  - Medical
- **Soft tissue density, food with gastric dilation on radiographs**
  - Medical
- **Dorsal and cranial displacement of pylorus with dilation of gastric fundus on right lateral radiograph, premature ventricular contractions on ECG, elevated lactate**
  - Surgical (immediate)

*Continued*
<table>
<thead>
<tr>
<th>Condition</th>
<th>Signalment</th>
<th>History and Chief Complaint</th>
<th>Physical Examination Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regenerative anemia, melena, loss of abdominal detail on radiographs if perforation present</td>
<td>Medical unless perforation present</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loss of detail on radiographs if perforation and peritonitis present</td>
<td>Medical unless perforation present</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C-shaped abnormal gas pattern with plication on radiographs</td>
<td>Surgical (immediate)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dilatation of bowel cranial to foreign object, radiopaque object in stomach or intestines, hypochloremic metabolic acidosis on bloodwork if pyloric outflow obstruction is present</td>
<td>Surgical (immediate)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elevated or decreased WBCs; foreign material, WBCs, and bacteria on abdominal fluid, elevated lactate and decreased glucose on abdominal fluid assessment</td>
<td>Medical unless perforation present</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Target-shaped soft tissue density on abdominal US, soft tissue density with gas dilation cranially on abdominal radiographs</td>
<td>Surgical (immediate); medical management of primary cause</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Colonic distension with hard feces on radiographs</td>
<td>Medical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increased or decreased WBCs, septic abdominal effusion</td>
<td>Surgical (immediate)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elevated T Bili, ALT, ALP, and WBCs; hypoechoic hepatic parenchyma on US; hepatomegaly</td>
<td>Medical after biopsy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elevated T Bili, ALT, ALP, and WBCs; hyperechoic foci in gallbladder or sludge on US; free gas in wall of gall bladder</td>
<td>Surgical (immediate)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abdominal effusion, bile pigment in effusion</td>
<td>Surgical (immediate)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elevated T Bili, ALP, ALT</td>
<td>Surgical (immediate)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elevated or decreased WBCs; elevated T Bili, ALP, and ALT; free gas in hepatic parenchyma on RADS; hypoechoic mass with hyperechoic material in hepatic parenchyma on US</td>
<td>Surgical (immediate)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heteroechoic liver with hyperechoic center on US</td>
<td>Surgical (immediate)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mixed echogenic mass on US; soft tissue mass density on radiographs; elevated ALP, ALT, T Bili; hypoglycemia</td>
<td>Surgical (immediate or delayed)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elevated T Bili, ALP, ALT, amylase, and/or lipase; elevated or decreased WBCs; hypocalcemia; focal loss of detail in right cranial quadrant on radiographs; hypoechoic to hyperechoic pancreas with hyperechoic peripancreatic fat on US; abdominal and/or pleural effusion on radiographs and US</td>
<td>Medical in most cases unless abscess or phlegmon is present</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pancreatic soft tissue mass effect on radiographs and US elevated amylase and lipase, hypoglycemia, elevated serum insulin</td>
<td>Surgical if mass identified, otherwise medical management of hypoglycemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Splenomegaly on radiographs, hyperechoic spleen with no blood flow on US</td>
<td>Surgical (immediate)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Soft tissue mass effect and loss of abdominal detail on radiographs, cavitated mass with abdominal effusion on US</td>
<td>Surgical (immediate)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Condition</td>
<td>Management</td>
<td></td>
<td></td>
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<tr>
<td>--------------------------------------------------------------------------</td>
<td>-----------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperechoic spleen with no blood flow on abdominal US, abdominal effusion, thrombocytopenia</td>
<td>Surgical (immediate)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loss of abdominal detail on radiographs, peritoneal effusion on US, hemoabdomen on abdominocentesis</td>
<td>Medical unless refractory hypotension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnosis based primarily on clinical signs</td>
<td>Medical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fracture of the os penis on radiographs</td>
<td>Largely medical unless urethral tear</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnosis based primarily on clinical signs</td>
<td>Medical, although prepuce may need to be incised to allow replacement of penis into sheath</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prostatomegaly on radiographs and US, hypoechoic prostate on US, pyuria and bacteriuria on UA</td>
<td>Medical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prostatomegaly on radiographs and US, hypoechoic to hyperechoic prostate on US, bacteriuria and pyuria on UA</td>
<td>Surgical (delayed)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prostatomegaly on radiographs and US, prostatic mineralization on radiographs and US</td>
<td>Medical, surgical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypoechoic kidneys on US, pyuria on UA, elevated WBCs, azotemia</td>
<td>Medical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pyuria, bacteriuria on UA</td>
<td>Medical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pyelectasia in abdominal US, azotemia</td>
<td>Surgical (immediate)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Renomegaly on radiographs, azotemia</td>
<td>Surgical (immediate)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Renal mass on US, renomegaly on radiographs</td>
<td>Surgical (delayed)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calculi in renal pelvis on radiographs and US, azotemia</td>
<td>Medical unless both kidneys affected</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ureteral calculi on radiographs and US, hydronephrosis, azotemia</td>
<td>Medical unless both kidneys affected</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ureteral calculi on radiographs and US, hydronephrosis, fluid or soft tissue density on US, azotemia</td>
<td>Surgical (delayed until electrolyte stabilization)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnosis largely based on physical examination findings</td>
<td>Medical unless cannot pass urethral catheter</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Azotemia, no peritoneal effusion, lack of urine output or outflow with ureteral catheterization, double-contrast cystourethrogram indicated</td>
<td>Surgical (delayed until electrolyte stabilization)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transitional cellular casts on UA, hematuria, mass effect or thickened irregular urethra on US or cystourethrogram</td>
<td>Surgical and medical management</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypoechoic swollen testicle on testicular US</td>
<td>Surgical (immediate)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fluid - or gas-filled tubular structure on abdominal US or abdominal radiographs</td>
<td>Surgical (immediate)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Continued**
<table>
<thead>
<tr>
<th>Condition</th>
<th>Signalment</th>
<th>History and Chief Complaint</th>
<th>Physical Examination Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soft tissue tubular structure on radiographs, fluid-filled uterus on US, azotemia, isosthenuria, elevated T Bili, ALT, ALP</td>
<td></td>
<td></td>
<td>Surgical (immediate)</td>
</tr>
<tr>
<td>Pneumoperitoneum on radiographs, abdominal effusion, degenerative neutrophils and bacteria on abdominal fluid cytology</td>
<td></td>
<td></td>
<td>Surgical (immediate)</td>
</tr>
<tr>
<td>Elevated WBCs, increased density to bony endplates on radiographs</td>
<td></td>
<td></td>
<td>Medical</td>
</tr>
<tr>
<td>Hypocalcemia, markedly elevated CK</td>
<td></td>
<td></td>
<td>Medical</td>
</tr>
<tr>
<td>Diagnosis of exclusion</td>
<td></td>
<td></td>
<td>Medical</td>
</tr>
<tr>
<td>Decreased intervertebral disk space on radiographs, evidence of disk herniation and cord compression on myelogram or MRI scan</td>
<td></td>
<td></td>
<td>Surgical (immediate)</td>
</tr>
<tr>
<td>Elevated protein and neutrophils on CSF analysis</td>
<td></td>
<td></td>
<td>Medical</td>
</tr>
<tr>
<td>Muscle biopsy</td>
<td></td>
<td></td>
<td>Medical</td>
</tr>
<tr>
<td>Mass effect and loss of abdominal detail on radiographs, mass and peritoneal effusion on US, anemia</td>
<td></td>
<td></td>
<td>Surgical (immediate)</td>
</tr>
<tr>
<td>Degenerative neutrophils, plant material, bile pigment, or bacteria on abdominal fluid cytology; peritoneal effusion on US, loss of abdominal detail on radiographs, elevated or decreased WBCs</td>
<td></td>
<td></td>
<td>Surgical (immediate)</td>
</tr>
<tr>
<td>Elevated or decreased WBCs, retroperitoneal mass effect on radiographs or US</td>
<td></td>
<td></td>
<td>Surgical (immediate)</td>
</tr>
</tbody>
</table>

ALP, Alkaline phosphatase; ALT, alanine aminotransferase; CK, creatine kinase; CSF, cerebrospinal fluid; DPL, diagnostic peritoneal lavage; ECG, electrocardiogram; MRI, magnetic resonance imaging; SQ, subcutaneous; RADS, radiographs; T bili, total bilirubin; UA, urinalysis; US, ultrasound; WBC, white blood cell.


ANAPHYLACTIC (ANAPHYLACTOID) SHOCK

Anaphylactic shock occurs as an immediate hypersensitivity reaction to a variety of inciting stimuli (Box 1-24). In animals, the most naturally occurring anaphylactic reaction results from wasp or bee stings. Most other reactions occur as a result of an abnormal sensitivity to items used in making medical diagnoses or treatment.

<table>
<thead>
<tr>
<th>BOX 1-23 INDICATIONS TO PERFORM EXPLORATORY LAPAROTOMY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penetrating abdominal injury</td>
</tr>
<tr>
<td>Presence of bacterial on abdominal fluid</td>
</tr>
<tr>
<td>Presence of greater than 500 mcL of white blood cells in lavage fluid effluent, particularly if degenerative neutrophils are present</td>
</tr>
<tr>
<td>Presence of food or plant material in lavage fluid</td>
</tr>
<tr>
<td>Presence of creatinine, blood urea nitrogen, potassium, or lactate in abdominal fluid greater than that in peripheral blood</td>
</tr>
<tr>
<td>Presence of glucose in abdominal fluid less than 50 mg/dL or less than in peripheral blood</td>
</tr>
<tr>
<td>Presence of bilirubin in lavage fluid</td>
</tr>
<tr>
<td>Pneumoperitoneum on radiographs</td>
</tr>
<tr>
<td>Continued evidence of peritoneal irritation</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>BOX 1-24 INCITING ALLERGENS THAT CAN CAUSE ANAPHYLACTOID REACTIONS, ANGIONEUROTIC EDEMA, OR URTICARIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Adrenocorticotropic hormone</td>
</tr>
<tr>
<td>• Antihistamines, antitoxins (foreign serums)</td>
</tr>
<tr>
<td>• Benzocaine</td>
</tr>
<tr>
<td>• Chloramphenicol</td>
</tr>
<tr>
<td>• Erythromycin</td>
</tr>
<tr>
<td>• Food</td>
</tr>
<tr>
<td>• Heparin</td>
</tr>
<tr>
<td>• Hypersensitization and skin testing</td>
</tr>
<tr>
<td>• Insect stings</td>
</tr>
<tr>
<td>• Insulin</td>
</tr>
<tr>
<td>• Iodinated contrast media</td>
</tr>
<tr>
<td>• Lidocaine</td>
</tr>
<tr>
<td>• Oxytocin</td>
</tr>
<tr>
<td>• Penicillin</td>
</tr>
<tr>
<td>• Penicillinase</td>
</tr>
<tr>
<td>• Procaine</td>
</tr>
<tr>
<td>• Salicylates</td>
</tr>
<tr>
<td>• Streptomyacin</td>
</tr>
<tr>
<td>• Tetracaine</td>
</tr>
<tr>
<td>• Tetracycline</td>
</tr>
<tr>
<td>• Tranquilizers</td>
</tr>
<tr>
<td>• Vaccines</td>
</tr>
<tr>
<td>• Vancomycin</td>
</tr>
<tr>
<td>• Vitamins</td>
</tr>
</tbody>
</table>
During an anaphylactic reaction, activation of C5a and the complement system results in vascular smooth muscle dilation and the release of a cascade of inflammatory mediators, including histamine, slow-reacting substance of anaphylaxis, serotonin, heparin, acetylcholine, and bradykinin.

Clinical signs associated with anaphylaxis differ between dogs and cats. In dogs, clinical signs may include restlessness, vomiting, diarrhea, hematochezia, circulatory collapse, coma, and death. In cats, clinical signs often are associated with respiratory system abnormalities. Clinical signs may include ptyalism, pruritus, vomiting, incoordination, bronchoconstriction, pulmonary edema and hemorrhage, laryngeal edema, collapse, and death.

**Immediate Action and Treatment**

The most important steps to remember in any emergency are the ABCs of airway, breathing, and circulation. First, establish an airway through endotracheal intubation or emergency tracheostomy, if necessary. Concurrently, an assistant should establish vascular or intraosseous access to administer drugs and fluids (Box 1-25).

**Differential Diagnosis**

Differential diagnoses to consider for anaphylactic shock include the following:
- Any cause of vomiting, diarrhea
- Toxin
- Internal hemorrhage
- CHF
- Lower airway disease
- Upper airway obstruction

**Management**

The patient should be hospitalized until complete resolution of clinical signs. After initial stabilization and treatment, it is important to maintain vascular access and continue intravenous fluid therapy until the patient is no longer hypotensive and vomiting and diarrhea have resolved. In cases of fulminant pulmonary hemorrhage and edema, administer supplemental oxygen until the patient is no longer hypoxemic or orthopneic on room air. Normalize and maintain BP using positive inotropes (dobutamine, 3 to 10 mcg/kg/min CRI) or pressors (dopamine, 3 to 10 mcg/kg/min IV CRI; see discussion of shock). If blood-tinged vomitus or diarrhea has been observed, administer antibiotics to decrease the risk of bacterial translocation and sepsis (cefoxitin, 22 mg/kg IV tid; metronidazole, 10 mg/kg IV tid). Also consider using gastroprotectant drugs (famotidine, 0.5 to 1.0 mg/kg IV; ranitidine, 0.5 to 2.0 mg/kg PO, IV, IM bid; sucralfate, 0.25 to 1.0 g PO tid; omeprazole, 0.7 to 1.0 mg/kg PO sid).

**ANGIONEUROTIC EDEMA AND URTICARIA**

A second and less serious form of allergic reaction manifests as angioneurotic edema and urticaria. In most cases, clinical signs develop within 20 minutes of an inciting allergen. Although this type of reaction causes patient discomfort, it rarely poses a life-threatening threat.

### Box 1-25 Immediate Treatment of Anaphylactic Shock

1. Administer epinephrine (0.01 mL/kg 1:1000 epinephrine IV or IO). If vascular access cannot be established, administer the epinephrine IM (0.2 to 0.5 mL/kg). Repeat epinephrine dose in 10 to 15 minutes if clinical signs are not resolving.
2. Start intravenous crystalloid fluids (Normosol-R, Plasma-Lyte M, lactated Ringer’s solution) at one fourth of a calculated shock dose (90 mL/kg/hr in dogs, 44 mL/kg/hr in cats).
3. Administer a short-acting steroid (dexamethasone sodium phosphate [Dex SP], 0.25 to 1.0 mg/kg IV).
4. Administer antihistamines. Administer diphenhydramine (2 mg/kg IM BID as needed). Administer famotidine (0.5 to 1.0 mg/kg IV).

*IM, Intramuscularly; IO, intraosseous; IV, intravenously.*
problem. Most animals have mild to severe swelling of the maxilla and periorbital regions. The facial edema also may be accompanied by mild to severe generalized urticaria. Some animals may paw at the face, rub at the eyes, or have vomiting or diarrhea.

**Immediate Action and Treatment**

The treatment for angioneurotic edema involves suppressing the immune response by administration of short-acting glucocorticoid drugs and blocking the actions of histamine by the synergistic use of histamine-1 (H₁) and H₂ receptor blockers (Box 1-26).

**Differential Diagnosis**

In some cases, the inciting cause is a known recent vaccination or insect sting. Many times, however, the inciting cause is not known and is likely an exposure to a stinging insect or arachnid. Differential diagnoses for acute facial swelling and/or urticaria include acetaminophen toxicity (cats), anterior caval syndrome, lymphadenitis, vasculitis, hypoalbuminemia, and contact dermatitis.

**Management**

Observe animals that have demonstrated angioneurotic edema for a minimum of 20 to 30 minutes after injection of the short-acting glucocorticoids and antihistamines. Monitor BP to make sure that the patient does not have concurrent anaphylaxis and hypotension. After partial or complete resolution of clinical signs, the animal can be discharged to its owner for observation. In dogs, mild vomiting or diarrhea may occur within 1 to 2 days after this type of reaction. Wherever possible, exposure to the inciting allergen should be avoided.

**Additional Reading**


**Anesthetic Complications and Emergencies**

Complications observed while a patient is under anesthesia can be divided into two broad categories: (1) those related to equipment malfunction or human error and (2) the patient’s physiologic response to the cardiorespiratory effects of the anesthetic drugs. Careful observation of the patient and familiarity with anesthetic equipment, drug protocols, and monitoring equipment are necessary for the safest anesthesia to occur. Despite this, however, anesthetic-related complications are frequent and need to be recognized and treated appropriately.

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**BOX 1-26 IMMUNE RESPONSE SUPPRESSION AGENTS FOR ANGIONEUROTIC EDEMA**

- Administer short-acting glucocorticoid:
  - Dexamethasone sodium phosphate (Dex SP), 0.25 to 1.0 mg/kg IV, SQ, IM
- Administer antihistamines:
  - Diphenhydramine, 2 mg/kg IM BID as needed
  - Famotidine, 0.5 to 1.0 mg/kg IV, SQ, IM

*IM, Intramuscularly; IV, intravenously; SQ, subcutaneously.*
Many anesthetic drugs have a dose-dependent depressive effect on the respiratory system and cause a decrease in respiratory rate and tidal volume, leading to hypoventilation. Respiratory rate alone is not a reliable indicator of the patient’s oxygenation and ventilatory status. The respiratory tidal volume can be measured with a Wright respirometer. Perform pulse oximetry and capnography as noninvasive measures of the patient’s oxygenation and ventilation.

Ventilation can be impaired as a result of anesthetic drugs, patient position, pneumothorax, pleural effusion (chylothorax, hemothorax, pyothorax), equipment malfunction, rebreathing of carbon dioxide, thoracic wall injury, or alveolar fluid (pulmonary edema, hemorrhage, or pneumonia). Problems such as a diaphragmatic hernia, GDV, or gravid uterus can impede diaphragmatic excursions once the patient is placed on its back and can lead to impaired ventilation. The work of breathing also may be increased because of increased resistance of the anesthesia circuit and increased dead space ventilation. This is particularly important in small toy breeds.

Clinical signs of inadequate ventilation and respiratory complications include abnormal respiratory pattern, sudden changes in heart rate, cardiac dysrhythmias, cyanosis, and cardiopulmonary arrest. End-tidal carbon dioxide, or capnography, gives a graphic display of adequacy of ventilation. Rapid decreases in end-tidal carbon dioxide can be caused by disconnection or obstruction of the patient’s endotracheal tube or poor perfusion, namely, cardiopulmonary arrest (see Capnometry [End-Tidal Carbon Dioxide Monitoring]).

Postoperatively, hypoventilation can occur because of the residual effects of the anesthetic drugs, hypothermia, overventilation during intraoperative support, surgical techniques that compromise ventilation (thoracotomy, cervical disk surgery, atlantooccipital stabilization), postoperative bandaging of the abdomen or thorax, ventilatory muscle fatigue, or injury to the CNS.

Cardiac output is a function of heart rate and stroke volume. Factors that influence stroke volume include vascular and cardiac preload, cardiac afterload, and cardiac contractility. The patient’s cardiac output can be affected adversely by the negative inotropic and chronotropic and vasodilatory effects of anesthetic drugs, all leading to hypotension. Bradycardia, tachycardia, cardiac dysrhythmias, and vascular dilation can lead to hypotension and inadequate organ perfusion. Table 1-25 lists the normal heart rate and BP in dogs and cats.

**Bradycardia**

Bradycardia is defined as a heart rate below normal values. Many anesthetic drugs can cause bradycardia. Causes of bradycardia include use of narcotics or $\alpha_2$-agonist drugs, deep plane of anesthesia, increased vagal tone, hypothermia, and hypoxia. Table 1-26 lists the causes of bradycardia and the necessary immediate action or treatment.

<table>
<thead>
<tr>
<th>Species</th>
<th>Normal Heart Rate (beats/min)</th>
<th>Normal Blood Pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dogs (large)</td>
<td>60-100</td>
<td>100-160</td>
</tr>
<tr>
<td>Dogs (medium)</td>
<td>80-120</td>
<td>60-90</td>
</tr>
<tr>
<td>Dogs (small)</td>
<td>90-140</td>
<td></td>
</tr>
<tr>
<td>Cats</td>
<td>140-200</td>
<td>100-160</td>
</tr>
</tbody>
</table>

**Table 1-25 Normal Parameters for Heart Rate and Blood Pressure in Dogs and Cats**
Tachycardia

Tachycardia is defined as a heart rate above normal values. Common causes of tachycardia include vasodilation, drugs, inadequate anesthetic depth and perceived pain, hypercapnia, hypoxemia, hypotension, shock, and hyperthermia. Table 1-27 lists the causes and immediate action or treatment for tachycardia.

<table>
<thead>
<tr>
<th>Cause</th>
<th>Immediate Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasodilator drugs</td>
<td>Allow time for drug to wear off.</td>
</tr>
<tr>
<td>Atropine</td>
<td>Allow time for drug to wear off.</td>
</tr>
<tr>
<td>Glycopyrrolate</td>
<td></td>
</tr>
<tr>
<td>Sympathomimetic drugs</td>
<td>Allow time for the drug to wear off; administer a β-blocker; turn off infusion.</td>
</tr>
<tr>
<td>Epinephrine</td>
<td></td>
</tr>
<tr>
<td>Isoproterenol</td>
<td></td>
</tr>
<tr>
<td>Dopamine</td>
<td></td>
</tr>
<tr>
<td>Ketamine</td>
<td>Allow time for drug to wear off.</td>
</tr>
<tr>
<td>Inadequate anesthetic depth</td>
<td>Increase anesthetic depth.</td>
</tr>
<tr>
<td>Hypercapnia</td>
<td>Increase ventilation (assisted ventilation).</td>
</tr>
<tr>
<td>Hypoxemia</td>
<td>Increase gas flow and oxygenation.</td>
</tr>
<tr>
<td>Hypotension</td>
<td>Decrease anesthetic depth; administer an intravenous crystalloid or colloid bolus, positive inotropic drug, positive chronotropic drug, or pressor.</td>
</tr>
<tr>
<td>Hyperthermia</td>
<td>Apply ambient or active cooling measures; administer dantrolene sodium if malignant hyperthermia is suspected.</td>
</tr>
</tbody>
</table>

Hypotension

Hypotension is defined as physiologically low BP (mean arterial pressure less than 65 mm Hg). A mean arterial BP less than 60 mm Hg can result in inadequate tissue perfusion and oxygen delivery. The coronary arteries are perfused during diastole. Inadequate diastolic BP, less than 40 mm Hg, can cause decreased coronary artery perfusion and myocardial hypoxemia that can predispose the heart to dysrhythmias. Causes of perianesthetic hypotension include peripheral vasodilation by anesthetic drugs, bradycardia or tachycardias, hypothermia, inadequate cardiac preload from vasodilation or hemorrhage, decreased venous return from patient position or surgical manipulation of viscera, and decreased cardiac contractility. Table 1-28 lists possible causes of hypotension and immediate actions to take.
Cardiac Dysrhythmias

ECG monitoring is useful for the early detection of cardiac dysrhythmias during the perianesthetic period. Clinical signs of cardiac dysrhythmias include irregular pulse rate or pressure, abnormal or irregular heart sounds, pallor, cyanosis, hypotension, and an abnormal ECG tracing. Remember that the single best method of detecting cardiac dysrhythmias is with your fingertips (palpate a pulse or apex heartbeat) and ears (auscultate the heart). Confirm the dysrhythmia by auscultating the heart rate and rhythm, identify the P waves and the QRS complexes, and evaluate the relationship between the P waves and QRS complexes. Is there a P wave for every QRS complex, and a QRS complex for every P wave? During anesthesia, fluid, acid-base, and electrolyte imbalances can predispose the patient to dysrhythmias. Sympathetic and parasympathetic stimulation, including during the process of intubation, can predispose the patient to dysrhythmias. If the patient's plane of anesthesia is too light, perception of pain can cause catecholamine release, sensitizing the myocardium to ectopic beats. Atrioventricular (AV) blockade can be induced with the administration of α₂-agonist medications, including xylazine and medetomidine. Thiobarbiturates (thiopental) can induce ventricular ectopy and bigeminy. Although these dysrhythmias may not be harmful in the awake patient, anesthetized patients are at a particular risk of dysrhythmia-induced hypotension. Carefully monitor and treat all dysrhythmias (see Cardiac Dysrhythmias). Box 1-27 lists steps to take to prevent perianesthetic dysrhythmias.

### Cardiac Dysrhythmias

<table>
<thead>
<tr>
<th>Cause</th>
<th>Immediate Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypothermia</td>
<td>Provide ambient rewarming.</td>
</tr>
<tr>
<td>Hypocalcemia*</td>
<td>Administer calcium chloride (10 mg/kg IV) or calcium gluconate (23 mg/kg).</td>
</tr>
<tr>
<td>Increased anesthetic</td>
<td>Decrease vaporizer setting and anesthetic depth. Reverse with opioids or α₂-agonists.</td>
</tr>
<tr>
<td>Vasodilation</td>
<td>Administer an intravenous crystalloid bolus (10 mL/kg).</td>
</tr>
<tr>
<td></td>
<td>Administer an intravenous colloid bolus (5 mL/kg).</td>
</tr>
<tr>
<td></td>
<td>Administer a pressor (phenylephrine 1-3 mcg/kg/min).</td>
</tr>
<tr>
<td>Negative inotropy</td>
<td>Decrease anesthetic depth.</td>
</tr>
<tr>
<td></td>
<td>Administer ephedrine (0.1-0.25 mg/kg IV).</td>
</tr>
<tr>
<td></td>
<td>Administer dobutamine (2-20 mcg/kg IV CRI).</td>
</tr>
<tr>
<td></td>
<td>Administer dopamine (2-10 mcg/kg/min).</td>
</tr>
<tr>
<td></td>
<td>Administer norepinephrine (0.05-0.4 mcg/kg/min IV CRI).</td>
</tr>
<tr>
<td></td>
<td>Administer epinephrine (0.05-0.4 mcg/kg/min IV CRI).</td>
</tr>
<tr>
<td>Bradycardia</td>
<td>Administer atropine (0.01-0.04 mg/kg IV or SQ).</td>
</tr>
<tr>
<td></td>
<td>Administer glycopyrrolate (0.005-0.02 mg/kg IV, SQ).</td>
</tr>
</tbody>
</table>

[CRI, Constant rate infusion; IV, intravenously; SQ, subcutaneously.]

*Hypocalcemia caused by chelation from ethylenediaminetetraacetic acid (EDTA) with multiple blood product transfusion (cats are particularly susceptible).

### Postanesthetic Complications

Delayed recovery can be caused by a number of factors, including excessive anesthetic depth, hypothermia, residual action of narcotics or tranquilizers, delayed metabolism of anesthetic drugs, hypoglycemia, hypocalcemia, hemorrhage, and breed or animal predisposition. Careful

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**Table 1-28**: Causes and Treatment of Perianesthetic Hypotension

<table>
<thead>
<tr>
<th>Cause</th>
<th>Immediate Action</th>
</tr>
</thead>
<tbody>
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<tr>
<td></td>
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</tr>
<tr>
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[CRI, Constant rate infusion; IV, intravenously; SQ, subcutaneously.]

*Hypocalcemia caused by chelation from ethylenediaminetetraacetic acid (EDTA) with multiple blood product transfusion (cats are particularly susceptible).
monitoring of the patient’s BP, acid-base and electrolyte status, anesthetic depth, PCV, and vascular volume intraoperatively and taking care with supportive measures to prevent abnormalities can hasten anesthetic recovery and avoid postoperative complications.

Additional Reading

**BLEEDING DISORDERS**

The presentation of a patient with a bleeding disorder often is a diagnostic challenge for the veterinary practitioner (Boxes 1-28 and 1-29). In general, abnormal bleeding can be caused by five major categories: (1) vascular trauma, (2) defective production of hemostatic factors, (3) dilution of hemostatic factors, (4) use or toxicity of systemic anticoagulants, and (5) DIC. A clotting disorder should be suspected in any patient with a history of development of spontaneous deep hematomas, unusually prolonged bleeding after traumatic injury, bleeding at multiple sites throughout the body involving multiple organ systems, delayed onset of severe
hemorrhage after bleeding, and an inability on the practitioner’s part to find an organic cause of bleeding. The signalment, history, clinical signs, and results of coagulation tests often can aid in making a rapid diagnosis of the primary cause of the disorder and in the selection of appropriate case management. When taking a history, ask the following important questions:

- What is the nature of the bleeding?
- What sites are affected?
- How long has the bleeding been going on?
- Has your animal had any previous or similar episodes?
- Is there any possibility of any toxin exposure?
- If so, when and how much did your animal consume?
- Is there any possibility of trauma?
- Does your animal run loose outdoors unattended?
- Have you ever traveled, and if so, where?
- Has your animal been on any medications recently or currently?
- Has your animal been vaccinated recently?
- Have any known relatives of your animal had any bleeding disorders?
- Have you seen any other abnormal signs?

Abnormalities found on physical examination may aid in determining whether the hemorrhage is localized or generalized (e.g., bleeding from a venipuncture site versus bleeding diathesis). Note whether the clinical signs are associated with a platelet problem and superficial hemorrhage or whether deep bleeding can be associated with abnormalities of the coagulation cascade. Also, make an attempt to identify any concurrent illness that can predispose the patient to a bleeding disorder (e.g., pancreatitis, snakebite, sepsis, immune-mediated hemolytic anemia, or severe trauma and crush or burn injury).
Abnormalities associated with coagulopathies include petechiae and ecchymoses, epistaxis, gingival bleeding, hematuria, hemarthrosis, melena, and hemorrhagic cavity (pleural and peritoneal or retroperitoneal) effusions.

**Specific Coagulopathies**

**Disseminated Intravascular Coagulation**

DIC is a complex syndrome that results from the inappropriate activation of the clotting cascade, leading to disruption of the normal balance between thrombosis and fibrinolysis. The formation of diffuse microthrombi with concurrent consumption of platelets and activated clotting factors leads to end-organ thrombosis with various degrees of clinical hemorrhage. In animals, DIC always results from some other pathologic process, including various forms of neoplasia, crush and heat-induced injury, sepsis, inflammation, and immune-mediated disorders (Box 1-30). The pathophysiologic mechanisms involved in DIC include vascular endothelial damage, activation and consumption of platelets, release of tissue procoagulants, and consumption of endogenous anticoagulants.

**Diagnosis of Disseminated Intravascular Coagulation**

Because DIC always results from some other disease process, diagnosis of DIC is based on a number of criteria when evaluating various coagulation test findings, peripheral blood smears, platelet count, and end products of thrombosis and fibrinolysis. There is no one definitive criterion for the diagnosis of DIC (Box 1-31). Thrombocytopenia occurs as platelets are consumed during thrombosis. It is important to remember that trends in decline in platelet numbers are just as important as thrombocytopenia when making the diagnosis. In some cases the platelet count still may be within the normal reference range but has significantly decreased in the last 24 hours. Early in DIC the

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<table>
<thead>
<tr>
<th>BOX 1-30</th>
<th>DISORDERS ASSOCIATED WITH DISSEMINATED INTRAVASCULAR COAGULATION IN THE DOG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neoplasia</td>
<td>Septic peritonitis</td>
</tr>
<tr>
<td>Hemangiosarcoma</td>
<td>Gangrenous mastitis</td>
</tr>
<tr>
<td>Lymphoma</td>
<td>Pyothorax</td>
</tr>
<tr>
<td>Inflammation</td>
<td>Heartworm disease</td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>Immune-mediated disease</td>
</tr>
<tr>
<td>Heat-induced injury</td>
<td>Immune-mediated hemolytic anemia</td>
</tr>
<tr>
<td>Gastric dilatation-volvulus</td>
<td>Trauma</td>
</tr>
<tr>
<td>Mesenteric torsion</td>
<td>Crush injury</td>
</tr>
<tr>
<td>Sepsis</td>
<td>Burn injury</td>
</tr>
<tr>
<td>Gram-negative and gram-positive sepsis</td>
<td>Snake envenomation</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>BOX 1-31</th>
<th>LABORATORY FINDINGS ASSOCIATED WITH A DIAGNOSIS OF DISSEMINATED INTRAVASCULAR COAGULATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Red blood cell fragments</td>
<td></td>
</tr>
<tr>
<td>• Thrombocytopenia</td>
<td></td>
</tr>
<tr>
<td>• Rapid or prolonged activated partial thromboplastin time</td>
<td></td>
</tr>
<tr>
<td>• Rapid or prolonged prothrombin time</td>
<td></td>
</tr>
<tr>
<td>• Rapid or prolonged activated clotting time</td>
<td></td>
</tr>
<tr>
<td>• Hypofibrinogenemia</td>
<td></td>
</tr>
<tr>
<td>• Positive fibrin degradation products without concurrent hepatic disease</td>
<td></td>
</tr>
<tr>
<td>• Decrease in antithrombin concentration</td>
<td></td>
</tr>
<tr>
<td>• Positive D-dimer test</td>
<td></td>
</tr>
</tbody>
</table>

*More than one of the above criteria should be present to aid in the diagnosis of disseminated intravascular coagulation.*
procoagulant cascade dominates, with hypercoagulability. ACT, APTT, and PT may be rapid and shorter than normal. In most cases, we do not recognize the hypercoagulable state in our critically ill patients. Later in DIC, as platelets and activated clotting factors become consumed, the ACT, APTT, and PT become prolonged. Antithrombin (AT), a natural anticoagulant, also becomes consumed, and AT levels decline. AT levels can be measured at commercial laboratories and in some large veterinary institutions. The end products of thrombosis and subsequent fibrinolysis also can be measured. Fibrinogen levels may decline, although this test is not sensitive or specific for DIC. Fibrin degradation (split) products also become elevated. Fibrin degradation products are normally cleared by the liver, and levels of these also become elevated in cases of hepatic failure because of lack of clearance. More recently, cage-side D-dimer tests have become available to measure the breakdown product of cross-linked fibrin as a more sensitive and specific monitor of DIC.

Management of Disseminated Intravascular Coagulation

Management of DIC first involves treating the primary underlying cause. By the time DIC becomes evident, rapid and aggressive treatment is necessary. If you are suspicious of DIC in any patient with a disease known to incite DIC, then ideally, to achieve the best possible prognosis, you should begin treatment before the hemostatic abnormalities start to occur. Treatment involves replacement of clotting factors and AT and prevention of further clot formation. To replenish clotting factors and AT, administer fresh whole blood or fresh frozen plasma. Heparin requires AT as a cofactor to inactivate thrombin and other activated coagulation factors. Administer heparin (50 to 100 units/kg SQ q6-8h of unfractionated heparin; or fractionated enoxaparin [Lovenox], 1 mg/kg SQ bid). Aspirin (5 mg/kg PO bid in dogs; every third day in cats) also can be administered to prevent platelet adhesion. Management of DIC also involves Rule of 20 monitoring and case management to maintain end-organ perfusion and oxygen delivery (see Rule of 20).

Congenital Defects of Hemostasis

Factor VIII Deficiency (Hemophilia A)

Hemophilia A is a sex-linked recessive trait that is carried by females and manifests in males. Female hemophiliacs can result when a hemophilic male is bred with a carrier female. Hemophilia A has been reported in cats and a number of dog breeds, including Miniature Schnauzer, Saint Bernard, Miniature Poodle, Shetland Sheepdog, English and Irish Setters, Labrador Retriever, German Shepherd Dog, Collie, Weimaraner, Greyhound, Chihuahua, English Bulldog, Samoyed, and Vizsla. Mild to moderate internal or external bleeding can occur. Clinical signs of umbilical cord bleeding can become apparent in some animals shortly after weaning. Gingival hemorrhage, hematrhosis, gastrointestinal hemorrhage, and hematomas may occur. Clotting profiles in animals with factor VIII deficiency include prolonged APTT and ACT. The PT and buccal mucosa bleeding time are normal. Affected animals have low factor VIII activity but normal to high levels of factor VIII–related antigen. Carrier females can be detected by low (30% to 60% of normal) factor VIII activity and normal to elevated levels of factor VII–related antigen.

Von Willebrand disease

Von Willebrand disease is a deficiency or defect in von Willebrand protein. A number of variants of the disease have been described: Von Willebrand disease type 1 is associated with a defect in factor VIIIR protein concentration, and von Willebrand disease type 2 is associated with a defect in VIIIR (vWF). Type 1 von Willebrand disease is most common in veterinary medicine. Von Willebrand disease has been identified in more than 29 breeds of dogs, with an incidence that varies from 10% to 60% depending on the breed of origin. Affected breeds include Doberman Pinschers, German Shepherd Dogs, Scottish Terriers, and standard Manchester Terriers, Golden Retrievers, Chesapeake Bay Retrievers, www.ajlobby.com
Miniature Schnauzers, and Pembroke Welsh Corgis. Two forms of genetic expression occur. The first is autosomal recessive disease, in which homozygous von Willebrand disease individuals have a bleeding disorder, whereas heterozygous individuals carry the trait but are clinically normal. The second variant of genetic expression involves an autosomal dominant disease with incomplete expression such that heterozygous individuals are affected carriers and homozygous individuals are severely affected. Von Willebrand disease has high morbidity but fortunately a low mortality. Dogs with 30% or less than normal vWF tend to hemorrhage. Platelet counts are normal, but bleeding times can be prolonged. The APTT can be slightly prolonged when factor VIII is less than 50% of normal. Routine screening tests are nondiagnostic for this disease, although in a predisposed breed with a normal platelet count, a prolonged buccal mucosa bleeding time strongly supports a diagnosis of von Willebrand disease. Documentation of clinical bleeding with low or undetectable levels of factor VIII antigen or platelet-related activities of vWF support a diagnosis of von Willebrand disease. Recessive animals have zero vWF antigen (a subunit of factor III); heterozygotes have 15% to 60% of normal. In the incompletely dominant form, levels of vWF antigen are reduced (less than 7% to 60%). Clinical signs in affected animals include epistaxis, hematuria, diarrhea with melena, penile bleeding, lameness, hemorrhosis, hematoma formation, and excessive bleeding with routine procedures such as nail trimming, ear cropping, tail docking, surgical procedures (spay, neuter), and lacerations. Estrus and postpartum bleeding may be prolonged. A DNA test to detect carriers of the vWF gene is available through VetGen (Ann Arbor, Michigan) and Michigan State University. Patients with von Willebrand disease should avoid drugs known to affect platelet function adversely (sulfonamide, ampicillin, chloramphenicol, antihistamines, theophylline, phenothiazine tranquilizers, heparin, and estrogen).

Factor IX (Christmas factor) Deficiency (Hemophilia B)

Hemophilia B is an X-linked recessive trait that occurs with less frequency that hemophilia A. The disease has been reported in Scottish Terriers, Shetland and Old English Sheepdogs, Saint Bernards, Cocker Spaniels, Alaskan Malamutes, Labrador Retrievers, Bichon Frises, Airedale Terriers, and British Shorthair cats. Carrier females have low (40% to 60% of normal) factor IX activity. Clinical signs are more severe than for hemophilia A.

Factor VII Deficiency

Congenital deficiencies of factor VII have been reported as an autosomal, incompletely dominant characteristic in Beagles. Heterozygotes have 50% factor VII deficiency. Bleeding tends to be mild. The PT is prolonged in affected individuals.

Factor X Deficiency

Factor X deficiency has been documented in Cocker Spaniels and resembles fading-puppy syndrome in newborn dogs. Internal or umbilical bleeding can occur, and affected dogs typically die. Bleeding may be mild in adult dogs. In severe cases, factor X levels are reduced to 20% of normal; in mild cases, factor X levels are 20% to 70% of normal.

Factor XII (Hageman Factor) Deficiency

Factor XII deficiency has been documented as an inherited autosomal recessive trait in domestic cats. Heterozygotes can be detected because they have a partial deficiency (50% of normal) of factor XII. Homozygote cats have less than 2% factor XII activity. Deficiency of Hageman factor usually does not result in bleeding or other disorders.

Factor XI Deficiency

Factor XI deficiency is an autosomal disease that has been documented in Kerry Blue Terriers, Great Pyrenees, and English Springer Spaniels. In affected individuals, protracted bleeding may be observed. Homozygotes have low factor XI activity (<20% of normal), and heterozygotes have 40% to 60% of normal activity.
Management of Congenital Defects of Hemostasis

The management of congenital defects of hemostasis typically involves replenishing the clotting factor that is present. Usually, this can be accomplished in the form of fresh frozen plasma transfusion (20 mL/kg). If anemia is present because of severe hemorrhage, fresh whole blood or packed RBCs also can be administered. Recent research has investigated the use of recombinant gene therapy in the treatment of specific factor deficiencies in dogs; however, the therapy is not yet available for use in clinical practice.

In cases of von Willebrand disease, administration of fresh frozen plasma (10 to 20 mL/kg) or cryoprecipitate (1 unit/10 kg body mass) provides vWF, factor VIII, and fibrinogen. Doses can be repeated until hemorrhage ceases. 1-Desamino-8-D-arginine vasopressin (DDAVP) also can be administered (1 mcg/kg SQ or IV diluted in 0.9% saline given over 10 to 20 minutes) to the donor and patient to increase the release of stored vWF from endothelial cells. A fresh whole blood transfusion can be obtained from the donor and immediately administered to the patient, or it can be spun down and the fresh plasma administered if RBCs are not needed. Administer a dose of DDAVP to any affected dog before initiating any elective surgical procedures. A supply of fresh frozen plasma and RBCs should be on hand, should uncontrolled hemorrhage occur.

Acquired Disorders of Hemostasis

Platelets are essential to normal blood coagulation. After a vessel has been damaged, release of vasoactive amines causes vasoconstriction and sluggish flow of blood in an attempt to squelch hemorrhage. Platelets become activated by platelet activating factor, and attach to the damaged vascular endothelium. Normal platelet adhesion depends on mediators such as calcium, fibrinogen, vWF antigen, and a portion of factor VIII. After adhesion, the platelets undergo primary aggregation and release a variety of chemical mediators, including adenosine diphosphate, prostaglandins, serotonin, epinephrine, thromboplastin, and thromboxane A2, that promote secondary aggregation and contraction. Platelet abnormalities can include decreased platelet production (thrombocytopenia), decreased platelet function (thrombocytopenia), increased platelet destruction, increased platelet consumption, and platelet sequestration.

Thrombocytopenia

Thrombocytopenia refers to platelet function abnormalities. Alterations in platelet function can affect platelet adhesion or aggregation or release of vasoactive substances that help form a stable clot (Box 1-32). In von Willebrand disease there is a deficiency in vWF antigen that results in altered platelet adhesion. Vascular purpuras are reported and have been seen in collagen abnormalities such as Ehlers-Danlos syndrome, which can be inherited as an autosomal dominant trait with complete penetrance and has been recognized in German Shepherd Dogs, Dachshunds, Saint Bernards, and Labrador Retrievers.

<table>
<thead>
<tr>
<th>DRUGS</th>
<th>Causes of Thrombocytopenia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin or other nonsteroidal antiinflammatory drugs</td>
<td>Dysproteinemia</td>
</tr>
<tr>
<td>Heparin</td>
<td>von Willebrand disease defects</td>
</tr>
<tr>
<td>Phenothiazine tranquilizers</td>
<td></td>
</tr>
<tr>
<td>Cephalosporin</td>
<td></td>
</tr>
<tr>
<td>UREMIA</td>
<td>INHERITED</td>
</tr>
<tr>
<td>Liver disease</td>
<td>Thrombasthenic thrombopathia of</td>
</tr>
<tr>
<td></td>
<td>Otterhounds</td>
</tr>
<tr>
<td></td>
<td>Glanzmann thrombasthenia of Great</td>
</tr>
<tr>
<td></td>
<td>Pyrenees</td>
</tr>
<tr>
<td></td>
<td>Thrombopathy of Bassett Hound and</td>
</tr>
<tr>
<td></td>
<td>American Eskimo Dog (Spitz)</td>
</tr>
<tr>
<td></td>
<td>Cyclic hematopoiesis of the gray Collie</td>
</tr>
<tr>
<td></td>
<td>Platelet storage pool disease of American Cocker Spaniel</td>
</tr>
</tbody>
</table>
Thrombasthenic thrombopathia is a hereditary autosomal dominant abnormality that has been described in Otterhounds, Foxhounds and Scottish Terriers. In this condition, platelets do not aggregate normally in response to adenosine diphosphate and thrombin stimulation.

Evaluation of platelet function is based on a total platelet count, buccal mucosa bleeding time, and thromboelastography. Platelet function defects (thrombocytopenia and thrombocytopenia) can affect both sexes. Clinical signs can resemble von Willebrand disease. In most cases, buccal mucosa bleeding time will be prolonged, but platelet count and clotting test results will be normal.

Thrombocytopenia

Platelet count can be decreased because of problems with production, increased consumption, sequestration, or destruction. Causes of accelerated platelet destruction are typically immune-mediated autoantibodies, drug antibodies, infection, and isoimmune destruction. Consumption and sequestration usually are caused by DIC, vasculitis, microangiopathic hemolytic anemia, severe vascular injury, hemolytic uremic syndrome, and gram-negative septicemia. Primary thrombocytopenia with no known cause has been called idiopathic thrombocytopenia. In approximately 80% of the cases, thrombocytopenia is associated with immune-mediated destruction caused by immune-mediated hemolytic anemia, systemic lupus erythematosus, rheumatoid arthritis, DIC, and diseases that affect the bone marrow. In systemic lupus erythematosus, 20% to 30% of the affected dogs have concurrent idiopathic thrombocytopenia. When immune-mediated hemolytic anemia and idiopathic thrombocytopenia are present in the same patient, the disease is called Evans syndrome. PF-3 is a non-complement-fixing antibody that is produced in the spleen and affects peripheral and bone marrow platelets and megakaryocytes. Antibodies directed against platelets can be measured by a PF-3 release test. Platelet counts with immune-mediated destruction typically are less than 50,000 platelets per microliter. Infectious causes of thrombocytopenia include Ehrlichia canis, Anaplasma phagocytophilum (formerly Ehrlichia equi), and R. rickettsii (Rocky Mountain spotted fever). Primary immune-mediated thrombocytopenia has an unknown cause and most frequently is seen in middle-aged to older female dogs. Breed predispositions include Cocker Spaniels, German Shepherd Dogs, Poodles (toy, miniature, standard), and Old English Sheepdogs.

Thrombocytopenia usually manifests as petechiae, ecchymoses of skin and mucous membranes, hyphema, gingival and conjunctival bleeding, hematuria, melena, and epistaxis. To make a diagnosis of idiopathic thrombocytopenia purpura, measure the severity of thrombocytopenia (<50,000 platelets per microliter) and analyze the peripheral blood smear for evidence of platelet fragmentation or microthrombocytosis; normal to increased numbers of megakaryocytes in the bone marrow, detection of antiplatelet antibody, and increased platelet counts after starting glucocorticoid therapy are expected, and other causes of thrombocytopenia should be eliminated. If tick-borne illnesses are suspected, antibody titers for E. canis, A. phagocytophilum, and R. rickettsii should be performed.

Treatment of immune-mediated thrombocytopenia involves suppression of the immune system to stop the immune-mediated destruction and to stimulate platelet release from the bone marrow. Traditionally, the gold standard to suppress the immune system is to use glucocorticoids (prednisone or prednisolone, 2 to 4 mg/kg PO bid divided, or dexamethasone, 0.1 to 0.3 mg/kg IV or PO q12h). More recently, human serum IgG also has been used (0.2 to 0.5 g/kg IV in saline over 8 hours; pretreat with 1 mg of diphenhydramine per kilogram 15 minutes before starting infusion). Vincristine (0.5 mg/m² IV once) can stimulate the release of platelets from the bone marrow if megakaryocytic precursors are present; however, the platelets released may be immature and potentially non-functional. Treatment with fresh whole blood or packed RBCs is appropriate if anemia is present; however, unless specific platelet-rich plasma has been purchased from a blood
bank, fresh whole blood contains relatively few platelets, which are short-lived (2 hours) and will not effectively raise the platelet count at all. Finally, long-term therapy is usually in the form of azathioprine (2 mg/kg PO once daily, tapered to 1 mg/kg daily to every other day after 1 week) and cyclosporine (10 to 25 mg/kg PO divided). If a tick-borne illness is suspected, administer doxycycline (5 to 10 mg/kg PO bid) for 4 weeks or if titers come back negative.

Thrombocytopenia also can occur in the cat. Causes for thrombocytopenia in cats include infections (29%), neoplasia (20%), cardiac disease (7%), primary immune-mediated disease (2%), and unknown causes (20%). In one study of cats with feline leukemia and myeloproliferative disease, 44% had thrombocytopenia.

**Vitamin K–Antagonist Rodenticide Intoxication**

Warfarin and coumarin derivatives are the major class of rodenticides used in the United States. Vitamin K–antagonist rodenticides inhibit the epoxidase reaction and deplete active vitamin K, causing a depletion of vitamin K–dependent coagulation factors (II, VII, IX, X) within 24 hours to 1 week of ingestion, depending on the ingested dose. Affected animals can spontaneously hemorrhage anywhere in the body. Clinical signs can include hemoptysis, respiratory difficulty, cough, gingival bleeding, epistaxis, hematuria, hyphema, conjunctival bleeding, petechiae and ecchymoses, cavity hemorrhage (pleural, peritoneal, retroperitoneal) with acute weakness, lethargy or collapse, hemorrhaxis with lameness, deep muscle bleeds, and intracranial or spinal cord hemorrhage. Diagnosis of vitamin K antagonism includes prolonged PT. A PIVKA (protein induced by vitamin K absence or antagonism) test also can be performed, if possible.

Treatment of vitamin K antagonist rodenticide intoxication and other causes of vitamin K deficiency involves supplementation with vitamin K₁ (phytonadione, 5 mg/kg SQ once with 25-gauge needle in multiple sites, and then 2.5 mg/kg PO bid to tid for 30 days). Never administer injections of vitamin K IM, because of the risk of causing deep muscle hematomas, or IV, because of the risk of anaphylaxis. The PT should be rechecked 2 days after the last vitamin K capsule has been administered, because some of the second-generation warfarin derivates are fat-soluble, and treatment may be required for an additional 2 weeks.

**Table 1-30** summarizes criteria for interpreting coagulation profiles.

<table>
<thead>
<tr>
<th>Disorder</th>
<th>BMBT</th>
<th>ACT</th>
<th>PT</th>
<th>APTT</th>
<th>Platelets</th>
<th>Fibrogen</th>
<th>FDPs</th>
<th>D Dimers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thrombocytopenia</td>
<td>↑</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>↓</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>↑</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Von Willebrand disease</td>
<td>↑</td>
<td>↑/N</td>
<td>N</td>
<td>D/N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Hemophilia</td>
<td>N</td>
<td>↑</td>
<td>N</td>
<td>↑</td>
<td>↑</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Warfarin toxicity</td>
<td>N</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>N/↓</td>
<td>N/↓</td>
<td>N/↑</td>
<td>N</td>
</tr>
<tr>
<td>Disseminated intravascular coagulopathy</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
<td>N/↓</td>
<td>↑</td>
<td>↑</td>
</tr>
</tbody>
</table>

ACT, Activated clotting time; APTT, activated partial thromboplastin time; BMBT, buccal mucosa bleeding time; FDP, fibrin degradation products; N, normal; PT, prothrombin time.
Additional Reading


BURNS

Thermal Injury

Thermal burns are fortunately a relatively infrequent occurrence in veterinary patients. Box 1-33 lists various causes of malicious and accidental burns. The location of the burn is also important in assessing its severity and potential to cause loss of function. Burns on the perineum, feet, face, and ears are considered to be the most severe because of loss of function and severe pain. Often the severity of thermal injury is difficult to assess in animals because the hair coat potentially can mask clinical signs and because the thermal injury can continue after the animal has been removed from the heat source. The skin cools slowly and warms slowly, considerations that become important when initiating therapy for burns. The severity of thermal injury is associated with the temperature to which the animal is exposed, the duration of contact, and the ability of the tissue to dissipate heat. The tissue closest to the heat source undergoes necrosis and has decreased blood flow.

The severity of thermal burn injury is associated directly with the temperature to which the animal is exposed, the percentage of total body surface area affected, the thickness of injured tissue, and whether underlying complications with other body systems occur. Prognosis largely depends on the total body surface area affected (Table 1-31).

<table>
<thead>
<tr>
<th>BOX 1-33 CAUSES OF THERMAL INJURY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Automobile engines</td>
</tr>
<tr>
<td>Automobile exhaust systems</td>
</tr>
<tr>
<td>Boiling water</td>
</tr>
<tr>
<td>Cooking oil (hot)</td>
</tr>
<tr>
<td>Electric heating pads</td>
</tr>
<tr>
<td>Hair dryers</td>
</tr>
<tr>
<td>Heat lamps</td>
</tr>
<tr>
<td>Heat packs</td>
</tr>
<tr>
<td>Improperly grounded electrosurgical units</td>
</tr>
<tr>
<td>Semiliquids (e.g., hot tar)</td>
</tr>
<tr>
<td>Solar exposure</td>
</tr>
<tr>
<td>Steam</td>
</tr>
<tr>
<td>Stove</td>
</tr>
</tbody>
</table>
Superficial partial-thickness, or first-degree, burns offer the most favorable prognosis. The affected epidermis initially appears erythematous and then quickly desquamates within 3 to 6 days. In most cases fur grows back without a scar. Deep partial-thickness, or second-degree, burns involve the epidermis and dermis and are associated with subcutaneous edema, inflammation, and pain. Deep partial-thickness burns heal from deeper adnexal tissues and from the wound edges and are associated with an increased chance of scarring and depigmentation. The most severe type is the full-thickness, or third-degree, burn, in which thermal injury destroys the entire thickness of the skin and forms an eschar. Thrombosis of superficial and deeper skin vasculature and gangrene occur. Treatment involves sequential wound debridement. Healing occurs by second intention and reepithelialization or by wound reconstruction. In most cases scarring is extensive in affected areas.

Burns over greater than 20% of total body surface area will have systemic effects, including impaired cardiovascular function, pulmonary dysfunction, and impaired immune function. Burned tissue, with capillary damage, has increased permeability. The release of inflammatory cytokines, oxygen-derived free radical species, prostaglandins, leukotrienes, histamine, serotonin, and kinins results in increased vascular permeability and leakage of plasma proteins into the interstitium and extravascular space.

Immediate Action and Treatment
At the time of presentation, first examine the patient and ascertain whether airway obstruction, impaired ventilatory function, circulatory shock, or pain is present. If necessary, establish an airway with endotracheal intubation or emergency tracheostomy. Next, cool the burned area(s) with topical cool water. Use care to avoid overcooling and iatrogenic hypothermia. The best approach is to cool only one portion of the patient’s body at a time, then dry, and repeat the process for all affected areas to avoid overcooling and iatrogenic hypothermia. Establish vascular access and administer appropriate and judicious analgesic drugs and intravenous fluid therapy. Whenever possible, avoid placing a catheter through an area of burned or damaged skin. In the early stages of burn injury, shock doses of intravenous crystalloid fluids usually are not required. Later, however, as severe tissue exudation occurs, protein and fluid losses can become extensive, necessitating aggressive crystalloid and colloid support to treat hypovolemia and hypoproteinemia. Flush the eyes with sterile saline and examine behind the third eyelids for any particulate matter. Stain the corneas to make sure that superficial corneal burns are not present. Treat superficial corneal burns with triple antibiotic ophthalmic ointment.

Next, assess the total body surface area affected, as this will determine prognosis. Depending on the extent of the damage, decide whether the burn is superficial and local therapy is indicated or whether more severe injuries exist that may involve systemic therapy or possibly euthanasia.

Differential Diagnosis
In most cases the diagnosis of thermal burns is based on a clinical history of having been in a house fire, in a clothes dryer, or under a heating lamp. Too frequently, however, thermal burns become apparent days after an elective surgical procedure in which the patient was placed on a faulty heating pad rather than a circulating warm water or warm air blanket. Superficial burns appear as singed fur with desquamating, easily epilated hair. This condition

<table>
<thead>
<tr>
<th>Body Region</th>
<th>Percent of Body Surface Area</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head</td>
<td>9%</td>
</tr>
<tr>
<td>Torso</td>
<td>18%</td>
</tr>
<tr>
<td>Forelimb (per limb)</td>
<td>9%</td>
</tr>
<tr>
<td>Hindlimb (per limb)</td>
<td>18%</td>
</tr>
</tbody>
</table>

TABLE 1-31 Percent Burn Estimation: Rule of Nines

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also can resemble a superficial or deeper dermatophytosis if the history is unknown. Other differential diagnoses include immune-mediated vasculitis or erythema multiforme. Unless the superficial dermis is blistered, it may be difficult to distinguish among a thermal burn, a chemical burn, or an electrical burn if the trauma went unnoticed.

Management

Management of burn injury largely depends on the depth of injury and the total body surface area affected. Partial thickness burns and those affecting less than 15% of the total body surface area will require support in the form of antibiotic ointment and systemic analgesic drugs.

Burns affecting greater than 15% of total body surface area or deep-thickness burns require more aggressive therapy. Central venous catheters can be placed to administer crystalloid and colloid fluids, parenteral nutrition if necessary, antibiotics, and analgesic drugs. Monitor perfusion parameters closely, including heart rate, BP, capillary refill time, and urine output. Respiratory function can be impaired because of concurrent smoke inhalation, thermal damage to the upper airways and alveoli, and carboxyhemoglobin or methemoglobin intoxication. Respiratory function also can be impaired because of burn injury to the skin around the thoracic cage. Thoracic radiographs may reveal patchy interstitial to alveolar infiltrates associated with pulmonary edema, pneumonia, and atelectasis. Bronchoscopy often reveals edema, inflammation, particulate matter, and ulceration of the tracheobronchial tree. In some cases, upper airway inflammation is so severe that an emergency tracheostomy must be performed to treat airway obstruction. Administer supplemental humidified oxygen at 50 to 100 mL/kg/min via endotracheal tube, tracheostomy, nasal or intratracheal tube, or hood oxygen if respiratory function and hypoxemia are present. Perform blood work, including determination of hematocrit, albumin, BUN, creatinine, and glucose levels, at the time of presentation. Monitor serum electrolytes, albumin, and colloid oncotic pressure closely because derangements can be severe as burns become exudative.

The goal of fluid therapy in the burn patient is to establish and maintain intravascular and interstitial fluid volume, normalize electrolyte and acid-base status, and maintain serum albumin and oncotic pressure. In the first 24 hours after burn injury, direct fluid therapy to maintaining the patient’s metabolic fluid requirements. Crystalloid fluids in the form of Normosol-R, Plasma-Lyte M, or lactated Ringer’s solution can be administered according to the patient’s electrolyte and acid-base status (see Fluid Therapy). Monitor urine output, and keep it at 1 to 2 mL/kg/hr. Avoid overhydration in the early stages of burn injury. In affected burn patients, calculate the amount of fluid that should be administered over a 24-hour period from the formula 1 to 4 mL/kg × percent total body surface area. Administer half of this calculated dose over the first 8 hours and the remaining half over the next 16 hours. In cats, administer only 50% to 75% of this calculated volume. To administer this volume while also avoiding fluid overload is often difficult in critically ill patients with pulmonary involvement associated with smoke inhalation injury. Avoid colloids in the first 6 hours after burn injury. Monitor the patient closely for serous nasal discharge, chemosis, and rales, which may signify pulmonary edema.

As burns become exudative, weigh the patient at least twice daily. Infused fluid should equal fluid output in the form of urine and wound exudates. Acute weight loss signifies acute fluid loss and that crystalloid fluid infusion should be more aggressive. Ideally, keep the patient’s serum albumin equal to or greater than 2.0 g/dL and total protein between 4.0 and 6.5 g/dL using a combination of fresh frozen plasma or concentrated human albumin. Adjunct colloidal support can be provided with synthetic colloids including hetastarch or hemoglobin-based oxygen carriers (HBOCs). Keep serum potassium within 3.5 to 4.5 mEq/L using potassium chloride or potassium phosphate supplementation. If potassium supplementation exceeds 80 to 100 mEq/L and the patient continues to have severe refractory hypokalemia, administer magnesium chloride (0.75 mEq/kg/day) to enhance potassium retention. If anemia occurs, administer packed RBCs or whole blood (see Blood Component Therapy).
Lavage wounds daily with lactated Ringer’s solution or 0.9% sodium chloride solution. Place wet-to-dry bandages or bandages soaked in silver sulfadiazine or nitrofurazone ointment over the wounds. Depending on the thickness of the burn, epilation and eschar formation and separation may take 2 to 10 days. At each bandage change, debride devitalized tissue to normal tissue. Perform staged partial or total escharectomy, and leave the wound to heal by second intention or by reconstruction using skin advancement flaps or grafts. Maintain meticulous sterility at all times, given that burn patients are at high risk for infection. Administer broad-spectrum antibiotics including cefazolin and enrofloxacin. Perform wound culture if a resistant bacterial infection is suspected.

**Electrical Injury**

The most common cause of electrical injury is associated with an animal chewing on low-voltage alternating current electrical cords in the household. Damage is caused by the current flowing through the path of least resistance, causing heat and thrombosis of vessels and neurons. In some cases, the owner witnesses the event. In other cases, the owner presents the patient because of vague nonspecific signs, and characteristic abnormalities on physical examination support a diagnosis of electrical injury. Burns on the face, paws, commissures of the mouth, tongue, and soft palate may be present. Electrical injury causes a massive release of catecholamines and can predispose the patient to noncardiogenic pulmonary edema within 36 hours of the incident. Clinical signs may be isolated to the pulmonary system, including orthopnea, pulmonary crackles, and cyanosis.

**Immediate Action and Treatment**

Assess the patient’s lips, tongue, soft palate, gingivae, and commissures of the mouth. Early after electrical injury the wound may appear small and white, black, or yellow. Later the wound may become larger as tissue sloughs because of damaged vascular supply. Assess the patient’s respiratory status. Auscultate the lungs to determine whether pulmonary crackles are present. If the patient’s condition is stable, thoracic radiographs may demonstrate an interstitial to alveolar lung pattern in the dorsocaudal lung fields. Measure the patient’s heart rate, BP, oxygenation as determined by pulse oximetry or arterial blood gas, and urine output. Immediate treatment consists of judicious use of analgesics for the burn injury, antibiotics (cefazolin, 22 mg/kg q8h; cephalexin, 22 mg/kg q8h), and humidified supplemental oxygen (50 to 100 mL/kg/min). Direct fluid therapy at meeting the patient’s metabolic fluid requirements. Because of the risk of development of noncardiogenic pulmonary edema, avoid overzealous administration of crystalloid fluids.

**Differential Diagnosis**

Differential diagnoses for the patient with electrical burn injury include chemical or thermal burn, immune-mediated glossitis, cardiogenic pulmonary edema, and pneumonia.

**Management**

Management of the patient with electrical burn injury primarily involves the administration of analgesic agents, supplemental humidified oxygen, and topical treatment of electrical burns. The noncardiogenic pulmonary edema is typically unresponsive to diuretics (e.g., furosemide), bronchodilators (e.g., aminophylline), and splanchnic vascular dilators (e.g., low-dose morphine). The use of glucocorticoids has no proven benefit and may impair respiratory immune function and is therefore contraindicated. Oral burns may require debridement and advancement flaps if large defects or oronasal fistulas develop. If oral injury is severe, place an esophagostomy or percutaneous gastrostomy tube to ensure adequate nutrition during the healing process. If an animal survives the initial electrical incident, prognosis is generally favorable with aggressive supportive care.
**Chemical Injury**

Chemical burns are associated with a number of inciting causes, including oxidizing agents, reducing agents, corrosive chemicals, protoplasmic poisons, desiccants, and vesicants. The treatment for chemical burns differs slightly from that for thermal burns, so it remains important to investigate the cause of the burn when providing initial treatment, whenever possible. At the scene, advise the owner to wrap the patient in a clean towel or blanket for transport. Chilling can be avoided by then wrapping the patient in a second or third blanket. Placement of ointments should be avoided. Encourage immediate transport to the nearest triage facility.

**Immediate Action and Treatment**

The first and foremost consideration when treating a patient with chemical burns is to remove the animal from the inciting cause or offending agent. Make no attempt to neutralize alkaline or acid substances because the procedure potentially could cause an exothermic reaction, leading to thermal injury in addition to the chemical injury.

Remove collars or leashes that may act as tourniquets or constricting devices. Flush affected areas with copious amounts of cool water for several minutes, not cooling more than 10% to 20% of the body at any one time to prevent iatrogenic hypothermia. Support breathing by extending the patient’s head and neck.

Carefully clip the fur over affected areas for further evaluation of the extent of the injury. Lavage exposed eyes with sterile saline, and stain the cornea to evaluate for any corneal burns. Debride any wounds carefully, knowing that the full extent of the wound may not manifest itself for several days. Then cover the wounds with antibiotic burn ointment such as silver sulfadiazine and an occlusive dressing.

**Differential Diagnosis**

Without a history of exposure, the differential diagnosis for any chemical burn includes thermal burn, necrotizing vasculitis, erythema multiforme, and superficial or deep pyoderma.

**Management**

Contact the local or national animal poison control center regarding whether to attempt neutralization. Perform daily bandage changes with staged debridement as the full extent of the wound manifests itself. Place antimicrobial ointment and silver sulfadiazine ointment over the wound to prevent infection. The routine use of antibiotics may promote the development of a resistant bacterial infection. First-generation cephalosporin can be administered. If a more serious infection develops, perform culture and susceptibility testing to direct appropriate antibiotic therapy. The wound can heal by second intention or may require reconstructive repair for definitive closure.

**Radiation Injury**

The primary cause of radiation injury in small animal patients is radiation therapy for neoplastic conditions. The goal of radiation therapy is to kill neoplastic cells. An unfortunate side effect is damage to adjacent normal tissue that results in necrosis, fibrosis, and impaired circulation to the affected area. Radiation burns result in dermatitis, mucositis, impaired surgical wound healing, and chronic nonhealing wounds. In many cases, secondary radiation injury to normal tissue can be prevented or its degree decreased with careful radiation planning and mapping of the radiation field, such that radiation exposure of normal tissue is limited to the smallest extent possible. With the advent of three-dimensional imaging modalities such as computed tomography (CT) and magnetic resonance imaging (MRI), this has become more routine in veterinary oncology to date.

Radiation injury can occur early and appear at the later stage of the course of radiation therapy. Late effects can be delayed and occur 6 months to years after treatment. The degree of radiation injury is categorized based on the depth of tissue affected. First-degree changes
cause cutaneous erythema. Second-degree changes cause superficial desquamation. Third-degree changes cause deeper moist desquamation, and fourth-degree changes are associated with complete dermal destruction and ulceration. During the early stages of radiation injury, affected tissues may appear erythematosus and edematous. Wound exudates may be moist, or the skin may appear dry and scaly with desquamation or ulceration. Later the area may scar and depigment or may have induration, atrophy, telangiectasia, keratosis, and decreased adnexal structures.

**Immediate Action and Treatment**

Treatment for radiation dermatitis is to irrigate the area with warmed saline and to protect the area from self-mutilation. No-bite, or Elizabethan, collars or loose clothing can be used to protect the area from patient-induced injury. Mucositis can be treated with topical green tea baths and the administration of an oral solution of L-glutamine powder (4 g/m²). Local irrigation with Xylocaine or lidocaine viscous jelly can be used in dogs but should be avoided in cats because of the risk of inducing hemolytic anemia and neurotoxicity. Topical and systemic antibiotics (cephalexin, 22 mg/kg PO tid) also can be administered. Avoid antibiotics that can be sensitized by radiation (e.g., metronidazole).

**Differential Diagnosis**

Because most radiation burns are associated with a known exposure to radiation therapy, the cause of the patient’s injury usually is known. If an animal is presented to you with a scar, however, differential diagnoses may include nasal planum solar dermatitis, pemphigus foliaceus, discoid lupus, superficial necrolytic dermatitis, superficial or deep pyoderma, chemical burn, and thermal burn.

**Management**

Treatment of radiation injury involves making the patient as comfortable as possible with analgesic drugs, preventing self-mutilation, and using staged debridement techniques. Wounds can heal by second intention or may require reconstructive surgery.

**Additional Reading**


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**CARDIAC EMERGENCIES**

**Cardiac Arrest and Cardiopulmonary Cerebral Resuscitation**

Cardiopulmonary arrest is the abrupt cessation of spontaneous and effective ventilation and perfusion. Cardiac arrest must be treated rapidly and aggressively to have any chance of success. The goal of CPCR is to perform effective thoracic compressions such that an adequate amount of oxygen is delivered to the brain and other vital tissues. At the time of admission into the hospital, all patients, regardless of their disease process, should have a plan in the event that cardiopulmonary arrest occurs. Do the owners want to proceed with
CPCR? Should you proceed with intubation, cardiac compressions and drugs, or do the owners want you to perform open-chest CPCR?

Among the most important aspects of cardiopulmonary resuscitation are to anticipate whether a patient is rapidly decompensating and likely to arrest and to be prepared at all times. Stock a crash cart at all times with the equipment and drugs necessary in the event that cardiopulmonary resuscitation is required (Box 1-34).

By having routine drills in the hospital on cadavers or stuffed animals, your emergency team can become efficient at performing the responsibilities and jobs required for successful CPCR. The staff should know how to recognize impending signs of a decompensating patient, clinical signs of cardiac arrest, how to call for an emergency in the hospital, how to intubate patients, and how to start cardiac compressions, hook up an electrocardiograph, and draw up the drugs required to treat various arrhythmias.

Conditions that predispose a patient to cardiopulmonary arrest include vagal stimulation, cellular hypoxia, septicemia, endotoxemia, severe acid-base and electrolyte derangements, prolonged seizures, pneumonia, pleural or pericardial effusion, severe multisystemic trauma, electrical shock, urinary obstruction or damage, acute respiratory distress syndrome (ARDS), and use of anesthetic agents. The acute onset of bradycardia, change in mucous membrane color and capillary refill time, change in respiratory pattern, and change in mentation are signs of possible deterioration and impending cardiopulmonary arrest.

The diagnosis of cardiopulmonary arrest is based on the absence of effective ventilation, severe cyanosis, absence of a palpable pulse or apex heartbeat, absence of heart sounds, and ECG evidence of asystole or other nonperfusing rhythm such as electrical-mechanical dissociation (EMD; i.e., pulseless electrical activity [PEA]) or ventricular fibrillation.

Immediate Action and Treatment

Cardiopulmonary Cerebral Resuscitation

The goals of CPCR are to obtain airway access, provide artificial ventilation and supplemental oxygen, implement cardiac compressions and cardiovascular support, recognize and treat dysrhythmias and arrhythmias, and provide stabilization and treatment for cardiovascular, pulmonary, and cerebral function in the event of a successful resuscitation. Even with aggressive treatment and management, the overall success of CPCR is less than 5% in critically ill or traumatized patients and 20% to 30% in anesthetized patients.

Basic Life Support

Basic life support involves rapid intubation to gain airway access, artificial ventilation, and cardiac compressions to promote blood flow and delivery of oxygen to the brain and other important tissues (Figure 1-26). Perform the ABCs or CABs of CPCR, where A is airway, B

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**BOX 1-34  ITEMS TO STOCK IN THE CRASH CART**

- Laryngoscope (various size blades)
- Endotracheal tubes (various sizes)
- Cotton roll gauze to tie in endotracheal tube
- Stylette for intubation
- Rigid catheter (tomcat and long urinary) to assist with intubation and endotracheal drug administration
- 3-, 6-, and 12-mL syringes, taken out of case and attached to 22-gauge needles
- 22-gauge needles
- Ambu bag and oxygen source
- Electrocardiogram monitor
- Epinephrine
- Atropine
- Naloxone
- Calcium gluconate or calcium chloride
- Magnesium chloride
- Amiodarone
- 0.9% saline
- 50% dextrose
- Laceration pack for slash tracheostomy
- Intravenous catheters
- 1-inch white tape
- Emergency drug table—drugs in appropriate doses and volumes and routes of administration for animals of various sizes

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Figure 1-26: Basic cardiopulmonary life support. ECG, Electrocardiogram; CPCR, cardiopulmonary cerebral resuscitation.
is breathing, and C is compression and circulation. Recently, the paradigm has shifted to CABs. While a team member is grabbing an endotracheal tube, clearing the airway of foreign debris, and establishing airway access through endotracheal intubation, a second person starts external cardiac compressions to deliver oxygen that is in the bloodstream to the vital organs. The patient should be positioned in dorsal (>7 kg) or lateral (<7 kg) recumbency for external cardiac compressions. Approximately 80 to 120 external compressions should be performed over the patient’s sternum. A team member should palpate for a peripheral pulse to determine whether cardiac compressions are actually effective. If a peripheral pulse cannot be palpated for every chest compression, change the patient’s position and have a larger individual perform compressions, or initiate open-chest cardiac resuscitation. Once the patient has been intubated, tie in the endotracheal tube and attach it to an oxygen source (anesthetic machine or mechanical ventilator or Ambu bag) for artificial ventilation. The oxygen flow rate should be 150 mL/kg/min. Give two long breaths, and then 12 to 16 breaths per minute. Simultaneous ventilation with thoracic compression increases the pressure difference in the thorax and allows more forward flow of oxygenated blood through the great vessels into the periphery. If possible, a third team member can initiate interposed abdominal compressions, compressing the abdomen when the thoracic cage is relaxed, to improve forward flow. If only one person is available to perform the thoracic compressions and ventilation, give two breaths for every 15 compressions (i.e., 15 thoracic compressions followed by two long breaths, and then start thoracic compressions again). The Jen Chung maneuver can be performed by placing a 25- to 22-gauge hypodermic needle through the skin of the nasal philtrum and twisting the needle into the periosteum to stimulate respirations. This maneuver appears to work better in cats than dogs at return to spontaneous respiration.

Advanced Life Support

Advanced life support during CPCR involves ECG, pulse oximetry and capnometry monitoring, administration of drugs, and the administration of intravenous fluids (in select cases). Most of the drugs used during CPCR can be administered directly into the lungs from the endotracheal tube (intratracheal tube). Therefore only in select instances is it necessary to establish vascular or intraosseous access during CPCR (Figure 1-27). If an animal experiences cardiopulmonary arrest because of extreme hemorrhage or hypovolemia, inappropriate vasodilation caused by sepsis or systemic inflammation, or vasodilation resulting from anesthesia, the administration of shock volumes (90 mL/kg/hr in dogs and 44 mL/kg/hr in cats) is appropriate. If a patient is euvoletic and experiences cardiopulmonary arrest, however, an increase in circulating fluid volume actually can impair coronary artery perfusion by increasing diastolic arterial BP and is therefore contraindicated. Place a capnograph on the end or side of the endotracheal tube to measure end-tidal carbon dioxide.

Recognition and Treatment of Common Nonperfusing Cardiac Rhythms During Cardiopulmonary Cerebral Resuscitation

Asystole: “He’s flatlined”

Asystole is one of the most common rhythm disturbances that causes cardiac arrest in small animal patients. One of the most important things to do when the ECG indicates asystole is to make sure that the ECG monitor is working properly and that all ECG leads are attached properly to the patient. If asystole is truly present, reverse any opiate, α₂-agonist, or benzodiazepine drugs with their appropriate reversal agents. Low-dose epinephrine (0.02 to 0.04 mg/kg diluted with 5 mL sterile saline) can be administered directly into the endotracheal tube via a rigid or red rubber catheter. If vascular access is available, epinephrine (0.02 to 0.04 mg/kg) can be administered IV. No drug should ever be administered directly into the heart by intracardiac injection. Unless the heart is in the veterinarian’s hand during open-chest CPR, intracardiac injection is risky and potentially could lacerate a coronary artery or cause the myocardium to become more irritable and refractory to other therapies, if a drug is delivered into the myocardium and not into the ventricle. For these reasons, intracardiac injections are contraindicated.
Administer atropine (0.04 mg/kg IV, IO) immediately after the epinephrine. Atropine, a vagolytic drug, serves to decrease tonic vagal inhibition of the sinoatrial and AV nodes and increase heart rate. Administer atropine and epinephrine every 2 to 5 minutes during asystole while cardiac compressions, interposed abdominal compressions, and artificial ventilation are continued. Although discontinuation of thoracic compressions can decrease the chance of success during CPCR, you must intermittently evaluate the ECG monitor for any
rhythm change that may necessitate different drug therapies. If the cardiac arrest was not witnessed or more than 2 to 5 minutes have passed without successful return to a perfusing rhythm, perform open-chest CPR, if the client wishes. Administer sodium bicarbonate (1 to 2 mEq/kg IV) every 10 to 15 minutes during CPR. Sodium bicarbonate is the only drug used in CPR that should not be administered intratracheally because of inactivation of pulmonary surfactant.

**Electrical-Mechanical Dissociation**

EMD, also known as pulseless electrical activity, is an electrical rhythm that may look wide, bizarre, and irregular with no associated mechanical contraction of the ventricles. The rhythm can appear different from patient to patient. EMD is one of the more common nonperfusing rhythms observed during cardiopulmonary arrest in small animal patients (Figure 1-28).

When EMD is identified, first confirm the rhythm and proceed with CPR as previously described. EMD is thought to be associated with high doses of endogenous endorphins and high vagal tone. The treatment of choice for EMD is high-dose atropine (4 mg/kg IV, intratracheal [IT] [10 times the normal dose]) and naloxone hydrochloride (0.03 mg/kg IV, IO, IT). Administer epinephrine (0.02 to 0.04 mg/kg diluted in 5 mL sterile 0.9% saline IT). If the rhythm does not change within 2 minutes, consider open-chest cardiac massage.

**Ventricular Fibrillation**

Ventricular fibrillation can be coarse (Figure 1-29). Patients with coarse ventricular fibrillation are easier to defibrillate than those with fine defibrillation. If ventricular fibrillation is identified, initiate CPR as described previously (Figure 1-30). If an electrical defibrillator is available, administer 5 J/kg of direct current externally. When a patient in cardiopulmonary arrest is attached to ECG leads, it is important to use contact electrode paste, water-soluble gel such as K-Y jelly, or water, rather than any form of alcohol. Electrical defibrillation of a patient who has alcohol on the ECG leads can lead to fire and thermal burns. Reverse any opioid, α₂-agonist, and phenothiazine drugs that have been administered to the patient. If fine ventricular fibrillation is identified, administer epinephrine (0.02 to 0.04 mg/kg in 5 mL sterile 0.9% saline IT) to attempt to convert fine ventricular fibrillation to coarse ventricular fibrillation. After administration of epinephrine, repeat electrical defibrillation. If an electrical defibrillator is not available, chemical defibrillator

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**Figure 1-28:** Electrical-mechanical dissociation (EMD), also known as pulseless electrical activity (PEA). The complexes often appear wide and bizarre without a palpable apex beat or functional contraction of the heart. This is just one example of EMD, as many shapes and complexes may be observed.

**Figure 1-29:** Rhythm strip of ventricular fibrillation.
drugs can be used. Amiodarone (0.5 mg/kg IV) is the treatment of choice for ventricular fibrillation. If amiodarone is unavailable, magnesium chloride can be administered (30 mg/kg IV, IO, IT). Even if an electrical defibrillator is available, magnesium chloride can increase the success of converting ventricular fibrillation to asystole or some other rhythm during CPCR. Amiodarone (0.5 mg/kg IV) also can be used to convert ventricular fibrillation. If drug therapy and external thoracic compressions are ineffective after 2 minutes, consider open-chest CPCR.

Open-Chest Cardiopulmonary Cerebral Resuscitation

Perform open-chest CPCR immediately if a pathologic condition exists that prevents enough of a change in intrathoracic pressure that closed-chest CPCR will not be effective in promoting forward blood flow (Box 1-35).

To perform open-chest CPCR, place the patient in right lateral recumbency. Clip a wide strip of fur over the left fifth to seventh intercostal space and quickly aseptically scrub over the clipped area. Using a No. 10 scalpel blade, incise over the fifth intercostal space through the skin and subcutaneous tissue to the level of the intercostal muscles. With Mayo scissors, make a blunt stab incision through the intercostal muscles in the left sixth intercostal
space. Make sure that the person who is breathing for the patient deflates the lungs as you make the stab incision to avoid iatrogenic lung puncture. After the stab incision, open the tips of the Mayo scissors and quickly open the muscle dorsally and ventrally to the sternum with a sliding motion. Avoid the internal thoracic artery at the sternum and the intercostal arteries at the caudal aspect of each rib. Cut the rib adjacent to the sternum and push it behind the rib in front of and at the caudal aspect of the incision to allow more room and better visualization if a rib-spreading retractor is not available. Visualize the heart in the pericardial sac. Visualize the phrenic nerve, and incise the pericardium just ventral to the phrenic nerve. Make sure to not cut the phrenic nerve. Grasp the heart in your hand(s) and gently squeeze it from apex to base, allowing time for the ventricle to fill before the next “contraction.” If the heart does not seem to be filling, administer fluids IV or directly into the right atrium. The descending aorta can be cross-clamped with a Rummel tourniquet or red rubber catheter to improve perfusion to the brain and heart.

Management

Postresuscitation Care and Monitoring (Prolonged Life Support)

Postresuscitation care involves careful monitoring and management of the adverse effects of hypoxia and reperfusion injury on the brain and other vital organs. The first 4 hours after an arrest are most critical, because this is the time period in which an animal is most likely to rearrest unless the underlying cause of the initial arrest has been determine and treated (Table 1-32). Until an animal is adequately ventilating on its own, artificial ventilation by manual bagging or attaching the patient to a mechanical ventilator with supplemental

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
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<tbody>
<tr>
<td><strong>Advanced Life Support</strong></td>
<td></td>
</tr>
<tr>
<td>Atropine</td>
<td>0.04 mg/kg IV, IO; 0.4 mg/kg IT</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>0.5 mg/kg IV</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>0.02-0.04 mg/kg IV, IO, IT</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>0.04-0.08 mcg/kg/min IV CRI for third-degree atrioventricular block</td>
</tr>
<tr>
<td>Magnesium chloride</td>
<td>30 mg/kg IV, IO, IT</td>
</tr>
<tr>
<td>Naloxone</td>
<td>0.03 mg/kg IV, IO, IT</td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>1-2 mEq/kg IV, IO; <em>never administer</em> intratracheally</td>
</tr>
<tr>
<td><strong>Postresuscitation or Prolonged Life Support</strong></td>
<td></td>
</tr>
<tr>
<td>Furosemide</td>
<td>1 mg/kg IV</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>1-2 mg/kg IV, followed by 50-100 mcg/kg/min CRI</td>
</tr>
<tr>
<td>Mannitol</td>
<td>0.51 g/kg IV</td>
</tr>
</tbody>
</table>

CRI, Constant rate infusion; IO, intraosseously; IT, intratracheally; IV, intravenously.
oxygen must continue. The efficacy of oxygenation and ventilation can be monitored using a Wright respirometer, pulse oximetry, capnometry, and arterial blood gas analyses (see also Pulse Oximetry and Capnometry [End-Tidal Carbon Dioxide Monitoring]). Once an animal has been extubated, administer supplemental oxygen (50 to 100 mL/kg/min) (see Oxygen Supplementation).

The brain is sensitive to ischemia and reperfusion injury. The effects of cellular hypoxia and reperfusion include the development of oxygen-derived free radical species that contribute to cerebral edema. Administer mannitol (0.5 to 1 g/kg IV over 5 to 10 minutes), followed by furosemide (1 mg/kg IV) 20 minutes later, to all patients that have experienced cardiopulmonary arrest and have had successful resuscitation. Mannitol and furosemide work synergistically to decrease cerebral edema formation and scavenge oxygen-derived free radical species.

The combination of cardiac arrest, myocardial ischemia and acidosis, and external or internal cardiac compressions often makes the myocardium irritable and predisposed to dysrhythmias after successful CPCR. Start lidocaine (1 to 2 mg/kg IV, followed by 50 to 100 mcg/kg/min IV CRI) in all patients after successful resuscitative efforts. Monitor the ECG continuously for the presence of cardiac dysrhythmias and recurrence of nonpulsing rhythms. Perform direct or indirect BP monitoring. If a patient’s systolic BP is less than 80 mm Hg, diastolic pressure is less than 40 mm Hg, or mean arterial BP is less than 60 mm Hg, administer positive inotropic drugs (dobutamine, 1 to 20 mcg/kg/min) and pressor agents (epinephrine, 0.02 to 0.04 mg/kg IV, IO, IT) to improve cardiac contractility, cardiac output, and core organ perfusion.

The kidneys are sensitive to decreased perfusion and cellular hypoxia. Place a urinary catheter and monitor urine output. In a euvolemic patient, normal urine output should be no less than 1 to 2 mL/kg/hr. If urine output is low, administer low-dose dopamine (3 to 5 mcg/kg/min IV CRI) in an attempt to dilate afferent renal vessels and improve renal perfusion.

Maintain acid-base and electrolyte status within normal reference ranges. Monitor serum lactate as a rough indicator of organ perfusion and cellular oxygen extraction. The presence of elevated or rising serum lactate in the face of aggressive cardiorespiratory and cerebral support makes prognosis less favorable.

Additional Reading


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Cardiac Dysrhythmias Requiring Emergency Management

Cardiac dysrhythmias can encompass a wide range of clinical syndromes that vary in their clinical significance and signs, depending on the rate and frequency and whether coexisting cardiac disease is present. Ventricular and supraventricular dysrhythmias can occur because of primary myocardial disease or some other, secondary underlying disease process, including thoracic trauma, sepsis, systemic inflammatory response syndrome (SIRS), pancreatitis, GDV, splenic disease, hypoxia, uremia, and acid-base and electrolyte disturbances. Common cardiac causes of dysrhythmias include dilative cardiomyopathy, end-stage degenerative valvular disease, infectious endocarditis, myocarditis, and cardiac neoplasia. In the cat, hypertrophy, restrictive and unclassified cardiomyopathies, and hyperthyroidism are the most common causes of dysrhythmias. In addition to arising from structural cardiac or systemic disease, dysrhythmias can occur as an adverse effect of some drugs, including digoxin, dobutamine, aminophylline, and anesthetic agents.

Immediate Action

Immediate action depends largely on recognition of the primary or secondary cause of the dysrhythmia and treatment of the dysrhythmia and underlying cause.

Differential Diagnosis

Diagnosis of cardiac dysrhythmias is based on abnormal physical examination findings on thoracic or cardiac auscultation, the presence of abnormal pulse rhythm and its quality, and recognition of ECG abnormalities. The ECG is critical to the accurate diagnosis of dysrhythmias.

Ventricular Dysrhythmias

Ventricular dysrhythmias arise from ectopic foci in the ventricles that cause the wave of depolarization to spread from cell to cell rather than spread through fast-conducting tissue. This causes the QRS complex to appear wide and bizarre, unless the ectopic focus originates close to the AV node high in the ventricle. Other ECG features of ventricular dysrhythmias include a T wave polarity that is opposite to the QRS complex and nonrelated P waves. Ventricular dysrhythmias may manifest as isolated ventricular premature complexes, couplets, or triplets; bigeminy; or ventricular tachycardia. Relatively slow ventricular tachycardia is known as an idioventricular rhythm and is not as hemodynamically significant as faster ventricular tachycardia. Idioventricular rhythm usually is less than 130 beats/min and may alternate spontaneously with sinus arrhythmias (Figures 1-31 to 1-34).

Figure 1-31: Unifocal premature ventricular complexes (PVCs). All the PVCs are the same shape and size and originate from the same ectopic focus in the ventricle. Note that this rhythm is actually an example of ventricular bigeminy.
Supraventricular Dysrhythmias

Supraventricular dysrhythmias arise from ectopic foci in the atria and are commonly associated with atrial dilatation and structural heart disease such as advanced acquired or congenital heart disease, cardiomyopathies, cardiac neoplasia, or advanced heartworm disease. Occasionally, supraventricular dysrhythmias may be associated with respiratory or other systemic illness. Sustained supraventricular tachycardia in the absence of underlying structural heart or systemic disease is disturbing and should alert the clinician that an accessory pathway conduction disturbance may be present, particularly in Labrador Retrievers.

Supraventricular dysrhythmias can manifest as isolated premature complexes (atrial premature complexes or contractions), sustained or paroxysmal supraventricular tachycardia (atrial tachycardia), or atrial fibrillation or flutter. In the dog, atrial fibrillation most commonly is associated with dilative cardiomyopathy. Rarely and primarily in giant breed dogs, lone atrial fibrillation can occur with no underlying heart disease. Atrial fibrillation and the resultant sustained elevation in ventricular rate are presumed to progress to dilative cardiomyopathy in such breeds. By comparison, atrial fibrillation is relatively uncommon in cats because of the small size of their atria but is associated most commonly with hypertrophic and restrictive cardiomyopathy.

The ECG is critical to the diagnosis of a supraventricular dysrhythmia. The ECG usually demonstrates a normal appearance to the QRS complex unless aberrant conduction occurs in the ventricles, in which case the QRS can be wide but still originate from above the AV node. In most cases of a supraventricular dysrhythmia, some evidence of atrial
activity including P waves, atrial flutter, or atrial fibrillation is apparent. In some cases, it may be difficult to diagnose the exact rhythm without slowing the rate down mechanically or through pharmacologic intervention. Once a rhythm diagnosis has been made, appropriate treatment strategies can be implemented (Figures 1-35 and 1-36).

Management

Ventricular Dysrhythmias

Treatment of ventricular dysrhythmias largely depends on the number of ectopic foci discharging, the rate and character of the dysrhythmia, and whether the presence of the abnormal beats is of adverse hemodynamic consequence, including risk of sudden death. Many ventricular dysrhythmias, including slow idioventricular rhythms, ventricular bigeminy, or intermittent ventricular premature complexes, do not warrant antiarrhythmic therapy unless the patient is hypotensive and the dysrhythmia is thought to be contributing to the hypotension. In such cases, correction of the underlying disease process including hypoxia, pain, or anxiety often alleviates or decreases the incidence of the dysrhythmia.

More serious ventricular dysrhythmias that warrant antiarrhythmic therapy (Table 1-33) include sustained ventricular tachycardia (>160 beats/min in dogs; >220 beats/min in cats), multifocal ventricular premature complexes originating from more than one place in the ventricles, and the presence of R-on-T phenomena, in which the T wave of the preceding complex is superimposed on the QRS of the next complex with no return to isoelectric shelf in between complexes. Treat these ventricular dysrhythmias immediately and aggressively. In dogs, the mainstay of emergency treatment for ventricular dysrhythmias is lidocaine therapy. Administer lidocaine (1 to 2 mg/kg intravenous bolus) over a period of 5 minutes to prevent the adverse side effects of seizures or vomiting. The bolus can be repeated an additional three times (total dose 8 mg/kg) over

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
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<tbody>
<tr>
<td>Procainamide</td>
<td>10-20 mg/kg PO q6-8h</td>
</tr>
<tr>
<td>Sotalol</td>
<td>1-3 mg/kg PO q12h (start low, then titrate upward to effect)</td>
</tr>
<tr>
<td>Mexiletine*</td>
<td>4-10 mg/kg PO q8h</td>
</tr>
<tr>
<td>Atenolol</td>
<td>0.25-1.0 mg/kg PO q12-24h (start low, titrate upward to effect)</td>
</tr>
</tbody>
</table>

PO, Orally.

*Do not use for longer than 2 weeks because of idiosyncratic blindness.
15 minutes, or the patient can be placed on a CRI (50 to 100 mcg/kg/min) if control of ventricular tachycardia is accomplished. Also correct the patient’s magnesium and potassium deficiencies to maximize the success of lidocaine therapy in the treatment of ventricular tachycardia. Procainamide (4 mg/kg IV slowly over 3 to 5 minutes) also can be used to control ventricular tachycardia. If procainamide is successful at controlling ventricular tachycardia, administer it as a CRI (25 to 40 mcg/kg/min). Side effects of procainamide include vomiting, diarrhea, and hypotension.

Chronic oral therapy may or may not be necessary in the treatment of acute ventricular tachycardia. The decision to continue antiarrhythmic therapy depends on the underlying disease process and the expectation of persistent arrhythmogenesis of the underlying disease process. Oral antiarrhythmic therapy is warranted in cases in which a serious ventricular dysrhythmia is recognized but the animal does not require hospitalization, such as the syncopal Boxer with intermittent ventricular dysrhythmias and no evidence of structural heart disease. It deserves emphasis that asymptomatic, low-grade ventricular dysrhythmias probably do not require treatment. If maintenance therapy for ventricular dysrhythmias is needed, use an oral drug based on the underlying disease process, clinical familiarity, class of drug, administration frequency, owner compliance, concurrent medications, cost, and potential adverse side effects.

**Treatment of Ventricular Dysrhythmias in Cats**

In the cat the mainstay of antiarrhythmic therapy is the use of a β-adrenergic antagonist. In the acute management of ventricular dysrhythmias in cases of hypertrophic, restrictive, or unclassified cardiomyopathies, consider using injectable esmolol (0.05 to 1.0 mg/kg IV slowly to effect) or propranolol (0.02 to 0.06 mg/kg IV slowly to effect), particularly if the dysrhythmia results from hyperthyroidism. For chronic oral ventricular antiarrhythmic therapy in cats, propranolol (2.5 to 5.0 mg PO per cat q8h) or atenolol (6.25 to 12.5 mg PO per cat q12-24h) can be used.

**Supraventricular Dysrhythmias**

The decision to treat supraventricular dysrhythmias depends on the ventricular rate and the hemodynamic consequences of the dysrhythmia. For intermittent isolated atrial premature contractions, couplets, and triplets, usually no treatment is required. When the ventricular rate exceeds 180 beats/min, diastolic filling is shortened, causing the heart to not fill adequately. The consequence is decreased cardiac output and decreased coronary artery perfusion. The goal of therapy is rhythm control or, in most cases, rate control. In cases of atrial fibrillation and CHF; conversion to a normal sinus rhythm rarely can be achieved, although electrocardioversion or pharmaconversion can be attempted.

In the dog a vagal maneuver can be attempted by pressing on the eyeballs or massaging the carotid body. For sustained supraventricular tachycardia, diltiazem (0.25 mg/kg IV), esmolol (0.05 to 0.1 mg/kg, titrated upward to a cumulative dose of 0.5 mg/kg IV), or propranolol (0.04 to 0.1 mg/kg IV slowly to effect) can be administered in an attempt to slow the ventricular rate in emergent situations. Administer oral diltiazem (0.5 mg/kg PO q8h), diltiazem (Dilacor XR) (1.5 to 6 mg/kg PO q12-24h), propranolol (0.1 to 0.2 mg/kg tid, PO titrated up to a maximum of 1.5 mg/kg PO q8h), atenolol (0.25 to 1 mg/kg PO q12-24h), or digoxin (0.005 to 0.01 mg/kg bid or 0.22 mg/m² PO bid for dogs weighing more than 15 kg).

In the cat a vagal maneuver can be attempted by ocular or carotid massage. Diltiazem [Dilacor] (30 to 60 mg PO q12-24h), propranolol (2.5 to 10 mg PO q8h), or atenolol (6.25 mg PO q12-24h) also can be administered. If structural heart disease is present, treat pulmonary edema and start angiotensin-converting enzyme (ACE) inhibitor therapy. Table 1-34 summarizes the drugs used in the management of supraventricular dysrhythmias.

**Bradyarrhythmias**

Severe bradycardia often results from systemic disease, drug therapy, anesthetic agents, or hypothermia and thus rarely requires specific therapy except to treat or reverse the underlying mechanisms promoting bradycardia. Hemodynamically significant bradyarrhythmias that must be treated include atrial standstill, AV block, and sick sinus syndrome.
Atrial Standstill

Atrial standstill most commonly is associated with hyperkalemia and is seen most often in urinary obstruction, renal failure, urinary trauma with uroabdomen, and hypoadrenocorticism. Characteristic ECG abnormalities observed in atrial standstill are an absence of P waves, widened QRS complexes, and tall spiked T waves (Figure 1-37).

The treatment for hyperkalemia-induced atrial standstill is to correct the underlying cause and to drive potassium intracellularly and protect the myocardium from the adverse effects of hyperkalemia. Regular insulin (0.25 to 0.5 units/kg IV) followed by dextrose (1 g/unit insulin IV, followed by 2.5% dextrose CRI to prevent hypoglycemia) or sodium bicarbonate (1 mEq/kg IV) can be administered to drive potassium intracellularly. Calcium gluconate (0.5 mL/kg of 20% solution IV over 5 minutes) also can be administered as a cardioprotective drug until the cause of hyperkalemia has been identified and resolved. Also administer sodium chloride fluids (0.9% sodium chloride IV) to promote kaliuresis.

Less commonly, atrial standstill is associated with atrial cardiomyopathy or silent atrium syndrome. Persistent atrial standstill has been recognized without electrolyte abnormalities in the English Springer Spaniel and the Siamese cat. Short-term therapy for persistent atrial standstill includes atropine (0.04 mg/kg SQ) until definitive treatment by implantation of a cardiac pacemaker can be performed.

Third-Degree Atrioventricular Block

Complete or third-degree AV block or high-grade symptomatic second-degree AV block can be hemodynamically significant when ventricular rates are less than 60 beats/min in the dog. Classic clinical signs include weakness, exercise intolerance, lethargy, anorexia, syncope, and occasionally seizures. Advanced AV block usually is caused by advanced

![Figure 1-37](www.ajlobby.com) An example of atrial standstill caused by hyperkalemia in a blocked tomcat. Note that there are no P waves and that the ventricular QRS complexes are widened and blunted.
idiopathic degeneration of the AV node. Less commonly, AV block has been associated with digoxin toxicity, magnesium oversupplementation, cardiomyopathy, endocarditis, or infectious myocarditis (Lyme disease). An accurate diagnosis is made based on the ECG findings of nonconducted P waves with ventricular escape beats. First- and second-degree AV block may not be hemodynamically significant and therefore may not require therapy.

Initially treat third-degree (complete) or symptomatic high-grade second-degree atrioventricular block (<60 beats/min) with atropine (0.04 mg/kg SQ or IM). Perform a follow-up ECG in 15 to 20 minutes. Atropine is rarely successful in treating complete AV block. Also attempt treatment with isoproterenol (0.04 to 0.08 mcg/kg/min IV CRI or 0.4 mg in 250 mL 5% dextrose in water IV slowly), a pure β-agonist. Definitive treatment involves permanent pacemaker implantation. Consultation with a veterinary cardiologist who implants pacemakers is suggested. Never attempt to convert or treat the observed ventricular escape beats with lidocaine (Figure 1-38).

Sick Sinus Syndrome

Sick sinus syndrome most commonly is recognized in the Miniature Schnauzer, although any dog can be affected. Sick sinus syndrome usually results from idiopathic degeneration of the sinus node in the dog. In the cat, sinus node degeneration usually is associated with cardiomyopathy. Dysfunction of the sinus node may manifest as marked bradycardia with periods of sinus arrest followed by junctional or ventricular escape complexes. A variant of sick sinus syndrome is the presence of severe bradycardia followed by periods of supraventricular tachycardia, often termed bradycardia-tachycardia syndrome. The most common clinical signs are syncope, exercise intolerance, and lethargy.

Treatment of sick sinus syndrome involves permanent pacemaker implantation by a veterinary cardiologist. Less severe cases of sick sinus syndrome can be managed medically, at least short-term, with atropine (0.04 mg/kg IM) or Propantheline (0.25 to 0.5 mg/kg PO q8h).

Additional Reading


Congenital Heart Failure in Dogs and Cats

Presentation in the Dog

The majority of animals with CHF are older animals that have some acquired heart disease that developed later in life. Congenital defects are rarer than acquired heart disease. The most common congenital defect observed in dogs and in some cats is a patent ductus arteriosus.

The most common acquired cardiac disease in dogs is chronic valvular disease, or endocardiosis (mitral valve endocardiosis). In endocardiosis, the AV valves chronically lose the ability to close effectively, causing abnormalities in blood flow, including regurgitation during ventricular systole. In most cases, disease progression is chronic and slow, although acute exacerbations and onset of clinical signs can be associated with stress, rupture of chordae tendineae, or ingestion of a high-salt meal. Mitral valve disease tends to affect older toy breeds such as miniature Poodles, Chihuahuas, and younger Cavalier King Charles Spaniels.

The second most common cause of acquired heart disease is dilated cardiomyopathy, which is a disease of primary myocardial failure. In dilated cardiomyopathy the muscular wall of the heart becomes thin and weak as the myocardium dilates, causing a decrease in contractility and cardiac output. Secondary mitral and tricuspid valvular insufficiency may result from chronic stretching of the valve annulus. This type of heart disease typically is associated with giant breed dogs including Irish Wolfhounds, English Mastiffs, Great Danes, Boxers, and Doberman Pinschers. A rare form of the disease has been documented in young Labrador Retrievers. Acute exacerbation of dilated cardiomyopathy may be related to the development of a dysrhythmia, including atrial fibrillation.

Presentation in the Cat

In cats, hypertrophic cardiomyopathy is the most common form of acquired cardiac disease observed. CHF resulting from hypertrophic cardiomyopathy can occur in animals as young as 6 to 10 months of age. Hypertrophic cardiomyopathy is characterized by stiff, noncompliant ventricles that do not relax during diastole, resulting in an increase in left atrial pressures and left atrial enlargement. Other cardiomyopathies, including unclassified, restrictive, and dilated cardiomyopathies, are less common but also can occur in the cat. Cats often develop acute exacerbations of clinical signs because of stress or arterial embolization.
Immediate Action and Treatment

The rapid diagnosis of CHF often is made based on the history given by the owner, signalment, and physical examination findings (Box 1-36).

Typical physical examination findings include a cardiac murmur or gallop dysrhythmia, abnormal breath sounds, respiratory difficulty and orthopnea, tachycardia, weak pulse quality, cool peripheral extremities, and pale or cyanotic mucous membrane. Initiate immediate treatment based on physical examination findings and index of suspicion. In some cases, it is difficult to distinguish between CHF and feline lower airway disease (asthma) without performing thoracic radiographs. Let the animal rest and become stabilized before attempting any stressful procedures, including thoracic radiographs.

Immediate treatment consists of administering supplemental oxygen, decreasing circulating fluid volume with furosemide, dilating pulmonary and splanchnic capacitance vessels with topical nitroglycerine and morphine, and alleviating patient anxiety and stress (Box 1-37).

Differential Diagnosis

Primary differential diagnoses are made based primarily on the patient’s breed, age, clinical signs, history, and physical examination abnormalities. The most common differential diagnoses in a patient with CHF are cardiac abnormalities and respiratory disease (chronic bronchitis [asthma], pulmonary hypertension, cor pulmonale, neoplasia).

Postpone diagnostic tests in any patient with suspected CHF until the immediate treatments have taken effect and the patient is cardiovascularly more stable. In most cases, lateral and dorsoventral thoracic radiographs are one of the most important diagnostic tools in helping make a diagnosis of CHF. Increased perihilar interstitial to alveolar infiltrates are characteristic of pulmonary edema. Left atrial enlargement may be observed as a “backpack” sign at the caudal cardiac waist. Cardiomegaly of the right or left side also may be present in cases of valvular insufficiency. In cats, increased sternal contact and a classic valentine-shaped heart may be observed in cases of hypertrophic cardiomyopathy. Perform a vertebral heart score (sum) to measure cardiac size and determine whether cardiomegaly is present (Box 1-38).

Also obtain arterial BP and ECG readings to determine whether hypotension and dysrhythmias are present. Atrial fibrillation, ventricular premature contractions, and supraventricular tachycardia are common rhythm disturbances that can affect cardiac output adversely and influence treatment choices.

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**BOX 1-36  COMMON PRESENTING SIGNS OF CONGESTIVE HEART FAILURE AS REPORTED BY OWNERS**

- Lethargy
- Weakness
- Cough
- Respiratory difficulty
- Exercise intolerance

- Inappetence
- Weight loss
- Abdominal distension
- Syncope

**BOX 1-37  IMMEDIATE MANAGEMENT OF CONGESTIVE HEART FAILURE**

- Supplemental oxygen at 50 to 100 mL/kg/min to supply 40% to 50% oxygen
- Furosemide, 4 to 8 mg/kg IV, IM, every 30 minutes until the patient urinates and body weight decreases by 7%
- Nitroglycerine ointment (¼ to 1 inch topically) every 8 hours
- Morphine, 0.025 to 0.05 mg/kg IV (dog only)

*IM, Intramuscularly; IV, intravenously.*
The echocardiogram is a useful noninvasive and nonstressful method to determine the degree of cardiac disease present. The echocardiogram is largely user-dependent. The quality of the study is based on the experience of the operator and the quality of the ultrasound machine. Echocardiography can be a useful tool in making a diagnosis of pericardial effusion, dilated or hypertrophic cardiomyopathy, cardiac neoplasia, and endocarditis.

Management of Congestive Heart Failure in Dogs and Cats
The medical management of CHF is designed to improve cardiac output and relieve clinical signs. The immediate goal of therapy is to reduce abnormal fluid accumulation and provide adequate cardiac output by increasing contractility, decreasing preload and ventricular afterload, and/or normalizing cardiac dysrhythmias. Strict cage rest is of utmost importance when managing a patient with CHF.

After initial administration of furosemide, morphine, and oxygen, clinical signs of respiratory distress should show improvement within 30 minutes. If no improvement is observed, administer repeated doses of furosemide. Reevaluate severe cases that are refractory to this standard treatment protocol. Vasodilation should be the next step in the management of refractory cases, provided that a normal BP is present. Sodium nitroprusside is a potent balanced vasodilator that should be administered (1 to 10 mcg/kg/min IV CRI), taking care to monitor BP continuously because severe vasodilation and hypotension can occur. The goal of nitroprusside therapy is to maintain a mean arterial BP of 60 mm Hg. Sodium nitroprusside should not be considered in cases of refractory CHF with severe hypotension.

For more long-term management of CHF, ACE inhibitors including enalapril (0.5 mg/kg PO q12-24h), benazepril (0.5 mg/kg PO q24h), and lisinopril (0.5 mg/kg PO q24h) have become the mainstay of therapy to reduce sodium and fluid retention and decrease afterload. Balanced inodilators such as pimobendan (0.5 mg/kg PO) can also be administered both in the acute congestive failure setting and for long-term therapy. Start ACE inhibition as soon as a patient is able to tolerate oral medications.

Dobutamine (2.5 to 10 mcg/kg/min CRI diluted in 5% dextrose in water) can be administered to improve cardiac contractility, particularly in cases of dilated cardiomyopathy. At low doses, dobutamine, primarily a β-adrenergic agonist, will improve cardiac output with minimal effects on heart rate. Dobutamine must be given as a CRI with careful, continuous ECG monitoring. Despite the minimal effects of dobutamine on heart rate, sinus tachycardia or ventricular dysrhythmias may develop during infusion. Cats are more sensitive to the effects of dobutamine than dogs. Monitor carefully for seizures and facial twitching.

Digoxin is a cardiac glycoside that acts as a positive inotrope and negative chronotrope in the long-term management of CHF. Digoxin has a long (24 hours in dogs, 60 hours in cats) half-life and so has minimal use in the emergency management of CHF. In chronic management of CHF resulting from dilated cardiomyopathy or advanced mitral disease, however, digoxin is extremely useful. Oral digitalization protocols have been developed but are risky in that dysrhythmias and severe gastrointestinal side effects can occur.

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**BOX 1-38 VERTEBRAL HEART SUM TO DETERMINE CARDIOMEGALY**

The vertebral heart sum can be calculated by performing the following steps:
1. Measure the long axis of the heart from the apex to the carina on the lateral view and mark the distance on a sheet of paper.
2. Measure the length of the long axis of the heart in terms of vertebral bodies, starting by counting caudally from the fourth thoracic vertebra; count the number of vertebrae that are covered by the length of the long axis of the heart.
3. Measure the short axis of the heart at the caudal vena cava, perpendicular to the long axis of the heart.
4. Count the number of thoracic vertebrae covered by the short axis of the heart, starting at T4.
5. Add the two numbers together to yield the vertebral heart sum; a vertebral heart sum greater than 10.5 is consistent with cardiomegaly.
Cats with CHF often have fulminant pulmonary edema, pleural effusion, arterial thromboembolism, or some combination of all three. If the pleural effusion is significant, perform therapeutic thoracocentesis to relieve pulmonary atelectasis and improve oxygenation. Once the diagnosis of CHF has been made and initial management undertaken, formulate a plan for continued management and monitoring. Tailor the therapeutic plan to the patient based on the cause of the CHF, the presence of concurrent diseases, and response to therapy. An important and often overlooked part of the successful emergency management of CHF is open communication with the owner regarding the owner’s emotional and financial commitment to immediate and long-term management to ensure appropriate quality of life for each patient.

Additional Reading

**Canine Caval Syndrome of Heartworm Disease**
Caval syndrome resulting from severe heartworm disease is caused by the rapid maturation of a large quantity of adult worms in the right atrium and cranial and caudal venae cavae. Most cases of caval syndrome occur in regions of the world where heartworm disease is highly endemic and dogs spend a large portion of time living outdoors. Caval syndrome is recognized by the following clinical signs and results of biochemical analyses: acute renal and hepatic failure, enlarged right atrium and posterior vena cava, ascites, hemoglobinuria, anemia, acute collapse, respiratory distress, DIC, jugular pulses, circulating microfilariae, and sometimes tricuspid insufficiency.

**Immediate Action and Treatment**
Immediate action in cases of caval syndrome in dogs involves immediate stabilization of the cardiovascular and respiratory systems with supplemental oxygen, furosemide (4 mg/kg IV), and careful crystalloid fluid infusion.
Diagnosis
Diagnosis of caval syndrome is based on clinical signs of cardiogenic shock with right ventricular heart failure, intravascular hemolysis, and renal and hepatic failure. Thoracic radiographs reveal cardiomegaly of the right side and enlarged tortuous pulmonary arteries. A right axis deviation may be seen on ECG tracings. Clinopathologic changes observed include azotemia, inflammatory leukogram, regenerative anemia, eosinophilia, elevated hepatocellular enzyme activities, hemoglobinuria, and proteinuria. Circulating microfilariae may be observed on peripheral blood smears or in theuffy coat of microhematocrit tubes. Heartworm antigen test results will be strongly positive. Echocardiographic changes include visualization of a large number of heartworms in the right atrium, pulmonary arteries, and vena cava; tricuspid insufficiency; and right atrial and ventricular enlargement.

Management
Treatment involves surgical removal of as many of the adult heartworms as possible from the right jugular vein and right atrium. Glucocorticosteroids are recommended to decrease inflammation and microangiopathic disease associated with heartworm infection. For more long-term management, administer adulticide therapy several weeks after surgery, followed by routine microfilaricide therapy and then prophylaxis. Doxycycline (10 mg/kg PO bid) also should be administered for a period of 4 weeks, as Wolbachia, a species of bacteria, is often associated with the Dirofilaria immitis nematode. Adulcidal activity of ivermectin is augmented when doxycycline is used concurrently in the treatment of heartworm disease.

Additional Reading

Pericardial Effusion and Pericardiocentesis
Pericardial effusion often develops as a consequence of neoplasia in the older dog and cat. The most common types of neoplasia that affect the heart and pericardium include hemangiosarcoma, chemodectoma, mesothelioma, and metastatic neoplasia. More rarely, other causes of pericardial effusion include benign idiopathic pericardial effusion, coagulopathy, left atrial rupture in dogs with chronic mitral valvular insufficiency, infection, or pericardial cysts. Regardless of the cause of the effusion, the development of pericardial tamponade adversely affects cardiac output.

Cardiac output is a function of heart rate and stroke volume. Stroke volume depends on cardiac preload. The presence of pericardial effusion can impede venous return to the heart and thus adversely affect preload. In addition, as preload decreases, heart rate reflexively increases in an attempt to maintain normal cardiac output. As heart rate increases more than 160 beats/min, diastolic filling is impaired further, and cardiac output further declines. Animals with pericardial effusion often demonstrate the classic signs of hypovolemic or cardiogenic shock: anorexia, weakness, lethargy, cyanosis, cool peripheral extremities, tachycardia, weak thready pulses, hypotension, and collapse. Physical examination abnormalities may include muffled heart sounds, thready femoral pulses, pulsus paradoxus, jugular venous distension, weakness, tachycardia, cyanosis, and tachypnea. ECG findings may include low amplitude QRS complexes (<0.5 mV), sinus tachycardia, ventricular dysrhythmias, or electrical alternans (Figure 1-39). Thoracic radiographs often demonstrate a globoid cardiac silhouette, although the cardiac silhouette rarely may appear
normal with concurrent clinical signs of cardiogenic shock in cases of acute hemorrhage. In such cases the removal of even small amounts of pericardial effusion by pericardiocentesis can increase cardiac output exponentially and alleviate clinical signs (Table 1-35). Unless an animal is dying before your eyes, ideally perform an echocardiogram to attempt to determine whether a right atrial, right auricular, or heart base mass is present before pericardiocentesis.

### Pericardiocentesis

Before attempting pericardiocentesis, assemble all of the required supplies (Box 1-39).

To perform pericardiocentesis, follow this procedure:

1. Place the patient in sternal or lateral recumbency.
2. Attach ECG leads to monitor the patient for dysrhythmias during the procedure.
3. Clip a 6-cm square caudal to the right elbow over the fifth to seventh intercostal space.

### Differential Diagnosis of Pericardial Effusion

<table>
<thead>
<tr>
<th>Type of Pericardial Effusion</th>
<th>Cause</th>
<th>Characteristic Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemorrhagic</td>
<td>Heart base tumors, Hemangiosarcoma, Metastatic neoplasia, Benign idiopathic pericardial effusion, Physical trauma, Left atrial rupture</td>
<td>Usually brachycephalic breeds; &gt;8 years old; blood usually nonclotting.</td>
</tr>
<tr>
<td>Transudate</td>
<td>Coagulopathy, Congestive heart failure, Hypoproteinemia, After peritoneo-pericardial diaphragmatic hernia</td>
<td>Radiograph or echocardiogram will usually demonstrate pericardial lesion.</td>
</tr>
<tr>
<td>Exudate</td>
<td>Infectious pericarditis, Suppurative pericarditis</td>
<td>Exudate in distemper, leptospirosis, and systemic fungal infection. Foreign body or hematologic spread of inflammatory process.</td>
</tr>
</tbody>
</table>
Aseptically scrub the clipped area, and infuse 1 to 2 mg of 2% lidocaine per kilogram mixed with a small amount of sodium bicarbonate just dorsal to the sternum at the sixth intercostal space. Bury the needle to the hub, and inject the lidocaine as you withdraw the needle.

While the local anesthetic is taking effect, assemble the intravenous extension tubing, three-way stopcock, and 60-mL syringe.

Wearing sterile gloves, make a small nick incision in the skin to decrease drag on the needle and catheter during insertion.

Slowly insert the needle and catheter, watching for a flash of blood in the hub of the needle and simultaneously watching for cardiac dysrhythmias on the ECG monitor.

Once a flash of blood is observed in the hub of the needle, advance the catheter off of the stylette further into the pericardial sac, and remove the stylette.

Attach the length of intravenous extension tubing to the catheter, and have an assistant withdraw the fluid slowly.

Place a small amount of fluid in a red-topped tube, and watch for clots. Clot formation could signify that you have penetrated the right ventricle inadvertently or that active hemorrhage is occurring. Withdraw as much of the fluid as possible, and then remove the catheter. Monitor the patient closely for fluid reaccumulation and recurrence of clinical signs of cardiogenic shock.

**Additional Reading**


Ware WA: Cardiac tamponade and pericardiocentesis. In Silverstein DC, Hopper K, editors: *Small animal critical care medicine*, St Louis, 2009, Elsevier.

**EAR EMERGENCIES**

**FOREIGN BODIES**

Foreign bodies within the ear canal (e.g., foxtails) can manifest as emergencies because of acute inflammation and pressure necrosis of the tissue of the external auditory meatus causing pain and discomfort. Clinical signs may be limited to incessant head shaking or scratching of the ear canal.

**Immediate Action and Treatment**

Complete examination of the ear canal and removal of any foreign body often require administration of a short-acting anesthetic agent. Once the animal has been restrained sufficiently and placed under anesthesia, carefully examine the ear canal and remove any foreign material with alligator forceps. Stimulation of the ear canal can cause awakening and shaking of the head. Use care to not perforate the tympanum or cause trauma to the
ear canal with the forceps. Heat-fix any purulent material within the ear canal and examine it cytologically for bacteria or fungal organisms. Gently irrigate the ear canal with warm sterile saline to remove excessive debris and exudates. Use care to avoid excessive pressure (>50 mm Hg) to avoid iatrogenic damage to the tympanic membrane.

**Management**
After removal of all debris and detritus, gently wipe the internal and external ear canal with sterile gauze. Place a topical antimicrobial-antifungal-steroid ointment such as Otomax in the ear every 8 to 12 hours. If pain and discomfort are severe, systemically effective opioids or NSAIDs may be required.

**Otitis Externa**
Otitis externa is a common emergency that causes excessive head shaking, scratching, and purulent malodorous aural discharge.

**Immediate Action and Treatment**
Clean the ear canal with an irrigating solution such as Epi-Otic and wipe it clean of debris. Perform a complete aural examination to determine whether a foreign body or tumor is present and whether the tympanic membrane is intact. Heat-fix any discharge and examine it cytologically for bacteria and fungal organisms. After careful cleansing, instill a topical antibiotic-antifungal-steroid ointment.

**Management**
In severe cases in which the ear canal has scarred and closed down with chronicity, consider administering systemically effective antibiotics (cephalexin, 22 mg/kg PO tid) and antifungal agents (ketoconazole, 10 mg/kg PO q12h) instead of topical therapy. Systemically effective steroids (prednisone or prednisolone, 0.5 mg/kg PO q12h) may be indicated in cases of severe inflammation to decrease pruritus and patient discomfort.

**Otitis Interna**
Presentation of a patient with otitis interna often is characterized by torticollis, head tilt, nystagmus, circling to the affected side, or rolling. Fever, pain, vomiting, and severe depression may accompany clinical signs. Most cases of severe otitis interna are accompanied by severe otitis media. Both conditions must be treated simultaneously. The most common causes of otitis interna are *Staphylococcus aureus*, *Pseudomonas* species, *E. coli*, and *Proteus* species. Otitis interna can develop by infection spreading across the tympanic membrane, through the eustachian tubes, or by hematogenous spread from the blood supply to the middle ear. In most cases of otitis media, the tympanic membrane is ruptured.

**Immediate Action and Treatment**
Perform a culture and susceptibility test of the debris behind the tympanic membrane and within the aural canal. Carefully clean the external ear canal. Medicate with a topical combination antibiotic, antifungal, and antibiotic ointment. Administer high-dose antibiotics (cephalexin, 22 mg/kg PO q8h, or enrofloxacin, 10 to 20 mg/kg PO q24h).

**Management**
If the tympanic membrane is not ruptured but appears swollen and erythematous, a myringotomy may need to be performed. If clinical signs of otitis media persist despite topical and systemic therapy, x-ray, CT, or MRI examination of the tympanic bullae may be required.

**Aural Hematoma**
Chronic shaking of the head and ears or aural trauma (bite wounds) causes disruption of the blood vessels and leads to the development of unilateral or bilateral aural hematomas. Aural hematomas are clinically significant because they cause patient discom
fort and are often caused by the presence of some other underlying problem such as otitis externa, atopy, or aural foreign bodies. Acute swelling of the external ear pinna with fluid is characteristic of an aural hematoma. In some cases, swelling can be so severe that the hematoma breaks open, bathing the patient and external living environment in blood.

**Immediate Action and Treatment**

When a patient has an aural hematoma, investigate the underlying cause. Perform a complete aural examination to determine whether an aural foreign body, otitis externa, or atopy is present. Carefully examine and gently clean the inner ear canal. Treat underlying causes.

**Management**

Management of an aural hematoma involves draining the hemorrhagic fluid from the aural tissue and tacking the skin down in multiple places to prevent reaccumulation of fluid until the secondary cause is resolved. Many techniques have been described to surgically tack down the skin overlying the hematoma. After the animal has been placed under general anesthesia, lance the hematoma down the middle with a scalpel blade and remove the fluid and blood clot. Tack down the skin with multiple through-and-through interrupted or mattress sutures through the ear. Some clinicians prefer to suture through and attach a sponge or length of x-ray film to the front and back of the ear for stabilization and support. More recently, a laser has been used to drill holes in the hematoma and tack the skin down in multiple areas. Compress the ear against the head with a compression bandage, whenever possible, for 5 to 7 days after the initial surgery, and then recheck the ear. The patient must wear an Elizabethan collar until the surgical wound and hematoma heal to prevent self-mutilation. Also systemically treat underlying causative factors, such as otitis externa, with antibiotics, antifungals, and steroids as indicated. Investigate and treat other underlying causes such as hypothyroidism or allergies.

**Additional Reading**


**ELECTRICAL INJURY AND ELECTRICAL SHOCK**

Electrical injury usually is observed in young animals after they have chewed on an electrical cord. Other causes of electrical injury include use of defective electrical equipment or being struck by lightning. Electrical current passing through the body can produce severe dysrhythmias, including supraventricular or ventricular tachycardia and first- and third-degree AV block. The electrical current also can produce tissue destruction from heat and electrothermal burns. Exposure to electrical current also commonly results in noncardiogenic pulmonary edema caused by massive catecholamine release and increase in pulmonary vascular pressures during the event. Ventricular fibrillation can occur, although that depends on the intensity and path of the electrical current and duration of contact.

Clinical signs of electrical injury include acute onset of respiratory distress with moist rales, and localized necrosis or thermal burns of the lips and tongue. Often the skin at the commissures of the mouth appears white or yellow and firm to the touch. Muscle fasciculations, loss of consciousness, and ventricular fibrillation may occur. Thoracic radiographs often reveal an increased interstitial to alveolar lung pattern in
The dorsocaudal lung fields. Noncardiogenic pulmonary edema can develop up to 24 to 36 hours after the initial incident. The first 24 hours are the most critical for the patient, and then prognosis improves.

The most important aspects of the treatment of the patient with noncardiogenic pulmonary edema are to minimize stress and to provide supplemental oxygen, with positive pressure ventilation, when necessary. Although treatment with vasodilators (low-dose morphine) and diuretics (furosemide) can be attempted, noncardiogenic pulmonary edema is typically resistant to vasodilator and diuretic therapy. Positive inotropes and pressor drugs may be necessary to treat shock and hypotension. Opioid drugs (morphine, hydromorphone, oxymorphone) may be useful in controlling anxiety until the pulmonary edema resolves. Administer broad-spectrum antibiotics (cefazolin; amoxicillin and clavulanic acid [Clavamox]) to treat thermal burns. Use analgesic drugs to control patient discomfort. If thermal burns are extensive and prohibit adequate food intake, place a feeding tube as soon as the patient’s cardiovascular and respiratory functions are stable and the patient can tolerate anesthesia.

Additional Reading
Mann FA: Electrical and lightning injuries. In Silverstein DC, Hopper K, editors: Small animal critical care medicine, St Louis, 2009, Elsevier.

EMERGENCIES OF THE FEMALE REPRODUCTIVE TRACT AND GENITALIA

Uterine Prolapse
Prolapse of the uterus occurs in the immediate postparturient period in the bitch and queen. Excessive straining during or after parturition causes the uterus to prolapse caudally through the vagina and vulva. Immediate intervention is necessary. Examine the bitch or queen for a retained fetus. Treatment consists of general anesthesia to replace the prolapsed tissue. If the uterus is edematous, physical replacement may be difficult or impossible. Application of a hypertonic solution such as hypertonic (7%) saline or dextrose (50%) to the exposed endometrium can help shrink the tissue. That, combined with gentle massage to stimulate uterine contraction and involution and lubrication with sterile lubricating jelly, can aid in replacement of the organ into its proper place. To ensure proper placement in the abdominal cavity and to prevent recurrence, perform an exploratory laparotomy and hysteropexy. Postoperatively, administer oxytocin (5 to 20 units IM) to cause uterine contraction. If the uterus contracts, it is usually not necessary to suture the vulva. Administer antibiotics postoperatively. Recurrence is uncommon, even with subsequent pregnancies.

If the tissue is damaged or too edematous to replace or if the tissue is devitalized, traumatized, or necrotic, perform an ovariohysterectomy. In some instances, replacement of the damaged tissue is not necessary before removal.

Pyometra
Pyometra occurs in both dogs and cats. The disease process occurs as a result of infection overlying cystic endometrial hyperplasia under the constant influence of progesterone. During the 2-month luteal phase after estrus or following copulation, artificial insemination, or administration of hormones (particularly estradiol or progesterone), the myometrium becomes relaxed and produces a quiescent environment for bacterial proliferation.

Clinical signs of pyometra are associated with the presence of bacterial endotoxin and sepsis. Affected animals become lethargic and anorexic early in the course of pyometra. Polyuria with secondary polydipsia is often present because of the influence of bacterial endotoxin on renal tubular concentration. If the cervix is open, purulent or mucoid
vaginal discharge may be observed. Later in the course of pyometra, vomiting, diarrhea, and progressive debilitation resulting from sepsis occur. Diagnosis is based on clinical signs in an intact queen or bitch and radiographic or ultrasonographic evidence of a fluid-filled tubular density in the ventrocaudal abdomen, adjacent to the urinary bladder (Figures 1-40 and 1-41).

Treatment of open and closed pyometra involves correction of fluid and electrolyte abnormalities, administration of broad-spectrum antibiotics, and ovariohysterectomy. Closed pyometra is a life-threatening septic condition. Open pyometra also can become
life-threatening and so should be treated aggressively. In closed pyometra, conservative medical therapy is not advised. Administration of prostaglandins and oxytocin do not reliably cause the cervix to open and can result in ascending infection from the uterus into the abdomen or uterine rupture, both of which can result in severe peritonitis.

For animals with an open pyometra, ovariohysterectomy is the most reliable treatment for chronic cystic endometrial hyperplasia. Although less successful than ovariohysterectomy, medical therapy may be attempted in breeding bitches as an alternative to surgery. The most widely used medical treatment in the breeding queen and bitch is administration of prostaglandin F<sub>2α</sub>. This drug has not been approved for use in the queen or bitch in the United States. To proceed with medical management of pyometra, first determine the size of the uterus. Start the patient on antibiotic therapy (ampicillin, 22 mg/kg IV q6h, or enrofloxacin, 10 mg/kg PO q24h). Administer the prostaglandin F<sub>2α</sub> (250 mcg/kg SQ q24h) for 2 to 7 days until the size of the uterus approaches normal. Measure serum progesterone concentrations if the bitch is in diestrus. As the corpus luteum degrades under the influence of prostaglandin F<sub>2α</sub>, serum progesterone levels will decline.

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**Acute Metritis**

Acute metritis is an acute bacterial infection of the uterus that typically occurs within 1 to 2 weeks after parturition. The most common organism observed in metritis is *E. coli* ascending from the vulva and vaginal vault. Sepsis can progress rapidly. Clinical signs of acute metritis include inability to nurse puppies, anorexia, lethargy, foul-smelling purulent-sanguineous vaginal discharge, vomiting, or acute collapse.

Physical examination may reveal fever, dehydration, and a turgid distended uterus. Septic inflammation will be observed on vaginal cytologic examination. An enlarged uterus can be observed on abdominal radiographs and ultrasonography.

Treatment of acute metritis is directed at restoring hydration status with intravenous fluids and treating the infection with antibiotics. Because the primary cause of metritis is *E. coli* infection, start enrofloxacin therapy (10 mg/kg IV or PO once daily). As soon as the patient’s cardiovascular status is stable enough for anesthesia, perform an ovariohysterectomy. If the patient’s condition is not critical and the animal is a valuable breeding bitch, medical therapy can be attempted. Medical management of acute bacterial metritis includes administration of oxytocin (5 to 10 units q3h for three treatments) or administration of prostaglandin F<sub>2α</sub> (250 mcg/kg/day for 2 to 5 days) to evacuate the uterine exudate and increase uterine blood flow. Either drug should be used concurrently with antibiotics.

**Uterine Rupture**

Rupture of the gravid uterus is rare in cats and dogs but has been reported. Uterine rupture may occur as a consequence of parturition or result from blunt abdominal trauma. Fetuses expelled into the abdominal cavity may be resorbed but more commonly cause the development of peritonitis. If fetal circulation is not disrupted, the fetus actually may live to term. Uterine rupture is an acute surgical emergency. An ovariohysterectomy with removal of the extrauterine puppies and membranes is recommended. If only one horn of the uterus is affected, a unilateral ovariohysterectomy can be performed to salvage the remaining unaffected puppies and preserve the breeding potential of the valuable bitch. If uterine rupture occurs because of pyometra, peritonitis is likely, and copious peritoneal lavage should be performed at the time of surgery. The patient should be placed on 7 to 14 days of antibiotic therapy (amoxicillin or amoxicillin and clavulanic acid [Clavamox] with enrofloxacin).
VAGINAL PROLAPSE

Vaginal prolapse occurs from excessive proliferation and hyperplasia of vaginal tissue while under the influence of estrogen during proestrus (Figure 1-42). The hyperplastic tissue usually recedes during diestrus but reappears with subsequent heat cycles. Vaginal prolapse can be confused with vaginal neoplasia. The former condition occurs primarily in younger animals, whereas the latter condition occurs primarily in older animals. Treatment for vaginal hyperplasia or prolapse generally is not required if the tissue remains within the vagina. The proliferation can lead to dysuria or anuria, however. In some cases, the tissue becomes dried out and devitalized or becomes traumatized by the animal. Such extreme cases warrant immediate surgical intervention. The treatment for vaginal prolapse consists of ovariohysterectomy to remove the influence of estrogen, placement of an indwelling urinary catheter if the patient is dysuric, and protection of the hyperplastic tissue until it recedes on its own. Although surgical resection of the hyperplastic tissue has been recommended, excessive hemorrhage after removal can occur, and so the procedure should not be attempted. The patient should wear an Elizabethan collar at all times to prevent self-mutilation. Administer broad-spectrum antibiotics for a minimum of 7 to 14 days or until the hyperplastic tissue recedes. Keep the tissue clean with saline solution.

EMERGENCIES OF PREGNANCY AND PARTURITION

dystocia

Dystocia, or difficult birth, can occur in the dog and cat but is more common in the dog. A diagnosis of dystocia is made based on the time of onset of visible labor and the time in which the last puppy or no puppy has been born, the intensity and timing of contractions, the timing of when the amniotic membranes first appear, the condition of the bitch, and the timing of gestation. Causes of dystocia can be maternal or fetal and include primary or secondary uterine inertia, narrowing of the pelvic canal, hypocalcemia, psychological disturbances, and uterine torsion. Maternal-fetal disproportion, or large fetus size in relation to the bitch or queen, also can result in dystocia (Box 1-40).

Obtain an abdominal radiograph for all cases of suspected dystocia at the time of presentation to determine the size of the fetus, the presentation of the fetus (both anterior and posterior presentations can be normal in the bitch or queen, but fetal malpositioning can cause dystocia), and whether there is radiographic evidence of a uterine rupture.

Figure 1-42: Vaginal prolapse in a bitch.
or torsion. If maternal-fetal disproportion, uterine torsion, or uterine rupture is observed, take the patient immediately to surgery. If the puppies or kittens are in a normal position for birth, medical management can be attempted.

Clip the perineum and aseptically scrub it. Wearing sterile gloves, insert a lubricated finger into the vagina and palpate the cervix. Massage (or “feather”) the dorsal wall of the vagina to stimulate contractions. Place an intravenous catheter, and administer oxytocin (2 to 20 units IM), repeating up to three times at 30-minute intervals. In some cases, hypoglycemia or hypocalcemia can contribute to uterine inertia. Administration of a calcium-containing solution (lactated Ringer’s solution) with 2.5% dextrose is advised. Alternately, administer 10% calcium gluconate (100 mg/5 kg IV slowly). If labor has not progressed after 1 hour, immediately perform a cesarean section.

**Uterine Torsion**

Uterine torsion is an uncommon emergency seen in the gravid and nongravid uterus and has been reported in dogs and cats. The onset of clinical signs of abdominal pain and straining as if to whelp or queen or defecate is usually acute and constitutes a surgical emergency. In some cases there may have been a history of delivery of a live or dead fetus. Vaginal discharge may or may not be present. Radiographs or ultrasound examination reveals a fluid-filled or air-filled tubular density in the ventral abdomen. Treatment consists of placing an intravenous catheter, stabilizing the patient’s cardiovascular status with intravenous fluids and sometimes blood products, and performing an immediate ovariohysterectomy. If there are viable fetuses, the uterus should be delivered en mass and the puppies or kittens delivered.

**Spontaneous Abortion**

The expulsion of one or more fetuses before term is known as spontaneous abortion. In dogs and cats, it is possible to expel or abort one or more fetuses and still carry viable fetuses to term and deliver normally. Clinical signs of spontaneous abortion include vaginal discharge and abdominal contractions. In some cases the fetus is found, or there may be evidence of fetal membranes or remnants. Causes of spontaneous abortion in dogs include *Brucella canis*, herpesvirus, coronavirus, and toxoplasmosis. In cats, herpesvirus, coronavirus, and FeLV can cause spontaneous abortion. In both species, trauma, hormonal factors, environmental pathogens, drugs, and fetal factors also can result in spontaneous abortion.

**Pregnancy Termination in the Bitch and Queen**

The safest method of pregnancy termination in the bitch or queen is by performing an ovariohysterectomy. Oral diethylstilbestrol is not an effective mechanism of pregnancy termination in the bitch. A so-called mismating shot, an injection of estradiol cypionate (0.02 mg/lb IM) is effective at causing termination of an early pregnancy but can be associated with severe side effects, including bone marrow suppression and pyometra. Estradiol cypionate is not approved for use in the bitch or queen and is not recommended.

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**Box 1-40 Diagnostic Criteria for Dystocia**

- Fetus lodged in birth canal
- Presence of vaginal stricture or band of tissue preventing normal delivery
- Prolonged gestation (>70 days)
- Drop in rectal temperature (<100° F) with no evidence of labor
- Green vaginal discharge and no evidence of delivery of fetus
- No puppies delivered after 2 to 3 hours of a visible amniotic sac
- Strong contractions with no puppy or kitten delivered after 30 minutes
- Weak, infrequent contractions with no puppy delivered in 4 hours from onset of labor
- More than 2 hours have passed with no evidence of further contraction or delivery of a puppy
- Signs of systemic illness or pain: depression, weakness, sepsis
Prostaglandin $F_{2\alpha}$ is a natural abortifacient in the bitch if treatment is started within 5 days of cytologic evidence of diestrus (noncornified epithelium on a vaginal smear). The prostaglandin $F_{2\alpha}$ causes lysis of the corpora lutea and a rapid decline in progesterone concentration. The prostaglandin $F_{2\alpha}$ is administered for a total of eight injections (250 mcg/kg q12h for 4 days), along with atropine (100 to 500 mcg/kg SQ). Side effects can occur within 5 to 40 minutes of injection and include restlessness, panting, salivation, abdominal pain, urination, vomiting, and diarrhea. Walking the patient for 20 to 30 minutes after each treatment sometimes decreases the intensity of the reactions.

Bitches in the first half of the pregnancy often resorb the embryos. If prostaglandin $F_{2\alpha}$ is administered in the second half of the pregnancy, the fetuses are aborted within 5 to 7 days of treatment. Measure serum progesterone concentrations at the end of treatment to ensure complete lysis of the corpus luteum. Prostaglandin $F_{2\alpha}$ is not approved for pregnancy termination in the bitch.

In cats, prostaglandin $F_{2\alpha}$ can terminate pregnancy after day 4 of gestation. Prostaglandin $F_{2\alpha}$ should be used only in healthy queens (100 to 250 mcg/kg SQ q24h for 2 days). Side effects in the queen are similar to those observed in the bitch but typically have a shorter duration (2 to 20 minutes). Prostaglandin $F_{2\alpha}$ is not approved for use in cats in the United States. The use of prostaglandin $F_{2\alpha}$ does not preclude breeding and pregnancy at a later date.

**Additional Reading**


**EMERGENCIES OF THE MALE GENITALIA AND REPRODUCTIVE TRACT**

**Figure 1-43** illustrates conditions of the male genitalia and reproductive tract that require emergent care.

**Scrotal Trauma**

In the dog and cat the majority of injuries to the scrotum are associated with animal fights or shearing and abrasive injuries sustained in accidents involving automobiles. Scrotal injuries should be categorized as superficial or penetrating.

Treatment of superficial injuries to the scrotum includes cleaning the wound with dilute antimicrobial cleanser and drying it. Administer antiinflammatory doses of steroids (prednisolone, 0.5 to 1.0 mg/kg PO q12-24h) or NSAIDs (carprofen, 2.2 mg/kg PO q12h in dogs) for the first several days after scrotal injury to prevent or treat edema. Administer topical antibiotic ointment until the wound heals. In most cases, place an Elizabethan...
collar to prevent self-mutilation. Prognosis is generally favorable; however, semen quality may be affected for months after injury because of scrotal swelling and increased scrotal temperature.

Penetrating injuries to the scrotum are more serious and are associated with severe swelling and infection. Surgically explore and debride penetrating scrotal wounds. Administer systemically effective antibiotics and analgesics. In extreme cases, particularly those that involve the testicle, consider castration and scrotal ablation. 

**Acute Scrotal Dermatitis**

Scrotal dermatitis is common in intact male dogs and can be associated with direct physical injury, self-infliction from licking, chemical irritation, burns, or contact dermatitis. In affected animals, the scrotum can become extremely inflamed, swollen, and painful. If left untreated, pyogranulomatous dermatitis can develop.

Make an attempt to determine whether an underlying systemic illness is present that could predispose the animal to scrotal dermatitis. Widespread vasculitis with scrotal edema, pain, fever, and dermatitis has been associated with *R. rickettsii* infection (Rocky Mountain spotted fever). *B. canis* also has been associated with scrotal irritation and dermatitis. If scrotal dermatitis follows from an infectious cause, empiric use of glucocorticosteroids potentially can make the condition worse by suppressing immune function. Empiric treatment with antibiotics also potentially can confound making an accurate diagnosis.

Treatment of scrotal dermatitis is to eliminate predisposing causes, if possible. Keep an Elizabethan collar in place at all times to prevent self-mutilation. Bathe the scrotum with a mild antimicrobial soap and dry it to remove any offending chemical irritants.
Topical medications, including tar shampoo, tetracaine, neomycin, and petroleum, can cause further irritation and are contraindicated. Use oral or parenteral administration of glucocorticosteroids or NSAIDs to control discomfort and inflammation.

**Scrotal Hernia**

Scrotal hernias occur when the contents of the abdomen (intestines, fat, mesentery, omentum) protrude through the inguinal ring into the scrotal sac. Like inguinal hernias, scrotal hernias are surgical emergencies only if intestinal incarceration or vascular obstruction occurs. Differential diagnoses for scrotal hernias include epididymitis, orchitis, testicular torsion, and testicular neoplasia.

Definitive therapy for a scrotal hernia involves exploratory laparotomy and surgical reduction of the contents of the hernia, surgical correction of the rent in the inguinal ring, and castration.

**Testicular Trauma**

Trauma to the epididymis or testicle can cause testicular pain and swelling of one or both testes. Treat penetrating trauma to the testicle by castration to prevent infection and self-mutilation. Administer oral antibiotics (amoxicillin or amoxicillin-clavulinate) for 7 to 10 days after the injury. Nonpenetrating injuries to the scrotum and testicle rarely may cause acute testicular hemorrhage or hydrocele formation. Palpation of the affected area often reveals a peritesticular, soft, compliant area. Treatment consists of cool compresses on the scrotum and testicle and administration of antiinflammatory doses of glucocorticosteroids or NSAIDs. If the swelling does not resolve spontaneously in 5 to 7 days, consider surgical exploration and drainage. Increased scrotal temperature and testicular inflammation can affect semen quality for months after the initial incident.

**Testicular Torsion**

Testicular torsion, or torsion of the spermatic cord, causes rotation of the testicle, ultimately causing obstruction to venous drainage. Testicular torsion often is associated with a neoplastic mass of a retained testicle within the abdomen but also can be observed with nonneoplastic testes located within the scrotum. The predominant clinical signs are pain, stiff stilted gait, and the presence of an abnormally swollen testicle (if located within the scrotum). If an intraabdominal testicular torsion is present, pain, lethargy, anorexia, and vomiting can occur (see Acute Condition in the Abdomen). An intraabdominal mass may be palpable. Perform an abdominal or testicular ultrasound, preferably with color flow Doppler to evaluate perfusion to the testicle. Treatment involves surgical removal of the involved testes.

**Infectious Orchitis and Epididymitis**

Bacterial infections of the testicle or epididymis most commonly are caused by ascending infections of the normal bacterial flora of the prepuce or urethra. Common inhabitants include *E. coli*, *S. aureus*, *Streptococcus* species, and *Mycobacterium canis*. *B. canis* and *R. rickettsii* are also capable of causing orchitis and epididymitis in the dog. Clinical signs of orchitis or epididymitis include testicular enlargement, stiff stilted gait, and reluctance to walk. Physical examination often reveals a fever and self-induced trauma to the scrotum from licking or chewing at the inflamed area. Collect a semen sample by ejaculation, and culture it to identify the causative organism. Alternately, collect samples by needle aspiration of the affected organ(s) and test serologically for *B. canis*.

Treatment of infectious orchitis involves a minimum of 3 to 4 weeks of specific antimicrobial therapy, based on culture and susceptibility testing, whenever possible. If a bacterial culture cannot be performed, initiate fluoroquinolone therapy (enrofloxacin, 10 mg/kg PO q24h). Doxycycline (5 mg/kg PO bid for 7 days) has been shown to suppress but not eradicate *B. canis* infection. Testicular inflammation and increased temperature can affect sperm quality for months after infection.
**Acute Prostatitis**

The most common causes of acute prostatitis are associated with acute bacterial infection (*E. coli*, *Proteus* species, *Pseudomonas* species, and *Mycoplasma* species). Less common causes include fungal infection (*Blastomyces dermatitidis*) or anaerobic bacterial infection.

Acute prostatitis is characterized by fever, caudal abdominal pain, lethargy, anorexia, blood in the ejaculate, hematuria, dyschezia, and occasionally stranguria or dysuria. The patient often appears in pain and depressed and may be dehydrated on physical examination. Symmetric or asymmetric prostatomegaly and prostate pain may be evident on rectal palpation. In severely affected dogs, clinical signs of tachycardia, hyperemic or injected mucous membranes, bounding pulses, lethargy, dehydration, and fever may be present because of sepsis. Death can occur within 2 days if a prostatic abscess ruptures.

Diagnosis of acute prostatitis is confirmed based on the presenting clinical signs, neutrophilic leukocytosis (with or without a left shift), and positive urine culture results. Prostatic samples may be obtained from the prostatic portion of the ejaculate, prostatic massage, urethral discharge, urine, or (less commonly) prostatic aspirate. Although semen samples can yield positive bacterial cultures, dogs with acute prostatitis are often unwilling to ejaculate. Radiography may reveal an enlarged prostate, but this alone does not confirm the diagnosis of prostatitis. Abdominal ultrasound often reveals prostatic abscessation and allows for the collection of samples from the affected area(s) via prostatic aspirate. Aspiration of the affected tissue potentially can wick infection into periprostatic tracks. Cytologic examination of the patient's ejaculate or prostatic wash from a dog with acute prostatitis reveals numerous inflammatory cells, and such samples may contain bacterial organisms.

The treatment of a patient with acute prostatitis is directed at correcting dysuria and constipation associated with prostatic enlargement. Enrofloxacin (10 mg/kg PO sid) can penetrate the inflamed prostatic tissue and is effective in treating gram-negative and *Mycoplasma* species infections. Ciprofloxacin does not appear to penetrate prostatic tissue as readily. Alternatives to enrofloxacin therapy are trimethoprim-sulfamethoxazole (30 mg/kg PO q12h) or chloramphenicol (25 to 50 mg/kg PO q8h) for a minimum of 2 to 3 weeks. Castration is recommended because benign prostatic hyperplasia may be a predisposing factor in the development of acute prostatitis. Do not perform castration until the patient has been on antibiotic therapy for a minimum of 7 days, to prevent the surgical complication of scirrhous cords. Finasteride (Proscar, 0.1–0.5 mg/kg PO q24h), an antiandrogen 5α-reductase inhibitor, may help reduce the size of prostatic tissue until the effects of castration are observed. If a prostatic abscess is present, perform marsupialization, surgical drainage, or ultrasonographic drainage. Surgical therapy is associated with a large incidence of complications, including incontinence, chronic drainage from fistulas and stomas, septic shock, and death.

**Os Penis Fracture**

Fracture of the os penis is an uncommon condition encountered in male dogs. Os penis fractures can occur with minimal soft tissue damage but cause hematuria and dysuria. On physical examination, urethral obstruction and crepitus in the penis are found. A lateral abdominal radiograph is usually sufficient to document the fracture. Treatment consists of conservative therapy, in most cases, and consists primarily of analgesia administration. If the urethra also is damaged, place a urethral catheter for 5 to 7 days to allow the urethral mucosa to heal. Fractures of the os penis that are comminuted or severe enough to cause urethral obstruction require open reduction and fixation, partial penile amputation, or antescrotal (prescrotal) urethrostomy.

**Laceration**

Lacerations of the penis cause significant bleeding because of the extensive vascular supply to the penis. Dogs and cats tend to lick penile lacerations and prevent adequate clot formation. Sedation or general anesthesia often is required to evaluate and treat the laceration. After sedation or general anesthesia, place a urinary catheter and examine the penis under
a stream of cold water. Small lacerations can be managed with cold compresses and one to several absorbable sutures. Extensive suturing usually is not required. Prevent erection by isolating the patient from females in estrus or allowing excitement or excessive activity. Place an Elizabethan collar to prevent self-mutilation. Initiate systemic antibiotic therapy to prevent infection.

**Paraphimosis**

The inability to withdraw the penis into the prepuce in male dogs or cats is known as paraphimosis. Paraphimosis usually develops after an erection in young male dogs and in older dogs after coitus. Mucosal edema, hemorrhage, self-mutilation, and necrosis requiring penile amputation can occur if left untreated. Treatment consists of applying cold water to the penis and reducing edema with application of an osmotic substance such as sugar. Examine the base of the penis for hair rings that can prevent retraction of the penis into the prepuce. Rinse the penis carefully with cold water and lubricate it with sterile lubricant and replace it into the prepuce. If the penis cannot be reduced easily into the prepuce, anesthetize the patient and make a small incision at the lateral aspect of the preputial opening. Replace the penis and close the incision with absorbable suture. Place a purse-string suture and leave it in place for several days to prevent recurrence. Instill topical antimicrobial ointment with steroids into the prepuce several times a day. In severe cases, a urinary catheter may need to be placed to prevent urethral obstruction, until penile swelling and edema resolve. Place an Elizabethan collar to prevent excessive licking during the healing process.

**Urethral Prolapse**

Prolapse of the distal urethra is a condition usually confined to intact male English Bulldogs, although isolated incidences also have been reported in Yorkshire and Boston Terriers. The exact cause of this condition is unknown but usually is associated with a condition that causes increased intraabdominal pressure or urethral straining, including sexual excitement, coughing, vomiting, obstructed airway or brachycephalic airway syndrome, urethral calculi, genitourinary tract infection, and masturbation.

The urethral prolapse usually appears as a mushroom-tip congested, irritated mass at the end of the penis that may or may not bleed (Figure 1-44). In some cases, bleeding occurs or worsens with sexual excitement. Clinical signs associated with the prolapsed urethra

![Image of urethral prolapse](https://via.placeholder.com/150)

**Figure 1-44:** Example of urethral prolapse. This condition is most commonly observed in intact male Bulldogs, although it has been associated with neoplasia and urethral calculi in other breeds.
include excessive licking of the prepuce, stranguria, and preputial bleeding. Once the mass is observed, other differential diagnoses include transmissible venereal tumor, urethral polyp, trauma, urethritis, and neoplasia. In most cases, however, the prolapse occurs in intact young dogs, making neoplastic conditions less likely.

Treatment for urethral prolapse should occur at the time of diagnosis to prevent self-induced trauma and infection. Immediate therapy includes manual reduction of the prolapsed tissue and placement of a purse-string suture around an indwelling urinary catheter. The purse-string suture can remain in place for up to 5 days until definitive repair. Until the time of surgery, place an Elizabethan collar on the patient to prevent self-mutilation. Several forms of surgical correction have been described. In some cases, surgical resection of the prolapsed tissue with apposition of the urethral and penile mucosa can be attempted. More recently, a technique involving placement of several mattress sutures to reduce and secure the prolapsed tissue has been described. Recurrence of prolapse can occur with either technique, particularly if the inciting event recurs. Because there may be a genetic predisposition in this breed and because the prolapse can recur with sexual excitement, neutering should strongly be recommended.

**Additional Reading**


**ENVIRONMENTAL AND HOUSEHOLD EMERGENCIES**

**Frostbite**

Local freezing or frostbite most commonly affects the peripheral tissues of the ears, tail, paws, and genitalia that are sparsely covered with fur, are poorly vascularized, and may have been traumatized previously by cold. Clinical signs of frostbite are paleness and appearance of a blanched pink to white discoloration to the skin. The skin also may appear black and necrotic.

**Immediate Action and Treatment**

Immediate treatment consists of slowly rewarming the affected area with moist heat at 29.5° C (85° F) or by immersion in warm water baths. Analgesics may be required to alleviate patient discomfort. Carefully dry the injured areas and protect them from further trauma.

**Management**

The use of prophylactic antibiotics is controversial because it can promote resistant bacterial infection. Use of antibiotics should be based on the presence of infection. Treatments that are ineffective and may be harmful include rubbing of the affected areas, pressure bandages, and ointments. Corticosteroids can decrease cellular immunity and promote infection and are therefore contraindicated. Many frostbitten areas that appear nonviable can regain function gradually. Use care when removing areas of necrotic tissue. Affected areas may take several days to a week before fully manifesting areas of demarcation between healthy viable and necrotic nonviable tissue.
**Hypothermia**

Chilling of the entire body from exposure or immersion in extremely cold water results in a decrease in core body temperature and physiologic processes that becomes irreversible when the body temperature falls below 24° C (75° F). Mild hypothermia occurs at 32° to 37° C, moderate hypothermia at 28° to 32° C, and severe hypothermia below 28° C. The duration of exposure and the general condition of the animal influences its ability to survive.

Clinical signs and consequences associated with hypothermia include shivering, vasoconstriction, mental depression, hypotension, sinus bradycardia, hypoventilation with decreased respiratory rate, increased blood viscosity, muscle stiffness, atrial and ventricular irritability, decreased level of consciousness, decreased oxygen consumption, metabolic (lactic) acidosis, respiratory acidosis, and coagulopathies including DIC.

**Immediate Action and Treatment**

If the animal is breathing, administer warm, humidified oxygen at 4 to 10 breaths per minute. If the animal is not breathing or is severely hypoventilating, endotracheal intubation with mechanical ventilation may be necessary. Place an intravenous catheter and infuse warmed crystalloid fluids. If the blood glucose is less than 60 mg/dL, add supplemental dextrose (2.5%) to the crystalloid fluids. Monitor the core body temperature and ECG closely. Rewarming should occur in the form of external circulating warm water blankets, radiant heat, and circulating warm air blankets (Bair Hugger). Never use a heating pad, to avoid iatrogenic thermal burn injury. Severe hypothermia may require core rewarming in the form of intraperitoneal fluids (10 to 20 mL of lactated Ringer’s solution per kilogram, warmed to 39.4° C [103° F]). Place a temporary peritoneal dialysis catheter, and repeat the dialysis every 30 minutes until the patient’s body temperature reaches 36.6° to 37.7° C (98° to 100° F).

**Management**

The body temperature should rise slowly, ideally no more than 1° F per hour. Because the response of the body to drugs is unpredictable, avoid administering drugs whenever possible, until the body temperature returns to normal. Complications observed during rewarming include DIC, cardiac dysrhythmias including cardiac arrest, pneumonia, pulmonary edema, CNS edema, ARDS, and renal failure.

**Hypothermia and Heat-Induced Illness (Heat Stroke)**

Heat stroke and heat-induced illness in dogs can be associated with excessive exertion, exposure to high environmental temperatures, stress, and other factors that cause an inability to dissipate heat. Brachycephalic breeds, obese animals, dogs with laryngeal paralysis, and older animals with cardiovascular disease can be particularly affected. Hyperthermia is defined as a rectal temperature of 41° to 43° C (105° to 110° F). Clinical signs of hyperthermia include congested hyperemic mucous membranes, tachycardia, and panting. More severe clinical signs include collapse (heat prostration), ataxia, vomiting, diarrhea, hyper salivation, muscle tremors, loss of consciousness, and seizures. Heat-induced illness can affect all major organ systems in the body because of denaturation of cellular proteins and enzyme activities, inappropriate shunting of blood, hypotension, decreased oxygen delivery, and lactic acidosis. Cardiac dysrhythmias, interstitial and intracellular dehydration, intravascular hypovolemia, central nervous dysfunction, slough of gastrointestinal mucosa, oliguria, and coagulopathies can be seen as organ function declines. Excessive panting can result in respiratory alkalosis. Poor tissue perfusion results in metabolic acidosis. Loss of water in excess of solutes such as sodium and chloride can lead to a free water deficit and severe hypernatremia. A marked increase in PCV occurs because of the free water loss. Severe abnormalities in electrolytes and pH can lead to cerebral edema and death.

**Immediate Action and Treatment**

Treatment goals for the patient with heat-induced illness are to lower the core body temperature and support cardiovascular, respiratory, renal, gastrointestinal, neurologic, and hepatic functions. At the scene the veterinarian or caretaker can spray the animal with tepid (not
cold) water. Immersion in cold water or ice baths is absolutely contraindicated. Cold water and ice will cause extreme peripheral vasoconstriction, inhibiting the patient’s ability to dissipate heat through conductive and convective cooling mechanisms. As a result, core body temperature will continue to rise despite the good intentions of attendants at the scene. Animals that have been cooled to the point of hypothermia have a worse prognosis. Once the animal has been presented to the veterinarian, the goal is to cool the animal’s body temperature with towels soaked in tepid water, cool intravenous fluids, and fans until the temperature has decreased to 103° F. Organ system monitoring and support are based on the severity and duration of the heat stroke and the ability of the body to compensate and respond to treatment.

Management
Management of the patient with heat-induced illness involves prompt aggressive cooling without being overzealous and creating iatrogenic hypothermia. Administer cool intravenous crystalloid fluids to replenish volume and interstitial hydration and correct the patient’s acid-base and electrolyte abnormalities. Management consists of Rule of 20 monitoring (see Rule of 20), taking care to evaluate, restore, and maintain normal cardiac rhythm, BP, urine output, and mentation. Administer antibiotics if there are any signs of gastrointestinal bleeding that will predispose the patient to bacterial translocation. Monitor results of baseline chemistry tests including a complete blood count, biochemical panel, platelet count, coagulation tests, and urinalysis. Treat coagulopathies including DIC aggressively and promptly (see also Disseminated Intravascular Coagulation). Severe changes in mentation including stupor or coma worsen a patient’s prognosis. After initial therapy, monitor the patient for a minimum of 24 to 48 hours for secondary organ damage, including renal failure, myoglobinuria, cerebral edema, and DIC. Dogs that are going to die of heat-induced illness usually die within the first 24 hours. Animals that survive longer than 24 hours have a more favorable prognosis.

Additional Reading

Malignant Hyperthermia
Malignant hyperthermia is a syndrome that involves impaired muscular calcium metabolism. Malignant hyperthermia has been recognized as a consequence of exertion in Labrador Retrievers and in sensitized animals placed under anesthesia. Clinical signs of malignant hyperthermia are severe muscle spasm or fasciculation, unstable BP, metabolic or respiratory acidosis, and a rapidly increased end-tidal carbon dioxide under anesthesia. The patient’s temperature often rises above 42° C. Cellular death can result if the malignant hyperthermia is not recognized and treated rapidly.
Immediate Action and Treatment
Immediate treatment consists of cooling the patient with cooling measures as for hyperthermia and heat-induced illness (see the previous discussion) and eliminating the cause (e.g., exertion, anesthesia, or neuromuscular blockers such as succinylcholine). If the patient is under general anesthesia, hyperventilate the patient to help eliminate carbon dioxide and respiratory acidosis. Administer dantrolene sodium (1 to 2 mg/kg IV) to stabilize the sarcoplasmic reticulum and decrease its permeability to calcium.

Management
Animals with malignant hyperthermia should avoid any predisposing factors, including exertion, hyperthermia, and anesthesia. After an episode of malignant hyperthermia, administer crystalloid fluids IV to aid in the elimination of myoglobin. Monitor renal function closely for myoglobinuria and pigment damage to the renal tubular epithelium. Monitor and correct acid-base and electrolyte changes.

Additional Reading

Snakebite: Nonpoisonous
Sometimes it is difficult to assess whether an animal has been bitten by a poisonous or nonpoisonous snake. In Colorado, the bull snake closely resembles the prairie rattlesnake. Both snakes make similar noise and can be alarming if noticed on a hike or in the backyard. Whenever possible, identify the offending reptile but never risk being bitten. Know what types of venomous creatures are in the geographic area of the practice.

If an animal has been bitten by a nonpoisonous snake, usually the bite marks are small with multiple small tooth punctures, and the bite is relatively nonpainful. Usually local reaction is negligible. However, large boas or pythons also can inflict large crushing injuries that can cause severe trauma, including bony fractures.

Treatment for a nonpoisonous snakebite involves clipping the bite wound and carefully cleaning the area with antimicrobial scrub solution. Broad-spectrum antibiotics (e.g., amoxicillin-clavulanate, 16.25 mg/kg PO q12h) are indicated because of the extensive bacterial flora in the mouths of snakes. Monitor all snakebite victims for a minimum of 8 hours after the incident, particularly when the species of the offending reptile is in question. If clinical signs of envenomation occur, modify the patient’s treatment appropriately and aggressively.

Snakebite: Poisonous
The two major groups of venomous snakes in North America are the pit viper and the coral snake. All venomous snakes are dangerous. The severity of any given bite depends on the toxicity of the venom, the amount of venom injected, the site of envenomation, the size of the animal bitten, and the time from bite and envenomation to appropriate medical intervention.

Pit Viper Envenomation
The majority of reptile envenomations in the United States are inflicted by pit vipers, including the water moccasin (cottonmouth), copperhead, and numerous species of rattlesnakes. Pit vipers are characterized by a deep pit located between the eye and nostril, elliptic pupils, and retractable front fangs (Figure 1-45).

Localized clinical signs of pit viper envenomation may include the presence of bleeding puncture wounds, local edema close to puncture wounds, immediate severe pain or collapse, edema, petechiae, and ecchymosis with subsequent tissue necrosis. Systemic signs of pit viper envenomation may include hypotension, shock, coagulopathies, lethargy, weakness, muscle fasciculations, lymphangitis, rhabdomyolysis, and neurologic signs including respiratory depression and seizures. Neurologic signs largely are associated with
envenomation by the Mojave and canebrake rattlesnakes, although a potent neurotoxin, Mojave toxin A, also has been identified in other subspecies of rattlesnake.

Clinical signs of envenomation may take several hours to appear. Hospitalize all suspected victims and monitor them for a minimum of 24 hours. The severity of envenomation cannot be judged solely on the basis of local tissue reaction. First aid measures by animal caretakers do little to prevent further envenomation. The most important aspect of initiating therapy is to transport the animal to the nearest veterinary emergency facility.

**Immediate Action and Treatment**

To determine whether an animal has been envenomated by a pit viper, examine a peripheral blood smear for the presence of echinocytes. Echinocytes will appear within 15 minutes of envenomation and may disappear within 48 hours. Other treatment should be initiated

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**Figure 1-45:** Characteristics of poisonous snakes. (From Parrish HM, Carr CA: Bites by copperheads (*Ancistrodon contortrix*) in the United States, *JAMA* 201:927, 1967.)
as rapidly and aggressively as possible, although controversy exists regarding whether some therapies are warranted. The mainstay of therapy is to improve tissue perfusion with intravenous crystalloid fluids, prevent pain with judicious use of analgesic drugs, and, when necessary, reverse or negate the effects of the venom with antivenin. Because pit viper venom consists of multiple fractions, treat each envenomation as a complex poisoning.

Obtain vascular access and administer intravenous crystalloid fluids (one fourth of a calculated shock dose) according to the patient’s perfusion parameters of heart rate, BP, and capillary refill time (see also Management of the Shock Patient and Fluid Therapy). Opioid analgesics are potent and should be administered at the time of presentation. (See also Pharmacologic Means to Analgesia: Major Analgesics.)

The use of diphenhydramine and glucocorticoids has fallen out of favor, as there is no benefit to their use, and in humans the use of glucocorticoids with snakebite has been shown to increase patient morbidity and mortality.

Polyvalent antivenin is necessary in many cases of pit viper envenomation, except in most cases of prairie rattlesnake (*Crotalus viridis viridis*) envenomation in Colorado. A recent study demonstrated no difference in outcome with or without the use of antivenin in cases of prairie rattlesnake envenomation. Clinically, however, patients that receive antivenin are more comfortable and leave the hospital sooner than those that do not receive antivenin. The exact dose of antivenin is unknown in small animal patients. Administer a dose of at least one vial of antivenin to neutralize circulating venom. Mix antivenin with a swirling, rather than a shaking motion, to prevent foaming. Mix the antivenin with a 250-mL bag of 0.9% saline, and then administer it slowly over a period of 2 to 4 hours. In smaller patients the antivenin can be diluted in a smaller volume of fluid, depending on the patient’s body weight, and given over the same period of time. During the administration of antivenin, monitor the animal closely for clinical signs of angioneurotic edema, urticaria, tachyarrhythmias, vomiting, diarrhea, and weakness during the infusion. Administration of antivenin into the bite site is relatively contraindicated and ineffective because uptake is delayed, and systemic effects are the more life-threatening.

**Management**

Management of pit viper envenomation largely involves maintenance of normal tissue perfusion with intravenous fluids, decreasing patient discomfort with analgesia, and negating circulating venom with antivenin. Hydrotherapy to the affected bite site with tepid water is often soothing to the patient. The empiric use of antibiotics is controversial but is recommended because of the favorable environment created by a snakebite (i.e., impregnation of superficial gram-positive bacteria and gram-negative bacteria from the mouth of the snake into a site of edematous necrotic tissue). Administer amoxicillin-clavulanate (16.25 mg/kg PO q12h) or cepalexin (22 mg/kg PO q8h). Also consider administration of NSAIDs (carprofen, 2.2 mg/kg PO q12h). Monitor the patient closely for signs of local tissue necrosis and the development of thrombocytopenia and coagulopathies including DIC (see Management of Disseminated Intravascular Coagulation). Treat coagulopathies aggressively to prevent end-organ damage.

**Coral Snake Envenomation**

Coral snakes are characterized by brightly colored bands encircling the body, with red and black separated by yellow. “Red on black, friend of Jack; red on yellow, kill a fellow.” Types of coral snakes include the Eastern coral, Texas coral, and Sonoran coral snakes. Clinical signs of coral snake envenomation may include small puncture wounds, transient initial pain, muscle fasciculations, weakness, difficulty swallowing or dysphagia, ascending lower motor neuron paralysis, miotic pinpoint pupils, bulbar paralysis, respiratory collapse, and severe hemolysis. Clinical signs may be delayed for as long as 18 hours after the initial bite.
Immediate Action and Treatment

Immediate treatment with antivenin is necessary in cases of coral snake envenomation before the clinical signs become apparent, whenever possible. Support respiration during paralysis with mechanical ventilation. Secure the patient’s airway with a cuffed endotracheal tube to prevent aspiration pneumonia.

Management

Clinical signs will progress rapidly once they develop. Rapid administration with antivenin is the mainstay of therapy in suspected coral snake envenomation. Respiratory and cardiovascular support should occur with mechanical ventilation and intravenous crystalloid fluids. Keep the patient warm and dry in a quiet place. Turn the patient every 4 to 6 hours to prevent atelectasis and decubitus ulcer formation. Maintain cleanliness using a urinary catheter and closed urinary collection system. Perform passive range of motion and deep muscle massage to prevent disuse atrophy of limb muscles and function. Treat aspiration pneumonia aggressively with broad-spectrum antibiotics (ampicillin, 22 mg/kg IV q6h, with enrofloxacin, 10 mg/kg IV q24h, and then change to oral once tolerated and the patient is able to swallow) for 2 weeks past the resolution of radiographic signs of pneumonia; intravenous fluids; and nebulization with sterile saline and coupage chest physiotherapy. Several weeks may elapse before a complete recovery.

Additional Reading


Black Widow Spider Bite

The adult black widow spider (Latrodectus species) can be recognized by a red to orange hourglass-shaped marking on the underside of a globous, shiny, black abdomen. The immature female can be recognized by a colorful pattern of red, brown, and beige on the dorsal surface of the abdomen. Adult and immature females are equally capable of envenomation. The male is unable to penetrate the skin because of its small size. Black widow spiders are found throughout the United States and Canada. Black widow spider venom is neurotoxic and acts presynaptically, releasing large amounts of acetylcholine and norepinephrine. There appears to be a seasonal variation in the potency of the venom; it is lowest in the spring and highest in the fall. In dogs, envenomation results in hyperesthesia, muscle fasciculations, and hypertension. Muscle rigidity without tenderness is characteristic. Affected animals may demonstrate clinical signs of acute abdominal pain. Tonic-clonic convulsions may occur but are rare. In cats, paralytic signs predominate and appear early as an ascending lower motor neuron paralysis. Increased salivation, vomiting, and diarrhea may occur. Serum biochemistry profiles often reveal significant elevations in creatine kinase and hypocalcemia. Myoglobinemia and myoglobinuria can occur because of extreme muscle damage.

Management

Management of black widow spider envenomation should be aggressive in the cat and dog, particularly when the exposure is known. In many cases, however, the diagnosis is made based on clinical signs, biochemical abnormalities, and lack of another apparent cause. Antivenin (one vial) is available and should be administered after pretreatment with

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diphenhydramine. If antivenin is unavailable, administer a slow infusion of calcium-containing fluid such as lactated Ringer’s solution with calcium gluconate while carefully monitoring the patient’s ECG.

**Brown Spider Bite**

**Fiddleback, Brown Recluse, Arizona Brown, Loxosceles Species**

The small brown nonaggressive brown spider is characterized by a violin-shaped marking on the cephalothorax. The neck of the violin points toward the abdomen. Brown spiders are found primarily in the southern half of the United States but have been documented as far north as Michigan. The venom of the brown spider has a potent dermatonecrotic effect and starts with a classic bull’s-eye lesion. The lesion then develops into an indolent ulcer into dependent tissues promoted by complement fixation and influx of neutrophils into the affected area. The ulcer can take months to heal and often leaves a disfiguring scar. Systemic reactions are rare but can include hemolysis, fever, thrombocytopenia, weakness, and joint pain. Fatalities are possible.

**Management**

Immediate management of an animal with brown spider envenomation is difficult because there is no specific antidote and because clinical signs may be delayed until necrosis of the skin and underlying tissues becomes apparent through the patient’s fur 7 to 14 days after the initial bite. Dapsone has been recommended at a dose of 1 mg/kg PO tid for 10 days. Surgical excision of the ulcer may be helpful if performed in the early stages of wound appearance. Glucocorticosteroids may be of some benefit if used within 48 hours of the bite. The ulcer should be left to heal by second intention. Deep ulcers should be treated with antibiotics.

**Additional Reading**


**Other Poisonous Creatures**

**Bufo Species Toxicosis**

*Bufo* toad species (*Bufo marinus*, also known as the cane toad, marine toad, or giant toad; and the Colorado River toad or Sonoran desert toad, *Bufo alvarius*) can be associated with severe cardiac and neurotoxicity if an animal licks the toad’s skin. The severity of toxicity depends largely on the size of the dog. Toxins in the cane toad, *B. marinus*, include catecholamines and vasoactive substances (epinephrine, norepinephrine, serotonin, dopamine) and bufotoxins (bufagins, bufotoxin, and bufotenine), the mechanism of which is similar to that of cardiac glycosides. Clinical signs can range from ptyalism, weakness, ataxia, extensor rigidity, opisthotonus, and collapse to seizures. Clinical signs associated with *B. alvarius* toxicity are limited largely to cardiac dysrhythmias, ataxia, and salivation.

**Immediate Action and Treatment**

The animal should have its mouth rinsed out thoroughly with tap water even before presentation to the veterinarian. If the animal is unconscious or actively seizing and cannot protect its airway, flushing the mouth is contraindicated. Once an animal is presented to the veterinarian, the veterinarian should place an intravenous catheter and monitor the patient’s
ECG and BP. Attempt seizure control with diazepam (0.5 mg/kg IV) or pentobarbital (5 to 15 mg/kg IV to effect). Ventricular dysrhythmias can be controlled first with esmolol (0.1 mg/kg). If esmolol is ineffective, administer a longer-acting parenteral β-antagonist such as propranolol (0.05 mg/kg IV). Ventricular tachycardia also can be treated with lidocaine (1 to 2 mg/kg IV, followed by 50 to 100 mcg/kg/min IV CRI).

Management
Case management largely depends on supportive care and treating clinical signs as they occur. Monitor baseline acid-base and electrolyte balance because severe metabolic acidosis may occur that should be treated with intravenous fluids and sodium bicarbonate (0.25 to 1 mEq/kg IV). Monitor ECG, BP, and mentation changes closely. Control seizures and cardiac dysrhythmias.

Additional Reading

Gila Monster (Heloderma suspectum) and Mexican Bearded Lizard (Heloderma horridum) Bites
Lizards of the family Helodermatidae are the only two poisonous lizards in the world. They are found in the Southwestern United States and Mexico. The venom glands are located on either side of the lower jaw. Because these lizards are typically lethargic and nonaggressive, bite wounds are rare. The lizards have grooved teeth that introduce the venom with a chewing motion as the lizard holds tenaciously to the victim. The majority of affected dogs are bitten on the upper lip, which is very painful.

Management
There are no proven first aid measures for bites from Gila monsters or Mexican bearded lizards. The lizard can be disengaged by inserting a prying instrument between the jaws and pushing at the back of the mouth. The teeth of the lizard are brittle and break off in the wound. Topical irrigation with lidocaine and probing with a needle will aid in finding and removing the teeth from the victim. Bite wounds will bleed excessively. Irrigate wounds with sterile saline or lactated Ringer’s solution, and place compression on the affected area until bleeding ceases. Monitor the patient for hypotension. Establish intravenous access, and administer intravenous fluids according to the patient’s perfusion parameters. Antibiotic therapy is indicated because of the bacteria in the lizard’s mouth. Because no antidote is available, treatment is supportive according to patient signs.

FRACTURES AND MUSCULOSKELETAL TRAUMA
The majority of musculoskeletal emergencies are the result of external trauma, most commonly from motor vehicle accidents. Blunt trauma invokes injury to multiple organ systems as a rule, rather than an exception. Because of this, massive musculoskeletal injuries are assigned a relatively low priority during the initial triage and treatment of a traumatized animal. Perform a rapid primary survey and institute any lifesaving emergency therapies. Adhere to A CRASH PLAN or the ABCs of resuscitation (see Initial Emergency Examination, Management, and Triage).

Although musculoskeletal injuries are assigned a relatively lower priority, the degree of recovery from these injuries and financial obligation for fracture repair sometimes become critical factors in a client’s decision whether to pursue further therapy. One of the most important deciding factors is the long-term prognosis for the patient to have a good quality of life after fracture repair.

The initial management of musculoskeletal injuries is important in ensuring the best chance for maximal recovery with minimal complications after definitive surgical fracture
repair. This is particularly important for open fractures, spinal cord compromise, multiple fractures, open joints, articular fractures, physeal fractures, and concomitant ligamentous or neurologic compromise (Box 1-41).

**Immediate Action and Treatment**

Immediately after the initial primary survey of a patient, perform a more thorough examination, including an orthopedic examination. Multiple injuries often are observed in falls from a height (e.g., “high-rise syndrome”), motor vehicle accidents, gunshot wounds, and encounters with other animals (e.g., “big-dog–little-dog”). Address the most life-threatening injuries, and palliate musculoskeletal injuries until more definitive repair can be attempted when the patient’s condition is more stable.

In animals with the history of potential for multiple injuries, search thoroughly and meticulously for areas of injury to the spinal column and extremities and for small puncture wounds. Helpful signs that can provide a clue as to an underlying injury include swelling, bruising, abnormal motion, and crepitus (caused by subcutaneous emphysema or bony fracture). If the patient is alert, look for areas of tenderness or pain. In unconscious or depressed patients, reexamine the patient after the patient becomes more mentally alert. In obtunded patients because of the early response and attenuation of pain. Unconscious or immobile patients must have radiographic examination of the spinal column after stabilization and support. Palpate the skull carefully for obvious depressions or crepitus that may be associated with a skull fracture. Localization of the injury can be determined by motion in abnormal locations, swelling caused by hemorrhage or edema, pain during gentle movement or palpation, deformity, angular change, or a significant increase or decrease in normal range of motion of bones and joints. Perform a rectal examination in all cases to palpate for pelvic fractures and displacement.

Once the diagnosis of a fracture or luxation has been confirmed, look for any evidence of skin lacerations or punctures near the fracture site. In long-haired breeds, clipping the fur near the fracture site often is necessary to perform a thorough examination of the area. If any wounds are found, the fracture is classified as an open fracture until proven otherwise. In some cases the open fracture is obvious, with a large section of bone fragment protruding through the skin. In other cases the puncture wound may be subtle, with only a small amount of blood or a pinpoint hole in the skin surface. Characteristics observed with open fractures include bone penetration, fat droplets or marrow elements in blood coming from the wound, subcutaneous emphysema on radiographs, and lacerations in the area of a fracture. Protect the patient from further injury or contamination of wounds. Excessive palpation to intentionally produce crepitus is inappropriate because it causes severe patient
discomfort and has the potential to cause severe soft tissue and neurologic injury at the fracture site. Sedation and analgesia aid in making the examination more comfortable for the patient and allow localization of the injury and comparison with the opposite extremity. Higher-quality radiographs can be performed to determine the extent of the injury when the animal is sedated adequately and pain is controlled.

**Initial Fracture Management**

Sedate the patient judiciously with analgesic drugs. Opioid drugs work well for orthopedic pain, produce minimal cardiopulmonary depression, and can be reversed with naloxone if necessary. Handle the fracture site gently to avoid causing further pain and soft tissue injury at the fracture site. Rough or careless handling of a fracture site can cause a closed fracture to penetrate through the skin and become an open fracture. Cover open fractures immediately to prevent contamination of the fracture with nosocomial infection from the hospital. Administer a first-generation cephalosporin (cephalexin, 22 mg/kg PO q8h, or cefazolin, 22 mg/kg IV q8h). The bandage also serves to control hemorrhage and prevent desiccation of the bones and surrounding soft tissue structures. Leave the initial bandages in place until the patient’s cardiopulmonary status has been determined to be stable and more definitive wound management can occur in a clean, preferably sterile location.

Examine the neurologic status and cardiovascular status of the limb before and after treatment. Determine the vascular status of the limb by checking the color and temperature of the limb, the state of distal pulses, and the degree of bleeding from a cut nail bed. In patients with severe cardiovascular compromise and hypotension caused by hemorrhagic shock, the viability of the limb may be in question until the cardiovascular status and BP are normalized. Reduction of the fracture or straightening of gross deformities may return normal vascularity to the limb. When checking neurologic status, examine for motor and sensory function to the limb. Swelling may increase pressure on the nerves as they run through osteofascial compartments, resulting in decreased sensory or motor function, or neuropraxia. Diminished function often returns to normal once the swelling subsides. Serial physical examinations in the patient and response to initial stabilization therapy can lead to a higher index of suspicion that more occult injuries are present, such as a diaphragmatic hernia, perforated bowel, lacerated liver or spleen, or uroabdomen.

To prevent ongoing trauma, reduce any fracture and then stabilize the site above and below the fracture. A modified Robert Jones splint or bandage often works well for fractures involving the distal extremities. Fractures of the humerus or femur are difficult to immobilize without the use of spica or over-the-hip coaptation splints to prevent mobility. Inappropriate bandaging of humerus or femur fractures can result in a fulcrum effect and worsen the soft tissue and neurologic injuries.

Further displacement of vertebral bodies or luxations can cause cord compression or laceration such that return to function becomes impossible. Immediately place any patient with a suspected spinal injury on a flat surface, and tape down the animal to prevent further movement until the spine has been cleared by a minimum of two orthogonal radiographic views (lateral and ventrodorsal views performed as a cross-table x-ray technique).

**Open Musculoskeletal Injury**

Wounds associated with musculoskeletal trauma are common and include injury to the bones, joints, tendons, and surrounding musculature (Box 1-42). Major problems associated with these cases are the presence of soft tissue trauma that makes wound closure hazardous or impossible because of the risk of infection. Chronic deep infection of traumatized wounds can cause delayed healing and sequestra to develop, particularly if there is avascular bone or cartilage within the wound.

In the early management of an open fracture, the areas should be splinted without pulling any exposed bone back into the soft tissue. The wound should not be probed or soaked, as nosocomial bacteria and other external contaminants can be introduced
into the wound, leading to severe infection. Because of the risk of actually causing infection, probing, flushing, or replacing tissues back into the wound should be performed at the time of formal debridement when the patient is physiologically stable. Bactericidal antibiotic therapy with a first-generation cephalosporin should be started immediately to obtain adequate concentrations of antibiotics at the fracture site. The duration of antibiotic therapy should ideally be limited to 2 to 3 days to prevent the risk of superinfection.

Treatment
Treatment of open musculoskeletal injury involves three considerations: initial inspection and wound debridement, stabilization and repair, and wound bandaging.

Initial inspection and wound debridement include the following steps:
1. After the patient's cardiovascular status has been stabilized and it has been determined that the patient can withstand anesthesia, place the animal under general anesthesia and remove the temporary splint.
2. Keeping the wound covered, shave the surrounding fur.
3. Remove the covering and then place sterile lubricant jelly over the wound. Shave the fur to the edges of the wound margin.
4. Wash away any entrapped fur and the lubricant jelly.
5. Complete an antiseptic scrub of the surrounding skin.
6. If the wound is a small puncture (e.g., gunshot pellets or bites), probe the wound with a sterile hemostat. Perform thorough debridement if tissues deep to the hole are cavitated. If not deep, create a hole for drainage.
7. Flush the wound with a physiologic solution (lactated Ringer's solution is preferred).
8. Debride the wound from outward to inward. Cut away damaged areas of skin and deeper tissues to open up underlying cavitations and tissue injury.

**Box 1-42 Classification of Open Wounds by Degree of Soft Tissue Injury**

**Type I Wound**
Minimal soft tissue trauma and devitalization.
When associated with a fracture, wound is created from the inside out by penetration of bone fragments through the skin, or from a low-energy gunshot.
Simple or comminated fracture pattern.
Good stability of the two main bone segments.
Treatment and prognosis are good and similar to those of a closed injury if wound is debrided and stabilized within 6 to 8 hours.

**Type II Wound**
Moderate soft tissue contusion and devitalization.
When associated with a fracture, wound is created from the outside in.
Major deep injury with considerable soft tissue stripping from bone and muscle damage.
Simple or comminated fracture pattern.
Prognosis is good if wound is debrided within 6 hours of injury and provided rigid stabilization with a bone plate or external fixator.

**Type III Wound**
Results from major external force.
Severe damage and necrosis of skin, subcutaneous tissue, muscle, nerve, bone, tendon, and arteries.
Soft tissue damage may vary from crush injury to shearing injury associated with bite wounds or low-speed automobile accidents.
Requires immediate and delayed sequential debridement and rigid external fixation.
Can require prolonged healing times.
Guarded prognosis.
9. Continuously irrigate with warm physiologic solution (lactated Ringer’s solution is preferred). The stream must be strong enough to flush debris out of the bottom of the wound. To accomplish this, attach a 20-gauge needle to a 35-mL syringe (will deliver 7 psi). Excise any obviously devitalized tissue.

10. Do not remove any bone fragments that are firmly attached to soft tissue. Do not cut into healthy soft tissue to find bullet or bone fragments, unless the bullet can cause injury to joints or nerve tissue.

11. Do a primary repair of tendons and nerves if the wound is type I and recent (within 8 hours of the initial injury). If the wound is too severe or if there is obvious infection, tag the ends of the tendons and nerves for later repair.

It is best to stabilize and repair open fractures as soon as the patient’s cardiovascular and respiratory status can tolerate general anesthesia, provided that adequate stabilization is possible. If this is not possible because of the level of experience of the surgeon or the lack of necessary equipment, it is best to perform wound management and place a temporary splint until definitive repair can be performed.

Wound bandaging is discussed in the section on bandaging techniques.

**Articular Cartilage Injury**

Structural injuries to the joints are common and can involve both ligaments and articular cartilage injuries. Cartilage does not heal well; therefore, injuries involving articular cartilage can lead to a significant loss of function and degenerative joint disease (osteoarthritis). Cartilage injuries that are superficial evoke a short-lived enzymatic and metabolic response that does not stimulate enough cellular growth to repair the defect. Superficial lesions remain as defects but do not progress to chondromalacia or osteoarthritis. Deep cartilage lacerations that extend to subchondral bone produce an exuberant healing response from the cells of the underlying cartilage. In many cases, this material undergoes degeneration and leads to osteoarthritis. Impact injuries to surface cartilage can cause chondrocyte and underlying bone injury. These lesions rapidly progress to osteoarthritis; however, they may be totally or partially reversible.

**Ligamentous Injuries**

Treatment of grade I injuries requires short-term coaptation splints; such injuries carry a good prognosis. Grade II injuries require surgical treatment with a suture stent and consistent postoperative coaptation splints to heal and maintain good function. Healing of grade III injuries often is a problem, and suture stents or surgical reapproximation may be indicated. Failure to immobilize joints that are frequently flexed (elbow and stifle) can result in late complications of ligament repair. Ligamentous injuries of joints, particularly the collateral ligaments of the stifle, elbow, and hock, and carpal hyperextension injuries are commonly missed and may require surgical fixation, including arthrodesis (Box 1-43).

**Fractures in the Immature Animal**

Fractures in immature animals differ from those in adults in that young puppies and kittens have a great ability to remodel bone. Remodeling is dependent on the age of the patient and the location of the fracture. The younger the puppy or kitten and the closer the fracture to

<table>
<thead>
<tr>
<th>BOX 1-43 CLASSIFICATION OF LIGAMENTOUS INJURIES</th>
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<tr>
<td><strong>Grade I sprain:</strong> Rupture of a portion of the ligament with minimal lengthening. Preservation of anatomic and mechanical integrity.</td>
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<tr>
<td><strong>Grade II sprain:</strong> A portion of the ruptured ligament is stretched. Ligament is longer but still intact.</td>
</tr>
<tr>
<td><strong>Grade III sprain:</strong> Complete ligament disruption.</td>
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the epiphysis or growth plate, the greater the potential for remodeling and the development of angular limb deformities. Remodeling occurs more effectively in long-limbed breeds of dogs than in short-limbed breeds. Fractures through the growth plate of immature animals may potentially cause angular limb deformities, joint dislocations or incongruity, and osteoarthritis. This form of injury is commonly observed in the distal ulnar growth plate and the proximal and distal radial growth plates.

**HIGH-RISE SYNDROME**

High-rise syndrome in cats is seen in cats that fall from a height, usually greater than 30 feet. It occurs most frequently in high-rise buildings in urban areas where cats lie on window ledges and suddenly fall out the window. The most common lesions observed in cats that fall from heights are thoracic injuries (rib and sternal fractures, pneumothorax, and pulmonary contusions) and facial and oral trauma (lip avulsions, mandibular symphyseal fractures, fractures of the hard palate, and maxillary fractures). Limb and spinal cord fractures and luxations, radius and ulna fractures, abdominal trauma, urinary tract trauma, and diaphragmatic hernias are also common. The injuries sustained are often found in combination, rather than as an isolated injury of one area of the body.

Follow the mnemonic A CRASH PLAN when managing a cat with high-rise syndrome, and immediately treat the animal for shock. Following cardiovascular and respiratory stabilization, evaluate thoracic and abdominal radiographs, including those of the spine. Evaluate the bladder closely, making sure that the cat is able to urinate effectively. Examine the hard palate, maxilla, and mandibular symphysis for fractures. Palpate the pelvis and carefully manipulate all limbs to examine for fractures or ligamentous injuries. Finally, perform a complete neurologic examination. Patients that fall less than five stories often have a more guarded prognosis than patients that fall from higher levels.

**Additional Reading**


**GASTROINTESTINAL EMERGENCIES**

**Oral Cavity**

Sometimes the owner witnesses the ingestion of a foreign body during play, such as throwing a stick or fetching a ball. Cats tend to play with string or thread that becomes caught around the base of the tongue. In many cases, however, ingestion of the foreign object is not witnessed, and diagnosis is made based on clinical signs and physical examination.

Foreign bodies lodged in the oral cavity often cause irritation and discomfort, including difficulty breathing and difficulty swallowing. Often, an animal paws at its mouth in an attempt to dislodge a stick or bones wedged across the roof of the mouth. Irritation, inability to close the mouth, and blockage of the oropharynx can result in excessive drooling. The saliva may appear blood-tinged owing to concurrent soft tissue trauma (Figures 1-46 and 1-47).

Obstruction of the glottis by a foreign body (e.g., tennis ball or toy) can result in cyanosis secondary to an obstructed airway and hypoxemia. In many cases the object is small enough to enter the larynx but too large to be expelled. If a foreign object is lodged in the mouth for more than several days, halitosis and purulent discharge may be present.

Many animals are anxious at the time of presentation and may require sedation or a light plane of anesthesia to remove the foreign object. The animal may bite personnel and may have bitten the owner during his or her attempt to remove the object from the mouth en route to the hospital. Propofol (4 to 7 mg/kg IV) with diazepam (0.5 to 1 mg/kg IV) is an
excellent combination for a light plane of anesthesia. Exercise caution when anesthetizing a patient with a ball lodged in the airway, as further compromise of respiratory function may occur and cause worsening of the hypoxemia.

Before inducing anesthesia, assemble all supplies necessary to remove the object. Make sure that rigid towel clamps, sponge forceps, and bone forceps are on hand, because the

Figure 1-46: Excessive ptyalism and gagging or excessive swallowing should increase suspicion of the presence of an esophageal or pharyngeal foreign body.

Figure 1-47: Radiograph of a chicken bone lodged in the patient's pharynx.
foreign object is often very slippery with saliva. Hemostats and Carmalt forceps may slip and not be useful in the removal of the foreign object.

Place a peripheral intravenous catheter to secure vascular access before anesthetic induction. Have available the supplies necessary for an emergency tracheostomy if the foreign object cannot be removed by usual methods. Induce a light plane of anesthesia and then grasp the object with the sponge forceps or towel clamps, and extract. Monitor the cardiorespiratory status of the animal at all times during the extraction process. If you are unable to remove the object, and if severe respiratory distress, including cyanosis, bradycardia, or ventricular dysrhythmias, develops, perform a tracheostomy distal to the site of obstruction.

Once the foreign body has been removed, administer supplemental flow-by oxygen until the animal awakens. If laryngeal edema or stridor on inspiration is present, administer a dose of dexamethasone sodium phosphate (0.25 mg/kg IV, IM, SQ) to decrease inflammation. The patient should be carefully monitored for 24 hours, because noncardiogenic pulmonary edema can develop secondary to airway obstruction.

**ESOPHAGEAL FOREIGN BODIES**

Esophageal foreign bodies pose a serious medical emergency. It is helpful if the owner witnessed ingestion of the object and noted rapid onset of clinical signs. In many cases, however, ingestion is not witnessed, and the diagnosis must be made based on clinical signs, thoracic radiographs, and results of a barium swallow. The most common clinical signs are excessive salivation with drooling, gulping, and regurgitation after eating. Many animals will make repeated swallowing motions. Some animals exhibit a rigid “sawhorse” stance, with reluctance to move immediately after foreign body ingestion and esophageal entrapment.

After completing a physical examination, evaluate cervical and thoracic radiographs to determine the location of the esophageal obstruction. Esophageal foreign objects are lodged most commonly at the base of the heart, the carina, or just orad to the lower esophageal sphincter. If the object has been lodged for several days, pleural effusion and pneumomediastinum may be present secondary to esophageal perforation. Endoscopy is useful for both diagnosis and removal of the foreign object; however, it is invasive and requires general anesthesia (Figure 1-48).

Remove foreign objects lodged in the esophagus with a rigid or flexible endoscope after the patient has been placed under general anesthesia. Evaluate the integrity of the esophagus both before and after removal of the material because focal perforation or pressure necrosis can be present. Necrosis of the mucosa and submucosa of the esophagus often leads to stricture formation or perforation.

Attempt to retrieve the object with a flexible fiberoptic endoscope if available. Rigid tube endoscopy can also be performed. In many cases, smooth objects that cannot be easily grasped can be pushed into the stomach and allowed to dissolve or may be removed by gastrostomy. If the foreign body is firmly lodged in the esophagus and cannot be pulled or pushed into the stomach, or if perforation has already occurred, the prognosis for return to function without strictures is not favorable. In such cases, referral to a surgical specialist is recommended for esophagostomy or esophageal resection.

After removal of the object, carefully examine the esophagus and then administer gastroprotectant agents (famotidine, 0.5 mg/kg PO bid; sucralfate slurry, 0.5 to 1.0 g/dog PO tid) for a minimum of 5 to 7 days. To rest the esophagus, the patient should receive nothing per os (NPO) for 24 to 48 hours. If esophageal irritation or erosion is moderate to severe, a percutaneous gastrostomy tube should be placed for feeding until the esophagus heals. Perform repeat endoscopy every 7 days to evaluate the healing process and to determine whether stricture formation is occurring.

**STOMACH**

Persistent vomiting immediately or soon after eating is often associated with a gastric foreign body. In some cases, the owner knows that the patient has ingested a foreign body of some kind. In other cases, continued vomiting despite lack of response to conservative
treatment (NPO status, antiemetics, gastroprotectant drugs) prompts further diagnostic procedures, including abdominal radiographs and bloodwork. Obstruction to gastric outflow and vomiting of hydrochloric acid often cause a hypochloremic metabolic acidosis. Radiopaque gastric foreign bodies may be observed on plain films. Radiolucent cloth material may require a barium series to delineate the shape and location of the foreign body (Figure 1-49).

Treatment consists of removal with flexible endoscopy or a simple gastrotomy. Most animals with uncomplicated gastric foreign bodies are relatively healthy, but any metabolic and electrolyte abnormalities should be corrected before anesthesia and surgery.

**Small Intestinal Obstruction**

Small intestinal obstruction can be caused by foreign bodies, tumors, intussusception, volvulus, or strangulation within hernias. Regardless of the cause, clinical signs of small intestinal obstruction depend on the location and degree of obstruction and whether the bowel has perforated. Clinical signs associated with a high small intestinal obstruction are usually more severe and more rapid in onset compared with partial or complete obstruction of the jejunum or ileum. Complete obstructions that allow no fluid or chyme to pass are worse than partial obstructions, which can cause intermittent clinical signs interspersed with periods of normality (Table 1-36).

The most common clinical signs associated with a complete small intestinal obstruction are anorexia, vomiting, lethargy, depression, dehydration, and sometimes abdominal pain. Early clinical signs may be limited to anorexia and depression, making a diagnosis challenging unless the owner has a suspicion that the animal ingested some kind of foreign object. Obstructions cranial to the common bile duct and pancreatic papillae lead to vomiting of gastric contents, namely hydrochloric acid, and a hypochloremic metabolic alkalosis. Obstructions caudal to the common bile duct and pancreatic papillae result in loss of other electrolytes and sometimes mixed acid-base disorders.

**Figure 1-48:** Example of an esophageal foreign body. Common locations are the carina and thoracic inlet.
Eventually, all animals with small intestinal obstruction vomit and have fluid loss into dilated segments of bowel, leading to dehydration and electrolyte abnormalities. Increased luminal pressure causes decreased lymphatic drainage and bowel edema. The bowel wall eventually becomes ischemic and may rupture.

Linear foreign bodies should be suspected in any vomiting patient, particularly cats. String or thread often is looped around the base of the tongue and can be visualized in many cases by a thorough oral examination. To look properly under the tongue, grasp the top of the animal’s head with one hand, and pull the lower jaw open with the index finger of the opposite hand while pushing up the thumb simultaneously on the tongue in between the intermandibular space. Thread and string can be observed lying along the ventral aspect of the tongue. In some cases, if a linear foreign body is lodged very caudally, it cannot be visualized without heavy sedation or anesthesia.

Linear foreign bodies eventually cause bowel obstruction and perforation of the intestines along the mesenteric border. The foreign material (e.g., string, thread, cloth, pantyhose) becomes lodged proximally, and the intestines become plicated as the body

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**Figure 1-49:** Lateral abdominal radiograph with two radiopaque densities within the lumen of the small intestine, consistent with rocks.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Onset</th>
<th>Progression of Vomiting</th>
<th>Frequency</th>
<th>Volume of Vomit</th>
<th>Tenesmus</th>
<th>Abdominal Distension</th>
</tr>
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<tbody>
<tr>
<td>High small bowel</td>
<td>Rapid</td>
<td>Rapid</td>
<td>Frequent</td>
<td>Large volume</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Low small bowel</td>
<td>Slower</td>
<td>Slower</td>
<td>Less frequent</td>
<td>Small volume</td>
<td>Diarrhea</td>
<td>Present</td>
</tr>
<tr>
<td>Large bowel</td>
<td>Subacute to chronic</td>
<td>Slow</td>
<td>Occasional</td>
<td>Scant</td>
<td>Often with diarrhea</td>
<td>Present</td>
</tr>
</tbody>
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Eventually, all animals with small intestinal obstruction vomit and have fluid loss into dilated segments of bowel, leading to dehydration and electrolyte abnormalities. Increased luminal pressure causes decreased lymphatic drainage and bowel edema. The bowel wall eventually becomes ischemic and may rupture.
attempts to push the material caudally through the intestines (Figure 1-50). Continued peristalsis eventually causes a sawing motion of the material and perforation of the mesenteric border of the intestines. Once peritonitis occurs, the prognosis is less favorable unless prompt and aggressive treatment is initiated.

Reevaluate any patient that does not respond to conservative symptomatic therapy, performing a complete blood count and serum biochemical panel (including electrolytes) and obtaining abdominal radiographs.

Intestinal masses may be palpable on physical examination and are often associated with signs of discomfort or pain when palpating over the mass. Radiography and abdominal ultrasound are the most useful diagnostic aids. Plain radiographs may be diagnostic when the foreign object is radiodense or there is characteristic dilation or plication of bowel loops. As a rule of thumb, the width of a loop of small bowel should be no larger than twice the width of a rib. Diagnosis of small intestinal obstruction or ileus can be based on the appearance of stacking loops of dilated bowel. Comparison of the width of the bowel with the width of a rib is often performed. With mild dilation, the bowel width is three to four times the rib width; with extensive dilation, five to six times the rib width (Figure 1-51). In cases of linear foreign bodies, C-areas (comma-shaped areas) of gas trapped in the plicated bowel will appear stacked on one another. Blunt, wedge-shaped areas of gas or square linear areas of gas adjacent to a distended bowel loop are characteristic of a foreign body lodged in the intestine. Contrast radiography is indicated when confirmation of the suspected diagnosis is necessary and ultrasonography is not available. Contrast material may outline the object or abruptly stop oral to the obstruction.

The definitive treatment of any type of small intestinal foreign body is surgical removal. Linear foreign bodies sometimes pass, but they should never be left untreated in a patient that is demonstrating clinical signs of inappetence, vomiting, lethargy, and dehydration. The timing of surgery is critical because the risk of intestinal perforation increases with time. Before surgery, correct any acid-base and electrolyte abnormalities with intravenous fluid therapy. Administer broad-spectrum antibiotics. Perform an enterotomy or intestinal resection and anastomosis as soon as possible once the patient’s acid-base status and electrolyte status have been corrected.
Clinical signs of a foreign body in the large bowel are usually nonexistent. In most cases, if a foreign object has passed successfully through the small bowel, it will pass through the large bowel without incident unless bowel perforation and peritonitis occur. Penetrating foreign bodies such as needles often cause localized or generalized peritonitis, abdominal pain, and fever. Hematochezia may be present if the foreign object causes abrasion of the rectal mucosa.

Symptomatic patients should have abdominal radiographs taken. Colonoscopy or exploratory laparotomy should be performed if survey radiographs are suggestive of a large intestinal obstruction or perforation. In most cases, large intestinal foreign bodies will pass without incident. Surgery is required to treat perforations, peritonitis, or abscesses.

Foreign bodies in the rectum and anus often are the result of ingestion of bones, wood material, needles, and thread or malicious external insertion. Often the material can pass through the entire gastrointestinal tract and then get stuck in the anal ring. Clinical signs include hematochezia and dyschezia with straining to defecate. Diagnosis is made by visual examination of the item in the anus, or by careful digital palpation after heavy sedation or short-acting general anesthesia. Radiography is helpful in locating needles that have penetrated the rectum and lodged in the perirectal or perinatal tissues. Treatment consists of careful removal of the needle digitally or surgically.

Intussusception is the acute invagination of one segment of bowel (the intussusceptum) into another (the intussucipiens). The proximal segment always invaginates into the distal segment of bowel. Intussusception most commonly occurs in puppies and kittens younger than 1 year of age but can occur in an animal of any age with hypermotility of the small bowel, gastrointestinal parasites, and severe viral or bacterial enteritis. Intussusception occurs primarily in the small bowel in the jejunum, ileum, and ileocolic junction.
Clinical signs include vomiting, abdominal discomfort, and hemorrhagic diarrhea. Usually, hemorrhagic diarrhea is the first noticeable sign and in puppies may be caused by parvoviral enteritis, with secondary intussusception. Usually, the obstruction is partial with mild clinical signs. More serious clinical signs develop as the obstruction becomes more complete. Differential diagnoses include hemorrhagic gastroenteritis (HGE), parvoviral enteritis, gastrointestinal parasites, intestinal foreign body, bacterial enteritis, and other causes of vomiting and diarrhea.

The diagnosis of intussusception is often made based on palpation of a sausage-shaped firm, tubular structure in the abdomen accompanied by clinical signs and abdominal pain. Plain radiographs may demonstrate segmental or generalized dilated segments of bowel, depending on the duration of the problem. Ultrasonographs of the palpable mass resemble the layers of an onion, with hyperechoic intestinal walls separated by less echogenic edema.

Treatment consists of correction of the patient's acid-base and electrolyte abnormalities with intravenous fluids and surgical reduction or removal of the intussusception with resection and anastomosis. Although enteroplication has been suggested, the technique has fallen out of favor because of the increased risk of later obstruction. The primary cause of intestinal inflammation and hypermotility must be identified and corrected.

**Gastric Dilatation-Volvulus**

Gastric dilatation can occur with or without volvulus in the dog. GDV occurs primarily in large- and giant-breed dogs with deep chests, such as the Great Dane, Labrador Retriever, Saint Bernard, German Shepherd Dog, Gordon Setter, Irish Setter, Standard Poodle, Bernese Mountain Dog, and Bassett Hound. The risk of GDV increases with age; however, it can be seen in dogs as young as 4 months old. Deep, narrow-chested breeds are more likely to develop GDV than dogs with broader chests. The overall mortality for surgically treated GDV ranges from 10% to 18%, with most deaths occurring in patients that required splenectomy and partial gastrectomy.

Clinical signs of GDV include abdominal distension, unproductive vomiting or retching, lethargy, weakness, sometimes straining to defecate, and collapse. The owner may think that the animal is vomiting productively because of the white foamy froth (saliva) that is not able to pass into the twisted stomach. In some cases there is a history of the dog’s being fed a large meal or consuming a large quantity of water before the onset of clinical signs. Instruct the owner of any patient with a predisposition for and clinical signs of GDV to transport the animal to the nearest veterinary facility immediately.

Physical examination often reveals a distended abdomen with a tympanic area on auscultation. In dogs with very deep chests, it may be difficult to appreciate abdominal distension if the stomach is tucked under the rib cage. Depending on the stage of shock, the patient may have sinus tachycardia with bounding pulses, cardiac dysrhythmias with pulse deficits, or bradycardia. The mucous membranes may appear red and injected or pale with a prolonged capillary refill time. The patient may appear anxious and attempt to retch unproductively. If the patient is nonambulatory at the time of presentation, the prognosis is more guarded.

The definitive diagnosis of GDV is based on clinical signs, physical examination findings, and radiographic appearance of gas distension of the gastric fundus with dorsocranial displacement of the pylorus and duodenum (the “double-bubble” or “Popeye arm” sign) (Figure 1-52). In simple gastric dilatation without volvulus, there is gas distension of the stomach, with anatomy appearing normal on radiography. With “food bloat,” or gastric distension from overconsumption of food, ingesta are visible in the distended stomach (Figure 1-53).

As soon as a patient with possible GDV is presented, place a large-bore intravenous catheter in the cephalic vein(s) and assess the patient’s ECG, BP, heart rate, capillary refill time, and respiratory function. Obtain blood samples for a complete blood count, serum biochemistry profile, immediate lactate measurement, and coagulation tests before taking any radiographs. Rapidly infuse a colloid (hydroxyethyl starch, 5 mL/kg IV bolus) along
with shock volumes of a crystalloid fluid (up to 90 mL/kg (see section on shock). Monitor perfusion parameters (heart rate, BP, capillary refill time, and ECG) and titrate fluid therapy according to the patient’s response. The use of short-acting glucocorticosteroids is not recommended. No detailed studies have proved them to be beneficial versus not using glucocorticosteroids in the patient with GDV.

Attempt gastric decompression, either with placement of an orogastric tube or by trocharization. To place an orogastric tube, position the distal end of the tube at the level of the patient’s last rib (Figure 1-54) and place it adjacent to the animal’s thorax; then put a piece of tape around the tube where it comes out of the mouth, once it is in place. Put a
roll of 2-inch tape in the patient’s mouth behind the canine teeth and then secure the roll in place by taping the mouth closed around the roll of tape. Lubricate the tube with lubricating jelly and slowly insert the tube through the center of the roll of tape into the stomach. The passing of the tube does not rule out volvulus.

In some cases, the front legs of the patient need to be elevated, and the caudal aspect of the patient lowered (front legs standing on a table with back legs on the ground) to allow gravity to pull the stomach down to allow the tube to pass. Once the tube has been passed, air within the stomach is relieved, and the stomach can be lavaged. The presence of gastric mucosa or blood in the efflux from the tube makes the prognosis more guarded.

If an orogastric tube cannot be passed, clip and aseptically scrub the patient’s lateral abdomen and then insert a 16-gauge over-the-needle catheter. “Pinging” the animal’s side with simultaneous auscultation allows determination of the location that is most tympanic—that is, the proper location for catheter insertion.

Once intravenous fluids have been started in the animal, take a right lateral abdominal radiograph to document GDV. If no volvulus is present, the owner may elect for more conservative care, and the animal should be monitored in the hospital for a minimum of 24 hours. Because some cases of GDV intermittently twist and untwist, the owner should be cautioned that although the stomach is not twisted at that moment, volvulus can occur at any time. If radiographs demonstrate food bloat, induce emesis (apomorphine, 0.04 mg/kg IV) or perform orogastric lavage under general anesthesia. Documentation of GDV constitutes a surgical emergency.

After diagnosis of GDV, continue administration of intravenous fluids. Serum lactate measurements greater than 6.0 mmol/L are associated with an increased risk of gastric necrosis, requirement for partial gastrectomy, and increased mortality. If serum lactate does not decrease by more than 4 mmol/L, or 42.5% of original serum lactate concentrations, after fluid resuscitation and surgery, the prognosis worsens. Administer fresh frozen plasma (20 mL/kg) to patients with thrombocytopenia or prolonged PT, APTT, or ACT. Cardiac dysrhythmias, particularly ventricular dysrhythmias, are common in cases of GDV and are thought to occur secondary to ischemia and proinflammatory cytokines released during volvulus and reperfusion. Lidocaine (1 to 2 mg/kg followed by 50 mcg/kg/min IV CRI) can be used to preemptively treat cardiac dysrhythmias that are associated with ischemia-reperfusion injury, or administration can be started when ventricular dysrhythmias are present. Correct any electrolyte abnormalities, including hypokalemia and hypo-

Figure 1-54: Measure the orogastric tube from the patient’s mouth to the last rib and mark the tube to prevent pushing it in too far.
magnesemia. The use of NSAIDs (flunixin meglumine, carprofen, ketoprofen) that can potentially decrease renal perfusion and predispose to gastric ulcers is absolutely contraindicated. Administer analgesic drugs (fentanyl, 2 mcg/kg IV bolus, followed by 3 to 20 mcg/kg/hr IV CRI; or hydromorphone, 0.1 mg/kg IV) before anesthetic induction. After a balanced anesthesia protocol has been carried out, the patient should be taken immediately to surgery for gastric derotation and gastropexy.

Postoperatively, assess the patient’s ECG, BP, platelet count, coagulation parameters, and gastric function (see section Rule of 20). If no resection is required, the animal can be given small amounts of water beginning 12 hours after surgery. Depending on the severity of the patient’s condition, small amounts of a bland diet can be offered 12 to 24 hours postoperatively. Continue supportive care with analgesia and crystalloid fluids until the patient is able to tolerate oral analgesic drugs (tramadol, 1 to 3 mg/kg PO q8-12h). Once the patient is ambulatory and able to eat and drink on its own, it can be released from the hospital; instruct the owner to feed the animal multiple small meals throughout the day for the first week.

**Small Intestinal Mesenteric Volvulus or Torsion**

When the intestines twist around the root of the mesentery, a small intestinal or mesenteric volvulus occurs. The problem is most common in the young German Shepherd Dog, although it has been observed in other large and giant breeds. Predisposing factors include pancreatic atrophy, gastrointestinal disease, trauma, and splenectomy.

Clinical signs of mesenteric volvulus include vomiting, hemorrhagic diarrhea, bowel distension, acute onset of clinical signs of shock, abdominal pain, brick-red mucous membranes (septicemia), and sudden death.

Diagnosis is based on an index of suspicion and the presence of clinical signs in a predisposed breed. Plain radiographs often reveal grossly distended loops of bowel in a palisade gas pattern. In some dogs, multiple, tear-drop–shaped, gas-filled loops appear to rise from a focal point in the abdomen. Usually, massive distension of the entire small bowel is observed (Figure 1-55). The presence of pneumoperitoneum or lack of abdominal detail secondary to the presence of abdominal fluid is characteristic of bowel perforation and peritonitis.

**Figure 1-55:** Severe generalized distension of the small intestine, characteristic of a mesenteric volvulus. This constitutes an immediate surgical emergency, and the prognosis is often poor. This condition is most common in young German Shepherd Dogs but can be observed in any breed.
In a patient with mesenteric volvulus, immediate aggressive action is necessary for the animal to have any chance of survival. Treatment consists of massive volumes of intravenous crystalloid and colloid fluids (see section on intravenous therapy), broad-spectrum antibiotics (ampicillin, 22 mg/kg IV tid to qid, with enrofloxacin, 10 mg/kg IV once daily), and surgical correction of the bowel. Because of the massive release of proinflammatory cytokines, bacterial translocation, and ischemia, treatment for shock is of paramount importance (see sections on the Rule of 20 and shock). The prognosis for any patient with mesenteric volvulus is poor.

**Large Intestinal Obstruction**

**Obstipation**

Obstipation (obstructive constipation) is most common in the older cat. In cases of simple constipation, rehydrating the animal with intravenous fluids and stool softeners is often sufficient for it to regain the ability to have a bowel movement. Obstipation, however, is caused by a dynamic ileus of the large bowel that eventually leads to megacolon. Affected cats usually are anorexic, lethargic, and extremely dehydrated. Treatment consists of rehydration with intravenous crystalloid fluids, correction of electrolyte abnormalities, enemas, and promotility agents such as cisapride (0.5 mg/kg PO q8-24h). The use of phosphate enemas in cats is absolutely contraindicated because of the risk of causing acute, fatal hyperphosphatemia. In many cases the patient should be placed under general anesthesia, and manual deobstipation performed with warm water soapy enemas and a gloved finger to relieve and disimpact the rectum. Stool softeners such as lactulose and docusate stool softener (DSS) may also be used. Predisposing causes of obstipation such as narrowing of the pelvic canal, perineal hernia, and tumors should be ruled out.

**Tumors of the Gastrointestinal Tract**

**Adenocarcinoma**

Adenocarcinoma is the most common neoplasm of the gastrointestinal tract that causes partial to complete obstruction. Adenocarcinomas tend to be annular and constricting, and they may cause progressive obstruction of the lumen of the small or large bowel. Siamese cats tend to have adenocarcinomas in the small intestine, whereas in dogs the tumor tends to occur in the large intestine.

Clinical signs of adenocarcinoma are both acute and chronic and consist of anorexia, weight loss, and progressive vomiting that occur over weeks to months. Effusion may be present if metastasis to peritoneal surfaces has occurred.

Diagnosis is based on clinical signs and physical examination findings of a palpable abdominal mass, radiographic evidence of an abdominal mass and small or large intestinal obstruction, or ultrasonographic evidence of an intestinal mass.

Treatment consists of surgical resection of the affected bowel segment. The prognosis for long-term survival (10 to 12 months) is good if the mass is completely resected and if other clinical signs of cachexia or metastasis are observed at the time of diagnosis. Median survival is 15 to 30 weeks if metastases to lymph nodes, liver, or the peritoneum are absent at the time of diagnosis. In dogs the prognosis is more guarded.

**Leiomyoma and Leiomyosarcoma**

Leiomyoma and leiomyosarcoma are tumors that can cause partial or complete obstruction of the bowel. Clinical signs are often referred to progressive anemia, including weakness, lethargy, inappetence, and melena. Hypoglycemia can be observed as a paraneoplastic syndrome or because of sepsis and peritonitis secondary to bowel perforation. Leiomyomas are most commonly observed at the cecocolic junction or in the cecum. Surgical resection and anastomosis are usually curative and carry a favorable prognosis.
**Strangulated Hernias**

Incarceration of a loop of bowel into congenital or acquired defects in the body wall can cause small bowel obstruction. Pregnant females and young animals with congenital hernias are most at risk. Rarely, older animals with perineal hernias and animals of any age with traumatic hernias can be affected. Clinical signs are consistent with a small intestinal obstruction: anorexia, vomiting, lethargy, abdominal pain, and weakness. Diagnosis is often made based on physical examination of a reducible or nonreducible mass in the body wall. Hernias whose contents are reducible are usually asymptomatic. Treatment consists of supportive care and rehydration, administration of broad-spectrum antibiotics, and surgical correction of the body wall hernia. In some cases, intestinal resection and anastomosis of the affected area is necessary when bowel ischemia occurs.

**Bowel Perforation**

The potential for bowel perforation should be suspected whenever there is any penetrating injury (knife, gunshot wound, bite wound, stick impalement) of the abdomen. Injuries that result in bowel ischemia and rupture can also occur secondary to nonpenetrating blunt trauma or shear forces (e.g., big dog–little dog or cat). Perforation of the stomach and small and large intestines can occur with use of NSAIDs.

Diagnosis of bowel perforation first depends on the alertness to the possibility that the bowel may have been perforated or penetrated. As a general rule, all penetrating injuries of the abdomen should be investigated by exploratory laparotomy. DPL can be performed; however, early after penetrating injury of the bowel, DPL findings may be negative or nondiagnostic until peritonitis develops. Whenever any patient with blunt or penetrating abdominal trauma does not respond to initial fluid therapy, or responds and then deteriorates, the index of suspicion for bowel injury should be raised. The findings of pneumoperitoneum on abdominal radiographs or of intracellular bacteria, extracellular bacteria, bile pigment, or bowel contents in fluid obtained by abdominocentesis or DPL fluid or cloudy appearance of such fluids (see sections on abdominocentesis and diagnostic peritoneal lavage) warrant immediate surgical exploration.

Treatment largely consists of stabilizing the patient's cardiovascular and electrolyte status with intravenous fluids, administering broad-spectrum antibiotics, and performing definitive surgical exploration and repair of injured structures.

**Rectal Prolapse**

Prolapse of the rectum is observed most frequently secondary to parasitism and gastrointestinal viral infections in young puppies and kittens with chronic diarrhea. Older animals with rectal prolapse often have an underlying problem such as a tumor or mucosal lesion that causes straining and dyschezia. The diagnosis of a rectal prolapse is made based on physical examination findings. The diagnosis of rectal prolapse is sometimes difficult to distinguish from small intestinal intussusception. In rare cases the intussusception can invaginate through the large bowel, rectum, and anus. The two entities are distinguished from each other by inserting a lubricated thermometer or blunt probe into the cul-de-sac formed by the junction of the prolapsed mucosa and mucocutaneous junction at the anal ring. Inability to insert the probe or thermometer indicates that the rectal mucosa is prolapsed. Passage of the probe signifies that the prolapsed segment is actually the intussusceptum.

Treatment can be performed easily if the prolapse is acute and the rectal mucosa is not too irritated or edematous. The presence of severely necrotic tissue warrants surgical intervention. To reduce an acute rectal prolapse, after placing the patient under general anesthesia, lubricate the prolapsed tissue and gently push it back into the rectum, using a lubricated syringe or syringe casing. Apply a loose purse-string suture, leaving it in place for a minimum of 48 hours. Deworm the patient and administer stool softeners. If a rectal prolapse cannot be reduced, or if the tissue is nonviable, surgical intervention is warranted.

In patients in which viable tissue does not stay reduced with a purse-string suture, a colopexy can be performed during a laparotomy. First, place tension on the colon to reduce the prolapse, and then suture the colon to the peritoneum of the lateral abdominal wall.
with two to three rows of 2-0 or 3-0 monofilament suture material. If the prolapsed tissue is nonviable, it must be amputated. Place four stay sutures at 90-degree intervals through the wall of the prolapse at the mucocutaneous junction. Resect the prolapse distal to the stay sutures and then reestablish the rectal continuity by suturing the seromuscular layers together in one circumferential line and the mucosal layers together in the other. Replace the suture incision into the anal canal. After surgery, deworm the patient and administer a stool softener and analgesic drugs. Avoid using thermometers or other probes in the immediate postoperative period because they may disrupt suture lines.

**Acute Gastritis**

Acute gastritis may be associated with a variety of clinical conditions, including oral hemorrhage, ingestion of highly fermentable indigestible foods or garbage, toxins, foreign bodies, renal or hepatic failure, inflammatory bowel disease, and bacterial and viral infections. Diarrhea often accompanies or follows acute gastritis. HGE often occurs as a shocklike syndrome with a rapidly rising hematocrit level. Clinical signs of gastritis include depression, lethargy, anterior abdominal pain, excessive water consumption, vomiting, and dehydration. Differential diagnosis of acute gastritis includes pancreatitis, hepatic or renal failure, gastrointestinal obstruction, and toxicities (Box 1-44).

The diagnosis is often a diagnosis of exclusion of other causes (see preceding text). A careful and thorough examination of the vomitus may be helpful in arriving at a diagnosis. A complete blood count, serum biochemistry profile including amylase and lipase, parvovirus test (in young puppies), fecal flotation and cytology, abdominal radiographs (plain and/or contrast studies), and abdominal ultrasound may be warranted to rule out other causes of acute vomiting.

While diagnostic tests are being performed, treatment consists of withholding all food and water for a minimum of 24 hours. After calculating the patient’s degree of dehydration, administer a balanced crystalloid fluid to normalize acid-base and electrolyte status. Control vomiting with antiemetics such as metoclopramide, prochlorperazine, chlorpromazine, dolasetron, ondansetron, and maropitant (Table 1-37). If vomiting is accompanied by diarrhea, administer broad-spectrum antibiotics (cefazolin, 22 mg/kg IV q8h, with metronidazole, 10 mg/kg IV q8h; or ampicillin, 22 mg/kg IV q6-8h, with enrofloxacin, 10 mg/kg IV q24h) to decrease the risk of bacterial translocation and bacteremia and septicemia. Although antacids (famotidine, ranitidine, cimetidine) do not have a direct antiemetic effect, their use can decrease gastric acidity and esophageal irritation during vomiting. If gastritis is secondary to uremia or NSAID use, administer gastroprotectant and antiemetic drugs (ranitidine, 1 mg/kg PO q12h; sucralfate, 0.25 to 1 g/dog PO q8h; or omeprazole, 0.5 to 1 mg/kg PO q24h) to decrease acid secretion and coat areas of gastric ulceration (see Table 1-37). Once food and water can be tolerated, the patient can be placed on an oral diet and medications, and intravenous fluids can be discontinued.

**Hemorrhagic Gastroenteritis**

HGE is severe hemorrhagic vomiting and diarrhea of acute onset, most commonly observed in young small-breed dogs (e.g., Poodles, Miniature Dachshunds, Miniature Schnauzers) 2 to 4 years of age. Clinical signs develop rapidly and include vomiting and fetid diarrhea.

<table>
<thead>
<tr>
<th>BOX 1-44 CAUSES OF ACUTE GASTRITIS</th>
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<tbody>
<tr>
<td>• Bacterial toxin</td>
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<tr>
<td>• Brain lesion</td>
</tr>
<tr>
<td>• Dietary indiscretion</td>
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<tr>
<td>• Drugs</td>
</tr>
<tr>
<td>• Food allergy</td>
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<tr>
<td>• Hepatic failure</td>
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<tr>
<td>• Infectious disease</td>
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<tr>
<td>• Renal failure</td>
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<tr>
<td>• Stress</td>
</tr>
<tr>
<td>• Toxic chemicals</td>
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<td>• Trauma</td>
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with hemorrhage, often strawberry jam–like in appearance. The hematocrit can rise from 55% to 75%. Often, the animal is extremely hypovolemic but has no apparent signs of abdominal pain. There is no known cause of HGE, although *Clostridium perfringens*, *E. coli*, *Campylobacter* species, and viral infections have been suggested but not consistently confirmed. Other differential diagnoses of hematemesis and hemorrhagic diarrhea include coronavirus, parvovirus, vascular stasis, sepsis, hepatic cirrhosis with portal hypertension, and other causes of severe shock.

Immediate treatment consists of placement of a large-bore intravenous catheter and replenishment of intravascular fluid volume with crystalloid fluids (up to 90 mL/kg) while carefully monitoring the patient’s hematocrit and total protein. Administer broad-spectrum antibiotics (ampicillin, 22 mg/kg IV q6-8h, and enrofloxacin, 10 mg/kg IV q24h) because of the high risk of bacterial translocation and sepsis. Control vomiting with antiemetic drugs. Monitor the patient’s platelet count and coagulation tests for impending DIC, and administer fresh frozen plasma and heparin as needed (see section on disseminated intravascular coagulation). When vomiting has ceased for 24 hours, offer the animal small amounts of water and then a bland diet (e.g., boiled chicken and rice or boiled ground beef and rice mixed with low-fat cottage cheese).

**Pancreatitis**

Pancreatitis occurs most frequently in dogs but can occur in cats as well. In dogs the onset of pancreatitis is sometimes preceded by ingestion of a fatty meal or the administration of drugs (e.g., potassium bromide or glucocorticoids). Glucocorticoids can increase the viscosity of pancreatic secretions and induce ductal proliferation, resulting in narrowing and obstruction of the lumen of the pancreatic duct. Pancreatitis can also occur after blunt or penetrating abdominal trauma, high duodenal obstruction causing outflow obstruction of the pancreatic papilla, pancreatic ischemia, duodenal reflux, biliary disease, and hyperadrenocorticism.

In cats, acute necrotizing pancreatitis is associated with anorexia, lethargy, hyperglycemia, icterus, and sometimes acute death. Chronic pancreatitis is more common in cats and results in intermittent vomiting, anorexia, weight loss, and lethargy. Predisposing causes of chronic pancreatitis in cats include pancreatic flukes, viral infection, hepatic lipodosis, drugs, organophosphate toxicity, and toxoplasmosis.

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**TABLE 1-37  Antiemetic Drugs and Dosages**

<table>
<thead>
<tr>
<th>Drugs (Proprietary Name)</th>
<th>Suggested Dosages*</th>
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<tbody>
<tr>
<td><strong>Phenothiazines</strong></td>
<td></td>
</tr>
<tr>
<td>Chlorpromazine (Thorazine)</td>
<td>0.2-0.5 mg/kg IM q8h, 0.05 mg/kg IV q4h, 1.0 mg/kg per rectum q8h (dog)</td>
</tr>
<tr>
<td>Prochlorperazine (Compazine)</td>
<td>0.1 mg/kg IM q6h, 0.5 mg/kg IV, IM q8h</td>
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<tr>
<td><strong>Serotonin Antagonists</strong></td>
<td></td>
</tr>
<tr>
<td>Dolasetron (Anzemet)</td>
<td>0.1-0.3 mg/kg IV q24h</td>
</tr>
<tr>
<td>Ondansetron (Zofran)</td>
<td>0.6-1.0 mg/kg IV q12h</td>
</tr>
<tr>
<td><strong>Others</strong></td>
<td></td>
</tr>
<tr>
<td>Metoclopramide (Reglan)*</td>
<td>0.2-0.5 mg/kg SQ q8h, 1.0-2.0 mg/kg/day IV, 3 mg/kg IM q8h (dog)</td>
</tr>
<tr>
<td>Neurokinin receptor antagonist</td>
<td>1 mg/kg SQ once daily for no more than 5 consecutive days</td>
</tr>
<tr>
<td>Maropitant</td>
<td>2 mg/kg PO once daily for no more than 5 consecutive days</td>
</tr>
</tbody>
</table>

*All doses apply to dogs and cats unless otherwise noted.
†Do not use until a gastrointestinal obstruction has been ruled out.

IM, Intramuscularly; IV, intravenously; PO, orally; SQ, subcutaneously.

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IM, Intramuscularly; IV, intravenously; PO, orally; SQ, subcutaneously.
*All doses apply to dogs and cats unless otherwise noted.
†Do not use until a gastrointestinal obstruction has been ruled out.
Clinical signs of acute pancreatitis include sudden severe vomiting, abdominal pain, and lethargy. Depending on the severity of pancreatic inflammation, depression, hypotension, and SIRS may be present. Subacute cases may have minimal clinical signs. Severe pancreatic edema can result in vascular changes and ischemia that perpetuates severe inflammation. Hypovolemic shock and DIC can also decrease pancreatic perfusion. Severe pancreatic edema, autolysis, and ischemia lead to pancreatic necrosis. Duodenal irritation is manifested as both vomiting and diarrhea. Pain may be localized to the right upper abdominal quadrant or may be generalized if peripancreatic saponification occurs. Differential diagnosis of pancreatitis is the same as for any other cause of vomiting.

Complications that occur in patients with severe pancreatitis include dehydration, acid-base and electrolyte abnormalities, hyperlipemia, hypotension, and localized peritonitis. Hepatic necrosis, lipodisosis, congestion, and abnormal architecture can develop. Inflammatory mediators (bradykinin, phospholipase A, elastase, myocar-dial depressant factor, and bacterial endotoxins) stimulate the inflammatory cascade and can lead to SIRS, with severe hypotension, clotting system activation, and DIC. Electrolyte imbalances and hypovolemia secondary to vomiting all can lead to multiple organ dysfunction syndrome (MODS) and ultimately death. If a patient survives an episode of acute pancreatitis, long-term sequelae can include diabetes mellitus. Monitor patients with recurrent pancreatitis for clinical signs of polyuria and polydipsia (PU/PD), polyphagia, hyperglycemia, and glucosuria.

The diagnosis of pancreatitis is based on the presence of clinical signs (which may be absent in cats), laboratory findings, and ultrasonographic evidence of pancreatic edema and increased peripancreatic echogenicity. Serum biochemistry analyses can sometimes support a diagnosis of pancreatitis; however, serum amylase and lipase are often unreliable indicators of pancreatitis, depending on the chronicity of the process in the individual patient. Both serum amylase and lipase are excreted in the urine. Impaired renal clearance or function can cause artifactual elevations of serum amylase and lipase in the absence of pancreatic inflammation. Furthermore, serum lipase levels can be elevated as a result of gastrointestinal obstruction (e.g., foreign body). Early in the course of the disease, levels can be two to six times normal, but they may decrease to within normal ranges at the time of presentation to the veterinarian. The transient nature of amylase elevation makes this test difficult to interpret, and it is not highly sensitive if a normal value is found. Lipase levels also increase later in the course of the disease. Amylase and lipase should be tested concurrently with the rest of the biochemistry profile.

Other changes often observed are elevations in BUN and creatinine levels secondary to dehydration and prerenal azotemia, hyperglycemia, and hyperlipemia. Hypocalcemia can occur secondary to peripancreatic fat saponification, and its presence warrants a more negative prognosis. A more specific measure is pancreatic lipase immunoreactivity, which becomes elevated in dogs and cats with pancreatitis. This test, combined with ultrasonographic or CT evidence of pancreatitis, is the most sensitive and specific test available for making an accurate diagnosis. However, because the results of this test take time to obtain, animals must be treated in the meantime.

Abdominal effusion or fluid from DPL can be compared with serum amylase and lipase activity. Abdominal lipase and amylase concentrations in the fluid greater than in the peripheral blood are characteristic of chemical peritonitis associated with pancreatitis. WBC counts greater than 1000 cells/mm³, the presence of bacteria, toxic neutrophils, glucose levels less than 50 mg/dL, or lactate levels greater than in serum are characteristic of septic peritonitis, and immediate exploratory laparotomy is warranted. If a biopsy sample obtained during laparotomy does not demonstrate inflammation, this does not rule out pancreatitis, because disease can be focal in nature and yet cause severe clinical signs.

Abdominal radiographs may sometimes reveal a loss of abdominal detail or a ground glass appearance in the right upper quadrant. Pancreatic edema and duodenal irritation can displace the gastric axis toward the left with dorsomedial displacement of the proximal duodenum (the “backward 7” or “shepherd’s crook” sign). Ultrasonography and CT are more sensitive in making a diagnosis of pancreatitis.
Treatment of pancreatitis is largely supportive in nature and is designed to correct hypovolemia and electrolyte imbalances, prevent or reverse shock, maintain vital organ perfusion, alleviate discomfort and pain, and prevent vomiting (see Rule of 20). When treating pancreatitis in dogs, all food and water should be restricted. However, food should not be withheld from cats with chronic pancreatitis. Give fresh frozen plasma to replenish α₂-macroglobulins. Administer antiemetics such as chlorpromazine (use with caution in a hypovolemic or hypotensive patient), dolasetron, ondansetron, or metoclopramide to prevent or control vomiting. Analgesic drugs can be provided in the form of CRI (fentanyl, 3-7 mcg/kg/hr IV CRI, and lidocaine, 30 to 50 mcg/kg/min IV CRI), intrapleural injection (lidocaine, 1 to 2 mg/kg q8h), or intermittent parenteral injections (morphine, 0.25 to 1 mg/kg SQ, IM; hydromorphone, 0.1 mg/kg IM or SQ). Because the pancreas must be rested, consider using parenteral nutrition.

**ACUTE HEPATIC FAILURE**

Acute hepatic failure may be associated with toxins, adverse reaction to prescription medication, and bacterial or viral infections. The most frequent clinical signs observed in a patient with acute hepatic failure are anorexia, lethargy, vomiting, icterus, bleeding, and CNS depression or seizures (associated with hepatic encephalopathy [HE]). Differential diagnosis and causes of acute hepatic failure are listed in Box 1-45.

Diagnosis of acute hepatic failure is based on clinical signs and biochemical evidence of hepatocellular (aspartate aminotransferase [AST], alanine aminotransferase [ALT]) and cholestatic (ALP, total bilirubin, γ-glutamyltransferase [GGT]) enzyme elevations. Ultrasonography may be helpful in distinguishing the architecture of the liver, but unless a mass or abscess is present, it cannot provide a specific diagnosis of the cause of the hepatic damage.

Management of the patient with acute hepatic failure includes correction of dehydration and acid-base and electrolyte abnormalities, as shown in the following list:

- **Hypoalbuminemia:** Plasma or concentrated albumin. Plasma also is an excellent source of clotting factors that can become depleted.
- **Clotting abnormalities:** Vitamin K₁ (2.5 mg/kg SQ or PO q8-12h) to restore available vitamin K₁.
- **Severe anemia:** Fresh or stored blood.
- **Gastric hemorrhage:** Gastroprotectant drugs (omeprazole, ranitidine, famotidine, cimetidine, sucralfate).

### BOX 1-45 CAUSES OF ACUTE HEPATIC FAILURE

<table>
<thead>
<tr>
<th><strong>ENDOGENOUS HEPATOTOXINS</strong></th>
<th>Mebendazole</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacterial endotoxins</td>
<td>Methoxyflurane</td>
</tr>
<tr>
<td><strong>ENVIRONMENTAL TOXINS</strong></td>
<td>Phenazopyridine</td>
</tr>
<tr>
<td>Aflatoxin</td>
<td>Phenytoin</td>
</tr>
<tr>
<td>Carbon tetrachloride</td>
<td>Sulfonamides (trimethoprim sulfadiazine, tetracycline)</td>
</tr>
<tr>
<td>Dimethylnitrosamine</td>
<td><strong>INFECTIOUS AGENTS</strong></td>
</tr>
<tr>
<td>Heavy metals, herbicides</td>
<td>Infectious canine hepatitis</td>
</tr>
<tr>
<td>Pesticides</td>
<td><em>Salmonella</em> species</td>
</tr>
<tr>
<td>Phosphorus</td>
<td><em>Leptospira</em> species</td>
</tr>
<tr>
<td>Pyrrolizidine alkaloids</td>
<td>Feline infectious peritonitis virus</td>
</tr>
<tr>
<td>Selenium</td>
<td><em>Toxoplasma gondii</em></td>
</tr>
<tr>
<td><strong>EXOGENOUS DRUGS</strong></td>
<td><em>Bacillus piliformis</em> (Tyzzer’s disease)</td>
</tr>
<tr>
<td>Acetaminophen</td>
<td><strong>OTHERS</strong></td>
</tr>
<tr>
<td>Arsenicals</td>
<td>Pancreatitis</td>
</tr>
<tr>
<td>Azathioprine</td>
<td>Septicemia</td>
</tr>
<tr>
<td>Carprofen</td>
<td>Inflammatory bowel disease</td>
</tr>
<tr>
<td>Griseofulvin</td>
<td>Acute hemolytic anemia</td>
</tr>
<tr>
<td>Halothane</td>
<td></td>
</tr>
<tr>
<td>Ketoconazole</td>
<td></td>
</tr>
</tbody>
</table>
Hypoglycemia: Dextrose supplementation (2.5% to 5%).
Hepatic failure, particularly when hypoglycemia is present: Broad-spectrum antibiotics (ampicillin, 22 mg/kg IV q6-8h; with enrofloxacin, 10 mg/kg IV q24h).
HE: Lactulose or Betadine enemas.
Cerebral edema: Mannitol (0.5 to 1.0 g/kg IV over 10 to 15 minutes) followed by furosemide (1 mg/kg IV 20 minutes later). Deterioration of clinical signs may signify the development of cerebral edema.

Additional Reading

HYPERTENSION: SYSTEMIC
Systemic hypertension is a recognized syndrome in dogs and cats and occurs most commonly secondary to acute or chronic renal failure, and less commonly as a primary idiopathic disease entity. Risk factors for the development of systemic hypertension include obesity, diabetes mellitus, and chronic kidney disease.
hypertension in dogs and cats include renal insufficiency, hyperadrenocorticism, hyperthyroidism, pheochromocytoma, diabetes mellitus, polycythemia vera, hyperaldosteronism, hypertensive encephalopathy, acromegaly, intracranial hemorrhage, and CNS trauma.

Often, systemic hypertension is diagnosed when the animal is seen by the veterinarian because of some other clinical sign, such as acute blindness, retinal detachment, hyphema, epistaxis, and CNS signs following intracranial hemorrhage. Diagnosis of systemic hypertension is often difficult in the absence of clinical signs and without performing invasive or noninvasive BP monitoring. Normal BP measurements in dogs and cats are listed in Table 1-38.

Hypertension is defined as a consistent elevation in systolic BP >200 mm Hg, consistent diastolic BP >110 mm Hg, and consistent mean arterial BP >130 mm Hg. The effects of systemic hypertension include left ventricular hypertrophy, cerebrovascular accident, renal vascular injury, optic nerve edema, hyphema, retinal vascular tortuosity, retinal hemorrhage, retinal detachment, vomiting, neurologic defects, coma, and excessive bleeding from cut surfaces.

Patients with systemic hypertension should have a thorough diagnostic workup to determine the underlying cause. Although uncommon, hypertensive emergencies can occur with pheochromocytoma, acute renal failure, and acute glomerulonephritis. Sodium nitroprusside (1 to 10 mcg/kg/min IV CRI) or diltiazem (0.3 to 0.5 mg/kg IV given slowly over 10 minutes, followed by 1-5 mcg/kg/min) can be used to treat systemic hypertension. With the use of sodium nitroprusside or diltiazem, monitor carefully for hypotension.

Diagnosis is based on consistent elevations in systolic, diastolic, and/or mean arterial BP. Because many of the clinical signs associated with systemic hypertension involve hemorrhage into some closed cavity, other causes of hemorrhage, such as vasculitis, thrombocytopenia, thrombocytopenia, and hepatic or renal failure, should be investigated (see section on coagulation disorders). Diagnostic testing is based on clinical signs and index of suspicion for an underlying disease and may include a complete blood count; urinalysis; urine protein/creatinine ratio; adrenocorticotropic hormone (ACTH) stimulation test; thoracic and abdominal radiographs; thoracic and abdominal ultrasound; tick serology; brain CT or MRI; and assays of serum electrolytes, aldosterone concentration, T4, endogenous thyroid-stimulating hormone (TSH), plasma catecholamine, and growth hormone.

Management of systemic hypertension involves treatment of the primary underlying disorder, whenever possible. Long-term adjunctive management includes sodium restriction in the form of cooked or prescription diets to decrease fluid retention. Obese animals should be placed on dietary restrictions and undergo a weight reduction program. Thiazide and loop diuretics may be used to decrease sodium retention and circulating blood volume. α-adrenergic and β-adrenergic blockers may be used, but they are largely ineffective as monotherapeutic agents for treating hypertension. Calcium channel blockers and ACE inhibitors are the mainstay of therapy in the treatment of hypertension in dogs and cats (Table 1-39).
TABLE 1-39  Drugs Used to Treat Systemic Hypertension

<table>
<thead>
<tr>
<th>Drug</th>
<th>Canine Dosage</th>
<th>Feline Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angiotensin-Converting Enzyme Inhibitors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enalapril</td>
<td>0.5-1.0 mg/kg PO q12-24h</td>
<td>0.25-0.5 mg/kg PO q12-24h</td>
</tr>
<tr>
<td>Benazepril</td>
<td>0.25-0.5 mg/kg PO q12-24h</td>
<td>Same as canine dosage</td>
</tr>
<tr>
<td>Cl-Adrenergic Blocker</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prazosin</td>
<td>0.5-2.0 mg PO q12h</td>
<td>Not used</td>
</tr>
<tr>
<td>β-Adrenergic Blockers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Propranolol</td>
<td>2.5-10.0 mg PO q8-12h</td>
<td>2.5-5.0 mg PO q8-12h</td>
</tr>
<tr>
<td>Atenolol</td>
<td>0.25-1.0 mg/kg PO q12-24h</td>
<td>6.25-12.5 mg PO q12-24h</td>
</tr>
<tr>
<td>Calcium Channel Blocker</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amlodipine</td>
<td>0.05-0.2 mg/kg PO q24h</td>
<td>0.625-1.25 mg PO q24h</td>
</tr>
<tr>
<td>Thiazide Diuretic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hydrochlorothiazide</td>
<td>1 mg/kg PO q12-24h (cats and dogs)</td>
<td></td>
</tr>
<tr>
<td>Loop Diuretic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Furosemide</td>
<td>2.0-4.0 mg/kg PO q12-24h</td>
<td></td>
</tr>
<tr>
<td>Phthalazine Derivative</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hydralazine</td>
<td>0.5-2.0 mg/kg PO q8-12h</td>
<td>2.5 mg PO q12-24h</td>
</tr>
</tbody>
</table>

PO, Orally.

Additional Reading

METABOLIC EMERGENCIES

Diabetic Ketoacidosis
DKA is a potentially fatal and terminal consequence of unregulated insulin deficiency and possible glucagon excess. In the absence of insulin, unregulated lipolysis results in the β-hydroxylation of fatty acids by abnormal hepatic metabolism. As a result, ketoacids—namely, acetoacetic acid, β-hydroxybutyric acid, and acetone—are produced. Early in the course of the disease, patients exhibit clinical signs associated with diabetes mellitus: weight loss, polyuria, polyphagia, and polydipsia. Later, as ketoacids stimulate the chemoreceptor trigger zone, vomiting and dehydration occur, with resulting hypovolemia, hypotension, severe depression, abdominal pain, oliguria, and coma. At the time of presentation, often a strong odor of ketones (acetone) is present on the patient’s breath.

Physical examination often reveals dehydration, severe depression or coma, and hypovolemic shock. In extreme cases the patient exhibits a slow, deep Kussmaul respiratory pattern in an attempt to blow off excess CO₂ to compensate for the metabolic acidosis. A serum biochemistry profile and complete blood count often reveal prerenal azotemia, severe hyperglycemia (blood glucose >400 mg/dL), hyperosmolarity (>330 mOsm/kg), lipemia, hypernatremia (sodium >145 mEq/L), elevated hepatocellular and cholestatic enzyme...
activities, high anion gap, and metabolic acidosis. Although a whole-body potassium deficit is usually present, the serum potassium may appear artifactually elevated in response to metabolic acidosis. With severe metabolic acidosis, potassium moves extracellularly in exchange for a hydrogen ion. Phosphorus too moves intracellularly in response to acidosis, and serum phosphorus is usually decreased. Hypophosphatemia (>2 mg/dL) can result in intravascular hemolysis. Urinalysis often reveals 4+ glucosuria, ketonuria, and a specific gravity of 1.030 or greater. The urine of all diabetic animals should be cultured to rule out a urinary tract infection or pyelonephritis.

Treatment of a patient with DKA presents a therapeutic challenge. Treatment is aimed at providing adequate insulin to normalize cellular glucose metabolism, correcting acid-base and electrolyte imbalances, rehydrating and restoring perfusion, correcting acidosis, providing carbohydrate sources for use during insulin administration, and identifying any precipitating cause of the DKA.

Obtain blood samples for a complete blood count and serum biochemistry electrolyte profiles. Whenever possible, insert a central venous catheter for fluid infusion and procurement of repeat blood samples. Calculate the patient’s dehydration deficit and maintenance fluid requirements and give appropriate fluid and electrolytes over a period of 24 hours. It is advisable to rehydrate patients with severe hyperosmolarity for a minimum of 6 hours before starting insulin administration. Use a balanced electrolyte solution (e.g., Plasma-Lyte A, Normosol-R, lactated Ringer’s solution) or 0.9% saline solution for maintenance and rehydration. Balanced electrolyte solutions contain small amounts of potassium and bicarbonate precursors that aid in the treatment of metabolic acidosis. Treat animals with severe metabolic acidosis with an \( \text{HCO}_3^- \geq 11 \) mEq/L or a pH < 7.1 with supplemental bicarbonate (0.25 to 0.5 mEq/kg). Add supplemental dextrose to the patient’s fluids as a carbohydrate source during insulin infusion.

Both insulin and carbohydrates are necessary for the proper metabolism of ketone bodies in patients with DKA. The rate and type of fluid and amount of dextrose supplementation will change according to the patient’s blood glucose concentration. Serum potassium will drop rapidly as the metabolic acidosis is corrected with fluid and insulin administration. Measure serum potassium every 8 hours, if possible, and supplement accordingly (see section on fluid therapy for chart of potassium supplementation). If the patient’s potassium requirement exceeds 100 mEq/L, or if the rate of potassium infusion approaches 0.5 mEq/kg/hr in the face of continued hypokalemia, magnesium should be supplemented. Magnesium is required as a cofactor for many enzymatic processes and for normal function of the Na,K-ATPase pump. Hypomagnesemia is a common electrolyte disturbance in many forms of critical illness. Replenishing magnesium (MgCl\(_2\), 0.75 mEq/kg/day IV CRI) often helps to correct the refractory hypokalemia observed in patients with DKA. Patients with hypophosphatemia that approaches 2.0 mmol/L should receive potassium phosphate (0.01 to 0.03 mmol/kg/hr IV CRI). When providing potassium phosphate supplementation, be aware of the additional potassium added to the patient’s fluids, so as to not exceed recommended rates of potassium infusion. To determine the amount of potassium chloride (KCl) to add along with potassium phosphate (KPO\(_4\)), use the following formula:

\[
m\text{Eq K}^+ \text{derived from KCl} = \text{Total mEq of K}^+ \text{to be administered over 24 hours} - m\text{Eq in which K}^+ \text{is derived from KPO}_4
\]

Clinical signs of severe hypophosphatemia include muscle weakness, rhabdomyolysis, intravascular hemolysis, and decreased cerebral function that can lead to depression, stupor, seizures, or coma.

**Insulin Administration**

Regular insulin can be administered either IM or as a CRI in the treatment of patients with DKA. Subcutaneous insulin should not be administered. Because of the severe dehydration present in most patients with DKA, subcutaneous insulin is poorly absorbed and is not effective until hydration has been restored.
In the low-dose intravenous method, place regular insulin (1.1 units/kg for a cat, and 2.2 units/kg for a dog) in 250 mL of 0.9% saline solution. Run 50 mL of this mixture through the intravenous line to allow the insulin to adsorb to the plastic tubing. Administer the patient’s insulin fluid rate according to blood glucose levels (Table 1-40). Adjust the patient’s total fluid volume according to changes in the insulin fluid rate as necessary. In many cases, multiple bags of fluids are necessary because they must be changed when fluctuations in blood glucose concentrations occur in response to therapy. Infusion of the insulin mixture should be in a separate intravenous catheter. To replenish hydration, use a second intravenous line for the more rapid infusion of non–insulin-containing fluids.

To administer the regular insulin IM, first give 0.22 unit/kg IM and then recheck the patient’s blood glucose every hour. Additional injections of regular insulin (0.11 unit/kg IM) should be administered based on the patient’s response to subsequent injections. Once the patient’s blood glucose falls to 200 to 250 mg/dL, add 2.5% to 5% dextrose to the fluids to maintain the blood glucose concentration at 200 to 300 mg/dL. Continue intramuscular injection of regular insulin (0.1 to 0.4 unit/kg q4-6h) until the patient is rehydrated, no longer vomiting, and able to tolerate oral fluids and food without vomiting. Even in patients with intramuscular regular insulin therapy, a central venous catheter should be placed for frequent blood sample collection. As the patient begins to respond to therapy, monitor electrolytes, glucose, and acid-base status carefully. Hypokalemia, hypophosphatemia, and hypomagnesemia can occur. When the patient’s hydration and acid-base status has normalized and the patient is able to tolerate oral food and water, a longer-acting insulin can be administered as for treatment of a patient with uncomplicated diabetes.

**HYPEROSMOLAR NONKETOTIC DIABETES**

Extreme hyperosmolality can result in a coma, if uncorrected. In patients with diabetes mellitus, hyperglycemia and hypernatremia secondary to osmotic diuresis and free water loss can lead to severe hyperosmolality. In dogs, normal serum osmolality is <300 mOsm/L of serum. Hyperosmolality is expected when serum osmolality is >340 mOsm/L. If equipment for determining serum osmolarity is not available, osmolarity can be calculated by the following formula:

\[ \text{Osm} / \text{L} = 2(\text{Na} + \text{K}) + (\text{glucose} / 18) + (\text{BUN} / 2.8) \]

Patients with severe dehydration, hyperglycemia, hypernatremia, and azotemia may experience cerebral edema without ketonemia. Treatment is directed solely at rehydrating the patient and slowly reducing blood glucose levels using a hypotonic solution such as 0.45% NaCl + 2.5% dextrose or 5% dextrose in water (D5W). After the initial rehydration period, administer potassium supplementation conservatively.

| TABLE 1-40 Type of Fluid and Rate of Insulin Infusion, Based on Patient’s Blood Glucose Concentration, in the Treatment of Diabetic Ketoacidosis* |
|----------------|------------------|------------------|
| Blood Glucose (mg/dL) | Rate of Insulin/0.9% NaCl Infusion (mL/hr) | Other Fluid Type (mL/hr) |
| >250 | 10 | 0.9% NaCl |
| 200-250 | 7 | 0.45% NaCl + 2.5% dextrose |
| 150-200 | 5 | 0.45% NaCl + 2.5% dextrose |
| 100-150 | 5 | 0.45% NaCl + 2.5% dextrose |
| <100 | 0 | 0.45% NaCl + 5% dextrose |

*1.1 Units of regular insulin per kilogram in 250-mL bag 0.9% saline for cats, and 2.2 units regular insulin per kilogram per 250-mL bag 0.9% saline for dogs.
Hypoglycemia

RBCs and the brain absolutely depend on the oxidation of glucose for energy. Hypoglycemia can be caused by various systemic abnormalities that can be related to intestinal malabsorption of nutrients, impaired hepatic glycogenolysis or gluconeogenesis, and inadequate peripheral use of glucose. Clinical signs of hypoglycemia are extremely variable and can include weakness, tremors, nervousness, polyphagia, ataxia, tachycardia, muscle twitching, incoordination, visual disturbances, and generalized seizures. Clinical signs typically occur when serum glucose levels are <60 mg/dL. The combination of the clinical signs listed previously, documentation of low serum glucose, and alleviation of clinical signs on glucose administration is known as Whipple’s triad.

Whenever a patient with hypoglycemia is presented, consider the following important factors: the age at onset, the nature of the hypoglycemic episode (transient, persistent, or recurrent), and the pattern based on the patient’s history (Box 1-46).

Treatment of hypoglycemia is directed at providing glucose supplementation and determining any underlying cause. Administer supplemental dextrose (25% to 50% dextrose, 2 to 5 mL/kg IV; or 10% dextrose, 20 mL/kg PO) as quickly as possible. Do not attempt oral glucose supplementation in any patient having a seizure or if the airway cannot be protected. Administer intravenous fluids (e.g., Normosol-R, lactated Ringer’s solution, 0.9% saline solution) with 2.5% to 5% supplemental dextrose until the patient is eating and able to maintain euglycemia without supplementation. In some cases (e.g., insulinoma), eating or administration of supplemental dextrose can promote insulin secretion and exacerbate clinical signs and hypoglycemia. In cases of refractory hypoglycemia secondary to iatrogenic insulin overdose, glucagon (50 ng/kg IV bolus, then 10 to 40 ng/kg/min IV CRI) can also be administered, along with supplemental dextrose. To make a glucagon infusion of 1000 ng/mL, reconstitute 1 mL (1 mg/mL) of glucagon according to the manufacturer’s instructions and add this amount to 1000 mL of 0.9% saline solution.

Hypocalcemia: Eclampsia (Puerperal Tetany)

The diagnosis of eclampsia (puerperal tetany) is often made on the basis of history and clinical signs. Clinical signs can become evident when total calcium decreases to <8.0 mg/dL in dogs and <7.0 mg/dL in cats. The disease is often observed in small, excitable dogs, and stress may play a complicating role in the cause. In most bitches the disease manifests itself 1 to 3 weeks after parturition. In some cases, however, clinical signs can develop before parturition occurs. Hypophosphatemia may accompany hypocalcemia. Clinical signs of

<table>
<thead>
<tr>
<th>Box 1-46</th>
<th>Causes of Hypoglycemia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Accelerated Glucose Removal</strong></td>
<td>Neonatal hypoglycemia</td>
</tr>
<tr>
<td>Insulin overdose</td>
<td>“Toy breed hypoglycemia”</td>
</tr>
<tr>
<td>Ethanol poisoning</td>
<td>“Hunting breed hypoglycemia”</td>
</tr>
<tr>
<td>Salicylate toxicity</td>
<td>Starvation</td>
</tr>
<tr>
<td>Propranolol</td>
<td>Hepatic enzyme insufficiencies</td>
</tr>
<tr>
<td>Functional islet cell tumor</td>
<td>Hypoadrenocorticism</td>
</tr>
<tr>
<td>Toxicity</td>
<td>Hepatic insufficiency</td>
</tr>
<tr>
<td>Oral hypoglycemic agents</td>
<td>Malabsorption and starvation</td>
</tr>
<tr>
<td>Renal glucosuria</td>
<td>Large mesodermal tumors</td>
</tr>
<tr>
<td>Hepatoma</td>
<td>Sepsis</td>
</tr>
<tr>
<td>Endotoxemia</td>
<td>Increased extrahepatic glucose substrate utilization</td>
</tr>
<tr>
<td><strong>Failure of Glucose Secretion</strong></td>
<td>Renal failure</td>
</tr>
<tr>
<td>Functional hypoglycemia (nonrecognizable lesion)</td>
<td>Extrahepatic tumors</td>
</tr>
</tbody>
</table>
hypocalcemia include muscle tremors or fasciculations, panting, restlessness, aggression, hypersensitivity, disorientation, muscle cramping, hyperthermia, stiff gait, seizures, tachycardia, a prolonged QT interval on ECG, PU/PD, and respiratory arrest.

Treatment of eclampsia consists of slow, cautious calcium supplementation (10% calcium gluconate, 0.15 ml/kg IV over 30 minutes). Severe refractory tetanus can be controlled with intravenous diazepam. Supportive care includes intravenous fluid administration and cooling (see section on hyperthermia and heat-induced illness). Instruct the owner to give the patient oral calcium supplements (e.g., 1 or 2 tablets of Tums bid or tid) after discharge from the hospital. Also instruct the owner about how to wean the puppies, allowing the bitch to dry up, in order to prevent recurrence. Recurrence with subsequent pregnancies is common, particularly in patients that receive calcium supplementation during gestation (Table 1-41).

Hypercalcemia

Hypercalcemia can occur from a variety of causes. The GOSH DARN IT mnemonic can be used to remember the various causes of hypercalcemia in small animal patients (Box 1-47).

The gastrointestinal, renal, and nervous systems are most commonly affected, particularly when serum total calcium rises above 16.0 mg/dL. Clinical signs of severe hypercalcemia include muscle weakness, vomiting, seizures, and coma. ECG abnormalities include prolonged PR interval, rapid QT interval, and ventricular fibrillation. The most serious clinical signs are often seen when hypercalcemia is observed in combination with hyperphosphatemia or hypokalemia. Pay special attention to the “calcium × phosphorus product.” If this product exceeds 70, dystrophic calcification can occur, leading to renal failure. Renal complications include PU/PD, dehydration, and loss of renal tubular concentrating ability. Renal blood flow and the glomerular filtration rate (GFR) are impaired when serum total calcium exceeds 20 mg/dL. The extent, location, and number of renal tubular injuries are the main factors in determining whether renal damage secondary to hypercalcemia is reversible or irreversible.

Emergency therapy of hypercalcemia is warranted when severe renal compromise, cardiac dysfunction, or neurologic abnormalities are present, or if no clinical signs occur but the calcium × phosphorus product exceeds 70. The treatment of choice is correction of the underlying cause of hypercalcemia, whenever possible. In some cases the results of diagnostic tests take time, and emergency therapy should be initiated immediately, before a definitive cause of the hypercalcemia is found. Emergency management of hypercalcemia consists of reduction of serum calcium levels. Administer intravenous fluids (0.9% saline solution) to expand extracellular fluid volume and promote calciuresis. To promote diuresis, initial intravenous fluid rates should approach two to three times maintenance levels (120 to 180 mL/kg/day). Potassium supplementation may be required to prevent iatrogenic hypokalemia. Administration of a loop diuretic such as furosemide (2 to 5 mg/kg IV) will promote calcium excretion. Calcitonin (4 IU per kilogram IM q12h for cats and 8 IU per kilogram SQ q24h for dogs) can be administered to decrease serum calcium levels. In severe refractory hypercalcemia secondary to cholecalciferol toxicity, more aggressive calcitonin therapy (4 to 7 IU per kilogram SQ q6-8h) can be attempted. Side effects of calcitonin treatment include vomiting and diarrhea. Alternatively, bisphosphonates (pamidronate, 1.02 to 2.0 mg/kg IV) are useful in rapidly reducing serum calcium concentrations.

Glucocorticosteroids reduce calcium release from the bone, decrease intestinal absorption of calcium, and promote renal calcium excretion. Administer glucocorticosteroids only after the underlying cause of hypercalcemia has been determined and appropriate therapy started. Because many forms of neoplasia can result in hypercalcemia as a paraneoplastic syndrome, empiric use of glucocorticosteroids can induce multiple drug resistance, making the tumor refractory to the effects of chemotherapeutic agents.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Preparation</th>
<th>Available Calcium</th>
<th>Dosage</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Parenteral Calcium</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcium gluconate</td>
<td>10% solution</td>
<td>9.3 mg/mL</td>
<td>a. Slowly IV to effect (0.5-1.5 mL/kg IV)</td>
<td>Stop if bradycardia or shortened QT interval occurs; infusion to maintain normal Ca; may be given SQ</td>
</tr>
<tr>
<td>Calcium chloride</td>
<td>10% solution</td>
<td>27.2 mg/mL</td>
<td>b. 5-15 mg/kg/hr IV</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>c. 1-2 mL/kg diluted 1:1 with saline SQ tid</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>5-15 mg/kg/hr IV</td>
<td></td>
</tr>
<tr>
<td><strong>Oral Calcium</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcium carbonate</td>
<td>Many sizes</td>
<td>40% tablet</td>
<td>25-50 mg/kg/day</td>
<td>Most common calcium supplement</td>
</tr>
<tr>
<td>Calcium lactate</td>
<td>325-, 650-mg tablets</td>
<td>13% tablet</td>
<td>25-50 mg/kg/day</td>
<td></td>
</tr>
<tr>
<td>Calcium chloride</td>
<td>Powder</td>
<td>27.2%</td>
<td>25-50 mg/kg/day</td>
<td>May cause gastric irritation</td>
</tr>
<tr>
<td>Calcium gluconate</td>
<td>Many sizes</td>
<td>10%</td>
<td>25-50 mg/kg/day</td>
<td></td>
</tr>
<tr>
<td><strong>Vitamin D</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitamin D&lt;sub&gt;1&lt;/sub&gt;</td>
<td>Capsules, syrup</td>
<td>—</td>
<td>Initial: 4000-6000 units/kg/day</td>
<td></td>
</tr>
<tr>
<td>(ergocalciferol)</td>
<td>parenteral</td>
<td></td>
<td>Maintenance: 1000-2000 units/kg PO</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(intramuscular)</td>
<td></td>
<td>once daily to once weekly</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dihydrotachysterol</td>
<td>Tablets, capsules,</td>
<td>—</td>
<td>Initial: 0.02-0.03 mg/kg/day PO</td>
<td></td>
</tr>
<tr>
<td></td>
<td>oral solution</td>
<td></td>
<td>Maintenance: 0.01-0.02 mg/kg PO</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>q24-48h</td>
<td></td>
</tr>
<tr>
<td>1,25-dihydroxyvitamin</td>
<td>Capsules</td>
<td>—</td>
<td>2.5-3.5 mg/kg/day PO</td>
<td></td>
</tr>
<tr>
<td>D&lt;sub&gt;3&lt;/sub&gt; (calcitriol)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


*IV, Intravenously; SQ, subcutaneously.

*Do not mix calcium solution with bicarbonate-containing fluids, as precipitation may occur.

†Calculate dose on elemental calcium content.
Hypoadrenocorticism is most commonly observed in young to middle-aged female dogs, but it can occur in animals of any age, gender, and breed. Clinical signs, which are referable to deficiency in glucocorticoid (cortisol) and mineralocorticoid (aldosterone) hormones, may develop slowly over time, leading to a waxing and waning course; acute clinical signs occur when >90% of the adrenal functional reserve has been destroyed. In such cases, complete adrenocortical collapse can result in an addisonian crisis. Lack of aldosterone causes a lack of renal sodium and water retention, and impaired potassium excretion. The most significant clinical signs associated with hypoadrenocorticism are depression, lethargy, weakness, anorexia, shaking, shivering, vomiting, diarrhea, weight loss, abdominal pain, weakness, hypotension, dehydration, and inappropriate bradycardia (Box 1-47).

The diagnosis of hypoadrenocorticism is made based on the patient’s clinical signs in combination with electrolyte abnormalities that include hyperkalemia, hyponatremia, and hypocholesterolemia. Serum sodium concentration (115 to 130 mEq/L) is often greatly reduced, and serum potassium is elevated (>6.0 mEq/L). A sodium:potassium ratio of <27 is characteristic of hypoadrenocorticism, although not exactly pathognomonic. Electrocardiographic changes associated with hyperkalemia include inappropriate bradycardia, absence of p waves, elevated spiked T waves, and widened QRS complexes. Other, more variable bloodwork abnormalities include a lack of a stress leukogram, eosinophilia, hypoglycemia, hyperphosphatemia, hypercalcemia, azotemia, and hypocholesterolemia. A definitive diagnosis of hypoadrenocorticism is based on an ACTH stimulation test. In patients with hypoadrenocorticism, baseline cortisol levels are usually low, with a lack of appropriate cortisol release after administration of ACTH analogue. Rarely, animals with “atypical” hypoadrenocorticism lose glucocorticoid-secreting ability from the zona fasciculata but retain mineralocorticoid secretory ability from the zona glomerulosa. Atypical addisonian patients have normal serum electrolytes but still have clinical signs of vomiting, diarrhea, weakness, lethargy, inappetence, muscle wasting, and weight loss. The diagnosis is more difficult in such cases because of the presence of normal electrolytes. An ACTH stimulation test should be considered, particularly in predisposed breeds.

Treatment of hypoadrenocorticism includes placement of a large-bore intravenous catheter, infusion of intravenous crystalloid fluids (0.9% saline solution), and replenishment of glucocorticoid and mineralocorticoid hormones. Administer dexamethasone or dexamethasone–sodium phosphate (0.5 to 1.0 mg/kg IV). Dexamethasone will not

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**BOX 1-47 CAUSES OF HYPERCALCEMIA**

- Granulomatous (fungal disease)
- Osteogenic
- Spurious (laboratory error)
- Hyperparathyroidism
- Vitamin D toxicosis
- Addison’s disease (hypoadrenocorticism)
- Renal failure
- Neoplasia (lymphoma, multiple myeloma, osteosarcoma)
- Idiopathic (cats)
- Toxins and drugs (overzealous calcium administration; thiazide diuretics)

**ACUTE ADRENOCORTICAL INSUFFICIENCY (HYPOADRENOCORTICISM, ADDISON’S DISEASE)**

**BOX 1-48 BREED PREDISPOSITION TO HYPOADRENOCORTICISM**

- Bassett Hound
- Bearded Collie
- Great Dane
- Great Pyrenees
- Portuguese Water Dog
- Standard Poodle
- West Highland White Terrier

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interfere with the ACTH stimulation test, unlike longer-acting steroids (e.g., prednisolone, methylprednisolone sodium succinate, triamcinolone). Depending on the severity of the patient’s condition, consider monitoring using the Rule of 20. Administer antimetics and gastroprotectant drugs to treat nausea, vomiting, and hematemesis. Give the patient broad-spectrum antibiotics (ampicillin, 22 mg/kg IV q6h) if hematochezia or hemorrhagic diarrhea is present. If severe gastrointestinal blood loss occurs, whole blood, packed RBCs, or fresh frozen plasma may be required. Control hypoglycemia with 2.5% to 5.0% dextrose. Use sodium bicarbonate, regular insulin with dextrose, or calcium gluconate to correct severe hyperkalemia with atrial standstill (see section on atrial standstill).

Chronic therapy for hypoadrenocorticism consists of mineralocorticoid and glucocorticosteroids supplementation for the rest of the animal’s life. Mineralocorticoid supplementation can be in the form of desoxycorticosterone pivalate (DOCP) (2.2 mg/kg IM) or fludrocortisone acetate (0.1 mg/2.5 to 5 kg body weight daily). Fludrocortisone acetate possesses both mineralocorticoid and glucocorticoid activities and can be used as the sole daily treatment of hypoadrenocorticism. (Because fludrocortisone is poorly absorbed in some dogs, it may not completely normalize electrolyte abnormalities in these animals.) DOCP is primarily a mineralocorticoid. Give supplemental glucocorticosteroids in the form of prednis(ol)one (0.25 to 1 mg/kg/day).

In dogs, iatrogenic hypoadrenocorticism can be caused by abrupt discontinuation of glucocorticosteroid treatment. Long-term glucocorticosteroid supplementation can downregulate the pituitary gland’s excretion of endogenous ACTH and the zona fasciculata’s ability to excrete cortisol. However, the zona glomerulosa’s ability to secrete aldosterone does not appear to be affected. Clinical signs of iatrogenic hypoadrenocorticism include inability to compensate for stress, weakness, lethargy, vomiting, diarrhea, and collapse. Treatment of iatrogenic hypoadrenocorticism is the same as for naturally occurring disease. After immediate emergency treatment, the patient should be weaned slowly from exogenous glucocorticosteroid supplementation.

Thyrotoxicosis
Severe hyperthyroidism can manifest as a medical emergency as a result of hypermetabolism. Clinical signs in affected cats with severe thyrotoxicosis include fever, severe tachycardia (heart rate >240 beats/min), vomiting, hypertension, CHF with pulmonary edema, and fulminant collapse. Clinical signs typically manifest as an end stage of chronic debilitation associated with hyperthyroidism and are often preceded by polyphagia, weight loss, cardiac murmur, PU/PD, vomiting, and diarrhea.

Treatment of thyrotoxicosis includes antagonizing the adrenergic activity by administration of a β-adrenergic blocker (esmolol, 25 to 50 mcg/kg/min, or propranolol, 0.02 mg/kg/hr). Administration of glucocorticosteroids (dexamethasone, 1 mg/kg) may inhibit the conversion of thyroxine (T₄) to the active form triiodothyronine (T₃) and decrease peripheral tissue responsiveness to T₃, effectively blocking its effects. Correct hypoglycemia with supplemental dextrose (2.5%). Use care to avoid overhydration in a patient with cardiac failure or insufficiency. Start the patient on methimazole as quickly as possible, and consider the use of radioactive iodine therapy.

Additional Reading
NEUROLOGIC EMERGENCIES

Four classes of neurologic injuries can seriously jeopardize a patient's life: head injuries, spinal cord and vertebral column injuries, coma, and seizure. The separate entities are discussed in this section.

HEAD INJURIES

Head injuries can be associated with skin and superficial lacerations, concussions, fractures, and hemorrhage (intracranial and extracranial). Fractures may be extracranial, linear, or depressed intracranial. Hemorrhage can be extradural, intradural, subdural, subarachnoid, or intracerebral. Immediately perform a baseline physical examination of an animal with head trauma at the time of presentation to assess neurologic status and determine whether progressive deterioration exists (Table 1-42).

During the initial examination, note the patient's ABCs (airway, breathing, and circulation). If necessary, establish an airway. Always supply supplemental oxygen to maintain Spo₂ >90%. Place an intravenous catheter and start increments of a shock dose of intravenous fluids (1/4 of 90 mL/kg for dogs: 44 mL/kg for cats). In order to maintain cerebral perfusion pressure, BP must be normalized. If other concurrent injuries are suspected (e.g., pulmonary contusions), administer synthetic colloid fluids (hydroxyethyl starch, 5 to 10 mL/kg IV) to normalize BP. Although the use of colloids is controversial because of their potential to leak into the calvarium, the benefits of reestablishing cerebral perfusion far outweigh the risks of their use. Hypertonic saline (7.5% NaCl, 3 to 5 mL/kg IV) can also be administered over 10 to 15 minutes to expand intravascular volume. Maintain blood glucose within normal reference ranges whenever possible, because hyperglycemia is a negative prognostic indicator in cases of head trauma. If tremors or seizures cause hyperthermia or increased metabolism, active cooling of the patient is warranted (see sections on hyperthermia and heat-induced injury). All patients with head trauma should receive care and monitoring based on the Rule of 20 (see Rule of 20).
Examine the patient’s level of consciousness, response to various stimuli, pupil size and reactivity to light, physiologic nystagmus, and cranial nerve deficits. In dogs, damage to the midbrain often produces coma and decerebrate rigidity. Initial consciousness followed by unconsciousness or stupor usually involves an injury to the brainstem. Brainstem lesions can be caused by compressive skull fractures, extradural or subdural hematomas, or herniation through the foramen magnum from cerebral edema (Box 1-49).

The patient’s pupil size and response to light can be used to localize a diagnosis and give a rough prognosis for severity of disease and possibility for return to function. Pupils can be normal in size, mydriatic, or miotic. Whenever a pupil appears miotic, direct ocular injury with uveitis or secondary miosis from brachial plexus injury should be ruled out. The eyes should always be examined to rule out ocular trauma.

In a patient with head trauma, a change from dilated to constricted to normal pupil size is suggestive of improvement in clinical function. Bilateral mydriatic pupils that are unresponsive to light in an unconscious animal are a grave prognostic sign and usually indicate an irreversible severe midbrain contusion. Bilateral miotic pupils with normal nystagmus and ocular movements are associated with diffuse cerebral or diencephalic lesions. Miotic pupils that become mydriatic indicate a progressive midbrain lesion with a poor prognosis. Unilateral, slowly progressive pupillary abnormalities in the absence of direct ocular injury are characteristic of brainstem compression or herniation caused by progressive brain swelling. Asymmetric pupils are seen in patients with rostral brainstem lesions and can change rapidly. Unresponsive pupils that are seen in the midposition occur with brainstem lesions that extend into the medulla and are a grave sign.

### BOX 1-49 LEVELS OF CONSCIOUSNESS

| Alertness: Alert, responsive, appropriate reaction to external stimuli |
| Depression: Appears lethargic and has sluggish response to external stimuli |
| Confusion: May appear confused or aggressive |
| Delirium: Vocalization, inappropriate response to external stimuli |
| Semicoma: Unconscious but responds to external noxious stimuli |
| Coma: Unconscious with no response to noxious stimuli |
Visual deficits are common with intracranial injury. Lesions that are less severe and limited to the cerebrum produce contralateral menace deficits with normal pupillary light response. Bilateral cerebral edema can cause blindness with a normal response to light if the midbrain is not disturbed. A patient that is severely depressed and recumbent may not respond to menacing gestures, even when visual pathways are intact. Ocular, optic tract, optic nerve, or optic chiasm lesions can interfere with vision and the pupillary light response. Brainstem contusion and cerebral edema may produce blindness and dilated unresponsive pupils owing to disturbance of the oculomotor area.

Examine all cranial nerves carefully. Cranial nerve abnormalities can indicate direct contusion or laceration of the neurons in the brainstem or where they exit the skull. Cranial nerves that are initially normal then later lose function indicate a progressively expanding lesion. When specific cranial nerve deficits are present, the prognosis is considered guarded.

Clinical signs such as rolling to one side, torticollis, head tilt, and abnormal nystagmus are usually associated with petrosal bone or cerebellomedullary lesions that produce vestibular neuron dysfunction. Fractures of the petrosal temporal bone often cause hemorrhage and cerebrospinal fluid (CSF) leak from the external ear canal. If the lesion is limited to the membranous labyrinth, the loss of balance will be toward the injured side and the quick phase of the nystagmus will be toward the injured side.

Normal physiologic nystagmus requires that the pathway be between the peripheral vestibular neurons and the pontomedullary vestibular nuclei to the nuclei of the cranial nerves that innervate the extraocular muscles (III, IV, VI). Severe brainstem lesions disrupt this pathway. Disruption of the pathway manifests as an inability to produce normal physiologic nystagmus by moving the patient’s head from side to side. In patients with severe CNS depression, this reflex may not be observed.

Next, assess postural changes and motor function abilities. A loss of the normal oculocerephalic (doll’s eye) reflex is an early sign of brainstem hemorrhage and a late sign of brainstem compression and herniation.

Any intracranial injury may be accompanied by a concurrent cervical spinal cord injury. Handle animals with such injuries with extreme care to avoid causing further damage. Whenever there is uncertainty whether a spinal cord lesion exists, strap the patient down to a flat surface and obtain radiographs of the spine. At least two orthogonal views may be required to see fractures; however, do not manipulate the patient until radiography has been completed. Cross-table views, in which the Bucky is turned perpendicular to the patient’s spine, with a radiograph plate secured behind the patient, may be required to minimize patient motion. In patients with cerebral lesions, hemiparesis usually resolves within 1 to 3 days.

Evaluation of cranial nerve function at frequent intervals may reveal an initial injury or a progressively expanding lesion in the brain. Signs of vestibular disorientation, marked head tilt, and abnormal nystagmus occur with contusions of the membranous labyrinth and fracture of the petrous temporal bone. Hemorrhage and CSF otorrhea may be visible from the external ear canal. Rolling movements indicate an injury to the cerebellar-medullary vestibular system.

Respiratory dysfunction and abnormal respiratory patterns are sometimes observed with severe head injury. Lesions of the diencephalon produce Cheyne-Stokes respirations, in which the patient takes progressively larger and larger breaths, pauses, then takes progressively smaller and smaller breaths. Mesencephalic lesions cause hyperventilation and can result in respiratory alkalosis. Medullary lesions result in a choppy, irregular respiratory pattern. Clinical signs of respiratory dysfunction in the absence of primary respiratory damage indicate a guarded prognosis.

After injury, seizures may be associated with intracranial hemorrhage, trauma, or an expanding intracranial mass lesion. Immediately begin medical therapy to control the seizure. Administer diazepam (0.5 mg/kg IV or 0.1 to 0.5 mg/kg/hr IV CRI) to treat seizures. If diazepam is not effective in combination with other treatments to control intracranial edema, consider giving pentobarbital (5 to 15 mg/kg IV to effect). Loading doses of
phenobarbital (16 to 20 mg/kg IV divided into four or five doses, given every 20 to 30 minutes) may be beneficial in preventing further seizures.

Severe refractory seizures or decreased mentation may be associated with cerebral edema and increased intracranial pressure. Mannitol, an osmotic diuretic, is effective at reducing cerebral edema (0.5 to 1.0 g/kg IV over 10 to 15 minutes). Mannitol also acts as a free radical scavenger that can inhibit the effects of cerebral ischemia-reperfusion injury. Mannitol works synergistically with furosemide (1 mg/kg IV given 20 minutes after the mannitol infusion). Corticosteroids have not been demonstrated to be beneficial in the treatment of head trauma and may induce hyperglycemia. Hyperglycemia has been shown to be a negative prognostic indicator in cases of head trauma. Also, glucocorticoids can suppress immune system function and impair wound healing. Because of the known risks and lack of known benefits of glucocorticosteroids, their use in treatment of head trauma is contraindicated.

The prognosis for any patient with severe head trauma is guarded. Management of head trauma patients may include intense nursing care for a period of weeks to months, depending on the presence and extent of concurrent injuries. If progressive loss of consciousness occurs, surgery for decompression of compressive skull injuries should be considered.

The most common injury associated with head trauma in small animals is a contusion with hemorrhage in the midbrain and pons. Subdural or extradural hemorrhage with space-occupying blood clots is uncommon. Diagnostic tests of head trauma may include skull radiographs, CT, and MRI of the brain. Special studies can help detect edema and hemorrhage in the brain and brainstem and aid in arriving at an accurate diagnosis and prognosis. A CSF tap is contraindicated in patients with head trauma because of the risk of causing a rapid decrease in intracranial pressure and brainstem herniation. If a compressive skull fracture is present, the patient should be stabilized for surgery to remove the compression. Surgery to alleviate increased intracranial pressure is rarely performed in veterinary medicine because of the poor prognosis and results. In some cases when a lesion can be localized to one area, 1- to 2-cm burr holes can be placed through the skull over the affected area of the cerebrum, exposing the underlying brain tissue. Blood clots can be removed through the holes. The bone flap may or may not be replaced, depending on the surgeon’s preference and the degree of brain swelling.

**Spinal Cord Injuries**

Spinal cord injuries may be associated with trauma, disk rupture, fractures, and dislocation of the spinal column. Proceed with caution when moving a patient with suspected spinal cord injury. Avoid flexion, extension, and torsion of the vertebral column. All animals that are unconscious after a traumatic event should be considered to have cervical or thoracolumbar spinal injury until proved otherwise by radiography, CT, or MRI. The animal should be moved onto a flat surface (e.g., board, door, window, picture frame) and taped down to prevent motion and further displacement of vertebrae. Sedation with analgesics or tranquilizers may be necessary to keep the animal immobile and to minimize patient motion. Whenever possible, avoid the use of narcotics in patients with head trauma because of the risk of increasing intracranial pressure. As in other emergencies, the ABCs should be evaluated, and the patient treated for shock, hemorrhage, and respiratory compromise. Once the cardiovascular and respiratory systems have been evaluated and stabilized, a more thorough neurologic examination can be performed.

**Thoracolumbar Disease: Herniated Disks and Trauma**

Protrusion of an intervertebral disk indicates that the disk is bulging into the vertebral canal as a result of dorsal shifting of the nuclear pulposus disk material. *Disk extrusion* refers to the rupture of the outer disk membrane and extrusion of the nuclear material into the vertebral column. In dogs and cats, there are 36 intervertebral disks that potentially can cause a problem. Chondrodystrophic breeds of dogs are predisposed to endochondral ossification and include the Dachshund, Shih Tzu, French Bulldog, Bassett Hound, Welsh Corgi, American Spaniel, Beagle, Lhasa Apso, and Pekingese.
Initial examination of the patient with suspected intervertebral disk disease includes identifying the neuroanatomic location of the lesion based on clinical signs and neurologic deficits and then establishing a prognosis. The neurologic examination should be carried out without excessive manipulation of the animal. The presence of pain, edema, hemorrhage, or a visible deformity may localize an area of vertebral injury. Once an area of suspected lesion is localized based on physical examination findings, take radiographs to establish a diagnosis and to institute therapy. In most cases, the animal must receive a short-acting anesthetic for proper radiographic technique and to prevent further injury. Lateral and cross-table ventrodorsal or dorsoventral radiographs require less manipulation of the animal compared with traditional ventrodorsal and dorsoventral projections. Myelography is often required to delineate the location of the herniated disk material.

Prognosis in spinal cord injury depends on the extent of the injury and the reversibility of the damage. Perception of noxious stimuli, or the presence of “deep pain,” by the animal when the stimulus is applied caudal to the level of the lesion is a good sign. To apply a noxious stimulus, apply firm pressure to a toe on one of the rear limbs using a thick hemostat or a pair of pliers. Flexion or withdrawal of the limb is simply a local spinal reflex and should not be perceived as a positive response to or patient perception of the noxious stimulus. Turning of the head, vocalization, dilation of the pupils, change in respiratory rate or character, or attempts to bite are behaviors that are more consistent with perception of the noxious stimulus. Absence of perception of the noxious stimulus (“loss of deep pain”) is a very poor prognosis for return to function.

Focal lesions are usually associated with vertebral fractures and displacement of the vertebral canal. Focal lesions in one or more of the spinal cord segments from T3 to T4 can cause complete dysfunction of the injured tissue as a result of concussion, contusion, or laceration. The degree of structural damage cannot be determined from the neurologic signs alone. Transverse focal lesions result in paraplegia, with intact pelvic limb spinal reflexes and analgesia of the limbs and body caudal to the lesion. Clinical signs in patients with spinal injury are summarized in Table 1-43.

**Treatment of Spinal Cord Injuries**

Carefully evaluate the cardiovascular and respiratory status of patients with spinal injuries. Immediately address specific injuries such as pneumothorax, pulmonary contusions, hypovolemic shock, and open wounds. If there is palpable or radiographic evidence of a vertebral lesion causing compressive injury, surgery is the treatment of choice unless the displacement has compromised most or all of the vertebral canal. Displacements through 50% to 100% of the vertebral canal are associated with a poor prognosis, particularly if deep pain is absent caudal to the lesion. In the absence of a

<table>
<thead>
<tr>
<th>Location of Lesion</th>
<th>Postural and Reflex Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cranial to C6</td>
<td>Spastic tetraplegia or tetraparesis Hyperreflexive, all four limbs Severe injury: possible death from respiratory failure</td>
</tr>
<tr>
<td>C6-T2</td>
<td>Tetraparesis or tetraplegia Depressed thoracic limb spinal reflexes (lower motor neuron) Hyperreflexive pelvic limbs (upper motor neuron)</td>
</tr>
<tr>
<td>T1-T3</td>
<td>Horner syndrome (prolated nictitans, enophthalmos, and miosis)</td>
</tr>
<tr>
<td>T3-L3</td>
<td>Schiff-Sherrington syndrome (extensor rigidity of thoracic limbs, flaccid paralysis with atonia, areflexia, and analgesia of pelvic limbs)</td>
</tr>
</tbody>
</table>
radiographic lesion and in the presence of continued neurologic deficits, an MRI or CT scan or myelography is warranted to localize a potentially correctable lesion. Surgical exploration can be considered: with the objectives of providing spinal cord decompression by hemilaminectomy or laminectomy with removal of disk material or blood clots, realign and stabilize the vertebral column, and perform a meningo(myelogram, if necessary. Place the patient on a backboard or other rigid surface, taped down for transport and sedated, to be transported to a surgical specialist. The presence of worsening or ascending clinical signs may signify ascending-descending myelomalacia and is characteristic of a very poor prognosis. In acute spinal trauma, the use of glucocorticoids has been the mainstay of therapy; however, controversy exists regarding whether they actually offer any benefit. Traditional glucocorticosteroid therapy is listed in Box 1-50. More recently, the use of propylene glycol has proved to be beneficial in the treatment of acute traumatic herniated disk. High-dose glucocorticoids should be used only for the first 48 hours after initial injury. Side effects of glucocorticosteroid therapy include gastric and intestinal ulceration. The prophylactic use of gastroprotectant drugs will not prevent gastrointestinal ulcer formation; however, if signs of gastrointestinal ulcer are present, institute gastroprotectant therapy.

**Management**

Management of the patient with spinal cord injury includes aggressive nursing care and physical therapy. Many patients with spinal cord injury have little to no control over bladder function, which results in chronic dribbling or retention of urine and overdistension of the urinary bladder with overflow incontinence. Urinary bladder retention can lead to urinary tract infection, bladder atony, and overflow incontinence. Manual expression of the bladder several times a day may be enough to keep the bladder empty. Alternatively, place a urinary catheter to maintain patient cleanliness and to keep the bladder decompressed. (See the discussion of urinary catheterization in Section 5.)

Paralytic ileus and fecal retention are frequent complications of spinal cord injury. To help prevent constipation, provide highly digestible foods and maintain the patient’s hydration with oral and intravenous fluids. Mild enemas or stool softeners can also be used to treat fecal retention. To prevent decubital ulcer formation, turn the patient every 4 to 6 hours, and use clean, dry, soft padded bedding. Apply deep muscle massage and passive range of motion exercises to prevent disuse atrophy of the muscles and dependent edema.

**Injuries to the Peripheral Nervous System**

The radial nerve innervates the extensor muscles of the elbow, carpus, and digits. The radial nerve also supplies sensory innervation to the distal craniolateral surface of the forearm and the dorsal surface of the forepaw. Injuries to the radial nerve at the level of the elbow result in an inability to extend the carpus and digits. As a result, the animal walks and bears weight on the dorsal surface of the paw. There is also loss of cutaneous sensation, which leads to paw injury. Injuries to the radial nerve above the elbow (in the shoulder area) result in an inability to extend the elbow and bear weight on the affected limb. It can take weeks before the full extent of the injury and any return to function are manifested. The animal may need to be placed in a carpal flexion sling or have eventual amputation if distal limb injury or self-mutilation occurs.

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**BOX 1-50 CORTICOSTEROID DOSAGE IN ACUTE SPINAL TRAUMA**

<table>
<thead>
<tr>
<th>Corticosteroid</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prednisolone sodium succinate or methylprednisolone</td>
<td>20 to 30 mg/kg intravenously (IV) once, then 10 to 15 mg/kg IV at 3, 6, and 9 hours</td>
</tr>
</tbody>
</table>


*Potentially useful for injuries less than 8 hours old.*
**Brachial Plexus**

**Sciatic Nerve**
The sciatic nerve primarily innervates the caudal thigh muscles that flex the stifle and extend the hip. The tibial branch of the sciatic nerve innervates the caudal leg muscles that extend the tarsus and flex the digits. The tibial nerve provides the sole cutaneous sensory innervation to the plantar aspect of the paw and digits. The peroneal branch of the sciatic nerve provides the sole sensory cutaneous innervation to the dorsal surface of the paw (Table 1-44). Sciatic nerve injury may occur with pelvic fractures, particularly those that involve the body of the ilium at the greater ischiatic notch, or with sacroiliac luxations that contuse the L6 and L7 spinal nerves that pass ventral to the sacrum to contribute to the sciatic nerve. With sciatic nerve injury, there is decreased stifle flexion and overflexion of the hock (tibial nerve), and the animal walks on the dorsal surface of the paw (peroneal nerve). Clinical signs of tibial or peroneal damage are seen with femur fractures or with inadvertent injection of drugs into the caudal thigh muscles.

**Femoral Nerve**
The femoral nerve innervates the extensor muscles of the stifle. The saphenous branch of the femoral nerve provides the sole cutaneous innervation to an area on the medial distal thigh, the leg, and the paw. The femoral nerve is protected by muscles and is rarely injured in pelvic fractures. Clinical signs of femoral nerve injury are inability to support weight on the pelvic limb, absence of a patellar reflex, and analgesia in the area of cutaneous innervation.

**Coma**
Coma is complete loss of consciousness, with no response to noxious stimuli. In some animals presented in a coma or stuporous state, the immediate cause will be apparent. In other cases, however, a careful and thorough diagnostic workup must be performed. A coma scale devised to assist in the clinical evaluation of the comatose patient is shown in Table 1-45. Whenever an animal is presented in a comatose state, immediately secure the airway by placing an endotracheal tube (see section on endotracheal intubation). If necessary, provide respiratory assistance or, at a minimum, supplemental oxygen. Control existing hemorrhage and treat shock, if present.

Take a careful and thorough history from the owner. Make careful note of any seizure, trauma, or toxin exposure, and whether prior episodes of coma have ever occurred. Perform a careful physical examination, taking note of the patient’s temperature, pulse, and respiration. An elevated temperature may suggest the presence of systemic infection, such as pneumonia or hepatitis, or a brain lesion with loss of hypothalamic thermoregulatory control. Very high temperatures associated with shock and coma are often observed in animals with

<table>
<thead>
<tr>
<th>Location of Injury</th>
<th>Clinical Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>C6-T2 nerve roots</td>
<td>Radial nerve paralysis</td>
</tr>
<tr>
<td>Musculocutaneous nerve</td>
<td>Inability to flex the elbow</td>
</tr>
<tr>
<td>Axillary or thoracodorsal</td>
<td>Dropped elbow nerve</td>
</tr>
<tr>
<td>Median and ulnar nerves</td>
<td>Loss of cutaneous sensation on the caudal surface of the forearm and palmar and lateral surfaces of the paw; inability to flex the carpus and digits</td>
</tr>
<tr>
<td>C8-T1 nerve roots</td>
<td>Radial, median, or ulnar nerve injury</td>
</tr>
<tr>
<td>C6-C7 nerve roots</td>
<td>Musculocutaneous, suprascapular, and axillary injury</td>
</tr>
<tr>
<td>C7-T3</td>
<td>Horner syndrome (miosis, enophthalmos, and prolapsed nictitans)</td>
</tr>
</tbody>
</table>
heat stroke (see section on heat stroke and heat-induced illness). Circulatory collapse or barbiturate overdose can produce coma and hypothermia.

Abnormal respiratory patterns also may be observed in a comatose patient. Hypoventilation may occur with elevated intracranial pressure or barbiturate overdose. Rapid respiratory rate may be associated with pneumonia, metabolic acidosis (DKA, uremia), or brainstem injury.

Examine the skin for any bruises or external trauma. Examine the mucous membranes and make note of color and capillary refill time. Icterus with petechiae or ecchymotic hemorrhage in a comatose patient may be associated with end-stage hepatic failure and HE. Smell the patient’s breath for the odor of ketones, which may signify DKA or end-stage hepatic failure.

Finally, conduct a complete neurologic evaluation. The presence of asymmetric neurologic signs may suggest an intracranial mass lesion (e.g., hemorrhage, neoplasia, injury). Usually, toxicities or metabolic disturbances (e.g., DKA, HE) cause symmetric clinical signs of neurologic dysfunction, with cerebral signs predominating. In HE, pupils are usually normal in size and responsive to light. With toxicities the pupils are abnormal in size and may be unresponsive to light.

Obtain a complete blood count and serum biochemistry profile and perform urinalysis and specific tests for glucosuria and ketonuria. Findings of a drastically elevated blood glucose with glucosuria, ketonuria, and high specific gravity are characteristic of DKA. Fever
and uremic encephalopathy are characterized by severe azotemia with a low urine specific gravity. If barbiturate intoxication is suspected, save urine for later toxin analysis. Evaluate urine sediment for calcium oxalate crystaluria, which may indicate ethylene glycol toxicity. Calculate plasma osmolality (see following section) to check for nonketotic hyperosmolar diabetes mellitus. Elevated blood ammonia levels may be associated with HE.

**Metabolic Causes of Altered Level of Consciousness**

**Diabetic Coma**

In uncontrolled diabetes mellitus, hyperosmolarity can result in clinical signs of disorientation, prostration, and coma. Plasma osmolality can be calculated from the following formula:

\[
\text{mOsm} / \text{L} = 2(\text{Na} + \text{K}) + (\text{glucose} / 18) + (\text{BUN} / 2.8)
\]

Clinical signs of hyperosmolarity can occur when the plasma osmolarity exceeds 340 mOsm/L. Treatment of DKA or nonketotic hyperosmolar syndrome is aimed at reducing ketoacid production, stimulating carbohydrate use, and impeding peripheral release of fatty acids. The treatment of choice is rehydration and provision of supplemental regular insulin and a carbohydrate source (see section on diabetic ketoacidosis). During ketosis, insulin resistance may be present. Slow rehydration with 0.9% saline solution or other balanced crystalloid fluids (e.g., Normosol-R, Plasma-Lyte A, lactated Ringer’s solution) should occur, with the goal of rehydration over 24 to 48 hours. Too-rapid rehydration can result in cerebral edema and exacerbation of clinical signs.

**Hepatic Coma**

HE is characterized by an abnormal mental state associated with severe hepatic insufficiency. The most common cause of HE is congenital or acquired portosystemic shunts. Acute hepatic destruction can also be caused by toxins, drugs, or infectious causes. HE is considered a medical emergency (Table 1-46). Absorption of ammonia and other nitrogenous substances from the gastrointestinal tract is thought to be one of the complicating factors in HE. Prevent absorption of ammonia and other nitrogenous substances from the gastrointestinal tract by restricting dietary protein to 15% to 20% for dogs and to 30% to 35% (on a dry matter basis) for cats. Dietary protein should be from a nonanimal plant source (e.g., soybean) whenever possible. Caloric requirements are met with lipids and carbohydrates.

<table>
<thead>
<tr>
<th>TABLE 1 - 46  Hepatic Encephalopathy</th>
<th>Clinical Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Grade of Hepatic Encephalopathy</strong></td>
<td><strong>Clinical Signs</strong></td>
</tr>
<tr>
<td>1</td>
<td>Listlessness, depression, mental dullness</td>
</tr>
<tr>
<td></td>
<td>Personality changes</td>
</tr>
<tr>
<td></td>
<td>Polyuria</td>
</tr>
<tr>
<td>2</td>
<td>Ataxia</td>
</tr>
<tr>
<td></td>
<td>Disorientation</td>
</tr>
<tr>
<td></td>
<td>Compulsive pacing or circling, head-pressing</td>
</tr>
<tr>
<td></td>
<td>Apparent blindness</td>
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<tr>
<td></td>
<td>Personality changes</td>
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<tr>
<td></td>
<td>Salivation</td>
</tr>
<tr>
<td></td>
<td>Polyuria</td>
</tr>
<tr>
<td>3</td>
<td>Stupor</td>
</tr>
<tr>
<td></td>
<td>Severe salivation</td>
</tr>
<tr>
<td></td>
<td>Seizures</td>
</tr>
<tr>
<td>4</td>
<td>Coma</td>
</tr>
</tbody>
</table>

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Also, prescribe cleansing enemas to rid the colon of residual material, and antibiotic therapy to reduce gastrointestinal tract bacteria. Neomycin (15 mg/kg q6h) can be administered as a retention enema. Metronidazole (7.5 mg/kg PO, q8-12h) or amoxicillin-clavulanate (16.25 mg PO q12h) can also be administered. Administer lactulose (2.5 to 5.0 mL q8h for cats; 2.5 to 15 mL q8h for dogs) to trap ammonia in the colon to prevent absorption (see Table 1-46). Administer lactulose PO to an alert animal or as a retention enema to a comatose animal. If lactulose is not available, Betadine retention enemas will change colonic pH and prevent ammonia absorption. A side effect of lactulose administration (PO) is soft to diarrheic stool.

**Emergency Treatment of Seizures**

A seizure is a transient disturbance of brain function that is sudden in onset, ceases spontaneously, and has a tendency to recur, depending on the cause. Most seizures are generalized and result in a loss of consciousness and severe involuntary contraction of the skeletal muscles, resulting in tonic-clonic limb activity and opisthotonus. Mastication, salivation, urination, and defecation are common. Partial (petit mal) seizures range from limited limb activity, facial muscle twitching, and periodic behavioral abnormalities to brief loss of consciousness. Similar clinical signs also can occur with syncopal episodes. Conduct a careful cardiac examination in any patient with a history of petit mal seizures. Seizures of any form constitute a medical emergency, particularly when they occur in clusters, or as status epilepticus.

Most seizures are of short duration and may have subsided by the time the animal is presented for treatment. Whenever a seizure occurs, however, it is important that the animal not inadvertently injure itself or a bystander. It is important to evaluate whether the patient has a coexisting disease that can predispose it to seizures, such as hepatic failure, uremia, diabetes mellitus, hypoglycemia, toxin exposure, insulin-secreting tumors, and thiamine deficiency. Many toxins are responsible for clinical signs of tremors or seizures (see section on poisons and toxins). Treatment of a primary disease entity can help control seizures in some cases, provided that the underlying cause is investigated and treated.

Status epilepticus, a state of continuous uncontrolled seizure activity, is a medical emergency. When an animal is in a state of status epilepticus, immediately place a lateral or medial saphenous intravenous catheter and administer diazepam (0.5 mg/kg IV) to help control the seizure. In most cases, the seizure must be controlled before a diagnostic workup is attempted. Whenever possible, however, blood samples should be collected before administration of any anticonvulsant agent because of the risk of incorrect test results. For example, the propylene glycol carrier in diazepam can cause a false-positive ethylene glycol test result when using an in-house testing kit.

Whenever possible, check blood glucose levels, particularly in young puppies or kittens, to evaluate and treat hypoglycemia as a cause of seizures. If hypoglycemia exists, administer 25% dextrose (1 g/kg IV). If diazepam partially controls the status epilepticus, administer a CRI (0.1 mg/kg/hr in 5% dextrose in water). Diazepam is sensitive to light, and the bag and infusion line must be covered to prevent degradation of the drug. If diazepam fails to control status epilepticus, give pentobarbital (5 to 15 mg/kg IV to effect) or propofol (3 to 7 mg/kg IV, then 0.4 mg/kg/min IV CRI to effect). The animal’s airway should be intubated and protected while the patient is kept in the drug-induced coma. Protracted cases of seizures may require mannitol and furosemide therapy to treat cerebral edema.

Administer intravenous fluids (balanced crystalloid at maintenance doses [see section on intravenous fluid therapy]). The patient should be turned every 4 to 6 hours to prevent atelectasis. Insert a urinary catheter for cleanliness, and place the animal on soft dry padded bedding to prevent decubital ulcer formation. Depending on the length of time that the patient is rendered unconscious, apply passive range of motion exercises and deep muscle massage to prevent disuse atrophy of the muscles and dependent or disuse edema. Monitor the patient’s oxygenation and ventilation status by arterial blood gas measurement or pulse oximetry and capnometry (see the discussion of blood gas
monitoring, pulse oximetry, and capnometry in Section 5). Administer supplemental oxygen to any patient that is hypoxemic secondary to hypoventilation or other causes. Severe refractory seizures can result in the development of neurogenic pulmonary edema. Lubricate the animal's eyes every 4 hours to prevent drying out and corneal abrasions. Depending on the cause of the seizure, administer phenobarbital (at a loading dose of 16 to 20 mg/kg IV given in four to five injections, every 20 to 30 minutes; make sure that the patient is rousable between injections) or levetiracetam (Keppra, 20 mg/kg PO Q8 hours or 20 mg/kg IV slow bolus).

Seizures in cats often are associated with structural brain disease. The occurrence of partial focal seizures is unequivocally associated with a focal cerebral lesion and acquired structural brain disease. An initial high frequency of seizures is also a strong indication that structural brain disease is present. Seizure activity in cats may occur as mild generalized seizures or complex partial seizures and may be associated with systemic disorders such as FIP virus, toxoplasmosis, Cryptococcus infection, lymphosarcoma, meningiomas, ischemic encephalopathy, and thiamine deficiency.

Thiamine deficiency in the cat can be a medical emergency characterized by dilated pupils, ataxic gait, cerebellar tremor, abnormal oculocephalic reflex, and seizures. Treatment consists of administration of thiamine (50 mg/day) for 3 days.

Additional Reading

**OCULAR EMERGENCIES**

An ocular emergency is any serious condition that causes or threatens to cause severe pain, deformity, or loss of vision. Treat ocular emergencies immediately, within 1 to several hours after the emergency, whenever possible ([Boxes 1-51 and 1-52](#)).

To assess the location and degree of ocular injury, perform a complete ocular examination. In some cases, short-acting sedation or general anesthesia in conjunction with topical local anesthetic may be necessary to perform the examination, because of patient discomfort and blepharospasm. The equipment listed in **Box 1-53** may be necessary and may be invaluable in making an accurate diagnosis.

To perform a systematic and thorough ocular examination, first obtain a history from the owner. Has there been any prior incident of ocular disease? Is there any history of trauma or known chemical irritant or exposure? Did the owner attempt any irrigation or medical techniques before presentation? When was the problem first noticed? Has it changed at all since the owner noticed the problem?

After a history has been obtained, examine the patient’s eyes for discharge, blepharospasm, or photophobia. If any discharge is present, note its color and consistency. Do not attempt to force the eyelids open if the patient is in extreme discomfort. Administer a short-acting sedative and topical local anesthetic such as 0.5% proparacaine. Note the posi-

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**BOX 1-51 OCULAR EMERGENCIES REQUIRING IMMEDIATE THERAPY**

- Penetrating injury to the globe
- Proptosis of the globe
- Glaucoma
- Corneal laceration
- Acute corneal abrasion or ulcer
- Acute iritis
- Lid laceration
- Descemetocoele
- Orbital cellulitis
- Chemical burns
- Ocular foreign bodies
- Hyphema

**BOX 1-52 OCULAR EMERGENCIES THAT CAN CAUSE A SUDDEN LOSS OF VISION**

- Hyphema
- Traumatic lid swelling
- Exposure keratitis
- Sudden acquired retinal degeneration
- Retinal hemorrhage
- Retinal detachment
- Intracranial damage
- Vitreous hemorrhage
- Corneal edema
- Acute glaucoma
- Retinal detachment
- Retinal edema
- Traumatic avulsion of the optic nerve
- Proptosis of the globe

**BOX 1-53 EQUIPMENT NEEDED TO PERFORM AN OCULAR EXAMINATION**

- Loupe
- Direct ophthalmoscope
- Fine-tooth forceps
- Lacrimal probe
- Fluorescein sterile strips
- Proparacaine (0.5%)
- Short-acting mydriatics (tropicamide 1%)
- Monocular indirect ophthalmoscope
- Transilluminator
- Lid retractor
- Sterile saline eye wash in irrigation bottle
- Sterile cotton-tipped swabs
- Schiotz tonometer or Tono-Pen
tion of the globe within its orbit. If the eye is exophthalmic, strabismus and protrusion of the third eyelid are often visible. Exposure keratitis may be present. In cases of retrobulbar or zygomatic salivary gland inflammation, the patient will resist opening the mouth and exhibit signs of discomfort or pain. Note any swelling, contusions, abrasions, or lacerations of the eyelids. Note whether the lids are able to close completely and cover the cornea. If a laceration of the lid is present, determine the depth of the laceration. Palpate the orbit for fractures, swelling, pain, crepitus, and cellulitis.

Examine the cornea and sclera for penetrating injury or foreign material. The use of lid retractors or small forceps can be very helpful in these cases. If a wound appears to penetrate completely into the globe, look for loss of uveal tissue, lens, or vitreous. Do not put any pressure on the globe, because intraocular herniation may result. Examine the conjunctiva for hemorrhage, chemosis, lacerations, and foreign bodies. Examine the superior and inferior conjunctival cul-de-sacs for foreign material. In such cases, placement of a topical anesthetic and use of a moistened cotton swab are invaluable to sweep the conjunctival fornix to pick up foreign bodies. Use small, fine-tipped forceps to retract the third eyelid away from the globe and examine behind the third eyelid for foreign bodies.

Next, examine the cornea for opacities, ulcers, foreign bodies, abrasions, or lacerations. Place a small amount of fluorescein stain mixed with sterile water or saline on the dorsal sclera. Close the eye to disperse the stain over the surface of the cornea, then flush gently with sterile saline irrigation. Examine the cornea again for any defects. A linear defect perpendicular to the long axis of the eye should alert the clinician to investigate the conjunctiva for dystechia.

Record the pupil size, shape, and response to light (both direct and consensual). Examine the anterior chamber and note its depth and whether hyphema or aqueous flare is present. Is the lens clear and is it in the normal position? Lens luxation can cause the lens tissue to touch the cornea and cause acute corneal edema. Measure intraocular pressure with a Schiotz tonometer or Tono-Pen. Finally, dilate the pupil and examine the posterior chamber using a direct or indirect ophthalmoscope to look for intraocular hemorrhage, retinal hemorrhage, retinal detachment, tortuous retinal vessels, optic neuritis, and inflammation.

**Specific Conditions and Treatment**

The basic surgical instruments listed in Box 1-54 may be useful in the treatment of ocular lacerations and other ophthalmic injuries.

**Injuries of the Eyelids**

**Lid Laceration**

Bite wounds and automobile trauma commonly cause lacerations and abrasions of the lid margins. The lids can be considered to be two-layer structures, with the anterior composed of the skin and orbicularis muscle and the posterior layer composed of the tarsus and conjunctiva. The openings of the meibomian glands in the lid margin form the approximate line separating the lids into anterior and posterior segments. Splitting the lid into these two segments facilitates the use of sliding skin flaps to close wound defects, if necessary.

**Box 1-54   Basic Instruments for Treatment of Ocular Emergencies**

- Castroviejo or Barraquer lid speculum
- Bishop-Harmon tissue forceps
- Stevens tenotomy scissors
- Castroviejo corneal scissors
- Castroviejo needle holder; standard jaws with lock
- Beaver knife handle and No. 64 blades
- Lacrimal cannula, straight 22 gauge
- Barraquer iris repository
- Foreign body spud
- Enucleation scissors, medium curve
- Suture material: 6-0 silk, 4-0 nylon, 7-0 collagen, 6-0 ophthalmic gut, 7-0 nylon

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Clean and thoroughly but gently irrigate the wound with sterile saline solution before attempting any lid laceration repair. Use sterile saline solution to irrigate the wound and conjunctiva. A 1% povidone-iodine scrub can be used on the skin, taking care to avoid getting any scrub material in the soft tissues of the eye. Drape the eye with an adhesive ocular drape, if possible, to prevent further wound contamination.

Trim the ragged wound edges, but be very conservative with tissue debridement. Leave as much tissue as possible to ensure proper wound contracture with minimal lid deformity. Close a small lid wound with a figure-of-eight or two-layered simple interrupted suture of absorbable suture material or nylon in the skin. The lid margins must be absolutely apposed to prevent postoperative lid notching.

Ecchymosis of the Lids
Direct blunt trauma to the eye can cause severe ecchymosis because of the excellent vascular supply of the eyelids. Other associated ocular injuries such as orbital hemorrhage, proptosis, and corneal laceration may also occur. Trauma, allergic reactions, inflammation of the sebaceous glands (hordeolum), thrombocytopenia, and vitamin K antagonist rodenticide intoxication can all cause ecchymoses of the lids.

Treat eyelid ecchymoses initially with cool compresses, followed by warm compresses. Resorption of blood can occur 3 to 10 days after the initial insult. Ocular allergies respond well to topical application (dexamethasone ophthalmic ointment q6-8h) and systemic administration of glucocorticosteroids, along with cool compresses.

Conjunctival Lacerations
In order to fully assess the conjunctiva for abnormalities, it may be necessary to carefully dissect it away from the underlying sclera. When performing this dissection, do not place undue pressure on the globe because of the risk of herniation of the intraocular contents through a scleral wound.

Repair large conjunctival lacerations with 6-0 absorbable sutures, using an interrupted or continuous pattern. Carefully approximate the margins of the conjunctiva to prevent formation of inclusion cysts. When large areas of the conjunctiva have been damaged, advancement flaps may be required to close the defect.

Subconjunctival Hemorrhage
Subconjunctival hemorrhage is a common sequela of head trauma, and it may also be observed in various coagulopathies. By itself it is not a serious problem, but it may signify severe underlying intraocular damage. A complete ocular examination is indicated. Other causes of subconjunctival hemorrhage include thrombocytopenia, autoimmune hemolytic anemia, hemophilia, leptospirosis, vitamin K antagonist rodenticide intoxication, severe systemic infection or inflammation, and prolonged labor (dystocia). Uncomplicated subconjunctival hemorrhage usually clears on its own within 14 days. If the conjunctiva is exposed because of swelling and hemorrhage, administer a topical protective triple antibiotic ophthalmic ointment every 6 to 8 hours until the conjunctival hemorrhage resolves.

Chemical Injuries
Toxic, acid, and alkaline chemical injuries to the eye can sometimes occur. The severity of the injury caused by ocular burns depends on the concentration, type, and pH of the chemical and on the duration of exposure. Weak acids do not penetrate biologic tissue very well. The hydrogen ion precipitates the protein on contact and therefore provides some protection to the corneal stroma and intraocular contents. Precipitation of corneal proteins produces a ground-glass appearance in the cornea.

Alkaline solutions and very strong acids penetrate tissues rapidly, causing saponification of the plasma membrane, denaturation of collagen, and vascular thrombosis within the conjunctiva, episclera, and anterior uvea.

Severe pain, blepharospasm, and photophobia are produced by exposure of free nerve endings in the corneal epithelium and conjunctiva. Severe alkaline burns cause an increase
in intraocular pressure. Intraocular prostaglandins are released, and the intraocular aqueous pH increases, producing changes in the blood-aqueous barrier and secondary uveitis. Uveitis with anterior synechia formation, eventual chronic glaucoma, phthisis, secondary cataract, and corneal perforation can occur.

Healing of the corneal epithelium is usually accomplished by neovascularization and sliding and increased mitosis of the corneal epithelium. Severe stromal burns within the cornea heal by degradation and removal of necrotic debris, followed by replacement of the collagen matrix and corneal epithelial cells. The release of collagenase, endopeptidase, and cathepsins from polymorphonuclear cells serves to cause further corneal breakdown. In severe cases, only PMNs may be present, and fibroblasts may never invade the corneal stroma.

All chemical burns should be washed copiously with any clean aqueous solution available. If any sticky paste or powder is adherent to the conjunctival sac, remove it with moist cotton swabs and irrigation. Begin mydriasis and cycloplegia by topical application of 1% atropine ophthalmic drops or ointment. Start antibiotic therapy with triple antibiotic ophthalmic ointment or Gentocin ointment every 6 to 8 hours. Treat secondary glaucomas with topical carbonic anhydrase inhibitors. To avoid fibrinous adhesions and symblepharon formation, keep the conjunctival cul-de-sacs free of proteinaceous exudate, which can form adhesions. Analgesics are required for pain. Oral nonsteroidal antiinflammatory agents such as carprofen, ketoprofen, meloxicam, or aspirin are recommended.

Persistent epithelial erosions may require a conjunctival flap left in place for 3 to 4 weeks or placement of a topical collagen shield (contact lens). Topical antibiotics, mydriatics, and lubricants (Lacri-Lube or Puralube ointment) should also be used.

Strong acid or alkali burns can result in severe corneal stromal loss. In the past, topical N-acetylcysteine (10% Mucomyst) has been recommended. This treatment is very painful. Other treatments are also available, such as ethylenediaminetetraacetic acid (EDTA) (0.2 M solution) and patient serum to inhibit mammalian collagenase activity. To prepare patient serum, obtain 10 to 12 mL of whole blood from the patient. Spin it down in a serum separator tube after a clot forms and then place the serum in a red-topped tube on the patient's cage. (The contents of the tube are viable for 4 days without refrigeration.) Apply the serum topically to the affected eye every 1 to 2 hours. Avoid using topical steroids because they inhibit fibroblast formation and corneal healing. In severe cases, if conjunctival swelling and chemosis also are present, antiinflammatory doses of oral steroids can be administered in the short term. Oral steroids and NSAIDs should never be administered to the patient concurrently, because of the risk of gastrointestinal ulcer and perforation.

**Corneal Abrasions**

Corneal abrasions are associated with severe pain, blepharospasm, lacrimation, and photophobia. Animals with such intense pain are often difficult to examine until analgesia has been administered. Topical use of proparacaine (0.5% proparacaine hydrochloride) is usually sufficient to permit relaxation of the eyelids so that the eye can be examined. Using a focal source of illumination and an eye loupe, examine the cornea, inferior and superior conjunctival fornices, and medial aspect of the nictitans for foreign bodies. Place a sterile drop of saline on a fluorescein-impregnated strip, and touch the superior conjunctiva once to allow the stain to spread onto the surface of the eye. Irrigate the eye to remove excess stain and then examine the corneal surface for any areas of stain uptake. If an area of the cornea persistently remains green, there is damage to the corneal epithelium in that area.

Initial treatment consists of application of a topical mydriatic (1 drop of 1% atropine in affected eye q12h) to prevent anterior synechiae and improve cycloplegia. Triple antibiotic ointment is the treatment of choice (a ¼-inch strip in the affected eye q8h) until the ulcer heals. In some cases, nonhealing ulcers (e.g., Boxer ulcer, indolent ulcer) form in which the epithelial growth does not adhere to the underlying cornea. Gently debride the loose edges of the ulcer or erosion with a cotton swab and topical anesthesia. More severe cases in which only minimal healing has occurred after 7 days of treatment require grid keratectomy, in
which a 25-gauge needle is used to gently scratch the surface of the abrasion or ulcer in the form of a grid to promote neovascularization. Apply a topical anesthetic before performing the procedure. A collagen contact lens also may be required to promote wound healing. All corneal abrasions should be reevaluated in 48 hours, and then every 4 to 7 days thereafter until they have healed.

**Acute Infectious Keratitis**

Acute infectious keratitis secondary to bacterial infection is characterized by mucopurulent ocular discharge, rapidly progressing epithelial and corneal stromal loss, inflammatory cellular infiltrates into the corneal stroma, and secondary uveitis, often with hypopyon formation. Confirmation of infectious keratitis is based on corneal scrapings and a positive Gram stain. Initial treatment for bacterial keratitis consists of systemic antibiotics and topical ciprofloxacin (0.3% eye drops or ointment).

**Penetrating Corneal Injury**

Penetrating injuries through the cornea may result in prolapse of intraocular contents. Frequently, pieces of uveal tissue or fibrin effectively but temporarily seal the defect and permit the anterior chamber to re-form. Avoid manipulation of these wounds until the animal has been anesthetized, as struggling or excitement can promote loss or dislodgement of the temporary seal and cause the intraocular contents to be extruded.

Superficial corneal lacerations need not be sutured and can be treated the same as a superficial corneal ulcer or abrasion. If the laceration penetrates more than 50% of the thickness of the cornea or extends more than 3 to 4 mm, it should be sutured. When placing sutures in the cornea, it is helpful to use magnification. Referral to a veterinary ophthalmologist is advised. If a veterinary ophthalmologist is not available, use 7-0 or 8-0 silk, collagen, or nylon sutures on a micropoint spatula-type needle. Use a simple interrupted suture pattern and leave the sutures in place for a minimum of 3 weeks. Because many corneal lacerations are jagged and corneal edema forms, most of the wound edges cannot be tightly juxtaposed. In such cases, pull a conjunctival flap across the wound to prevent leakage of aqueous fluid. Never suture through the full thickness of the cornea; rather, the suture should pass through the middle third of the cornea.

After closure of the corneal wound, the anterior chamber must be re-formed to prevent anterior synechia formation with secondary glaucoma. Taking care to avoid iris injury, use a 25- or 26-gauge needle to insert sterile saline at the limbus. Any defect in the suture line will be apparent because of leakage of the fluid from the site and should be repaired.

Incarceration of uveal tissue in corneal wounds is a difficult surgical problem. Persistent incarceration of uveal tissue can result in development of a chronic wick in the cornea, a shallow anterior chamber, chronic irritation, edema, vascularization of the cornea, and intraocular infection that can lead to panophthalmitis. Referral to a veterinary ophthalmologist is strongly recommended.

**Ocular Foreign Body**

The most common foreign bodies associated with ocular injuries in small animals are birdshot, BB pellets, and glass. The site of intraocular penetration of the foreign bodies may be obscured by the eyelids. A foreign body entering the eye may penetrate the cornea and fall into the anterior chamber or become lodged in the iris. Foreign bodies may occasionally penetrate the lens capsule, producing cataracts. Some metallic high-speed foreign bodies may penetrate the cornea, iris, and lens to lodge in the posterior wall of the eye or vitreous chamber.

Direct visualization of a foreign body is the best means of localization. Examination of the eye with an indirect ophthalmoscope or biomicroscope (if available) is invaluable for locating foreign bodies. Indirect visualization of the ocular foreign body can also be achieved through radiographic techniques. Three separate views should be obtained to determine
the plane of location of the foreign object. CT or MRI may prove useful, although scatter from the foreign body may make it difficult to directly visualize with these techniques. Ocular ultrasound is perhaps the most useful and refined radiographic technique for locating intraocular foreign bodies.

Before any foreign body is removed from the eye, the risks and surgical danger of removing it must be weighed against the risks of leaving it in place. Metallic foreign bodies in the anterior chamber are much easier to remove than nonmagnetic ones. Attempted removal of foreign objects from the vitreous chamber of the eye has consistently produced poor results. For the best chance of recovery, ocular foreign bodies should be removed by a veterinary ophthalmologist whenever possible.

Ocular Trauma

Blunt trauma to the globe can result in luxation or subluxation of the lens. The subluxated lens may move anteriorly and make the anterior chamber more shallow. Trembling of the iris (iridodonesis) may be noticed when the lens is subluxated. In complete luxation, the lens may fall totally into the anterior chamber and obstruct aqueous outflow, causing secondary glaucoma. Alternatively, the lens may be lost into the vitreous cavity. Luxation of the lens is almost always associated with rupture of the hyaloid membrane and herniation of the vitreous through the pupillary space.

Emergency surgery for lens luxation is required if the lens is entirely within the anterior chamber or incarcerated within the pupil, causing a secondary pupillary block glaucoma. Acute elevation in intraocular pressure can cause vision loss within 48 hours; thus, lens removal should be accomplished as quickly as possible. Referral to a veterinary ophthalmologist is recommended.

Severe trauma to the globe or a direct blow to the head can result in retinal or vitreous hemorrhage. There may be large areas of subretinal or intraretinal hemorrhage. Subretinal hemorrhage assumes a discrete globular form, and the blood appears reddish-blue in color. The retina is detached at the site of hemorrhage. Superficial retinal hemorrhage may assume a flame-shaped appearance, and preretinal or vitreous hemorrhage assumes a bright-red amorphous appearance, obliterating the underlying retinal architecture. Retinal and vitreous hemorrhage secondary to trauma usually resorbs spontaneously over a 2- to 3-week period. Unfortunately, vitreous hemorrhage, as it organizes, can produce vitreous traction bands that eventually produce retinal detachment.

Expulsive choroid hemorrhage can occur at the time of injury and usually leads to retinal detachment, severe visual impairment, and total loss of vision. Treatment of vitreal and retinal hemorrhage includes rest and correction of factors that may predispose to intraocular hemorrhage. More complicated cases may require vitrectomy performed by a veterinary ophthalmologist.

Hyphema

Hyphema refers to blood in the anterior chamber of the eye. The most common traumatic cause of hyphema is an automobile accident. Hyphema may also occur because of penetrating ocular wounds and coagulopathies. Blood within the eye may come from the anterior or posterior uveal tract. Trauma to the eye may result in iridodialysis or a tearing of the iris at its root, permitting excessive bleeding from the iris and ciliary body. Usually, simple hyphema resolves spontaneously in 7 to 10 days and does not cause vision loss. Loss of vision that follows bleeding into the anterior chamber is associated with secondary ocular injuries such as glaucoma, traumatic iritis, cataract, retinal detachment, endophthalmitis, and corneal scarring.

Treatment of hyphema must be individualized, but there are severe general principles of treatment. First, stop ongoing hemorrhage and prevent further bleeding whenever possible. This may involve correction of the underlying cause, if a coagulopathy is present. Next, aid in the elimination of blood from the anterior chamber, control secondary glaucoma, and treat associated injuries, including traumatic iritis. Finally, detect and treat any late complications of glaucoma.
In most cases of traumatic hyphema, little can be done to arrest or prevent ongoing hemorrhage. It is best to restrict the animal’s activity and prohibit exertion. Rebleeding can occur within 5 days, and intraocular pressure must be monitored closely. After 5 to 7 days, the blood in the anterior chamber will change color from a bright red to bluish-black (“eight-ball hemorrhage”). If total hyphema persists and intraocular pressure rises despite therapy, surgical intervention by a veterinary ophthalmologist may be necessary.

The primary route of escape of RBCs from the anterior chamber is via the anterior drainage angle. Iris absorption and phagocytosis play a minor role in the removal of blood from the anterior chamber. Because of the associated traumatic iritis in hyphema, topical administration of a glucocorticoid (1% dexamethasone drops or 1% prednisolone drops) is advised to control anterior chamber inflammation. A cycloplegic agent (1% atropine) should also be used.

The formation of fibrin in the anterior chamber of the eye secondary to hemorrhage can produce adhesions of the iris and secondary glaucoma (see section on glaucoma secondary to hyphema) by blocking the trabecular network. Hyphema secondary to retinal detachment (Collie ectasia syndrome) and end-stage glaucoma are extremely difficult to treat medically and have a poor prognosis.

**Proptosis**

Proptosis of the globe is common secondary to trauma, particularly in brachycephalic breeds. Proptosis of the globe in dolichocephalic breeds requires a greater degree of initiating contusion than the brachycephalic breeds because the orbits are so much deeper. Therefore, secondary damage to the eye and CNS associated with proptosis of the globe may be greater in the Collie or Greyhound than in the Pug.

When proptosis occurs, carefully evaluate the cardiovascular system for evidence of hypovolemic or hemorrhagic shock. Examine the respiratory and neurologic systems. Be sure to establish an airway and treat shock, if present. Control hemorrhage and stabilize the cardiovascular system before attempting to replace the globe within its orbit or perform enucleation. During the initial management of the cardiovascular and respiratory systems, the eye should be covered with an ophthalmic grade ointment or sponges soaked in sterile saline to prevent the globe from drying out. Proptosis of the globe can be associated with serious intraocular problems including iritis, chorioretinitis, retinal detachment, lens luxation, and avulsion of the optic nerve.

Stain the surface of the eye with fluorescein to look for topical abrasions or ulcers. Carefully examine the sclera, cornea, and conjunctiva for penetrating injuries that may allow aqueous leakage. Evaluate the size, location, and response to light of the pupil. A reactive pupil is better than a mydriatic fixed pupil. Topical administration of a mydriatic (atropine 1%) to prevent persistent miosis and synechia formation is indicated, along with topical and oral antibiotics and oral analgesic therapy.

Reposition the proptosed globe with the patient under general anesthesia. Make a lateral canthotomy incision to widen the palpebral fissure. Lavage the globe with sterile saline irrigation to remove any external debris. Place a copious amount of triple antibiotic ophthalmic ointment on the surface of the eye and then gently press the globe into the orbit using the flat side of a scalpel handle or a moistened sterile surgical sponge. Do not probe the retroorbital space with a needle or attempt to reduce intraocular pressure by paracentesis. When the globe is replaced in the orbit, close the lateral canthotomy incision with simple interrupted sutures. Place three nonpenetrating mattress sutures in the lid margins but do not draw them together. Tighten the lid sutures through small pieces of a red rubber catheter or length of intravenous extension tubing to prevent the sutures from causing lid necrosis. Leave the medial canthus of the eye open in order to allow topical treatment.

Postoperative treatment is directed at preventing further iritis and preventing infection. Administer systemic broad-spectrum antibiotics (Clavamox, 16.25 mg/kg PO bid) and analgesic drugs. Apply topical triple antibiotic ophthalmic ointment (¼ inch in affected eye q6-8h) and atropine (1% in affected eye q12h) to prevent infection, cycloplegia, and
anterior synechiae. Antiinflammatory doses of systemic steroids can also be added to the treatment if severe peri orbital inflammation is present. Systemic steroids should never be used in conjunction with NSAIDs, because of the risk of gastrointestinal ulceration and perforation.

The sutures should remain in place for a minimum of 3 weeks. After this time, remove the sutures and inspect the globe. If proptosis recurs, repeat the treatment.

After proptosis, strabismus is common secondary to peri orbital muscle injury. Even after extensive treatment, vision in the eye may still be lost. Nonvisual eyes can remain in place, but phthisis may develop.

**Glaucoma Secondary to Hyphema**

Carbonic anhydrase inhibitors such as acetazolamide and dichlorphenamide decrease aqueous secretion and may effectively reduce intraocular pressure if the trabecular outflow is still functioning at 40% of its capacity. An eye with a poorly functional trabecular outflow system will respond poorly to therapy with carbonic anhydrase inhibitors. Osmotic agents such as mannitol or glycerol may be helpful in controlling glaucoma secondary to hyphema. Reduction in vitreous chamber size can make the anterior chamber deeper and may allow increased aqueous outflow. Evacuation of blood or blood clots from the anterior chamber is not advisable unless the glaucoma cannot be controlled medically or there is no indication after a prolonged period of time that blood is being resorbed.

Tissue plasminogen activator (t-PA) has proved to be useful in lysing blood clots and preventing excessive fibrin formation. The t-PA is reconstituted to make a solution of 250 mcg/mL, which is then frozen at −70° C in 0.5-mL aliquots. The thawed, warmed reconstituted t-PA is injected into the anterior chamber.

Blind probing of the anterior chamber of the eye and surgical intervention in an attempt to remove blood clots can cause serious complications such as rebleeding, lens luxation, iris damage, and damage to the corneal epithelium and therefore is not advised.

**Acute Glaucoma**

Acute glaucoma is a rise in intraocular pressure that is not compatible with normal vision. Glaucoma may manifest as early acute congestive or noncongestive glaucoma or as end-stage disease. Cardinal signs of glaucoma are a sudden onset of pain, photophobia, lacrimation, deep episcleral vascular engorgement, edematous insensitive cornea, shallow anterior chamber depth, dilated unresponsive pupil, loss of visual acuity, and buphthalmia. Intraocular pressure usually exceeds 40 mm Hg but may be normal or only slightly increased if glaucoma is secondary to anterior uveitis.

Most forms of clinical glaucoma in dogs are secondary to some other intraocular problem. Primary glaucoma is recognized in some breeds, including the Bassett Hound, Cocker Spaniel, Samoyed, Bouvier des Flandres, and some Terrier breeds, either from goniodygenesis or a predisposition to lens luxation. Other common causes of acute glaucoma are anterior uveitis and intumescent lens secondary to rapid cataract development, particularly in dogs with diabetes mellitus.

Treatment involves investigation of the underlying cause of the sudden rise in intraocular pressure and rapid reduction in intraocular pressure. Permanent visual impairment is often associated with chronically buphthalmic globes or the presence of rippling or striae formation on the cornea. Referral to a veterinary ophthalmologist is recommended.

If the eye is still visual and not buphthalmic, the prognosis is favorable, depending on the cause of the acute glaucoma. Treatment to reduce intraocular pressure consists of improving aqueous outflow, reducing intraocular volume with osmotic agents, and reducing aqueous formation (Table 1-47).

The use of topical mydriatic agents in acute glaucoma is contraindicated because of the risk of making lens luxation or anterior uveitis worse. Referral to a veterinary ophthalmologist for emergency surgery is indicated in cases of iris bombe, intumescent lens, or lens subluxation.
Administer osmotic agents to reduce the size of the vitreous body and the amount of aqueous. Osmotic agents create an osmotic gradient between the intraocular fluids and the vascular bed, thus allowing osmotic removal of fluid independent of the aqueous inflow and outflow systems. If no other treatments are available, oral glycerol (50%, 1-2 ml/kg or 1-2 g/kg PO) can be used to effectively reduce intraocular pressure. An adverse side effect of oral glycerol treatment is protracted vomiting. Do not use glycerol in a diabetic patient. Mannitol (0.5-1 g/kg IV over 10-20 minutes) also effectively reduces intraocular pressure but does not cause vomiting.

Carbonic anhydrase inhibitors can be used to reduce intraocular volume by reducing aqueous production. Oral administration of dichlorphenamide, methazolamide, and acetazolamide (2 to 4 mg/kg) is usually not very effective alone in reducing aqueous volume and intraocular pressure and also can cause metabolic acidosis. Topical carbonic anhydrase inhibitors appear to be more effective (dorzolamide, Trusopt) when used in conjunction with topical β-blockers (timolol, 0.5% solution only BID). The most effective treatment for acute pressure reduction is use of a topical prostaglandin inhibitor (latanoprost). Usually just one or two drops effectively reduces intraocular pressure in the emergency stages, until the patient can be referred to a veterinary ophthalmologist the following day.

Additional Reading
ONCOLOGIC EMERGENCIES

Many clinical conditions that are presented as emergencies may be caused in part or wholly by the presence of a neoplasm. Paraneoplastic signs are summarized in Table 1-48. Prompt identification of the neoplasia combined with knowledge of treatment, expected response to therapy, and long-term prognosis can aid owners and practitioners in making appropriate treatment decisions.

HEMORRHAGE OR EFFUSION

Hemorrhage or effusion can occur in any body cavity as a result of the presence of benign or malignant tumors. Tumors secrete anticoagulants to allow angiogenesis to grow unchecked. Hemorrhage often occurs as a result of rupture of a neoplasm or invasion of a neoplasm into a major vascular structure. Effusion may be the result of direct fluid production by the mass or may be caused by obstruction of lymphatic or venous flow.

Hemorrhagic effusions in the abdominal cavity occur most commonly with neoplastic masses of the spleen or liver. The most common causes are hemangiosarcoma and hepatocellular carcinoma. Clinical signs associated with acute abdominal hemorrhage, regardless of the cause, are related to hypovolemic shock and decreased perfusion and include pale mucous membranes, tachycardia, anemia, lethargy, and acute collapse. Treatment for abdominal hemorrhage includes placement of a large-bore peripheral cephalic catheter and starting one fourth of a shock dose (90 mL/kg for dogs and 44 mL/kg for cats) of intravenous crystalloid fluids, taking care to carefully monitor perfusion parameters of heart rate, capillary refill time, mucous membrane color, and BP. Administer intravenous colloids such as hydroxyethyl starch or pentastarch (5 to 10 mL/kg IV bolus) to restore intravascular volume and normotension. Treat severe anemia with whole blood or packed RBCs to improve oxygen-carrying capacity and oxygen delivery (see sections on transfusion medicine and treatment of shock). Confirm the presence of hemoabdomen abdominocentesis (see section on abdominocentesis). The presence of nonclotting hemorrhagic effusion is consistent with free blood. PCV of the fluid is usually the same or higher than that of the peripheral blood. An abdominal compression bandage can be placed while further diagnostics are being performed.

In cases of acute hemoabdomen, obtain right lateral, left lateral, and ventrodorsal or dorsoventral thoracic radiographs to help rule out obvious metastasis. Monitor the patient’s ECG and correct dysrhythmias as necessary (see section on cardiac dysrhythmias). Surgery is indicated once the patient is stabilized. In some cases, hemorrhage is so severe that the patient should be taken immediately to surgery.

When recommending surgery for a hemorrhaging intraabdominal mass, it is important to discuss likely diagnoses and long-term prognosis with the owner. Hemangiosarcoma usually involves the spleen or liver or both. The presence of free abdominal hemorrhage is associated with a malignant tumor in 80% of cases. Even when free abdominal hemorrhage is not present, the tumor is malignant in 50% of cases. Approximately 66% (two thirds) of masses in the spleen are malignant (hemangiosarcoma, lymphoma, mast cell tumor, malignant fibrous histiocytoma, leiomyosarcoma, fibrosarcoma), and approximately one third are benign (hematoma, hemangioma).

Hepatocellular carcinoma usually affects one liver lobe (usually the left), and surgery is the treatment of choice. With complete surgical excision, median survival in dogs is longer than 300 days. If diffuse disease is observed at the time of surgery, the prognosis is poor.

Nonhemorrhagic effusions are associated with mesothelioma, lymphoma, carcinomatosis, or any mass that causes vascular or lymphatic obstruction. Clinical signs of respiratory distress and abdominal distension with nonhemorrhagic effusions are usually slowly progressive in onset and not as severe as those observed with hemorrhage. Treatment is usually aimed at identification of the underlying cause.

Obtain a fluid sample via thoracocentesis or abdominocentesis. To obtain further cells for cytologic evaluation, aspirate fluid from the thoracic or abdominal mass with ultrasonic guidance. Cytologic evaluation of the fluid will often elucidate the causative
<table>
<thead>
<tr>
<th>TABLE 1-48 Paraneoplastic Syndromes in Dogs and Cats</th>
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</thead>
<tbody>
<tr>
<td><strong>Paraneoplastic Syndrome</strong></td>
</tr>
<tr>
<td>----------------------------</td>
</tr>
<tr>
<td>Neutropenia</td>
</tr>
<tr>
<td>Sepsis</td>
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<tr>
<td>Thrombocytopenia</td>
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<tr>
<td>Anemia</td>
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<tr>
<td>Erythrocytosis</td>
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<td>DIC</td>
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</tbody>
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*Continued*
<table>
<thead>
<tr>
<th>Paraneoplastic Syndrome</th>
<th>Causes and Clinical Signs</th>
<th>Tumor Type</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypergammaglobulinemia</td>
<td>Increased serum viscosity following increased immunoglobulin G production by tumor, ocular hemorrhage and retinal detachment, dementia, seizures, petechiae, bleeding, occult infection</td>
<td>Plasma cell tumor, multiple myeloma</td>
<td>Treatment of underlying cause, melphalan and prednisone</td>
</tr>
<tr>
<td>Acute tumor lysis</td>
<td>Acute tumor cell death after chemotherapy, acute collapse and shock, vomiting, atrial standstill from hyperkalemia, bradycardia, muscle twitching</td>
<td>Lymphoma, leukemia</td>
<td>Crystalloid fluid syndrome therapy, treatment of hyperkalemia, monitoring of electrolyte status</td>
</tr>
<tr>
<td>Hypercalcemia</td>
<td>Parathyroid-related peptide, increased osteoclast activity; vomiting, diarrhea, constipation, polyuria and polydipsia, bradycardia, stupor, hypertension, weakness, seizures</td>
<td>Lymphoma, apocrine gland adenocarcinoma, multiple myeloma, mammary adenocarcinoma, parathyroid adenoma, parathyroid adenocarcinoma</td>
<td>Administration of 0.9% sodium chloride intravenously, prednisolone, bisphosphonates, furosemide, salmon calcitonin</td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>Sepsis, insulin secretion or insulin-like peptide secretion from tumor, catecholamine release, weakness, seizures</td>
<td>Pancreatic beta cell tumor (insulinoma), leiomyosarcoma, leiomyoma, oral melanoma, hepatoma, hepatocellular carcinoma</td>
<td>Surgical removal of tumor, supplemental dextrose in intravenous fluids, parathyroid hormone–related peptide, prednisone, propranolol</td>
</tr>
</tbody>
</table>

DIC, Disseminated intravascular coagulation; G-CSF, granulocyte colony-stimulating factor.
tumor type. An abdominal ultrasound can determine the degree of metastasis. Perform therapeutic abdominocentesis or thoracentesis if the effusion is causing respiratory difficulty. Rapid reaccumulation of the fluid potentially can cause hypoproteinemia and hypovolemic shock.

Mesothelioma is a rare tumor most commonly observed in urban environments. In humans, mesothelioma has been associated with exposure to asbestos. It is sometimes difficult to differentiate between reactive mesothelial cells and malignant mesothelial cells. Treatment is aimed at controlling the neoplastic effusion. Intracavitary cisplatin has been demonstrated to slow rates of fluid reaccumulation but is largely a palliative therapy. Lymphoma is another type of tumor that can cause thoracic or abdominal effusion. Cytologic evaluation of the fluid usually reveals abundant lymphoblasts. Treatment with multiagent chemotherapy protocols, with or without adjunctive radiation therapy, can prevent tumor remission and stop fluid accumulation.

Carcinomatosis occurs as a result of diffuse seeding of the abdominal cavity with malignant carcinomas and carries a poor prognosis. Carcinomatosis may occur de novo or from metastasis of a primary tumor. Treatment consists of fluid removal when respiratory difficulty occurs, with or without intracavitary cisplatin as a palliative measure. Cisplatin should never be used in cats because of fatal acute pulmonary edema.

**Thoracic Cavity**

Clinical signs of hemorrhagic thoracic effusion include acute respiratory distress, anemia, hypovolemic or cardiogenic shock, and collapse. Hemorrhagic thoracic effusions are rare in association with neoplastic effusions. A notable exception is intrathoracic hemorrhage in young dogs with osteosarcoma of the rib. Hemorrhage can result when a primary lung tumor erodes through a vessel. Hemangiosarcoma of the lungs or right auricular area can also result in hemorrhagic thoracic effusion. In many cases, hemorrhage may be confined to the pericardial sac with a right auricular mass, causing a globoid cardiac silhouette on thoracic radiographs.

Treatment consists of pericardiocentesis (see section on pericardial effusion and pericardiocentesis) and placement of a pericardial window, or the mass may be removed if it is in the right auricular appendage and resectable. Although surgery can resolve clinical signs of right-sided heart failure, metastatic disease often develops soon afterward.

Nonhemorrhagic thoracic effusion is more common than hemorrhagic thoracic effusion and is caused most commonly by mesothelioma, lymphoma, carcinomatosis, and thymoma. Clinical signs develop gradually and include respiratory difficulty, cyanosis, and cough. Supplemental oxygen should be administered. In many cases, thoracentesis can be therapeutic and diagnostic. Obtain thoracic radiographs both before and after thoracentesis to determine whether a mass effect is present. After identification of a cause, definitive therapy can be instituted.

Mesotheliomas are rare and are associated with diffuse serosal disease. They are more common in dogs than in cats. Effusions caused by mesotheliomas can affect the pleural or pericardial cavities. Treatment is directed at removing effusion fluid and controlling reaccumulation with use of intracavitary platinum compounds; carboplatin and cisplatin can be used in dogs. (Cisplatin and carboplatin should never be used in cats.) Chemical or physical pleurodesis may be helpful in controlling reaccumulation of fluid, but it is very painful in small animal patients.

Thoracic effusion secondary to lymphoma often is associated with an anterior mediastinal mass. T-cell lymphoma is the most common type of mediastinal mass observed in dogs. B-cell lymphoma is associated with a decreased response to chemotherapy and shorter survival times. Treatment consists of combination chemotherapy with or without radiation therapy to decrease mass size.

Carcinomatosis is a diffuse disease of the pleural cavity that often is a result of metastasis from a primary pulmonary carcinoma or mammary adenocarcinoma. Treatment is similar to that for mesothelioma and is aimed at controlling the effusion and delaying its recurrence.
Thymomas have been documented in both dogs and cats. Dogs most commonly are presented with a cough, whereas cats are presented with clinical signs of respiratory distress and a restrictive respiratory pattern associated with the presence of pleural effusion. An anterior mediastinal mass is often observed on thoracic radiographs. In some cases the pleural effusion must be drained via thoracocentesis before a mass is visible. Ultrasound-guided aspiration and cytologic evaluation of the mass reveal a malignant epithelial tumor with small lymphocytes and mast cells. Prognosis is good if the tumor can be completely excised. Treatment consists of surgical removal with or without presurgical radiation therapy to shrink the mass. Paraneoplastic syndromes of myasthenia gravis have been documented in dogs with thymomas. If megaesophagus or aspiration pneumonia is present, the prognosis is more guarded because of the high rate of complications.

**Neoplasia Causing Organ System Obstruction**

**Urinary Tract**

Obstructive lesions affecting the urinary tract can be extramural (intraabdominal, pelvic, or retroperitoneal) or intramural (urethral, bladder, or urethral wall). Transitional cell carcinoma is the most common type of bladder tumor observed in dogs. Prostatic adenocarcinoma, or neoplasia of the sublumbar lymph nodes (lymphoma, adenocarcinoma from apocrine gland adenocarcinoma), also can cause urethral obstruction. Treatment is aimed at relieving the obstruction and then attempting to identify the cause of the disease. To alleviate the obstruction, pass a urinary catheter whenever possible. Perform cystocentesis only as a last resort because of the risk of seeding the peritoneal cavity with tumor cells if transitional cell carcinoma is the cause of the obstruction. Institute supportive therapy including intravenous fluids and correction of electrolyte abnormalities.

Plain radiographs may reveal a mass lesion or may not be helpful without double contrast cystography. Abdominal ultrasound is more sensitive in identifying a mass lesion in the urinary bladder. Masses in the pelvic urethra are difficult to visualize with ultrasonography. Double contrast cystourethrography is preferred. Once the patient has been stabilized, biopsy or surgery is indicated to identify the cause of the mass and attempt resection. Urine tests for transitional cell carcinoma are available for identification of transitional cell carcinoma in the dog.

Complete surgical excision of transitional cell carcinoma or removal of benign tumors of the urinary bladder yields a favorable prognosis. Poorer prognosis is seen with incomplete excision. Many transitional cell carcinomas are located in the trigone region of the bladder and cannot be completely excised. The NSAID piroxicam is helpful in alleviating clinical signs for a reported 7-month median survival. In some dogs, cisplatin and carboplatin may delay recurrence of transitional cell carcinoma.

Tumors of the prostate gland are always malignant and occur with equal frequency in castrated and uncastrated male dogs. Diagnosis of prostatic tumors is based on ultrasonographic evidence of a mass effect or prostatomegaly and on transrectal or transabdominal aspiration or biopsy. Surgery, chemotherapy, and radiation therapy generally are unrewarding over the long term, although palliative radiation therapy may relieve clinical signs for 2 to 6 months.

**Gastrointestinal Obstruction**

Luminal tumors of the gastrointestinal tract typically cause obstruction, with slowly progressive clinical signs including vomiting, inappetence, and weight loss or with acute severe protracted vomiting. Extraluminal obstructive lesions usually arise from adhesions, or strangulation may occur, resulting in obstruction. Perforation of the mass through the gastric or intestinal wall can cause peritonitis. Treatment consists of initial stabilization and rehydration, evaluation for evidence of metastasis, and surgical resection of the affected area in cases of adenocarcinoma, leiomyoma, leiomyosarcoma, and obstructive or perforated lymphoma.
Gastric and intestinal adenocarcinoma are the most common gastrointestinal tumors observed in dogs. Affected animals typically have a history of anorexia, weight loss, and vomiting. Obtain an abdominal ultrasound before performing any surgery. Fine needle aspirates of the mass and adjacent lymph nodes are usually diagnostic and can determine whether there is local metastasis. Many tumors are not resectable, and metastasis occurs in approximately 70% of cases. Dogs with smaller tumors that can be resected typically have longer survival times.

Leiomyosarcomas occur in the intestines of dogs and carry a more favorable prognosis than adenocarcinoma if the mass can be completely resected. With complete resection, the average survival time is longer than 1 year. The paraneoplastic syndrome of hypoglycemia has been observed with this tumor type.

Gastrointestinal lymphoma is the most common tumor of the gastrointestinal tract observed in cats. In comparison, it is relatively rare in dogs. Unless there is complete obstruction or perforation of the gastrointestinal tract, surgical treatment for gastrointestinal lymphoma is not indicated. Rather, multiple chemotherapy drugs are used in combination to achieve remission and resolution of the clinical signs of anorexia, weight loss, and vomiting. Treatment responses unfortunately are poor.

Mast cell tumors of the gastrointestinal tract typically manifest as gastrointestinal ulceration and hemorrhage in up to 83% of patients. The gastrointestinal hemorrhage that occurs with mast cell tumors results from increased acid secretion as a result of histamine receptor stimulation. Treatment consists of histamine or proton pump inhibition (ranitidine, famotidine, cimetidine, or omeprazole). Bowel perforation is a rare complication.

### Paraneoplastic Syndromes

#### Chemotherapy-Related Toxicities

Many chemotherapy agents exert their effects on rapidly dividing normal and neoplastic cells. Normal tissues that are commonly affected include the bone marrow, gastrointestinal tract, skin and hair follicles, and reproductive organs. Some drugs have unique organ-specific toxicities that must be monitored. Knowledge and recognition of the expected type and onset of complications can alleviate their severity by permitting rapid treatment when complications occur (see Table 1-48).

#### Bone Marrow Toxicity

Neutropenia is the most common bone marrow toxicity observed secondary to chemotherapy in small animal patients (Table 1-49). In most cases the neutropenia is dose-dependent. The nadir, or lowest neutrophil count, is typically observed 5 to 10 days after chemotherapy treatment. Once the nadir occurs, bone marrow recovery is observed, with an increase in circulating neutrophils within 36 to 72 hours (see Table 1-49).

Treatment of myelosuppression is largely supportive to treat or prevent sepsis. Prophylactic antibiotics are recommended in the afebrile patient with a neutrophil count <2000/mcL. Acceptable antibiotics include trimethoprim-sulfamethoxazole (TMX) and amoxicillin-clavulanate.

<table>
<thead>
<tr>
<th>Degree of Myelosuppression</th>
<th>Time of Nadir</th>
<th>Causative Agent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild to none</td>
<td>Not observed</td>
<td>Vincristine (low-dose), L-asparaginase, glucocorticosteroids</td>
</tr>
<tr>
<td>Moderate</td>
<td>7-10 days</td>
<td>Melphalan, cisplatin, mitoxantrone, actinomycin D</td>
</tr>
<tr>
<td>Severe</td>
<td>7-10 days</td>
<td>Doxorubicin, cyclophosphamide, vinblastine</td>
</tr>
</tbody>
</table>
Granulocyte colony-stimulating factor (G-CSF) (e.g., Neupogen) is a recombinant human product that stimulates the release of neutrophils from the bone marrow, and its use shortens the recovery time after myelosuppressive drug therapy. Disadvantages of G-CSF include antibody production in response to the drug within 4 weeks of use and its high cost. To prevent ongoing neutropenia, subsequent chemotherapy doses should be decreased by 25%, and the interval between treatments increased. Whenever possible, overlap of myelosuppressive drugs should be avoided.

**Gastrointestinal Toxicity**

Acute gastrointestinal toxicity can occur within 6 to 12 hours after administration of cisplatin and actinomycin D. In many cases, pretreatment with the antiemetic metoclopramide, butorphanol, chlorpromazine, dolasetron or ondansetron can prevent chemotherapy-induced nausea and vomiting. Vomiting can also occur as a delayed side effect 3 to 5 days after treatment with doxorubicin (Adriamycin), actinomycin D, methotrexate, and Cytoxan. In delayed reactions, vomiting and diarrhea are caused by damage to intestinal crypt cells. Treatment consists of administration of antiemetics, intravenous fluids, and a bland highly digestible diet. Doxorubicin also can cause hemorrhagic colitis within 5 to 7 days of administration. Treatment includes a bland diet, metronidazole, and tylosin tartrate (Tylan powder). Paralytic ileus can be observed 2 to 5 days after administration of vincristine. This side effect is more common in humans than animals and can be treated with metoclopramide once a gastrointestinal obstruction has been ruled out.

**Cardiotoxicity**

Doxorubicin (Adriamycin) causes a dose-dependent dilative cardiomyopathy when the cumulative dose reaches 90-240 mg/m². In many cases, however, clinical signs do not occur until the cumulative dose is 240 mg/m². The myocardial lesions are irreversible. Treatment of cardiac dysrhythmias is dependent on the type of dysrhythmia (see section on treatment of dysrhythmias). Discontinue doxorubicin and administer diuretics and positive inotropic therapy for dilative cardiomyopathy in order to delay the progression of CHF (see sections on treatment of congestive heart failure). If abnormalities are shown on electrocardiography performed before beginning therapy, substitute liposome-encapsulated doxorubicin or mitoxantrone in the chemotherapy protocol. Cardioprotectant drugs such as vitamin E, selenium, and N-acetylcysteine have shown some promise in the prevention of doxorubicin-induced cardiotoxicity.

**Urinary Bladder Toxicity**

Cyclophosphamide can cause a sterile hemorrhagic cystitis. Damage to the urinary bladder mucosa and vessels is caused by the toxic metabolite acrolein. Clinical signs of sterile hemorrhagic cystitis include a history of cyclophosphamide administration, stranguria, hematuria, and pollakiuria. Treatment for sterile hemorrhagic cystitis is discontinuation of the drug, treatment of any underlying urinary tract infection with antibiotic therapy based on susceptibility testing, and intravesicle drug administration. In extremely refractory cases, surgical debridement and cauterization of the bladder mucosa may be necessary.

Prevention of sterile hemorrhagic cystitis includes emptying the bladder frequently and administering the drug in the morning. Concurrent administration of prednisone can induce PU/PD. If sterile hemorrhagic cystitis occurs, chlorambucil can be substituted as a chemotherapeutic agent.

**Anaphylactic Reactions**

Anaphylactic reactions have been observed with the administration of L-asparaginase, Adriamycin, etoposide, and paclitaxel. The risk of anaphylaxis increases with repeated administration, although in some animals anaphylaxis will occur on the first exposure to the drug. Treatment consists of administration of epinephrine, diphenhydramine,
famotidine, and glucocorticosteroids, as with any other life-threatening allergic reaction (see section on treatment of allergic reactions). To decrease the risk of an adverse reaction, give diphenhydramine (For dogs up to 20 lbs, 10 mg IV; For dogs 20–60 lbs, 20 mg IV; and for dogs over 60 lbs, 30 mg IV) 15 to 30 minutes before drug administration. Slowing the rate of intravenous infusion also can decrease the chance of an anaphylactic reaction.

**Acute Tumor Lysis Syndrome**

Acute tumor lysis results from massive destruction of neoplastic cells after chemotherapy or radiation, particularly in veterinary patients with lymphoma. The release of cellular contents after cell death can result in hyperkalemia, hyperphosphatemia, azotemia, hyperuricemia, and hypocalcemia. Risk factors for acute tumor lysis include high tumor burden and preexisting renal insufficiency. Treatment for suspected acute tumor lysis includes intravenous fluid diuresis, antiemetics if the patient is vomiting, and broad-spectrum antibiotics if diarrhea is present.

**Species-Specific Toxicities**

Cisplatin can cause a fatal irreversible pulmonary edema in cats, even at low dosages. 5-Fluorouracil (5-FU) can cause a severe neurotoxicity in cats that results in ataxia and seizures. Never use cisplatin or 5-FU in cats.

**Additional Reading**


**POISONS AND TOXINS**

Poisoning cases benefit from a rapid, organized approach. Key points in this approach are giving appropriate advice over the telephone, being able to access information sources, and providing appropriate treatment. Only a few classes of poisons account for the majority of toxicities reported in dogs and cats.

Every veterinarian should develop a familiarity with the clinical management of rodenticide and insecticide toxicity and be prepared with antidotes on hand. Beyond the most common toxins, the spectrum of possibilities is endless, and the veterinarian must rely on appropriate information resources. It is important to have available a comprehensive source of pharmaceutical and plant identification resources.

Remarkably, considering the myriad potentially toxic substances to which an animal can be exposed, relatively few specific antidotes are commonly used in veterinary medicine. Because of the lack of specific antidotes, the veterinarian must treat each toxicity with general methods of poison management, applying basic critical care in the treatment of specific clinical signs associated with the poison exposure or toxicity. The adage “Treat the patient, not the poison” often comes into play when the exact toxic substance is unknown or has no specific antidote.

**Advising Clients over the Phone**

Before an animal arrives, the staff should be prepared to ask specific questions over the phone and provide initial advice for clients, particularly if the animal lives some distance from the hospital (Box 1-55).
Toxicology Resources

It is important to have access to a database of information on toxic substances. Thousands of potentially toxic substances are available on the market today. The American Society for the Prevention of Cruelty to Animals (ASPCA) Animal Poison Control Center provides direct access to veterinary toxicologists 24 hours a day, 365 days a year. For additional information, call the nearest veterinary school or emergency center (Box 1-56). Also, see Section 6 for a table of emergency hotlines.

Human Poison Control Centers

Check your local telephone book for a poison control center listing under emergency numbers, usually found inside the front cover. Although these numbers are for organizations dealing with human poisonings, they have access to extensive poison and toxin databases and can potentially provide useful information for veterinarians, particularly regarding antidotal substances suitable for out-of-the-ordinary toxins and human medications. Information on the toxic ingredients in thousands of medications, insecticides, pesticides, and other registered commercial products has been confidentially placed by the government in these poison control centers. As new products are marketed, information regarding toxin ingredients is forwarded to the centers.

Internet

Various e-mail discussion lists can serve as an informative resource for practitioners, but access generally requires an initial subscription and may have the disadvantage of delayed response times. They are useful for ideas on standard and long-term therapy, but not
emergency stabilization. An exception to this is the Veterinary Interactive Network (VIN), which posts message board communications. Previous communications from veterinarians who treated a case with the same poison or toxin can be accessed with a subscription.

**Manufacturers**

Many manufacturers operate an information service about their products. If the product label or name is available, check for a telephone number that may route you to a specialist.

**Essential Steps in Emergency Treatment of Toxicities**

There are six essential steps in treating toxicities:

1. Performing a physical examination
2. Stabilizing the patient’s vital signs
3. Taking a thorough history
4. Preventing continued absorption of the toxin
5. Administering specific antidotes when available
6. Facilitating clearance or metabolism of the absorbed toxin

It is most important to provide symptomatic and supportive care both during and after emergency treatment.

**Physical Examination**

Immediately on presentation, perform a brief but thorough physical examination. Obtain a minimum database as well as serum, urine, or orogastric lavage samples for later toxicologic analyses. It is important at this time to systematically evaluate the patient’s physical status, focusing particularly on the toxins most common to a particular geographic location and the organ systems most commonly affected by toxins in veterinary medicine—namely, the neurologic and gastrointestinal tracts. A checklist is useful when performing a complete physical examination (Box 1-57).

**Minimum Database**

The minimum database includes a urine sample, PCV, total protein, serum urea, and serum glucose. The information obtained from these simple cage-side tests is useful for determining dehydration, hemoconcentration, azotemia (renal or prerenal), and hypoglycemia or...
**Box 1-57 Physical Examination Checklist**

### Eyes, Ears, Nose, and Throat
- What is the pupil size?
- What is the pupil reactivity to light?
- Are the ocular examination findings normal?
- What is the sensitivity to light or sound?
- Nose: Is it moist, dry, bubbling, or frothy, or caked with dirt?
- Throat: Are there any characteristic odors on the breath?
- Are there any traces of foreign material on the tongue or in the crevices of the teeth or gums?
- Are there petechiae or ecchymosis on the gums or bleeding from the gum line?

### Cardiovascular System
- What is the mucous membrane color? Is it normal and pink, or dark red (Injected), pale, or icteric?
- What is the capillary refill time? Is it fast, normal, or slow?
- What is the patient’s heart rate?
- Are there any pulse deficits or dysrhythmias auscultated?
- What is the patient’s blood pressure?
- What is the quality of the femoral pulse? Is it synchronous with the heart rate, or are there dropped pulses? Is the pulse bounding, normal, thready, or not palpable?
- What is shown on the patient’s electrocardiogram?

### Respiratory System
- What is the patient’s respiratory rate?
- What is the patient’s respiratory character? Is it normal, fast, shallow, or labored?
- What do you hear on thoracic auscultation? Do you hear harsh airway sounds or pulmonary crackles?

### Gastrointestinal and Hepatic Systems
- What is the patient’s rectal temperature?
- Is there excessive salivation?
- Is there evidence of vomiting or diarrhea?
- Is abdominal palpation painful?
- Do the intestinal loops feel normal, or are they fluid-filled or gas-filled?
- What are the color and consistency of the feces?

### Urogenital System
- Is there a palpable urinary bladder?
- Is there urine production?
- What is the color of the urine?

### Musculoskeletal and Neurologic
- What is the patient’s gait?
- Is the patient weak or recumbent?
- Is the patient ataxic?
- Does the patient display signs of hypermetria?
- Are there muscle fasciculations?
- Is there increased extensor tone?
- What is the patient’s attitude?
- Score the animal’s level of consciousness on a simple scale:
  - Alert
  - Responds to voice
  - Responds to touch
  - Responds to painful or noxious stimulus
  - Unresponsive: unconscious

### Integument
- Are there wet patches that smell of a particular substance?
- Is there any evidence of erythema or ulcerations?
- Does a black light cause the muzzle, paws, prepuce, or vulva to fluoresce?

### Peripheral Lymph Nodes
- Should be normal in poisonings

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hyperglycemia. When appropriate, obtain samples for serum biochemistry profiles, serum electrolytes, blood gases, serum osmolality, complete hemogram, and coagulation profiles. Samples of serum, urine, and any vomitus or orogastric lavage contents should be collected and saved for toxicologic analyses as required later.

**Stabilization of Vital Signs**

Stabilization of vital signs includes four major goals of treatment: maintain respiration, maintain cardiovascular function, control CNS excitation, and control body temperature. In any patient with clinical signs of respiratory distress or respiratory dysfunction, supplemental oxygen should be administered via flow-by, oxygen hood, oxygen cage, or nasal, nasopharyngeal, or transtracheal oxygen sources. Ventilatory assistance may be necessary. Irritant or corrosive substances can cause damage to the oropharyngeal mucosa to such an extent that airway obstruction occurs. When necessary, a temporary tracheostomy should be performed. Arterial blood gases, pulse oximetry, and capnometry may be required to monitor oxygenation and ventilation.

At the time of presentation, immediately place an intravenous catheter for administration of intravenous fluids, inotropes, antiarrhythmics, and antidotes, if necessary. The initial fluid of choice is a balanced crystalloid solution such as Normosol-R, Plasma-Lyte M, or lactated Ringer’s solution. Fluid therapy can later be changed based on the patient’s acid-base and electrolyte status. Some toxins can cause severe dysrhythmias and hypertension or hypotension. Monitor BP and perform electrocardiography, and correct any abnormalities according to standard therapy (see sections on hypotension and cardiac dysrhythmias). Some toxins cause hemolysis, methemoglobinemia, Heinz body anemia, and coagulopathies. Whole blood, fresh frozen plasma, or packed RBCs should be available and used if necessary. Treat methemoglobinemia with a combination of ascorbic acid and N-acetylcysteine.

Many toxins affect the CNS, producing clinical signs of excitation and/or seizures. Diazepam is the drug of choice for most but not all seizures and tremors. If an animal has CNS excitation secondary to the ingestion of selective norepinephrine reuptake inhibitors, avoid using diazepam, as it can potentially exacerbate clinical signs. Muscle relaxants such as guaifenesin (110 mg/kg IV) or methocarbamol (50 to 220 mg/kg IV not to exceed 330 mg/kg/day) may be required to control muscle spasm and tremors associated with some toxicities. Consider animals that are in status epilepticus because of toxin exposure at high risk. Such patients may not require the full dose of anesthetics or sedatives for seizure control. Give phenobarbital (bolus 2-5 mg/kg IV, can repeat every 20 minutes 2 times) or pentobarbital (5 to 15 mg/kg IV to effect) for longer-term management of seizures.

Core body temperature can easily increase or decrease secondary to increased muscle activity or coma. Animals may be hypothermic or hyperthermic, depending on the toxin ingested and the stage of toxicity. Manage hyperthermia with circulating hot water or hot air blankets, or place bubble wrap or Saran wrap around the animal’s peripheral extremities. Manage hyperthermia by placing lukewarm wet towels on the patient until the rectal temperature has decreased to 39.5°C (103°F). (See section on hyperthermia and heat-induced illness.) If sedatives or anesthetics have been used, initial hyperthermia may initially resolve because of hypothalamic loss of thermoregulatory control; cool water bathing should not be performed.

**Obtain a Thorough History**

When the patient is first presented to the veterinarian, have the owner complete a toxicologic history form (Figure 1-56) while the animal is being initially assessed and vital signs are being stabilized. When initial stabilization of vital signs has been accomplished, the veterinarian can discuss the patient’s history with the owner. In urgent situations, the veterinarian should obtain a brief history as an initial procedure (Box 1-58).

Knowing when the animal was last seen as normal provides a time frame in which the toxic substance was most likely accessed, allowing differential diagnoses to be ranked in some order of probability by rate of onset. In eliciting a history from the owner about the animal’s access to poisons, it is important not to take anything for granted. Many
Toxicologic History Form

Date: 
Time: 

Patient information: 
Name of animal: 
Age: 
Breed: 
Gender and Neuter status: 
Weight: 
Vaccinations last given: 
Any current medications (including heartworm prevention and nutriceuticals)

Today’s Problem
When did you first notice that something was wrong with your pet?
When was the last time you noticed your pet act normally?
What was the first abnormal sign noticed?
What other conditions have developed and what are they?
How soon did other signs develop?
Have the signs become better or worse since you first saw them?

Information on any suspected poison
What is the name of the product?
Do you have the container with you today?
Is it a liquid concentrate, dilute spray, or solid?
How long ago do you think that your pet was exposed to the poison?
Where do you think it happened?
Do you have any over-the-counter or prescription medications that your animal may have had access to?
Did you give any medications to your animal?
Is there any possibility of recreational drug exposure?

Your pet’s recent activity
Did your pet eat this morning or last night?
What is he/she normally fed?
Is there a chance that your pet may have gotten into the garbage?
Have you fed table scraps or anything new recently? If so, what?
Has your pet been off your property in the last 24-48 hours?
 Does your pet run loose unattended?
Has your pet had any antiflea/tick medication within the last week?

Your pet’s environment
Is your animal kept inside or outside of the house?
Is your pet kept in a fenced-in yard or allowed to run loose unattended?
Does your pet have access to neighboring properties (even for a short time)?
Where has your pet been in the last 24 hours?
Has your pet traveled outside of your immediate geographic location? If so, when?
Has your pet been to rural areas in the last week?

Your household’s recent activity
Has there been any gardening work recently?
Does your pet have access to a compost pile?
Any fertilizers or weed killer used in the last week?
Any construction work or renovation recently?
Any mouse or rat poison in your house, yard, or garage?
Any cleaning products used inside or outside the house within the last 48 hours? If so, which?
Have you changed your radiator fluid or does a car leak antifreeze?

Figure 1-56: Example of a thorough history form when a toxin is suspected.
owners do not realize how poisonous some substances can be, such as insecticide products, garbage, cleaning chemicals, and over-the-counter (OTC) drugs commonly used by humans. Many owners will deny that an animal could have ingested anything that might be toxic, not wanting to believe that the source of the toxin is within their household or property, particularly if recreational drug exposure is suspected. It is useful to phrase questions in a neutral fashion—for example, “Is such-and-such present on the premises?” rather than “Could the dog have eaten such-and-such?” If recreational drug exposure is suspected, another way to question the owners is to ask whether they have had any guests in their house recently who may have had such-and-such (e.g., marijuana, cocaine, methamphetamine). This approach serves to minimize the suggestion of any bias or preconceptions.

When questioning an owner about recent events, it is useful to realize and acknowledge that disruption in the household routine is a distinct factor in the occurrence of accidents, including poisonings. Examples of such disruptive events include moving from the house, a family member being ill or in the hospital, and renovations or recent construction. While these events are occurring, the safeguards followed by a normally careful owner may be disrupted. Often, doors or gates may be left open, animals may be outside instead of inside (or vice versa), and inexperienced people may be pet-sitters. Once owners are made aware of the importance of assessing such risks, they are often able to provide insight into otherwise baffling circumstances.

Prevent Continued Absorption of the Toxin

Various methods can be used to remove toxins from the gastrointestinal tract, including emesis, orogastric lavage, cathartics, and enemas. Adsorbents, ion exchange resins, or precipitating or chelating agents may be used. Removal of a toxic substance from the body surface may be necessary, depending on the toxin. The use of both emesis and orogastric lavage is less and less frequent in human medicine because of the risk of aspiration pneumonia and doubts about their efficacy. Currently, management of poisonings in human medicine relies heavily on the use of activated charcoal combined with sorbitol as a cathartic, when appropriate, and supportive critical care. It should be emphasized, however, that the majority of poisonings in humans are the result of overdoses of drugs (illicit or otherwise) that involve a relatively small volume and rapid absorption and for which this treatment is appropriate. Furthermore, adoption of the approach rests on the availability of a hospital intensive care infrastructure, which is not always available in veterinary practice.

Emetics

Induce emesis if the animal’s physiology and neurologic status are stable (i.e., the animal does not have respiratory depression or is not actively seizing, obtunded, unable to swallow or protect its airway). Do not administer the same emetic more than twice. If the emetic doesn’t work after two doses, give a different emetic or perform orogastric lavage under general anesthesia. Emetics are strictly contraindicated for toxicity from petroleum-based products and corrosives because of the risk of aspiration pneumonia and further esophageal damage. Emetics may also be of little value if poisons with antiemetic properties have been ingested, such as benzodiazepines, tricyclic antidepressants, and marijuana (Table 1-50).
Various emetics traditionally have been recommended for use in veterinary medicine. Many have fallen out of favor because of the risk of causing adverse consequences and side effects. Apomorphine (0.04 mg/kg IV or 0.25 mg/kg into conjunctival sac) remains the standard but is less useful in certain situations in which the poison causes CNS excitation or stimulation. It is ineffective in cats. Other emetics include xylazine and hydrogen peroxide. Do not use table salt because of the risk of severe oropharyngeal irritation and hypernatremia. Do not use mustard powder or dishwashing liquid detergent because of the risk of severe oropharyngeal, esophageal, and gastric irritation.

### Table 1-50: List of Emetics and Recommended Doses

<table>
<thead>
<tr>
<th>Name</th>
<th>Mechanism of Action</th>
<th>Dose and Onset</th>
<th>How Supplied</th>
<th>Adverse Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apomorphine</td>
<td>Dopaminergic receptor stimulation in chemoreceptor trigger zone; causes both CNS depression and stimulation and some respiratory depression</td>
<td>0.3-0.4 mg/kg or 0.25 mg/kg into conjunctival sac</td>
<td>6.25-mg tablets, can be compounded into sterile capsules for intravenous use</td>
<td>Respiratory and CNS depression Undesirable CNS excitement in metaldehyde toxicosis</td>
</tr>
<tr>
<td>Hydrogen peroxide</td>
<td>Gastric irritation</td>
<td>1-2 mL/kg or 5 ml (1 tsp) for every 10 lbs</td>
<td>3% solution PO, can be repeated once every 10 minutes</td>
<td>Protracted vomiting; some formulations have a stabilizing factor that can be converted to acetaminophen; use caution in very small dogs and in cats</td>
</tr>
<tr>
<td>Xylazine</td>
<td>Central α₂-agonist stimulation</td>
<td>0.44 mg/kg IM</td>
<td>Solution</td>
<td>Sedation, bradycardia, respiratory depression</td>
</tr>
</tbody>
</table>

CNS, Central nervous system; IM, intramuscularly; IV, intravenously; PO, orally.

Orogastric Lavage

Orogastric lavage is described in detail in the section on emergency procedures. Gastric lavage is contraindicated in treatment of toxicity from petroleum-based compounds and acid or alkali ingestion. The procedure can be messy but is very effective if performed within 1 to 2 hours of ingestion of the poison. To prevent aspiration, the patient should be placed under general anesthesia. Keep the animal’s head lowered during the procedure to prevent aspiration of stomach contents into the trachea. It is sometimes helpful to put the animal in both right and left lateral recumbency to allow complete emptying of gastric contents. Repeat the procedure until the fluid runs clear from the stomach. In some cases in which solid material has been ingested, this process can take a long time, so be prepared with a large volume of warm water.

Following successful evacuation and lavage, administer a slurry of activated charcoal through the orogastric tube before removing it. Keep the endotracheal tube cuffed and in
place until the animal is semiconscious, is starting the fight the tube, and is visibly able to swallow and protect its airway.

**Enemas**

Enemas are useful to facilitate the action of cathartics and in cases in which the poison is a solid material (e.g., compost, snail bait, garbage) (Box 1-59). It is best to use just lukewarm water. Commercially available phosphate enema solutions can cause severe electrolyte disturbances (hyperphosphatemia, hyponatremia, hypocalcemia, and hypomagnesemia) and acid-base abnormalities (metabolic acidosis); therefore they are absolutely contraindicated in small animal patients.

The fluid volume required depends on the size of the animal and the state of its lower gastrointestinal tract. As with orogastric lavage, continue the procedure until the water runs clear. If difficulty is encountered emptying the lower gastrointestinal tract, repeat the enema in 1 or 2 hours, rather than being overzealous on the first attempt.

**Cathartics**

Cathartics are useful for hastening gastrointestinal elimination of toxins, and they are particularly useful for elimination of most solid toxicants (e.g., compost, garbage, snail bait). Cathartics can be used in conjunction with activated charcoal. Do not use magnesium-based cathartics in patients with CNS depression, because hypermagnesemia can worsen this disorder and also cause cardiac rhythm disturbances.

Activated charcoal (1-4 g/kg PO or 5-20 ml of a 20% slurry) is the safest and to date the most effective adsorbent for the treatment of ingested toxins. Activated charcoal can be administered after emesis or orogastric lavage or can be administered as the sole treatment. Various preparations are available on the market, including dry powder, compressed tablets, granules, liquid suspensions, and concentrated paste preparations. Commercially available products are relatively inexpensive and should be used whenever possible for ease of administration. Vegetable-origin activated charcoal is the most efficient adsorbent and binds compounds with weak, nonionic bonds. Some preparations are combined with sorbitol to provide simultaneous administration of an adsorbent and a cathartic; this combination has been shown to be most efficacious.

Repeated administration of activated charcoal every 4 to 6 hours has been shown to be beneficial in the management of a toxin that undergoes enterohepatic recirculation. Administering an oily cathartic or mixing the activated charcoal with food only serves to reduce the absorptive surface of the activated charcoal and therefore is not recommended. In general, substances that are very soluble and are rapidly absorbed are not well adsorbed by activated charcoal, including alkalis, nitrates, mineral acids, ethanol, methanol, ferrous sulfate, ammonia, and cyanide.

### BOX 1-59  EQUIPMENT NEEDED FOR ENEMA ADMINISTRATION

**Tubing**
- Flexible red rubber catheters
- Foley balloon-tipped catheters if a retention enema is required

**Obstetric Lubricant**
- Use nonsterile nonspermicidal water-soluble lubricants (K-Y Jelly)

**Fluid Reservoir**
- Old intravenous fluid bag
- Enema bag
- 60- to 120-ml syringe

**Fluid**
- Warm water, with or without hand or liquid dish soap
Kaolin and bentonite are clays that have been used as adsorbents. Both are usually less effective than activated charcoal. However, they are reported to be better adsorbents than activated charcoal for the herbicide paraquat.

**Ion Exchange Resins**

Ion exchange resins can ionically bind certain drugs or toxins. Cholestyramine is one such resin, commonly used in human medicine to bind intestinal bile acids and thereby decrease cholesterol absorption. Its application in toxicology extends to the absorption of fat-soluble toxins such as organochlorine and certain acidic compounds such as digitalis. Ion exchange resins also have been used to delay or reduce the absorption of phenylbutazone, warfarin, chlorothiazide, tetracycline, phenobarbital, and thyroid preparations.

**Precipitating, Chelating, and Diluting Agents**

Precipitating, chelating, and diluting agents are used primarily in the management of heavy metal intoxications, such as alkaloids or oxalates. They work by binding preferentially to the metal ion and creating a more soluble complex that is amenable to renal excretion. Chelating agents that are available include calcium EDTA, deferoxamine, and D-penicillamine. D-Penicillamine has a wide application for a number of metal toxicities but tends to be used for long-term chronic therapy because it can be administered PO. Various agents used for non-specific dilution of toxins, including Milk of Magnesia and egg whites, although old-fashioned, still have wide application in many cases in which low-grade irritants have been ingested.

**Eliminating Poison from the Skin**

Bathing the animal is an important aspect of treatment for topical exposures to toxins such as insecticidal products, petroleum-based products, and aromatic oils. Bathing an animal is not an innocuous procedure. To avoid hypothermia and shock, use warm water at all times. Actively dry the animal to further minimize the risk of hypothermia. When bathing the animal, use rubber gloves and a plastic apron to avoid exposure to noxious agents.

In most cases, a mild dishwashing soap is appropriate. Medicated or antibacterial shampoos are less appropriate in this situation. For petroleum-based products in particular, Dawn dishwashing liquid, which “cuts the grease,” works well to remove the oils. If Dawn is not available, mechanics’ hand cleaners or coconut oil–based soaps can be used instead. As a general principle, best results are obtained by barely wetting the patient’s fur until the detergent is worked well into the fur, keeping the amount of water to a minimum until ready for the rinse. Oil-based paint is best removed by clipping rather than by attempting removal with solvents, because solvents are also toxic.

To remove powder products, brush and vacuum the animal before bathing it to eliminate further toxic exposure. With caustic alkaline or acidic products, the primary treatment is to dilute and flush the skin with warm water; do not attempt neutralization. Neutralization can cause an exothermic reaction that causes further damage to the underlying tissues.

**Eliminating Poison from the Eyes**

For ocular exposures, irrigate the eyes for a minimum of 20 to 30 minutes with warm (body temperature) tap water or warmed 0.9% sterile saline solution. The use of neutralizing substances is not recommended because of the risk of causing further ocular damage. After adequate irrigation, treat chemical burns of the eyes with lubricating ointments and possibly a temporary tarsorrhaphy. Atropine may be indicated as a cycloplegic agent. Systemic NSAIDs can be used to control patient discomfort.

Daily follow-up examinations are required because epithelial damage may be delayed, especially with alkali burns, and it is difficult to predict the final extent of ocular damage. Topical glucocorticosteroids are contraindicated if the corneal epithelium is not intact. If severe conjunctival swelling is present with a corneal ulcer, parenteral glucocorticosteroids can be administered to help alleviate inflammation, but NSAIDs should not be used simultaneously because of the risk of gastrointestinal ulceration or perforation.
Administer Antidotes
Whenever possible, administer specific antidotes to negate the effects of the toxin and prevent conversion of the substance to the toxic metabolite. Three categories of agents are used in the management of poisonings.

The first category is specific antidotes. Unfortunately, few specific antidotes are available for use in veterinary medicine.

The second, broader category of antidotes includes those drugs used in the symptomatic management of clinical signs, which are part of our routine veterinary stock. Drugs such as atropine, sedatives, steroids, antiarrhythmics, and β-blockers fall into this category.

The third category comprises nonspecific decontaminants such as activated charcoal, cathartics, and emetics. These were discussed previously.

Facilitate Clearance or Metabolism of Absorbed Toxin
Many patients benefit from efforts to enhance clearance or metabolism of the absorbed toxins. Some specific therapies have been developed for this purpose, including 4-methylpyrazole for ethylene glycol toxicity and specific antibodies such as Digibind (digoxin immune Fab [ovine]) for digitalis toxicity. Other strategies are aimed at promoting renal excretion. Renal excretion strategies include diuresis, ion trapping, and peritoneal dialysis or hemodialysis (see section on peritoneal dialysis). Diuresis and ion trapping are applicable to a large number of toxins and are discussed here in more detail. Other toxins respond to urine acidification and urine alkalinization.

Enhancing renal excretion of substances is most useful for those organic substances that are present in significant concentrations in the plasma. Substances that are nonionic and lipid-soluble, such as certain herbicides, are likely to be less affected by attempts to promote rapid renal elimination.

Before diuresis or ion trapping is started, intravenous fluid therapy should be adequate as determined by normal CVP, urine output, and mean arterial BP. If any of these values is less than normal, use other measures to ensure adequate renal perfusion, including but not limited to CRI of dopamine.

Simple fluid diuresis can influence the excretion of certain substances. The use of mannitol as an osmotic diuretic may reduce the passive reabsorption of some toxic substances in the proximal renal convoluted tubule by reducing water reabsorption. Dextrose (50%) can be used as an osmotic diuretic. Furosemide can be used to promote diuresis, but again, there is no substitute for intravenous fluid therapy. The use of mannitol, dextrose, and furosemide is contraindicated in hypotensive or hypovolemic patients. Take care to avoid causing dehydration with any diuretic; CVP monitoring is strongly recommended.

Urine Acidification and Alkalination
Ion trapping is based on the principle that ionized substances do not cross renal tubular membranes easily and are not well reabsorbed. If the urinary pH can be changed so that the toxin’s chemical equilibrium shifts to its ionized form, then that toxin can be “trapped” in the urine and excreted. Alkaline urine favors the ionization of acidic compounds, and acidic urine favors the ionization of alkaline compounds. Toxins that are amenable to ion trapping are mostly weak acids and weak bases.

Ammonium chloride can be used to promote urinary acidification. Contraindications to the use of ammonium chloride include preexisting metabolic acidosis, hepatic or renal insufficiency, and hemolysis or rhabdomyolysis leading to hemoglobinuria or myoglobinuria. Signs of ammonia intoxication include CNS depression and coma. When performing urine acidification, frequently check the serum potassium concentration and urine pH.

Urine alkalinization can be performed with use of sodium bicarbonate. Contraindications to the use of sodium bicarbonate include metabolic alkalosis (particularly with concurrent use of furosemide), hypocalcemia, and hypokalemia. As with urine acidification, monitor the serum potassium concentration and urine pH frequently.
Supportive and Symptomatic Care of the Poisoning Patient

The major steps in management of poisonings discussed here must be accompanied by application of the fundamentals of critical care. Respiratory and cardiovascular support have been discussed previously. Renal and gastrointestinal function and analgesia are particularly important in the management of the poisoning patient.

Maintenance of renal perfusion is a priority in the poisoning patient. Fluid, electrolyte, and acid-base balance must be controlled and be accurate. Poisoning patients are at particularly high risk for renal damage and acute renal failure, whether by primary toxic insult to the renal parenchyma or by acute or prolonged renal hypoperfusion. For this reason, a protocol that aims at preventing oliguria and ensuing renal failure is one of the therapeutic strategies that should be routinely employed. This protocol is described in Box 1-60.

Gastrointestinal Protectants

Gastrointestinal protectant drugs may be indicated for the management of poisons that are gastrointestinal irritants or ulcerogenic. Commonly used gastroprotectant drugs include cimetidine, ranitidine, famotidine, omeprazole, sucralfate, and misoprostol.

Antiemetics

Antiemetics may be used to suppress intractable vomiting. Metoclopramide is commonly used, although newer antiemetics such as maropitant is now considered to be the drug of choice for centrally mediated nausea. Antiemetics that work by different mechanisms can be used in combination as necessary. Examples are dopamine D₂-receptor antagonists such as prochlorperazine, 5-hydroxytryptamine antagonists such as ondansetron and dolasetron, and H₁-receptor antagonists such as diphenhydramine and meclizine.

Analgesics

Analgesics are more appropriate to treat poisonings than once thought. Common effects of poisons including severe gastroenteritis and topical burns or ulcerations may warrant the use of analgesics. Longer-acting analgesics such as morphine, hydromorphone, and buprenorphine are particularly useful.

Nutritional Support

Nutritional support may be necessary in the form of enteral or parenteral feeding in patients that have esophageal or gastric damage or that need to be sedated for long periods of time.

<table>
<thead>
<tr>
<th>BOX 1-60</th>
<th>MAINTENANCE OF RENAL PERFUSION</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Administer crystalloid intravenous fluids at maintenance rates using a balanced electrolyte solution.</td>
</tr>
<tr>
<td>2.</td>
<td>Perform urinary catheterization and collection to monitor urine output.</td>
</tr>
<tr>
<td>3.</td>
<td>Monitor serum urea nitrogen and creatinine every 12 hours.</td>
</tr>
<tr>
<td>4.</td>
<td>Monitor serum electrolytes every 6 to 8 hours.</td>
</tr>
<tr>
<td>5.</td>
<td>Monitor central venous pressure every 2 to 4 hours.</td>
</tr>
<tr>
<td>6.</td>
<td>Treat oliguria, defined as a drop in urine output to less than 1 mL/kg/hr.</td>
</tr>
<tr>
<td>7.</td>
<td>Initiate a fluid challenge with a crystalloid or colloid (5 mL/kg) bolus.</td>
</tr>
<tr>
<td>8.</td>
<td>Start dopamine at 3 to 5 mcg/kg/min if no response to the crystalloid or colloid bolus occurs within 30 minutes.</td>
</tr>
<tr>
<td>9a.</td>
<td>Consider mannitol (0.5 to 1 g/kg IV) administration if no response to dopamine occurs within 30 minutes.</td>
</tr>
<tr>
<td>9b.</td>
<td>Consider furosemide (4 to 8 mg/kg IV, or 0.66 to 1 mg/kg/hr IV CRI) if no response to dopamine or mannitol occurs in 30 to 60 minutes.</td>
</tr>
<tr>
<td>10.</td>
<td>If hypertensive and oliguric or anuric, consider diltiazem 0.1 to 0.5 mg/kg IV, then 1 to 5 mcg/kg/min; must be careful to monitor for hypotension.</td>
</tr>
<tr>
<td>11.</td>
<td>If no response to furosemide, peritoneal dialysis or hemodialysis is indicated immediately, particularly if anuria is present.</td>
</tr>
</tbody>
</table>

CRI, Constant rate infusion; IV, intravenously.
Endoscopy may be useful in assessing the degree of esophageal and gastric damage, particularly after ingestion of caustic substances.

**Treatment of Specific Toxins**

**Acetaminophen (Paracetamol)**

Acetaminophen (paracetamol) is the active ingredient in Tylenol and many OTC cold products.

*Pathophysiology:* Acetaminophen is converted in the liver to N-acetyl-P-benzoquinonimine, a toxic substance that can cause oxidative injury of RBCs and hepatocytes.

*Clinical Signs:* Clinical signs of acetaminophen toxicity include respiratory distress from lack of oxygen-carrying capacity, cyanosis, methemoglobinemia (chocolate-brown appearance of the blood and mucous membranes), lethargy, vomiting, and facial and paw swelling (cats).

*Toxic Dose:* The toxic dose of acetaminophen is 100 mg/kg for dogs, and 50 mg/kg for cats.

*Treatment:* Treatment of acetaminophen toxicity includes induction of emesis or orogastric lavage if the substance has been ingested within 30 minutes. Activated charcoal should also be administered. In cases of severe anemia, give supplemental oxygen along with a packed RBC transfusion. Administer intravenous fluids to maintain renal and hepatic perfusion. *N*-acetylcysteine (140 mg/kg PO or IV once, then 70 mg/kg IV PO or IV q6h for seven treatments), vitamin C (100 mg/kg PO q8h [feline], 100 to 500 mg/kg PO q6h [canine]), and cimetidine (5 to 15 mg/kg IV, IM, SQ, PO q6-12) are the treatments of choice for methemoglobinemia in patients with acetaminophen toxicity.

**Acids and Corrosives**

*Pathophysiology:* Hydrochloric, nitric, and phosphoric acids cause chemical burns through contact with the skin and/or eyes. Localized superficial coagulative necrosis occurs on contact.

*Clinical Signs:* Usually the patient’s skin is painful to the touch or the animal may lick or chew at an irritated area that is not visible under the hair coat.

*Toxic Dose:* Toxic dose is dependent on concentration of solution that comes in contact with skin, eyes, or oral mucosa.

*Treatment:* If the chemical is swallowed, do not induce emesis or perform orogastric lavage, because of the risk of worsening esophageal irritation. Rinse the patient’s skin and eyes with warm water or warm saline for a minimum of 1⁄2 hour. Use analgesics and treat corneal ulcers (see section on corneal ulcers) as required. Do not attempt chemical neutralization, because of the risk of causing an exothermic reaction and worsening tissue injury.

**Aflatoxin**

Aflatoxin (*Aspergillus flavus*) is found in moldy feed grains; it also has been reported after ingestion of moldy bread.

*Clinical Signs:* Clinical signs of toxicity occur after ingestion and include vomiting, diarrhea, and acute hepatitis, and coagulopathy; abortion may occur in pregnant bitches.

*Toxic Dose:* LD<sub>50</sub> 0.5 to 1.5 mg/kg (canine), 0.55 mg/kg (feline)

*Treatment:* Treatment of suspected aflatoxin ingestion consists of gastric decontamination, administration of activated charcoal, intravenous fluids, and hepatic supportive care (*S*-adenosylmethionine [SAMe] (20 mg/kg PO q24h [canine], 200 mg PO q24h [feline]), milk thistle (50 to 250 mg PO q24h).

**Alcohols**

Drinking (ethanol), rubbing (isopropyl), and methyl (methanol) alcohols can be harmful if ingested.
**Pathophysiology:** Alcohols cause disruption of neuronal membrane structure, impaired motor coordination, CNS excitation followed by depression, and stupor that can lead to cardiac and respiratory arrest, depending on the amount ingested.

**Toxic Dose:** 4.1 to 8.0 g/kg PO

**Clinical Signs:** Affected animals may appear excited and then ataxic and lethargic. Contact or inhalant injury can occur, causing dermal irritation and cutaneous hyperemia. Methanol also can cause hepatotoxicity.

**Treatment:** Induce and maintain a patent airway, and stabilize the patient’s cardiovascular and respiratory status. Control CNS excitation with diazepam (0.5 to 1 mg/kg IV), if necessary, and control the patient’s body temperature (both hypothermia and hyperthermia). Induce vomiting if the patient is alert and can protect its airway; otherwise, perform orogastric lavage with the patient under general anesthesia with a cuffed endotracheal tube in place. Alcohols do not bind well with activated charcoal. Treat dermal exposure by bathing the area with warm water.

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**Alkalis and Caustics**

**Pathophysiology:** If ingested, sodium or potassium hydroxide can cause severe contact dermatitis or irritation of the gastrointestinal tract. Esophageal burns and full-thickness coagulative necrosis can occur.

**Toxic Dose:** Toxic dose is dependent on the concentration of solution that comes in contact with animal.

**Treatment:** If an animal ingests a caustic alkali substance, feed the animal four egg whites mixed with 1 quart of warmed water. Perform endoscopy within 24 hours to evaluate the extent of injury and to place a feeding tube, in severe cases. Do not induce emesis, and do not perform orogastric lavage, because of the risk of worsening esophageal irritation. In cases of contact exposure to the skin or eyes, rinse the exposed area with warm water baths for at least 30 minutes. Administer gastroprotectant, antiemetic, and analgesic drugs as necessary. Avoid neutralization, which can cause a hyperthermic reaction and worsen injury to the skin and gastrointestinal tract.

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**Amitraz**

Amitraz is the active ingredient in ascaricides and antitick and antimite products such as Mitaban and Taktic.

**Pathophysiology:** Amitraz exerts its toxic effects by causing α-adrenergic stimulation.

**Toxic Dose:** 10 to 20 mg/kg

**Clinical Signs:** Clinical signs are similar to those observed with administration of xylazine: bradycardia, CNS depression, ataxia, hypotension, hyperglycemia, hypothermia, cyanotic mucous membranes, polyuria, mydriasis, emesis, and coma.

**Treatment:** Treatment of amitraz intoxication includes cardiovascular support with intravenous crystalloid fluids and induction of emesis in asymptomatic animals. If clinical signs are present, orogastric lavage may be required. Many toxic compounds are impregnated in a collar form. If the patient has ingested a collar and does not vomit it, it should be removed using endoscopy or gastrotomy. Administer activated charcoal to prevent or delay absorption of the toxic compound. Yohimbine (0.11 mg/kg IV slowly) or atipamezole (50 mcg/kg IM), both α-adrenergic antagonists, are the treatment(s) of choice to reverse the clinical signs of toxicity. Avoid the use of atropine, because it can potentially increase the viscosity of respiratory secretions and cause gastrointestinal ileus, thus promoting increased absorption of the toxic compound.

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**Ammonia, Cleaning**

Ammonium hydroxide, or cleaning ammonia, can be caustic at high concentrations (see Alkalis and Caustics) and cause severe injury to the respiratory system if inhaled.
Pathophysiology: Severe respiratory injury, pulmonary edema, or pneumonia can occur, resulting in respiratory distress. Ingestion of ammonia can cause severe irritation to the gastrointestinal tract, vomiting, and esophageal injury.

Clinical Signs: Clinical signs are directly related to the amount and concentration ingested.

Treatment: If ammonia is ingested, administer a dilute solution of egg white.

Administer gastroprotectant, antiemetic, and analgesic drugs as necessary. If pneumonia or pulmonary edema occurs secondary to aspiration of ammonia into the airways and alveolar spaces, treatment is largely supportive with supplemental oxygen administration, antibiotics, fluid therapy, and mechanical ventilation as necessary. Diuretics may or may not be useful in the treatment of pulmonary edema secondary to ammonia inhalation.

Amphetamine

Amphetamines may be in the form of prescription medications or illicit drugs such as methamphetamine.

Pathophysiology: Amphetamines cause CNS excitation owing to neurosynaptic stimulation, resulting in hypersensitivity to noise and motion, agitation, tremors, vomiting, diarrhea, and seizures.

Clinical Signs: Clinical signs of amphetamine toxicity include muscle tremors, tachyarrhythmias, mydriasis, ptyalism, and hyperthermia.

Treatment: Amphetamines are rapidly absorbed from the gastrointestinal tract. Treatment includes administration of intravenous fluids to maintain hydration and renal perfusion and correction of hyperthermia. Administer sedative drugs such as chlorpromazine (10-18 mg/kg IV) to control agitation and tremors, and diazepam (0.5 to 1 mg/kg IV) or levetiracetam (20 mg/kg PO q8h) to control seizures. Urinary acidification can promote excretion and prevent reabsorption from the urinary bladder. In severe cases, treat cerebral edema with a combination of mannitol (0.5 to 1 g/kg IV) followed by furosemide (1 mg/kg IV) to control increased intracranial pressure.

Antifreeze See Ethylene Glycol.

Antihistamines

Antihistamines (loratadine, diphenhydramine, doxylamine, clemastine, meclizine, dimenhydrinate, chlorpheniramine, cyclizine, terfenadine, hydroxyzine) are available as OTC and prescription allergy and anti-motion sickness products.

Pathophysiology: These agents cause sedation at lower doses and agitation, tremors, and seizures at higher doses.

Clinical Signs: Clinical signs of antihistamine toxicity include restlessness, nausea, vomiting, agitation, seizures, hyperthermia, and tachyarrhythmias.

Toxic Dose: Toxic dose is dependent on the type of antihistamine ingested.

Treatment: Treatment of antihistamine intoxication is largely symptomatic and supportive, as there is no known antidote. If ingestion is recent (within 1 to 2 hours) and the patient is not actively seizing and can protect its airway, induce emesis or perform orogastric lavage, followed by administration of activated charcoal and a cathartic. Monitor the patient's heart rate, rhythm, and BP. Treat cardiac arrhythmias, if present, with appropriate therapies (see section on cardiac dysrhythmias). Administer cooling measures and intravenous fluids to treat hyperthermia. A CRI of methocarbamol (55 to 220 mg/kg IV to effect) can be used to control muscle tremors.

ANTU (α-Naphthylthiourea)

α-Naphthylthiourea (ANTU) is manufactured as a white or blue-gray powder.
**Pathophysiology:** ANTU is a gastric irritant that induces emesis if stomach is empty. The toxin is well absorbed from the stomach in the presence of food and causes increased pulmonary capillary permeability and pulmonary edema.

**Clinical Signs:** Clinical signs include vomiting, ptyalism, cough, increased respiratory rate and effort, cyanosis, respiratory difficulty, and respiratory failure. Ataxia and weakness may be observed.

**Toxic Dose:** The toxic dose in dogs is 10 to 40 mg/kg, and in cats is 75 to 100 mg/kg. Younger dogs appear to be more resistant to its toxic effects.

**Treatment:** Treatment of ANTU toxicity includes respiratory support. Mechanical ventilation may be required in severe cases of pulmonary edema. If an animal does not vomit, orogastric lavage should be performed. Administer gastrointestinal protectant, antiemetic, and analgesic drugs. Cardiovascular support in the form of intravenous crystalloids should be administered with caution, because of the risk of exacerbating increased capillary permeability and causing pulmonary edema.

**Arsenic**
Inorganic arsenic (arsenic trioxide, sodium arsenite, sodium arsenate) is the active ingredient in many herbicides, defoliants, and insecticides, including ant killers.

**Pathophysiology:** Arsenic compounds interfere with cellular respiration by combining with sulfhydryl enzymes.

**Clinical Signs:** Clinical signs of toxicity include severe gastroenteritis, muscle weakness, capillary damage, hypotension, renal failure, seizures, and death. In many cases, clinical signs are acute in onset.

**Toxic Dose:** The toxic dose of sodium arsenate is 100 to 150 mg/kg; that of sodium arsenite is 1 to 25 mg/kg. Sodium arsenite is less toxic, although cats are very susceptible.

**Treatment:** Treatment of arsenic toxicity involves procuring and maintaining a patent airway. Administer intravenous crystalloid fluids to correct hypotension and hypovolemia, and normalize acid-base and electrolyte balance. If no clinical signs are present and if the compound was ingested within 2 hours, induce emesis. If clinical signs are present, perform orogastric lavage followed by administration of activated charcoal. If dermal exposure has occurred, thoroughly bathe the animal to prevent further absorption. Dimercaprol (BAL, 3 to 4 mg/kg IM q8h) can be administered as a chelating agent.

**N-acetylcysteine (Mucomyst)** (for cats, 140 to 240 mg/kg PO IV, then 70 mg/kg PO IV q6h for 3 days; for dogs, 280 mg/kg PO or IV, then 140 mg/kg PO IV q4h for 3 days) has been shown to decrease arsenic toxicity in rats.

**Aspirin** *(Acetylsalicylic Acid, Salicylate)*

**Pathophysiology:** Aspirin causes inhibition of the production of prostaglandins, a high anion gap metabolic acidosis, gastrointestinal ulceration, hypophosphatemia, and decreased platelet aggregation when ingested in high quantities.

**Clinical Signs:** Clinical signs of aspirin toxicity include tachypnea, vomiting, anorexia, lethargy, hematemesis, and melena.

**Toxic Dose:** >50 mg/kg/24 hours in dogs; >25 mg/kg/24 hours in cats

**Treatment:** Treatment of aspirin toxicity is largely supportive. If the ingestion was recent (within the last hour), induce emesis or perform orogastric lavage followed by administration of activated charcoal. Administer intravenous crystalloid fluids to maintain hydration and correct acid-base abnormalities. Administer synthetic prostaglandin analogues (misoprostol, 5 mcg/kg PO), gastroprotectant drugs, and antiemetics. Alkalinization of the urine can enhance excretion.

**Atomoxetine** See Strattera (Selective Norepinephrine Reuptake Inhibitor).

**Baclofen** Baclofen is a GABA agonist.
**Pathophysiology:** Baclofen is a centrally acting muscle relaxant, causing increased serotonergic stimulation.

**Clinical Signs:** Clinical signs of toxicity include vomiting, ataxia, vocalization, disorientation, seizures, hypoventilation, coma, and apnea.

**Toxic Dose:** Clinical signs can occur at doses as low as 1.3 mg/kg.

**Treatment:** Treatment of baclofen ingestion includes induction of emesis if the animal is asymptomatic. Otherwise, perform orogastric lavage. Emesis or orogastric lavage should be followed by administration of activated charcoal. Perform intravenous crystalloid fluid diuresis to promote elimination of the toxin, maintain renal perfusion, and normalize body temperature. Supplemental oxygen or mechanical ventilation may be required for hypoventilation or apnea. If seizures occur, avoid the use of diazepam, which is a GABA agonist and can potentially worsen clinical signs. Control seizures with intravenous phenobarbital (bolus 2-5 mg/kg IV, can repeat every 20 minutes 2 times), pentobarbital (5 to 15 mg/kg IV slowly to effect), propofol (3 to 6 mg/kg IV, then 8 to 12 mg/kg/hr IV CRI to effect), or levetiracetam (20 mg/kg PO q8h). Supportive care (eye lubrication, urinary catheter placement for patient cleanliness, passive range-of-motion exercises, soft heavy bedding to prevent decubitus ulcer formation) is required. Clinical signs usually resolve in several days. Seizures warrant a more guarded prognosis.

**Barbecue Lighter Fluids** See Fuels.

**Barbiturates**

Phenobarbital, pentobarbital, and thiopental are used in veterinary medicine as anticonvulsants and for anesthetic induction. Accidental or iatrogenic exposure can occur.

**Pathophysiology:** Barbiturates such as phenobarbital are GABA agonists and induce CNS depression by inhibiting acetylcholine, norepinephrine, and glutamine.

**Clinical Signs:** Clinical signs of barbiturate overdose or toxicity include weakness, lethargy, hypotension, hypoventilation, stupor, coma, and death. Paradoxical reactions and agitation or excitement can occur.

**Toxic Dose:** Toxic dose is dependent on substance ingested.

**Treatment:** Treatment of barbiturate toxicity includes maintenance and support of the cardiovascular and respiratory systems. If clinical signs are absent and the patient can protect its airway, induce emesis, then administer repeated doses of activated charcoal. Perform orogastric lavage if emesis is contraindicated. Administer supplemental oxygen if hypoventilation occurs. Some animals may require mechanical ventilation. Administer intravenous fluids to control perfusion and BP. Positive inotropic drugs may be required if dose-dependent decrease in cardiac output and BP occurs. Alkalization of the urine and peritoneal dialysis can be performed to enhance excretion and elimination. Hemodialysis should be considered in severe cases, if available.

**Batteries**

Given the large number of battery-powered toys, remote control devices, and electronic hand-held personal data equipment, ingestion of batteries is commonplace in veterinary patients.

**Pathophysiology:** Automotive and dry cell batteries contain sulfuric acid, which can be irritating on contact with the eyes, skin, and gastrointestinal tract. Button batteries, which contain sodium or potassium hydroxide, cause contact irritation and sometimes esophageal perforation if chewed.

**Clinical Signs:** Clinical signs are largely dependent on the area of contact with sodium or potassium hydroxide. Dermal injury can occur in the form of erythema and blistering. Gastrointestinal signs include vomiting, hematemesis, inappetence, and melena.

**Toxic Dose:** Clinical signs are largely dependent on the amount ingested.
**Treatment:** To treat exposure, rinse the eyes and skin with copious amounts of warm tap water or sterile saline solution for a minimum of 30 minutes. If ingestion occurred, administer gastroprotectant and antiemetic drugs. Induction of emesis and orogastric lavage is absolutely contraindicated because of the risk of aspiration pneumonia and worsening esophageal irritation. No attempt should be made at performing neutralization because of the risk of causing an exothermic reaction and worsening tissue damage. Administer analgesics to control discomfort.

**Benzoyl Peroxide**

Benzoyl peroxide is the active ingredient in many OTC acne preparations.

**Pathophysiology:** Ingestion can result in production of hydrogen peroxide, gastroenteritis, and gastric dilatation. Topical exposure can cause dermal irritation and blistering.

**Clinical Signs:** Clinical signs include vomiting, abdominal distension, dermal erythema, and blistering.

**Toxic Dose:** Signs of toxicity are dependent on dose ingested.

**Treatment:** If an animal has ingested benzoyl peroxide, do not induce emesis, because of the risk of worsening esophageal irritation. Instead, perform orogastric lavage. Administer gastroprotectant and antiemetic medications and closely observe the patient for signs of gastric dilatation.

**β-Adrenergic Agonists (Asthma Inhalers and Medications)**

β-Adrenergic agonists, including terbutaline, albuterol (salbutamol), and metaproterenol, are commonly used in inhaled form for the treatment of asthma. Animals commonly are exposed to the compounds after chewing on their owners’ inhalers.

**Pathophysiology:** B1 agonist stimulation results in tachycardia; beta-2 agonist stimulation results in vasodilation, hypotension, and reflex tachycardia. Potassium shifts intracellularly and results in severe hypokalemia.

**Clinical Signs:** Clinical signs of β-adrenergic stimulation include tachycardia, muscle tremors, and agitation. Severe hypokalemia can occur.

**Toxic Dose:** In most cases, dependent on amount left in inhaler, puncture of inhaler allows entire contents to be absorbed.

**Treatment:** Treatment of β-adrenergic agonist intoxication includes treatment with β-blockers (propranolol (0.02 to 0.08 mg/kg IV slowly to effect), esmolol (0.05 to 0.1 mg/kg IV slowly to effect, then 50 to 200 mcg/kg/min IV CRI), atenolol (0.5 to 1 mg/kg PO q12h [canine], 6.25 to 12.5 mg/cat PO q12-24h [feline]), intravenous fluids, and intravenous potassium supplementation. Diazepam (0.5 to 1 mg/kg IV) or acepromazine (0.025-0.2 mg/kg IV) may be administered for sedation and muscle relaxation.

**Bismuth Subsalicylate (Pepto-Bismol)** See Aspirin.

**Bleach, Chlorine (Sodium Hypochlorite)**

Sodium hypochlorite is available in dilute (3% to 6%) or concentrated (50% industrial strength or swimming pool) solutions for a variety of purposes.

**Pathophysiology:** Sodium hypochlorite can cause severe contact irritation and tissue destruction, depending on the concentration. Affected animals may have a bleached hair coat.

**Clinical Signs:** Clinical signs include bleached hair coat, erythema, blistering, vomiting and hematemesis, ptyalism, and inappetence if ingested.

**Toxic Dose:** Clinical signs are dependent on quantity ingested. Even small quantities can cause dermal irritation and gastrointestinal disturbance.

**Treatment:** Treatment of exposure includes dilution with copious amounts of warm water or saline baths and ocular lavage. Induction of emesis and orogastric lavage is absolutely
contraindicated because of the risk of causing further esophageal irritation. To treat ingestion, give the animal milk or large amounts of water, in combination with gastroprotectant and antiemetic drugs, to dilute the contents in the stomach. Administration of sodium bicarbonate or Milk of Magnesia is no longer recommended.

**Bleach, Nonchlorine**

Nonchlorine bleaches (sodium peroxide or sodium perborate) have a moderate toxic potential if ingested.

*Pathophysiology:* Sodium peroxide can cause gastric distension. Sodium perborate can cause severe gastric irritation, with vomiting and diarrhea; renal damage and CNS excitation followed by depression can occur, depending on the amount ingested.

*Clinical Signs:* Clinical signs include vomiting, diarrhea, agitation followed by lethargy or depression, and gastric dilation.

*Toxic Dose:* Six-percent sodium hypochlorite (undiluted) can cause irritation to the skin, eyes, and orogastric mucosa.

*Treatment:* To treat dermal or ocular exposure, rinse the skin or eyes with copious amounts of warm tap water or sterile saline for a minimum of 30 minutes; treat ocular injuries as necessary, if corneal burns have occurred. If the bleach has been ingested, do induce emesis and perform orogastric lavage. Administer Milk of Magnesia (2 to 3 mL/kg).

**Boric Acid, Borate**

Boric acid is the active ingredient in many ant and roach killers.

*Pathophysiology:* Pathophysiology is unknown.

*Clinical Signs:* Clinical signs include vomiting (blue-green vomitus), blue-green stools, renal damage, and CNS excitation and depression.

*Toxic Dose:* 1 to 3 g/kg

*Treatment:* Treatment of boric acid or borate ingestion includes gastric decontamination with induction of emesis or orogastric lavage, followed by administration of a cathartic to hasten elimination. Activated charcoal is not useful to treat ingestion of this toxin. Administer intravenous fluid therapy to maintain renal perfusion. Administer gastroprotectant and antiemetic drugs as necessary.

**Botulism**

*Clostridium botulinum* endospores can be found in carrion, food, garbage, and the environment. Ingestion of endospores and *C. botulinum* endotoxin rarely can cause generalized neuromuscular blockade of spinal and cranial nerves.

*Clinical Signs:* Clinical signs include miosis, anisocoria, lower motor neuron weakness, and paralysis. Respiratory paralysis, megaesophagus, and aspiration pneumonia can occur. Clinical signs usually develop within 6 days of ingestion.

*Toxic Dose:* Ingestion of preformed botulinum toxin causes toxicity; signs dependent on amount ingested.

*Differential Diagnoses:* Differential diagnoses include acute polyradiculoneuritis (coonhound paralysis), bromethalin intoxication, and tick paralysis.

*Treatment:* Treatment of botulism is largely supportive; although an antitoxin exists, it often is of no benefit. Treatment may include administration of intravenous fluids, frequent turning of the patient and passive range-of-motion exercises to prevent disuse muscle atrophy, and supplemental oxygen administration or mechanical ventilation. Administer amoxicillin, ampicillin, or metronidazole. Recovery may be prolonged, up to 3 to 4 weeks in some cases.

**Bromethalin**

Bromethalin is the active ingredient in some brands of mouse and rat poisons. It usually is packaged as 0.01% bromethalin in green or tan pellets and is packaged in 16- to 42.5-g packs.
**Pathophysiology:** Pathophysiology involves uncoupling of oxidative phosphorylation.

**Clinical Signs:** An acute syndrome of vomiting, tremors, extensor rigidity, and seizures occurs within 24 hours of ingestion of high doses. Delayed clinical signs occur within 3 to 7 days of ingestion of a lower dose and include posterior paresis progressing to ascending paralysis, CNS depression, and coma.

**Toxic:** The toxic dose for dogs is 6.25 mg/kg (dog) and 1.8 mg/kg (cat). Bromethalin causes toxicity by uncoupling of oxidative phosphorylation. An acute syndrome of vomiting, tremors, extensor rigidity, and seizures occurs within 24 hours of ingestion of high doses. Delayed clinical signs occur within 3 to 7 days of ingestion of a lower dose and include posterior paresis progressing to ascending paralysis, CNS depression, and coma.

**Treatment:** Treatment of known bromethalin ingestion includes induction of emesis or orogastric lavage, and repeated doses of activated charcoal every 4 to 6 hours for 3 days, because bromethalin undergoes enterohepatic recirculation. Supportive care includes intravenous fluids, anticonvulsants, muscle relaxants (methocarbamol up to 220 mg/kg/day IV to effect), frequent turning of the patient, and passive range-of-motion exercises. Supplemental oxygen and/or mechanical ventilation may be required in patients with coma and severe hypoventilation. Administer mannitol (0.5 to 1 g/kg) in conjunction with furosemide (1 mg/kg IV) if cerebral edema is suspected.

**Caffeine**

The majority of caffeine toxicities occur in dogs that ingest coffee beans.

**Pathophysiology:** Caffeine causes phosphodiesterase inhibition.

**Clinical Signs:** Clinical signs include cardiac tachyarrhythmias, CNS stimulation (hyperexcitability and seizures), diuresis, gastric ulcers, vomiting, and diarrhea. Muscle tremors and seizures can occur, resulting in severe hyperthermia.

**Toxic Dose:** LD<sub>50</sub> of 140 mg/kg

**Treatment:** Treatment of caffeine toxicity is largely symptomatic and supportive, as there is no known antidote. If clinical signs are not apparent and the patient is able to protect its airway, induce emesis. Alternatively, orogastric lavage can be performed, followed by administration of activated charcoal. Administer diazepam (0.5 to 1 mg/kg IV) to control seizures. Administer β-adrenergic blockers (e.g., esmolol, 50 to 100 mcg/kg IV bolus, 50 to 200 mcg/kg/min IV CRI; propranolol, 0.04 to 0.1 mg/kg IV slowly to effect; atenolol, 0.5 to 2 mg/kg PO q12h [canine], 6.25 to 12.5 mg/cat PO q12-24h [feline]) to control tachyarrhythmias. Give intravenous fluids to maintain hydration and correct hyperthermia. The patient should be walked frequently or have a urinary catheter placed to prevent reabsorption of the toxin from the urinary bladder.

**Carbamates**

Carbamate compounds are found in agricultural and home insecticide products. Examples of carbamates include carbofuran, aldicarb, propoxur, carbaryl, and methiocarb.

**Pathophysiology:** Carbamate compounds function by causing acetylcholinesterase inhibition. Toxic amounts cause CNS excitation, muscarinic acetylcholine overload, and SLUD (salivation, lacrimation, urination, and defecation). Miosis, vomiting, and diarrhea result from muscarinic overload. Nicotinic overload produces muscle tremors. Toxicity can result in seizures, coma, and death.

**Toxic Dose:** The toxic dose of each compound varies.

**Clinical Signs:** Toxic amounts cause CNS excitation, muscarinic acetylcholine overload, and SLUD (salivation, lacrimation, urination, and defecation). Miosis, vomiting, and diarrhea result from muscarinic overload. Nicotinic overload produces muscle tremors. Toxicity can result in seizures, coma, and death.

**Treatment:** Treatment of carbamate intoxication includes maintaining an airway and, if necessary, using artificial ventilation. Administer intravenous crystalloid fluids to control
the patient’s hydration, BP, and temperature. Cooling measures may be warranted. Induce emesis if the substance was ingested within 60 minutes and the animal is asymptomatic. Give repeated doses of activated charcoal if the animal can swallow and protect its airway. Control seizures with diazepam (0.5 mg/kg IV). Bathe the patient thoroughly. Atropine (0.2 mg/kg IV) is useful in controlling some of the muscarinic signs associated with the toxicity. Pralidoxime hydrochloride (2-PAM) is not useful in cases of carbamate intoxication. Control muscle tremors with methocarbamol (up to 220 mg/kg IV) or guaifenesin (110 mg/kg IV).

Carbon Tetrachloride
In humans, ingestion or inhalation of 3 to 5 mL of carbon tetrachloride can be fatal. The prognosis is grave. *Clinical Signs:* Clinical signs of carbon tetrachloride toxicity include vomiting and diarrhea, then progressive respiratory and CNS depression. Ventricular dysrhythmias and hepatorenal damage ensue. *Toxic Dose:* Toxic at very small quantities. *Treatment:* Treatment of carbon tetrachloride inhalation includes procurement and maintenance of a patent airway with supplemental oxygen, and cardiovascular support. To treat ingestion, administer activated charcoal, and give intravenous fluids to maintain hydration and support renal function.

Chlorinated Hydrocarbons
Chlorinated hydrocarbons include dichlorodiphenyltrichloroethane (DDT), methoxychlor, lindane, dieldrin, aldrin, chlordane, chlorecone, Perthane, toxaphene, heptachlor, mirex, and endosulfan. *Pathophysiology:* Chlorinated hydrocarbons exert their toxic effects by an unknown mechanism and can be absorbed through the skin and the gastrointestinal tract. *Toxic Dose:* The toxic dose of each compound varies. *Clinical Signs:* Clinical signs are similar to those observed in organophosphate toxicity: CNS excitation, seizures, SLUD (salivation, lacrimation, urination, defecation), excessive bronchial secretions, vomiting, diarrhea, muscle tremors, and respiratory paralysis. Secondary toxicity from toxic metabolites can cause renal and hepatic failure. Chronic exposure may cause anorexia, vomiting, weight loss, tremors, seizures, and hepatic failure. The clinical course can be prolonged in small animal patients. *Treatment:* Treatment of chlorinated hydrocarbon toxicity is largely supportive in nature, as there is no known antidote. Procure and maintain the patient’s airway. Normalize the body temperature to prevent hyperthermia. If the substance was just ingested and the patient is not demonstrating any clinical signs, induce emesis. If the patient is symptomatic, perform orogastric lavage followed by activated charcoal administration. Bathe the patient thoroughly in cases of topical exposure. Administer intravenous crystalloid fluids to maintain hydration. These compounds do not appear to be amenable to fluid diuresis.

Chlorphenoxy Herbicides
Chlorphenoxy derivatives are found in 2,4-diphenoxycetic acid (2,4-D); 2,4,5-trichlorophenoxyactic acid (2,4,5-T); 2-methyl-4-chlorophenoxyactic acid (MCPA); meta-chlorophenylpiperazine (mCPP); and Silvex. *Pathophysiology:* Chlorphenoxy derivatives exert their toxic effects by an unknown mechanism. *Clinical Signs:* Gastroenteritis (vomiting, diarrhea) and muscle rigidity. *Toxic Dose:* The LD₅₀ of 2,4-D is 100 mg/kg; however, the toxic dose appears to be much lower in small animal patients.
Treatment: Treatment of chlorphenoxy derivative toxicity is largely supportive in nature, as there is no known antidote. Secure the patient’s airway and administer supplemental oxygen as necessary. Control CNS excitation with diazepam (0.5 mg/kg IV). Intravenous crystalloid fluid diuresis and urinary alkalinization can promote elimination. Administer gastroprotectant and antiemetic drugs as needed.

Chocolate
Pathophysiology: The toxic effects of chocolate are related to theobromine and interference with or inhibition of phosphodiesterase.
Clinical Signs: Clinical signs include CNS stimulation (tremors, anxiety, seizures), myocardial stimulation (tachycardia and tachyarrhythmias), diuresis, and (at very high doses) gastrointestinal ulceration. Potential side effects include gastroenteritis and pancreatitis owing to the fat content of the chocolate.
Toxic Dose: Various types of chocolate have different concentrations of theobromine and thus can cause clinical signs of toxicity with ingestion of varying amounts of chocolate, depending on the type. The toxic dose of theobromine is 100 to 150 mg/kg in dogs. Milk chocolate contains 44 mg/oz (154 mg/100 g) of chocolate and has a low toxic potential. Semisweet chocolate contains 150 mg/oz (528 mg/100 g), and baking chocolate contains 390 mg/oz (1365 mg/100 g). Semisweet and baking chocolate, being the most concentrated, have a moderate to severe toxic potential, even in large dogs.
Treatment: Treatment of chocolate toxicity includes obtaining and maintaining a protected airway (if necessary), administering intravenous fluid diuresis, inducing emesis or performing orogastric lavage followed by administration of repeated doses of activated charcoal, and placing a urinary catheter to prevent reabsorption of the toxin from the urinary bladder. With treatment, the condition of most dogs returns to normal within 12 to 24 hours (t₁/₂ = 17.5 hours in dogs).

Cholecalciferol
Cholecalciferol is found in some rodenticides and also as active vitamin D in dietary or vitamin supplements (OTC and prescription).
Pathophysiology: Increased intestinal and renal reabsorption of calcium occurs, causing an increase in serum calcium and dystrophic mineralization of the kidneys and liver.
Clinical Signs: Clinical signs include lethargy, anorexia, vomiting, constipation, and renal pain within 2 to 3 days of ingestion. Seizures, muscle twitching, and CNS depression may be observed at very high doses. As renal failure progresses, PU/PD, vomiting and hematemesis, uremic oral ulcers, and melena may be observed.
Toxic Dose: 2 to 3 mg/kg
Treatment: If the compound was ingested recently (within 2 to 4 hours) induce emesis or perform orogastric lavage, followed by administration of activated charcoal. Check the patient’s serum calcium once daily for 3 days after ingestion. If clinical signs of toxicity or hypercalcemia are present, decrease serum calcium with loop diuretics (furosemide, 2 to 5 mg/kg PO or IV q12h) and glucocorticosteroids (prednisone or prednisolone, 2 to 3 mg/kg PO bid) to promote renal calcium excretion. In severe cases, salmon calcitonin (4 to 6 IU per kg SQ q2-12h in dogs) or bisphosphonate compounds (pamidronate 1 to 2 mg/kg in 150 mL 0.9% saline, administered IV over 2 hours) may be required. Correct acid-base abnormalities with intravenous crystalloid fluid diuresis and sodium bicarbonate, if necessary. (See section on hypercalcemia.)

Coal, Tar-Based See Hydrocarbons, Aromatic.

Coumarins See Vitamin K–Antagonist Rodenticides.
Cresol See Hydrocarbons, Aromatic.

Deicers See Ethylene Glycol and Alcohols.

Denture Cleaners
Denture cleaners contain sodium perborate as the active compound.  
Pathophysiology: Sodium perborate can cause severe direct irritation of the mucous membranes and may also act as a CNS depressant.  
Clinical Signs: Clinical signs are similar to those seen if bleach or boric acid compound is ingested, namely vomiting, diarrhea, CNS excitation then depression, and renal failure.  
Treatment: Treatment for ingestion of denture cleaner includes gastric decontamination along with induction of emesis or orogastric lavage and administration of a cathartic to hasten elimination. Activated charcoal is not useful for treatment of ingestion of this toxin. Administer gastroprotectant and antiemetic drugs as necessary.

Deodorants
Deodorants are usually composed of aluminum chloride and aluminum chlorohydrate. Both have a moderate potential for toxicity.  
Pathophysiology: Contact irritation occurs.  
Clinical Signs: Ingestion of deodorant compounds can cause oral irritation or necrosis, gastroenteritis, and nephrosis.  
Treatment: Treatment of deodorant ingestion includes orogastric lavage and administration of antiemetic and gastroprotectant drugs.

Detergents, Anionic
Anionic detergents include sulfonated or phosphorylated forms of benzene.  
Pathophysiology: Pathophysiology involves denaturation of proteins and contact irritation.  
Clinical Signs: Anionic detergents cause significant mucosal damage and edema, gastrointestinal irritation, CNS depression, seizures, and possible hemolysis. Ocular exposure can cause corneal ulcers and edema.  
Toxic Dose: Dishwashing liquid is an example of an anionic detergent that can be toxic at doses of 1 to 5 g/kg.  
Treatment: Treatment of anionic detergent exposure is largely symptomatic, as there is no known antidote. To treat topical toxicity, flush the patient’s eyes and skin with warmed tap water or 0.9% saline solution for a minimum of 30 minutes, taking care to avoid hypothermia. To treat ingestion, feed the patient milk and large amounts of water to dilute the toxin. Do not induce emesis, because of the risk of worsening esophageal irritation. To dilute the toxin, perform orogastric lavage, followed by administration of activated charcoal. Closely monitor the patient’s respiratory status, because oropharyngeal edema can be severe. If necessary, perform endotracheal intubation in cases of airway obstruction. Monitor the patient for signs of intravascular hemolysis. Administer intravenous crystalloid fluids to maintain hydration until the patient is able to tolerate oral fluids.

Detergents, Cationic, and Disinfectants
Cationic detergents and disinfectants include quaternary ammonia compounds, isopropyl alcohol, and isopropanol.  
Pathophysiology: Quaternary ammonia compounds have a serious toxic potential and cause severe irritation and corrosion of the mucous membranes and skin.  
Clinical Signs: Some compounds also can cause clinical signs similar to those observed with anticholinesterase compounds, including muscle tremors, seizures, paralysis, and coma. Methemoglobinemia can occur.
Treatment: Treatment of cationic detergent exposure includes careful bathing and ocular rinsing of the patient for a minimum of 30 minutes, taking care to avoid hypotension. Secure the patient’s airway and monitor the patient’s respiratory status. Administer supplemental oxygen, if necessary. Place an intravenous catheter and administer intravenous crystalloid fluids to maintain hydration. Do not induce emesis, because of the risk of causing further esophageal irritation. Give milk or large amounts of water PO, as tolerated by the patient, to dilute the toxin.

Detergents, Nonionic
Nonionic detergents include alkyl and aryl polyether sulfates, alcohols, and sulfonates; alkyl phenol; polyethylene glycol; and phenol compounds. Phenols are particularly toxic in cats and puppies.
Pathophysiology: Some compounds can be metabolized to glycolic and oxalic acid, causing renal damage similar to that observed with ethylene glycol toxicity.
Clinical Signs: Clinical signs of exposure include severe gastroenteritis and topical irritation. Some compounds can be metabolized to glycolic and oxalic acid, causing renal damage similar to that observed with ethylene glycol toxicity.
Treatment: Topical and ocular exposure should be treated with careful bathing or ocular irrigation for at least 30 minutes. Administer activated charcoal to prevent absorption of the compound. As tolerated, give dilute milk or straight tap water PO to dilute the compound. Administer antiemetic and gastroprotectant drugs to control vomiting and decrease gastrointestinal irritation. Administer intravenous crystalloid fluids to maintain hydration and decrease the potential for renal tubular damage. Monitor the patient’s acid-base and electrolyte status and correct any abnormalities with appropriate intravenous fluid therapy.

Dichlone
Dichlone (Phygon) is a dipyridyl compound that is a CNS depressant.
Pathophysiology: Dichlone reacts with thiol enzymes to cause methemoglobinemia and hepatorenal damage.
Clinical Signs: Clinical signs include CNS depression, somnolence, PU/PD, then vomiting and uremic ulceration.
Toxic Dose: The LD₅₀ in rats is 25 to 50 mg/kg.
Treatment: To treat dichlone ingestion, induce emesis or perform orogastric lavage, followed by administration of activated charcoal and a cathartic. Procure and maintain a patent airway. Perform intravenous fluid diuresis to maintain renal perfusion. N-acetylcysteine (140 mg/kg PO or IV once then 70 mg/kg PO or IV q6h for seven treatments) may be useful in the treatment of methemoglobinemia.

Diethyltoluamide (DEET)
Diethyltoluamide (DEET) is the active ingredient in many insect repellants (e.g., Off, Cutters, Hartz Blockade).
Pathophysiology: The mechanism of action of DEET is not fully understood, but it acts as a lipophilic neurotoxin within 5 to 10 minutes of exposure. Cats appear to be particularly sensitive to DEET.
Clinical Signs: Clinical signs of toxicity include aimless gazing, hypersalivation, chewing motions, and muscle tremors that progress to seizures. Recumbency and death can occur within 30 minutes of exposure at high doses.
Toxic Dose: A lethal dermal dose is 1.8 g/kg; if ingested, the lethal dose is much less. The toxic dose of dermal exposure in dogs is 7 g/kg.
Treatment: Treatment of DEET toxicity is largely supportive, as there are no known antidotes. Procure and maintain a patent airway and perform mechanical ventilation, if necessary. Place an intravenous catheter and administer intravenous crystalloid fluids to
control hydration and treat hypotension as necessary. Treat seizures with diazepam (0.5 mg/kg IV) or phenobarbital. Because of the rapid onset of clinical signs, induction of emesis is contraindicated. Perform orogastric lavage if the compound was ingested within the last 2 hours. Administer multiple repeated doses of activated charcoal. Cooling measures should be implemented to control hyperthermia. If dermal exposure has occurred, bathe the patient thoroughly to avoid further exposure and absorption.

**Diquat**

Diquat is a dipyridyl compound that is the active ingredient in some herbicide compounds.  

*Pathophysiology:* Like paraquat, diquat induces its toxic effects by causing the production of oxygen-derived free radical species.  

*Clinical Signs:* Clinical signs of diquat intoxication include anorexia, vomiting, diarrhea, and acute renal failure. Massive dehydration and electrolyte imbalances can occur as a result of fluid loss into the gastrointestinal tract.  

*Toxic Dose:* The LD$_{50}$ of diquat is 25 to 50 mg/kg.  

*Treatment:* Treatment of diquat intoxication is similar to that for paraquat ingestion. If the animal ingested diquat within 1 hour of presentation, induce emesis. In clinical cases, orogastric lavage may be required. Both emesis and orogastric lavage should be followed by administration of kaolin or bentonite as an adsorbent, rather than activated charcoal. Place an intravenous catheter and administer crystalloid fluids to restore volume status and maintain renal perfusion. Monitor urine output. If oliguria or anuria occurs, treatment with mannitol, furosemide, and dopamine may be considered.

**Ecstasy**

Ecstasy (3,4-methylenedioxymethamphetamine; MDMA) is a recreational drug used by humans.  

*Pathophysiology:* Ecstasy causes release of serotonin. A urine drug screening test can be used to detect the presence of MDMA.  

*Toxic Dose:* Clinical signs occur at 9 mg/kg; death occurs at doses greater than 15 mg/kg (canine).  

*Clinical Signs:* Clinical signs of intoxication are related to the serotonin syndrome (excitation, hyperthermia, tremors, and hypertension), and seizures may be observed.  

*Treatment:* Treatment of ecstasy intoxication is largely supportive, as there is no known antidote. Administer intravenous fluids to maintain hydration, correct acid-base status, and treat hyperthermia. Serotonin antagonist drugs (cyproheptadine 1.1 mg/kg PO in dogs—repeat in 4-6 hours until signs resolve; 2-4 mg PO per cat—repeat in 4-6 hours until signs resolve) can be dissolved and administered per rectum to alleviate clinical signs. Intravenous propranolol (0.05 to 0.08 mg/kg IV to effect) has additional antiserotonin effects. Administer diazepam (0.5 to 2 mg/kg IV) to control seizures. If cerebral edema is suspected, administer mannitol (0.5 to 1 mg/kg IV), followed by furosemide (1 mg/kg IV).

**Ethylene Glycol**

Ethylene glycol is most commonly found in antifreeze solutions but is also in some paints, photography developer solutions, and windshield wiper fluid.  

*Pathophysiology:* Ethylene glycol in itself is only minimally toxic. However, when it is metabolized to glycolate, glyoxal, glyoxylate, and oxalate, the metabolites cause an increased anion gap metabolic acidosis and precipitation of calcium oxalate crystals in the renal tubules, renal failure, and (ultimately) death.  

*Testing:* Colorimetric tests that can be performed in most veterinary hospitals can detect larger quantities of ethylene glycol in the patient’s serum. In a dog with clinical signs of ethylene glycol intoxication and renal impairment or failure, a negative result of a test for the presence of calcium oxalate crystalluria means that there is no more ethylene glycol in
the patient’s serum because it has all been metabolized. Cats are very sensitive to the toxic effects of ethylene glycol. In many cases a cat may have ingested a toxic dose, but because the sensitivity of the assay is low, test results will be negative. Lack of treatment can result in death.

**Clinical Signs:** There are three phases of ethylene glycol intoxication. In the first 1 to 12 hours after ingestion (stage I), the patient may appear lethargic, disoriented, and ataxic. In stage II (12 to 24 hours after ingestion), the patient improves and appears clinically normal. In stage III (24 to 72 hours after ingestion), the patient demonstrates clinical signs of renal failure (PU/PD) that progress to uremic renal failure (vomiting, lethargy, oral ulceration). Finally, seizures, coma, and death occur.

**Toxic Dose:** The toxic dose in dogs is 6.6 mL/kg, and in cats is 1.5 mL/kg. The toxin is absorbed quite readily from the gastrointestinal tract and can be detected in the patient’s serum within an hour of ingestion.

**Treatment:** Begin treatment of known ethylene glycol ingestion immediately. Induce emesis or perform orogastric lavage and administer repeated doses of activated charcoal. Place an intravenous catheter and perform crystalloid fluid diuresis with a known antidote. The treatment of choice for dogs and cats is administration of 4-methylpyrazole (4-MP), which directly inhibits alcohol dehydrogenase, thus preventing the conversion of ethylene glycol to its toxic metabolites. The doses for dogs and cats differ. For dogs, the initial dose is 20 mg/kg IV, followed by 15 mg/kg at 12 and 24 hours and 5 mg/kg at 36 hours. In cats, treatment is with 4-MP at 6.25 the dose for dogs (that is, 125 mg/kg IV once, then 31.25 mg/kg IV at 12, 24, and 36 hours after ingestion). 4-MP is effective only if administered within 3 hours of ingestion of ethylene glycol.

Cats will demonstrate signs of sedation and hypothermia with this treatment. If 4-MP is not available, administer ethanol (600 mg/kg IV loading dose, followed by 100 mg/kg/hr, or as a 20% solution [for dogs, 5.5 mL/kg IV q4h for five treatments, then q6h for five more treatments; for cats, 5 mL/kg q8h for four treatments]). Grain alcohol (190 proof) contains approximately 715 mg of ethanol per milliliter. Antiemetics and gastroprotective agents should be considered. Urinary alkalinization and peritoneal dialysis may enhance the elimination of ethylene glycol and its metabolites.

**Fertilizers**

Many fertilizers are on the market and may be composed of urea or ammonium salts, phosphates, nitrates, potash, and metal salts. Fertilizers have a moderate toxic potential, depending on the type and amount ingested.

**Pathophysiology:** Fertilizers are a contact irritant, causing oxidative damage to RBCs and hemoglobin, forming methemoglobin.

**Clinical Signs:** Clinical signs of fertilizer ingestion include vomiting, diarrhea, metabolic acidosis, and diuresis. Nitrates or nitrites can cause formation of methemoglobin and chocolate-brown blood. Electrolyte disturbances include hyperkalemia, hyperphosphatemia, hyperammonemia, and hyperosmolality.

**Toxic Dose:** Toxic dose is dependent on type of fertilizer ingested.

**Treatment:** Treatment of fertilizer ingestion includes cardiovascular support and administration of milk or a mixture of egg whites and water, followed by induction of emesis or orogastric lavage. Correct electrolyte abnormalities as they occur (see section on hyperkalemia). Administer antiemetic and gastroprotectant drugs, as necessary. Administer intravenous fluids to control hydration and maintain BP. N-acetylcysteine may be useful if methemoglobinemia is present.

**Fipronil**

Fipronil is the active ingredient in Frontline, a flea-control product; it is also found in household termite-control products.

**Pathophysiology:** Fipronil exerts its effects by GABA antagonism and can cause CNS excitation.
**Clinical Signs:** Clinical signs include muscle fasciculations, tremors, and seizures.

**Toxic Dose:** >0.3 mg/kg/day

**Treatment:** Treatment of fipronil toxicity includes treatment of CNS excitation, treatment of hyperthermia by cooling measures, and administration of activated charcoal.

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**Fire Extinguisher (Liquid)**

Fire extinguisher fluid contains chlorobromomethane or methyl bromide, both of which have a serious toxic potential.

**Pathophysiology:** Fire extinguisher fluid is a contact irritant. When it is ingested, the compounds can be converted to methanol and cause high anion gap metabolic acidosis.

**Clinical Signs:** Signs attributable to dermal or ocular irritation can occur. If ingested, the compounds can be converted to methanol and cause high anion gap metabolic acidosis, CNS excitation and depression, aspiration pneumonitis, and hepatorenal damage.

**Treatment:** To treat ocular or dermal exposure to fire extinguisher fluids, flush the eyes or skin with warmed tap water or 0.9% saline solution for a minimum of 30 minutes. Do not induce emesis or perform orogastric lavage to treat ingestion, because of the risk of causing severe aspiration pneumonitis. Gastroprotectant and antiemetic drugs may be used, if indicated. Administer intravenous fluids to maintain hydration and renal perfusion. Supplemental oxygen or mechanical ventilation may be required in severe cases of aspiration pneumonitis.

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**Fireplace Colors**

Fireplace colors contain salts of heavy metals—namely, copper rubidium, cesium, lead, arsenic, antimony, barium, selenium, and zinc—all of which have moderate toxic potential, depending on the amount ingested and the size of the patient.

**Clinical Signs:** Clinical signs are largely associated with gastrointestinal irritation (vomiting, diarrhea, anorexia). Zinc toxicity can cause intravascular hemolysis and hepatorenal damage.

**Treatment:** To treat ingestion of fireplace colors, administer cathartics, activated charcoal, and gastroprotectant and antiemetic drugs. Place an intravenous catheter for intravenous crystalloid fluid administration to maintain hydration and renal perfusion. Specific chelating agents may be useful in hastening elimination of the heavy metals.

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**Fireworks**

Fireworks contain oxidizing agents (nitrates and chlorates) and metals (mercury, copper, strontium, barium, and phosphorus).

**Pathophysiology:** Fireworks are a contact irritant.

**Clinical Signs:** Ingestion of fireworks can cause HGE and methemoglobinemia.

**Treatment:** To treat firework ingestion, induce emesis or perform orogastric lavage and administer activated charcoal. Administer specific chelating drugs if the amount and type of metal are known, and administer gastroprotectant and antiemetic drugs. If methemoglobinemia occurs, administer N-acetylcysteine; a blood transfusion may be necessary.

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**Fuels**

Fuels such as barbecue lighter fluid, gasoline, kerosene, and oils (mineral, fuel, lubricating) are petroleum distillate products.

**Pathophysiology:** Fuels have low toxic potential if ingested but can cause severe aspiration pneumonitis if as little as 1 mL is inhaled into the tracheobronchial tree.

**Clinical Signs:** CNS depression, mucosal damage, hepatorenal insufficiency, seizures, and corneal irritation can occur.

**Treatment:** If fuels are ingested, administer gastroprotectant and antiemetics drugs. Do not induce emesis or perform orogastric lavage, because of the risk of aspiration pneumonia. To
treat topical exposure, rinse the skin and eyes copiously with warm tap water or 0.9% saline solution. Administer antiemetic and gastroprotectant drugs, as necessary. Administer intravenous fluids to maintain hydration and treat acid-base and electrolyte abnormalities.

**Furniture Polish** See Fuels.

**Gasolines** See Fuels.

**Glue, Children’s**
Children’s glue contains polyvinyl acetate.
*Pathophysiology:* Children’s glue has a very low toxic potential. If inhaled, the compound can cause pneumonitis.
*Clinical Signs:* Clinical signs are attributable to pneumonitis: coughing, increased respiratory effort.
*Treatment:* Treatment for polyvinyl acetate exposure should be performed as clinical signs of pneumonitis (increased respiratory effort, cough, lethargy, respiratory distress) occur.

**Glue, Superglue**
Superglue contains methyl-2-cyanoacrylate.
*Pathophysiology:* Superglue is a contact irritant and causes dermal irritation.
*Clinical Signs:* Clinical signs include dermal irritation, glue stuck on fur, and erythema.
*Toxic Dose:* Toxic dose is dependent on quantity to which patient is exposed.
*Treatment:* Do not induce emesis. Do not bathe the animal, and do not apply other compounds (acetone, turpentine) in an attempt to remove the glue from the skin. The fur can be shaved, using care to avoid damaging the underlying skin. The affected area should be allowed to exfoliate naturally.

**Gorilla Glue**
Gorilla Glue, or Elmer’s Probond, contains the compound diphenylmethane diisocyanate, which, when ingested and exposed to moisture in the stomach and esophagus, expands to more than four times its original volume, then solidifies. The result is that the glue can adhere to the esophagus and stomach and cause obstruction.
*Pathophysiology:* Pathophysiology involves physical expansion and then adherence of glue product to esophagus and stomach, causing a mechanical obstruction to outflow.
*Clinical Signs:* Clinical signs include ptyalism, vomiting, inappetence, lethargy, abdominal pain, abdominal distension, and hematemesis.
*Toxic Dose:* Any ingestion can result in esophageal foreign body.
*Treatment:* Treatment involves physical (surgical) removal of glue from esophagus and/or stomach.

**Glyophosate**
Glyphosate is an herbicide found in Roundup and Kleenup.
*Pathophysiology:* If applied properly, the product has a very low toxic potential.
*Clinical Signs:* Clinical signs of toxicity include dermal and gastric irritation, including dermal erythema, anorexia, and vomiting. CNS depression can occur.
*Treatment:* Treatment includes thorough bathing in cases of dermal exposure, and induction of emesis or orogastric lavage followed by administration of activated charcoal. Administer antiemetic and gastroprotectant drugs as necessary. Administer intravenous crystalloid fluids to prevent dehydration secondary to vomiting.

**Grapes and Raisins**
Grapes and raisins have been reported to cause renal failure in some dogs.
Pathophysiology: The toxic principle and mechanism of toxicity is unknown. Clinical Signs: Clinical signs occur within 24 hours of ingestion of raisins or grapes and include vomiting, anorexia, lethargy, and diarrhea (often with visible raisins or grapes in the fecal matter). Within 48 hours, dogs demonstrate signs of acute renal failure (PU/PD, vomiting) that can progress to anuria.

Toxic Dose: The toxic dose is unknown.

Treatment: To treat known ingestion of raisins or grapes, induce emesis or perform orogastric lavage, followed by repeated doses of activated charcoal. If clinical signs of vomiting and diarrhea are present, administer intravenous fluids and monitor urine output. Aggressive intravenous fluid therapy, in conjunction with maintenance of renal perfusion, is necessary. In cases of anuric renal failure, dopamine (1 to 3 mcg/kg/min IV CRI), furosemide (4 to 8 mg/kg or 0.7 to 1 mg/kg/hr IV CRI), and mannitol (0.5 to 1 g/kg IV) can be useful in increasing urine output. Peritoneal dialysis or hemodialysis may be necessary in cases of severe oliguric or anuric renal failure. Calcium channel blockers such as amlodipine (0.1 to 0.4 mg/kg PO q24h [canine] and 0.625 to 1.25 mg/cat q24h [feline]) and diltiazem (0.1 to 0.25 mg/kg IV slowly to effect, then 2 to 6 mcg/kg/min CRI) can be used to treat systemic hypertension. Supportive care includes treatment of hyperkalemia and administration of gastroprotectant and antiemetic drugs and (if the animal is eating) phosphate binders.

Hashish See Marijuana (Cannabis sativa).

Hexachlorophene See Detergents, Nonionic.

Hydrocarbons, Aromatic
Aromatic hydrocarbons include phenols, cresols, toluene, and naphthalene. All have a moderate toxic potential if ingested.

Pathophysiology: Oxidative damage to red blood cells and hepatocytes.

Clinical Signs: Clinical signs include CNS depression, hepatorenal damage, muscle tremors, pneumonia, methemoglobinemia, and intravascular hemolysis.

Treatment: If an aromatic hydrocarbon is ingested, do not induce emesis, because of the risk of aspiration pneumonia. A dilute milk solution or water can be administered to dilute the compound. Perform orogastric lavage. Carefully monitor the patient’s respiratory and cardiovascular status. Administer supplemental oxygen if aspiration pneumonia is present. To treat topical exposure, thoroughly rinse the eyes and skin with copious amounts of warm tap water or 0.9% saline solution.

Ibuprofen See Nonsteroidal Antiinflammatory Drugs.

Imidacloprid
Imidacloprid is the compound used in the flea product Advantage.

Pathophysiology: Clinical signs of toxicity are related to nicotinic cholinergic stimulation.

Clinical Signs: Clinical signs include neuromuscular excitation followed by collapse. The compound may induce respiratory paralysis.

Toxic Dose: Even small amounts can be toxic if used on cats

Treatment: To treat imidacloprid toxicity, procure and maintain a patent airway with supplemental oxygen administration. Control CNS excitation with diazepam (0.5 to 1 mg/kg IV), phenobarbital (10 to 20 mg/kg IV slowly), or propofol (3 to 6 mg/kg IV, then 0.1-0.6 mg/kg/min IV CRI to effect). Administer enemas to hasten gastrointestinal elimination, and administer activated charcoal. Bathe the animal thoroughly to prevent further dermal absorption. Closely monitor the patient’s oxygenation and ventilation status. If severe hypoventilation or respiratory paralysis occurs, initiate mechanical ventilation.
Iron and Iron Salts

Lawn fertilizers are a common source of iron salts.  
**Pathophysiology:** Pathophysiology involves gastroenteritis and myocardial toxicity.  
**Clinical Signs:** Clinical signs include vomiting, hematemesis, lethargy, and inappetence.  
**Toxic Dose:** Iron and iron salts can cause severe gastroenteritis, myocardial toxicity, and hepatic damage if high enough doses are ingested.  
**Treatment:** Treatment of ingestion of iron and iron salts includes cardiovascular support in the form of intravenous fluids and antiarrhythmic drugs, as needed. Induce emesis or perform orogastric lavage for gastric decontamination. A cathartic can be administered to promote elimination from the gastrointestinal tract. Antiemetic and gastroprotectant drugs should be administered to prevent nausea and vomiting. In some cases, radiographs can aid in making a diagnosis of whether the compound was actually ingested. Iron toxicity can be treated with the chelating agent deferoxamine.

Ivermectin

Ivermectin is a GABA agonist that is used in commercial heartworm prevention and anthelminthic compounds and can be toxic in predisposed breeds, including Collies, Collie crosses, Old English Sheepdogs, and some Terriers.  
**Pathophysiology:** Ivermectin is a GABA neurotransmitter agonist in susceptible animals (hereditable mdr1 gene defect allows ivermectin to cross blood-brain barrier).  
**Clinical Signs:** Clinical signs of ivermectin toxicity include vomiting, ataxia, hypersalivation, agitation, tremors, hyperactivity, hyperthermia, hypoventilation, coma, seizures, signs of circulatory shock, bradycardia, and death. Clinical signs often occur within 2 to 24 hours after ingestion or iatrogenic overdose. Blood ivermectin levels can be measured, but diagnosis is often made based on clinical signs and knowledge of exposure in predisposed breeds. There is no known antidote. The clinical course can be prolonged for weeks to months before recovery occurs.  
**Treatment:** To treat known exposure, induce emesis or perform orogastric lavage if the substance was ingested within 1 hour of presentation and the patient is not symptomatic. Administer activated charcoal. Control seizures with phenobarbital (10 to 20 mg/kg IV slowly), pentobarbital (5 to 15 mg/kg IV slowly to effect), or propofol (3 to 6 mg/kg IV, then 0.1-0.6 mg/kg/min IV CRI to effect). Diazepam, which potentially can worsen central nervous system stimulation, is contraindicated. Administer intravenous fluids to maintain perfusion and hydration, and treat hyperthermia. Supportive care may be necessary, including supplemental oxygen (or mechanical ventilation, if necessary), frequent turning of the patient and passive range-of-motion exercises, placement of a urinary catheter to maintain patient cleanliness and monitor urine output, lubrication of the eyes, and parenteral nutrition (see Rule of 20). Specific antidotes used to treat ivermectin toxicity include physostigmine and picrotoxin. Physostigmine therapy was beneficial in some patients for a short period; picrotoxin caused severe violent seizures and therefore should be avoided.

Kerosene  See Fuels.

Lead

Lead is ubiquitous and is found in some paints, car batteries, fishing equipment (sinkers), and plumbing materials.  
**Pathophysiology:** Lead causes toxicity by inhibiting sulfur-containing enzymes, leading to increased RBC fragility and CNS damage.  
**Clinical Signs:** Clinical signs of hyperexcitability, dementia, vocalization, seizures, and lower motor neuron polyneuropathy can occur. Affected animals may appear blind, or vomiting, anorexia, and constipation or diarrhea may occur.  
**Toxic Dose:** Lead can be toxic at doses of 3 mg/kg. If ingestion of more than 10 to 25 mg of lead per kilogram occurs, death can result.
Testing: If lead toxicity is suspected, blood and urine lead levels can be measured.

Treatment: Treatment of lead toxicity is supportive and is directed at treatment of clinical signs. Control seizures with diazepam or phenobarbital. If cerebral edema is present, administer mannitol (0.5 to 1.0 g/kg IV), followed by furosemide (1 mg/kg IV 20 minutes after mannitol). Sodium or magnesium sulfate should be administered as a cathartic. Initiate chelation therapy with dimercaprol (2 to 5 mg/kg IM q4h for 2 days, then 2 to 5 mg/kg IM q8h on Day 3, then 2 to 5 mg/kg IM q12h for 10 days) or penicillamine (10 to 15 mg/kg PO q12h). If a lead object is identified in the gastrointestinal tract on radiographs, remove the object using endoscopy or exploratory laparotomy.

**d-Limonene, Linalool**

_d-Limonene_ and _linalool_ are components of citrus oil extracts used in some flea-control products.

*Clinical Signs:* Clinical signs of toxicity include hypersalivation, muscle tremors, ataxia, and hypothermia.

*Toxic Dose:* The toxic dose is unknown, but cats appear to be very sensitive to exposure.

*Treatment:* Treatment of _d-Limonene_ and _linalool_ exposure includes treatment of hypothermia, administration of activated charcoal to prevent further absorption, and careful, thorough bathing to prevent further dermal exposure.

**Loperamide**

Loperamide is an opioid derivative that is used to treat diarrhea.

*Pathophysiology:* Loperamide has opioid effects on the GI tract and causes CNS signs at central opioid receptors at higher doses.

*Clinical Signs:* Clinical signs of loperamide intoxication include constipation, ataxia, nausea, and sedation; vomiting and cramping may occur.

*Toxic Dose:* >0.6 mg/kg—vomiting, cramping; >1.25 mg/kg—ataxia and CNS depression; >5 mg/kg—hemorrhagic enteritis and posterior paresis or paralysis

*Treatment:* Induce emesis or perform orogastric lavage, followed by administration of activated charcoal and a cathartic. Naloxone may be beneficial in the temporary reversal of ataxia and sedation.

**Macadamia Nuts**

Ingestion of even small quantities of macadamia nuts can be toxic to some dogs. Macadamia nuts are often covered with chocolate, so a combination of macadamia nut and theobromine toxicity may be present.

*Pathophysiology:* The toxic principle in macadamia nuts is unknown.

*Clinical Signs:* Ingestion of macadamia nuts can cause clinical signs of vomiting, ataxia, and ascending paralysis in dogs.

*Toxic Dose:* >2.4 mg/kg

*Treatment:* There is no known antidote. Treatment consists of supportive care, including administration of intravenous fluids and antiemetics and placement of a urinary catheter for patient cleanliness. Clinical signs resolve in most cases within 72 hours.

**Marijuana (Cannabis sativa)**

Delta-9 tetrahydrocannabinol (THC) is the active ingredient in the plant _Cannabis sativa_. Largely known for its use as a recreational drug, marijuana is now sold for medicinal purposes in some states. Extraction of THC oil from the plant product into butter or oil and use in food products and baked goods have increased the potential for THC toxicity in companion animals.

*Pathophysiology:* Marijuana interacts with norepinephrine, dopamine, serotonin, and acetylcholine neurotransmitters in the frontal cortex and cerebellum.
Clinical Signs: Marijuana is a hallucinogen that can cause CNS depression, ataxia, mydriasis, increased sensitivity to motion or sound, salivation, and tremors. Along with these findings, a classic clinical sign is the sudden onset of dribbling urine.

Testing: Urine can be tested with drug test kits for tetrahydrocannabinoid, the toxic compound in marijuana. However, urine drug screening tests are not always sensitive or specific in urine of dogs, as dog metabolites of THC sometimes differ from those of humans.

Toxic Dose: The lethal dose is >3 g/kg; clinical signs occur at much lower doses.

Treatment: There is no known antidote for marijuana toxicity; therefore treatment is largely symptomatic. Place an intravenous catheter and administer intravenous fluids to support hydration. Administer atropine if severe bradycardia exists. Induction of emesis can be attempted but, because of the antiemetic effects of THC, is usually unsuccessful. Orogastric lavage can be performed, followed by repeated doses of activated charcoal. Clinical signs usually resolve within 12 to 16 hours.

Matches

“Strike Anywhere” matches, safety matches, and the striking surface of matchbook covers contain iron phosphorus or potassium chlorate.

Clinical Signs: Both compounds have a low toxic potential but can cause clinical signs of gastroenteritis and methemoglobinemia if large quantities are ingested.

Toxic Dose: Even small amounts can be caustic and cause gastric irritation.

Treatment: Treatment of match and matchbook ingestion includes gastric decontamination with induction of emesis or orogastric lavage and administration of activated charcoal and a cathartic. If methemoglobinemia occurs, administer N-acetylcysteine, intravenous fluids, and supplemental oxygen.

Metaldehyde

Metaldehyde is the active ingredient in most brands of snail bait.

Pathophysiology: The exact mechanism of toxicity is unknown but may involve inhibition of GABA channels.

Clinical Signs: Clinical signs associated with metaldehyde toxicity include severe muscle tremors, CNS excitation, and hyperthermia, which occur within 15 to 30 minutes of ingestion. Diarrhea and convulsions can develop. If hyperthermia is severe, renal failure secondary to myoglobinuria and DIC can result. Delayed hepatic failure has been described days after initial recovery. If metaldehyde toxicosis is suspected, analysis of urine, serum, and stomach contents is warranted.

Toxic Dose: 180 mg/kg

Treatment: To treat metaldehyde toxicity, procure and maintain a patent airway and control CNS excitation and muscle tremors. If an animal has just ingested the metaldehyde and is not symptomatic, induce emesis. If clinical signs are present, perform orogastric lavage. Both emesis and orogastric lavage should be followed by administration of one dose of activated charcoal. Administer intravenous fluids to control hyperthermia, prevent dehydration, and correct acid-base and electrolyte abnormalities. Methocarbamol (55 to 220 mg/kg IV slowly to effect) is the treatment of choice to control muscle tremors. Diazepam (0.5 to 1 mg/kg IV) can be used to control seizures if they occur.

Methiocarb See Carbamates.

Mineral Spirits See Fuels.

Mothballs See Naphthalene.
Mushrooms
Not all mushrooms are edible. Mushroom species can appear similar, and mushrooms can be toxic to humans and domestic animals. Mushroom toxicity is most common in areas of the country with moist environments that allow mushrooms to grow and where animals have free roaming access to the outdoors.

Pathophysiology: Mushroom ingestion most commonly causes activation of the autonomic nervous system.

Clinical Signs: Clinical signs include tremors, agitation, restlessness, hyperexcitability, and seizures. In some cases SLUD (salivation, lacrimation, urination, and defecation) is seen. Some mushrooms (Amanita species) also can cause hepatocellular toxicity. Clinical signs include vomiting, anorexia, lethargy, and progressive icterus.

Toxic Dose: Toxic dose is dependent on type of mushroom ingested.

Treatment: Treatment of mushroom toxicity is largely supportive. If the mushroom was ingested within the last 2 hours, induce emesis or perform orogastric lavage and then administer activated charcoal. Symptomatic treatment includes intravenous fluids to promote diuresis and treat hyperthermia and skeletal muscle relaxants to control tremors and seizures (methocarbamol, 55 to 220 mg/kg IV slowly to effect; diazepam, 0.5 to 1 mg/kg IV). If Amanita ingestion is suspected, administer hepatoprotectant agents including milk thistle (50 to 250 mg PO q24h).

Mycotoxins (Tremorgenic Mycotoxins)
Mycotoxins from Penicillium species are found in moldy foods, cream cheese, and nuts.

Pathophysiology: These mycotoxins have an unknown mechanism of action, although it is suspected to involve failure of neurotransmitter release at peripheral and CNS. A vomitogenic action of toxin on chemoreceptor trigger zone occurs.

Clinical Signs: Clinical signs of intoxication include vomiting, tremors, agitation, hyperesthesia, and seizures.

Testing: If tremorgenic mycotoxin toxicity is suspected, a sample of the patient’s serum and gastric contents or vomitus can be submitted to the Michigan State University Veterinary Toxicology Laboratory for tremorgen assay.

Toxic Dose: Small amounts are toxic, depending on the amount ingested and the size of the animal.

Treatment: There is no known antidote. Perform orogastric lavage, followed by administration of activated charcoal. Repeated doses of activated charcoal are recommended, as the toxin undergoes enterohepatic circulation. Control tremors and seizures with methocarbamol (55 to 220 mg/kg IV to effect), diazepam (0.5 to 1 mg/kg IV), phenobarbital (10 to 20 mg/kg IV slowly), or pentobarbital (5 to 15 mg/kg IV to effect). Administer intravenous fluids to control hyperthermia and maintain hydration. In cases in which cerebral edema is suspected secondary to severe refractory seizures, administer intravenous mannitol (0.5 to 1 g/kg IV) and furosemide (1 mg/kg IV).

Naphthalene
Naphthalene is the active ingredient in mothballs and has a high toxic potential.

Pathophysiology: Oxidative damage to red blood cells and hemoglobin, hepatotoxic

Clinical Signs: Clinical signs associated with naphthalene toxicity include vomiting, methemoglobinemia, CNS stimulation, seizures, and hepatic toxicity. A complete blood count often reveals Heinz bodies and anemia.

Toxic Dose: 411 mg/kg (dogs), smaller amounts in cats

Treatment: Do not induce emesis if naphthalene ingestion is suspected. If the ingestion was within 1 hour of presentation, perform orogastric lavage. Control seizures with diazepam (0.5 to 1 mg/kg IV) or phenobarbital (10 to 20 mg/kg IV slowly) Administer intravenous fluids to control hyperthermia and maintain hydration. N-acetylcysteine (140 mg/kg PO or
IV once, then 70 mg/kg IV or PO q6h for seven treatments) can play a role in the treatment of methemoglobinemia. A packed RBC transfusion may be necessary if anemia is severe. Observe the patient for clinical signs associated with hepatitis.

Nicotine
Nicotine toxicity occurs in animals as the result of ingestion of cigarettes, nicotine-containing gum, and some insecticides.
Pathophysiology: Nicotine stimulates autonomic ganglia at low doses and blocks autonomic ganglia and the neuromuscular junction at high doses. Absorption after ingestion is rapid.
Clinical Signs: Clinical signs include hyperexcitability and SLUD (salivation, lacrimation, urination, and defecation). Muscle tremors, respiratory muscle fatigue or hypoventilation, tachyarrhythmias, seizures, coma, and death can occur.
Toxic Dose: LD$_{50}$ is 9.2 mg/kg
Treatment: If the patient is presented within 1 hour of ingestion and has no clinical signs, induce emesis, followed by administration of repeated doses of activated charcoal. In patients with clinical signs of toxicity, perform orogastric lavage. Administer intravenous fluids to maintain hydration and promote diuresis, and treat hyperthermia. Administer atropine (0.022 to 0.044 mg/kg IV, IM, SQ) to treat cholinergic symptoms. Urinary acidification can promote nicotine excretion.

Nonsteroidal Antiinflammatory Drugs
NSAIDs include ibuprofen, ketoprofen, carprofen, diclofenac, naproxen, celecoxib, valdecoxib, rofecoxib, and deracoxib.
Pathophysiology: NSAIDs cause inhibition of prostaglandin synthesis.
Clinical Signs: Clinical signs include gastrointestinal ulceration, renal failure, and hepatotoxicity. Ibuprofen toxicity has been associated with seizures in dogs, cats, and ferrets.
Toxic Dose: The toxic dose varies with the specific compound ingested.
Treatment: To treat NSAID toxicity, induce emesis or perform orogastric lavage, followed by administration of multiple repeated doses of activated charcoal. Place an intravenous catheter for crystalloid fluid diuresis to maintain renal perfusion. Administer the synthetic prostaglandin analogue misoprostol (5 to 7.5 mcg/kg PO or per rectum Q12h, dog; 5 mcg/kg PO or per rectum Q12h, cat) to help maintain gastric and renal perfusion. Control seizures, if present, with intravenous diazepam (0.5 to 1 mg/kg IV). Administer gastroprotectant and antiemetic drugs to control vomiting and gastrointestinal hemorrhage. Continue intravenous fluid diuresis for a minimum of 48 hours, with frequent monitoring of the patient’s BUN and creatinine. When the BUN and creatinine levels are normal or have plateaued for 24 hours, slowly decrease fluid diuresis 25% per day until maintenance levels are restored.

Oils (Lubricating, Fuel, Mineral) See Fuels.

Onions, Garlic, and Chives
Onions, garlic, and chives contain sulfoxide compounds that can cause oxidative damage of RBCs, leading to Heinz body anemia, methemoglobinemia, and intravascular hemolysis.
Pathophysiology: Pathophysiology involves oxidative damage of RBCs, leading to Heinz body anemia, methemoglobinemia, and intravascular hemolysis.
Clinical Signs: Clinical signs of toxicity include weakness, lethargy, tachypnea, tachycardia, and pale mucous membranes. Vomiting and diarrhea can occur. Intravascular hemolysis can cause hemoglobinuria and pigment damage of the renal tubular epithelium. Heinz bodies may be observed on cytologic evaluation of the peripheral blood smear.
Toxic Dose: Ingestion of >0.5% of onions per body weight of animal
Treatment: Treatment of onion, chive, and garlic toxicity includes administration of intravenous fluid diuresis and induction of emesis or orogastric lavage, followed by administration of activated charcoal and a cathartic. In cases of severe anemia, packed RBC transfusion should be considered.

Opiates
Opiate drugs include heroin, morphine, oxymorphone, fentanyl, meperidine, and codeine.
Pathophysiology: Opiate compounds bind to specific opioid receptors throughout the body.
Clinical Signs: Clinical signs include miosis or mydriasis (cats) and CNS excitation, followed by ataxia and CNS depression, leading to stupor and coma. Hypoventilation, bradycardia, hypoxia, and cyanosis can occur.
Toxic Dose: Toxic dose is dependent on type of substance or opiate ingested and body weight of animal.
Treatment: To treat known overdose or ingestion of an opiate compound, induce emesis (in asymptomatic animals) or perform orogastric lavage, followed by administration of activated charcoal. Administer intravenous fluids and supplemental oxygen to support the cardiovascular and respiratory systems. Mechanical ventilation may be necessary until hypoventilation resolves. Administer repeated doses of naloxone (0.04 mg/kg IV, IM, SQ [canine], 0.005 to 0.01 mg/kg IV, IM, SQ [feline]) as a specific antidote to reverse clinical signs of narcosis and hypoventilation. If seizures are present (meperidine toxicity), administer diazepam (0.5 to 1 mg/kg IV).

Organophosphates
Organophosphate compounds traditionally are used in flea-control products and insecticides. Common examples of organophosphates include chlorpyrifos, coumaphos, diazinon, dichlorvos, and malathion.
Pathophysiology: Organophosphate toxicity causes acetylcholinesterase inhibition.
Clinical Signs: Clinical signs include CNS stimulation, including tremors and seizures. Muscarinic acetylcholine overload causes the classic SLUD signs of salivation, lacrimation, urination, and defecation. Miosis, excessive bronchial secretions, muscle tremors, and respiratory paralysis can occur. An intermediate syndrome of generalized weakness, hypoventilation, and eventual paralysis with ventral cervical ventroflexion that may require mechanical ventilation has been described.
Testing: If organophosphate toxicity is suspected, whole-blood acetylcholinesterase activity can be measured and will be low.
Toxic Dose: The toxic dose varies, depending on the particular compound and individual animal sensitivity.
Treatment: Treatment of toxicity includes careful and thorough bathing in cases of dermal exposure and, if the substance was ingested, gastric decontamination with induction of emesis or orogastric lavage, followed by administration of activated charcoal, and administration of the antidote 2-PAM (50 mg/kg IV over 30 minutes [canine], 20 mg/kg IV over 30 minutes [feline]). Atropine can help control the muscarinic clinical signs. Supportive care in the form of cooling measures, intravenous crystalloid fluids, and supplemental oxygen or mechanical ventilation may be required, depending on the severity of clinical signs.

Paint and Varnish Removers See Fuels.

Paintballs
Paintballs are gelatin capsules that contain multiple colors of paint in a sorbitol or glycerol carrier. Ingestion of large amounts of paintballs can cause neurologic signs, electrolyte abnormalities, and occasionally death.
Pathophysiology: When large quantities of these osmotically active sugars are ingested, osmotic shifts of fluid cause a sudden onset of neurologic or gastrointestinal signs, including ataxia, seizures, and osmotic diarrhea caused by massive fluid shifts into the gastrointestinal tract. The loss of water in excess of solute can result in hypernatremia, a free water deficit, and increased serum osmolality.

Clinical Signs: Clinical signs include ataxia, seizures, and diarrhea.

Treatment: After orogastric lavage, treatment of ingestion includes administering warm water enemas to help speed the movement of the paintballs through the gastrointestinal tract. Do not administer activated charcoal (usually in a propylene glycol carrier), because the compound’s cathartic action will pull more fluid into the gastrointestinal tract. Baseline electrolytes should be obtained and then carefully monitored. If severe hypernatremia develops, administer hypotonic solutions such as 0.45% NaCl + 2.5% dextrose or 5% dextrose in water after calculating the patient’s free water deficit. Because of the large volume of fluid loss, intravenous fluid rates may seem excessive but are necessary to normalize acid-base, electrolyte, and hydration status. In most cases these patients can survive if the problem is recognized promptly and corrected with careful electrolyte monitoring, aggressive decontamination strategies, and intravenous fluid support.

Paints and Varnishes See Fuels.

Paracetamol See Acetaminophen.

Paraffin Wax See Fuels.

Paraquat
Paraquat, a dipyridyl compound, is the active ingredient in some herbicides. Pathophysiology: Pathophysiology involves oxygen-derived free radical species in the lungs, and eventually multiorgan failure and death.

Clinical Signs: Clinical signs include initial CNS excitation, then intractable vomiting, ataxia, stupor, and coma. Seizures may be noted. Within 2 to 3 days, clinical signs associated with severe respiratory distress and ARDS can develop, leading to death. Chronic effects include pulmonary fibrosis, if the patient survives the initial toxicity period. The prognosis for paraquat toxicity is generally unfavorable.

Toxic Dose: The LD₅₀ of paraquat is 25 to 50 mg/kg.

Treatment: To treat paraquat ingestion, remove the toxin from the gastrointestinal tract as rapidly as possible after ingestion. There are no known antidotes. If the compound was ingested within the past hour and the animal is able to protect its airway, induce emesis. Otherwise, perform orogastric lavage. Activated charcoal is not as effective as clay or bentonite adsorbents for removing this particular toxin. Early in the course of paraquat toxicity, oxygen therapy is contraindicated because of the risk of producing oxygen-derived free radical species. Later, oxygen therapy, including mechanical ventilation, is necessary if ARDS develops. Experimentally, free radical scavengers (N-acetylcysteine, vitamin C, vitamin E, SAMe) have been shown to be useful in preventing damage caused by oxygen-derived free radical species. Hemoperfusion may be useful in eliminating the toxin, if it is performed early in the course of toxicity.

Pennies See Zinc and Zinc Oxide.

Pennyroyal Oil
Pennyroyal oil is an herbal flea-control compound that contains menthofuran as its toxic compound. Two plant species (Mentha pulegium and Hedeoma pulegioides) contain pennyroyal oil.
Pathophysiology: Pathophysiology involves glutathione depletion in cats. The toxic agent pulegone is metabolized to a hepatotoxic metabolite called menthofuran.

Clinical Signs: Menthofuran is hepatotoxic and may cause gastrointestinal hemorrhage and coagulopathies, seizures, and death.

Toxic Dose: Toxic dose is unknown; toxic dose as low as 2 g/kg in dogs has been reported.

Treatment: To treat toxicity, administer a cathartic and activated charcoal and antiemetic and gastroprotectant drugs, and thoroughly bathe the animal to prevent further dermal exposure.

Petroleum Distillates See Fuels.

Phenobarbital See Barbiturates.

Phencyclidine (Angel Dust)
Phencyclidine ("angel dust") is an illicit recreational drug that causes both CNS depression and excitation, decreased cardiac output, and hypotension; death can occur at higher doses.

Pathophysiology: The drug is a nonnarcotic, nonbarbiturate anesthetic.

Clinical Signs: Clinical signs include mydriasis, tachycardia, tonic-clonic seizures, tremors, muscle rigidity, jaw snapping, opisthotonus, and death.

Toxic Dose: 1.1 mg/kg (cats), 2.5 mg/kg (dogs)

Treatment: To treat phencyclidine toxicity, place an intravenous catheter and administer intravenous fluids and antiarrhythmic drugs to maintain organ perfusion. Administer supplemental oxygen, and administer diazepam (0.5 to 1 mg/kg IV) to control seizures. Urine alkalinization can help eliminate the compound.

Phenylephrine
Phenylephrine is an α-adrenergic agonist in many OTC decongestant preparations.

Pathophysiology: Pathophysiology involves stimulation of α-adrenergic receptors, causing vasoconstriction and tachycardia.

Clinical Signs: Clinical signs of intoxication include mydriasis, tachypnea, agitation, hyperactivity, and abnormal flybiting and staring behavior. Tachycardia, bradycardia, hypertension, hyperthermia, and seizures can occur.

Toxic Dose: 1 mg/kg—vomiting; 3 mg/kg—tachyarrhythmias, hypertension

Treatment: To treat phenylephrine toxicity, place an intravenous catheter and give intravenous fluids to maintain hydration, promote diuresis, and treat hypertension. Administer prazosin (1 to 4 mg/kg PO q8-12h [canine], 0.5 mg PO q8-12h [feline]) or sodium nitroprusside (1 to 2 mcg/kg/min IV CRI, titrating up slowly until desired BP is reached, carefully monitoring for hypotension) to treat hypertension, antiarrhythmic drugs as necessary, and diazepam (0.5 to 1 mg/kg IV) to control seizures.

Phenylpropanolamine
Phenylpropanolamine has both α- and β-adrenergic agonist effects and is used primarily in the treatment of urinary incontinence in dogs. The drug was taken off of the market for use in humans because of the risk of stroke.

Pathophysiology: Phenylpropanolamine is an α- and β-adrenergic receptor agonist, causing vasoconstriction, tachyarrhythmias, and hypertension at higher doses.

Clinical Signs: Clinical signs of phenylpropanolamine intoxication include hyperactivity, hyperthermia, mydriasis, tachyarrhythmias or bradycardia, hypertension, agitation, and seizures.

Toxic Dose: 1-5 mg/kg—vomiting, tachycardia, hyperactivity; 5 to 10 mg/kg—vomiting, tachycardia, hyperactivity, hyperthermia, hypertension
Treatment: To treat toxicity, administer prazosin (1 to 4 mg/kg PO q8-12h [canine], 0.5 mg PO q8-12h [feline]) or nitroprusside (1 to 2 mcg/kg/min IV CRI, titrating up slowly until desired BP is reached, carefully monitoring for hypotension) to control hypertension; a β-blocker (esmolol, 50 to 100 mcg/kg IV bolus, 50 to 200 mcg/kg/min IV CRI; propranolol, 0.04 to 0.1 mg/kg IV slowly to effect; atenolol, 0.5 to 1 mg/kg PO q12h [canine], 6.25 to 12.5 mg/cat PO q12-24h [feline]) to control tachyarrhythmias; diazepam (0.5 to 1 mg/kg IV) to control seizures; and intravenous fluids to maintain hydration and promote diuresis. Urine acidification may aid in facilitating excretion. If bradycardia occurs, do not use atropine.

Photographic Developer Solutions See Detergents, Nonionic.

Pine Oil Disinfectants See Detergents, Nonionic and Alcohols.

Piperazine
Piperazine is a relative of ivermectin used for anthelmintic purposes.
Pathophysiology: Piperazine is a GABA agonist.
Clinical Signs: Clinical signs include cervical and truncal ataxia, tremors, seizures, coma, and death.
Toxic Dose: 30 to 55 mg/kg
Treatment: If ingestion was recent and if no clinical signs of toxicity are present, induce emesis or perform orogastric lavage, followed by administration of a cathartic and activated charcoal. There is no known antidote. Treatment includes supportive care in the form of intravenous fluids and administration of phenobarbital or methocarbamol to control seizures and tremors. Diazepam, a GABA agonist, is contraindicated, because it can potentially worsen clinical signs. Urine acidification may hasten elimination. Clinical signs can last from 3 to 5 days.

Pseudoephedrine
Pseudoephedrine is an α- and β-adrenergic agonist that is a component of many OTC decongestants and is used in the manufacture of crystal methamphetamine.
Pathophysiology: Pathophysiology involves α- and β-adrenergic receptor agonist activity, causing vasoconstriction, tachyarrhythmias, and hypertension.
Clinical Signs: Clinical signs of toxicity include severe restlessness, tremors, mydriasis, agitation, hyperthermia, tachyarrhythmias or bradycardia, hypertension, and seizures.
Toxic Dose: Clinical signs of toxicity are seen at 3 mg/kg.
Treatment: To treat toxicity, administer activated charcoal, intravenous fluids to promote diuresis and treat hypertension, chlorpromazine to combat α-adrenergic effects, a β-blocker (propranolol, 0.04 to 0.1 mg/kg IV slowly to effect; esmolol, 50 to 100 mcg/kg IV bolus, 50 to 200 mcg/kg/min IV CRI; atenolol, 0.5 to 1 mg/kg PO q12h [canine], 6.25 to 12.5 mg/cat PO q12-24h [feline]) to treat β-adrenergic effects, and cyproheptadine (0.5 to 1.1 mg/kg POq12h or per rectum dog, 2 mg/k cat PO or per rectum) to combat serotoninergic effects.

Pyrethrin and Pyrethroids
Pyrethrin and pyrethroid compounds are extracted from chrysanthemums and include allethrin, decamethrin, tralomethrin, fenpropathrin, prallethrin, sumithrin, permethrin, tetramethrin, cyfluthrin, and resmethrin.
Pathophysiology: Pyrethrin and pyrethroid compounds cause depolarization and blockade of nerve membrane potentials, causing clinical signs of tremors, seizures, respiratory distress, and paralysis. Contact dermatitis can occur.
Clinical Signs: Clinical signs include tremors, seizures, respiratory distress, and paralysis.
The oral toxicity is fairly low; however, the compounds can be significantly harmful if inhaled or applied to the skin.

Testing: To distinguish between pyrethrin or pyrethroid toxicity and organophosphate toxicity, acetylcholinesterase levels should be obtained; they will be normal if pyrethrins are the cause of the animal’s clinical signs.

Toxic Dose: Spot-On flea products labeled for dogs contain 45% to 60% permethrin. Permethrin is toxic to cats.

Treatment: Treatment of toxicity is supportive, as there is no known antidote. Carefully bathe the animal in lukewarm water to prevent further oral and dermal exposure. Both hyperthermia and hypothermia can worsen clinical signs. Administer activated charcoal to decrease enterohepatic recirculation. Atropine (0.02 to 0.04 mg/kg IV, IM, SQ) may control clinical signs of excessive salivation. To control muscle tremors, administer methocarbamol (50 to 220 mg/kg IV slowly to effect). Administer diazepam (0.5 to 1 mg/kg IV) or phenobarbital (10 to 20 mg/kg IV slowly) to control seizures, as necessary.

Radiator Fluids See Ethylene Glycol.

Raisins See Grapes and Raisins.

Rotenone
Rotenone is used as a common garden and delousing insecticide. Fish and birds are very susceptible to rotenone toxicity.

Pathophysiology: Rotenone inhibits mitochondrial electron transport and nerve conduction.

Clinical Signs: Clinical signs of tissue irritation and hypoglycemia can occur after topical or oral exposure. If the compound is inhaled, CNS depression and seizures can occur.

Treatment: To treat toxicity, perform orogastric lavage, followed by administration of a cathartic and activated charcoal. Bathe the animal carefully to prevent further dermal exposure and further ingestion. Administer diazepam (0.5 to 1 mg/kg IV) or phenobarbital (10 to 20 mg/kg IV slowly) to control seizures. The prognosis generally is guarded.

Rubbing Alcohol See Alcohols.

Rust Removers See Acids and Corrosives.

Salicylates See Aspirin.

Salt, Thawing
Salt used for thawing ice commonly contains calcium chloride, a compound that has a moderate toxic potential.

Pathophysiology: Calcium chloride produces strong local irritation and can cause gastroenteritis and gastrointestinal ulcers if ingested.

Clinical Signs: Clinical signs include erythema, vomiting, and hematemesis.

Treatment: Treatment of ingestion includes dilution with milk, water, or egg whites. Perform orogastric lavage, followed by administration of activated charcoal. Administer intravenous crystalloid fluids to maintain hydration. Administer antiemetic and gastroprotectant drugs to treat gastroenteritis and vomiting.

Shampoos, Nonmedicated See Detergents, Nonionic.

Shampoos, Selenium Sulfide
Selenium sulfide shampoos (e.g., Selsun Blue) have a low toxic potential and primarily cause gastroenteritis.
Pathophysiology: Pathophysiology involves gastric irritation.
Clinical Signs: Clinical signs include vomiting.
Treatment: Treatment of ingestion includes dilution with water, milk, or egg whites and administration of activated charcoal. Carefully and thoroughly rinse the skin and eyes to prevent further exposure. Administer antiemetic and gastroprotectant drugs in cases of severe gastroenteritis.

Shoe Polish See Hydrocarbons, Aromatic.

Silver Polish
Some silver polishes contain the alkali substance sodium carbonate and cyanide salts and have a serious toxic potential.
Pathophysiology: Pathophysiology involves disruption of electron transport chain.
Clinical Signs: Ingestion results in rapid onset of vomiting and possibly cyanide toxicity.
Treatment: To treat ingestion, monitor and maintain the patient's respiration and cardiovascular status and administer intravenous crystalloid fluids. Induce emesis, followed by administration of activated charcoal. Administer sodium nitrite or sodium thiosulfate IV for cyanide toxicity.

Soaps (Bath, Bar Soap)
Bath soap (bar soap) usually has low toxic potential and causes mild gastroenteritis with vomiting if ingested.
Pathophysiology: Bar soaps cause gastric irritation.
Clinical Signs: Clinical signs include vomiting, hematemesis.
Toxic Dose: Small quantities may be toxic.
Treatment: To treat ingestion, include dilution with water, administration of intravenous fluids to maintain hydration, and administration of antiemetic and gastroprotectant drugs to treat gastroenteritis.

Sodium Fluoroacetate (1080, 1081)
Sodium fluoroacetate is a colorless, odorless, tasteless compound that causes uncoupling of oxidative phosphorylation.
Pathophysiology: Pathophysiology involves uncoupling of oxidative phosphorylation, prevention of adenosine triphosphate (ATP) production and cellular metabolism, and cerebral edema.
Clinical Signs: Clinical signs of toxicity include CNS excitation, seizures, and coma secondary to cerebral edema. The prognosis is guarded.
Toxic Dose: The toxic dose in dogs and cats is 0.05 to 1.0 mg/kg.
Treatment: To treat toxicity, procure and maintain a patent airway, monitor and stabilize the cardiovascular status, and control hyperthermia. Perform orogastric lavage, followed by administration of activated charcoal. If clinical signs are not present at the time of presentation, induce emesis. Administer intravenous fluids and supplemental oxygen, as necessary.

Strattera (Selective Norepinephrine Reuptake Inhibitor)
Strattera (atomoxetine hydrochloride) is a selective norepinephrine reuptake inhibitor used in the treatment of attention-deficit/hyperactivity disorder (ADHD) in humans.
Pathophysiology: Pathophysiology involves accumulation of norepinephrine in CNS.
Clinical Signs: Clinical signs of toxicity include cardiac tachyarrhythmias, hypertension, disorientation, agitation, trembling, tremors, and hyperthermia.
Toxic Dose: Peak serum concentrations occur in dogs within 3 to 4 hours of ingestion, with a peak half-life at 4 to 5 hours after ingestion.
Treatment: Treatment of intoxication is largely symptomatic and supportive in nature. First, induce emesis if the patient is conscious and has an intact gag reflex. Orogastric lavage can also be performed. Administer one dose of activated charcoal to prevent further absorption of the compound from the gastrointestinal tract. Identify cardiac dysrhythmias and treat accordingly. Control hypertension with sodium nitroprusside (1 to 2 mcg/kg/min IV CRI, titrating up slowly until desired BP is reached, carefully monitoring for hypotension) or diltiazem (0.1 to 0.25 mg/kg IV slowly to effect, then 2 to 6 mcg/kg/min CRI). Administer acepromazine (0.02 to 0.05 mg/kg IV) or chlorpromazine (0.2 to 0.5 mg/kg IM q8h, 0.05 mg/kg IV q4h) to control agitation. Do not use diazepam, because it can potentially worsen clinical signs. Administer intravenous fluids to maintain hydration and promote diuresis.

Strychnine
Strychnine is the active ingredient in pesticides used to control rodents and other vermin. Pathophysiology: Strychnine antagonizes spinal inhibitory neurotransmitters. Clinical Signs: Clinical signs include severe muscle tremors, muscle rigidity, and seizures. Clinical signs are stimulated or exacerbated by noise, touch, light, and sound. Mydriasis, hyperthermia, and respiratory paralysis can occur. Testing: If strychnine toxicity is suspected, gastric contents should be collected and saved for analysis. Toxic Dose: The toxic dose in dogs is 0.75 mg/kg, and in cats is 2 mg/kg. Strychnine antagonizes spinal inhibitory neurotransmitters. Treatment: If the animal is asymptomatic at the time of presentation, induce emesis. If clinical signs are present, perform orogastric lavage. Both emesis and orogastric lavage should be followed by the administration of activated charcoal. Administer intravenous crystalloid fluids to support the cardiovascular system, aid in cooling measures, and improve renal diuresis. Treat CNS stimulation with methocarbamol (55 to 220 mg/kg IV to effect), diazepam (2-5 mg/kg IV), or phenobarbital (10 to 20 mg/kg IV slowly). The animal should have cotton packed in its ears to prevent noise stimulation, and should be placed in a quiet, dark room.

Styptic Pencil
Styptic pencils contain potassium alum sulfate, a compound with a low toxic potential. Pathophysiology: Ingestion of styptic pencils is corrosive because of the release of sulfuric acid during hydrolysis of the salt. Styptic pencils are a gastric irritant. Clinical Signs: Clinical signs include vomiting. Treatment: Treatment of ingestion includes dilution with Milk of Magnesia or water, administration of antiemetic and gastroprotectant drugs, and administration of intravenous crystalloid fluids to maintain hydration. Do not induce emesis, because of the risk of causing further esophageal irritation.

Sunscreen See Zinc and Zinc Oxide.

Tar See Fuels.

Tea Tree Oil (Melaleuca Oil)
Tea tree (Melaleuca) oil is an herbal-origin flea-control product. The toxic principles in tea tree oil are monoterpenes. Pathophysiology: The pathophysiology is unknown. Clinical Signs: Clinical signs include neuromuscular weakness and ataxia and hepatic failure. Toxic Dose: Direct application of 100% tea tree oil results in a toxic dose. Treatment: Treatment of tea tree oil toxicity includes administration of cathartics and activated charcoal to prevent further absorption. Carefully bathe the animal to prevent further dermal exposure.
Tetanus

Tetanus spores from *Clostridium tetani* organisms are ubiquitous in the soil and feces, particularly in barnyards. Cases have been reported in dogs after tooth eruption and after abdominal surgeries performed with cold sterilization packs. Anaerobic wound infections can contain tetanus spores.

**Pathophysiology:** The neurotoxin from *C. tetani* inhibits spinal inhibitory neurons, causing motor neuron excitation.

**Clinical Signs:** Extensor muscle rigidity ("sawhorse stance"), erect ears, and risus sardonicus (a sardonic grin) are characteristic features of tetanus.

**Treatment:** Administer tetanus antitoxin if toxin has not already been bound in the CNS. To eliminate the source of the toxin (e.g., abscess), open and debride all wounds. Intravenous administration of ampicillin or penicillin G is the treatment of choice for tetanus. Supportive care in the form of skeletal muscle relaxants, intravenous fluids and parenteral nutrition, and nursing care to prevent decubitus ulcer formation is required. In extreme cases, mechanical ventilation may be necessary.

**Toilet Bowl Cleaners** See Acids and Corrosives.

**Triazines**

Triazine compounds include atrazine, prometone, and monuron (Telvar).

**Pathophysiology:** The toxic mechanism of triazine compounds is unknown.

**Clinical Signs:** Clinical signs include salivation, ataxia, hyporeflexia, contact dermatitis, hepatorenal damage, muscle spasms, respiratory difficulty, and death.

**Treatment:** Treatment of triazine exposure includes cardiovascular and renal support in the form of intravenous crystalloid fluids, inotropic drugs, and antiarrhythmic agents, as necessary. If the exposure is recent, induce emesis. Perform orogastric lavage in animals that cannot protect the airway. Emesis and orogastric lavage should be followed by the administration of activated charcoal and a cathartic. Carefully bathe the patient to prevent further dermal absorption.

**Tricyclic Antidepressants**

A variety of tricyclic antidepressants are available for use in both humans and animals, including amitriptyline, amoxapine, desipramine, doxepin, fluoxetine (Prozac), fluvoxamine (Luvox), imipramine, nortriptyline, paroxetine (Paxil), protriptyline, sertraline (Zoloft), and trimipramine.

**Pathophysiology:** Selective serotonin reuptake inhibitors (SSRIs) are rapidly absorbed from the digestive tract, with peak serum concentrations occurring 2 to 8 hours after ingestion. The elimination half-life for each drug differs in dogs but typically last 16 to 24 hours. SSRIs inhibit the reuptake of serotonin, causing serotonin to accumulate in the brain. This can cause "serotonin syndrome."

**Clinical Signs:** Clinical signs include trembling, seizures, hyperthermia, ptyalism or hypersalivation, cramping or abdominal pain, vomiting, and diarrhea. Other clinical signs of SSRI intoxication include depression, tremors, bradycardia, tachyarrhythmias, and anorexia. Any animal that has ingested an SSRI should be promptly treated and carefully observed for at least 72 hours for side effects.

**Toxic Dose:** Toxic dose is dependent on type of tricyclic antidepressant ingested.

**Treatment:** The treatment of suspected SSRI intoxication involves gastric decontamination if the patient is not depressed and has an intact gag reflex. Perform orogastric lavage and administer activated charcoal to prevent further toxin absorption and hasten elimination from the gastrointestinal tract. Treat other clinical signs symptomatically. Administer intravenous diazepam to control seizures. Treat tachyarrhythmias according to type. Administer methocarbamol (55 to 220 mg/kg IV to effect) to control muscle tremors. Cyproheptadine (2 mg/kg), a serotonin antagonist, can be dissolved in water and administered per rectum.
Turpentine See Fuels.

Vitamin K–Antagonist Rodenticides

Vitamin K–antagonist rodenticides are commonly found in pelleted or block form.

Pathophysiology: These agents inhibit the activation of the vitamin K–dependent coagulation factors II, VII, IX, and X.

Clinical Signs: Clinical signs of hemorrhage occur within 2 to 7 days of exposure. Hemorrhage can occur anywhere in the body and can manifest as petechiation of the skin or mucous membranes, hemorrhagic sclera, epistaxis, pulmonary parenchymal or pleural hemorrhage, gastrointestinal hemorrhage, pericardial hemorrhage, hematuria, retroperitoneal hemorrhage, hemarthrosis, and CNS hemorrhage. Clinical signs include respiratory distress, cough, bleeding from the gums or into the eyes, ataxia, paresis, paralysis, seizures, hematuria, joint swelling, lameness, lethargy, weakness, inappetence, and collapse.

Testing: Diagnosis is made based on clinical signs and a prolonged ACT or PT. The PIVKA (proteins induced by vitamin K antagonism) test may be helpful but usually cannot be performed in house. Slight thrombocytopenia may be present secondary to hemorrhage; however, blood levels usually do not reach the critical level of <50,000 platelets per microliter to cause clinical signs of hemorrhage. In some cases, severe stress-induced hyperglycemia and glucosuria may be present but resolve within 24 hours.

Treatment: If the rodenticide was ingested within the last 2 hours, induce emesis. Alternatively, orogastric lavage can be performed in an uncooperative patient. Both emesis and orogastric lavage should be followed by administration of activated charcoal. The stomach contents can be submitted for analysis. After successful treatment, administer oral vitamin K for 30 days after the exposure; or a check PT 2 days after gastric decontamination. If the PT is prolonged, administer fresh frozen plasma and vitamin K. If the PT is normal, gastric decontamination was successful, and no further treatment is necessary.

If an animal shows clinical signs of intoxication, administer activated clotting factors in the form of fresh frozen plasma (20 mL/kg) and vitamin K$_1$ (5 mg/kg SQ in multiple sites with a 24-gauge needle). Packed RBCs or fresh whole blood may be required if the patient is also anemic. Supportive care in the form of supplemental oxygen may be necessary in cases of pulmonary or pleural hemorrhage. After initial therapy and discharge, the patient should receive vitamin K$_1$ (2.5 mg/kg PO q8-12h for 30 days), and PT should be checked 2 days after the last vitamin K capsule is administered. In some cases, depending on the type of anticoagulant ingested, an additional 2 weeks of vitamin K$_1$ therapy may be required.

Window Cleaner See Ethylene Glycol.

Xylitol

Xylitol is a sugar alcohol that, when ingested by humans, does not cause a significant increase in blood glucose and therefore does not stimulate insulin release from the human pancreas. In dogs, however, xylitol causes a massive rapid and dose-dependent release of insulin from pancreatic beta cells.

Pathophysiology: This agent causes insulin release from pancreas. After insulin release, clinically significant hypoglycemia can develop.

Toxic Dose: >0.1 g/kg hypoglycemia, >0.5 g/kg hepatotoxicity

Clinical Signs: Clinical signs include hypoglycemia, vomiting, weakness, ataxia, mental depression, hypokalemia, hypoglycemic seizures, and coma. Clinical signs associated with xylitol ingestion can be seen within 30 minutes of ingestion and can last for more than 12 hours, even with aggressive treatment. Cases of acute hepatic necrosis, with vomiting, icterus, coagulopathies, and death, can occur.
Treatment: Known xylitol ingestion should be treated as for other toxin ingestion. If no neurologic abnormalities exist at the time the patient is seen, induce emesis, followed by administration of activated charcoal. It remains unknown at this time whether activated charcoal actually delays or prevents the absorption of xylitol from the canine gastrointestinal tract. If clinical signs have already developed, perform orogastric lavage and gastric decontamination. Blood glucose concentrations should be analyzed and maintained with supplemental dextrose as a CRI (2.5% to 5%) until normoglycemia can be maintained with multiple frequent small meals. Hypokalemia may develop because it is driven intracellularly by the actions of insulin. Treat hypokalemia with supplemental potassium chloride by infusion, not to exceed 0.5 mEq/kg/hr.

Zephiran See Detergents, Cationic, and Disinfectants.

Zinc and Zinc Oxide
Pennies minted in the United States after 1982 contain large amounts of zinc rather than copper. Other sources of zinc include zinc oxide ointment and hardware such as that found in metal bird cages.
Pathophysiology: Zinc toxicity causes intravascular hemolysis, anemia, gastroenteritis, and renal failure.
Clinical Signs: Clinical signs include vomiting, lethargy, icterus, hemoglobinemia, hemoglobinuria, and diarrhea.
Toxic Dose: The toxic dose is unknown. Pennies minted after 1982 contain large quantities of zinc, not copper.
Treatment: If zinc toxicity is suspected, take an abdominal radiograph to document the presence of the metal in the stomach or intestines. (If zinc-containing ointment was ingested, this will not be visible on radiographs.) Induce emesis or perform orogastric lavage, depending on the size of the object ingested. Often, small objects such as pennies can be retrieved using endoscopy or surgical gastrotomy or enterotomy. Always take an additional radiograph after the removal procedure to ensure that all objects have been successfully removed. Administer intravenous fluids to maintain renal perfusion and promote fluid diuresis. Administer gastroprotectant and antiemetic drugs. Chelation therapy with succimer, calcium EDTA, dimercaprol, or penicillamine may be necessary. Do not administer calcium EDTA if the patient is dehydrated, because renal failure can result. Severe anemia should be treated with packed RBCs.

Additional Reading
Fletcher DJ, Murphy LA: Anticholinergic poisonings. In Silverstein DC, Hopper K, editors: Small animal critical care medicine, St Louis, 2009, Elsevier.
Fletcher DJ, Murphy LA: Cyclic antidepressant drug overdose. In Silverstein DC, Hopper K, editors: Small animal critical care medicine, St Louis, 2009, Elsevier.
Gfeller RW, Messonnier SP: Handbook of small animal toxicology and poisonings, ed 2, St Louis, 2004, Mosby.

**RESPIRATORY EMERGENCIES**

Respiratory emergencies consist of any problem that impairs delivery of oxygen to the level of the alveoli or diffusion of oxygen across the alveolar capillary membrane into the pulmonary capillary network. Decreased respiratory rate or tidal volume can result in hypoxia and buildup of carbon dioxide, or hypercarbia, leading to respiratory acidosis. Conditions most frequently encountered result in airflow obstruction, prevention of normal lung expansion,
interference with pulmonary gas exchange (ventilation-perfusion mismatch), and alterations of pulmonary circulation. Evaluation of the patient with respiratory distress is often challenging, because the most minimal stress can cause rapid deterioration, or even death in critical cases. Careful observation of the patient from a distance often allows the clinician to determine the severity of respiratory distress and localize the lesion based on the patient’s respiratory pattern and effort.

Animals in respiratory distress often have a rapid respiratory rate (>30 breaths per minute). As respiratory distress progresses, the patient may appear anxious and start open-mouth breathing. The animal often develops an orthopneic posture, characterized by neck extension, open-mouthed breathing, and elbows abducted or pulled away from the body. Cyanosis of the mucous membranes often indicates extreme decompensation. Clinical signs of respiratory distress can develop acutely or from decompensation of a more chronic problem that was preceded by a cough, noisy respirations, or exercise intolerance.

Localization of the cause of respiratory distress is essential to successful case management. In any patient with clinical signs of respiratory distress, the differential diagnosis should include primary pulmonary parenchymal disease, airway disease, thoracic cage disorders, CHF, dyshemoglobinemias (carbon monoxide, methemoglobin), and anemia. Careful observation of the patient’s respiratory pattern can aid in making a diagnosis of upper airway disease or obstruction, primary pulmonary parenchymal disease, pleural space disease, and abnormalities of the thoracic cage. It is often helpful to rest a hand on the patient and breathe along with the patient’s effort, to confirm the periods of inhalation and exhalation.

The pharynx, larynx, and extrathoracic trachea constitute the upper airway. Obstructive lesions are associated with a marked inspiratory wheeze or stridor and slow deep inspiratory effort. Auscultation of the larynx and trachea may reveal more subtle obstructions of normal air flow. Stridor can usually be auscultated without the use of a stethoscope. Lung sounds are usually normal. The neck should be carefully palpated for a mass lesion, tracheal collapse, and subcutaneous emphysema. Subcutaneous emphysema suggests tracheal damage or collapse secondary to severe trauma. In some cases there is a history of voice, or bark, change secondary to laryngeal dysfunction. Differential diagnosis is usually based on the patient’s signalment, history, and index of suspicion of a particular disease process. Differential diagnoses of upper airway obstruction are listed in Box 1-61.

Diseases of the pleural space often are associated with a restrictive respiratory pattern. Inspiratory efforts are short, rapid, and shallow, and there is often a marked abdominal push. The pattern has been referred to as a “choppy” “dysynchronous” respiratory pattern. Depending on the disease present, lung sounds may be muffled ventrally and enhanced dorsally. Percussion of the thorax reveals decreased resonance if fluid is present. Increased resonance is present with pneumothorax. Decreased compressibility of the anterior thorax may be present with an anterior mediastinal mass lesion, particularly in cats and ferrets. A pneumothorax or diaphragmatic hernia is commonly associated with evidence of trauma, with or without rib fractures. Respiratory distress caused by hemothorax may be exacerbated by anemia. Differential diagnoses for patients with evidence of pleural cavity disease include pneumothorax, diaphragmatic hernia, neoplasia, and various types of pleural effusion.

**BOX 1-61 DIFFERENTIAL DIAGNOSES OF UPPER AIRWAY OBSTRUCTION**

- Abscess
- Brachycephalic airway syndrome
- Granuloma
- Laryngeal collapse
- Laryngeal paralysis
- Nasopharyngeal polyp
- Neoplasia
- Obstructive laryngitis
- Pharyngeal foreign body
- Tracheal collapse
- Tracheal foreign body
- Traumatic fracture of larynx or tracheal cartilage
Primary pulmonary parenchymal disease can involve the intrathoracic airways, alveoli, interstitial space, and pulmonary vasculature. A rapid, shallow, restrictive respiratory pattern may be observed with a marked push on exhalation, particularly with obstructive airway disease such as chronic bronchitis (asthma) in cats. Crackles or wheezes are heard on thoracic auscultation. Differential diagnoses for pulmonary parenchymal disease include cardiogenic and noncardiogenic pulmonary edema, pneumonia, feline bronchitis (asthma), pulmonary contusion, aspiration pneumonitis, pulmonary thromboembolism (PTE), neoplasia, infection (bacterial, fungal, protozoal, viral), and/or chronic bronchitis.

Other abnormal respiratory patterns may be evident, and warrant further consideration. Tachypnea present in the absence of other signs of respiratory distress can be a normal response to nonrespiratory problems, including pain, hyperthermia, and stress. A restrictive respiratory pattern with minimal thoracic excursions can be associated with diseases of neuromuscular function, including ascending polyradiculoneuritis, botulism, and tick paralysis. If adequate ventilation cannot be maintained by the patient, mechanical ventilation may be indicated. Kussmaul respiration manifests as very slow, very deep respirations when a metabolic acidosis is present. This type of respiratory pattern typically is observed in patients with severe diabetic ketoacidosis and renal failure in a compensatory attempt to blow off carbon dioxide. Cheyne-Stokes respiration is usually observed with a defect in the central respiratory control center. The classic pattern of Cheyne-Stokes respiration is normal or hyperventilation followed by a period of apnea or hypoventilation. In cases of lower cervical cord damage or damage to the central respiratory control center in the CNS, the diaphragm alone may assume most of the ventilatory movement. With diaphragmatic fatigue, severe hypoventilation and resultant hypoxemia may require mechanical ventilation.

**Immediate Management**

Immediate management of any patient in respiratory distress is to minimize stress at all costs. Relatively benign procedures such as radiography or intravenous catheter placement can be fatal in patients with severe respiratory compromise. Stabilization should always precede further diagnostic evaluation. In some cases, sedation may be required before any diagnostics are performed, to prevent further stress. All patients should receive some form of supplemental oxygen, either by mask, cage, or flow-by techniques. In cases in which a severe pneumothorax or pleural effusion is suspected, perform therapeutic and diagnostic thoracocentesis bilaterally to allow lung reexpansion and alleviate respiratory distress, whenever possible. If thoracocentesis alone is not effective at maintaining lung reexpansion, place a thoracostomy tube (particularly in cases of tension pneumothorax). If hypovolemic or hemorrhagic shock is present, initiate treatment while stabilizing the respiratory system (see section on shock).

If an animal is suspected of having an upper airway obstruction, reestablish airflow. In cases of laryngeal paralysis, tracheal collapse, and brachycephalic airway syndrome, sedation is often very useful in alleviating the distress of airway obstruction. In cases of laryngeal collapse, however, sedation may make the condition worse. If laryngeal edema is severe, administer a dose of short-acting glucocorticosteroids (dexamethasone sodium phosphate) to decrease laryngeal inflammation and edema. If a foreign body is lodged in the pharynx, perform the Heimlich maneuver by thrusting bluntly several times on the patient’s sternum. Objects such as balls or bones may be small enough to enter the larynx but too large to be expelled; rapid-acting general anesthesia will be necessary to facilitate dislodgement and removal. If the obstruction cannot be removed, bypassing the obstruction with an endotracheal tube or temporary tracheostomy should be considered.

In an emergency, a temporary transtracheal oxygen catheter can quickly be placed in the following manner. Connect a 20- or 22-gauge needle to a length of intravenous extension tubing and a 3-mL syringe. Place the male connector of the syringe into the female portion of the extension tubing. Cut off the syringe plunger and connect the resulting blunt end to a length of flexible tubing attached to a humidified oxygen source. Run the oxygen at 10 L/min to provide adequate oxygenation until a tracheostomy can be performed. (See sections on oxygen supplementation and tracheostomy.)
Once the animal’s condition has been stabilized, specific diagnostic tests, including arterial blood gas analyses, thoracic radiographs, and/or transtracheal wash, can be performed, depending on the patient’s condition and needs. Specific therapies for management of upper airway obstruction, pleural space disease, and pulmonary disease are discussed next.

Management of Upper Airway Obstruction

Upper airway obstruction can occur as a result of intraluminal or extraluminal mass lesions or foreign bodies in the oropharynx (abscess, neoplasia), laryngeal paralysis, trauma, and anatomic abnormalities. Clinical signs of an upper airway obstruction are associated with an animal’s extreme efforts to inhale air past the obstruction. Marked negative pressure occurs in the extrathoracic airways and can cause worsening of clinical signs. Mucosal edema and inflammation further worsen the obstruction.

Therapy for upper airway obstruction is aimed at breaking the cycle of anxiety and respiratory distress. Administer the anxiolytic tranquilizer acepromazine (0.02 to 0.05 mg/kg IV, IM, SQ) to decrease patient anxiety. Many animals develop hyperthermia from increased respiratory effort and extreme anxiety. Implement cooling measures in the form of cool intravenous fluids and wet towels soaked in tepid water placed over the animal (see section on hyperthermia). Administer supplemental oxygen in a manner that is least stressful for the animal. Short-acting glucocorticosteroids can also be administered (dexamethasone sodium phosphate, 0.25 mg/kg IV, SQ, IM) to decrease edema and inflammation.

If the airway obstruction is severe and there is no response to initial measures to alleviate anxiety and decrease inflammation, establish control of ventilation by placement of an endotracheal tube (see section on endotracheal intubation), tracheal oxygen catheter, or temporary tracheostomy. To obtain airway control, administer a rapid-acting anesthetic (propofol, 4 to 7 mg/kg IV to effect), and intubate with a temporary tracheostomy. An intratracheal oxygen catheter can be placed with sedation and/or a local anesthetic (see technique for transtracheal wash).

Laryngeal Paralysis

Laryngeal paralysis is a congenital or acquired condition that occurs primarily in large-breed dogs secondary to denervation of the arytenoid cartilages by the recurrent laryngeal nerve. Congenital laryngeal paralysis occurs in the Bouvier des Flandres, Siberian Husky, and Bull Terrier. Acquired laryngeal paralysis occurs in Labrador Retrievers, Saint Bernards, and Irish Setters. Acquired laryngeal paralysis can be idiopathic, acquired secondary to trauma to the recurrent laryngeal nerve, or can be a component of systemic neuromuscular disease. Although rare, this condition also occurs in cats.

With dysfunction of the recurrent laryngeal nerve, the intrinsic laryngeal muscles atrophy and degenerate. As a result, the vocal folds and arytenoid cartilage move in a paramedian position within the airway and fail to abduct during inhalation, causing airway obstruction. Laryngeal paralysis can be partial or complete, unilateral or bilateral. In many cases a change in bark is noted before the development of clinical signs of respiratory distress or exercise intolerance. When a patient demonstrates severe inspiratory stridor (with or without hyperthermia), initiate stabilization with anxiolytic tranquilizers, supplemental oxygen, and cooling measures. Once the patient’s condition has been stabilized, definitive measures to accurately document and assess the patient’s airway should be considered. Place the patient under very heavy sedation with short-acting barbiturates or propofol (4 to 7 mg/kg IV) and observe the arytenoid cartilages closely in all phases of respiration. Administer just enough drug to allow careful examination without getting bitten. If the arytenoid cartilages do not abduct during inhalation, administer Dopram (doxapram hydrochloride, 1 to 5 mg/kg IV) to stimulate respiration.

Absent or paradoxical laryngeal motion (closed during inspiration and open during exhalation) is characteristic of laryngeal paralysis. Correction of the defect involves documentation and treatment of any underlying disorder and surgical repair of the area to open the airway. Partial laryngectomy, arytenoid lateralization (“tie-back” surgery), or removal of the vocal folds has been used with some success. Aspiration pneumonitis is common after these procedures.
Brachycephalic Airway Syndrome and Laryngeal Collapse

Brachycephalic airway syndrome is associated with a series of anatomic abnormalities that collectively increase resistance to airflow. Affected animals typically have stenotic nares, an elongated soft palate, and a hypoplastic trachea. Components of the syndrome can occur alone or in combination. In severe cases, laryngeal saccular edema and eversion, and eventual pharyngeal collapse, can occur secondary to the severe increase in intrathoracic airway pressure required to overcome the resistance of the upper airways. Specific airway anomalies can be identified with general anesthesia and laryngoscopy.

Severe respiratory distress should be treated as discussed previously. Treatment requires surgical correction of the anatomic abnormalities. In animals with laryngeal collapse, surgical correction may not be possible, and a permanent tracheostomy may be required. Because an elongated soft palate and stenotic nares can be identified before the onset of clinical signs, surgical correction to improve airflow when the animal is young may decrease the negative intrathoracic pressure necessary to move air past these obstructions. The chronic consequences of everted laryngeal saccules and laryngeal collapse potentially can be prevented.

Tracheal Collapse

Tracheal collapse is common in middle-aged and older toy and small-breed dogs. The owner typically reports a chronic cough that is readily induced by excitement or palpation of the trachea. The cough often sounds like a “goose honk.” Diagnostic confirmation is obtained by lateral radiography or fluoroscopy of the cervical and thoracic trachea during all phases of respiration. Acute decompensation is uncommon but does occur, particularly with excitement, exercise, and increased environmental temperatures or ambient humidity.

Therapy of the patient with acute respiratory distress secondary to tracheal collapse includes sedation, administration of supplemental oxygen, and provision of cooling measures to treat hyperthermia. Cough suppressants (hydrocodone bitartrate–homatropine methylbromide, 0.25 mg/kg PO q8-12h, or butorphanol, 0.5 mg/kg PO q6-12h) are useful.

Tracheal collapse is a dynamic process that usually involves both the upper and lower airways. Because of this, bypassing the obstruction is often difficult. Tracheal stents have been used with limited success in combination with treatment of chronic lower airway disease.

Trauma

Crush or bite injuries to the neck can result in fractures or avulsion of the laryngeal or tracheal cartilages. Bypassing the obstructed area may be necessary until the patient is stable and can undergo surgical correction of the injury. If there is avulsion of the cranial trachea, it may be difficult to intubate the patient. A long, rigid urinary catheter can be inserted past the area of avulsion into the distal segment, and an endotracheal tube passed over the rigid catheter, to establish a secure airway. Neck injury can also result in damage to the recurrent laryngeal nerve and laryngeal paralysis.

Foreign Bodies

Foreign bodies can lodge in the nasal cavity, pharynx, larynx, and distal trachea. Signs of foreign bodies in the nares include acute sneezing and pawing at or rubbing the muzzle on the ground. If the object is not removed, sneezing continues and a chronic nasal discharge develops. Respiratory distress is uncommon, but the foreign body is severely irritating. Pharyngeal and tracheal foreign bodies can cause severe obstruction to airflow and respiratory distress. Diagnosis of a foreign body is based on the patient history, physical examination findings, and thoracic or cervical radiographs. Smaller foreign bodies lodged in the distal airways may not be apparent radiographically but can cause pulmonary atelectasis.

Foreign bodies of the nose or pharynx can often be removed with alligator forceps with the patient under anesthesia. If removal is not possible with forceps, flushing the nasal cavity from cranial to caudal (pack the back of the mouth with gauze to prevent aspiration) can sometimes dislodge the foreign material into the gauze packing. Rhinoscopy may be necessary. If an endoscope is not available, an otoscope can be used.
Foreign objects lodged in the trachea can be small and function like a ball valve during inhalation and exhalation, causing episodic hypoxia and collapse. When attempting to remove these objects, suspend the patient with its head down. Remove the object with an alligator forceps, using a laryngoscope to aid in visualization. Foreign bodies lodged in the trachea or bronchi require removal with endoscopic assistance.

**Intraluminal Masses**

Nasopharyngeal polyps (in cats, tumors, obstructive laryngitis, granulomas, abscesses, and cysts) can cause upper airway obstruction. Clinical signs are usually gradual in onset. The lesions can be identified through careful laryngoscopic examination performed with the patient under general anesthesia. The nasopharynx above the soft palate should always be included in the examination. Pedunculated masses and cysts are excised at the time of evaluation. Biopsy of diffusely infiltrative masses is indicated for histologic examination and prognosis. It is impossible to distinguish obstructive laryngitis from neoplasia based on gross appearance alone. Whenever possible, material should be collected from abscesses and granulomas for cytologic evaluation and bacterial culture.

**Extraluminal Masses**

Extraluminal masses impinge on and slowly compress the upper airways, resulting in slow progression of clinical signs. Masses are usually identified by palpation of the neck. Enlarged mandibular lymph nodes, thyroid tumors, and other neoplasms may be present. Diagnosis is usually based on a combination of radiography and ultrasonography. CT and/or MRI is helpful in identifying the full extent and invasiveness of the lesion. Definitive diagnosis is made with fine-needle aspiration or biopsy. Many thyroid tumors bleed excessively.

**Pleural Cavity Disease**

The inside of each side of the hemithorax is covered in parietal pleura. The lung lobes are covered in visceral pleura. The two surfaces are in close contact with each other and are contiguous at the hilum under normal circumstances. Pneumothorax refers to free air within the pleural space, accumulating between the parietal and visceral pleura. The term pleural effusion refers to fluid accumulation in that area but does not reflect the amount or type of fluid present. The mediastinal reflections of the pleura typically are thin in dogs and cats and usually, but not always, connect. Bilateral involvement of pneumothorax or pleural effusion is common. Both pneumothorax and pleural effusion compromise the lungs’ ability to expand and result in hypoxia and respiratory distress.

**Pneumothorax**

Pneumothorax can be classified as open or closed, simple or complicated, and tension pneumothorax. An open pneumothorax communicates with the external environment through a rent in the thoracic wall. A closed pneumothorax results from tears in the visceral pleura but does not communicate with the outside. A tension pneumothorax occurs as a result of a tear in the lung or chest wall that creates a flap valve, such that air is allowed to leave the lung and accumulate in the pleural space during inhalation, and closes to seal off exit of air from the pleural space during exhalation. Tension pneumothorax can cause rapid decline in cardiopulmonary status and death if not recognized and treated immediately. A simple pneumothorax is one that can be controlled with a simple thoracocentesis. Complicated pneumothorax involves repeated accumulation of air, requiring placement of a thoracic drainage catheter.

In many cases, pneumothorax develops as a result of trauma. Spontaneous pneumothorax occurs with rupture of cavitory lesions of the lung that may be congenital or acquired as a result of prior trauma, heartworm disease, airway disease (emphysema), paragonimiasis, neoplasia, or lung abscess. Pneumothorax also rarely occurs as a result of esophageal tears or esophageal foreign bodies.
Rapid circulatory and respiratory compromise after traumatic pneumothorax can develop as a result of open or tension pneumothorax, rib fractures, airway obstruction, pulmonary contusions, hemothorax, cardiac dysrhythmias, cardiac tamponade, and hypovolemic shock. Any patient that is rapidly decompensating after a traumatic episode must be quickly assessed, and emergency therapy initiated (see section on immediate management of trauma, A CRASH PLAN).

Diagnosis of pneumothorax is usually made based on a history of trauma; a rapid, shallow, restrictive respiratory pattern; and muffled heart and lung sounds on thoracic auscultation. The clinical signs and history alone should prompt the clinician to perform a bilateral diagnostic and therapeutic thoracocentesis before taking thoracic radiographs (see section on thoracocentesis). The stress of handling the patient for radiography can be deadly in severe cases of pneumothorax. Although the mediastinum on both sides of the thorax connects, it is necessary to perform thoracocentesis on both sides to ensure maximal removal of free air in the pleural space and allow maximal lung expansion. If negative pressure cannot be obtained, or if the patient rapidly reaccumulates air, place a thoracostomy tube connected to continuous suction. (See section on thoracostomy tube placement.)

**Management of Open Sucking Chest Wounds in Pneumothorax**

Treat all penetrating wounds to the thorax as open sucking chest wounds unless proved otherwise. To “close” an open sucking chest wound, clip the fur around the wound as quickly as possible, and place sterile lubricant jelly or antimicrobial ointment circumferentially around the wound. Cut a sterile glove to provide a covering. Place the covering over the wound, making sure to cover all of the sterile lubricant, thus creating a seal to close the wound temporarily from the external environment. Evaluate the patient’s thorax via thoracocentesis while placing a thoracostomy tube. Once the patient’s condition is stable, the open chest wound can be surgically explored, lavaged, and definitively corrected. All animals with open chest wounds should receive antibiotics (first-generation cephalosporin) to prevent infection. After stabilization, radiographs can be taken and evaluated. Pneumothorax is confirmed by evidence of elevation of the cardiac silhouette above the sternum, increased density of the pulmonary parenchymal tissue, free air in between the parietal and visceral pleura (making the outline of the lungs visible), and absence of pulmonary vascular structures in the periphery. Parenchymal lesions within the lungs are best identified after as much air as possible has been removed from the thorax. Obtain left and right lateral and ventrodorsal or dorsoventral views. A standing lateral view may reveal air- or fluid-filled cavitary masses. If underlying pulmonary disease is suspected as a cause of spontaneous pneumothorax, a transtracheal wash, fecal flotation, and heartworm test may be indicated.

**Treatment of Pneumothorax**

Treatment of pneumothorax includes immediate bilateral thoracocentesis, covering of any open chest wounds, administration of supplemental oxygen, and placement of a thoracostomy tube if negative pressure cannot be obtained or if air rapidly reaccumulates. Serial radiography, CT, or MRI should be performed in dogs with spontaneous pneumothorax, because the condition can be associated with generalized pulmonary parenchymal disease. Strict cage rest is required until air stops accumulating and the thoracostomy tube can be removed. The patient’s chest tube should be aspirated every 4 hours after continuous suction is discontinued. If no air reaccumulates after 24 hours, the chest tube can be removed. Exercise restriction is indicated for a minimum of 1 week. If bullae or mass lesions are present, exploratory thoracotomy should be considered as a diagnostic and potentially therapeutic option for long-term management in prevention of recurrence.

**Pleural Effusion**

Pleural fluid cytologic analysis is indicated for all patients with pleural effusion before administration of antibiotics. The general term pleural effusion means a collection of fluid in the space between the parietal and visceral pleura but does not indicate what kind or how much fluid is present. Clinical signs associated with pleural effusion depend on how much
fluid is present and how rapidly the fluid has accumulated. Clinical signs associated with pleural effusion include respiratory distress, reluctance to lie down, labored breathing with an abdominal component on exhalation, cough, and lethargy. Auscultation of the thorax may reveal muffled heart and lung sounds ventrally and increased lung sounds dorsally, although pockets of fluid may be present, depending on the chronicity of the effusion. Percussion of the thorax may reveal decreased resonance.

In stable patients, the presence of pleural effusion can be confirmed radiographically. Radiographic confirmation of the pleural effusion should include right and left lateral and dorsoventral or ventrodorsal views. A handling or standing lateral view should be obtained if an anterior mediastinal mass is suspected. The standing lateral view will allow the fluid to collect in the costophrenic recess.

In patients with respiratory distress, muffled heart and lung sounds, and suspicion of pleural effusion, thoracocentesis should be performed immediately. Thoracocentesis can be both therapeutic and diagnostic. Radiography is contraindicated because the procedure can cause undue stress and exacerbation of clinical signs in an unstable patient. Pleural effusion can cause severe respiratory distress and can be the result of a number of factors that must be considered when implementing an appropriate treatment plan. Pathology of the pleura is almost always a secondary process except for primary bacterial pleuritis and pleural mesotheliomas. Causes of pleural effusion in the cat and dog include pyothorax, FIP, CHF, chylothorax, heartworm disease, hemothorax, hypoalbuminemia, lung lobe torsions, neoplasia, diaphragmatic hernia, and pancreatitis (Box 1-62).

In stable animals, diagnosis of pleural effusion can be made based on thoracic radiography or ultrasound. Thoracic radiographs can show whether the pleural effusion is unilateral or bilateral. Effusions in dogs and cats are usually bilateral. The lung parenchyma and the cardiac silhouette cannot be fully evaluated until most of the fluid has been evacuated from the pleural cavity. After thoracocentesis, radiography should be performed with left and right lateral and ventrodorsal or dorsoventral views. In cases of suspected heart failure, echocardiography also is necessary.

Pleural fluid cytologic analysis is indicated for all patients with pleural effusion. Collect specimens before administering antibiotics, whenever possible, because treatment with antibiotics can make a septic condition (pyothorax) appear nonseptic. The remainder of the diagnostic workup and treatment is based on the type of fluid present (Table 1-51). The fluid may be a transudate, nonseptic exudate, septic exudate or chylos, hemorrhagic, or neoplastic. Ultrasonographic evaluation of the thorax can be helpful in identifying intrathoracic masses, diaphragmatic hernias, lung lobe torsions, and cardiac abnormalities. Unlike radiography, ultrasonography is facilitated by the presence of fluid in the pleural space.

Pyothorax

*Pyothorax* refers to a septic effusion of the pleural cavity. The infection is generally the result of a combination of aerobic and anaerobic bacteria. Rarely, fungal organisms are present. The source of the underlying organisms is rarely identified, particularly in cats, but infection can be caused by penetrating wounds through the chest wall or esophagus, migrating foreign bodies (especially grass awns), and primary lung infections. The most common organisms associated with pyothorax in the cat are *Pasteurella* species, *Bacteroides* species, and *Fusobacterium* species. Fever is often present in addition to clinical signs of pleural effusion. Septic shock is uncommon.

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**Box 1-62 Physiologic Processes Associated with Pleural Effusion**

- Imbalance of transpleural or hydrostatic or protein osmotic forces
- Change in membrane permeability
- Decrease in rate of fluid reabsorption
- Combination of foregoing mechanisms
<table>
<thead>
<tr>
<th>Transudates</th>
<th>Modified Transudates</th>
<th>Nonseptic Exudates</th>
<th>Septic Exudates</th>
<th>Chylous Effusions</th>
<th>Hemorrhagic Effusions</th>
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<td>Rare</td>
<td>Variable number Nondegenerative</td>
<td>Moderate Nondegenerative</td>
<td>Moderate to high number Nondegenerative to degenerative</td>
<td>Acute: low number Chronic: moderate number Nondegenerative</td>
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<td>Transudates</td>
<td>Modified Transudates</td>
<td>Nonseptic Exudates</td>
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<td>Chylous Effusions</td>
<td>Hemorrhagic Effusions</td>
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<td>Idiopathic</td>
<td>Trauma</td>
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<td>failure</td>
<td>transudates</td>
<td>Feline infectious</td>
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<td>Chronic</td>
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<td>Pyothorax</td>
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Diagnosis of pyothorax is made based on cytologic analysis and the demonstration of intracellular and extracellular bacteria, toxin neutrophils and macrophages, and sometimes the presence of sulfur granules. Gram stains of the fluid can assist in the initial identification of some organisms. Bacterial cultures are indicated for bacteria identification and antibiotic susceptibility testing. Administration of antibiotics before cytologic evaluation can cause a septic effusion to appear nonseptic.

Emergency treatment for pyothorax involves placement of an intravenous catheter, intravenous fluids to treat hypovolemic shock, and broad-spectrum antibiotics (ampicillin, 22 mg/kg IV q6-8h; and enrofloxacin, 2.5 mg/kg IV q12h). Chloramphenicol (45 to 60 mg/kg PO q8h) also is an appropriate antibiotic to use for penetration into pockets of fluid. Administration of a β-lactam antibiotic (ampicillin or amoxicillin, 25 to 50 mg/kg PO q12h) with a β-lactamase inhibitor (amoxicillin clavulanate, 20 mg/kg PO q12h, or ampicillin sulbactam) is helpful for achieving better coverage of Bacteroides species.

Treatment of pyothorax differs in the cat and dog. In the cat, placement of one or two thoracic drainage catheters is recommended to allow continuous drainage of the intrathoracic abscess. Inadequate drainage can result in treatment failure. Fluid should be evaluated and the pleural cavity lavaged with 10 mL of warmed 0.9% saline or lactated Ringer’s solution per kilogram every 8 hours. Approximately 75% of the infused volume should be recovered after each lavage.

In dogs, or in cats with refractory pyothorax, perform an exploratory thoracotomy to remove any nidus of infection. A foreign body that can be removed at the time of surgery may be visible, but this finding is rare. Antibiotics are indicated for a minimum of 6 to 8 weeks after removal of the thoracostomy tube. Early diagnosis and aggressive treatment result in a good prognosis in the majority of patients with pyothorax. In cats, clinical signs of ptetalism and hypothermia at the time of presentation worsen the prognosis.

**Chylothorax**

*Chylothorax* refers to the abnormal accumulation of chyle (lymphatic fluid) in the pleural cavity. The cisterna chili is the dilated collection pool of lymphatic ducts in the abdomen that accumulate chyle before entry into the thoracic duct located within the thoracic cavity. The thoracic duct enters the thorax at the aortic hiatus. Numerous tributaries or collateral ducts exist. The functions of the lymphatic vessels collectively serve to deliver triglycerides and fat-soluble vitamins into the peripheral vascular circulation. Damage of the thoracic duct or lymphatic system or obstruction to lymphatic flow can result in the development of chylous effusion in the pleural or peritoneal space.

It is difficult to identify chylous effusions based on their milky appearance alone. To identify a chylous effusion versus a pseudochylous effusion, the triglyceride and cholesterol levels of the fluid must be compared with those of peripheral blood. Chylous effusions have a higher triglyceride and lower cholesterol levels than peripheral blood. Pseudochylous effusions have a higher cholesterol and lower triglyceride levels than peripheral blood.

Disease processes that can result in chylous effusions are listed in the Box 1-63. Clinical signs associated with chylous effusion are typical of any pleural effusion and of the disease process that caused the effusion. Weight loss may be evident, depending on the chronicity of the process.

<table>
<thead>
<tr>
<th>Causes of Chyloous Effusion</th>
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<tbody>
<tr>
<td>Cardiac disease</td>
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<td>Diaphragmatic hernia</td>
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<td>Heartworm disease</td>
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<td>Idiopathic cause</td>
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<td>Immune-mediated lymphadenitis</td>
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<td>Lung lobe torsion</td>
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<td>Pericardial disease</td>
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<td>Thoracic duct rupture</td>
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<td>Thoracic lymphangiectasia</td>
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<td>Thoracic neoplasia</td>
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<tr>
<td>Trauma</td>
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<tr>
<td>Venous thrombi</td>
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The diagnosis is made based on thoracocentesis, cytology, and biochemical evaluation of the fluid (i.e., triglyceride and cholesterol levels). The fluid often appears milky or blood-tinged but can be clear if the patient has significant anorexia. Typical cytologic characteristics are listed in Table 1-52. Lymphangiography can be used to confirm trauma to the thoracic duct, but this is usually not necessary unless surgical ligation is going to be attempted. The diagnostic evaluation must also attempt to identify an underlying cause.

Therapy for chylothorax is difficult and primarily involves documentation and treatment of the underlying cause. If an underlying cause is not found, treatment is largely supportive and consists of intermittent thoracocentesis to drain the fluid as it accumulates and causes respiratory dysfunction, nutritional support, and maintenance of fluid balance. A variety of surgical techniques, including ligation of the thoracic duct, pleural-peritoneal shunts, and pleurodesis, have been attempted but have had limited success. Most recently, the combination of thoracic duct ligation with subtotal pericardectomy has been shown to improve surgical success rates in the treatment of chylothorax. Rutin, a bioflavonoid, has been used with limited success in the treatment of idiopathic chylothorax in cats. Prognosis in many cases of chylothorax is guarded.

**Hemothorax**

Extensive hemorrhage into the pleural cavity can cause fulminant respiratory distress resulting from sudden hypovolemia and anemia and interference with lung expansion. Hemothorax typically is associated with trauma, systemic coagulopathy, lung lobe torsions, and erosive lesions within the thorax (usually neoplasia). Diagnosis of hemothorax involves obtaining a fluid sample via thoracocentesis. Hemorrhagic effusion must be differentiated from systemic blood inadvertently collected during the thoracocentesis procedure. Unless the hemorrhage is peracute, fluid in cases of hemothorax is rapidly defibrinated and will not clot, has a PCV less than that of venous blood, and contains RBCs and macrophages. Hemorrhagic effusions also usually contain a disproportionately higher number of WBCs compared with peripheral blood.
Hemothorax commonly is the sole clinical sign observed in animals with vitamin K–antagonist rodenticide intoxication and systemic coagulopathy. Whenever an animal demonstrates signs of a hemorrhagic pleural effusion, perform coagulation testing immediately to determine whether a coagulopathy exists. The PT test is fast and can be performed as a cage-side test (see section on coagulopathy).

Therapy for hemorrhagic pleural effusions should address the blood and fluid loss. Administer intravenous crystalloid fluids and RBC products (see section on transfusion therapy). When necessary, administer coagulation factors in the form of fresh whole blood or fresh frozen plasma, along with Vitamin K₁ (5 mg/kg/day SQ in multiple sites with a 25-gauge needle). If severe respiratory distress is present, evacuate the blood within the pleural space via thoracocentesis until clinical signs of respiratory distress resolve. Fluid that remains aids in the recovery of the patient, because RBCs and proteins eventually will be reabsorbed. Autotransfusion can be performed to salvage blood and reinfuse it into the anemic patient. In cases of neoplastic or traumatic uncontrollable hemorrhagic effusions, surgical exploration of the thorax is warranted.

**Diaphragmatic Hernia**

Diaphragmatic hernia, or a rent in the diaphragm, can result in the protrusion of abdominal organs into the thoracic cavity and impair pulmonary expansion. Organs that are commonly herniated into the thorax include the liver, stomach, and small intestines. Diaphragmatic hernia usually is secondary to trauma but can occur as a congenital anomaly. In cases of trauma, rib fractures, pulmonary contusions, traumatic myocarditis, hemothorax, and shock are also often present concurrently with diaphragmatic hernia. Respiratory distress can be caused by any one or a combination of these lesions. Animals with prior or chronic diaphragmatic hernias may have minimal clinical signs despite the presence of abdominal organs within the thorax. Clinical signs of acute or severe diaphragmatic hernia include respiratory distress, cyanosis, and shock.

A diagnosis of diaphragmatic hernia is made based on the patient’s history (traumatic event), clinical signs, and radiographs. In some cases, ultrasonography or contrast peritoneography is necessary to confirm the diagnosis. Contrast radiographs may show the presence of the stomach or intestines within the thorax after oral administration of barium. Never administer barium directly into the peritoneal cavity or in cases of suspected gastrointestinal rupture.

Treatment of a patient with a diaphragmatic hernia includes cardiovascular and respiratory system stabilization before attempting surgical repair of the diaphragm. If the stomach is within the thorax, or if the patient’s respiratory distress cannot be alleviated with medical management alone, immediate surgery is necessary. If the respiratory distress is minimal and the stomach is not located within the thorax, surgery can be postponed until the patient is a more stable anesthetic candidate. At the time of surgery, the abdominal organs are replaced into the abdominal cavity, and the rent in the diaphragm is closed. Air must be evacuated from the thorax after closure of the diaphragm. If chronic diaphragmatic hernia is repaired, the complication of reexpansion pulmonary edema can occur.

**Cardiac Changes Associated with Thoracic Trauma**

Cardiac injury is a common complication secondary to blunt thoracic trauma. In most cases, cardiac injury manifests as arrhythmias, including multiple premature ventricular contractions (PVCs), ventricular tachycardia, ST segment depression or elevation secondary to myocardial hypoxemia, and atrial fibrillation (see section on cardiac emergencies). Myocardial infarction and cardiac failure can occur. Careful and repeated assessments of the patient’s BP and ECG tracing should be a part of any diagnostic workup for a patient that has sustained blunt thoracic trauma.

**Rib Fractures and Flail Chest**

Rib fractures are associated with localized pain and painful respiratory movements. Radiographs are helpful to confirm the diagnosis. Careful palpation may reveal crepitus and instability of the fractured ribs. Common problems associated with rib fractures include
pulmonary contusions, pericardial laceration, traumatic myocarditis, diaphragmatic hernia, and splenic laceration or rupture.

A flail segment results from rib fractures of more than three adjacent ribs that produce a “floating segment” of the chest wall. The flail segment moves paradoxically with respiration—that is, it moves inward during inhalation and outward during exhalation. Respiratory distress is associated with the pain caused by the fractures and the presence of traumatic underlying pulmonary pathology.

Therapy for rib fractures and flail chest includes administration of supplemental oxygen, treatment of pneumothorax or diaphragmatic hernia, and administration of systemic and local anesthesia to alleviate the discomfort associated with the fractures. Although controversial, positioning the patient with the flail segment up may reduce pain and improve ventilation. Avoid the use of chest wraps, which do nothing to stabilize the flail segment and can further impair respiratory excursions. After administration of a systemic analgesic, administer a local anesthetic at the dorsocaudal and ventrocaudal segment of each fractured rib, and in one rib in front of and behind the flail segment. Often, pulmonary function will improve once the pain associated with rib fractures has been adequately treated. In rare cases in which the flail segment involves five or more ribs, surgical stabilization may be necessary. Single rib fractures or smaller flail segments are allowed to heal on their own.

**PULMONARY DISEASES**

**Feline Bronchitis (Feline Lower Airway Disease, Asthma)**

Feline bronchitis has a variety of names (bronchial asthma, asthma, acute bronchitis, allergic bronchitis, chronic asthmatic bronchitis, feline lower airway disease) and refers to the acute onset of respiratory distress secondary to narrowing of the bronchi. Cats may have an acute onset of a severe restrictive respiratory pattern associated with lower airway obstruction. Acute bronchitis in cats typically has an inflammatory component in the lower airways, resulting in acute bronchoconstriction, excessive mucus production, and inflammatory exudates. In cats with chronic bronchitis, there may be damage of the bronchial epithelium and fibrosis of the airways. These patients often have a history of intermittent exacerbation of clinical signs, intermittent cough, and periods of normality throughout the year. Because there appears to be an allergic or inflammatory component in feline bronchitis, clinical signs can be acutely exacerbated by stress and the presence of aerosolized particles such as perfume, smoke, and carpet powders. Causes of feline bronchitis include heartworm disease, parasitic infestation (lungworms), and (rarely) bacterial infection.

On presentation, the patient should be placed in an oxygen cage and allowed to rest while being observed from a distance. Postpone performing stressful diagnostic procedures until the patient’s respiratory status has been stabilized. After careful thoracic auscultation, administer a short-acting bronchodilator (terbutaline, 0.01 mg/kg SQ or IM) along with a glucocorticosteroid (dexamethasone sodium phosphate, 1 mg/kg IM, SQ, IV) to alleviate immediate bronchospasm and airway inflammation.

**Diagnosis**

Clinical signs of feline bronchitis are characterized by a short, rapid respiratory pattern with prolonged expiration with an abdominal push. Wheezes may be heard on thoracic auscultation. In some cases, no abnormalities are found on auscultation, but abnormalities become acutely worse when the patient is stimulated to cough by tracheal palpation. Radiographs may reveal a hyperinflated lung field with bronchial markings and caudal displacement of the diaphragm. In some cases, consolidation of the right middle lung lobe is present. A complete blood count and serum biochemistry profile can be performed, but results usually are unrewarding. In endemic areas, a heartworm test is warranted. Fecal examination by flotation and the Baermann technique is helpful in ruling out lungworms and other parasites. Bronchoalveolar lavage or transtracheal wash is useful for cytologic and bacterial examination.
Management

Long-term management of feline bronchitis includes isolation from environmental exposure to potential allergens (litter dust, perfumes, smoke, incense, carpet powders) and treatment of bronchoconstriction and inflammation with a combination of oral and inhaled glucocorticosteroids and bronchodilators (see Table 1-52). Antibiotic therapy is contraindicated unless a pure culture of a pathogen is documented. Oral therapy with steroids and bronchodilators should be used for a minimum of 4 weeks after an acute exacerbation and then gradually decreased to the lowest dose possible to alleviate clinical signs. Metered dose inhalers are now available (www.aerokat.com) for administration of inhaled bronchodilators (90 mcg of albuterol per puff) and steroids. Fluticasone (Flovent, 100 mcg/puff) can be administered initially every 12 hours for 1 week and then decreased to once daily, in most cases. Inhaled glucocorticosteroids are not absorbed systemically, and therefore patients do not develop the adverse side effects sometimes documented with oral glucocorticosteroid administration. Because it takes time for glucocorticosteroids to reach peak effects in the lungs, administration of inhaled glucocorticosteroids should overlap with oral prednisolone administration for 5 to 7 days. In some cats with previously undocumented cardiac disease, the use of glucocorticoids has been associated with sodium and fluid retention to such an extent that pulmonary vascular overload and pulmonary edema have occurred.

Pulmonary Contusions

Pulmonary contusions are a common sequela of blunt traumatic injury. A contusion basically is a bruise characterized by edema, hemorrhage, and vascular injury. Contusions may be present at the time of presentation or can develop over the first 24 hours after injury. A diagnosis of pulmonary contusion can be made based on auscultation of pulmonary crackles, presence of respiratory distress, and the presence of patchy interstitial to alveolar infiltrates on thoracic radiographs. Radiographic signs can lag behind the development of clinical signs of respiratory distress and hypoxemia by 24 hours.

Treatment of pulmonary contusions is supportive. Administer supplemental oxygen in a manner that is least stressful for the animal. Arterial blood gas analysis or pulse oximetry can be used to determine the degree of hypoxemia and monitor the response to therapy. Intravenous fluids should be administered with caution to avoid exacerbating pulmonary hemorrhage or fluid accumulation in the alveoli. Treat other conditions associated with the traumatic event. Possible complications of pulmonary contusions are rare but include bacterial infection, abscessation, lung lobe consolidation, and the development of cavitary lesions. The routine use of antibiotics or steroids in cases of pulmonary contusions is contraindicated unless external wounds are present. Empiric antibiotic use without evidence of external injury or known infection can potentially increase the risk of a resistant bacterial infection. Steroids have been shown to decrease pulmonary alveolar macrophage function and impair wound healing and are contraindicated.

Aspiration Pneumonia

Aspiration pneumonia can occur in animals as a result of abnormal laryngeal or pharyngeal protective mechanisms or can be secondary to vomiting during states of altered mentation, including anesthesia, recovery from anesthesia, and sleep. Megaesophagus, systemic polyneuropathy, myasthenia gravis, and localized oropharyngeal defects such as cleft palate can increase the risk of developing aspiration pneumonitis. Iatrogenic causes of aspiration pneumonia include improper placement of nasogastric feeding tubes, overly aggressive force-feeding, and oral administration of drugs. Aspiration of contents into the airways can cause mechanical airway obstruction, bronchoconstriction, chemical damage to the alveoli, and infection. Severe inflammation and airway edema are common. Pulmonary hemorrhage and necrosis can occur.

Diagnosis of aspiration pneumonia is based on clinical signs of pulmonary parenchymal disease, a history consistent with vomiting or other predisposing causes, and thoracic radiographs demonstrating a bronchointerstitial to alveolar pulmonary infiltrate. The most common site is the right middle lung lobe, although the pneumonia
can occur anywhere, depending on the position of the patient at the time of aspiration. A transtracheal wash or bronchoalveolar lavage is useful for bacterial culture and susceptibility testing.

Treatment of aspiration pneumonia includes antibiotic therapy for the infection, administration of supplemental oxygen, and loosening of the debris in the airways. Administer intravenous fluids to maintain hydration. Nebulization with sterile saline and chest physiotherapy (coupage) should be performed at least every 8 hours. Antibiotics to consider in the treatment of aspiration pneumonia include ampicillin and enrofloxacins, amoxicillin-clavulanate, ampicillin-sulbactam, trimethoprim sulfat, and chloramphenicol. The use of glucocorticosteroids is absolutely contraindicated. Continue antibiotic therapy for a minimum of 2 weeks after the resolution of radiographic signs of pneumonia.

**Pulmonary Edema**

Pulmonary edema arises from the accumulation of fluid in the pulmonary interstitial alveolar spaces and the airways. Ventilation-perfusion abnormalities result in hypoxia. Pulmonary edema can be caused by increased pulmonary vasculature hydrostatic pressure, decreased pulmonary oncotic pressure, obstruction of lymphatic drainage, or increased capillary permeability. Multiple factors can occur simultaneously. The most common cause of edema is increased pulmonary hydrostatic pressure resulting from left-sided CHF. Decreased plasma oncotic pressure with albumin <1.5 g/dL can also result in accumulation of fluid in the pulmonary parenchyma. Overzealous intravenous crystalloid fluid administration can result in dilution of serum oncotic pressure and vascular overload. Obstruction of lymphatic drainage is usually caused by neoplasia. Other causes of pulmonary edema include pulmonary thromboembolic disease, severe upper airway obstruction (noncardiogenic pulmonary edema), seizures, and head trauma.

Increased capillary permeability is associated with a variety of diseases that cause severe inflammation (SIRS). The resultant pulmonary edema contains a high amount of protein and is known as acute respiratory distress syndrome (ARDS). ARDS can be associated with pulmonary or extrapulmonary causes, including direct lung injury from trauma, aspiration pneumonia, sepsis, pancreatitis, smoke inhalation, oxygen toxicity, electrocution, and immune-mediated hemolytic anemia with DIC.

Diagnosis of pulmonary edema is made based on clinical signs of respiratory distress and the presence of crackles on thoracic auscultation. In severe cases, cyanosis and fulminant blood-tined frothy edema fluid may be present in the mouth and nostrils. Immediate management includes administration of furosemide (up to 8 mg/kg IV, IM, SQ, PO q1-2h [canine], up to 4 mg/kg IV, IM, SQ, PO q1-2h [feline]) and supplemental oxygen. Sedation with low-dose morphine sulfate (0.025 to 0.1 mg/kg IV) is helpful in dilating the splanchnic capacitance vasculature and relieving anxiety for the patient. If fluid overload is suspected secondary to intravenous fluid administration, fluids should be discontinued. Severely hypoalbuminemic patients should receive concentrated human albumin (2 mL/kg of a 25% solution) or fresh frozen plasma. Furosemide as a CRI (0.66 to 1.0 mg/kg/hr) also can dilate the pulmonary vasculature and decrease fluid accumulation in cases of ARDS. After initial stabilization of the patient, thoracic radiographs and an echocardiogram should be assessed to determine cardiac side, pulmonary vascular size, and cardiac contractility. Further diagnostic testing may be required to determine other underlying causes of pulmonary edema.

Heart failure is managed with vasodilators, diuretics, oxygen, and sometimes positive inotropes. Treatment ultimately consists of administration of supplemental oxygen, minimal stress and patient handling, and judicious use of diuretics. In cases of cardiogenic pulmonary edema, administer furosemide (up to 8 mg/kg IV, IM, SQ, PO q1-2h [canine], up to 4 mg/kg IV, IM, SQ, PO q1-2h [feline]) every 30 to 60 minutes until the patient loses 7% of its body weight. Positive inotropic and antiarrhythmic therapy may be necessary to improve cardiac contractility and control dysrhythmias. The clinician
should determine whether the cause of the pulmonary edema is secondary to CHF with pulmonary vascular overload, volume overload, hypoalbuminemia, or increased permeability (ARDS). Pulmonary edema secondary to ARDS typically is refractory to supplemental oxygen and diuretic therapy. In many cases, mechanical ventilation should be considered.

**PULMONARY THROMBOEMBOLISM**

A diagnosis of PTE is difficult to make and is based on clinical signs of respiratory distress consistent with PTE, lack of other causes of hypoxemia, a high index of suspicion in susceptible animals, the presence of a condition associated with PTE, and radiographic findings. Virchow's triad consists of vascular endothelial injury, sluggish blood flow with increased vascular stasis, and a hypercoagulable state as predisposing factors for thromboembolic disease. Clinical conditions that predispose an animal to PTE include hyperadrenocorticism, DIC, catheterization of blood vessels, bacterial endocarditis, protein-losing nephropathy or enteropathy, hyperviscosity syndromes, heat-induced illness, pancreatitis, diabetes mellitus, inflammatory bowel disease, and immune-mediated hemolytic anemia. Definitive diagnosis requires angiography or a lung perfusion scan.

Clinical signs associated with PTE include an acute onset of tachypnea, tachycardia, orthopnea, and cyanosis. If the embolism is large, the patient may respond poorly to supplemental oxygen administration. Pulmonary hypertension can cause a split second heart sound on cardiac auscultation. In some cases a normal thoracic radiograph is present in the face of severe respiratory distress. This is a classic finding in cases of PTE. Potential radiographic abnormalities include dilated, tortuous, or blunted pulmonary arteries; wedge-shaped opacities in the lungs distal to an obstructed artery; and interstitial to alveolar infiltrates. The right side of the heart may be enlarged.

Echocardiography can show right-sided heart enlargement, tricuspid regurgitation, pulmonary hypertension, and evidence of underlying cardiac disease, possibly with clots in the atria. Measurement of AT and D-dimer levels can be useful in the identification of hypercoagulable states, including DIC. Treatment of any patient with AT deficiency or DIC includes replenishment of AT and clotting factors in the form of fresh frozen plasma.

Treatment of PTE includes therapy for cardiovascular shock, oxygen supplementation, and thrombolytic therapy (see section on thromboembolic therapy). For short-term treatment, administer heparin (heparin sodium, 200-500 units/kg SQ once [canine]; 200-300 units/kg SQ once [feline], followed by 100 units of unfractionated heparin per kilogram q8h; or fractionated heparin). Thrombolytic therapy may include t-PA, streptokinase, or urokinase. Long-term therapy with low–molecular-weight heparin or warfarin may be required to prevent further thromboembolic events. Ideally, management should include treatment and elimination of the underlying disease.

**SMOKE INHALATION**

Smoke inhalation commonly occurs when an animal is trapped in a burning building. The most severe respiratory complications of smoke inhalation are seen in animals that are close enough to the flames to also sustain burn injuries (see section on burn injury). At the scene, many animals are unconscious from the effects of hypoxia, hypercapnia, carbon monoxide intoxication, and hydrogen cyanide gases that accumulate in a fire. Carbon monoxide produces hypoxia by avidly binding to and displacing oxygen binding to hemoglobin, resulting in severe impairment of oxygen-carrying capacity. The percentage of carboxyhemoglobin in peripheral blood depends on the amount of carbon monoxide in inhaled gases and the length of time of exposure. Clinical signs of carbon monoxide intoxication include cyanosis, nausea, vomiting, collapse, respiratory failure, loss of consciousness, and death.

Smoke inhalation of superheated particles also causes damage to the upper airways and respiratory tree. The larynx can become severely edematous and obstruct inspiration.
Emergency endotracheal intubation, tracheal oxygen, or tracheostomy tube may be required in the initial resuscitation of the patient, depending on the extent of airway edema. Inhalation of noxious gases and particles can cause damage to the terminal respiratory bronchioles. Specific noxious gases that can cause alveolar damage include combustible particles from plastic, rubber, and other synthetic products. Pulmonary edema, bacterial infection, and ARDS can result.

In any case of smoke inhalation, the first and foremost treatment is to get the animal away from the source of the flames and smoke and administer supplemental oxygen at the scene. At the time of presentation, carefully examine the animal’s eyes, mouth, and oropharynx. Suction soot and debris from the mouth and upper airways. Evaluate the patient’s respiratory rate, rhythm, and pulmonary sounds. Arterial blood gases should be analyzed with co-oximetry to evaluate the PaO₂ and carboxyhemoglobin concentrations. Evaluation of SaO₂ by pulse oximetry is not accurate in cases of smoke inhalation, as the PaO₂ may appear normal, even when large quantities of carboxyhemoglobin are present. Radiographs are helpful in determining the extent of pulmonary involvement, although radiographic signs may lag behind the appearance of clinical respiratory abnormalities by 16 to 24 hours. Bronchoscopy and bronchoalveolar lavage provide a more thorough and accurate evaluation of the respiratory tree; however, these procedures should be performed only in patients whose cardiovascular and respiratory status is stable.

Management of the patient with smoke inhalation includes maintaining a patent airway, administering supplemental oxygen, correcting hypoxemia and acid-base abnormalities, preventing infection, and treating thermal burns (see section on burn injury). If severe laryngeal edema is present, a temporary tracheostomy may be necessary to allow adequate oxygenation and ventilation. Glucocorticosteroids should not be empirically used in the treatment of smoke inhalation, because of the risk of decreasing pulmonary alveolar macrophage function and increasing the potential for infection. In cases of severe laryngeal edema, however, glucocorticosteroids may be necessary to decrease edema and inflammation. The use of empiric antibiotics is contraindicated unless clinical signs of deterioration and bacterial pneumonia develop.

**Epistaxis**

Epistaxis can be caused by facial trauma, a foreign body, bacterial or fungal rhinitis, neoplasia, coagulopathies, and systemic hypertension. Acute, severe bilateral hemorrhage without exudate is suggestive of a systemic disorder. A history of chronic nasal discharge usually accompanies nasal disease. Acute unilateral epistaxis can occur with nasal or systemic disease.

In most cases, cage rest is sufficient to temporarily diminish blood loss. Sedation (acepromazine, 0.02 to 0.05 mg/kg IV, IM, SQ) may be helpful in alleviating anxiety and decreasing BP. The hypotensive effects of acepromazine are potentially harmful if severe blood loss has occurred. If evidence of hypovolemia is present (see section on hypovolemic shock), intravenous fluid resuscitation should be administered.

Rapid assessment of clotting ability, with a platelet count estimate and clotting profile (ACT or APTT and PT), should be performed. If epistaxis secondary to vitamin K–antagonist rodenticide intoxication is suspected, administer vitamin K₁ and fresh frozen plasma or fresh whole blood.

Persistent hemorrhage from a nasal disorder can be treated with dilute epinephrine (1:100,000) into the nasal cavity with the nose pointed toward the ceiling to promote vasoconstriction. If this fails, the animal can be anesthetized, the nasal cavity packed with gauze, and the caudal oropharynx and external nares covered with umbilical tape to control hemorrhage. Rhinoscopy should be performed to determine the cause of ongoing hemorrhage. Continued excessive hemorrhage can be controlled with ligation of the carotid artery on the side of the hemorrhage or with percutaneous arterial embolization.
Additional Reading


SUPERFICIAL SOFT TISSUE INJURIES

Wounds have been classified in several ways according their degree of tissue integrity, causative force, degree of contamination and duration, and degree of contamination and infection (Table 1-53). There are also unique causes of wounds such as burns, psychogenic dermatoses, frostbite, decubital ulcers, and snake bite.

The animal should be transported to the nearest veterinary facility for definitive care. The wound should be covered or packed with dry gauze or clean linen to protect the wound and to prevent further hemorrhage and contamination. If an open fracture is present, the limb should be splinted without placing the exposed bone back into the wound. Replacing the exposed bone fragment back through the skin wound can cause further damage to underlying soft tissue structures and increase the degree of contamination of deeper tissues. If a spinal fracture is suspected, the patient should be transported on a stable flat surface to prevent further spinal mobilization and neurologic injury.

At the time of presentation, first refer to the ABCs of trauma care, taking care to evaluate and stabilize the patient’s cardiovascular and respiratory status. After a complete physical examination and history, ancillary diagnostic techniques can be performed if the patient is hemodynamically stable (see section on triage, assessment, and treatment of emergencies).

WOUND MANAGEMENT

Initially, every patient with a superficial wound should receive some degree of analgesia and an injection of a first-generation cephalosporin, preferably within 3 hours of the injury. Evaluate the wound after the patient’s cardiovascular and respiratory status have been stabilized. Always cover an open wound before taking an animal to the hospital, to prevent a
Evaluate limb wounds for neural, vascular, and orthopedic abnormalities. Carefully examine the structures deep to the superficial wounds. When there has been a delay in assessment of the wound, obtain samples for culture and antimicrobial susceptibility testing. If the wound is older and obviously infected, a Gram stain can help guide appropriate antimicrobial therapy pending results of culture and susceptibility testing. Place a support bandage saturated with a water-soluble antibiotic ointment or nonirritating antimicrobial solution (e.g., 0.05% chlorhexidine, if bone or joint tissue is not exposed) around the wound. In addition to a first-generation cephalosporin, other appropriate antibiotic choices include amoxicillin-clavulanate, trimethoprim-sulfadiazine, amoxicillin, and ampicillin. If gram-negative flora are present, administer enrofloxacin. Administer the antibiotics of choice for a minimum of 7 days unless a change of antibiotic therapy is indicated.

Modified from Swaim SF, Henderson RA: Small animal wound management, ed 2, Media, Pa, 1997, Williams & Wilkins.

<table>
<thead>
<tr>
<th>T A B L E 1 - 53</th>
<th>Classification of Soft Tissue Wounds</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Classification</strong></td>
<td><strong>Characteristics</strong></td>
</tr>
<tr>
<td>Open</td>
<td>Lacerations or skin loss</td>
</tr>
<tr>
<td>Closed</td>
<td>Crushing injuries and contusions</td>
</tr>
<tr>
<td><strong>Causative Force</strong></td>
<td></td>
</tr>
<tr>
<td>Abrasion</td>
<td>Loss of epidermis and portions of dermis, usually caused by shearing between two compressive surfaces</td>
</tr>
<tr>
<td>Avulsion</td>
<td>Tearing of tissue from its attachment because of forces similar to those causing abrasion but of a greater magnitude</td>
</tr>
<tr>
<td>Incision</td>
<td>Wound created by a sharp object; wound edges are smooth and there is minimal trauma in the surrounding tissues</td>
</tr>
<tr>
<td>Laceration</td>
<td>Irregular wound caused by tearing of tissue with variable damage to the superficial and underlying tissue</td>
</tr>
<tr>
<td>Puncture</td>
<td>Penetrating wound caused by a missile or sharp object; superficial damage may be minimal; damage to deeper structures may be considerable; contamination by fur and bacteria with subsequent infection is common</td>
</tr>
<tr>
<td><strong>Degree of Contamination and Duration</strong></td>
<td></td>
</tr>
<tr>
<td>Class I</td>
<td>0-6 hours with minimal contamination</td>
</tr>
<tr>
<td>Class II</td>
<td>6-12 hours with significant contamination</td>
</tr>
<tr>
<td>Class III</td>
<td>&gt;12 hours with gross contamination</td>
</tr>
<tr>
<td><strong>Degree of Contamination and Infection</strong></td>
<td></td>
</tr>
<tr>
<td>Clean wound</td>
<td>Surgically created under aseptic conditions; no invasion of the respiratory, gastrointestinal, or genitourinary tracts or of the oropharyngeal cavity</td>
</tr>
<tr>
<td>Clean contaminated wound</td>
<td>Minimal contamination, and contamination can be removed effectively; includes operative wounds involving the respiratory, gastrointestinal, and genitourinary tracts</td>
</tr>
<tr>
<td>Contaminated wound</td>
<td>Open traumatic wound with heavy contamination and possibly foreign debris; includes operative wounds with major breaks in aseptic technique and incisions in areas of acute nonpurulent inflammation adjacent to inflamed or contaminated skin</td>
</tr>
<tr>
<td>Dirty or infected wound</td>
<td>Old traumatic wound and wounds with clinical signs of infection or perforated viscera</td>
</tr>
</tbody>
</table>

nosocomial infection. Evaluate limb wounds for neural, vascular, and orthopedic abnormalities. Carefully examine the structures deep to the superficial wounds.

When there has been a delay in assessment of the wound, obtain samples for culture and antimicrobial susceptibility testing. If the wound is older and obviously infected, a Gram stain can help guide appropriate antimicrobial therapy pending results of culture and susceptibility testing. Place a support bandage saturated with a water-soluble antibiotic ointment or nonirritating antimicrobial solution (e.g., 0.05% chlorhexidine, if bone or joint tissue is not exposed) around the wound. In addition to a first-generation cephalosporin, other appropriate antibiotic choices include amoxicillin-clavulanate, trimethoprim-sulfadiazine, amoxicillin, and ampicillin. If gram-negative flora are present, administer enrofloxacin. Administer the antibiotics of choice for a minimum of 7 days unless a change of antibiotic therapy is indicated.
At the time of wound cleansing or definitive wound repair, the patient should be placed under general anesthesia with endotracheal intubation, unless the procedure will be brief (i.e., less than 10 minutes). In such cases, a short-acting anesthetic combination (analgésia + propofol, analgesia + ketamine and diazepam) can be administered to effect. Heavy sedation with infiltration of a local anesthetic may also be appropriate for very small wounds, depending on the location of the wound and temperament of the patient. Protect the wound by packing it with sterile gauze sponges soaked in sterile saline, or with water-soluble lubricating gel such as K-Y jelly.

Clip the fur surrounding the wound, moving from the inner edge of the wound outward, to help prevent wound contamination with fur or other debris. Scrub the wound and surrounding skin with an antimicrobial soap and solution such as dilute chlorhexidine until the area is free of all gross debris. Gross debris within the wound itself can be flushed using a 30-mL syringe filled with sterile saline or lactated Ringer’s solution and an 18-gauge needle. Pressure-lavage systems are also available for use, if desired. Grossly contaminated wounds can be rinsed first with warm tap water to eliminate gross contamination, and then prepared as just described.

Debride the wound, removing skin and other soft tissue that is not obviously viable. Obviously viable and questionable tissue should remain, and the wound should be left open for frequent reassessment on a daily basis. Remove any dark or white segments of skin. Questionable skin edges may or may not regain viability and should be left in place for 48 hours, so the wound can fully reveal itself. Excise grossly contaminated areas of fat and underlying fascia. Blood vessels that are actively bleeding should be ligated to control hemorrhage, if collateral circulation is present.

If nerve bundles are ligated cleanly in a clean wound, the nerve edges should be reapposed and anastomosed. If gross contamination is present, however, definitive neurologic repair should be delayed until healthy tissue is present. Excise contaminated muscle until healthy bleeding tissue is present. Anastamose tendon lacerations if the wound is clean and not grossly contaminated. If gross contamination is present, the tendon can be temporarily anastomosed and a splint placed on the limb until definitive repair of healthy tissue is possible.

Thoroughly lavage open wounds to a joint with sterile saline or lactated Ringer’s solution. Infusion of chlorhexidine or povidone-iodine solution into the joint can cause a decrease in cartilage repair and is contraindicated. Smooth sharp edges and remove any obvious fragments. Whenever possible, the joint capsule and ligaments should be partially or completely closed. After removal of bullets and metal fragments, the subcutaneous tissue and skin should be left open to heal by second intention or should be partially closed with a drain. The joint should then be immobilized.

Injuries and exposed bone should be carefully lavaged, taking care to remove any gross debris without pushing the debris further into the bone and wound. The bone should be covered with a moist dressing and stabilized until definitive fracture repair can be made. This type of injury typically is seen with shearing injuries of the distal extremities caused by interaction with slow-moving vehicles. Perform wet-to-dry or enzymatic debridement until a healthy granulation bed is present.

If large areas of contamination are present (e.g., necrotizing fasciitis), en bloc debridement may be necessary. En bloc debridement consists of complete excision of badly infected wounds without entering the wound cavity, to prevent systemic infection. This technique should be used only if there is sufficient skin and soft tissue to allow later closure and it can be performed without damaging any major nerves, tendons, or blood vessels.

**Open Wounds**

Open wounds often are managed by second intention healing, delayed primary closure, or secondary closure. See the section on wound management and bandaging for a more complete discussion of the use of various bandaging materials in the treatment of open wounds.
Closed Wounds

If an animal is presented very shortly after a wound has occurred and contamination and trauma are minimal, the wound can be closed after induction of anesthesia and careful preparation of the wound and surrounding tissues. Close any dead space under the skin with absorbable suture material in an interrupted suture pattern. Avoid incising major blood vessels or nerves. Close the subcutaneous tissues with absorbable suture material in an interrupted or continuous suture pattern. Take care that there is not too much tension on the wound, or else surgical dehiscence will occur with patient movement. Close the skin with nonabsorbable suture or surgical staples (2-0 to 4-0).

If there is any doubt at the time of repair about tissue status or inability to close all dead space, place a passive drain (Penrose drain) so that the proximal end of the drain is anchored in the proximal aspect of the wound with a suture(s). Leave the ends long so that the suture can be accurately identified at the time of drain removal. Pass the suture through the skin, through the drain, and out the other side of the skin. Place the rest of the drain into the wound and then secure it at the most ventral portion of the wound or exit hole in the most dependent area of the body, to allow drainage and prevent seroma formation. Close the subcutaneous tissue over the drain before skin closure. During wound closure, be sure to not incorporate the subcutaneous or skin sutures into the drain, or it will not be possible to remove the drain without reopening the wound. Bandage the area to prevent contamination. The drain can be removed once drainage is minimal (usually 3 to 5 days).

Active drains can be constructed or purchased; their use is indicated in wounds that are free of material that can plug the drain. To construct a small suction drain, remove the female portion or catheter hub at the end of a butterfly catheter. Fenestrate the tubing so that there are multiple side holes, taking care to avoid making the holes larger than 50% of the circumference of the tubing. Place the tubing into the wound via a small stab incision distal to the wound. Use a purse-string suture around the tubing to facilitate a tight seal and prevent the tubing from exiting the wound. After wound closure, insert the butterfly needle into a 5- to 10-mL evacuated blood collection tube to allow fluid to drain into the tube. Incorporate the tube into the bandage, and replace it when it becomes full.

Alternatively, the butterfly portion of the system can be removed and the tube fenestrated as described previously. Place the tube into the wound and suture it in place to create a tight seal. Secure the catheter hub to a syringe in which the plunger has been drawn back slightly to create suction. Insert a metal pin or 16- to 18-gauge needle through the plunger at the top of the barrel to hold it at the desired level. Incorporate the suction apparatus into the bandage and replace it when it becomes full.

Delayed Primary Closure

Delayed primary closure should be considered when there is heavy contamination, purulent exudate, residual necrotic debris, skin tension, edema and erythema, and lymphangitis. Delayed primary closure usually is made 3 to 5 days after the initial wound infliction and open wound management has been performed. Once healthy tissue is observed, the skin edges should be debrided and the wound closed as with primary closure.

Secondary Wound Closure

Secondary wound closure should be considered when infection and tissue trauma necessitate open wound management for more than 5 days. Secondary wound closure is performed after the development of a healthy granulation bed. This technique also is useful when a wound has dehisced and has formed granulation tissue.

If the wound edges can be manipulated into apposition and if epithelialization has not begun, the wound can be cleansed and the wound edges apposed and sutured. This is known as early secondary closure.
Late secondary closure should be performed whenever there is a considerable amount of granulation tissue, the edges of the wound cannot be manipulated into position, and epithelialization has already started. In such cases, the wound should be cleaned, and the skin edges debrided to remove the epithelium. The remaining wound edges are then sutured over the granulation tissue (Table 1-54).

**Additional Reading**


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**TABLE 1-54 Complicating Factors Involving the Management of Superficial Soft Tissue Wounds**

<table>
<thead>
<tr>
<th>Circumstance</th>
<th>Potential Problem(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Improper handling of animal during transport</td>
<td>Further tissue and neurologic damage may occur (e.g., improper limb or spine immobilization).</td>
</tr>
<tr>
<td>Inadequate assessment of animal’s general condition or wounded tissues</td>
<td>Animal’s condition may worsen or animal may die; tissue injuries may be overlooked.</td>
</tr>
<tr>
<td>Inadequate wound protection during assessment, resuscitation, or stabilization procedures</td>
<td>Further wound contamination may occur at veterinary facility.</td>
</tr>
<tr>
<td>Inadequate wound protection while preparing the surrounding area</td>
<td>Further wound contamination with fur and debris may occur.</td>
</tr>
<tr>
<td>Insufficient wound lavage</td>
<td>Wound infection may occur.</td>
</tr>
<tr>
<td>Hydrogen peroxide wound lavage</td>
<td>Lavage offers little bactericidal activity and contributes to irritation of tissues and delayed healing.</td>
</tr>
<tr>
<td>Povidone-iodine wound lavage</td>
<td>Lavage has short residual activity and absorption with large wound.</td>
</tr>
<tr>
<td>Overly aggressive initial layered debridement</td>
<td>Debridement may result in the removal of viable tissue.</td>
</tr>
<tr>
<td>En bloc debridement</td>
<td>Debridement results in removal of large amounts of tissue and a large defect for closure.</td>
</tr>
<tr>
<td>Use of drains</td>
<td>Potential exists for bacteria to ascend along the drain, for drain removal by the animal or breakage of the drain, and for possible tissue emphysema with air being sucked under the skin with patient movement.</td>
</tr>
<tr>
<td>Tube-type drains</td>
<td>Drains may cause postoperative discomfort; fenestrations may become occluded to stop intraluminal drainage.</td>
</tr>
<tr>
<td>Deeply placed sutures in the presence of a drain</td>
<td>Drain may be incorporated into the repair and prevent drain removal.</td>
</tr>
<tr>
<td>Active drains</td>
<td>High negative pressure may cause tissue injury; highly productive wounds may necessitate changing the evacuated blood tubes several times a day with constructed drains.</td>
</tr>
</tbody>
</table>

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SHOCK

Shock is defined as a state of inadequate circulating volume and inability to meet cellular oxygen demands. There are three types of shock: hypovolemic, cardiogenic, and septic. Early recognition of the type of shock present is crucial in the successful clinical management of shock syndrome. Tissue oxygen delivery is based on cardiac output and arterial oxygen concentration. Knowledge of the components of normal oxygen delivery is essential to the treatment of shock in the critical patient.

Oxygen delivery (\(\text{DO}_2\)) = Cardiac output (Q) × Arterial oxygen content (\(\text{CaO}_2\))

where Q = heart rate x stroke volume. Stroke volume is affected by preload, afterload, and cardiac contractility.

\[
\text{CaO}_2 = [(1.34 \times \text{Hb} \times \text{SaO}_2)] + (0.003 \times \text{PaO}_2)
\]

where Hb = hemoglobin concentration, \(\text{SaO}_2\) = oxygen saturation, and \(\text{PaO}_2\) = arterial partial pressure of oxygen in millimeters of mercury.

Therefore factors that can adversely affect oxygen delivery include inadequate preload or loss of circulating volume, severe peripheral vasoconstriction and increased afterload, depressed cardiac contractility, tachycardia and decreased diastolic filling, cardiac dysrhythmias, inadequate circulating hemoglobin, and inadequate oxygen saturation of hemoglobin. During septic shock, enzymatic dysfunction and decreased cellular uptake and use of oxygen also contribute to anaerobic glycolysis.

An inadequate circulating volume may develop secondary to maldistribution of available blood volume (traumatic, septic, and cardiogenic origin) or as a result of absolute hypovolemia (whole blood or loss of extracellular fluid). Normally the animal compensates by (1) splenic and vascular constriction to translocated blood from venous capacitance vessels to central arterial circulation, (2) arteriolar constriction to help maintain diastolic BP and tissue perfusion, and (3) an increase in heart rate to help maintain cardiac output. Arteriolar vasoconstrictions support perfusion to the brain and heart at the expense of other visceral organs. If vasoconstriction is severe enough to interfere with delivery of adequate tissue oxygen for a sufficient period of time, the animal may die.

Hypovolemic Shock

Hypovolemic shock can result from acute hemorrhage or from severe fluid loss from vomiting, diarrhea, or third spacing of fluids. Early in shock, baroreceptors in the carotid body and aortic arch sense a decrease in wall stretch from a decrease in circulating fluid volume. Tonic inhibition of sympathetic tone via vagal stimulation is diminished, and heart rate and contractility increase and peripheral vessels constrict to compensate for the decrease in cardiac output. The compensatory mechanisms protect and support blood supply to the brain and heart at the expense of peripheral organ perfusion. This is called early compensatory shock.

Early compensatory shock is characterized by tachycardia, normal to fast capillary refill time, tachypnea, and normothermia. As shock progresses, the body loses its ability to compensate for ongoing fluid losses. Early decompenatory shock is characterized by tachycardia, tachypnea, delayed capillary refill time, normotension to hypotension, and a fall in body temperature. End-stage decompenatory shock is characterized by bradycardia, markedly prolonged capillary refill time, hypothermia, and hypotension. Aggressive treatment is necessary for any hope of a favorable outcome.

Septic Shock

Septic shock should be considered in any patient with a known infection, recent instrumentation that could potentially introduce infection (indwelling intravenous or urinary catheter, surgery or penetrating injury), disorders or medical therapy that could compromise immune function (diabetes mellitus, immunodeficiency virus, parvovirus or feline
panleukopenia virus infection, stress, malnutrition, glucocorticoids, chemotherapy). The presence of bacteria, viruses or rickettsiae, protozoa, or fungal organisms in the blood constitutes septicemia. Septic shock is characterized by the presence of sepsis and refractory hypotension that is unresponsive to standard aggressive fluid therapy and inotropic or pressor support. Septic shock and other causes of inflammation can lead to SIRS. In animals, the presence of two or more of the criteria in Table 1-55 in the presence of suspected inflammation or sepsis constitutes SIRS.

Clinical signs associated with sepsis may be vague and nonspecific, including weakness, lethargy, vomiting, and diarrhea. Cough and pulmonary crackles may be associated with pneumonia. Decreased lung sounds may be associated with pyothorax. Abdominal pain and fluid may be associated with septic peritonitis. Vaginal discharge may or may not be present in patients with pyometra. Diagnostic tests should include a WBC count, serum biochemical profile, coagulation tests, thoracic and abdominal radiographs, and urinalysis.

The WBC count in a septic patient that is appropriately responding to the infection will be elevated, with a left-shifted neutrophilia and leukocytosis. A degenerative left shift, with leukopenia with elevated band neutrophils, suggests an overwhelming infection. Biochemical analyses may demonstrate hypoglycemia and nonspecific hepatocellular and cholestatic enzyme elevations. In the most severe cases, metabolic (lactic) acidosis, coagulopathies, and end-organ failure, including anuria and ARDS, may be present.

**Cardiogenic Shock**

Cardiogenic shock occurs as a result of cardiac output inadequate to meet cellular oxygen demands. Cardiogenic shock is associated with primary cardiomyopathies, cardiac dysrhythmias, pericardial fluid, and pericardial fibrosis. Abnormalities seen on physical examination often are similar to those seen in other categories of shock, but they can also include cardiac murmurs, dysrhythmias, pulmonary rales, bloody frothy pulmonary edema fluid from the nares or mouth, orthopnea, and cyanosis. It is important to distinguish the primary cause of shock before implementing treatment (Table 1-56), whenever possible, because the treatment for a suspected ruptured hemangiosarcoma differs markedly from the treatment for end-stage dilatative cardiomyopathy. The patient’s clinical signs may be similar and include a peritoneal fluid wave, but the treatment for hypovolemia can dramatically worsen the CHF secondary to dilatative cardiomyopathy.

When a patient has some form of shock, immediate vascular access is of paramount importance. Place a large-bore peripheral or central venous catheter for the infusion of crystalloid or colloid fluids, blood component therapy, and drugs. Monitor the patient’s cardiopulmonary status (by ECG), BP, oxygen saturation (as determined by pulse oximetry or arterial blood gas analyses), hematocrit, BUN, and glucose. Ancillary diagnostics, including thoracic and abdominal radiography, urinalysis, serum biochemistry profile, coagulation tests, complete blood count, abdominal ultrasound, and echocardiography, should be performed as determined by the individual patient’s needs and the type of shock.

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Dogs</th>
<th>Cats</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temperature</td>
<td>&lt;100° F or &gt; 103.5° F</td>
<td>&lt;100° F or &gt; 103.5° F</td>
</tr>
<tr>
<td>Heart rate</td>
<td>&gt; 120 beats/min in dogs</td>
<td>&lt; 140 or &gt; 250 beats/min in cats</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>&gt; 20 breaths/min or Paco₂ &lt; 32 mm Hg</td>
<td>&gt; 40 breaths/min or Paco₂ &lt; 32 mm Hg</td>
</tr>
<tr>
<td>White blood cell count</td>
<td>&gt; 18,000 cells/mL or &lt; 4000 cells/mL or &gt; 10% bands</td>
<td>19,000 cells/mL or &lt; 5000 cells/mL or &gt; 10% bands</td>
</tr>
</tbody>
</table>
**Management of the Shock Patient**

**The Rule of 20**

The following list, called the Rule of 20, is a guideline for case management of the shock patient. Consideration of each aspect of the Rule of 20 on a daily basis ensures that major organ systems are not overlooked. The list also provides a means to integrate and relate changes in different organ systems functions with one another.

1. **Fluid Balance**

The treatment of hypovolemic and septic shock requires the placement of large-bore intravenous catheters in peripheral and central veins. If vascular access cannot be obtained percutaneously or by cutdown methods, intrasosseous catheterization should be considered. Once vascular access is achieved, rapidly administer large volumes of crystalloid or colloid fluids. As a rule of thumb, administer \( \frac{1}{4} \) of a calculated shock dose of fluids—that is, \( \frac{1}{4} \times (90 \text{ mL/kg/hr}) \) in dogs and \( \frac{1}{4} \times (44 \text{ mL/kg/hr}) \) in cats—of a balanced crystalloid fluid (Normosol-R, Plasma-Lyte M, lactated Ringer’s solution, or 0.9% sterile saline). Reassess the patient’s perfusion parameters (heart rate, capillary refill time, BP, urine output) on a continual basis to direct further fluid therapy. Synthetic colloid fluids (hydroxyethyl starch) can also be administered in the initial resuscitation from shock. A guideline is to administer 5 to 10 mL/kg of hydroxyethyl starch as a bolus over 10 to 15 minutes and then reassess perfusion parameters.1 Hypertonic saline (0.7% NaCl, 4 mL/kg) can be used in cases of hemorrhagic shock to temporarily restore intravascular fluid volume by drawing fluid from the interstitial space. Because this type of fluid resuscitation is short-lived, hypertonic saline should always be used with another crystalloid or colloid fluid, and it should not be used in patients with interstitial dehydration. If hemorrhagic shock is present, the goal should be to return a patient’s BP to normal (not supraphysiologic) levels (i.e., systolic pressure 90 to 100 mm Hg, diastolic

---


pressure >40 mm Hg, and mean arterial pressure ≥60 mm Hg) to avoid iatrogenically causing clots to fall off and hemorrhage to start again.

In critically ill patients, fluid loss can be measured in the form of urine, vomit, diarrhea, body cavity effusions, and wound exudates. In addition, insensible losses (those that cannot be readily measured from sweat, panting, and cellular metabolism) constitute 20 mL/kg/day. Measurement of fluid “ins and outs” in conjunction with the patient’s CVP, hematocrit, albumin, and colloid oncotic pressure can help guide fluid therapy (see also section on fluid therapy).

2. Blood Pressure
Maintenance of normotension is necessary for adequate oxygen delivery to meet cellular energy demands. BP can be measured using direct arterial catheterization, or through indirect means such as Doppler plethysmography or oscillometric methods. The systolic pressure should remain at or greater than 90 to 100 mm Hg at all times. The diastolic pressure is very important, too, as it constitutes two thirds of the mean arterial pressure; it must be greater than 40 mm Hg for coronary artery perfusion. The mean arterial pressure should be greater than 60 mm Hg for adequate tissue perfusion.

If fluid resuscitation and pain management are not adequate in restoring BP to normal, vasoactive drugs including positive inotropes and pressors should be considered (Table 1-57).

In cases of cardiogenic shock, vasodilator drugs (Table 1-58) can be used to decrease vascular resistance and afterload. Low-dose morphine (0.025-0.05 mg/kg IV, IM) dilates splanchnic vessels and helps reduce pulmonary edema. Furosemide (1 mg/kg/hr) also can dilate pulmonary vasculature and potentially reduce edema fluid formation in cases of ARDS.

3. Heart Rate, Rhythm, and Contractility and Pulse Quality
Cardiac output is a function of both heart rate and stroke volume. Stroke volume (or the amount of blood that the ventricle pumps in 1 minute) is affected by preload, afterload, and contractility. During hypovolemic shock, there is a fall in cardiac preload owing to a decrease in circulating blood volume. During septic and cardiogenic shock, there is a decrease in contractility secondary to inherent defects of the myocardium or because of the negative inotropic effects of inflammatory cytokines such as tumor necrosis factor (TNF)-α, myocardial depressant factor, interleukin (IL)-1, and IL-10 released during sepsis and systemic inflammation. Afterload also may be increased because of the compensatory mechanisms and neurohumoral activation of the renin-angiotensin-aldosterone axis in hypovolemic or cardiogenic shock. As heart rate increases to compensate for a decline in cardiac output, myocardial oxygen demand increases and diastolic filling

<table>
<thead>
<tr>
<th>Drug</th>
<th>Receptor Activity</th>
<th>Dose (Intravenous)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td>DA₁, DA₂, α⁺⁺⁺,</td>
<td>5-25 mcg/kg/min (blood pressure support)*</td>
</tr>
<tr>
<td></td>
<td>β⁺⁺⁺</td>
<td>1-5 mcg/kg/min (renal afferent diuresis)</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>α⁺, β⁺⁺⁺</td>
<td>3-20 mcg/kg/min* (blood pressure support, positive inotrope)</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>α⁺⁺⁺, β⁺</td>
<td>0.05-0.3 mg/kg/min; 0.01-0.02 mg/kg</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>α⁺⁺⁺, β₀</td>
<td>1-3 mcg/kg/min CRI</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>α⁺⁺⁺, β⁺⁺⁺</td>
<td>0.02-0.5 mg/kg; 0.1-1 mcg/kg/min CRI</td>
</tr>
</tbody>
</table>

+++ , Strong receptor activity; +, weak receptor activity; 0, no receptor activity.
* Monitor for tachyarrhythmias at higher doses.
Because the coronary arteries are perfused during diastole, coronary perfusion can be impaired, and myocardial lactic acidosis can develop, causing a further decline in contractility. In addition to lactic acidosis, acid-base and electrolyte abnormalities, inflammatory cytokines, direct bruising of the myocardium from trauma, and areas of ischemia can further predispose the patient to ventricular or atrial dysrhythmias.

Cardiac dysrhythmias should be controlled whenever possible. Treatment of bradycardia should be directed at treating the underlying cause. Administer anticholinergic drugs such as atropine (0.04 mg/kg IM) or glycopyrrolate (0.02 mg/kg IM) as necessary. In cases of third-degree or complete AV block, administer a pure β-agonist such as isoproterenol.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism of Action</th>
<th>Dose and Method of Administration (Onset, Peak, Duration)</th>
<th>Potential adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Captopril</td>
<td>Angiotensin-converting enzyme inhibitor</td>
<td>0.5-2 mg/kg PO tid</td>
<td>Azotemia</td>
</tr>
<tr>
<td>Enalapril</td>
<td>Angiotensin-II–converting enzyme inhibitor</td>
<td>0.25-0.5 mg/kg/PO q12-24h</td>
<td>Azotemia</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>Direct arteriolar smooth muscle relaxant; little effect on venous capacitance vessels</td>
<td>0.2-2 mg/kg PO q12h</td>
<td>Blood dyscrasias, neuritis with prolonged use</td>
</tr>
<tr>
<td>Lisinopril</td>
<td>Angiotensin-II–converting enzyme inhibitor</td>
<td>0.25-0.5 mg/kg PO q12-24h</td>
<td></td>
</tr>
<tr>
<td>Morphine</td>
<td>Splanchnic capacitance vessel dilatation</td>
<td>0.025-0.05 mg/kg q6-8 hr IV, IM, SQ</td>
<td>Vomiting</td>
</tr>
<tr>
<td>Prazosin</td>
<td>α-Receptor blockade; arteriolar and venous dilatation</td>
<td>1 mg/15 kg PO BID-TID (canine); 0.5 mg PO TID (feline)</td>
<td>Anorexia, vomiting, diarrhea</td>
</tr>
<tr>
<td>Sodium nitroprusside</td>
<td>Direct arteriolar and venular dilatation</td>
<td>0.5-2 mcg/kg/min; increase dose incrementally every 3-5 minutes CRI; dilute in 5% dextrose in water Intravenous line with continuous blood pressure monitoring (immediately; 1 min; 2 min)</td>
<td>Hypotension, tolerance, cyanide toxicity at higher doses, avoid in hepatic or renal failure; thiocyanate accumulation (disorientation); is light sensitive and must be covered in foil and not kept for longer than 4 hours</td>
</tr>
</tbody>
</table>

CRI, Constant rate infusion; IM, intramuscularly; IV, intravenously; PO, orally; SQ, subcutaneously.
Perform passive rewarming if the patient is hypothermic. Correct any underlying electrolyte abnormalities such as hyperkalemia, hypomagnesemia, and hypermagnesemia.

Treat ventricular dysrhythmias such as multifocal PVCs, sustained ventricular tachycardia >160 beats/min, and R on T phenomenon (the T wave of the preceding beat occurs superimposed on the QRS complex of the next beat, and there is no return to isoelectric shelf), and administer treatment if runs of ventricular tachycardia cause a drop in BP. Intravenous lidocaine and procainamide are the first drugs of choice for ventricular dysrhythmias. Supraventricular tachycardia can impair cardiac output by impairing diastolic filling time. Control supraventricular dysrhythmias with calcium channel blockers, β-adrenergic blockers, or quinidine (Table 1-59).

### Table 1-59: Antiarrhythmic Drugs of Choice Used for the Treatment of Ventricular and Supraventricular Tachycardias

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism of Action</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lidocaine</td>
<td>Fast sodium channel inhibition</td>
<td>1-4 mg/kg IV slowly, then 50-100 mcg/kg/min (dog); 0.25-1.0 mg/kg IV (cat)*</td>
</tr>
<tr>
<td>Procainamide</td>
<td>Fast sodium channel inhibition</td>
<td>1-8 mg/kg IV, slow (canine); 3-8 mg/kg PO q6-8h (feline)</td>
</tr>
<tr>
<td>Tocainide</td>
<td>Fast sodium channel inhibition</td>
<td>5-20 mg/kg PO q8h† (dog)</td>
</tr>
<tr>
<td>Quinidine</td>
<td>Fast sodium channel inhibition</td>
<td>6-10 mg/kg PO qid</td>
</tr>
<tr>
<td>Propranolol</td>
<td>β-Adrenergic blocker</td>
<td>0.02-0.06 mg/kg IV; 0.2-1 mg/kg PO q8h</td>
</tr>
<tr>
<td>Esmolol</td>
<td>β-Adrenergic blocker</td>
<td>0.5 mg/kg IV, then 50-200 mcg/kg/min IV CRI</td>
</tr>
<tr>
<td>Verapamil</td>
<td>Slow calcium channel blocker</td>
<td>0.01-1 mg/kg IV; 0.5-5 mg/kg PO q8h (canine); 0.5-1 mg/kg PO q8h (feline)</td>
</tr>
<tr>
<td>Diltiazem</td>
<td>Calcium channel blocker</td>
<td>0.25 mg/kg IV, 0.5-1.5 mg/kg PO q8h (dogs); 1.75-2.5 mg/kg PO q8h (dogs and cats)</td>
</tr>
<tr>
<td>Pimobendan</td>
<td>Phosphodiesterase inhibition</td>
<td>Positive inotrope</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.1-0.3 mg/kg PO q12h</td>
</tr>
</tbody>
</table>

*CRI, Constant rate infusion; IV, intravenously; PO, orally.
* Use caution with lidocaine in cats because of neurotoxicity and seizures.
† Monitor for hypotension.
† Is not to be used for more than 2 weeks owing to idiosyncratic blindness.
5. Oncotic Pressure
Colloid oncotic pressure within the intravascular and interstitial spaces contributes to fluid flux. Oncotic pressure can be measured with a colloid osmometer. Normal oncotic pressure is 15 mm Hg. In cases of sepsis and SIRS, increased vascular permeability increases the tendency for leakage of fluids into the interstitial spaces. Colloids that can be administered until the source of albumin loss resolves include the synthetic colloids hydroxyethyl starch, concentrated human albumin (25% albumin, 2 mL/kg), canine albumin (3 to 6 mL/kg of the 16% solution), and plasma (20 mL/kg).

6. Oxygenation and Ventilation
Oxygenation and ventilation can be evaluated by arterial blood gas analysis or by the noninvasive means of pulse oximetry and capnometry (see sections on pulse oximetry and capnometry). Oxygen delivery can be impaired in cases of hypovolemic shock because of hemorrhage and anemia, resulting in a decrease in functional capacity to carry oxygen, and in cases of cardiogenic shock as a result of impaired ability to saturate hemoglobin caused by pulmonary edema in the lungs or decrease in cardiac output. In septic shock, decreases in cardiac output caused by inflammatory cytokines and a decrease in cellular oxygen extraction can lead to lactic acidosis. Increased cellular metabolism and decreases in respiratory function can lead to respiratory acidosis as CO₂ increases.

Administer supplemental oxygen via flow-by, nasal or nasopharyngeal catheter, oxygen hood, or oxygen cage. Supplemental oxygen should be humidified and delivered at 50 to 100 mL/kg/min. If oxygenation and ventilation are so impaired that the PaO₂ remains <60 mm Hg with the patient on supplemental oxygen, a PaCO₂ >60 mm Hg, or severe respiratory fatigue, develops, and mechanical ventilation should be considered.

7. Glucose
Glucose is a necessary fuel source for RBCs and neuronal tissues, and serum glucose should be maintained within normal reference ranges. Glucose supplementation can be administered as 2.5% to 5% solutions in crystalloid fluids, or in parenteral and enteral nutrition products.

8. Acid-Base, Electrolyte, and Lactate Status
Arterial and venous pH can be measured by performing blood gas analyses. Decrease in tissue perfusion, impaired oxygen delivery, and decreased oxygen extraction in the various forms of shock can lead to anaerobic metabolism and metabolic acidosis. In most cases, improving tissue perfusion and oxygen delivery with crystalloid and colloid fluids, supplemental oxygen, and inotropic drugs will help normalize metabolic acidosis. Serial measurements of serum lactate (normal, <2.5 mmol/L) can be used as a guide to evaluate the tissue response to fluid resuscitative efforts.

Serum electrolytes often become severely deranged in shock states. Serum potassium, magnesium, sodium, chloride, and total and ionized calcium should be maintained within normal reference ranges.

If metabolic acidosis is severe, sodium bicarbonate can be administered by calculating the following formula:

\[
\text{Base deficit} \times 0.3 \times \text{Body weight in kg} = \text{Bicarbonate to administer in mEq}
\]

Because iatrogenic metabolic alkalosis can occur, a conservative approach is to administer \(\frac{1}{4}\) of the calculated dose and then recheck the patient’s pH and bicarbonate levels. If the base excess is unknown, sodium bicarbonate can be administered in incremental doses of 1 mEq/kg until the pH is above 7.2. Complications associated with bicarbonate therapy include iatrogenic hypocalcemia, metabolic alkalosis, paradoxical CSF acidosis, hypotension, restlessness, and death.
9. **Coagulation**

Massive trauma, neoplasia, sepsis, and systemic inflammation can all lead to coagulation abnormalities, including DIC. Cage-side coagulation monitors are available for daily measurement of PT, APTT, and platelet counts. Fibrin degradation products (fibrin split products) become elevated in DIC, trauma, hepatic disease, and surgery. Coagulation proteins (clotting factors) and AT often are lost with other proteins in hypoproteinemia or are consumed when microclots are formed and then dissolved. AT levels can be measured by commercial laboratories. AT and clotting factors can be replenished in the form of fresh frozen plasma transfusions. A more sensitive and specific test for DIC is the detection of D dimers, which can be measured by commercial laboratories.

Treatment for DIC involves treatment and resolution of the underlying disease and administration of AT and clotting factors in the form of fresh frozen plasma (20 mL/kg) and heparin (unfractionated, 50 to 100 units/kg SQ tid; fractionated [Lovenox], 1 mg/kg SQ bid).

10. **Mentation**

Monitor the patient for changes in mental status, including stupor, coma, decreased ability to swallow and protect the airway, and seizures. Elevation of the patient's head can help to protect the airway and decrease the risk of increased intracranial pressure. Serum glucose should be maintained within normal levels to prevent hypoglycemia-induced seizures.

11. **Red Blood Cell and Hemoglobin Concentration**

One of the major components of oxygen delivery is the binding to hemoglobin. PCV must be kept above 20% to 30% for adequate cellular oxygen delivery. Acid-base status can adversely affect oxygen offloading at the tissue level if metabolic or respiratory alkalosis is present. Oxygen-carrying capacity and hemoglobin levels can be increased with administration of RBC component therapy.

12. **Renal Function**

Monitoring of renal function includes daily measurement of BUN, creatinine, and urine output. Normal urine output in a hydrated euvolemic patient is 1 to 2 mL/kg/hr. Fluid intake and output should be measured in cases of suspected oliguria or anuria. In patients with oliguria or anuria, furosemide can be administered as a bolus (4 to 8 mg/kg) or by CRI (0.66 to 1 mg/kg/hr). Mannitol should also be administered (0.5 to 1 g/kg over 10 to 15 minutes). Dopamine (1 to 5 mcg/kg/min CRI) can be administered to dilate renal afferent vessels and improve urine output.

13. **White Blood Cell Count, Immune Function, Antibiotic Dose and Selection**

The patient’s WBC count may be elevated, normal, or decreased, depending on the type of shock. The decision to administer antibiotics should be made on a daily basis. Superficial or deep *Staphylococcus* or *Streptococcus* infection usually can be treated with a first-generation cephalosporin (cefazolin, 22 mg/kg IV tid). If a known source of infection is present, administer a broad-spectrum antibiotic (cefoxitin, 22 mg/kg IV tid; ampicillin, 22 mg/kg qid, or enrofloxacin, 5-10 mg/kg IV once daily (dogs), 5 mg/kg IV once daily (cats) pending results of culture and susceptibility testing. If broader anaerobic coverage is required, metronidazole (10 mg/kg IV tid) should be considered. Gentamicin (6-8 mg/kg/day or 2-4 mg/kg q8h) is a good choice for gram-negative sepsis, provided that the patient is well hydrated and has normal renal function. Ideally, patients receiving any aminoglycoside antibiotic should have a daily urinalysis to check for renal tubular casts, which signify renal damage.

14. **Gastrointestinal Motility and Integrity**

In dogs the gut is the shock organ. Impaired gastrointestinal motility and vomiting should be aggressively treated with antiemetics and promotility drugs (dolasetron, 0.6 mg/kg IV once daily, and metoclopramide, 1 to 2 mg/kg/day IV CRI). Metoclopramide is contraindicated in cases of suspected gastrointestinal obstruction. Histamine-receptor
blockers such as famotidine (0.5 mg/kg bid IV) and ranitidine (0.5 to 2 mg/kg IV bid, tid) or proton-pump inhibitors (omeprazole, 0.5 to 1 mg/kg PO once daily) can be administered for esophagitis. Administer sucralfate (0.25 to 1 g PO tid or 0.5-1 mg/kg IV q25h) to treat gastric ulceration. If the gastrointestinal barrier function is diminished because of poor perfusion, infection, or inflammation, administer broad-spectrum antibiotics such as ampicillin (22 mg/kg IV qid) to prevent gastrointestinal bacterial translocation.

15. Drug Doses and Metabolism
The course of drug therapy should be reviewed daily, and the patient should be monitored for potential drug interactions. For example, metoclopramide and dopamine, working at the same receptor, can effectively negate the effects of each other. Cimetidine, a cytochrome P450 enzyme inhibitor, can decrease the metabolism of some drugs. Drugs that are avidly protein-bound may have an increase in unbound fraction with concurrent hypoalbuminemia or when hypoalbuminemia is present. Decreased renal function may impair the renal clearance of some drugs, requiring increased administration interval or decreased dose.

16. Nutrition
Nutrition is of utmost importance in any critically ill patient. Patients with septic shock may become hypermetabolic and require supraphysiologic nutrient caloric requirements, whereas others may actually become hypometabolic. Enteral nutrition is preferred whenever possible, because enterocytes undergo atrophy without luminal nutrient stimulation. A variety of enteral feeding tubes can be placed, depending on what portion of the gut is functional, to provide enteral nutrition in an inappetent patient. Loss of gastrointestinal mucosal barrier function may predispose patients to the development of bacterial translocation and may contribute to sepsis. If enteral nutrition is impossible because of protracted vomiting or gastrointestinal resection, glucose, lipid, and amino acid products are available that can be administered parenterally to meet nutrient needs until the gastrointestinal tract is functioning and the patient can be transitioned to enteral nutrition.

17. Analgesia and Pain Control
Assessment of pain in animals in shock can be challenging. Pain can result in the release of catecholamines and glucocounterregulatory hormones that can impair nutrient assimilation and lead to negative nitrogen balance, impaired wound healing, and immunocompromise. In any animal determined to be in pain, analgesic drugs should be administered to control pain and discomfort at all times. Opioids are cardiovascularly friendly, and their effects can easily be reversed with naloxone if adverse effects such as hypotension and hypoventilation occur.

18. Nursing Care and Patient Mobilization
If the patient is nonambulatory, rotate the animal from side to side every 4 to 6 hours to prevent lung atelectasis. Passive range-of-motion exercises and deep muscle massage should be performed to increase tissue perfusion, decrease dependent edema, and prevent disuse atrophy. Animals should be kept completely dry on soft, padded bedding to prevent the development of decubital ulcers.

19. Wound Care and Bandage Care
All bandages, wound sites, and catheter sites should be checked daily for the presence of swelling, erythema, and pain. Soiled bandages should be changed to prevent strike-through and contamination of the underlying catheter or wound.

20. TLC (Tender Loving Care)
Hospitalization can be a stressful experience for patient and client alike. Allowing brief visits and walks outside in the fresh air can improve a patient’s temperament and decrease stress. The preemptive use of analgesic drugs on a regular schedule (not PRN) should be used to prevent pain before it occurs. Pain decreases the patient’s ability to sleep. Lack of sleep can promote further stress and impaired wound healing.
**OTHER CONSIDERATIONS AND CONTROVERSIES IN SHOCK THERAPY**

**Glucocorticosteroids and Antiprostaglandins**

The use of glucocorticosteroids and antiprostaglandins in shock therapy remains a topic of wide controversy. Although the use of these agents potentially may stabilize membranes, decrease the absorption of endotoxin, and decrease prostaglandin release, the routine use of glucocorticosteroids and antiprostaglandins can decrease renal perfusion and gastrointestinal blood flow, promoting gastrointestinal ulceration and impaired renal function. The administration of supraphysiologic levels of glucocorticosteroids in patients in any type of shock can increase sodium and water retention, depress cellular immune function, and impair wound healing. In clinical studies of small animal patients, the routine use of glucocorticosteroids and antiprostaglandins has not demonstrated definite improved survival. The risks of therapy do outweigh the anecdotal reported benefits, and therefore the empiric use of glucocorticosteroids and antiprostaglandins in any shock patient is absolutely contraindicated. The administration of glucocorticosteroids to patients with cardiac disease has been shown to promote sodium and water retention and can actually predispose to the development of CHF.

**Additional Reading**


**THROMBOEMBOLISM: SYSTEMIC**

Systemic thromboembolism is most commonly recognized in cats with cardiomyopathies (hypertrophic, restrictive, unclassified, and dilatative) but can also occur in dogs with hyperadrenocorticism, DIC, SIRS, protein-losing enteropathy and nephropathy, and tumors affecting the aorta and vena cava. Thrombosis occurs through a complex series of mechanisms when the components of Virchow’s triad (hypercoagulable state, sluggish blood flow, and vascular endothelial injury or damage) are present. In cats, blood flow through a severely stretched left atrium is a predisposing factor to the development of clots and thromboembolism.
The most common site of embolism is the aortic bifurcation, or “saddle thrombus.” Other, less common locations of thromboembolism include the forelimbs, kidneys, gastrointestinal tract, and cerebrum. Diagnosis usually is made based on clinical signs of cool extremities, the presence of a cardiac murmur or gallop rhythm, auscultation of pulmonary crackles resulting from pulmonary edema, acute pain or paralysis of one or more peripheral extremities, respiratory distress, and pain and lack of a palpable pulse in affected limbs. The affected nail beds and paw pads are cyanotic, and nails do not bleed when cut with a nail clipper.

Client education is one of the most important aspects of emergency management of the patient with thromboembolic disease. Concurrent CHF occurs in 40% to 60% of cats with arterial thromboembolism. More than 70% of cats are euthanized during the initial thromboembolic event because of the poor long-term prognosis and the high risk of recurrence within days to months after the initial event, even with aggressive therapy. Although the long-term prognosis varies from 2 months to 2 years after initial diagnosis and treatment, in the majority of cats thromboembolic disease recurs within 9 months. Rectal temperature hypothermia and bradycardia on presentation are negative prognostic indicators.

Immediate treatment of a patient with CHF and thromboembolic disease involves management of the CHF with furosemide, oxygen, and vasodilators (morphine, nitropresside). Additional management includes analgesia (butorphanol, 0.1 to 0.4 mg/kg IV, IM) and prevention of further clot formation. Aspirin (10 mg/kg PO q48h) is beneficial because of its antiplatelet effects. Heparin works in conjunction with AT to prevent further clot formation (500 units/kg IV, followed by 250 to 300 units/kg SQ q8h in cats, and 100 to 200 units/kg SQ q8h in dogs). Acepromazine can cause peripheral vasodilation and decreased afterload but also can promote hypotension in a patient with concurrent CHF. Acepromazine (0.05 mg/kg SQ) should be used with extreme caution, if at all.

The use of thrombolytic therapies (streptokinase, t-PA, urokinase), has been shown to not improve outcome, and may increase the risk of hemorrhage, reperfusion injury, and mortality. For these reasons, the use of thrombolytic agents is not recommended.

In cats, the primary cause of arterial thromboembolism is cardiomyopathy. Once an animal’s condition is determined to be stable enough to allow diagnostic procedures, lateral and dorsoventral (DV) thoracic radiographs and an echocardiogram should be performed. Ultrasound of the distal aorta and renal arteries should also be performed to determine the location of the clot and help establish the prognosis.

Other diagnostic procedures to evaluate the presence and cause of thromboembolism include a complete blood count, serum biochemistry profile, urinalysis (to rule out protein-losing nephropathy), urine protein:creatinine ratio, AT levels, ACTH stimulation test (to rule out hyperadrenocorticism), heartworm antigen test (in dogs), thyroid profile (to rule out hyperthyroidism in cats and hypothyroidism in dogs), thoracic radiographs, arterial blood gas analyses, coagulation tests, and Coombs’ test. Selective and nonselective angiography can also be performed to determine the exact location of the thrombus.

Long-term management of thromboembolism involves management of the underlying disease process and preventing further clot formation. Most recently, the use of clopidogrel (Plavix, 3 to 5 mg/kg PO q24h [dog]; 18.75 to 37.5 mg PO q24h [cat]) has been recommended to help prevent clot formation. In the past the combination of heparin then warfarin was used; however, it was often difficult to regulate. This form of therapy involves first beginning heparin until the APTT becomes prolonged 1.5 times; then administering warfarin (0.06 to 0.09 mg/kg/day). Monitoring therapy based on prothrombin time and the international normalized ratio (INR, 2.0 to 4.0) is recommended. Low-dose aspirin 0.5 mg/kg PO q12-24h (canine), 25 mg/kg q56-84h (feline) also has been recommended. Physical therapy with warm water bathing, deep muscle massage, and passive range-of-motion exercises should be performed until the patient regains motor function. Future therapy may involve the use of platelet receptor antagonists to prevent platelet activation and adhesion.
Additional Reading


**URINARY TRACT EMERGENCIES**

Azotemia

Azotemia occurs when 75% or more of the nephrons are nonfunctional. The magnitude of the azotemia alone cannot be used to determine whether the azotemia is prerenal, renal, or postrenal in origin or whether the disease process is acute or chronic, reversible or irreversible, or progressive or nonprogressive. Before treatment for azotemia is begun, the location or cause of the azotemia must be identified. Take a thorough history and then perform a physical examination. Obtain blood and urine samples before initiating fluid therapy, for accurate assessment of the location of the azotemia.

For example, an azotemic animal with a history of vomiting and diarrhea that appears clinically dehydrated on physical examination should normally have a concentrated urine specific gravity (>1.045), reflecting the attempt to conserve fluid. If this level is found, the azotemia is much less likely to be renal in origin, and the azotemia will likely resolve after rehydration.

If, however, the urine specific gravity is isosthenuric or hyposthenuric (1.007 to 1.015) in the presence of azotemia and dehydration, primary intrinsic renal insufficiency is likely present. If the azotemia resolves with fluid therapy, the patient has prerenal and primary renal disease. If the azotemia does not resolve after rehydration, the patient has prerenal and primary renal failure. Dogs with hypoadrenocorticism can have both prerenal and primary renal disease secondary to the lack of mineralocorticoid (aldosterone) influence on the renal collecting duct and renal interstitial medullary gradient. Medullary washout can occur, causing isosthenuric urine in the presence of dehydration from vomiting and diarrhea. The patient often has azotemia from fluid loss (dehydration and urinary loss) and gastric or intestinal hemorrhage (elevated BUN). The prerenal component will resolve with treatment with glucocorticoids and crystalloid fluids, but the renal component may take several weeks to resolve, until the medullary concentration gradient is reestablished with the treatment and influence of mineralocorticoids. Drugs such as corticosteroids and diuretics can influence renal tubular uptake and excretion of fluid and cause a prerenal azotemia and isosthenuric urine in the absence of primary renal disease.

Treatment of azotemia includes calculation of the patient’s dehydration estimate and maintenance fluid volumes, and administering that volume over the course of 24 hours. Identify and treat underlying causes of prerenal azotemia (shock, vomiting, diarrhea). Monitor urine output closely. Once a patient is euvoletic, oliguria is defined as urine output <1 to 2 mL/kg/hr. Urine output should return to normal in patients with prerenal azotemia as rehydration occurs. If a patient remains oliguric after rehydration, consider the possibility of oliguric acute intrinsic renal failure, and administer additional fluid therapy based on the patient’s urine output, body weight, CVP, and response to other medical therapies.
**Prerenal Azotemia**

Prerenal azotemia is caused by conditions that decrease renal perfusion, including hypovolemic shock, severe dehydration, hypoadrenocorticism, CHF, cardiac tamponade, cardiac dysrhythmias, and hypotension. Once renal perfusion is restored, the kidneys can resume normal function. GFR decreases when the mean arterial BP falls to less than 80 mm Hg in a patient with normal renal autoregulation. Renal autoregulation can be impaired in some diseases. Passive reabsorption of urea from the renal tubules can occur during states of low tubular flow (dehydration, hypotension) even if glomerular filtration is not decreased. If renal hypoperfusion is not quickly restored, the condition can progress from prerenal disease to acute intrinsic renal failure. Prerenal and renal azotemia can coexist in animals with primary renal disease, as a result of vomiting and ongoing polyuria in the absence of any oral fluid intake. The treatment of prerenal azotemia consists of rehydration, antiemetic therapy, and treatment of the underlying cause of vomiting, diarrhea, or third spacing of fluids.

**Acute Intrinsic Renal Failure**

Acute intrinsic renal failure is characterized by an abrupt decline in renal function to the extent that azotemia and an inability to regulate solute and fluid balance occur. Patients with acute intrinsic renal failure may be oliguric or polyuric, depending on the cause and state of renal failure. In small animals, the most common causes of acute intrinsic renal failure are renal ischemia and toxins.

There are three phases of acute intrinsic renal failure: induction, maintenance, and recovery. During the induction phase, some insult (ischemia or toxin) to the kidneys occurs, leading to a defective concentrating mechanism, decreased renal clearance of nitrogenous waste (azotemia), and polyuria or oliguria. If treatment is initiated during the induction phase, progression to the maintenance phase potentially can be stopped. As the induction phase progresses, there is worsening of the urine-concentrating ability and azotemia. Renal tubular epithelial cells and renal tubular casts can be seen on examination of the urine sediment. Glucosuria may be present.

The maintenance phase of acute intrinsic renal failure occurs after a critical amount of irreversible nephron injury. Correction of the azotemia and removal of the cause of the problem do not result in return to normal function. In patients with oliguria, the extent of nephron damage is greater than that observed in patients with polyuria. The maintenance phase may last for several weeks to months. Recovery of renal function may or may not occur, depending on the extent of injury. The most serious complications (overhydration and hyperkalemia) are observed in patients with oliguria.

The recovery phase occurs with sufficient healing of damaged nephrons. Azotemia may resolve, but concentrating defects may remain. If the patient was oliguric in the maintenance phase, a marked diuresis develops during the recovery phase that may be accompanied by fluid and electrolyte losses. This phase may last for weeks to months.

Treatment of acute intrinsic renal failure consists of determining the cause and ruling out obstruction or uroabdomen whenever possible. A careful history can sometimes determine whether there has been exposure to nephrotoxic drugs, chemicals, or food items. If ingestion or exposure to a toxic drug, chemical, or food occurred recently (within 2 to 4 hours), induce emesis with apomorphine (0.04 mg/kg IV). Next, administer activated charcoal either PO or via stomach tube, to prevent further absorption of the toxin. Obtain blood and urine samples for toxicologic analysis (e.g., ethylene glycol) and to determine whether azotemia or abnormalities in the urine sediment exist. (See sections on ethylene glycol, grapes and raisins, and nonsteroidal antiinflammatory drugs.) Obtain a complete blood count, biochemical profile, and urinalysis to determine the presence of signs of chronic renal failure, including PU/PD and nonregenerative anemia. Radiographs and abdominal ultrasound can help in determining the chronicity of renal failure. Normal renal size is 2.5 to 3.5 times the length of L2 in dogs and 2.4 to 3.0 times the length of L2 in cats. Monitor the patient’s body weight at least twice a day to avoid overhydration.
Also monitor urine output; normal output is 1 to 2 mL/kg/hr. In cases of polyuric renal failure, massive fluid and electrolyte losses can occur. Place a urinary catheter for patient cleanliness and to facilitate urine quantitation. Measure fluid ins and outs (see section on fluid therapy). After the patient has been rehydrated, the amount of fluids administered should equal maintenance and insensible needs plus the volume of urine produced each day. If a urinary catheter cannot be placed or maintained, serial body weight measurements and CVP should be used to monitor the patient’s fluid balance and prevent overhydration.

If the patient is oliguric (urine output <1 to 2 mL/kg/hr), pharmacologic intervention is necessary to increase urine output. First, administer furosemide (2 to 4 mg/kg IV or 0.66 mg/kg/hr IV CRI). Repeat bolus doses of furosemide if there is no response to initial treatment. If necessary, administer low-dose dopamine (0.5-3 mcg/kg/min IV CRI) to increase renal afferent dilatation and renal perfusion. Dopamine and furosemide may be synergistic if administered together. If dopamine and furosemide therapy is ineffective, administer mannitol (0.25 to 0.5 g/kg IV) over 15-20 minutes once only. Diltiazem may be effective in inducing diuresis in oliguric patients with hypertension (0.1 to 0.5 mg/kg IV slowly, followed by 1 to 5 mcg/kg/min).

If polyuria is present, management is simplified because of the decreased risk of overhydration. If oliguria cannot be reversed, monitor the central venous pressure, body weight, and respiratory rate and effort, auscultate for crackles, and examine the patient carefully for signs of chemois and the presence of serous nasal discharge.

Correct hyperkalemia with sodium bicarbonate (0.25 to 1.0 mEq/kg IV) or with insulin (0.25 units/kg) plus dextrose (1 g/unit of insulin IV, followed by 2.5% dextrose IV CRI). Treat severe metabolic acidosis (pH <7.2 or \(\text{HCO}_3^- <12\text{ mEq/L}\)) with sodium bicarbonate. If anuria develops or oliguria is irreversible despite this therapy, begin peritoneal dialysis. Perform a renal biopsy to establish a diagnosis and prognosis (see section on renal biopsy). Administer gastroprotectant drugs and antiemetics to control nausea and vomiting. If possible, avoid the use of nephrotoxic drugs and general anesthesia. Initiate nutritional support in the form of an enteral feeding tube or parenteral nutrition as early as possible.

Once the patient enters the recovery phase, diuresis may occur, which can lead to dehydration and electrolyte imbalances (hyponatremia, hypokalemia). Dehydration and electrolyte imbalances can be treated with parenteral fluid and electrolyte supplementation.

**Postrenal Azotemia**

Postrenal azotemia is primarily caused by urethral obstruction or leakage from the urinary tract into the abdomen (uroabdomen). Complete urinary tract obstruction and uroabdomen are both ultimately fatal within 3 to 5 days if left untreated. In dogs, the most common causes of urethral obstruction are urinary (urethral) calculi or tumors of the urinary bladder or urethra. In male cats, feline urologic syndrome (FUS) is the most common cause of urethral obstruction, although there has been an increased incidence of urethral calculi observed in recent years. A ruptured urinary bladder is the most common cause of uroabdomen and is usually secondary to blunt trauma.

**Urinary Tract Obstruction**

Clinical signs of urinary tract obstruction include dysuria, hematuria, inability to urinate or initiate an adequate stream of urine, and a distended painful urinary bladder. Late in the course of obstructive disease, clinical signs referable to uremia and azotemia (vomiting, oral ulcers, hematemesis, dehydration, lethargy, and anorexia) occur.

The initial goal of treatment of urinary tract obstruction is to relieve the obstruction. In male dogs a lubricated catheter can be inserted past the area of obstruction with the animal under heavy sedation or general anesthesia (see section on urohydropulsion). Depending on the chronicity of the obstruction, serum electrolytes should be measured; an ECG should be obtained before any anesthetic drugs are administered, because of the cardiototoxic effects of hyperkalemia (see section on atrial standstill). Correct fluid, electrolyte, and acid-base abnormalities. If a urinary catheter cannot be placed, perform cystocentesis only as a last resort, because of the risk of urinary bladder rupture.
Definitive treatment includes identification and treatment of the underlying cause (tumor versus urinary calculi). In most cases, surgical intervention is necessary. If an unresectable tumor is present, a low-profile permanent cystostomy tube can be placed, if the owner desires. Administration of piroxicam (Feldene, 0.3 mg/kg PO q24-48h) with or without chemotherapy may shrink the tumor mass and delay the progression of clinical signs.

**Feline Lower Urinary Tract Disease**

A complete discussion of this disorder is beyond the scope of this text (see Additional Reading for other sources of information). Feline lower urinary tract disease can cause urethral obstruction, particularly in male cats. Clinical signs include stranguria, dribbling of small amounts of urine, lethargy, inappetence, and vomiting. Often owners call with the primary complaint of constipation, because the cat is making frequent trips to the litter box and straining. Cases with a duration of obstruction <36 hours are considered uncomplicated; those with a duration >36 hours are complicated.

Treatment of urethral obstruction includes stabilizing and normalizing the patient’s electrolyte status, induction of sedation or general anesthesia, and relieving the obstruction. Obtain blood samples for analysis of electrolyte abnormalities. Treat hyperkalemia (K+ >6.0 mEq/L) with sodium bicarbonate (0.25 to 1.0 mEq/kg IV), regular insulin (0.25 unit/kg IV) plus dextrose (1 g/unit of insulin IV), followed by 2.5% dextrose IV CRI to prevent hypoglycemia; or calcium gluconate (0.5-1 ml/kg over 10-20 minutes). Administer non-potassium-containing intravenous fluids in 0.9% saline solution. Obtain an ECG to assess for atrial standstill (see section on atrial standstill).

In some cases a urethral plug is visible at the tip of the penis. The urethral plug can sometimes be manually extracted or massaged from the penis, and the obstruction temporarily relieved. In such cases it is still necessary to pass a urethral catheter to flush sediment from the urethra and urinary bladder. Unless a patient is obtunded, administer an anesthetic such as ketamine, atropine, or propofol (4 to 7 mg/kg IV) with diazepam IV for patient comfort and muscle relaxation.

Once the patient is under anesthesia or heavily sedated, urinary catheterization should be performed. In some cases, it will be difficult to advance the catheter. Lubricate a closed-ended Tomcat catheter and pass the tip into the distal urethra. Fill a 12-mL syringe with sterile saline and sterile lubricant and connect the syringe to the hub of the catheter. Pulse the fluid into the catheter as you gently move the catheter tip back and forth against the urethral obstruction. When the catheter has been passed into the urinary bladder, obtain a urine sample for urinalysis. Drain the bladder and flush with sterile saline solution until the urine efflux appears clear. Remove the Tomcat catheter and insert a 3F to 5F red rubber tube or Argyle infant feeding catheter into the urethra for urine collection and quantitation. Secure the urinary catheter to prepuce with a butterfly strip of 1-inch adhesive tape secured around the catheter and then sutured to either side of the prepuce. The catheter should be connected to a closed urinary collection system for cleanliness and to reduce the risk of ascending bacterial infection. An Elizabethan collar should be placed at all times to prevent the patient from damaging or removing the catheter.

When the urethral obstruction has been relieved and the catheter placed, continue intravenous fluid diuresis to alleviate postrenal azotemia. Monitor the urine for bacteria and other sediment. In some cases, postobstructive diuresis can be severe. Carefully monitor fluid ins and outs, along with body weight, to maintain adequate hydration and perfusion. The urinary catheter can be removed after 24 to 48 hours. Palpate the bladder frequently to make sure that the patient is voiding normally and to detect the recurrence of obstruction.

In patients with severe penile or urethral trauma or edema, administer a short-acting steroid (dexamethasone sodium phosphate, 0.25 mg/kg IV, IM, SQ). At the time of initial diagnosis and again at the time of discharge, clients need to be instructed about the long-term management of feline lower urinary tract disease at home, and informed of the risks and consequences of recurrence.
UROABDOMEN

Uroabdomen can occur from trauma or leakage from the kidneys, ureter, or urinary bladder. Clinical signs of uroabdomen (azotemia, uremia, hyperkalemia) can also occur secondary to third spacing of urine and leakage into muscular tissue from a ruptured urethra. In most cases, urinary bladder trauma and rupture are secondary to blunt trauma. Abdominocentesis should be performed in any animal with suspected blunt abdominal trauma, and any fluid obtained should be analyzed for creatinine or potassium and compared with the patient’s serum levels. An abdominal effusion that has a low PCV and a potassium or creatinine level greater than that of the patient’s serum is consistent with the diagnosis of uroabdomen.

Uroabdomen is not a surgical emergency. However, medical management consists of placement of a temporary abdominal drainage catheter into the abdomen to facilitate removal of urine from the peritoneal cavity. To place the catheter, position the patient in dorsal or lateral recumbency and shave the ventral abdomen, as for any exploratory laparotomy. Aseptically scrub the clipped area, and instill a local anesthetic (lidocaine, 1 to 2 mg/kg) caudal and to the right of the umbilicus, through the skin, subcutaneous tissues, and rectus abdominus muscles, inserting the lidocaine as you pull the needle out, thus creating an anesthetized tunnel. Aseptically scrub the area again and drape with sterile field towels; then make a small stab incision through the skin. Bluntly dissect through the subcutaneous tissue to the level of the external rectus abdominis. Pick up the muscle with a thumb forceps, and make a small stab incision into the abdominal cavity. Cut multiple holes in the side of a 14F to 16F red rubber tube or thoracic drainage catheter, using care not to make the cut wider than 50% of the circumference of the tube. Insert the catheter into the abdominal cavity in a dorsal caudal direction. Make sure that all holes in the catheter are located within the abdominal cavity. Secure the tube by placing a purse-string suture around the tube entrance site in the abdominal musculature with absorbable suture material. Close the dead space in the subcutaneous tissues with absorbable suture. Close the skin around the tube with another purse-string suture secured using a finger-trap technique. Connect the tube to a closed urinary collection system and bandage the catheter to the abdomen. The tube can remain in place until the patient’s cardiorespiratory status is stabilized enough to allow anesthesia and definitive repair of the urinary tract defect.

Additional Reading
PATIENT EVALUATION

OWNER ASSESSMENT OF PET HEALTH: THE BEETTS TEST

Most pet owners, particularly first-time owners, have limited understanding of animal health issues and therefore are not well prepared to recognize early signs of illness in a pet dog or cat. The most common medical disorders are not recognized by owners at all (e.g., accumulation of dental tartar and gingival erosion), or their treatment is delayed until the pet has advanced illness. Ironically, few veterinary practices take time to teach pet owners how to (proactively) assess a pet’s health. Educating owners on how to recognize early changes in health status not only encourages early awareness of potential health problems but also supports earlier intervention by the veterinarian.

A simple-to-remember, owner-prescribed examination is the BEETTS (pronounced “beets”) test. This acronym represents a means for willing owners to assess significant changes in a pet’s activity or physical appearance that will alert them to common minor problems and possibly more serious health problems, thereby avoiding the consequences of delayed diagnosis and treatment.
B for Behavior
Know Your Pet!
Owners should be aware that subtle changes in behavior may be the first sign of underlying illness. Several such behavior changes—such as reduced or no appetite and associated weight loss, increased demand for water, frequent need to urinate or defecate, unexplained aggression, reluctance to play, difficulty standing, or persistent licking of skin (particularly in one location)—might signal early and serious illness. Such changes often justify a physical examination and laboratory profile.

E for Eyes
Asymmetry of the eyes and eyelids (denoting pain or injury) is as important as discoloration of the eye (cataracts, intraocular hemorrhage) or the accumulation of mucus on or around the eyelids. Pets with hair covering the eyes are reasonably considered at risk of serious eye disease owing to delayed diagnosis. Frequent evaluation of both eyes for clarity and absence of irritation (redness) is essential.

E for Ears
Owners should be alert for signs that might indicate an ear disease (e.g., head tilt or scratching, pain on manipulating the ears, ear abrasions, discoloration, discharge, malodor). If the pinnae hang below the opening of the external ear canal, owners should be willing to lift and look. Owners must be advised to avoid inserting any instrument or medication into a pet’s ears unless specifically instructed how to do so.

T for Teeth (and Gingiva)
The relationship between halitosis and serious dental or gingival disease is important, and such disease can easily be missed if the teeth are not examined on a regular basis. Owners are not encouraged to open the mouth of a dog or cat. However, most dogs will allow owners to lift the lip and visually examine the teeth for evidence of damaged or discolored teeth. It is sufficient for the owner to occasionally assess the outside appearance (labial surface) of the teeth and gingiva once or twice annually. Examination of the teeth of cats is generally not recommend because of the risk associated with being bitten.

T for Toes (and Toenails)
Most owners are not aware of when or how to examine a pet’s toenails. Although a cat’s toenails are periodically shed and usually do not require clipping, the toenails of sedentary dogs living predominantly indoors deserve attention as often as every month. Because dogs may resist any attempt to handle or examine the feet, owners are instructed to listen to their dog walk across a noncarpeted surface. If the owner can hear toenails clicking against the floor as the pet walks, clipping is indicated. Occasionally an owner will express interest in clipping their own pet’s toenails. It is important to advise owners of the risks inherent in what may appear to be a simple procedure: pain, bleeding, aggressive resistance, and even biting. Owners willing to attempt nail trimming at home should be instructed on what equipment to purchase and how to safely perform the procedure.

S for Skin (and Hair Coat)
Variability in hair coat type, length, and density makes examining the skin one of the home examinations that pet owners least often accomplish. The skin is the largest organ of the body, and serious disorders may develop many weeks or months before they become evident. This is particularly true in long-haired dogs and cats. In addition to routine brushing, which benefits all dogs and cats, and the occasional bath, owners are instructed to thoroughly touch their pet’s skin and hair coat in a systematic manner. One technique owners find appealing begins with the owner standing behind the pet. Starting at the pet’s head with one hand around each ear, the owner uses his or her fingers to “crawl” from the head...
to the tail while gently massaging the pet and touching the entire surface of the skin over the thorax and abdomen. Then, gently grasping one leg at a time with one hand, the owner massages each limb from the point closest to the body to the toes.

**CLINICIAN INITIAL ASSESSMENT: THE PROBLEM LIST**

A correct diagnosis is based on the clinician’s ability to assess and define constituent problems affecting the patient. This sounds simple enough. However, unless the term “problem” is defined, actually doing a thorough diagnostic assessment of the patient is simply not possible. When pursuing a diagnosis, the astute clinician works from a clear definition of a problem:

1. The clinical history: Any abnormality described by the owner (whether or not the owner’s interpretation is correct) is a problem.
2. The physical examination: Any abnormality discovered during the physical examination is a problem (see Organ System Examination).
3. Any imaging (radiographic or ultrasound) or laboratory abnormalities are considered to be problems.

The problem list, once acquired, becomes the foundation on which a diagnosis is built. Problems that are obviously related are grouped and may either confirm the diagnosis or suggest which additional diagnostic studies need to be performed to elucidate the diagnosis.

**CLINICAL HISTORY**

The clinical history is a critical component of the patient’s problem list and frequently is the single most revealing part of the diagnostic assessment of a given patient. It requires unique skills and experience to elicit an unbiased, pertinent clinical history about a pet’s illness. Some owners are astute observers and can readily communicate important information, whereas others either may not be aware of certain abnormalities or may purposely withhold information. The clinical history is centered around, but not limited to, the chief complaint. The chief complaint is the reason the patient is being presented. What should be recorded is a sign (vomiting), not a diagnosis (enteritis). Note the duration or frequency of the sign. Determine whether the duration or frequency has increased, decreased, or remained unchanged since onset. It is important to determine whether or not the pet’s overall condition since the onset of illness has improved, worsened, or remained the same.

Ask neutral questions—one that will not prejudice the owner’s answers—for example, “Tell me about your dog’s water consumption.” Questions requiring only a “yes” or “no” response tend to introduce bias—for example, “Is your dog current on its vaccinations?” If the answer is “yes,” further questions such as “Anything else?” “How do you mean?” or “Tell me about that” are helpful in inducing the owner to elaborate. Given the widespread use of preventive medications in pets (e.g., heartworm, flea, tick preventative), a complete list of drugs and medications is a critical part of the clinical history. If the same sequence of history taking and physical examination is followed each time, the procedure gradually requires less time, and important facts are less likely to be omitted.

The clinical history is not inherently distinct from the physical examination. It is not uncommon for the client to be completely unaware that a physical abnormality is present until it is pointed out. When examining the patient, any unusual or unexpected physical findings justify additional inquiry as to causal relationships with the pet’s environment, diet, exposure to other animals, and so forth. For example, discovering severe abrasions on the footpads of both rear feet should prompt additional inquiry into possible causes.

**Note:** Failing to obtain a thorough clinical history is the first step in a missed diagnosis!
PHYSICAL EXAMINATION

The physical examination is the means whereby the clinician evaluates the health status of the patient through a methodical, "hands-on" organ system evaluation. It is a critical step in problem definition and objective diagnostic assessment of the patient. The physical examination is predicated on the clinician’s ability to distinguish normal from abnormal.

The extent of examination carried out on the individual patient varies. The “well-patient” physical examination is characteristically employed when evaluating healthy animals presented for routine health care (i.e., there is no chief complaint). The basic elements involved in the physical examination include the following:

VITAL SIGNS

Temperature, pulse, respiration, and weight are the most fundamental health parameters evaluated when examining the patient. Capillary refill time (CRT) is commonly recorded (normal: < 2 seconds) but is a relatively poor test of peripheral vascular perfusion. Blood pressure (see Section 4) is more sensitive but requires some operator experience to obtain reliable readings and multiple measurements in the individual patient to obtain a reasonable value. Although weight is not strictly a “vital” sign, all patients should be weighed at every visit.

BEHAVIOR AND MENTATION

Observing the patient’s behavior, activity, and alertness in the examination room can be particularly helpful in assessing patients with significant neurologic (brain) disease. Even animals that are nervous or scared in the hospital can be assessed and should manifest reasonable awareness of their surroundings. Dogs and cats that are particularly aggressive need to be handled with extreme care, yet evaluated for possible organic brain or neurologic disease.

CONFORMATION AND BODY CONDITION SCORE

Several methods exist for documenting conformation and body condition in dogs and cats. One commonly applied scale entails use of a 5-point grading system. Making appropriate entries in the medical record allows the clinician to assess, over time, changes in conformation other than just weight. Parameters for the 5-point grading scale are listed in the following paragraphs.

Grade 1/5—Emaciated

- Ribs, backbone, and pelvic bones easily seen, even from a distance
- No body fat
- Obvious loss of muscle mass

See Figure 2-1.

Grade 2/5—Underweight

- Ribs can be seen and easily felt
- Pelvic bones are prominent
- Obvious waist and abdominal tuck

Grade 3/5—Ideal Body Score

- Ribs can be felt
- Waist obvious when viewed from above
- Abdominal tuck evident

Grade 4/5—Overweight

- Ribs hard to feel, covered by fat
- Noticeable fat deposits over back and base of tail
- Waist and abdominal tuck barely discernible
Grade 5/5—Obese
• Ribs cannot be felt under heavy fat covering
• Massive fat deposits over back and base of tail
• No waist or abdominal tuck
See Figure 2-2.

The 9-Point Body Condition Score
An alternative 9-point body condition score for cats and dogs has also been described. In this scoring format, a BCS of 4 to 5 represents the ideal weight and conformation. A BCS of less than 4 represents underfed, underweight dogs and cats, whereas a BCS of 6 to 7 and higher represents overfed, overweight animals.

Figure 2-1: Dog with a body condition score of 1 out of 5.

Figure 2-2: Cat with a body condition score of 5 out of 5.
Clinical evaluation of the sick patient entails obtaining a laboratory profile to assess and characterize biochemical and hematologic abnormalities. Laboratory test abnormalities are a critical component of the diagnostic evaluation and are necessarily included, individually, on the patient's problem list. In companion animal practice, performing a laboratory profile meets the standard of care accepted in veterinary medicine. Although the specific test methods used and analytes assayed will vary from practice to practice, the laboratory database for any ill dog or cat should include most or all of the tests listed in Table 2-1.

<table>
<thead>
<tr>
<th></th>
<th>Canine</th>
<th>Feline</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hematology</strong></td>
<td>Complete blood count (CBC) includes the following:</td>
<td>Complete blood count (CBC) includes the following:</td>
</tr>
<tr>
<td></td>
<td>• Total RBC count</td>
<td>• Total RBC count</td>
</tr>
<tr>
<td></td>
<td>• Hematocrit or packed cell volume</td>
<td>• Hematocrit or packed cell volume</td>
</tr>
<tr>
<td></td>
<td>• Hemoglobin</td>
<td>• Hemoglobin</td>
</tr>
<tr>
<td></td>
<td>• Total WBC count</td>
<td>• Total WBC count</td>
</tr>
<tr>
<td></td>
<td>• Differential cell count</td>
<td>• Differential cell count</td>
</tr>
<tr>
<td></td>
<td>• Total solids</td>
<td>• Total solids</td>
</tr>
<tr>
<td></td>
<td>• Estimation of platelet numbers</td>
<td>• Estimation of platelet numbers</td>
</tr>
<tr>
<td></td>
<td>• Reticulocyte count if patient’s hematocrit is low (e.g., &lt;30%)</td>
<td>• Aggregate reticulocyte count if patient’s hematocrit is low (e.g., &lt;30%)</td>
</tr>
<tr>
<td>Note:</td>
<td>Some laboratories will also provide values for the RBC indices: MCV, MCH, and MCHC.</td>
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</tr>
<tr>
<td><strong>Biochemistry</strong></td>
<td>Individual analytes included on biochemistry panels will vary among laboratories. (See Section 5 for a comprehensive review of the various analytes that are likely to be included.)</td>
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<tr>
<td></td>
<td>Includes the following:</td>
<td>Includes the following:</td>
</tr>
<tr>
<td></td>
<td>• Specific gravity, color, and appearance</td>
<td>• Specific gravity, color, and appearance</td>
</tr>
<tr>
<td></td>
<td>• Biochemistry, usually includes protein, glucose, ketones, blood (hemoglobin), and urobilinogen</td>
<td>• Biochemistry, usually includes protein, glucose, ketones, blood (hemoglobin), and urobilinogen</td>
</tr>
<tr>
<td></td>
<td>• Microscopic, includes a description of cell types and number as well as presence of crystals, casts, bacteria, fat</td>
<td>• Microscopic, includes a description of cell types and number as well as presence of crystals, casts, bacteria, fat</td>
</tr>
<tr>
<td><strong>Parasites</strong></td>
<td>Fecal flotation for intestinal parasites</td>
<td>Fecal flotation for intestinal parasites</td>
</tr>
<tr>
<td></td>
<td>Heartworm antigen test</td>
<td></td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td></td>
<td>Feline leukemia virus (antigen) test and feline immunodeficiency virus (antibody) test</td>
</tr>
</tbody>
</table>

**Note:** MCH, mean corpuscular (cell) hemoglobin; MCHC, mean corpuscular (cell) hemoglobin concentration; MCV, mean cell volume; RBC, red blood cell; WBC, white blood cell.
The availability of numerous supplemental laboratory tests (see Section 5) dictates careful assessment of the initial laboratory database described. The decision to perform additional or specialized laboratory tests is based on the clinician’s interpretation of initial test results and physical examination findings.

**Imaging and Special Diagnostics**

The results of any abnormal findings observed with conventional radiography, ultrasound examination, or echocardiography or as a result of performing special diagnostic procedures are defined and included individually on the patient’s problem list. Routine, special, and invasive diagnostic procedures are described in Section 4.

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**THE MEDICAL RECORD**

Documenting health care delivery and administration of medical procedures is said to date as far back as ancient Egypt, when early physicians recognized a need to record (on papyrus) details of surgery and prescriptions. Since that time, those involved in healing or treatment have acknowledged the importance of documenting health care and communicating details of successful procedures or potions either by written methods or through an oral tradition.

The problem-oriented medical record (POMR) was introduced into human medicine in 1969. This format for recording clinical information consists of a problem list; a database (i.e., results of the history, physical examination, and laboratory findings); and then, written out separately for each problem, a plan (diagnostic, therapeutic, and educational); with a daily SOAP note (subjective, objective, assessment, and plan) entered into the medical record as a progress note. A “Master” problem list served as an index for the reader so that each problem could be followed through until it was resolved. This system widely influenced note keeping by recognizing four distinct phases of the clinical decision-making process: collecting data, formulating problems (not necessarily diagnoses), devising a management plan, then reviewing the situation and revising the plan if necessary.

In veterinary medicine, a modified version of the POMR is commonly taught in veterinary schools. Unfortunately, despite the availability of published guidelines, there are no generally adhered to standards for either making medical record entries or maintaining individual patient records in veterinary medicine. This fact becomes most apparent when veterinary medical records are subpoenaed and become the subject of legal scrutiny.

The primary purpose of the medical record is to benefit the patient by documenting the standard of care provided at the time of need and support the continuity of health care by the same or another clinician in the future. The secondary purpose of the record is to provide a medicolegal record of the care provided should there be any reason to investigate the competence of the clinicians providing care. Hence the secondary purpose of the medical record is to demonstrate the competence of the clinicians. The medical record must be respected as a legitimate, legal document and must not jeopardize the primary and secondary purposes.

Thus, a health care record can operate in the interests of a number of people and has a potentially wide audience. It is a key element in individual care, acute and preventative care, supporting and authorizing clinical care, and decision support. It provides the basis for liability in case of negligence and is a source of health care statistics.

**MEDICAL RECORD CONTENT**

Legal standards outlining the content of veterinary medical records have not been established for veterinary medicine. However, the clinician and technical support staff should consider the following information as reasonable content to include in the patient’s medical record.
INPATIENT MEDICAL RECORD

1. Patient identification data, including patient’s name, date of birth, breed, gender, current owner’s address, and medical record identifier or number
2. Admission and discharge date (and time)
3. Medical history, to include the following:
   - Chief complaint (reason for presentation)
   - Details of present illness and current medications
   - Relevant past, social, and family histories
   - Summary of relevant past examinations
4. Statement on the conclusions or impressions drawn from the admission history and physical examination
5. Statement on the course of action planned for this episode of care and its periodic review, as appropriate
6. Diagnostic tests ordered or recommended and therapeutic orders
7. Evidence of informed consent, as appropriate
8. Clinical observations and progress notes, including the results of therapy and the patient’s response to treatment
9. Information on every medication ordered or prescribed and administered, to include dosage and any adverse drug reaction
10. Reports of all operative and other invasive procedures performed
11. Reports of any diagnostic and therapeutic procedures and test results
12. Final diagnosis(es)
13. Recommended: Conclusions at termination of hospitalization to include a discharge summary, condition at discharge, disposition of care, and provisions for follow-up care
14. If applicable, cause of death and necropsy results

OUTPATIENT MEDICAL RECORD

1. Patient identification data, including patient’s name, date of birth, breed, gender, current owner’s address, and medical record identifier or number
2. Admission and discharge date and time
3. Emergency care provided to the patient by the owner or other veterinarian before arrival, if any
4. History of disease or injury
5. Physical findings
6. Results of diagnostic tests, if applicable

Note: In human medicine, a list of significant medical diagnoses and conditions, known operative and invasive procedures, known adverse and allergic drug reactions, and medications known to be prescribed for or used by the patient must be started not later than the third outpatient visit.

EMERGENCY PATIENT MEDICAL RECORD

1. Patient identification data, including patient’s name, date of birth, breed, gender, current owner’s address, and medical record identifier or number
2. Admission and discharge date and time
3. Emergency care provided to the patient by the owner or other veterinarian before arrival, if any
4. History of disease or injury
5. Physical findings
6. Results of diagnostic tests, if applicable
7. Diagnosis(es)
8. Record of treatment
9. Conclusions at the termination of treatment are documented, to include the following:
   - Final disposition
   - Condition at discharge
   - Instructions for follow-up care
10. Notation when owner refused medical care
11. Patient transfer information provided to other facilities (e.g., an after-hours emergency practice), when applicable, to include the following:
   - Reason for transfer
   - Stability of patient
   - Acceptance by the receiving organization and location (address)
   - Responsibility during transfer
   - Documentation that relevant patient information accompanied the patient

THE ORGAN SYSTEM EXAMINATION

The sections that follow address indications, options, and techniques for performing examinations on the individual patient. The organ system examinations outlined here are merely intended to serve as a guide for evaluating the ill patient. The challenge for the clinician is not limited to the examination technique but entails determining which organ systems should become the subject of a more comprehensive examination.

THE ALIMENTARY TRACT

THE DENTAL EXAMINATION

Before examining specific areas of the alimentary system, carefully observe the general physical status of the animal, particularly noting any evidence of emaciation, abdominal enlargement or asymmetry, the position of the animal at rest, and body carriage while moving (e.g., tucked up abdomen, stiffness).

In most animals a routine examination of the mouth can be done without anesthesia or tranquilization. Gently retract the lips and examine the teeth and gingiva. When examining the teeth and gingiva of a cat or a puppy, using a cotton-tipped applicator to lift the lips and even open the mouth is particularly effective.

Normal Dentition: Canine

Formula for deciduous teeth: 2 (Di3/3 Dc1/1 Dm3/3) = 28 (total)
Formula for permanent teeth: 2 (I3/3 C1/1 P4/4 M2/3) = 42 (total)

Canine eruption dates are shown in Table 2-2.

In dogs, deciduous teeth should be in place by 7 to 8 weeks. Permanent teeth begin to replace the deciduous teeth by about 4 months of age. All permanent teeth should be in place at about 7 months. In some breeds, all the permanent teeth may not be erupted until about 1 year of age.

Normal Dentition: Feline

Formula for deciduous teeth: 2 (Di3/3 Dc1/1 Dm3/2) = 26 (total)
Formula for permanent teeth: 2 (I3/3 C1/1 PM3/2 M1/1) = 30 (total)

Feline eruption dates are shown in Table 2-3.

Examination

Examine individual teeth for caries, faulty enamel, exposure of roots, deposition of calculus and plaque, and periodontitis, as well as loose, crooked, or sharp-edged (fractured) teeth. Determine the apposition of the maxilla and mandible for prognathism (undershot jaw) or brachygnathism (overshot jaw). Several systemic abnormalities, including infectious disease, renal failure, hypoadrenocorticism, diabetes mellitus, and hypoparathyroidism, can produce oral pathology.
Dental Terminology (Figure 2-3)

- **Crown.** Portion above the gum line and covered by enamel.
- **Neck.** Construction of the tooth located at the gum line where the enamel ends and the dentin covered by cementum begins.
- **Root.** Portion below the gum line and covered by cementum.
- **Furcation.** Visible space between roots of a multirooted tooth representing advanced dental disease.
- **Apex.** Most terminal portion of the root.
- **Apical delta.** Numerous small openings found at the apex allowing the nerves and vessels of the tooth to enter.
- **Enamel.** Outer covering of the crown; very shiny, hard substance. It is the hardest substance in the body and is made up of less than 5% of organic material.
- **Dentin.** Dense, bonelike material underlying the enamel and making up the substance of the tooth. It can be sensitive to heat and cold and is made up of 26% to 28% organic material. It continues to be formed in the healthy tooth by odontoblasts, cells that line the pulp chamber.

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**TABLE 2-2 Eruption Dates of Deciduous and Permanent Teeth—Canine**

<table>
<thead>
<tr>
<th>Teeth</th>
<th>Deciduous (Weeks of Age)</th>
<th>Permanent (Months of Age)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incisor 1</td>
<td>4-5</td>
<td>4-5</td>
</tr>
<tr>
<td>Incisor 2</td>
<td>4-5</td>
<td>4-5</td>
</tr>
<tr>
<td>Incisor 3</td>
<td>5-6</td>
<td>4-5</td>
</tr>
<tr>
<td>Canine</td>
<td>3-4</td>
<td>5-6</td>
</tr>
<tr>
<td>Premolar 1</td>
<td>—</td>
<td>4-5</td>
</tr>
<tr>
<td>Premolar 2</td>
<td>—</td>
<td>5-6</td>
</tr>
<tr>
<td>Premolar 3</td>
<td>—</td>
<td>5-6</td>
</tr>
<tr>
<td>Premolar 4</td>
<td>—</td>
<td>5-6</td>
</tr>
<tr>
<td>Molar 1</td>
<td>4-6</td>
<td>4-5</td>
</tr>
<tr>
<td>Molar 2</td>
<td>4-6</td>
<td>5-6</td>
</tr>
<tr>
<td>Molar 3</td>
<td>6-8</td>
<td>6-7</td>
</tr>
</tbody>
</table>

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**TABLE 2-3 Eruption Dates of Deciduous and Permanent Teeth—Feline**

<table>
<thead>
<tr>
<th>Teeth</th>
<th>Deciduous (Weeks of Age)</th>
<th>Permanent (Months of Age)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incisor 1</td>
<td>2-3</td>
<td>3½-4</td>
</tr>
<tr>
<td>Incisor 2</td>
<td>2-4</td>
<td>3½-4</td>
</tr>
<tr>
<td>Incisor 3</td>
<td>3-4</td>
<td>4-4½</td>
</tr>
<tr>
<td>Canine</td>
<td>—</td>
<td>5</td>
</tr>
<tr>
<td>Premolar 1</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Premolar 2</td>
<td>—</td>
<td>4½-5*</td>
</tr>
<tr>
<td>Premolar 3</td>
<td>—</td>
<td>5-6</td>
</tr>
<tr>
<td>Premolar 4</td>
<td>—</td>
<td>5-6</td>
</tr>
<tr>
<td>Molar 1</td>
<td>—</td>
<td>4-5</td>
</tr>
<tr>
<td>Molar 2</td>
<td>4-5*</td>
<td>—</td>
</tr>
<tr>
<td>Molar 3</td>
<td>4-6</td>
<td>—</td>
</tr>
</tbody>
</table>

*Upper only.
Cementum. Layer of bony tissue that covers the root of the tooth and is attached to the alveolar bone by the periodontal ligament fibers.

Pulp. The soft tissues of the tooth: the nerves, which contain sensory fibers only, and the vessels coming in through the apical delta extending through the length of the tooth in the root canals.

Periodontal ligaments. Network of fibrous connective tissue that attaches the tooth to the alveolar bone and to other teeth and the gingiva to the alveolus.

Gingiva. Oral mucous membranes (or “gums”).

Surfaces of the Tooth

- Buccal (vestibular). The tooth surface toward the cheek (molars).
- Labial (vestibular). The tooth surface toward the lips (incisors, canines, premolars).
- Lingual. The tooth surface toward the tongue, lower jaw.
- Palatal. The tooth surface toward the tongue, upper jaw.
- Occlusal. The tooth surface that faces the opposing tooth in the maxilla or mandible.

There are no true occlusal surfaces in carnivores such as cats.

Contact. The tooth surface that faces adjacent teeth.

Mesial. The tooth surface closest to the midline (e.g., incisors).

Distal. The tooth surface most distant from the midline (e.g., molars).

Maxillary tooth. Any tooth positioned in the maxilla (upper dental arcade).

Mandibular tooth. Any tooth positioned in the mandible (lower dental arcade).

The Dental Record

Like all medical disciplines, dentistry has its special forms. Diagnostic and therapeutic procedures should be carefully recorded. Figures 2-4 and 2-5 are examples of dental record forms.
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2  PATIENT EVALUATION AND ORGAN SYSTEM EXAMINATION

Occlusion and Dentition

The major malocclusion defects that are inherited are brachygnathism and prognathism. In brachygnathism, the maxilla is longer than the mandible (overshot). In prognathism, the mandible is longer than the maxilla (undershot). This condition is the standard for brachycephalic breeds, including Boxers, Bulldogs, and Pekingese, but it is not anatomically normal. Any occlusion other than the normal “scissors” occlusion predisposes the patient to dental disease. There can be crowding or rotation of teeth (early onset of periodontal disease) in the short jaw and trauma to the soft tissues or teeth in the long and short jaw (abnormal attrition).

Supernumerary teeth occur occasionally in the maxilla and mandible and should be extracted if they are causing problems. Oligodontia (too few teeth) can be diagnosed in large or small breeds. Radiographs should be taken to confirm tooth absence. Enamel
hypoplasia, sometimes called "distemper teeth" (Figure 2-6), may occur subsequent to infection during the ameloblastic phase (2 to 5 months of age). Tetracyclines or tetracycline derivatives should be avoided in pregnant patients and patients younger than 5 months old. Yellow staining of deciduous or permanent teeth can result after 10 or more days of consecutive treatment.

**Dental or Periodontal Abscess**

Abscessed teeth are often associated with advanced periodontal disease. A periodontal probe is inserted into the gingival sulcus to locate periodontal pockets (Figure 2-7), where tissue and bone have been lost. Loose teeth can sometimes be salvaged using techniques such as root planing and subgingival curettage. If only one root of a multirooted tooth is involved, a dental bar in a high- or low-speed dental handpiece can be used to section the tooth. The affected root is removed and a pulpotomy performed on the remaining tooth. This is an especially useful procedure for small, old dogs with one root of the lower first molar involved and the other root healthy.

**Dental Fractures**

Fractured teeth with an exposed pulp chamber often form periapical abscesses. An infraorbital abscess indicates a problem with the upper fourth premolar. This problem will not resolve permanently unless endodontic therapy or extraction is performed. A fractured lower first molar may drain into the oral cavity or through the ventral aspect of the mandible. A fractured upper canine tooth may drain into the nasal cavity or through a fistulous tract at the level of the upper first or second premolar. An inapparent
(the tooth is in place) oronasal fistula may also be present (this is seen often in old, small-breed dogs secondary to periodontal disease). A fractured lower canine tooth may drain internally or externally. Endodontic therapy and crown restoration have returned many patients to dental health.

**Oral Cavity Examination**

The normal oral mucosa is pink, partially pigmented, or completely pigmented, depending on the breed. The oral cavity should be moist, and evidence of excessive salivation and mal-odororous breath should be lacking. Examine the gingiva for color; petechiae or gross hemorrhage; hypertrophy or recession of the gingiva; any discharge around the base of the teeth; or any inflammation, swelling, or growth. Examine the hard palate for the presence of
foreign bodies. Dogs and cats with a history of sneezing and nasal discharge must be examined for evidence of an oronasal fistula (dental probe of the medial aspect of the upper canine teeth) or cleft palate. Inflammation of the mucous membranes of the mouth, stomatitis, can be associated with a variety of primary infectious agents, as well as being secondary to systemic (metabolic) diseases (e.g., chronic renal failure). Stomatitis may be associated with foreign bodies; metabolic disorders (e.g., uremia, diabetes mellitus); heavy metal poisoning (e.g., thallium); viral infections (especially those of the respiratory system in the cat); mycotic infections associated with Candida albicans (Monilia); and chemical, thermal, or electrical burns.

Technique for examining the oral cavity varies among clinicians. However, the technique used to examine a dog may not work equally well in a cat. Use of disposable examination gloves is recommended whenever examining the oral mucosa, the tongue, or related tissues. Cooperative dogs will allow the clinician to open the mouth briefly and examine at least the dorsal surface of the tongue, the hard palate, the teeth (albeit to a limited extent), and the tonsils. One single-handed, yet professional-appearing, method of performing this examination is to place the thumb of one hand in direct contact with the hard palate while gently placing the fingers of the same hand across the nose. Most dogs will open the mouth, allowing at least a limited examination. To facilitate visualizing critical structures, the fingers of the opposite hand are used to gently depress the tongue and position the head as needed (Figure 2-8).

Examination of the gingiva and the buccal surface of the teeth in cats is safely accomplished using a cotton-tipped applicator (Figure 2-9). This technique is not only a more professional approach to examining the mouth, especially in the presence of the client, but it is better tolerated by many cats, and safer, than the use of one's fingers. The technique used to actually open the mouth of cats, and small dogs, entails gently touching the hard palate with a cotton-tipped applicator rather than a finger. The stem of the applicator may then be positioned behind the lower canine teeth to facilitate opening the mouth and visualizing the oral cavity.

Note: It is not possible to adequately examine any structure caudal to the oral cavity without administering an anesthetic.

Figure 2-8: Use of only the thumb of one hand to entice a cooperative dog to open its mouth. Use of the opposite hand to facilitate examination of the teeth and oral cavity in the same cooperative dog.
Periodontal Disease

Periodontal disease is the most common oral disease in dogs and cats. Eighty-five percent to 95% of dogs and cats over 6 years of age have periodontal disease that is completely preventable. Periodontal disease is progressive and has two phases: gingivitis (reversible) and periodontitis (irreversible, but usually controllable). It is caused by the accumulation of plaque on teeth. Plaque is a soft, sticky, bacteria-laden film of saliva and debris. The bacteria and bacterial byproducts cause soft tissue inflammation. Plaque mineralizes to form calculus, which then migrates into the gingival sulcus, causing further inflammation, periodontal ligament loss, bone loss, and eventually tooth loss. The overall health of the patient with periodontal disease can be expected to decline until such time as the destructive process is stopped.

Tongue

Examine the tongue for the presence of any abnormal discoloration, membrane or pseudo-membrane, foreign bodies, inflammation, ulcers, growth, or hyperplasia. Note whether the tongue protrudes normally and whether both halves are bilaterally symmetric. The underside of the tongue should be examined for ulcers, foreign bodies such as string wrapped around the base of the tongue (in cats), hyperplasia (indicating a gum chewer syndrome), and swelling of the lingual frenulum.

Palate, Pharynx, and Buccal Mucosa

The ability of the animal to swallow effectively should be tested by stimulating the pharyngeal area. Dysphagia (see Section 3) refers to difficulty in swallowing and can be associated with localized diseases of the oropharynx or central nervous system (CNS) diseases. Thorough examination of the soft palate, oropharynx, and buccal mucosa requires administration of an anesthetic. Use of a focal light source, a tongue depressor or laryngoscope, and a spay hook (for limited examination of the nasopharynx) is important. Samples may be cultured or tissue biopsy may be performed as needed.

Retropharyngeal tumors or abscesses may produce a ventral displacement of the pharynx and larynx. Careful digital exploration of the retropharyngeal tissues may reveal a palpable mass that otherwise is not visible. Fractures involving one or more bones of the hyoid apparatus may cause dysphagia. Persistent stertor (snorting) in dogs is characteristically associated with foreign matter, occasionally tumors, trapped above the soft palate in the nasopharynx. In dogs, melanoma, squamous cell carcinoma, and fibrosarcoma are the most common oral and pharyngeal neoplasms. In cats, squamous cell carcinoma and fibrosarcoma are the most common tumors involving the oral cavity.
Tonsils
Inspect the oral mucous membranes for changes in color, hemorrhage, inflammation, abrasions, ulceration, abnormal discharges, membranes or pseudomembranes, and abnormal growths. The tonsils should be examined for symmetry, size, color, and consistency; the surrounding tissues should be examined. Conclusive diagnosis of the cause of tonsillar enlargement may depend on the results of a biopsy. Examine the uvula and note its length. Foreign material frequently lodges at the opening of the posterior nares (choanae) subsequent to coughing or vomiting. However, examination of the nasopharynx without use of an endoscope (pharyngoscopy) strictly limits the quality of an examination. Pharyngoscopy is required to adequately visualize the posterior nares and perform a biopsy. Examine the hard and soft palate for the presence of tumors or foreign bodies. Fractures of the hard palate are frequently seen in cats that fall from high elevations.

**Note:** Halitosis is one of the most common complaints registered by owners about a pet dog. Halitosis may be caused by the accumulation of bacteria on teeth (plaque); ulcerations (including tumors) of the lip folds, tongue, or mucous membranes (buccal mucosa); and tonsillitis. Uremia produces an ammonia-like odor (that not everyone equally perceives); diabetic ketosis may cause the smell of acetone; suppurative lung disease may cause a putrid odor to the breath. Many owners delay having a pet's bad breath examined, believing that halitosis is nothing more than “dog breath.”

**Examination of the Cervical Esophagus**
Examination of the esophagus is limited to external evaluation and palpation of the ventral neck. Observation of swallowing can be simply evaluated by offering (forcing) a small dose of water administered orally. Animals that manifest painful, frequent, or spontaneous attempts to swallow have dysphagia (see Section 3) and should be subjected to a more comprehensive examination. Careful palpation of the neck for evidence of obstructive lesions is an important part of the examination. However, lesions inside the esophagus must be evaluated with the use of radiography or gastroscopy. The signs of esophageal disease include regurgitation; spontaneous, frequent, or painful swallowing; and weight loss. Dogs with regurgitation associated with an esophageal lesion frequently have aspiration pneumonia and pharyngitis.

**Abdominal Palpation**
Initial examination of the abdomen involves observing abdominal conformation while the patient is standing on the examination table or walking. Unusual abdominal enlargement as well as a particularly small or tensed abdomen should be noted. Determine whether the abdominal wall moves normally during respiration. Abnormal movement may reflect pain from peritonitis. Animals manifesting abdominal pain typically stand with the hindlegs drawn forward well under the body and the back appearing arched. The patient may walk with a shorter than normal stride. When in severe abdominal pain, some animals will assume a “praying position” (lie down with front legs extended, stand up on hindlegs). Look for soft tissue edema, as well as abnormal distension of the abdominal wall.

After visual inspection, palpate the abdomen. The most effective technique for abdominal palpation entails placing the patient in right (side down) lateral recumbency (Figure 2-10). This position places the spleen, located in the left upper portion of the abdomen, in a superficial position, accessible to examination. Right-handed individuals should place the left hand beneath the patient's abdomen, palm up. Using the fingertips of the left hand, gently depress the right abdominal wall to assess when the abdominal musculature relaxes.

**Note:** Dogs that are standing during abdominal palpation cannot completely relax abdominal muscles, making assessment of abdominal organs difficult.

With the right hand kept in position approximately over the left hand, the examination proceeds using the fingertips, not the metacarpals or palms, to discern the location, size, and consistency of abdominal organs. For consistency, the examination proceeds in a
clockwise manner starting at the lower left position of the lateral abdomen (i.e., at the level of the xiphoid cartilage) to assess the ventral-most aspect of the liver. The examination continues dorsally along and beneath the costal arch, assessing the liver and, if palpable, the spleen. In the fasted state the stomach of the dog and cat cannot normally be palpated. Large tumors of the gastric wall and gastric torsion may cause the stomach to be displaced and palpable.

At the junction of the costal arch and the lumbar vertebrae, the examiner should attempt to palpate the caudal pole of the left kidney. In some dogs and virtually all cats, the entire left kidney can be palpated. Palpation of the abdomen parallel to and below the lumbar vertebrae (using both hands, one on each side of the abdomen) usually allows palpation of the colon. Even an empty colon can be palpated in most patients.

Palpation continues as the clinician positions the hands at the level of the caudal abdomen. With the hands opposed above and below the urinary bladder, the clinician should attempt to assess the size, location, and consistency of the bladder. The prostate gland of male dogs cannot normally be palpated from this position. However, in some dogs, prostatic enlargement may be so extreme that the examiner can feel the ventral prostate gland on abdominal palpation. After examination of the caudal abdomen, attention turns to the center of the abdomen. Objectively, the location and consistency of the small bowel mass and occasionally the spleen can be palpated.

<table>
<thead>
<tr>
<th>ABDOMINAL PALPATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>What Can Normally Be Palpated</td>
</tr>
<tr>
<td>What Cannot Consistently Be Palpated</td>
</tr>
<tr>
<td>---------------------------------------------------------</td>
</tr>
<tr>
<td>Ventral aspect of the liver</td>
</tr>
<tr>
<td>Caudal pole of the left kidney</td>
</tr>
<tr>
<td>Left kidney</td>
</tr>
<tr>
<td>Right kidney (cat, occasionally dog)</td>
</tr>
<tr>
<td>Colon</td>
</tr>
<tr>
<td>Urinary bladder</td>
</tr>
<tr>
<td>Small bowel mass</td>
</tr>
<tr>
<td>Uterine horns (occasionally dog)</td>
</tr>
<tr>
<td>Spleen</td>
</tr>
<tr>
<td>Cranial aspect of the liver</td>
</tr>
<tr>
<td>Gallbladder</td>
</tr>
<tr>
<td>Stomach</td>
</tr>
<tr>
<td>Right kidney (dog)</td>
</tr>
<tr>
<td>Cecum</td>
</tr>
<tr>
<td>Intraabdominal lymph nodes</td>
</tr>
<tr>
<td>Prostate gland (see discussion of rectal examination)</td>
</tr>
<tr>
<td>Adrenal glands</td>
</tr>
<tr>
<td>Posterior vena cava and abdominal aorta</td>
</tr>
</tbody>
</table>
Most dogs, although fewer cats, tolerate this technique. Abdominal palpation in uncooperative cats can usually be achieved with the patient standing on the examination table. Obesity is perhaps the most significant factor to compromise abdominal palpation. Pregnancy, depending on the stage, can also make it very difficult to distinguish discrete anatomic structures.

**Note:** Abdominal palpation is a clinical technique that does improve with experience. It is this experience that allows the skilled clinician to distinguish normal internal anatomy from abnormal.

**Abdominal Percussion**

After palpation, percuss the abdomen. The normal abdomen yields a tympanitic note throughout except over a solid organ such as the liver, the spleen, or a full bladder. Increased accumulations of air in the stomach or abdomen may result in a greater area of tympanitic sounds.

Free fluid in the peritoneum (ascites) may shift as the patient is moved. When ascites is suspected, place one hand on one side of the abdomen over the lumbar area, and with the other hand “flick” or tap the opposite abdominal wall. A distinct impact is felt from one hand to the other if fluid under tension is present.

**Abdominal Auscultation**

Carry out auscultation in a quiet room. Normal bowel sounds occur at frequent and regular intervals as liquid ingesta mixes with air during peristalsis. The absence of bowel sounds is abnormal and justifies further evaluation (e.g., abdominal radiography). Increased and decreased bowel sounds are subjective assessments and may only be variations of normal. Borborygmus, an audible rumbling noise emanating from the abdomen as air passes through the intestines, is sufficiently loud in dogs that the owner may actually hear the noise without use of a stethoscope.

**Ballottement**

The maneuver to palpate an organ floating in a fluid-filled cavity (e.g., a uterus or tumor in a fluid-filled abdomen) is ballottement. In patients having significant ascites, it may be possible to identify abnormal structures by ballottement. However, definitive assessment requires additional examination techniques (e.g., abdominal ultrasound or surgical exploration).

**EXAMINATION OF THE RECTUM, ANUS, AND ANAL SACs**

The rectum is the caudal 5 to 6 cm of the colon that communicates with the anus. Its diameter varies with the breed and size of the animal. Innervation to the anorectal area is supplied by the pudendal nerve (formed by S1, S2, and S3), which also provides motor nerves to the external anal sphincter and to the skin of the anus and perianal region. The rectum and internal anal sphincter are supplied by nerves from the pelvic plexus. Tenesmus and dyschezia are the primary signs in anorectal diseases. Carefully examine the external anal area and perineum for evidence of inflammation, swelling, ulceration, evidence of fecal staining, and matting of hair.

In most adult dogs, examination of the intestinal tract is concluded by performing a rectal examination. Digital examination (a disposable examination glove is highly recommended) may reveal the color and consistency of the stool in the rectum, any narrowing of the rectum (stricture or mass lesions), the possibility of a fractured pelvis, asymmetry of the pelvic canal, impaction or tumors of the anal glands, and the presence of rectal polyps or tumors. In medium- to large-breed male dogs, the rectal examination is an opportunity to assess the size of the prostate gland. After digital examination of the rectum, direct visualization of the rectal canal can be accomplished by use of a proctoscope or an anoscope. This procedure, however, may require sedation or anesthesia. Digital examination of the prostate in cats and young dogs is not normally performed.
Additional Reading

CARDIOPULMONARY EXAMINATION

When initial physical examination findings suggest that further evaluation of the heart and lungs is warranted, the examination should encompass assessment of patient’s behavior, at rest and while active; character of respirations; pulse character; presence (or absence) of peripheral edema or ascites; and findings of careful cardiac auscultation, thoracic radiography, electrocardiography, and, when indicated, echocardiography. Familiarity with normal and conformational variations among breeds and species will allow the veterinarian to distinguish between normal and abnormal findings.

PATIENT ASSESSMENT

Dogs and cats with cardiovascular disease may appear healthy. Even the owner may not be aware of subtle signs suggestive of heart disease. However, animals with cardiovascular disease can manifest a wide range of clinical signs, most of which are not directly referable to the heart (e.g., cough, weakness, tachypnea, or weight loss). All patients undergoing evaluation for cardiovascular disease should be observed for general appearance, attitude, and body condition, as well as respiratory rate and character.

Body Condition
Note whether the patient is overweight or underweight. Animals with advanced heart disease can be very thin (cardiac cachexia). Also note whether abdominal distension is present. Abdominal effusion (ascites), hepatomegaly, and splenomegaly may be present in animals with right-sided heart failure.

Respiratory Character
Observe the animal for both respiratory rate and breathing effort while at rest. An increase in respiratory rate and effort in a dog or cat at rest suggests dyspnea. A normal animal should not use its abdominal muscles for breathing. A dyspneic animal may breathe with its mouth open and may recruit its abdominal muscles to aid in respiration. It may also have an anxious facial expression, with eyes bulging, nostrils flaring, and head and neck extended. There are several types of dyspnea, depending on which phase of respiration is most prolonged. Inspiratory dyspnea is characterized by a longer than normal inspiratory phase, usually accompanied by stridor (noisy breathing). Inspiratory dyspnea suggests upper airway obstruction, such as laryngeal paralysis or brachycephalic syndrome. Expiratory dyspnea is characterized by forced, abdominal respirations during the expiratory phase. This type of breathing pattern suggests asthma or chronic obstructive pulmonary disease. Dyspnea throughout inspiration and expiration is typical of pulmonary diseases and pulmonary edema. The terminology used to describe normal and abnormal lung sounds is described in Table 2-4.

Neck
Animals with collapsing trachea and tracheobronchitis may readily cough after gentle manipulation or palpation of the trachea. Beware, as even a normal animal will cough with excessive tracheal palpation. With the patient’s nose angled upward, palpate the entire neck for evidence of asymmetry. Particular attention should be paid to examination of the thyroid gland, especially in adult cats. In cats, a hyperplastic thyroid gland can migrate to a lower level in the neck and may be palpable in the jugular groove at the level of the thoracic inlet.

Jugular Vein
Assess the jugular vein for distension and pulsation. Jugular venous distension suggests increased central venous pressure (CVP) and is a sign of right-sided heart failure. Jugular venous distension may occur with conditions that cause tricuspid regurgitation or impaired
## Table 2-4

<table>
<thead>
<tr>
<th>Breath Sounds</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Normal Breath Sounds</strong></td>
<td></td>
</tr>
<tr>
<td>Bronchial sounds</td>
<td>Low-pitched sounds made as air passes in and out of large airways and the trachea. Loudest over the trachea and larynx. Least audible over the caudal thorax.</td>
</tr>
<tr>
<td>Bronchovesicular sounds (also referred to as “vesicular sounds”)</td>
<td>Soft, low-pitched sounds characterized as a rustling sound. Most audible over the peripheral lung fields during inspiration.</td>
</tr>
<tr>
<td><strong>Abnormal Breath Sounds</strong></td>
<td></td>
</tr>
<tr>
<td>Crackles (also called “rales”)</td>
<td>Discontinuous, clicking or popping sounds that originate in the airways. Most audible during inspiration but may be heard on expiration. Also described as “coarse crackles” (moist rales) and “fine crackles” (dry rales). Clinically significant because they are associated with presence of increased quantities of secretions in the lumen of bronchi and lower airways; may also be heard in patients with airway collapse.</td>
</tr>
<tr>
<td>Wheezes (also called “rhonchi”)</td>
<td>Continuous sounds described as a musical or whistling noise heard during either inspiration or expiration. Also described as sibilant wheezes (high pitched) and sonorous wheezes (low pitched). May be most audible at end expiration or early inspiration. Associated with restriction of airflow through the glottis, trachea, or lower airways. Careful auscultation of the larynx, trachea, and lung fields is critical to determine the point of maximum intensity and thereby localize the source of the airway restriction.</td>
</tr>
<tr>
<td>Friction rub (pleural)</td>
<td>Combination of both continuous and discontinuous sounds caused by movement of inflamed visceral pleura over inflamed parietal pleura.</td>
</tr>
<tr>
<td>“Silent lung”</td>
<td>The absence of breath sounds (in a patient that is obviously breathing) is as important as the presence of abnormally loud breath sounds. This term is used to describe profoundly decreased, or totally inaudible, breath sounds. Clinical significance is that this is associated with significant, life-threatening airway obstruction, pneumothorax, accumulation of a large volume of fluid in the pleural space, and space-occupying lesions (e.g., diaphragmatic hernia, neoplasia). Extreme obesity and low tidal volume (especially in cats) may also be interpreted as silent lung.</td>
</tr>
</tbody>
</table>

### Descriptive Terminology for Abnormal Respiration

**Dyspnea**: Difficult or labored breathing, usually associated with an increased respiratory rate at rest

**Tachypnea**: Abnormal increase in the rate of respiration

**Hyperpnea**: Abnormal increase in both rate and depth of respiration

**Orthopnea**: Inability to breathe unless in a sitting or standing position, often with elbows abducted; positional breathing at rest
filling of the right side of the heart, such as pericardial effusion, constrictive pericarditis, or a mass lesion in the right side of the heart or cranial thorax.

Also observe for jugular venous pulsation. A jugular pulse traveling more than one third the way up the neck is abnormal and is generally associated with tricuspid valve insufficiency (i.e., congenital tricuspid valve dysplasia, or tricuspid valve insufficiency caused by chronic degeneration, pulmonary stenosis, heartworm disease, and pulmonary hypertension). An arrhythmia causing atrioventricular (AV) dissociation, such as a ventricular premature complex and second- or third-degree AV block, may also cause a jugular pulse. The atria contract against a closed AV valve, causing the pulsation. In thin animals, beware of the carotid pulse beneath the jugular vein, which may be confused with a jugular pulse.

**Thorax**

With the patient standing, palpate the thorax for evidence of trauma or deformities. One may also assess the compressibility of the chest in a cat. Compressibility is variable even in a normal cat, but this physical examination maneuver may be helpful in cats with space-occupying lesions such as a thymic mass (feline leukemia virus [FeLV]–positive cat), which will cause decreased compressibility.

**Palpation of the Precordium**

Palpate the precordium (area of chest wall next to the heart) for the apex beat. The palm of the hand, ventral surface of the proximal metacarpals, and the fingertips should be used for optimal appreciation of the precordium. The apex beat is the point of maximum intensity (PMI) of the heartbeat. The normal location is at the left fifth intercostal space around the costochondral junction. Displacement of the normal apex beat may result from right-sided heart enlargement or a thoracic mass. The intensity of the apex beat may be decreased or increased. An increased apex beat may occur in thin animals, hyperdynamic states (anemia, hyperthyroidism), mitral valve regurgitation (or other volume-overloaded state with maintained myocardial contractility), and hypertrophic cardiomyopathy. Decreased intensity of the apex beat may occur in dilated cardiomyopathy and pleural or pericardial effusion.

**Precordial thrill**

Precordial thrill is the vibration felt on the surface of the chest caused by a murmur. The location of the thrill is always the PMI of the murmur.

**Thoracic Percussion**

Thoracic percussion be helpful in animals with restrictive breathing patterns and quiet breath sounds. This technique may not be helpful in obese animals. First, the flat part of a fingertip is placed in the intercostal space. Then the fingertip (usually the middle finger) of the other hand briskly strikes the finger on the intercostal space, producing a resonating sound. Percussion of the chest should be done systematically from dorsal to ventral, evaluating the entire chest. The sound produced by percussion may be increased or hyperresonant in pneumothorax or asthma. The resonance may be decreased with pleural effusion, consolidated lung (lung filled with fluid or exudate), or a thoracic mass.

**Abdomen**

Palpate for ascites and hepatic or splenic enlargement, which may accompany right-sided heart failure in the dog. Ascites is an uncommon sign of right-sided heart failure in the cat. Ballottement is a palpation technique used to help determine if abdominal distension is caused by fluid. One taps the abdomen once or twice with the fingertips to evaluate a fluid wave or rebound of an abdominal organ floating in fluid.

**Mucous Membranes**

The color of the mucous membranes is usually assessed in the mouth, but some animals have pigmented gums, and the vulva, penis, or conjunctiva should be examined. The normal mucous membrane color is pink. Alterations in color may suggest various conditions pertinent to the cardiopulmonary systems such as the following.
Cyanosis (blue discoloration) suggests hypoxia. If cyanosis is present, the caudal mucous membrane should also be observed for differential cyanosis. Animals with a reverse (right-to-left) patent ductus arteriosus (PDA) will have more cyanosis in the caudal mucous membrane (penis or vulva) than in the oral mucous membrane (differential or caudal cyanosis).

A pale mucous membrane (light pink or white discoloration described as pallor) suggests anemia or poor perfusion associated with low cardiac output. Hyperemia of the mucous membrane (bright red) suggests peripheral vasodilation as seen in septic shock or exercise.

Capillary Refill Time
Using the gingival mucosa (nonpigmented tissue is required) above the maxillary canine tooth, gently press and release a finger against the gingiva, causing it to blanch. The CRT is the time required for blood to reperfuse the mucosal capillaries and for the tissue to return to a pink color. Reperfusion of gingiva should be apparent within 2 seconds. Prolonged CRT may indicate poor peripheral perfusion or low cardiac output. In anemic patients, a CRT may not be feasible.

Note: A normal CRT may be observed in animals up to 1 hour after death.

Arterial Pulse
Assessment of the pulse, usually the femoral pulse, is a basic and important part of the cardiovascular physical examination. The normal pulse should feel full, with a rapid rise and fall. The arterial pulse is defined as the difference between systolic and diastolic blood pressures (e.g., in a normal dog, 120 mm Hg − 80 mm Hg = 40 mm Hg pulse pressure). Femoral pulses may be difficult to assess in normal cats and obese dogs and cats. Evaluate the pulse quality as well as the rate.

Cardiac Auscultation
Stethoscope
The main components of the stethoscope are the bell, diaphragm, tubing, and earpieces. The bell transmits both low-frequency and high-frequency sounds. The diaphragm attenuates low-frequency sounds and selectively transmits high-frequency sounds. The diaphragm also transmits louder sounds than the bell by virtue of its size. The tubing should not be too long because sounds attenuate in the longer tubing. The earpieces should fit comfortably without entering the ear canals. A properly fitted stethoscope is the first step to successful auscultation. In addition to a good stethoscope, a quiet room and properly restrained patient are essential. Whenever possible, perform auscultation with the animal in a standing position. Control respirations if necessary by holding the mouth closed or occluding a nostril transiently. In some animals, it may be useful to delay auscultation until the patient relaxes. Purring in cats may interfere with auscultation, and running tap water or the smell of alcohol may stop the purring.

<table>
<thead>
<tr>
<th>ABNORMAL PULSES</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pulse deficits:</strong> For every heartbeat, there should be a pulse. A pulse deficit occurs when there is a heartbeat without a corresponding pulse, as typically occurs with arrhythmias such as ventricular premature complexes or atrial fibrillation.</td>
</tr>
<tr>
<td><strong>Hyperkinetic pulse</strong> (syn: bounding, BB-shot, or water-hammer pulse): An unusually strong pulse that rises quickly and decays quickly. Associated conditions include patent ductus arteriosus and aortic insufficiency or hyperdynamic conditions such as anemia, fever, and hyperthyroidism.</td>
</tr>
</tbody>
</table>

Care should be taken not to confuse respiratory sounds, shivering, or rubbing of the hair for heart sounds.

Proper use of the bell and diaphragm is also important for accurate auscultation. Most of the auscultation should be performed with the diaphragm firmly placed on the chest. The bell is used to hear low-frequency sounds such as gallop sounds or low-frequency
murmurs. The bell should be used with light pressure; too much pressure on the bell tightens the skin and creates a diaphragm. Auscultation with the bell should be used in animals with an extra heart sound (auscultated with the diaphragm), all cats (to screen for gallop sounds), and dogs suspected of having cardiomyopathy or with congestive heart failure. Develop a systemic approach to auscultation. Determine heart rate and rhythm and correlate it with the femoral pulse. Auscultate over all valve areas for any abnormal heart sounds.

Normal Heart Rate and Rhythm
Certainly the heart rate can vary in a normal animal depending on the state of excitement and fitness. Sometimes true resting heart rates can be determined only in the home environment by the owner. In general, the heart rate increases (tachycardia) with excitement, pain, shock, hyperdynamic states (anemia, fever, hyperthyroidism), and congestive heart failure of various causes. Slow heart rates (bradycardia) may be noted in athletic animals or with hypothyroidism, increases in vagal tone, or a conduction abnormality of the heart (sick sinus syndrome, advanced AV block).

Heart rhythm is typically regular in cats and dogs. However, many normal dogs (especially brachycephalic breeds) have respiratory sinus arrhythmia. In respiratory sinus arrhythmia, the heart rate increases with inspiration and decreases with expiration.

Normal Heart Sounds
There are four principal anatomical areas for cardiac auscultation:

1. **Mitral valve (left AV valve).** The left fifth intercostal space around the costochondral junction—in the normal animal, the area of the apex beat. Usually in the standing animal, the area opposite to the point of the elbow. First heart sounds are heard better at the mitral valve.

2. **Aortic valve.** The left fourth intercostal space dorsal to the mitral valve (usually the level of the point of the shoulder). The second heart sound is heard better at the aortic and pulmonary valves.

3. **Pulmonary valve.** The left third intercostal space at the sternal border (usually at the axilla).

4. **Tricuspid valve (right AV valve).** The right third to fourth intercostal space at the costochondral junction.

The intensity of normal heart sounds may be increased with conditions such as those related to body condition, age, and hyperdynamic states (anemia, fever, hyperthyroidism). The intensity may also be decreased with obesity or in a heavily muscled animal, as well as in pathologic conditions such as pericardial effusion or pleural effusion.

The first heart sound (S₁) is associated with closure of the AV valves, causing the phonetic sound "lubb." S₁ is typically loudest over the mitral and tricuspid valve areas. The pulse occurs just after S₁.

The second heart sound (S₂) is associated with closure of the semilunar valves (pulmonary and aortic valves), causing the phonetic sound "dupp." S₂ is typically loudest over the pulmonary and aortic valves.

<table>
<thead>
<tr>
<th><strong>NORMAL HEART RATE (DOG)</strong></th>
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</thead>
<tbody>
<tr>
<td>Large dogs: 60 to 100 beats/min</td>
</tr>
<tr>
<td>Medium-size dogs: 80 to 120 beats/min</td>
</tr>
<tr>
<td>Small dogs: 90 to 140 beats/min</td>
</tr>
</tbody>
</table>

Transient Heart Sounds

**Split S₁.** Asynchronous closure of the AV valves. May be a normal variant in a large-breed dog. Other causes include a ventricular premature complex or bundle branch block.

**Split S₂.** Asynchronous closure of the pulmonic and aortic valves. Associated conditions include heartworm disease or other causes of pulmonary hypertension, pulmonic stenosis, aortic stenosis, and atrial septal defects.
**Mid systolic click.** A high-frequency sound in the middle of systole, usually associated with early mitral valve disease. May precede a murmur in some animals.

**S₃ gallop.** A low-frequency, diastolic sound associated with rapid ventricular filling. Abnormal in the small animal patient and represents ventricular stiffness. May be associated with dilated cardiomyopathy.

**S₄ gallop.** A low-frequency, diastolic sound associated with atrial contraction. Abnormal in the small animal and represents ventricular stiffness. May be associated with hypertrophic cardiomyopathy.

**Heart Murmurs**

A heart murmur is produced by an interruption of laminar blood flow through the heart or great vessels (i.e., turbulent blood flow). The majority of heart murmurs are caused by a lesion at a level of the heart valves, causing turbulence. However, some murmurs may be physiologic, such as murmurs associated with severe anemia or shock. Murmurs can be classified in several ways: timing in the cardiac cycle, intensity, location, PMI, quality (subjective), phonographic configuration, and frequency.

**Timing**

**Systolic murmur** occurs during systole (with pulse). These murmurs are heard between S₁ and S₂. Most murmurs are systolic, and associated conditions include mitral and tricuspid valve insufficiency, aortic or pulmonary stenosis, and ventricular septal defect (VSD).

**Diastolic murmur** occurs during diastole (after the pulse), after S₂. Diastolic murmurs are rare and occur most commonly with aortic insufficiency.

**Continuous murmur** occurs throughout systole and diastole. PDA is the most common cause of a continuous murmur. One could further classify the timing of the murmur by commenting on the duration and position in the cardiac cycle, such as holosystolic (entire systole) versus early-, mid-, or late-systolic murmurs.

**Intensity**

Intensity is a subjective determination of the loudness of the murmur. In most cases, murmur intensity does not correlate with the severity of heart disease. Murmurs are usually graded on a scale of I to VI (also, 1 to 6):

- **Grade I:** Very faint murmur requiring concentration and a quiet room to be heard
- **Grade II:** Soft murmur that is consistently auscultated over only one valve area
- **Grade III:** Moderate-intensity murmur, readily auscultable, usually radiating to another valve area
- **Grade IV:** Loud murmur without a precordial thrill, usually radiating to both sides of the chest
- **Grade V:** Loud murmur with a precordial thrill
- **Grade VI:** Loud murmur with a precordial thrill and still audible with the stethoscope off the chest wall

**Note:** It is customary to record the intensity of a patient’s heart murmur in accordance with the grading scale—for example, a moderate-intensity systolic murmur would be recorded as “3/6 systolic murmur.”

**Location**

Note the valve area where the murmur is loudest (PMI). Also note where the murmur radiates. In some cases of severe subaortic stenosis, the murmur can radiate up the carotid arteries. Figure 2-11 illustrates the location of the PMI for each of the heart valves.

**Note:** Closure of three of the four heart valves is heard best over the left thorax (pulmonary, aortic, and mitral), whereas the PMI for the tricuspid valve is on the right thorax.
Patient Evaluation and Organ System Examination

Quality

Regurgitant: Quality of murmur (plateau-shaped) that is the most common and is associated with AV valve insufficiency

Ejection: Quality of murmur (crescendo-decrescendo or diamond-shaped) that is associated with aortic and pulmonary stenosis

Machinery: Quality of murmur that is continuous, most commonly associated with PDA

Decrescendo: Quality of murmur that is most commonly associated with VSD or AV valve insufficiency

Frequency

Low frequency: low rumbling sound, usually aortic insufficiency, PDA

High frequency: usually aortic or pulmonary stenosis

Mixed frequency: usually AV valve insufficiency

Common Types of Heart Murmur (see Table 2-5)

Mitral valve insufficiency (regurgitation): Early systolic to holosystolic regurgitant (plateau) or occasionally decrescendo murmur with PMI over the left apex (mitral valve area). This is the most commonly heard murmur and is usually associated with chronic degenerative mitral valve disease in the older small-breed dog.

Figure 2-11: Point of maximum intensity for murmurs associated with the A, mitral valve (left thorax). B, Aortic valve (left thorax). C, Pulmonic valve (left thorax). D, The tricuspid valve (right thorax).
**TABLE 2-5  Characterization of Heart Murmurs Heard during Cardiac Auscultation**

<table>
<thead>
<tr>
<th></th>
<th>Mitral Insufficiency</th>
<th>Patent Ductus Arteriosus</th>
<th>Aortic Stenosis</th>
<th>Pulmonic Stenosis</th>
<th>Ventricular Septal Defect</th>
<th>Anemic Murmur</th>
<th>Physiologic (Functional) Murmur</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Timing</strong></td>
<td>Systolic</td>
<td>Continuous</td>
<td>Systolic</td>
<td>Systolic</td>
<td>Systolic</td>
<td>Systolic</td>
<td>Systolic</td>
</tr>
<tr>
<td><strong>Duration</strong></td>
<td>Holosystolic</td>
<td>Holosystolic, holodiastolic</td>
<td>Midsystolic (crescendo-decrescendo or diamond-shaped murmur)</td>
<td>Midsystolic (crescendo-decrescendo or diamond-shaped murmur)</td>
<td>Holosystolic</td>
<td>Early systolic</td>
<td>Early systolic</td>
</tr>
<tr>
<td><strong>Pitch</strong></td>
<td>Early—high frequency Later—mixed frequency</td>
<td>Mixed frequency with low-frequency components</td>
<td>Harsh mixed frequency, with some high-frequency components</td>
<td>High frequency</td>
<td>Mixed frequency</td>
<td>High frequency</td>
<td>High frequency</td>
</tr>
<tr>
<td><strong>Intensity</strong></td>
<td>Usually moderate to loud</td>
<td>Usually loud</td>
<td>Usually loud</td>
<td>Usually loud</td>
<td>Usually loud</td>
<td>Usually very soft; may wax and wane</td>
<td>Very soft; may wax and wane; usually disappears by 8 wk of age</td>
</tr>
<tr>
<td><strong>Valve area</strong></td>
<td>Mitral valve area</td>
<td>Anterior on chest in area of pulmonary and aortic valve areas; may have PMI on ventral sternum cranial to left foreleg</td>
<td>Aortic valve area</td>
<td>Pulmonic area on left</td>
<td>Mitral area on left; anterior mid thorax on right</td>
<td>Mitral area</td>
<td>Aortic area</td>
</tr>
<tr>
<td><strong>Radiation</strong></td>
<td>Rightward, craniocentral, or dorsal</td>
<td>Craniodorsal</td>
<td>Cranial and rightward; thoracic inlet</td>
<td>Tends not to radiate beyond thoracic inlet; radiates to right</td>
<td>Heard on both sides of chest, but PMI is on right side</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

*PMI, Point of maximal intensity.*
Tricuspid valve insufficiency (regurgitation): Systolic regurgitant murmur that is loudest in the tricuspid valve area (right apex).

Patent ductus arteriosus: Usually a loud continuous machinery murmur with PMI at the left heart base (pulmonary and aortic valve areas). PDA is the most common congenital defect in the dog.

Pulmonary stenosis: Usually a loud, high-frequency, harsh holosystolic crescendo-decrescendo ejection murmur with a PMI at the left heart base (pulmonary valve area). The murmur usually peaks in mid-systole and radiates well caudally and to the right.

Subaortic stenosis: Usually a loud, harsh mixed or high-frequency holosystolic crescendo-decrescendo ejection murmur with a PMI at the left heart base. Sometimes the PMI is the right heart base. The murmur radiates well to the right side, up the thoracic inlet, and up the carotids. It is sometimes associated with aortic insufficiency.

Ventricular septal defect: Usually a holosystolic murmur with PMI at the right side near the sternum. VSD is one of the most common congenital defects in the cat.

Radiography of the Heart

In the absence of echocardiography, survey lateral and ventrodorsal (VD) or dorsoventral (DV) radiographs are critical in assessing patients suspected of having cardiac disease. Figure 2-12 depicts the critical anatomic features of the canine heart as seen in properly positioned and exposed radiographs. In addition, knowledge of the position and size of major vessels becomes critical in the assessment of patients with heart disease. Changes in lung vasculature may be evident on radiographs. With pulmonary congestion, the pulmonary veins are engorged with blood. With pulmonary overcirculation, the pulmonary arteries and veins are engorged. On lateral radiographs, the veins appear indistinct and tortuous and are seen emanating from the area of the left atrium. On the other hand, the pulmonary arteries appear straight and branching, like a tree. On the DV view, veins are medial and arteries are lateral to each bronchus. Decompensated mitral insufficiency causes pulmonary venous congestion, heartworm disease, chronic lung disease, and congenital left-to-right shunt pulmonary artery enlargement.

The mediastinum is a compartment of the thorax between the medial aspects of the two pleural sacs. The mediastinal pleural layers are thin. Disorders such as pneumothorax and pleural effusion seldom remain unilateral. Signs related to abnormalities in the mediastinal area may be dysphagia, regurgitation, coughing and dyspnea, syncope, head and neck edema, thoracic pain, abdominal breathing, Horner syndrome, and emphysema.

Survey Radiographs of the Heart

1. The normal canine heart is on a 45-degree angle with the sternum.
2. The heart extends from T1 to T8.
3. Breed variation can greatly affect the appearance of the cardiac silhouette, as can the respiratory and cardiac cycle.
4. The heart of the cat assumes a more elongated and elliptic position than that of the dog; the feline heart occupies two to two-and-a-half intercostal spaces, and the caudal border is separated from the diaphragm by one or two intercostal spaces.
5. In VD and DV positions, the canine heart has a curved right border and a straight left border, with the long axis oriented at a 30-degree angle to the spine and to the left of the midline.
6. The feline heart is more oval in appearance in the VD position; in the DV view, the cardiac apex is just to the left of the midline. The ratio of the longitudinal axis to the transverse axis is 1.4:1.

Radiographic evaluation of the cardiovascular system and lungs is important in the differential diagnosis of cardiovascular disease. It is especially important to evaluate (1) enlargement of cardiac chambers; (2) dilation of great vessels; (3) increased or decreased pulmonary circulation; (4) venous congestion, pulmonary edema, and pleural effusion; and (5) mediastinal space.
When interpreting changes in cardiac size and shape, note the consistency of the radiographic technique. Short radiographic exposure times of \( \frac{1}{60} \) or \( \frac{1}{120} \) second with radiographs taken at full inspiration give the best results.

**Enlargement of Right Atrium**

Right atrial enlargement is usually associated with right ventricular enlargement.

1. Bulging cranial heart border on lateral view
2. Bulging at the 9- to 11-o'clock position on VD (DV) view.

*Figure 2-12:* A, Radiographic anatomy of the right side of the heart (left lateral thoracic radiograph). 4, Fourth rib; AV, junction of the azygos vein with the AVC; AVC, cranial vena cava; CA, right ventricular outflow tract (tricuspid valve [arrow]); LPA, left pulmonary artery; PA, main pulmonary artery; PVC, caudal vena cava; RA, right atrium; RPA, right pulmonary artery; RU, right auricle; RV, right ventricle. B, Radiographic anatomy of the left side of the heart (left lateral thoracic radiograph). A, ascending aorta; AA, aortic arch; AU, small portion of the left auricle; BA, brachiocephalic artery; DA, descending aorta; LSA, left subclavian artery; LV, left ventricle; PV, pulmonary veins; S, sinus of Valsalva (arrow points to aortic valve); T, tracheal bifurcation (carina).

(Continued)
Right Ventricle
1. Cranial border of the heart is more rounded with increased sternal contact, and the heart may be elevated dorsally on lateral view.
2. Overall width of heart is increased.
3. Elevation of trachea, cranial to tracheal bifurcation.
4. VD (DV) view: heart rounded from 6- to 11-o’clock position.
5. Distance between right heart border and thoracic wall is decreased.

Left Atrium
1. Bulging caudal dorsal heart border on lateral view.
2. Loss of caudal waist on lateral view.
3. Elevation of trachea, compression of main stem bronchi.
4. Bulging at 2- to 3-o’clock position on VD (DV) view.
5. Increased size of pulmonary veins.

Left Ventricle
1. Elongation of cardiac silhouette on either lateral or VD (DV) view.
2. Elevation of trachea.
3. Rounded caudal border of the heart.
4. Distance between left heart border and thoracic wall is decreased on VD (DV) view.

Biventricular Enlargement
1. Heart appears rounded on both views.
2. Increased sternal contact on lateral view, with elongation and widening of the heart shadow.
3. May mimic pericardial effusion if uniform and severe.
Decrease in Size of Cardiac Silhouette.
1. Heart elevated off the sternum.
2. Increase in ratio of longitudinal axis to transverse axis to more than 1.4:1.
3. Shifting of heart away from midline.
4. Small caudal vena cava.
5. Seen in Addison’s disease, hypothyroidism, shock, and pneumothorax.

Differential Diagnoses Based on Survey Radiographs (Table 2-6)
A severe degree of cardiomegaly with evidence of right-sided heart failure suggests advanced mitral and tricuspid valvular fibrosis, dilated cardiomyopathy, or pericardial effusion. Nonselective angiocardiography can be helpful in distinguishing among cardiomyopathy, congenital cardiac abnormalities, and pericardial effusion.

Radiographic Appearance of the Lungs in Left-Sided Heart Failure
1. Pulmonary congestion. Engorgement and distension of pulmonary veins, especially at the junction of the veins with the left atrium. Pulmonary radiodensity is unchanged.
3. Alveolar edema. Fluid enters the alveoli and peripheral bronchioles, creating alveolar radiodensity and air bronchograms. Alveolar radiodensity is most severe in the perihilar area.

The lung fields should also be carefully reviewed for evidence of vascular changes compatible with heartworm disease or pulmonary embolism.

<table>
<thead>
<tr>
<th>Differential Diagnoses for Patients with Cardiomegaly</th>
</tr>
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<tbody>
<tr>
<td><strong>Left-Sided Cardiomegaly</strong></td>
</tr>
<tr>
<td><strong>Dilation</strong></td>
</tr>
<tr>
<td>Mitral regurgitation</td>
</tr>
<tr>
<td>Right-to-left shunting (PDA)</td>
</tr>
<tr>
<td>Aortic insufficiency</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
</tr>
<tr>
<td>Feline hyperthyroidism</td>
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<td></td>
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<tr>
<td><strong>Hypertrophy</strong></td>
</tr>
<tr>
<td>Aortic stenosis</td>
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<tr>
<td>Hypertension</td>
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<tr>
<td>Hypertrophic cardiomyopathy</td>
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</table>

PDA, Patent ductus arteriosus; VSD, ventricular septal defect.
Note: In patients with right-to-left shunting PDA, the characteristic “machinery murmur” becomes systolic.
Disease Processes That Alter Mediastinal Position
1. Unilateral pleural or pulmonary masses
2. Unilateral pneumothorax or pleural effusion
3. Lung lobe collapse, agenesis, hypoplasia, or resection
4. Pleural adhesions
5. Hypostatic congestion of a lung

Diseases That Result in Mediastinal Widening
1. Accumulation of mediastinal fat or fluid
2. Inflammation secondary to tracheal or esophageal puncture
3. Hemorrhage
4. Tumor formation (lymphosarcoma, thymoma)
5. Heart base tumors
6. Enlargement of tracheobronchial lymph nodes
   - The intrathoracic trachea is about three times the width of the proximal third rib but
   increases in diameter on inspiration and decreases on expiration. The normal trachea enters
   the thoracic inlet in the dorsal third of the inlet. The intrathoracic trachea may collapse on
   expiration, which may extend to the carina and main stem bronchi.
   - Congenital tracheal hypoplasia is seen in the English bulldog. Tracheal compression or
     left main stem bronchus compression may be associated with enlargements of tracheobron-
     chial lymph nodes or of the left atrium.

Advanced Diagnostic Tests
Section 4 of this text describes in detail the basic requirements for performing advanced
cardiac examination, including electrocardiography.

Additional Reading
Cote E: Electrocardiography and cardiac arrhythmias. In Ettinger SJ, Feldman EC, editors: 
Herrtage ME: Cardiovascular disorders. In Schaer M, editor: Clinical Medicine of the Dog and 
Sisson DD: Pathophysiology of heart failure. In Ettinger SJ, Feldman EC, editors: Textbook of 
Veterinary Internal Medicine, ed 7, St Louis, 2010, Elsevier.

INTEGUMENT (SKIN, HAIR COAT, AND TOENAILS)

Clinical History
The owner’s chief complaint is often the major sign used in compiling a differential diagno-
sis for animals with skin disease. It is important that the questions presented to the client do
not suggest answers. The clinician should obtain a complete medical history. It is vital to use
a systematic, detailed method of examination and history taking so that important
information is not overlooked.

Some dermatologic disorders are age related, so age is important in the dermatology
history. For example, demodicosis usually begins in young dogs before sexual matur-
ity. Allergies tend to appear in more mature individuals, probably because repeated
exposure to the antigen must occur before clinical signs develop. Hormonal disorders tend to occur in animals 6 to 10 years of age, and most neoplasms develop in mature to older patients.

The sex of the patient obviously limits the incidence of certain problems, but it is especially important in sex hormone imbalances. Perianal adenomas are seen almost exclusively in male dogs. One should determine whether the patient is sexually intact and, if so, whether the skin problem bears any relationship to the estrous cycle.

Breed predilection determines the incidence of some skin disorders. For example, seborrhea is common in Cocker Spaniels; acanthosis nigricans is common among Dachshunds; adult-onset hyposomatotropism occurs in Pomeranians, Keeshonds, and Chow Chows; dermatomyositis is found in Shetland Sheepdogs and Collies; zinc-responsive dermatosis occurs in Siberian Huskies and Alaskan Malamutes; and many of the wire-coated terrier breeds (Scotties, Cairns, Sealyhams, West Highland Whites, Irish Terriers, and Welsh Terriers) seem to be particularly predisposed to allergic skin disease.

The following information should be obtained from the owner: date of onset, original locations of the lesions, description of the initial lesions, tendency toward progression or regression, factors affecting the course and duration, and previous treatment (home, proprietary, or pet shop remedies used, as well as prescribed therapies).

**EXAMINATION**

Recording historical facts, physical findings, and laboratory data in a systematic way is particularly important for patients with skin disease. Many dermatoses are chronic, and skin lesions are slow to change. For this reason, digital images of the patient’s lesions enable the clinician to document the location and extent of lesions.

Figure 2-13 illustrates a conventional record form for noting physical and laboratory findings for dermatology cases. The special form enables one to circle pertinent descriptive terms, saves time, and ensures that no important information is omitted. This form details only dermatologic data and should be used as a supplement to the general history and physical examination record. A special dermatologic history form, completed by the client, is also useful for patients with a documented allergy or other chronic skin disease (Figure 2-14). The examination should be performed with good lighting. Normal daylight without glare is best, but any artificial light of adequate candlepower is sufficient if it produces bright, uniform lighting. The lamp should be adjustable to illuminate all body areas. A combination loupe and light magnifies and illuminates the field. Before individual lesions are evaluated, the entire animal should be observed from a distance for a general impression of abnormalities and to observe distribution patterns. Palpation of the skin is important to establish the texture of the hair (coarse or fine, dry or oily). A change in the amount of hair present is often a dramatic finding. Alopecia (focal or generalized) refers to the lack of hair in areas where hair is normally present. The texture, elasticity, and thickness of the skin should be determined, and impressions of heat or coolness recorded. It is important to examine every inch of the skin and mucous membranes. It is easier to find important skin lesions in some breeds than in others, depending on the thickness of the coat. The density of an individual’s coat varies in different body areas. Lesions can be discerned more easily in sparsely haired regions. However, the clinician must part or clip the hair in many areas to observe and palpate lesions that are partially covered.

**Assessment of Individual Lesions**

The evolution of lesions should be determined from the history or by finding different stages of lesions on the same patient (Table 2-7). Papules often develop into vesicles and pustules, which may rupture to leave erosions or ulcers with epidermal collarettes and finally crusts. As lesions develop in special patterns, they also involute in characteristic ways. Acute lesions often appear suddenly and disappear quickly and completely. Chronic lesions may leave diagnostically important pigmentation or scars that persist for months or become permanent (e.g., chronic generalized demodicosis and juvenile cellulitis, respectively).
**DERMATOLOGY EXAMINATION**

<table>
<thead>
<tr>
<th>PRIMARY LESIONS (Check)</th>
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<tbody>
<tr>
<td>Macule</td>
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<td>Patch</td>
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<tr>
<td>Papule</td>
<td></td>
<td>Nodule</td>
</tr>
<tr>
<td>Pustule</td>
<td></td>
<td>Vesicle</td>
</tr>
<tr>
<td>Abscess</td>
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<table>
<thead>
<tr>
<th>SECONDARY LESIONS (Check)</th>
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<th></th>
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</thead>
<tbody>
<tr>
<td>Scale</td>
<td></td>
<td>Crust</td>
</tr>
<tr>
<td>Erosion</td>
<td></td>
<td>Ulcer</td>
</tr>
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<td>Excoration</td>
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<tr>
<td>Hyperpigmentation</td>
<td></td>
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</tr>
<tr>
<td>Hyperkeratosis</td>
<td></td>
<td></td>
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<tr>
<td>Sinus</td>
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</tbody>
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<table>
<thead>
<tr>
<th>SKIN CHANGES (Check)</th>
<th></th>
<th></th>
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<tbody>
<tr>
<td>Normal</td>
<td></td>
<td>Thick</td>
</tr>
<tr>
<td>Hypotonic</td>
<td></td>
<td>Hyperextensible</td>
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<td></td>
</tr>
</tbody>
</table>

**DISTRIBUTION OF LESIONS**

Weight [ ]

Figure 2-13: Physical examination form appropriate for use in patients presented because of complex or chronic dermatologic problems.

*(Continued)*
| HAIRCOAT CHANGES (Check)                  | -------------- | -------------- | -------------- |
| Alopecia          | Hypotrichosis  | Hypertrichosis  |
| Dry Coat          | Brittle Coat   | Oily Coat      |
| Easy Epilation    | 1st Hairs      | 2nd Hairs      | Both           |
| Hair Casts        | Color Associated Hair Loss |

| CONFIGURATION OF LESIONS (Check)          |    |
| Linear            | Follicular    | Grouped       |
| Annular           | Other         |               |

| PRURITUS (Check)            |    |
| Seasonal                   | Nonseasonal | Lesional      |
| Face                       | Ears        | Feet/Legs     | Rump          |
| Axillae                    | Abdomen     | Other         |

| CUTANEOUS PAIN (Check)      |    |
| Absent                      | Mild         | Moderate      | Severe        |

| PARASITES (Check)           |    |
| Fleas                       | Flea Dirt    | Lice          | Ticks         |
| Ear Mites                   | Other        |

*Can have either dog or cat outline here

| Ears L                  |    |
| R                       |    |
| Oral                    |    |
| Anogenital              |    |
| Footpads                |    |
| Nails                   |    |
| Other                   |    |

| LABORATORY               |    |
| Scrape                   |    |
| Scotch Tape              |    |
| Fungal Cult              |    |
| Wood's Light             |    |
| Hair Exam                |    |
| ID Hist                  | Flea 15   | Flea 24       |
| Cytophagy                | 1.         | 2.            |
| 2.                       | 3.         | 4.            |

| DIAGNOSIS/DIFFERENTIAL    |    |
|                          |    |

Figure 2-13:—cont’d
When was the problem first noted? Day Month Year
Where on the body did the problem begin?

Is the problem: Year Round Seasonal Unknown
If seasonal, in which season(s) is it worse? Spring Summer Fall Winter
If nonseasonal, is it worse in any season?

Does the animal itch (scratch, chew, lick, rub)? Yes No
Is the itching: Mild Moderate Severe Constant Periodic
Where does the animal itch? Check those areas which are itchy.

Face: Abdomen: Lower Back:
Ears: Front Feet/Legs: All Over:
Arm Pits: Back Feet/Legs:

What medications have been used?

Drug How Much How Often Did It Help?

Do parents, littermates, other animals in the house or other animals in the area have a similar problem? Yes No

On the reverse side, please provide any information which you feel is important.

Figure 2-14: Dermatology history form for owners to complete for patients with complex or chronic dermatologic problems.

(Continued)
DERMATOLOGY HISTORY

Chief Complaint:

Itching    Sores
Hair loss  Ear Disease
Other

Aside from the skin problem is the animal healthy?

Yes    No

(Please Specify)

Figure 2-14:—cont’d
Morphology of skin lesions is the essential feature of dermatologic diagnosis and sometimes is the only guide if laboratory procedures yield no useful information. Most skin diseases are characterized by a single type of lesion.

The clinician must learn to recognize primary and secondary lesions. A primary skin lesion is one that develops spontaneously as a direct reflection of underlying disease. A secondary skin lesion evolves from primary lesions or consists of artifacts induced by the patient or by external factors such as trauma or medications (Table 2-8). Careful inspection of the diseased skin will frequently reveal a primary lesion suggestive of a specific dermatosis. In many cases, however, the significant lesion must be differentiated from the mass of secondary debris. The ability to discover a characteristic lesion and understand its significance is the first step toward mastering dermatologic diagnosis. Variations are common, because early as well as advanced stages exist in most skin diseases. In addition, the appearance of skin lesions may change with medication, self-inflicted trauma, and secondary infection.

The following basic tests are indicated in assessing the severity and extent of dermatologic lesions in dogs and cats.

Several simple techniques for initial examination of skin lesions should be considered:

1. **Diascopy**: This technique entails pressing a clear piece of plastic or glass (a clean microscope slide) over an erythematous lesion. If the lesion blanches on pressure, the erythema is a result of vascular engorgement. If the lesion does not blanch, there is hemorrhage into the skin (petechiae or ecchymoses).

2. **Nikolsky sign**: This sign is elicited by applying pressure on a vesicle or pustule or at the edge of an ulcer or erosion or even on normal skin. The result is positive when the outer layer of the skin is easily rubbed off or pushed away. This indicates poor cellular cohesion, as found in the pemphigus complex, pemphigoid, and toxic epidermal necrolysis.

3. **Skin scrapings**: This procedure can be performed (gently) with a No. 10 surgical blade, mineral oil (as a vehicle to suspend material in), and a glass slide. Assessment centers on identification of ectoparasites, using a microscope.

4. **Wood’s lamp**: This ultraviolet light source with a cobalt or nickel filter is used to screen patients for the presence of dermatophytes. Some experience is required in making accurate interpretations, as keratinized skin and debris in the hair may appear to fluoresce. The Wood lamp test does not have a positive result with all dermatophytes. In fact, it is estimated that less than half of patients with fungal skin disease will have a positive Wood lamp test result. A negative Wood lamp test result does not rule out dermatophytosis as the diagnosis.

5. **Culture**: Submission of samples for bacterial or fungal cultures may be indicated in patients with particularly complex, chronic lesions that do not respond to empiric therapy. Hair samples from patients suspected of having a dermatophyte infection can be inoculated onto dermatophyte test medium (DTM). In addition, direct

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**Table 2-7 Classification of Skin Lesions**

<table>
<thead>
<tr>
<th>Primary Lesions (Lesions of First Diagnostic Importance)</th>
<th>Secondary Lesions (Evolutionary or Complicating Lesion of Secondary Diagnostic Importance)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Macule—patch</td>
<td>Pustule</td>
</tr>
<tr>
<td>Papule—plaque</td>
<td>Tumor—neoplasm</td>
</tr>
<tr>
<td></td>
<td>Nodule</td>
</tr>
<tr>
<td></td>
<td>Vesicle—bulla</td>
</tr>
<tr>
<td></td>
<td>Wheal</td>
</tr>
<tr>
<td>Scale—epidermal collaret</td>
<td>Comedo</td>
</tr>
<tr>
<td>Crust</td>
<td>Pigmentary abnormalities</td>
</tr>
<tr>
<td>Scar</td>
<td>(hyperpigmentation or hypopigmentation)</td>
</tr>
<tr>
<td>Erosion—ulcer</td>
<td>Lichenification</td>
</tr>
<tr>
<td></td>
<td>Hyperkeratosis—callus</td>
</tr>
</tbody>
</table>

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cytologic examination of skin scrapings and pustules may be helpful in assessing the underlying cause.

6. **Acetate tape:** Applying acetate (clear) adhesive tape to the hair coat may actually capture diagnostic organisms (e.g., *Cheyletiella* species mites [“walking dandruff”]). In addition, the acetate tape may be stained with lactophenol cotton blue or Diff-Quik for further cytologic evaluation.

7. **Skin biopsy:** Using 4-mm, 6-mm, or 8-mm punch biopsy instruments, full-thickness skin biopsies, performed in the appropriate locations, can be particularly useful in establishing a diagnosis. Location of the biopsy is critical in obtaining diagnostic tissue. Generally, multiple lesions should be biopsied. Surgical preparation of the skin is not recommended, as this will alter the histologic appearance of the sample and may compromise the ability to establish a diagnosis.

8. **Allergen-specific immunoglobulin E (IgE) serology:** Serum is submitted to a diagnostic laboratory for in vitro testing of atopic dermatitis.

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**Table 2-8 Systemic Diseases with Cutaneous Lesions**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Skin Lesions or Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atopy</td>
<td>Pruritus</td>
</tr>
<tr>
<td>Castration-responsive dermatosis</td>
<td>Alopecia</td>
</tr>
<tr>
<td>Cold agglutinin diseases</td>
<td>Erythema, purpura, necrosis, ulceration</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>Atrophy, ulceration, pyoderma, seborrhea</td>
</tr>
<tr>
<td>Dirofilariasis</td>
<td>Erythema, alopecia, pruritus, nodules</td>
</tr>
<tr>
<td>Erythema multiforme</td>
<td>Macules, papules, vesicles, wheals</td>
</tr>
<tr>
<td>Feline leukemia virus infection</td>
<td>Pyoderma, seborrhea, poor healing, cutaneous horns on footpads</td>
</tr>
<tr>
<td>Hepatocutaneous syndrome</td>
<td>Mucocutaneous crusts and ulcers, footpad hyperkeratosis and ulcers</td>
</tr>
<tr>
<td>Hyperadrenocorticism</td>
<td>Alopecia, hyperpigmentation, calcinosis cutis, pyoderma, seborrhea, phlebectasias, thin and hypotonic skin</td>
</tr>
<tr>
<td>Hypothyroidism</td>
<td>Alopecia, hypothermia, seborrhea, pyoderma, hyperpigmentation, myxedema, galactorrhea</td>
</tr>
<tr>
<td>Leishmaniasis</td>
<td>Erythema, nodules, ulceration, exfoliative dermatitis</td>
</tr>
<tr>
<td>Male feminizing syndrome</td>
<td>Alopecia, seborrhea, hyperpigmentation, gynecomastia, galactorrhea</td>
</tr>
<tr>
<td>Mycoses, deep</td>
<td>Nodules, ulceration, fistulas</td>
</tr>
<tr>
<td>Mycosis fungoides</td>
<td>Erythroderma, plaques, nodules, ulceration</td>
</tr>
<tr>
<td>Ovarian imbalances</td>
<td>Alopecia, hyperpigmentation, seborrhea</td>
</tr>
<tr>
<td>Pemphigus</td>
<td>Purulent exudate, crusting, vesiculation, ulceration or erosion</td>
</tr>
<tr>
<td>Pituitary dwarfism</td>
<td>Alopecia, cutaneous degeneration, hyperpigmentation</td>
</tr>
<tr>
<td>Sertoli cell tumor</td>
<td>Alopecia, gynecomastia, hyperpigmentation</td>
</tr>
<tr>
<td>Systemic lupus erythematosus</td>
<td>Pyoderma, seborrhea, ulceration, pruritus, erythema</td>
</tr>
<tr>
<td>Thallium toxicosis</td>
<td>Alopecia, erythema, ulceration</td>
</tr>
<tr>
<td>Toxic epidermal necrolysis</td>
<td>Ulceration, blisters, pain</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>Nodules, ulceration, fistulas</td>
</tr>
</tbody>
</table>

**Note:** IgE serology is not indicated for patients with suspected food allergy. Although the quality of serologic assays for allergic skin disease has improved, some concerns are expressed by dermatologists over the predictive value of these tests.
9. **Intradermal skin testing**: This testing platform is valuable in diagnosing and managing allergic skin disease but must be performed and interpreted by a dermatologist or an individual with considerable experience in administering and interpreting intradermal skin tests.

**Cutaneous Manifestations of Systemic Disease**

Cutaneous changes that accompany internal disease may result from simultaneous involvement of skin and internal organs with identical pathologic mechanisms (multicentric disease), direct extension of an internal disease process to the skin, immunologic manifestations of a deficient or hyperactive immune system, hormonal deficiency or excess, or metabolic derangements. These cutaneous changes may be obvious or subtle, mild or extensive, or incidental or specific. When the cutaneous change has a high correlation to a specific internal disease process, the lesions are referred to as a **marker** of internal disease (Table 2-9). Clinicians must examine the patient with dermatologic disease while considering internal pathologic factors.

Systemic disease should be suspected in patients with dermatologic disorders when the following conditions are observed:

1. Dermatologic disease concurrent with systemic illness, such as fever, depression, or clinical signs consistent with a particular organ system (e.g., diarrhea, lameness)
2. Bizarre or atypical dermatoses.
3. Chronic recurring dermatoses, including pyoderma, and scaling
4. Dermatoses in unexpected patients (e.g., age, breed, sex)
5. Dermatologic signs after illness or drug administration

**Distribution Patterns of Skin Lesions**

A dramatic change becomes apparent when a skin disorder affects an animal whose body is covered with a dense hair coat. Even the most casual observer is aware of the loss or hair in certain areas. The alopecic pattern, which is often sharply demarcated, assumes a new meaning when it is accurately interpreted. When alopecia and other hair changes are evaluated according to their distribution pattern over the entire body, significant diagnostic clues appear. Comparatively speaking, only on the human scalp is alopecia as striking and meaningful.

In animals, the primary or secondary skin lesions are often hidden under the hair coat; in fact, it requires painstaking observation to see them. In short-coated animals, using both hands to roll the skin into a horizontal fold enables visualization of the interface between individual hair shafts and the skin surface. Rolling the fold backward facilitates evaluation and distribution of subtle skin lesions. Only after the hair is clipped is it possible to see the distribution pattern of such lesions with ease and accuracy. Consequently, in animals there are two distinctly different patterns that aid in diagnosis: (1) the changes in external hair coat and (2) the definition and distribution of primary and secondary skin lesions. These two factors do not necessarily have a reciprocal relationship. In addition, it is important to recognize whether the lesions present a symmetric distribution on either side of the midline or an asymmetric distribution.

**Additional Reading**


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<table>
<thead>
<tr>
<th>Category</th>
<th>Representative Diseases</th>
<th>Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fungal</td>
<td>Deep mycoses</td>
<td>Nodules, draining tracts, suppurative-ulceration (can be aggressive in patients with P. insidiosum infection)</td>
</tr>
<tr>
<td></td>
<td>Blastomycosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Histoplasmosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Coccioidiomycosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pythium insidiosum</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lagenidium species</td>
<td></td>
</tr>
<tr>
<td>Viral</td>
<td>Canine herpesvirus-1 and calicivirus</td>
<td>Hyperkeratosis (foot pads and nasal planum), oral and cutaneous ulceration, keratitis</td>
</tr>
<tr>
<td></td>
<td>Feline herpesvirus (Felv) and feline immunodeficiency virus</td>
<td>Hyperkeratosis, gingivitis, stomatitis, pyoderma, recurrent abscesses, demodectic mange, dermatophytosis</td>
</tr>
<tr>
<td></td>
<td>Hyperkeratosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Canine distemper virus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Feline leukaemia virus (FeLV) and Feline immunodeficiency virus</td>
<td></td>
</tr>
<tr>
<td>Bacterial</td>
<td>Tick-borne infections</td>
<td>Plaques, nodules, draining tract, suppurative-ulceration</td>
</tr>
<tr>
<td></td>
<td>Rocky Mountain spotted fever</td>
<td></td>
</tr>
<tr>
<td>Parasitic</td>
<td>Leishmaniais (protozoa)</td>
<td>Icterus, petechiae, exfoliative dermatitis on the head, neck, and extremities.</td>
</tr>
<tr>
<td></td>
<td>Trypanosomiasis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ehrlichia species</td>
<td>Erythematous macule and papules (often associated with neoplasia)</td>
</tr>
<tr>
<td></td>
<td>Demodirosis (mite)</td>
<td>Erythematous macule and papules (often associated with neoplasia)</td>
</tr>
<tr>
<td></td>
<td>Demodicosis (mite)</td>
<td>Erythematous macule and papules (often associated with neoplasia)</td>
</tr>
<tr>
<td></td>
<td>Demodirosis (mite)</td>
<td>Erythematous macule and papules (often associated with neoplasia)</td>
</tr>
<tr>
<td></td>
<td>Dermoidosis (mite)</td>
<td>Erythematous macule and papules (often associated with neoplasia)</td>
</tr>
<tr>
<td>Immune-</td>
<td>Pemphigus and the bullous dermatoses</td>
<td>Erythema multiiforme</td>
</tr>
<tr>
<td>mediated</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Systemic lupus erythematosus (SLE)</td>
<td>erythema multiiforme</td>
</tr>
<tr>
<td></td>
<td>Ischemic vasculitis</td>
<td>erythema, superficial or deep pyoderma (secondary bacterial dermatis)</td>
</tr>
<tr>
<td></td>
<td>Hemolytic uremic syndrome</td>
<td>erythema, superficial or deep pyoderma (secondary bacterial dermatis)</td>
</tr>
<tr>
<td></td>
<td>Dermatomyositis</td>
<td>erythema, superficial or deep pyoderma (secondary bacterial dermatis)</td>
</tr>
<tr>
<td></td>
<td>Dermatomyositis</td>
<td>erythema, superficial or deep pyoderma (secondary bacterial dermatis)</td>
</tr>
</tbody>
</table>

(Continued)
<table>
<thead>
<tr>
<th>Category</th>
<th>Representative Diseases</th>
<th>Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neoplasia</td>
<td>Feline vaccine-associated sarcoma (VAS)</td>
<td>Aggressive fibrosarcoma associated primarily with adjuvanted FeLV and rabies vaccines in cats; may occur months to years after inoculation.</td>
</tr>
<tr>
<td></td>
<td>Multiple primary skin tumors</td>
<td>Mast cell tumor, lymphoma, squamous cell carcinoma, and so on; melanoma in skin of dogs is reported but is typically benign, despite histologic appearance of cells.</td>
</tr>
<tr>
<td></td>
<td>Nodular dermatofibrosis</td>
<td>Subcutaneous nodules (coalescing) with associated alopecia and hyperpigmentation; seen most often in German Shepherd Dogs, Boxers, and Golden Retrievers but is seen occasionally in other breeds; may be associated with renal disease.</td>
</tr>
<tr>
<td></td>
<td>Testicular neoplasia</td>
<td>Male feminization syndrome.</td>
</tr>
<tr>
<td></td>
<td>Pheochromocytoma</td>
<td>Intermittent flushing, especially of the pinnae; other systemic signs are associated with this tumor.</td>
</tr>
<tr>
<td></td>
<td>Paraneoplastic syndromes</td>
<td>Various focal and regional changes in skin are reported, including fissures, nodular skin disease, pemphigus vulgaris, necrotizing panniculitis, and exfoliative dermatitis in cats (thymoma).</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Hypothyroidism, canine</td>
<td>Dry hair coat, symmetric, nonpruritic (usually) truncal alopecia, hyperpigmentation, seborrheic skin disease.</td>
</tr>
<tr>
<td></td>
<td>Hyperthyroidism, feline</td>
<td>Poor hair coat, matting, excessive shedding, increased nail growth.</td>
</tr>
<tr>
<td></td>
<td>Hyperadrenocorticin (canine Cushing syndrome)</td>
<td>Symmetric, truncal alopecia, comedones, thinning of the skin, failure of hair regrowth after clipping; calcinosis cutis (approximately 5% of cases).</td>
</tr>
<tr>
<td></td>
<td>Diabetes mellitus</td>
<td>Otitis externa, pyoderma, demodectic mange, thin skin, pyoderma, seborrheic skin disease, and, in cats, xanthoma formation.</td>
</tr>
<tr>
<td>Nutritional</td>
<td>Vitamin E deficiency in cats</td>
<td>Pansteatitis.</td>
</tr>
</tbody>
</table>
OPHTHALMIC (OCULAR) EXAMINATION

GLOBE AND ADNEXA

Examination of the globe and external ocular structures should be conducted before any detailed examination of the eye is undertaken. Inspect the globe in normal daylight or room light and observe the relationship of the globe to the orbit and the eyelids. Note whether the eyes are in the same visual axis or whether atropia is present. Observe any undue prominence of either or both eyes. Note the presence of any other facial lesions (e.g., facial paralysis) that may affect the symmetry of the orbit. Inspect the external ocular structures (lids, conjunctiva, cornea, sclera, and lacrimal apparatus). Note the position of the eyelids; the size of the palpebral aperture; the position of the nictitating membrane; and the presence of nystagmus (involuntary, rapid oscillations of the eyes), anisocoria (unequal pupils), blepharospasm (tonic spasm of the eyelids), lagophthalmos (drooping of the eyelid), or ocular discharges.

Tonic Eye Reflexes

Tonic eye reflexes are used to assess extraocular muscle function and localization of lesions in the CNS. Cranial nerves III (oculomotor), IV (trochlear), and VI (abducent) innervate the extraocular striated muscles and are examined together. Cranial nerve IV innervates the obliquus dorsalis; cranial nerve VI innervates the rectus lateralis and part of the retractor bulbi; and cranial nerve III innervates the rectus medialis and rectus ventralis, obliquus ventralis, and levator palpebrae superioris. Pupillary dilation is controlled by preganglionic neurons in the first three thoracic spinal cord segments, the cranial thoracic and cervical sympathetic trunks, and postganglionic neurons in the cranial thoracic and cervical trunks, and in the cranial cervical and sympathetic nerves that course through the middle ear to reach the orbit and the dilator pupillae muscle. Parasympathetic fibers in cranial nerve III innervate the sphincter pupillae muscle. The integrity of cranial nerve III may be evaluated by examining (1) the size and symmetry of the pupils; (2) the reaction of the pupil to light; (3) the presence or absence of ptosis (drooping of the upper eyelid) because of paralysis of the levator palpebrae superioris muscle; and (4) the medial deviation of the eye, which occurs in oculomotor nerve palsy (different from humans). In oculomotor nerve palsy with a normal pupillary response, if all the extraocular muscles innervated by cranial nerve III are affected, an intracranial lesion should be suspected. If individual extraocular muscles are involved, a peripheral nerve lesion may exist. If an oculomotor nerve palsy exists in association with a dilated pupil, an intraorbital or intracranial lesion should be suspected.

Paralysis of the trochlear nerve produces a transient strabismus and results in a slight upward deviation of the eye (rarely seen). The affected animal may compensate for this by developing a head tilt. Paralysis of the abducent nerve results in a medial deviation of the affected eye with inability to gaze laterally.

It is important to assess tonic neck and eye reflexes when evaluating the extraocular muscles. When the nose is elevated, the forelimbs extend and the hindlimbs flex. As the nose is elevated, the eye should remain focused within the center of the palpebral fissure. Deviating the head to the left (or right) results in increased extensor tonus on the right (or left) side of the neck. Nystagmus is an abnormal, involuntary rapid movement of the eye that denotes a disorder (stimulation) at some level of the vestibular tract; movement may be horizontal, vertical, rotary, or mixed. In a normal animal, nystagmus can be observed during lateral deviation of the head (with the quick phase toward the side of the deviation). Normal tonic eye reflexes signify a healthy brainstem and peripheral vestibular system and motor efferent pathways to the eyes. Tonic eye reflexes are not dependent on vision.

Pupillary Light Reflexes

Cranial nerve II (optic) has its origin in the retina at the optic disk. About 66% of the optic nerve fibers in the cat and about 75% in the dog decussate (cross over) at the optic chiasm. The optic nerve has two components: one is composed of the fibers that pass to the
pupillary centers within the brainstem; the other is composed of fibers that synapse in the thalamus and project impulses to the visual cortex of the brain. The normal pupillary response requires that nerves II and III be intact. The normal direct pupillary light reflex entails centering a beam of focused light into one eye and observing pupillary constriction (miosis). Recovery should be immediate after removal of the light. The normal consensual pupillary light reflex entails directing a beam of focused light into one eye then noting pupillary constriction in the opposite eye.

**External Appearance of the Eye**

Note the color of the sclera, and look for nodules, hemorrhages, lacerations, cysts, and tumors. Normal sclera is white to blue-white. The sclera may appear blue when it is abnormally thinned and the uveal tract shows through. Look for staphylomas and for any injection of the scleral vessels and accompanying edema. Episcleritis can produce local scleral inflammation, whereas deep-seated ocular diseases such as glaucoma and uveitis produce generalized scleral vessel injection.

The cornea should be smooth, moist, free of blood vessels, and transparent. Note any ulceration or opacity of the cornea. Slight opacities are termed nebulae; dense ones are called leukomas. In puppies, the cornea tends to be hazy, which restricts ophthalmoscopic examination until the animals are 4 to 6 weeks of age. Diseases of the cornea, such as corneal inflammation (keratitis), pigmentation, degeneration, trauma, and neoplasia, frequently may alter its transparency. Test the corneal sensitivity by touching the cornea with a wisp of dry cotton.

Topically applied ophthalmic fluorescein stains are routinely used to diagnose lesions of the cornea. Any break in the epithelial barrier permits rapid penetration of fluorescein into the stroma as a deep green color outlining the lesion. It cases of deep corneal ulcers, fluorescein may actually penetrate into the anterior chamber. When the epithelial surface has regenerated, the green color no longer appears. Rose bengal dye stains cells and their nuclei. The dye selectively stains devitalized corneal and conjunctival epithelium a readily visible red. The main use of this dye has been in the identification of corneal and conjunctival lesions caused by keratitis sicca.

If an ulcer is present, note whether the borders are regular or irregular and whether the ulcer is superficial or deep. With ulcers that are progressive and deep, the prognosis is guarded. It is advisable to culture deep ulcers and to take scrapings of their borders. The scrapings should be stained with Giemsa, and the type of cells should be determined. If the ulcer appears to be deep, look for evidence of anterior synchiae, prolapsed iris, iridocyclitis, cataract, extrusion of the lens, fistula, or hemorrhage.

Note the presence of blood vessels in the cornea. The depth at which vascularization is taking place is usually directly related to the cause of the vascularization. Superficial vascularization is commonly associated with superficial keratitis, superficial ulcers, or pannus. Deep vascularization usually indicates a deep corneal stromal lesion, uveitis, or glaucoma.

Look for deposits on the posterior surface of the cornea (keratic precipitates). These precipitates vary in size and shape, but they are usually indicative of infectious disease (e.g., feline infectious peritonitis virus) (Figure 2-15).

**Assessment of Vision**

Objective signs and reflexes must be used to assess vision in dogs and cats. A vision test often used to assess vision is the “menace reaction.” This test involves passing the hand or an object in front of the animal’s eyes and noting the presence or absence of a blink reflex. However, movement of air across the cornea may prompt a blink reflex in a blind animal. Preferably, a cotton ball can be tossed repeatedly in front of an animal to assess its ability to visually track movement. In some cases, it may be important to assess vision in each eye independently. A temporary blindfold can be made with adhesive or masking tape sufficient to cover one eye. Tracking is assessed using a cotton ball. The test is repeated with the opposite eye covered. An obstacle course can also be valuable in assessing visual function. Styrofoam cylinders mounted on a platform can be used to create the course. The light
intensity in the examining room can be varied, and alternate patching of the eyes can be helpful for detecting lesions.

**Note:** Blind dogs and cats do memorize obstacles within their environment and at home, commonly giving the owner the impression that vision is normal.

**Examination of the Orbit**

Observe the orbit for size. Look for swelling, depression, fistulas, or laceration of the orbital margin. If the orbit is enlarged, note whether the swelling is hard or soft, painful or nonpainful. Retrobulbar abscesses produce exophthalmos (protrusion of the globe) accompanied by pain, immobility of the eye, chemosis (swelling of the conjunctiva), edema of the eyelids, and pain on opening of the mouth. Orbital tumors may not be painful. Orbital retrobulbar hemorrhage or orbital fracture may occur after severe head trauma from automobile accidents. Enophthalmos, abnormal retraction of the eye, may result from shrinkage of orbital contents (as in phthisis bulbi after ocular injury), from paralysis of the sympathetic nerve in Horner syndrome, or from loss of retrobulbar fat in emaciation and dehydration.

**Examination of the Eyelids**

Note any inflammation along the margins of the eyelids and any inability to close the lids (lagophthalmos). The eyelids should touch the globe, thus preventing accumulation of tears and debris. The cilia or eyelashes on the dog’s upper eyelids are arranged in three irregular rows. The lower eyelids of dogs and both eyelids of cats are devoid of cilia (eyelashes). When examining the lids for the presence of entropion (inversion of the eyelid margin) or ectropion (eversion of the eyelid margin), do not manipulate the head because this may distort the normal lid-globe relationship. The lids of dogs and cats have a very poorly developed tarsal plate, which makes manipulation relatively easy. Observe the edges of the lids for signs of entropion, ectropion, trichiasis (aberrant eyelashes directed at the cornea), or distichiasis (a double row of eyelashes, some of which are directed at the cornea). Observe the eyelids for symblepharon (adhesion between the conjunctiva of the eyelid and the globe) or for swelling, edema, redness, or localized inflammation, which may indicate an internal or external hordeolum (sty, or inflammation of the sebaceous glands of the lid margin).
glands of the eyelid). Examine the lid margins for indication of any growths. The most common benign, epithelial growth observed on the lids of older dogs is the papilloma. The most common benign, adnexal-derived growth observed in older dogs is the sebaceous gland adenoma.

**Examination of the Conjunctiva**

Note whether the conjunctiva is pale, injected (hyperemic), pigmented, hemorrhagic, or jaundiced. The inferior or ventral conjunctiva normally is more hyperemic than the upper conjunctiva. Pigmentation is occasionally present in normal dogs and cats, especially on the superior bulbar conjunctiva. Usually a few follicles are visible on the conjunctival surface, especially that of the third eyelid in normal animals. Note whether the conjunctiva is relatively smooth and dry, excessively moist, or abnormally congested. Note any lacerations or erosions of the conjunctiva. Lacerations or erosions may be demonstrated using fluorescein. After initial inspection of the conjunctiva, additional tests may be required, such as the Schirmer tear test, culture, cytologic examination, or the use of stains.

Conjunctivitis (also called “pink eye”) is a common, yet complex, presenting sign that can involve one or both eyes. It is critical, when assessing a dog or cat with a “red eye,” to determine whether the underlying problem is caused by congestion of superficial vessels of the eye or a problem deeper within the eye. The differential diagnoses for conjunctivitis are listed in Table 2-10.

<table>
<thead>
<tr>
<th>Table 2-10 Differential Diagnosis Distinguishing among Conjunctivitis, Iritis, and Glaucoma</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Onset</strong></td>
</tr>
<tr>
<td>Pain</td>
</tr>
<tr>
<td>None to mild irritation</td>
</tr>
<tr>
<td>Discharge</td>
</tr>
<tr>
<td>Vision</td>
</tr>
<tr>
<td>Conjunctiva</td>
</tr>
<tr>
<td>Cornea</td>
</tr>
<tr>
<td>Iris</td>
</tr>
<tr>
<td>Pupil</td>
</tr>
<tr>
<td>Anterior chamber</td>
</tr>
<tr>
<td>Tenderness</td>
</tr>
<tr>
<td>Intraocular pressure</td>
</tr>
<tr>
<td>Constitutional signs</td>
</tr>
</tbody>
</table>
Nictitating Membrane ("Third Eyelid")
The palpebral (outer) and bulbar (inner) surfaces of the nictitating membrane should be inspected. The anterior surface of the membrane is normally smooth, and the leading edge is frequently pigmented. The bulbar surface can be examined by placing two or three drops of topical anesthetic (proparacaine hydrochloride) onto the eye. With a cotton-tipped applicator or small, atraumatic thumb forceps, the third eyelid can be everted and the area of the glans nictitans can be examined. The bulbar surface normally contains a few small follicles. The following abnormalities are frequently associated with the third eyelid: laceration, eversion of the cartilage, protrusion, inflammation and hyper trophy of the glans nictitans (also called "cherry eye"), foreign bodies, and neoplasia.

Examination of the Lacrimal System
Excessive tearing (epiphora) and decrease of tear secretion (sicca) are important disorders that can be easily assessed by measuring tear production with Schirmer Tear Test strips. Basic tear secretion comes mainly from the tarsal and conjunctival glands and the accessory tarsal glands. Reflex tear production comes from the main lacrimal gland and accessory lacrimal glands. The Schirmer Tear Test is performed by placing a single tear strip over the lower eyelid and holding in place 1 minute. In normal dogs, wetting of Schirmer test papers ranges from 10 to 25 mm in 1 minute. Both eyes should be tested.

Note any swelling, redness, or pain in the area of the lacrimal puncta and the lacrimal sac. Excessive tearing may be real, apparent, or physiologic. When excessive tearing exists, it must be determined whether the tearing is real (i.e., caused by increased lacrimal secretion from chronic ocular irritation, as in distichiasis or trichiasis), is apparent (i.e., caused by partial or complete obstruction of the excretory duct system), or is physiologic (i.e., caused by transient stimulation, such as corneal drying resulting from the dog holding its head out the window of the car during the trip to the hospital).

Fluorescein dye can be used to assess patency of the nasolacrimal duct. To perform this examination, place a drop of fluorescein dye from a sterile fluorescein strip into the eye and add one or two drops of a sterile eyewash. After 2 to 5 minutes, examine the external nares with the aid of a cobalt blue filter or Wood light for the presence or absence of fluorescence. If dye is present, the lacrimal excretory system is patent and functioning. If epiphora exists but the primary dye test indicates that the lacrimal excretory system is patent, hypersecretion of tear fluid may be implicated as the cause of the epiphora.

Irrigation of the nasolacrimal system is indicated if the primary dye test is negative. In the dog, the nasolacrimal puncta are located 1 to 3 mm from the medial canthus on the mucocutaneous border of the upper and lower lids. In the dog, a 20- to 22-gauge (in the cat, a 23-gauge) nasolacrimal cannula should be used, often with the patient under topical anesthesia. A 3-mL syringe is filled with 1 to 2 mL of sterile saline. The lacrimal cannula is attached and passed into the lacrimal puncta of the upper lid; the technique is repeated on the lower lid (see Section 4 for a description of how to flush the nasolacrimal ducts).

Several points should be made about evaluating the nasolacrimal system. Brachycephalic breeds of dogs and cats may occasionally have a negative primary dye test, although no blockage in the nasolacrimal system exists. In flushing the nasolacrimal system of some animals, fluid may not appear at the nose; however, the animal may gag and exhibit swallowing movements, indicating that the fluid has entered the mouth and the system is patent.

Examination of the Anterior Chamber
Examine the anterior chamber, and observe its depth; note changes in the transparency of the ocular media, such as hypopyon, hyphema, fibrin, or foreign bodies. Look for anterior synechiae, and make sure the lens is in the normal position. The anterior drainage angle cannot be visualized readily in the dog without the use of a gonioscopic contact lens (discussed later). Large tumors and some anterior synechiae can be visualized with a loupe and a focal light source.
**NORMAL TEAR SECRETION**

- **Dogs:** 10 to 25 mm in 1 minute
- **Cats:** >10 mm in 1 minute

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**EXAMINATION OF THE IRIS**

The color of the iris in each eye may vary. Observe the shape and size of the iris. An iris that is thickened and muddy in color indicates an infiltration of the uveal tract. Look for evidence of atrophy, tears, synechiae, persistent pupillary membranes, iridodonesis, iridodialysis, nodules, tumors, cysts, or colobomas. Examine the pupillary border of the iris for signs of atrophy or posterior synechiae to the anterior lens capsule. Complete posterior synechia results in iris bombé and secondary glaucoma.

Examine the pupil of each eye by diffuse and focal illumination. Note the size, shape, and symmetry of the pupils and perform both direct and consensual pupillary light reflex. Note any inequalities between the two pupils. Note whether the pupil of one eye is equal in size to the pupil of the other and whether the size remains equal with changes in the degree of illumination. Inequality of pupil size (anisocoria) may be caused by physiologic or pathologic factors.

**Note:** Sympathetic stimulation dilates the pupil. Parasympathetic stimulation constricts the pupil.

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**EXAMINATION OF THE LENS**

The pupil must be dilated to properly examine the lens. The lens may be examined with a focal source of illumination, an ophthalmoscope or a slit lamp. Examine the lens for the presence of pigment, adhesions, and opacities (cataract), the position of the lens (subluxation or luxation), or absence of the lens (aphakia). Normal refractive changes in the lens occur with aging and can be observed in dogs older than 7 years and in cats older than 8 years of age. This condition is called nuclear sclerosis and appears as a cloudy, white, or light-blue pupil often interpreted by owners as cataracts. Animals with nuclear sclerosis have functional vision. True opacities of the lens, however, called cataracts, may impair vision and if complete can cause blindness.

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**EXAMINATION OF THE RETINA**

The fundus is the portion of the inner eye that includes the optic disk or papilla, retinal vessels, tapetum lucidum, and tapetum nigrum. Complete visualization of the fundus requires that the iris be dilated (using a 1% tropicamide topical ophthalmic solution). Dilation requires 15 to 20 minutes after instillation of the drug. Examine each fundus in a dark room. To examine the right fundus, hold the ophthalmoscope in the right hand and view the fundus with the right eye. Starting with the ophthalmoscope at the 0-diopter setting, hold the ophthalmoscope about 20 inches from the patient’s eye. Observe the pupil and the tapetal reflex. Bring the ophthalmoscope to within 1 inch of the patient’s eye, and place the setting on 1 to 3 diopters (red numbers 1 to 3 on the rotating scale) to view the optic disk and retina. If the disk is not seen immediately, follow the retinal vessels back to the disk. Inserting more positive diopters (black numbers) into the ophthalmoscope focuses the instrument on more anterior structures within the eye.

**Additional Reading**


OTIC (EAR) EXAMINATION

Among the most important aspects of the ear examination is careful observation of the patient at rest. Physical evidence of a painful ear (e.g., loss of hair around the ear, scratching or rubbing, frequent head shaking, or head tilt) may facilitate localizing which ear, and which part of the ear, is affected. Externally compare one ear with the other. Observe the skin for signs of inflammation (swelling, redness, or desquamation of the epithelium). Movement and handling of the normal pinna should not produce pain. Look for discharges or blood emanating from the external ear canal.

An otoscope is required to examine the auditory canal. Use a clean or sterile otoscope speculum. Avoid reusing an otoscope speculum unless it has been thoroughly cleaned and dried. When feasible, always examine the uninfected or normal ear first. To examine the ear, hold the otoscope with one hand and the pinna between the thumb and first two fingers of the other hand (Figure 2-16). Gently insert the speculum without using force, while observing the external ear canal through the speculum. Slowly and carefully draw the ear laterally and turn the tip of the instrument medially to straighten the external canal. Otoscopes are provided with specula of varying diameters.

Note: The largest diameter speculum can limit the insertion depth and therefore may not always permit the visualization, especially of the tympanic membrane.

The tympanic membrane (eardrum) is a thin gray membrane through which a white curved bone (the malleus) and blood vessels can be observed along the dorsal margin (Figure 2-17). Every effort should be made to visualize the eardrum. However, considering that a dog’s external ear canal is far more tortuous and much longer than a human external ear canal, deep examination of the ear canal in either a dog or cat is an uncomfortable procedure and may not be practical without sedation or general anesthesia. The tympanic membrane consists of a small upper portion, the pars flaccida, and a large lower part, the pars tensa. The membrane separates the horizontal portion of the external auditory canal from the middle ear. The posterior portion of the pars tensa is the part that is usually visualized to the greatest extent with the otoscope. The tense part of the tympanic membrane appears darker because the tympanic bulla of the middle ear can be seen through the eardrum.

Figure 2-16: Position of the otoscope during examination of the external ear canal. Once the speculum is properly positioned in the ear canal, the pinna must gently be pulled downward and away from the head to facilitate visualization of the entire ear canal and tympanic membrane.

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The eardrum can usually be seen in normal dogs younger than 1 year of age. It may be difficult to visualize the eardrum in older dogs, because the meatus is narrowed, the tense part of the eardrum is obscured by the flaccid part, the lining of the meatus obscures the eardrum, or the eardrum is ruptured, a common occurrence in dogs with chronic otitis externa.

Any abnormal changes in the tympanic membrane, such as swelling, redness, loss of translucency, or absence of the membrane, should be recorded. Any concern that the tympanic membrane may have been penetrated or that a thorough examination of the external ear canal is indicated justifies the use of general anesthesia.

Cleaning the External Ear Canal

Otitis externa is commonly associated with the accumulation of cerumen, exudate, bacteria, and tissue debris in the lumen and skin of the external ear canal. In many breeds, but especially Poodles, Bedlington Terriers, and Kerry Blue Terriers, the external ear canal contains hair that may prevent visualization through an otoscope. If the canal is filled with debris, the hair must be removed first. This can usually be accomplished by simple epilation (grab the hair and quickly pull). In dogs and cats with minor infections with only moderate accumulation of debris in the ear, it is recommended that a medical approach be used to treat the ear. Use of cotton-tipped applicators in an attempt to remove debris deep in the ear canal is not recommended. Instilling an oil-based product into the external ear canal three or four times daily and massaging will loosen debris and treat the ear infection. Ceruminolytic agents can be irritating and are generally not used. Cotton-tipped applicators may be used to remove debris from the outer part of the external ear canal.

Deep cleaning of the ear canal should be done in the anesthetized patient using magnification (i.e., through the otoscope). For all but very simple external cleaning, it is best to use gentle irrigation with warm-water solutions to clean the canal thoroughly and enable a complete otoscopic examination. Cotton-tipped applicators are not recommended as they will pack debris deep into the ear canal and in turn may cause rupture of the tympanic membrane.

Bacterial culture and sensitivity is indicated in patients with chronic otitis, inflamed tissues, or discharge in the external canal. Before the external canal can be examined visually, debris and discharges must be removed. For clearing of the ear before examination, a nontoxic cleaning agent should be instilled into the external ear canal. Numerous preparations are commercially available. Most solutions contain dilutions (2%) of acetic or boric...
acid, propylene glycol, and dioctyl sodium sulfosuccinate (DSS). Tris-EDTA solution is an
alkalinization-inducing agent that can be instilled into the external ear canal in 5-mL ali-
quots to soften and facilitate removal of debris. In the anesthetized patient, the combina-
tion of a pulsating water jet (warm water) and an ear loop is recommended for cleaning a
chronically infected ear (see Section 4). The irrigation stream is kept parallel to the external
ear canal and is applied with a rotating motion. The excess water and debris can be caught
in a sink or basin. The canal can then be reinspected and carefully dried with cotton or by
using an aspirator. This technique is contraindicated in patients known to have a ruptured
tympanic membrane.

**Additional Reading**
Logas D: Ear-flushing techniques. In Bonagura JD, Tweedt DC, editors: *Current Veterinary
Radlinsky MG, Mason DE: Diseases of the ear. In Ettinger SJ, Feldman EC, editors: *Textbook of
Veterinary Internal Medicine*, ed 7, St Louis, 2010, Elsevier.

**LYMPH NODES AND THYROID EXAMINATION**

**Clinical History**
Only occasionally does an owner present a dog or cat for evaluation of one or more enlarged
lymph nodes. Even less commonly is the dog or cat presented specifically for an enlarged
thyroid gland. The clinician should therefore be particularly compelled to examine all
peripheral lymph nodes and the entire ventral neck for evidence of abnormal enlargement
in lymph nodes or thyroid glands.

**Examination**
Any routine physical examination, at any age, includes assessment of the size, consistency
(degree of firmness), symmetry, and location of all peripheral lymph nodes and the thy-
roid glands. It is appropriate to include examination of the lymph nodes and thyroid
glands with examination of the skin and hair coat. In the normal patient, this examina-
tion will generally be characterized by the ability to palpate small, symmetric subman-
dibular lymph nodes and popliteal lymph nodes. Three additional pairs of peripheral
lymph nodes, superficial cervical (also called “prescapular”), axillary, and inguinal lymph
nodes, are sufficiently small (or anatomically out of reach, in the case of axillary lymph
nodes) that palpation is typically not possible in the normal patient. Considerable varia-
tion in lymph node size (and number) and texture is expected among dogs and cats of
similar age, weight, and breed. The examination should focus on whether or not there is
significant asymmetry or enlargement, lymphadenomegaly, of any individual or matched
pairs of nodes. When all, some, or even one lymph node is significantly large (by subjec-
tive examination), fine-needle aspiration and cytology are indicated. There is no known
clinical significance attached to lymph nodes that are particularly small, that cannot be
palpated, or that may be absent.

Examination of the right and left thyroid glands is equally important in both dogs and
cats. The preferred examination technique actually begins at the thoracic inlet, on either
side of the trachea, rather than the larynx. Enlarged, or hypertrophied, thyroid glands
(either individually or together) may migrate ventrally from their normal location adjacent
to the larynx. In some cases one or both enlarged thyroid glands may actually migrate into
the cranial thorax and, therefore may not be palpable. From the thoracic inlet, the examina-
tion proceeds by carefully palpating the neck on either side of the trachea, moving from the
thoracic inlet to the larynx.

The normal thyroid gland is difficult, or impossible, to delineate on physical examina-
tion. Any asymmetric or symmetric enlargement should be noted. Also, the ability to pa-
lpate one or both thyroid glands in a location other than on either side of the larynx must be
considered abnormal and is likely to represent a significant pathologic condition.
DIFFERENTIAL DIAGNOSES

The most serious consideration in any dog or cat with enlargement of one or more lymph nodes is lymphosarcoma, regardless of the patient’s age, breed, or gender. However, there are also several nonmalignant causes of lymphadenomegaly that may mimic lymphosarcoma. Examples include generalized skin disease, systemic infection, and recent vaccination. Fine-needle aspiration (see Sections 4 and 5 for additional information on collection and interpretation of lymph node aspiration cytology samples) is generally indicated in any patient with one or more enlarged, asymmetric lymph nodes. Lymph node biopsy (incisional) is strongly recommended in any patient with a lymph node that demonstrates cytologic evidence of neoplasia. Rarely, surgical removal of an entire lymph node (excisional biopsy) is indicated. FeLV and feline immunodeficiency virus (FIV) testing is essential in any cat with lymphadenomegaly.

Thyroid gland hyperplasia or enlargement is much more frequently reported in adult and older adult cats (feline hyperthyroidism) than in dogs. Most reported cases in cats are diagnosed as benign hyperplasia, although about 15% of affected cats are reported to have thyroid adenocarcinoma. Fine-needle aspiration and incisional biopsy in cats with thyroid gland enlargement are rarely performed. Instead, serum is submitted for thyroid hormone (T4) levels in an attempt to establish a diagnosis of feline hyperthyroidism (see Section 5).

In dogs, thyroid tumors account for less than 4% of all tumors and approximately 10% to 15% of all head and neck tumors. However, most thyroid masses reported in dogs are associated with carcinoma. Therefore palpation of an enlarged thyroid gland in a dog justifies incisional biopsy to confirm the underlying cause. Hyperthyroidism associated with benign hyperplasia of one or both thyroid glands is reported in dogs but is considerably less common than that reported in cats. Canine hypothyroidism, the most common thyroid disorder of dogs, is not associated with palpable changes in the size, consistency, or symmetry of the thyroid glands.

Additional Reading


MUSCULOSKELETAL (ORTHOPEDIC) EXAMINATION

Examination of the musculoskeletal system is indicated in any patient with lameness; difficulty walking, running, climbing, or jumping; and what the owner perceives is pain. The examination involves methods similar to those used in any other organ system by requiring the clinician to obtain a history, perform a physical examination, and order ancillary tests. Consideration of the patient’s body size, breed, age, lameness severity, onset, clinical course, and sometimes sex often provides important insights into the examination (Table 2-11). Certain body sizes and specific breeds are more at risk for particular orthopedic conditions. For example, large, rapidly growing dogs seem to be predisposed to conditions such as osteochondrosis dissecans of the shoulder, elbow, stifle, and tarsal joints and disorders of osteochondrosis in general. Hip dysplasias, fragmented coronoid processes, an ununited anconeal process, and bone tumors are additional examples of syndromes seen in larger dogs, whereas small, miniature, and toy breeds are predisposed to conditions such as Legg-Calvé-Perthes disease and medial patella luxation.

Age can determine what conditions are considered likely for diagnosis on the examination. Immature dogs will have a certain differential diagnosis, whereas mature dogs, older than 1 year, will have others. For example, a grade II forelimb lameness of insidious onset and progressive course may indicate osteochondritis dissecans of the shoulder, whereas the clinician would not initially consider this condition in a mature dog of the same breed with the same history.
<table>
<thead>
<tr>
<th>Condition</th>
<th>Small</th>
<th>Medium</th>
<th>Large</th>
<th>Giant</th>
<th>Cat</th>
<th>Breed</th>
<th>Sex</th>
<th>Lameness Grade</th>
<th>Age at Onset</th>
<th>Onset</th>
<th>Course</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip dysplasia</td>
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<td>2+</td>
<td>3+</td>
<td>3+</td>
<td>+</td>
<td>Several</td>
<td>II</td>
<td>Juvenile or Adult</td>
<td>Slow</td>
<td>Wax and wane, progressive, frequently bilateral</td>
<td></td>
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<tr>
<td>Cruciate syndrome</td>
<td>2+</td>
<td>2+</td>
<td>3+</td>
<td>2+</td>
<td>+</td>
<td>Rottweiler, Labrador, Newfoundland, Staffordshire Terrier</td>
<td>Mc</td>
<td>Any</td>
<td>Adult</td>
<td>Any</td>
<td>Wax and wane, progressive, frequently bilateral</td>
</tr>
<tr>
<td>Medial patella luxation</td>
<td>3+</td>
<td>2+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Toy breed</td>
<td>Fe</td>
<td>I, II, III</td>
<td>Juvenile or Adult</td>
<td>Slow</td>
<td>Intermittent, progressive, frequently bilateral</td>
</tr>
<tr>
<td>Lateral patella luxation</td>
<td>+</td>
<td>+</td>
<td>2+</td>
<td>3+</td>
<td>+</td>
<td>Flat-Coated Retriever, Great Dane, Saint Bernard, Irish Wolfhound</td>
<td>I, II, III</td>
<td>Juvenile or Adult</td>
<td>Slow</td>
<td>Intermittent, progressive, frequently bilateral</td>
<td></td>
</tr>
<tr>
<td>Bicipital tenosynovitis</td>
<td>+</td>
<td>+</td>
<td>2+</td>
<td>+</td>
<td></td>
<td></td>
<td>I, II</td>
<td>Adult</td>
<td>Slow</td>
<td>Intermittent, progressive, sometimes bilateral</td>
<td></td>
</tr>
</tbody>
</table>

(Continued)
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<tr>
<th>Condition</th>
<th>Small</th>
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<th>Large</th>
<th>Giant</th>
<th>Cat</th>
<th>Breed</th>
<th>Sex</th>
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<th>Age at Onset</th>
<th>Onset</th>
<th>Course</th>
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</thead>
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<tr>
<td>Mineralization of supraspinatus</td>
<td>+</td>
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<td>2+</td>
<td></td>
<td></td>
<td>Rottweiler, Labrador</td>
<td>I, II</td>
<td>Adult</td>
<td>Slow</td>
<td></td>
<td>Intermittent, progressive, sometimes bilateral</td>
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<td>tendon</td>
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<tr>
<td>Neoplasia</td>
<td>+</td>
<td>+</td>
<td>3+</td>
<td>3+</td>
<td>+</td>
<td></td>
<td>II, III</td>
<td>Adult</td>
<td>Slow</td>
<td></td>
<td>Progressive</td>
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<td>Panosteitis</td>
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<td>3+</td>
<td>+</td>
<td></td>
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<td>II</td>
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<td>Rapid</td>
<td>Variable, self-limiting, multiple limbs</td>
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<td>2+</td>
<td>3+</td>
<td>3+</td>
<td></td>
<td>Rottweiler, Labrador, German</td>
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<td>II</td>
<td>Juvenile</td>
<td>Slow</td>
<td>Wax and wane, progressive, frequently bilateral</td>
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<tr>
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<td></td>
<td></td>
<td></td>
<td>Shepherd Dog, Great Dane</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Legg-Calvé Perthes disease</td>
<td>3+</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Terrier, toy breed</td>
<td>II</td>
<td>Juvenile</td>
<td>Slow</td>
<td></td>
<td>Progressive, sometimes bilateral</td>
</tr>
<tr>
<td>Condition</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>Breed(s)</td>
<td>Sex</td>
<td>Age</td>
<td>Type</td>
<td>Description</td>
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</tr>
<tr>
<td>Fragmented coronoid process</td>
<td>+</td>
<td>3+</td>
<td>2+</td>
<td>Rottweiler, Labrador, Golden Retriever, German Shepherd Dog, Newfoundland, Chow Chow, Bernese Mountain Dog</td>
<td>M</td>
<td>I, II</td>
<td>Juvenile or Adult</td>
<td>Slow wax and wane, progressive</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ununited anconeal process</td>
<td>2+</td>
<td>3+</td>
<td>+</td>
<td>German Shepherd Dog, Bassett Hound, English Bulldog</td>
<td>M</td>
<td>II, III</td>
<td>Juvenile</td>
<td>Slow wax and wane, progressive</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertrophic osteodystrophy</td>
<td>+</td>
<td>3+</td>
<td>3+</td>
<td></td>
<td>III</td>
<td>Juvenile</td>
<td>Rapid</td>
<td>Variable, self-limiting, multiple limbs, painful, anorectic, febrile</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


3+, Frequent; 2+, sometimes; +, seldom; Fe, female; FeS, female spayed; M, male; Mc, male castrate.
Examining

Sometimes the most difficult aspect of the orthopedic examination is localizing the problem. Lameness, for example, is among the most common reasons a dog is presented for examination. However, it may be as difficult to localize the affected limb as it is to define a muscular or orthopedic cause.

Often it must be determined whether the lameness is caused by an orthopedic or a neurologic problem. A cursory examination of the spinal column and assessment of the neurologic status of the affected limb should precede orthopedic examination of the extremity. However, one must be careful when performing the neurologic evaluation. Several common orthopedic problems, such as bilateral hip or stifle abnormalities, can appear as neurologic conditions. Animals with these problems can be reluctant to bear full weight on either limb. However, the clinician can be misled by a cursory assessment of the neurologic status when evaluating proprioceptive deficits.

The presence of a “head bob” suggests lameness. The head and neck move upward as the problematic forelimb touches the floor and downward when the affected rear limb touches down. This action helps to reduce the load carried by the limb. The stride is shortened on the affected side; the animal will offload the abnormal limb more quickly than the normal one. The animal spends less time on the abnormal limb. Audible clicks are sometimes heard in young dogs with hip dysplasia or in dogs that have a meniscus abnormality secondary to rupture of the cranial cruciate ligament.

Palpation and Manipulation

It is best to first examine the affected limb without sedation, if possible, to determine the source of discomfort or instability. The limb is palpated and manipulated from the toes proximally. The patient is turned and the procedure repeated on the other side, as the contralateral limb can often serve as a normal control when bilateral conditions are not present. An equivocal asymmetric finding requires repeating the examination as many times as necessary to confirm or rule out its presence. Last, the animal is walked again, because a subtle lameness is often exacerbated by the manipulation.

- Localized signs of inflammation: pain, swelling, heat, redness, or altered function.
- Muscle atrophy, muscle tremors, muscle atrophy (significant) in the limb or region of the pain.
- Laxity, effusion, crepitation, localized heat, altered range of motion, or decreased joint stability.
- Limited range of motion in joint(s) when compared with the same joints on the opposite limb.
- Shift of body weight to unaffected limb.
- Digits of the normal limb spread farther apart than digits of the affected limb.
- Arched back as weight is shifted to forelimbs or to hindlimbs.
- Hindlimb stance is wide-based when weight is shifted to the hindlimbs.
- Nails of the affected limb are longer than those of the unaffected limbs. (Note: Nails that are unusually short or show abrasions on the dorsum of the nail may represent proprioceptive deficits rather than a musculoskeletal disorder.)
- Self-trauma (licking) of the skin over the affected bone or joint.
- Obvious conformational differences in specific breeds with a known and characteristic conformation or posture.

Cardinal Clinical Signs in the Patient with Orthopedic Disease

- Focal or regional inflammation (pain, swelling, heat, redness, and loss of function)
- Muscle atrophy
- Muscle tremors
- Muscle atrophy localized to the muscle group primarily responsible for moving the painful limb
- Laxity, effusion, crepitation
- Localized temperature increase

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Often the goal of palpation is only to localize the specific site that is causing the animal discomfort or pain. Putting together the location of pain with the other known information will frequently lead to a diagnosis. For example, pain on palpation of the elbow joint in an immature German Shepherd Dog should lead the clinician to think of an ununited anconeal process rather than fragmentation of the medial coronoid process of the ulna. Fragmentation of the medial coronoid process is more frequently seen in Retrievers, Rottweilers, Basset Hounds, and Bernese Mountain Dogs than in German Shepherd Dogs.

**Anterior Cruciate Rupture**

Rupture of the anterior cruciate in dogs can have a varied clinical presentation. Onset can be acute or chronically insidious. Any lameness grade is possible; however, grade II progressing to grade III lameness in middle-aged, neutered, obese dogs is common. Palpation for the cranial drawer test can reveal obvious laxity, but subtle laxity is common with early or late disease. Early, the ligament is degenerating but mostly intact, whereas late, the ligament is severely degenerated and torn, but there is much fibrosis of the joint preventing gross laxity. Frequent examination of the stifle joint for cruciate syndrome requires either sedation or general anesthesia to appreciate the subtleties. Palpation signs for cruciate ligament disease are a subtle or grossly positive cranial drawer test finding, increased internal rotation of the tibia on the femur, and medial joint thickening. When palpating for the cranial drawer, position the stifle in different flexion and extension angles while performing the maneuver. The normal joint will give an abrupt endpoint “thud” to the maneuver, as the ligament normally stretches. Minimal cranial translation of the tibia on the femur may indicate early degeneration and partial tearing or advanced degeneration and major tearing with fibrosis.

**Hip Dysplasia**

Clinical signs of hip dysplasia include lameness, gait abnormalities, reluctance to exercise, and pelvic limb muscle atrophy. Specific maneuvers intended to demonstrate Barlow, Ortolani, and Barden signs are useful to evaluate the degree of joint laxity, both when screening young puppies and in performing diagnostics in clinically lame dogs. None of the signs is a definitive test for hip dysplasia, but the techniques should be performed as sequential maneuvers in the examination. Pelvic radiography is mandatory for a definitive diagnosis of hip dysplasia, but it should not be the first step in the examination because other diagnoses or concurrent conditions may be missed.

**Radiography**

Along with the history and physical examination, radiographs play an important role in the examination of the orthopedic patient. Two standard views, craniocaudal and lateromedial, are usually sufficient to define an overt orthopedic problem. Less frequently, oblique views or “stress views” are needed to help define the situation. Rarely, special imaging techniques such as tomography, bone scans, and arthograms are needed. A common error made in veterinary medicine is to use the radiograph as a predictor of the severity of a problem and let it dictate clinical treatment or prognosis. For example, osteochondrosis dissecans of the shoulder joint in the dog may be demonstrated on the radiograph, but surgical exploration is not warranted unless lameness develops. It is difficult and often misleading to predict the severity of degenerative joint disease from radiographs alone; noncartilaginous changes, such as osteophytes, are what can be seen on radiographs. Conversely, inflammatory joint disease, when nonerosive, can be very severe, and the clinician will note minimal or no radiographic changes.

**Other Diagnostic Tests**

Besides radiographs, other diagnostic aids are used with the orthopedic examination. Arthrocentesis with joint fluid analysis is a common diagnostic aid (Table 2-12). Other tests include arthroscopy, rheumatoid factor testing, antinuclear antibody testing, Lyme disease testing, synovial membrane biopsy, and other serologic tests and measurement of immune complexes.
<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Degenerative</th>
<th>Hemarthrosis</th>
<th>Rheumatoid</th>
<th>Lupus Erythematosus (LE)</th>
<th>Neoplastic</th>
<th>Aseptic</th>
<th>Septic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color</td>
<td>None or straw-colored</td>
<td>Pale yellow</td>
<td>Red</td>
<td>Yellow to blood-tinged</td>
<td>Yellow to blood-tinged</td>
<td>Yellow to blood-tinged</td>
<td>Yellow to blood-tinged</td>
<td>Yellow to sanguineous</td>
</tr>
<tr>
<td>Turbidity</td>
<td>Clear</td>
<td>Clear to slight</td>
<td>Blood-tinged</td>
<td>Slight to moderate</td>
<td>Slight to moderate</td>
<td>Slight to moderate</td>
<td>Slight to moderate</td>
<td>Turbid to purulent</td>
</tr>
<tr>
<td>Viscosity</td>
<td>Normal</td>
<td>Normal</td>
<td>Reduced</td>
<td>Reduced</td>
<td>Reduced</td>
<td>Reduced</td>
<td>Reduced</td>
<td>Reduced</td>
</tr>
<tr>
<td>Mucin clot</td>
<td>Good</td>
<td>Good</td>
<td>Fair</td>
<td>Poor</td>
<td>Fair</td>
<td>Good</td>
<td>Fair</td>
<td>Poor</td>
</tr>
<tr>
<td>Red blood cells</td>
<td>Rare</td>
<td>Few</td>
<td>Many</td>
<td>Few to moderate</td>
<td>Few to moderate</td>
<td>Few to moderate</td>
<td>Few to moderate</td>
<td>Moderate</td>
</tr>
<tr>
<td>White blood cells</td>
<td>0.1-2.0 × 10^3 mcL</td>
<td>Few</td>
<td>Moderate</td>
<td>Marked</td>
<td>Marked</td>
<td>Moderate</td>
<td>Moderate to marked</td>
<td>Marked</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>1%-10%</td>
<td>Few</td>
<td>Moderate</td>
<td>Many</td>
<td>Many</td>
<td>Moderate</td>
<td>Moderate</td>
<td>Many</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>50%-60%</td>
<td>Moderate</td>
<td>Few</td>
<td>Few</td>
<td>Few to moderate</td>
<td>Few</td>
<td>Few to moderate</td>
<td>Few</td>
</tr>
<tr>
<td>Macrophages</td>
<td>Rare</td>
<td>Moderate</td>
<td>Few</td>
<td>Few</td>
<td>Few to moderate</td>
<td>Moderate</td>
<td>Moderate</td>
<td>Few</td>
</tr>
<tr>
<td>Synovial cells</td>
<td>Moderate</td>
<td>Moderate to many</td>
<td>Rare</td>
<td>Few</td>
<td>Few</td>
<td>Moderate</td>
<td>Few to moderate</td>
<td>Few</td>
</tr>
<tr>
<td>Synovial glucose–blood glucose ratio</td>
<td>0.8:1.0</td>
<td>0.8:1.0</td>
<td>1.0</td>
<td>0.5:0.8</td>
<td>0.5:0.8</td>
<td>0.5:0.8</td>
<td>0.5:0.8</td>
<td>&lt;0.5</td>
</tr>
<tr>
<td>Other</td>
<td>Rare neutrophils and red blood cells unless blood contamination</td>
<td>Phagocytes</td>
<td>LE cells</td>
<td>Neoplastic cells</td>
<td>Toxic changes to cells, microorganisms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------</td>
<td>---------------------------------------------------------------</td>
<td>------------</td>
<td>----------</td>
<td>------------------</td>
<td>---------------------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Causes</td>
<td>Conformation, age, osteochondrosis</td>
<td>Trauma, bleeding disorder</td>
<td>Rheumatoid arthritis</td>
<td>Lupus</td>
<td>Synovial, periosteal bone, connective tissue</td>
<td>Trauma, local inflammation, immune-mediated Lyme disease, viral, rickettsial, mycoplasmas</td>
<td>Hematogenous, wounds</td>
<td></td>
</tr>
</tbody>
</table>

Additional Reading

NERVOUS SYSTEM EXAMINATION
The neurologic examination is performed to (1) localize a lesion (or lesions) within the peripheral or CNS, (2) assess the extent of disease or injury involving the nervous system, (3) assess the nature of the disease or injury affecting the nervous system, and, if possible, (4) establish the cause of the neurologic signs.

The challenge of the neurologic examination is to determine whether the presenting sign is, in fact, primarily neurologic in origin or is secondary (e.g., compromised vascularity, neoplasia, immune-mediated response, toxin, or infection.)

CLINICAL HISTORY
The breed, sex, and age of the animal should be noted; considered together with the chief complaint, they may help direct the line of questioning in the historical review. Certain breeds are predisposed to specific neurologic ailments, and the age of the animal may reduce the possibility of certain neurologic diseases.

The history should include a summary of all past medical and surgical illnesses and the facts surrounding the present complaint. The line of questioning will be influenced by the chief complaint. However, general questions should include current medications, possible history of trauma, vaccination history, and the health status of littermates or other animals in the household.

EXAMINATION
A comprehensive neurologic examination can be divided into five parts: mental attitude and behavior, gait, postural reactions, spinal nerve reflexes, and cranial nerve reflexes. In this context, an intact reflex requires the function of only the peripheral nerves being tested and the segments of the spinal cord or brainstem in which the afferent axon enters and the cell bodies and axons of the efferent neurons are located. A reaction depends on the same components as the reflex, plus the ascending pathways through the white matter of the spinal cord and brainstem to the cerebellum and sensorimotor cortex of the cerebrum and the descending pathways that return from the cerebrum by way of its internal capsule and the white matter of the brainstem and spinal cord (Figure 2-18).
The precise order in which the parts of a neurologic examination are performed varies with the preference of the examiner and the attitude of the patient. An initial assessment should be made of the patient’s mental attitude and behavior. If the animal is resting quietly in a cage at the time of examination, the cranial nerve examination may be done first. If the animal is excited or apprehensive, it may be more convenient to perform the cranial nerve examination after the animal has been handled during the examinations of gait, postural reactions, and reflexes.

**Mental Attitude and Behavior**

The owner is often the best judge of subtle changes in a patient’s behavior and the time of onset (acute versus gradual). The various terms that characterize alterations in this attitude and behavior include lethargy (often described as depression), unresponsiveness, stupor, anxiety, disorientation, hyperactivity, and aggression.

Cervical spinal cord disease that produces recumbency will not alter the animal’s mental attitude, except that some animals may become frantic and hyperexcitable if they are unable to get up. The same degree of tetraplegia can occur with a brainstem lesion that severely alters the animal’s responsiveness to its environment.

**Gait**

The gait should be examined where the patient is allowed to move freely, unleashed, and where the ground surface is not slippery. A carpeted room is ideal. The degree of functional deficit dictates the necessity for further examination of strength and coordination. A patient that is tetraplegic—unable to support its weight or move its limbs when the weight is borne on them—need not have further tests performed for the postural reactions. A grade 0 paraplegic patient need not be examined for postural reactions in the pelvic limbs, but the thoracic limbs should be examined carefully. Occasionally, a patient with progressive myelitis may be paraplegic because of an extensive thoracolumbar spinal cord location of the lesion; the patient will also have an asymmetric thoracic limb gait because of a less severe focus of the lesion in the cervical spinal cord. An early sign in dogs with ascending myelomalacia associated with an acute severe intervertebral disk extrusion may be a hesitant, stumbling, awkward gait in the thoracic limbs.

![Schematic diagram illustrating the four principal regions of the spinal cord and the associated spinal cord segments.](https://www.ajlobby.com)
The severity of advanced pelvic limb dysfunction is evaluated best by holding the animal suspended at the base of the tail and observing its gait.

### GRADING SCALE FOR PELVIC LIMB FUNCTION

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>Normal strength and coordination</td>
</tr>
<tr>
<td>4</td>
<td>Can stand to support; minimal paraparesis and ataxia</td>
</tr>
<tr>
<td>3</td>
<td>Can stand to support but frequently stumbles and falls; mild paraparesis and ataxia</td>
</tr>
<tr>
<td>2</td>
<td>Unable to stand to support; when assisted, moves limbs readily but stumbles and falls frequently; moderate paraparesis and ataxia</td>
</tr>
<tr>
<td>1</td>
<td>Unable to stand to support; slight movement when supported by the tail; severe paraparesis</td>
</tr>
<tr>
<td>0</td>
<td>Absence of purposeful movement; paraplegia</td>
</tr>
</tbody>
</table>

### Postural Reactions

After observation of the gait for strength and coordination, the postural reactions can be tested, especially to determine whether there are less obvious deficits in strength and coordination when the gait appears to be normal. Each of these reactions requires that all major components of the peripheral nervous system and CNS be intact. They are not of localizing value by themselves.

#### Wheelbarrowing

The thoracic limbs may be tested by supporting the animal under the abdomen, so that the pelvic limbs are off the ground surface, and forcing the animal to walk on its thoracic limbs. The normal animal walks with symmetric movements of both thoracic limbs and with the head extended in normal position. Animals with lesions of the peripheral nerves of the thoracic limbs, cervical spinal cord, or brainstem may have asymmetric movements, with stumbling or knuckling over on the dorsum of the paw of the affected limb. Hypermetria is occasionally observed. With more severe lesions in this area, there is a tendency to carry the head flexed with the nose close to and occasionally reaching the ground surface for support. Animals with neuromuscular disease affecting neck muscles will carry their neck partially flexed and have difficulty with normal extension. If no deficit is observed, extend the neck while the animal is wheelbarrowed. This sometimes reveals a mild deficit, a tendency to knuckle over on the dorsum of the paw, which was not observed previously. This may be helpful to confirm a cervical spinal cord lesion in Great Danes or Doberman Pinschers that have a cervical vertebral malformation and show mild pelvic limb paresis and ataxia but no overt thoracic limb signs.

#### Hopping—Thoracic Limb

While still supporting the pelvic limbs, hop the animal on one thoracic limb while holding the other off the ground surface so that the limb to be tested supports the entire weight of the body. Move the dog forward and to each side but especially laterally, and observe the strength and coordination of the limb. Repeat this with the other thoracic limb and compare the response. Asymmetry occurs with paresis or ataxia. Hypermetria may be seen with general proprioceptive or cerebellar deficits. This is an effective way of determining minor deficits when the gait appears to be normal, as occurs with contralateral cerebral sensorimotor cortex lesions. An animal with neuromuscular disease that can still move its limbs will usually struggle to hop, but the animal will collapse when all of its weight is borne on the limb being tested. If the weight is held up, the limb to be tested will often respond fairly well, indicating that proprioceptive function is not impaired.

#### Extensor Postural Thrust

The same responses to tests can be obtained on the pelvic limbs. The extensor postural thrust reaction is performed by holding the animal off the ground surface by supporting it caudal to the scapulae, lowering it to the ground surface, and observing the animal extend
its pelvic limbs to support its weight. Moving the animal forward and backward in this position tests the symmetry of pelvic limb function, strength, and coordination.

**Hopping—Pelvic Limb**

Continuing to support the animal by the thorax so that the thoracic limbs are not in contact with the ground surface, hold up one pelvic limb, and force the animal to hop laterally or forward on the supporting limb. Both pelvic limbs should be tested this way and the responses compared. It is important to compare the pelvic limb hopping responses with each other and not with the response of the ipsilateral thoracic limb. Normally the hopping response of the pelvic limb seems more stiff or hypertonic, with a slightly larger excursion than that of the thoracic limb.

**Hemistanding and Hemiwalking**

The animal’s ability to stand and walk with the thoracic and pelvic limbs on one side can be tested by holding the opposite thoracic and pelvic limbs off the ground surface and forcing the animal to walk forward or to the side. These reactions are referred to as the hemistanding and hemiwalking reactions. With a large dog or uncooperative patient that resists hopping, you may be able to evaluate the hopping responses by observing the responses of the limbs during hemiwalking.

An animal with a unilateral lesion of the sensorimotor cortex or internal capsule may have a normal gait but show deficits in its postural reactions on the side opposite the lesion. Attempts to hemiwalk on the contralateral side are delayed or exaggerated (hypermetric) and spastic, and stumbling may occur. With unilateral cervical spinal cord lesions, the limbs on the same side as the lesion show a deficiency in the gait and are poorly responsive to postural reaction testing, including the animal’s inability to respond in the hemiwalking reaction.

**Placing**

Other postural reactions that can be tested include placing with the thoracic limbs. The animal is supported off the ground surface and its thoracic limbs are brought to the edge of a table or similar surface so that the dorsal surface of the paws makes contact. This test should be performed on both thoracic limbs simultaneously and individually, with and without blindfolding the animal. Vision can compensate for the sense of position when the general proprioceptive system is abnormal, so tactile placing (blindfolded animal) is tested before visual placing.

**Tonic Neck Reaction**

The tonic neck reaction involves extension of the head and neck so that the nose is directed dorsally. The normal patient responds by extension of all the joints of both thoracic limbs. An animal with disease of the general proprioceptive system in the cervical spinal nerves, cervical spinal cord, or medulla fails to extend its carpus or digits or both, and these joints passively flex so that the weight is borne on the dorsal surface of the paw. The same response may occur if an animal is paretic as a result of disease of the motor neurons that innervate the thoracic limb or in the white matter of the spinal cord that influences these motor neurons.

**Proprioceptive Positioning**

Proprioceptive positioning tests the afferent proprioceptive system by determining the animal’s ability to sense or feel when the paw has been flexed so that the weight is borne on its dorsal surface. With normal proprioception, the animal immediately returns the paw to its usual position. In patients with paresis, this test may also reveal findings of deficiency. Delayed proprioception may also be a manifestation of limb or pelvic pain. Sedatives and analgesics are also likely to delay proprioceptive positioning in otherwise normal animals.

**Spinal Nerve Reflexes**

Spinal nerve evaluation includes assessment of muscle tone and size, spinal reflexes, and cutaneous sensation. Muscle tone and spinal reflexes are evaluated best when the animal is in lateral recumbency and as relaxed as possible. It is important to test muscle tone, tendon
reflexes, and the flexor reflex to noxious stimuli, in that order, to maintain the animal's cooperation.

**Muscle Tone**

Muscle tone is evaluated by passive manipulation of the limbs individually. The degree of resistance is determined to be less than normal (hypotonic), normal, or more than normal (hypertonic). The last may be referred to as spasticity. The degree of spasticity varies from a mild increased resistance, to passive manipulation, to rigid extension. Hypotonia usually occurs with LMN disease, whereas UMN disease may be characterized by hypertonia or spasticity. However, normal muscle tone without spasticity can occur in some animals with UMN disease. The functional integrity of the LMN is necessary to cause muscle cell contraction to maintain muscle tone. Functional integrity of the LMN is also necessary to maintain the normal health of the muscle cell it innervates. When denervated, these cells degenerate. The degeneration is observed clinically as neurogenic atrophy and can be detected electromyographically by the production of abnormal potentials in resting muscle. The UMN influences the activity of the LMN to produce voluntary motor activity and to maintain muscle tone for support of the body against gravity. Although the UMN includes both facilitatory and inhibitory functions on the activity of the LMN, when the UMN is diseased the result usually observed is a release of the LMN from inhibition and overactivity of the facilitatory mechanism. This release is seen as hypertonia or spasticity.

Dogs that are tetraplegic should be held in a supporting position to observe the muscle tone in the limbs and any voluntary responses. Usually, dogs with cervical spinal cord lesions rostral to the brachial plexus have rigidly extended limbs, and the entire trunk and limbs feel stiff when the dog is held up and the limbs are moved along the ground surface. The hypertonia may be severe enough to permit the animal to stand unsupported. Tetraplegic dogs with diffuse neuromuscular diseases such as polyradiculoneuritis are hypotonic or atonic and appear and feel limp when held in a supporting position. There is no reflex tension of the limb, and no support is elicited by placing the paws on the ground. Instead, the limbs buckle under the weight of the body.

**Patellar Reflexes**

The most reliable tendon reflex is the patellar reflex. It is the only tendon reflex that is present in all normal animals. However, the reflex may normally be difficult to detect in older, large-breed dogs. It is obtained by lightly tapping the patellar tendon with the animal in lateral recumbency and as relaxed as possible for proper evaluation. A pediatric neurologic hammer is the most useful instrument. The reflex can be elicited in all normal dogs and is mediated by the femoral nerve through the L4-L6 spinal cord segments. The degree of normal response varies with the breed. Large-breed dogs have a brisker reflex than the short-limbed breeds such as the Dachshund. The response should be evaluated as absent (0), hyporeflexic (1), normal (2), hyperreflexic (3), or clonic (4). This reflex should be tested with the animal lying on each side. An absent reflex or hyporeflexia occurs when there is disease of a portion of the reflex arc. Hyperreflexia or clonus is often present as UMN disease.

**Biceps and Triceps Reflexes**

In the thoracic limb the biceps and triceps reflexes can be elicited in many dogs that are relaxed and in lateral recumbency. Lightly tapping the tendon of insertion of the triceps proximal to the olecranon elicits a slight extension of the elbow. The reflex is mediated by the radial nerve through the C7 and C8 and the T1 and T2 spinal cord segments. The biceps reflex is elicited by placing a finger on the distal ends of the biceps and brachialis muscles at the level of the elbow. Tapping this finger with the hammer elicits a slight flexion of the elbow. The muscle contraction can be palpated in some instances when no movement of the joint is seen. The musculocutaneous nerve mediates this reflex through the C6-C8 spinal cord segments. The normal animal has a mild reflex response to these
stimuli. In a few normal animals, these reflexes are difficult to elicit. They are absent when there is disease of some portion of the reflex arc. They may be hyperactive in some animals with disease of the UMN.

**Flexor Reflex—Pelvic Limb**
The flexor reflexes to noxious stimuli determine the integrity of the reflex arc as well as the pathway in the CNS that is concerned with the animal’s response to noxious stimuli. The most reliable stimulus is pressure exerted on the base of the toenail with hemostats. Many normal animals do not respond to the stimulus of a pin. The pelvic limb is maintained perpendicular to the long axis of the pelvis by placing a hand on the anterior surface of the limb above the stifle when applying the noxious stimuli. The normal animal with UMN lesions will flex the limb at the stifle. The flexor reflex is mediated by the sciatic nerve through the L6 and L7 spinal cord segments and the S1 segment. A depressed or absent flexor indicates a lesion in one of these structures. Abnormality of the motor portion of the sciatic nerve distal to the pelvis causes paralysis, hypotonia, and atrophy of the flexors of the stifle, tarsus, and digits, as well as of the extensors of the hip, tarsus, and digits. There is no resistance to flexion or extension of the tarsus. In the animal walking with a sciatic nerve paralysis, the tarsus is lower on the affected side and the paw may be placed on its dorsal surface; however, the limb is able to support weight as long as the femoral nerve is intact.

Sensory branches of the peroneal nerves supply the dorsal surface of the paw. The plantar surface is supplied by tibial nerve sensory branches. The medial side of the paw is supplied by the saphenous nerve, a branch of the femoral nerve. The saphenous nerve enters the spinal cord through the L4-L6 segments. A patient may have a contused sciatic nerve from a pelvic fracture and have no function of the muscles innervated by this nerve and analgesia of the lateral, dorsal, and plantar surfaces of the paw. However, the intact saphenous nerve provides sensation to the medial surface of the paw. If this area is stimulated, the animal will flex the hip with the intact innervation of the iliopsoas muscle, but the stifle, tarsus, and digits will fail to flex. For this reason, both the medial and lateral surfaces of the paw should be tested for reflex responses as well as nociception.

**Nociception (Outward Manifestation of Pain)**
Animals show signs of pain perception by a behavioral response (e.g., crying, biting), not a flexor reflex. The impulses generated by a noxious stimulus enter the spinal cord over the peripheral nerves and dorsal roots and are relayed to tracts in the lateral funiculi of the spinal cord bilaterally. These tracts ascend the spinal cord in the lateral funiculi and continue through the medulla, pons, and mesencephalon to specific nuclei in the thalamus for relay to the somatic sensory cerebral cortex. Pain may be evidenced when the impulses reach the thalamus or cerebrum.

**Flexor Reflex—Thoracic Limb**
In the thoracic limb, the thoracodorsal, axillary, musculocutaneous, median, ulnar, and radial nerves are responsible for flexion of the shoulder, elbow, carpus, and digits when a noxious stimulus is applied to the paw. These nerves arise from the C6-T2 spinal cord segments. The specific sensory nerve stimulated depends on the location of the stimulus. The median and ulnar nerves innervate the skin of the palmar surface of the paw; the radial nerve supplies the dorsal surface. In the forearm, the radial nerve supplies the skin on the cranial and lateral surfaces. The ulnar nerve supplies the caudal surface, and the musculocutaneous nerve supplies the medial surface. Be aware of the amount of overlap of the cutaneous innervation by these nerves. The thoracic limb is maintained in a position similar to that described for the pelvic limb. After application of a noxious stimulus, the normal animal and the animal with a UMN lesion will flex the limb at the elbow. A depressed or absent flexor reflex indicates a lesion in one of the structures that mediate the flexor reflex.
Crossed Extensor Reflex
In animals with UMN disease and release of the LMN, a crossed extensor reflex may be elicited in the recumbent animal when the flexor reflex is stimulated. The crossed reflex occurs in the limb opposite the one being tested for a flexor reflex. To avoid voluntary extension of the contralateral limb as a response to a noxious stimulus, the flexor reflex first should be elicited with as mild a stimulus as is necessary and the opposite limb observed for extension. When elicited in an animal in lateral recumbency, this is an abnormal reflex, indicative of UMN disease.

Perineal Reflex
The perineal reflex is elicited by stimulating the anus with a noxious stimulus and observing contraction of the anal sphincter and flexion of the tail. The reflex is mediated by branches of the sacral and caudal nerves through the sacral and caudal segments of the spinal cord.

Cutaneous Reflex
The cutaneous reflex is the contraction of the cutaneous trunci in response to mild stimulation of the skin of the trunk. It can be elicited in normal animals from the thoracic and most of the lumbar region. The regional segmental spinal nerves contain the sensory neurons that are stimulated. The impulses are carried into the related spinal cord segments and then relayed through the white matter of the spinal cord cranially to the C6 spinal cord segment. Here synapse occurs on LMNs of the lateral thoracic nerve that innervate the cutaneous trunci. When the cutaneous response is present, it indicates that the spinal cord white matter is intact from the level tested to the C8 spinal cord segment. This reflex may require multiple stimulation to elicit, and occasionally normal animals resist this stimulation; dehydrated animals and animals with advanced generalized muscle atrophy show no reflex.

Cranial Nerve Examination (See Also Table 2-13)
Indications for performing a cranial nerve examination are based on the initial evaluation of the patient’s behavior, attention to the surroundings, ability to see and track objects, ability to hear, posture, and gait. Much of the initial assessment can simply be observed by allowing the animal to walk around inside the examination room or, with larger dogs, outside. When evidence of a deficit or abnormality is present, methodical assessment of the cranial nerves is indicated. Defined cranial nerve deficits typically manifest ipsilateral to the lesion. Deficits of cranial nerves II and IV may manifest contralateral to the lesion. Figure 2-19 illustrates the point of origin, at the level of the brain and brainstem, for each of the 12 cranial nerves; the nerve type (sensory, motor, or mixed) is also depicted. Table 2-13 summarizes common presenting signs with individual cranial nerve examination responses.

Clinical Signs Associated with Intracranial Lesions
Medulla and Pons
Lesions in the medulla and pons result in spastic tetraparesis and ataxia of all four limbs or tetraplegia, ipsilateral spastic hemiparesis and ataxia (unilateral lesions), central vestibular signs, depression and irregular respirations and heartbeat, and hypalgesia of the trunk and limbs.

Signs of cranial nerve deficit include facial hypalgesia or analgesia (sensory, V); paresthesia or paralysis of masticatory muscles (motor, V); medial strabismus (VI); facial paresis or paralysis (VII); pharyngeal paresis (IX, X); tongue paresis (XII); and loss of balance, head tilt, and abnormal nystagmus (VIII).

Cerebellum
With diffuse lesions, the signs are symmetric ataxia with preservation of voluntary motor activity, dysmetric gait (hypermetria), truncal ataxia, head tremor, muscle hypertonia, occasional abnormal nystagmus, and bilateral menace deficit. With unilateral lesions, the signs are ipsilateral. The body and the head tilt toward the side of the lesion or occasionally away from the side of the lesion, and there may be ipsilateral menace deficit. With severe rostral...
Figure 2-19: Schematic demonstrating the points of origin and the target organs (sensory [green lines] and motor [black lines]) for each of the cranial nerves. (Modified from Hoerlein BF: Canine neurology, ed 3, Philadelphia, 1978, WB Saunders.)
<table>
<thead>
<tr>
<th>Nerve</th>
<th>Sign of Dysfunction</th>
<th>Test and Responses</th>
</tr>
</thead>
<tbody>
<tr>
<td>I Olfactory</td>
<td>Anosmia</td>
<td>Observe response to smell of food or some mild volatile oils</td>
</tr>
<tr>
<td>II Optic</td>
<td>Visual deficit, bumping objects</td>
<td>No menace response—failure to close eyelids or retract head when affected eye is menaced</td>
</tr>
<tr>
<td></td>
<td>Unilateral disease</td>
<td>Light in affected eye—no pupillary response from either eye</td>
</tr>
<tr>
<td></td>
<td>Mild mydriasis in affected eye (slight anisocoria) or none</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bilateral disease</td>
<td>Light in affected eye—no pupillary constriction</td>
</tr>
<tr>
<td>III Oculomotor</td>
<td>Marked mydriasis bilaterally</td>
<td>Examine with ophthalmoscope</td>
</tr>
<tr>
<td></td>
<td>Marked mydriasis</td>
<td>Light in affected eye—only pupil of normal eye constricts</td>
</tr>
<tr>
<td></td>
<td>Severe anisocoria</td>
<td>Light in normal eye—only pupil of normal eye constricts</td>
</tr>
<tr>
<td></td>
<td>Ventrolateral strabismus</td>
<td>Incomplete adduction of affected eye on moving head side to side</td>
</tr>
<tr>
<td></td>
<td>Ptosis</td>
<td>Inability to elevate upper eyelid completely</td>
</tr>
<tr>
<td>IV Trochlear</td>
<td>Slight extorsion (tilting) of eyeball, which may be visualized in the dog only by ophthalmoscopic examination of the position of the retinal veins</td>
<td></td>
</tr>
<tr>
<td>V Trigeminal</td>
<td>Dropped jaw; unable to close mouth if bilateral disease</td>
<td>Hypalgesia can be determined by patient’s lack of response to touching nasal septum with forceps</td>
</tr>
<tr>
<td></td>
<td>No motor deficit if unilateral disease</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Atrophy of muscles of mastication</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hypalgesia or analgesia of face</td>
<td></td>
</tr>
<tr>
<td>VI Abducent</td>
<td>Medial strabismus</td>
<td>Incomplete abduction of affected eye on moving head from side to side</td>
</tr>
<tr>
<td>VII Facial</td>
<td>Paresis or paralysis of facial muscles— inability to close palpebral fissure, drooped hypotonic lip with drooling of saliva, inability to move ear, but the ears will not droop in all patients (cats and some prick-eared dogs); incomplete dilation of nares on inspiration</td>
<td></td>
</tr>
</tbody>
</table>

TABLE 2-13 Evaluation of Cranial Nerves

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lesions, there may be opisthotonus and rigidly extended forelimbs, and the pelvic limbs are extended forward by hip flexion.

**Midbrain (Mesencephalon)**

With lesions in the midbrain area, the following signs occur: opisthotonus with rigid extension of all limbs (decerebration); spastic tetraparesis and ataxia of all four limbs; spastic hemiparesis if the lesion is unilateral (usually contralateral); depression; stupor (semicoma) or coma; and hypalgesia of the head, trunk, and limbs. Signs of cranial nerve deficit are ventrolateral strabismus (III) and mydriasis and nonreactive pupil (III). There is deviation of the eye in certain positions of the head, and the head and neck are flexed laterally, with the nose directed toward the shoulder with severe midline or unilateral lesions in the tegmentum. Visual deficits may be observed in acute lesions.

**Thalamus and Hypothalamus (Diencephalon)**

Bilateral lesions of the diencephalon produce the following signs: slows postural reactions bilaterally, mild ataxia, bilateral visual deficit with dilated unresponsive pupils (optic tracts), and bilateral hypalgesia.

Unilateral lesions are indicated by contralateral deficient postural reactions, contralateral visual deficit with normal pupils, contralateral hypalgesia (most noticeable in the head), and the adversive syndrome—propulsive circling and head and eye deviation, usually toward the side of the lesion.

With lesions that are either bilateral or unilateral, the manifestations are depression, stupor (semicoma) or coma, behavioral changes, seizures, and hypothalamo-hypophyseal

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**TABLE 2-13 Evaluation of Cranial Nerves—cont’d**

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Sign of Dysfunction</th>
<th>Test and Responses</th>
</tr>
</thead>
<tbody>
<tr>
<td>VII Vestibulocochlear</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cochlear</td>
<td>Deafness (unilateral is difficult to determine)</td>
<td>Lack of response to commands or any noise</td>
</tr>
<tr>
<td>Vestibular—Unilateral disease</td>
<td>Head tilt and ataxic toward side of lesion—lean, fall, circle toward side of lesion Abnormal resting or positional nystagmus with quick phase away from side of lesion</td>
<td>Unequal response on postrotatory testing—rapid movement away from side of lesion; extend the neck; eye on affected side will not elevate completely (vestibular strabismus); hold head to side or dorsally and observe for positional nystagmus</td>
</tr>
<tr>
<td>Vestibular—Bilateral disease</td>
<td>Crouched gait, stumble to either side No abnormal nystagmus, wide excursions of head</td>
<td>Inability to generate nystagmus on moving head from side to side or spinning—no postrotatory response</td>
</tr>
<tr>
<td>IX Glossopharyngeal</td>
<td>Dysphagia, gagging on eating</td>
<td></td>
</tr>
<tr>
<td>X Vagus</td>
<td>Dysphagia, gagging on eating</td>
<td></td>
</tr>
<tr>
<td>XI Accessory</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>XII Hypoglossal</td>
<td>May deviate toward affected side on protrusion</td>
<td></td>
</tr>
</tbody>
</table>
disorders of body temperature, glucose metabolism, appetite control, autonomic nervous system, water balance, gonadal function, and thyroid and adrenal function.

Cerebrum (Telencephalon)
Lesions in the cerebrum are evidenced by changes in several ways. Changes in behavior or temperament include depression (lethargy, obtundation); stupor (semicoma); lack of recognition of owner or environment and bewilderment; loss of trained habits; and irritable, hysterical maniacal, or aggressive behavior. In propulsion, the animal often paces and circles in one direction and turns the head and eyes in one direction; this direction is usually toward a unilateral lesion, called the advantageous syndrome (“turn to”). This may indicate rostral thalamic involvement in the lesion. Seizures are partial (contralateral face or limbs or both) or generalized (grand mal, psychomotor). The gait is usually normal, but contralateral postural reactions are deficient. Bilateral lesions produce blindness. Unilateral lesions produce a contralateral visual deficit with normal pupil responses to light. Occasionally, contralateral facial hypalgesia occurs. Rarely, the hypalgesia is observed in the contralateral trunk and limbs. Acute diffuse lesions may produce bilateral miosis. Pseudobulbar paresis rarely may be observed on voluntary movement: contralateral lower facial paresis (lip and nose), pharyngeal paresis, and tongue paresis.

Clinical Signs Associated with Spinal Cord Lesions
The objective of the neurologic examination in patients with spinal disorders is to localize the injury or disease. This is accomplished by identifying which of the four major spinal cord divisions are involved:

- **Cervical region:** Spinal cord segments C1 to C5 (Box 2-1)
- **Cervical enlargement:** Spinal cord segments C6 to T2 (Box 2-2)
- **Thoracolumbar region:** Spinal cord segments T3 to L3 (Box 2-3)
- **Lumbar enlargement:** Spinal cord segments L4 to the caudal segment, including the cauda equina (Box 2-4)

Cervical Region
The cervical region constitutes the critical region of the spinal cord because complete transection or myelopathy can result in respiratory arrest and death. Injuries involving less than complete myelopathy can spare respirations but still cause ataxia or paresis in all four limbs.

**BOX 2-1** REPRESENTATIVE DISORDERS LOCALIZED TO THE CERVICAL REGION OF THE SPINAL CORD

<table>
<thead>
<tr>
<th>Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intervertebral disk disease</td>
</tr>
<tr>
<td>Diskospondylitis</td>
</tr>
<tr>
<td>Cervical trauma</td>
</tr>
<tr>
<td>Ischemic myelopathy</td>
</tr>
<tr>
<td>Neoplasia</td>
</tr>
<tr>
<td>Atlantoaxial subluxation</td>
</tr>
<tr>
<td>Steroid-responsive meningitis</td>
</tr>
</tbody>
</table>

**BOX 2-2** REPRESENTATIVE DISORDERS LOCALIZED TO THE CERVICAL ENLARGEMENT REGION OF THE SPINAL CORD

<table>
<thead>
<tr>
<th>Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intervertebral disk disease</td>
</tr>
<tr>
<td>Diskospondylitis</td>
</tr>
<tr>
<td>Congenital vertebral anomalies</td>
</tr>
<tr>
<td>Neoplasia</td>
</tr>
<tr>
<td>Spinal trauma</td>
</tr>
</tbody>
</table>
In addition, cervical spinal injuries rarely result in tetraparesis but can cause hindlimb paralysis while sparing the thoracic limbs. Injury at this level is likely to result in normal to exaggerated muscle tone and spinal reflexes including normal anal tone. However, distinguishing between cervical myelopathy and lesions in the brainstem and cerebrum can be difficult. Neck pain, for example, can occur in patients with cervical disk disease, but it may also be present in patients with brain tumors or spinal root entrapment (also called root signature). (See Box 2-1.)

Cervical Enlargement
Ataxia and paresis involving all four limbs characterize lesions at the level of the cervical enlargement (spinal cord segments C6 to T2). Postural reactions and proprioception are depressed. It is possible for some patients to manifest paresis in the thoracic limbs and paralysis in the hindlimbs. Spinal reflexes may be normal; however, if spinal reflexes are abnormal, thoracic limb reflexes are likely to be depressed whereas abnormal hindlimb reflexes are expected to be exaggerated; muscle atrophy of the forelimbs can be significant. On the other hand, anal tone remains normal. Evidence of Horner syndrome (unilateral ptosis, miosis, enophthalmia, and prolapse of the third eyelid) can be a manifestation of injury at the level of the cervical enlargement. (See Box 2-2.)

Thoracolumbar Region
Most of the spinal cord injuries and diseases that manifest in dogs and cats occur in the thoracolumbar region. The most striking neurologic feature is that of normal thoracic limb function and gait in a patient with paresis, ataxia, or paralysis of the hindlimbs. Proprioception and postural reactions are normal in the thoracic limbs but are expected to be compromised (to absent) in both hindlimbs. Spinal reflexes involving the hindlimbs are normal to exaggerated. Although voluntary control of defecation may be lost, anal tone is still present. The ability of the patient to perceive pain (significant pressure applied to the toes) will range from normal to absent (usually a poor prognostic sign). Changes in bladder function are not consistent and are determined by the location, type, and severity of the injury. Rarely, however, will patients with significant spinal injury have normal bladder function and be able to voluntarily control urination. Some patients will be incontinent.
with a flaccid, easily expressed bladder, whereas in others the bladder will be difficult to express (also called an “upper motor neuron” bladder). (See Box 2-3.)

Lumbar Enlargement
Injury or disease involving the caudal-most region of the spinal cord will cause variable clinical manifestations ranging from near normal function to paresis, ataxia, and paralysis. Although thoracic limb function is normal, the hindlimb reflexes and muscle tone can be obviously reduced; and in chronic disease, significant muscle atrophy can be apparent. Anal tone is generally reduced and accompanied by fecal incontinence. Some patients may become severely constipated. Although the bladder may fill, the patient is unable to voluntarily urinate. The bladder is easily expressed (also called a “lower motor neuron” bladder). (See Box 2-4.)

Neurogenic Urinary Incontinence
Bladder dysfunction often accompanies severe spinal cord disease. Total LMN paralysis occurs with sacral spinal cord lesions. Severe or total focal thoracolumbar spinal cord lesions produce a UMN type of paralysis. Paralysis is less common with cervical spinal cord lesions unless the lesions are severe. With both LMN and UMN paralysis, retention of urine occurs. Overflow takes place with both but is more constant with LMN disease. Overflow is less frequent in UMN disease, because greater intraluminal pressure is required to overcome the tone in the striated urethral muscle. If the integrity of the bladder wall is retained, reflex urination may follow within a variable period of time. Reflex urination is more efficient in UMN disease, using the intact peripheral nerves and sacral spinal cord segments. In LMN disease, reflex urination must be mediated within the wall of the bladder and is very inefficient.

Additional Reading

REPRODUCTIVE TRACT EXAMINATION: CANINE—MALE

Clinical History
In addition to age, breed, history of breedings and breeding problems, body condition, vaccination, and comments on specific disease problems, establish the environmental setting, feeding, management practices, fertility, and breeding data about related animals. The degree of inbreeding may be important in evaluating sexual function. Previous information related to reproduction is particularly important and should include dates and results of all matings (especially for the previous year). Include comments about libido, breeding techniques, previous fertility and therapy, number of pups born, number weaned, and any abortions or deaths.

Examination
Inspection and palpation of the genital organs is the first procedure. A thickened scrotal wall may produce testicular degeneration from increased temperature. Palpate the spermatic cord and testes for size, symmetry, and consistency (firm resilience), and note whether the testes are both located in the scrotum. Small, soft testes indicate degeneration or hypoplasia; firm masses may be the result of inflammation, fibrosis, or tumors. Testicular tumors are relatively common in dogs over 8 years old and may, depending on tumor type, produce estrogens or testosterone that can cause mammary development, can alter reproductive ability or interest, and may cause life-threatening coagulopathies. The epididymis, palpable dorsal to the testis, may be prominent or firm because of fibrosis or ascending infection. The penis, prepuce, and external urethral orifice should be examined for frenula, hypospadias, phimosis, balanoposthitis, and, in older patients, neoplasia.
The prostate is the only accessory sex gland in the dog. Examine the prostate by rectal palpation. The prostate should be smooth, bilaterally symmetric (bilobed on rectal palpation), nonpainful, and, in most dogs, usually smaller than 3 cm in diameter. Nodules, fixation, and pain are found in carcinomas; nonpainful symmetric enlargements (often so large as to pull the organ into the abdomen) are seen with cysts or benign prostatic hypertrophy. The four major causes of prostatomegaly are benign hypertrophy; prostatitis, including abscessation of the prostate; prostatic cysts; and primary or secondary prostatic tumors. The appearance of an enlarged prostate on survey radiographs, combined with positive contrast retrograde urethrography and prostatic ultrasonography findings, can be helpful in the differential diagnosis. Hyperplastic and inflammatory prostatic diseases result in more symmetric prostatomegaly than do cystic, neoplastic, or prostatic abscess processes. Signs of acute bacterial prostatitis include urethral discharge, constipation, tenesmus, stilted gait, fever, depression, abdominal pain, dysuria, and leukocytosis. Chronic bacterial prostatitis may include signs associated with recurrent urinary tract infection, and the use of radiography, prostatic fluid evaluation, cultures, and biopsy may be necessary to establish a diagnosis.

Numerous additional testing procedures can be used in evaluating the animal with prostatic disease, including ejaculation and microscopic evaluation, prostatic biopsy, and prostatic aspiration and massage. Techniques for performing these procedures are described in Section 4.

REPRODUCTIVE TRACT EXAMINATION: CANINE—FEMALE

CLINICAL HISTORY
Disorders associated with breeding, especially failure to conceive, are among the most common female genital presentations. Blood in the urine or frequent urination are the most common presentations involving the female urinary tract. In evaluating the patient, the clinical history is particularly important and should include age, breed, body condition, vaccination status, and comments on specific disease problems. Determine the environmental setting, feeding, breeding, and other management practices concerning the animal and the fertility and breeding data for related animals. The stud dog’s records should also be examined. Pedigrees should be examined to determine inbreeding or possible genetic defects. Obtain information about the age at first estrus; number and frequency of estrous cycles; breedings; pregnancies; history of false pregnancies; urogenital problems; litters whelped; and numbers of pups born and weaned, with causes of abortions or deaths, if known. Treatments and prophylactic measures, especially if they involve sex hormones, should be investigated.

EXAMINATION
Inspection and palpation of the external genitalia provide limited but valuable information. The vulva is usually small and wrinkled, with good tone and free of discharge. Obese females may be difficult to examine, particularly when a “hooded” vulva is present. Note the size and condition of the clitoris. The vulva swells during proestrus and has a serosanguineous discharge; during estrus an odorless bloody or mucoid discharge is present. Exudates at other times, especially if fetid, suggest infection (open pyometra), neoplasia, or other endocrine problems. Digital examination of the vestibule and caudal vagina and abdominal palpation of the uterus should be performed in all bitches with a clinical history of vaginal discharge. Contrast vaginography, vaginoscopy, and ultrasonographic examination of the uterus and ovaries may be indicated when disease processes have been localized to these organs.

Individual mammary glands should be palpated and inspected for the presence of infection (mastitis), hypertrophy, or tumors. Acute infections result in hot, painful, and swollen areas; usually involve only one gland; and produce a purulent secretion. Chronic mastitis involves several glands; the glands are enlarged, firm, and nodular on palpation. Mammary gland tumors constitute about half of all neoplasms in the intact bitch, and about half of these are malignant. The median age of affected bitches is 10.5 years.
and mammary gland tumors are multicentric in about 50% of bitches. Of the benign tumor forms, the most frequently recognized histologic patterns are fibroadenomas (benign mixed tumors) (45%); simple adenomas; and benign mesenchymal tumors. Of the malignant tumors in the dog, the most common histologic types are solid carcinomas, tubular adenocarcinomas, papillary adenocarcinomas, anaplastic carcinomas, and sarcomas.

In malignant tumors that metastasize, tumor cells from glands 4 and 5 drain to the inguinal lymph nodes and via the thoracic duct to the lungs. The iliac lymph nodes may also be involved with metastasis. Tumor cells from glands 1 and 2 drain to the axillary lymph nodes and to the lungs and may involve the intrathoracic lymph nodes. Gland 3 appears to drain more commonly to the axillary lymph nodes. Hematogenous spread of mammary tumors with no lymph node involvement is also possible. Many malignant mammary tumors of the bitch metastasize widely and may affect abdominal as well as thoracic organs.

Fine-needle biopsy and cytologic examination may be helpful in distinguishing benign from malignant cell types. Multiple mammary gland tumors, which are present in 50% of dogs, may have different tumor types; thus, all tissues and regional lymph nodes should be sectioned.

Mammary gland neoplasia in the bitch is almost 100% preventable if ovariectomy is performed before the first estrous cycle. The incidence of mammary gland neoplasia can be markedly reduced if ovariectomy is done before the animal is 2.5 years old or before the first four estrous periods. Pharmacologic doses of progestational compounds have been associated with the development of mixed mammary gland tumors.

Mammary tumors occur more frequently in cats than in any other domestic animal except the dog. Ninety percent of the tumors observed are malignant. The tumors are usually adenocarcinomas and are seen most commonly in cats 7 years of age or older. The tumors usually ulcerate early in their development. The cat normally has four pairs of mammary glands. The cranial two glands on each side have a common lymphatic system and drain into the axillary lymph nodes. The caudal two mammary glands also have a common lymphatic system and drain into the superficial inguinal lymph nodes.

**Bacteriologic Examination**

Bacteriologic examination of mammary secretion, vaginal smear or culture, and intrauterine culture (collected at laparotomy) is very important if infectious processes are suspected. All breeding bitches should be tested for brucellosis; a positive rapid slide test result should be confirmed by laboratory tube tests or blood cultures.

Infertility, abortion, premature births, stillbirths, or neonatal deaths can be caused by many infectious agents, such as β-hemolytic streptococci, *Escherichia coli*, *B. canis*, and *Staphylococcus*, *Proteus*, *Pseudomonas*, and *Mycoplasma* species; canine distemper; adenovirus; and herpesvirus. Fetal resorption, mummification, and abortion may be caused by any of the infectious agents just listed or by numeric chromosomal abnormality, inherited metabolic disease, maternal endocrine abnormality (thyroid insufficiency), lack of uterine space, trauma, placental hemorrhage, hormone deficiency (progesterone), exogenous estrogen, myometrial cysts, hyperplasia, and endometritis. In evaluating these problems, supplement the bacteriologic examination with maternal serology and hormone analysis, pedigree studies of the sire and dam, and, particularly, aggressive diagnostic evaluation of the dead fetus(es) (karyotype, culture, histopathology, metabolic screening).

**Radiographic Examination**

Radiographic examination of the uterus can be performed easily if the organ is enlarged (pyometra, pregnancy, tumors). However, examination is limited to detection of an abnormally enlarged or displace uterus. Radiographic contrast studies, such as injection of a radiopaque dye through the cervix to delineate intrauterine disease (e.g., cystic hyperplasia, myometrial cysts), have been replaced today with abdominal ultrasound. Peritoneal laparoscopy is a technique that may be useful for direct observation of abdominal organs such as the uterus. The ovaries are embedded in fatty bursae and thus are difficult to visualize.
unless the examiner is skilled at incising the bursae. Exploratory laparotomy is sometimes necessary to completely examine the female reproductive tract. One can directly view and palpate the uterus, oviducts, and ovaries for malformations and pathologic changes that cannot be delineated in other ways. Placental sites or corpora lutea can be counted to determine embryonic death, and microbiologic samples and biopsy material can be obtained for laboratory evaluation. At the same time, surgical or medical measures may be performed for treatment of abnormalities.

**Pregnancy Examination**

Palpation of the uterus through the abdominal wall is the most practical method of pregnancy examination. At 20 to 22 days after ovulation, the uterus has distinct swellings 2 cm in diameter. After 28 days, these swellings have increased to about 3 to 5 cm in diameter, and this is the optimal time for diagnosis. (Diagnosis in the queen is easiest within 18 to 24 days and is difficult after 30 days.) By 35 days, the uterine swellings become confluent and diagnosis becomes more difficult. As pregnancy continues, individual fetuses may be palpated per rectum or through the abdominal wall.

Mammary glands enlarge at about 35 days of gestation, and the teats become enlarged and turgid. The nipples of a primiparous bitch are often quite red in color. Milk can be expressed from the teats during the last week of pregnancy.

Radiographs first show calcified fetal bitches 43 to 54 days after the first breeding in the bitch. Radiographs may be especially helpful if only one or two fetuses are present.

Today, abdominal ultrasonography is routinely performed to confirm a pregnancy diagnosis. Examination may reveal the presence of viable fetuses by the detection of fetal heartbeats as early as 24 to 28 days postbreeding.

**NORMAL BREEDING BEHAVIOR AND PHYSIOLOGY**

**The Intact Female Dog**

The pubertal estrus in the intact female dog (“bitch”) usually occurs at age 6 to 12 months; the reproductive life of the female dog is 8 to 10 years. The canine female is seasonally monestrous and ovulates spontaneously. The interval between estrous cycles ranges from 4 to 12 months, depending on the size and breed of the animal (e.g., Basenji, once a year; small breeds, two or three times a year; and large breeds, one or more times a year).

Proestrus lasts for 3 to 17 (mean, 9) days, during which time serum estradiol concentrations increase. Other characteristics are a bloody discharge from the vagina and a swollen vulva. The bitch attracts males but will not accept mating. Plasma estrogens reach a maximum level at the end of proestrus and then decrease.

Estrus lasts from 3 to 21 (mean, 9) days. Proestrus and estrus combined are called the “heat” period. The character of the discharge usually changes from bloody (during proestrus) to straw colored (during estrus) but may remain sanguineous without an adverse effect on fertility. The vulva is less turgid. The bitch is receptive and courts the male through foreplay, jumping, and trying to mount the male. The canine female presents the perineum in a lordosis-like posture and reflexively deviates the tail to one side. The bitch first refuses mating by the male at a variable time after the onset of estrus (usually 6 to 15 days). Ovulation usually occurs early in estrus (within the first 3 days), but some normal bitches may ovulate several days before to 11 days after onset of estrus. The ovum is not ready for fertilization until 48 hours later, after the second polar body has been extruded. The ovum lives 4 to 5 days, the transit time to the uterus being 4 to 10 days. Implantation occurs in 18 to 20 days, and an endotheliocchorial deciduate zonary placenta forms. The gestation cycle is 58 to 71 days from a single breeding or 62 to 64 days from ovulation.

Luteinizing hormone (LH) surges within 24 hours of the estrogen peak and causes ovulation. Progesterone increases gradually during estrus and is the cause of “behavioral” estrus. Serum progesterone concentration rises to about 2 ng/mL on the day of the LH surge; best reproductive performance (conception rate and litter size) occurs when the bitch is bred 4 days after the LH surge, which is also 2 days after ovulation. Progesterone reaches
a maximum 25 to 30 days after the LH surge and then gradually decreases to less than 1 mg/mL at parturition in the pregnant bitch. The progesterone decline is the cause of the temperature drop just before parturition. After ovum implantation, the hematocrit falls from 40% to 45% to 30%, which is probably a reflection of plasma volume expansion in the pregnant bitch; nonpregnant luteal-phase bitches also show a decline in hematocrit level from anestrous values but do not show the magnitude of decline seen in pregnancy.

_Diestrus_ (2 months) is the period when the corpus luteum produces progesterone, which is present in the bitch’s serum in concentrations exceeding 2 ng/mL. Diestrus begins on the first day of a predominantly noncornified vaginal smear and ends when serum progesterone declines to less than 2 ng/mL. Both pregnant and nonpregnant bitches have periods of diestrus lasting approximately 2 months.

_Anestrus_ (approximately 4 months) is the period during which the genital organs are relatively quiescent; the uterine lining regenerates.

**The Intact Male Dog**

Puberty in the male dog occurs at 6 to 12 months of age. Follicle-stimulating hormone (FSH) initiates spermatogenesis; LH increases the testosterone secretion of Leydig cells needed to complete spermatogenesis and to maintain accessory sex glands, secondary sex characteristics, and libido. Testosterone has a negative feedback effect on pituitary gonadotropins. Oxytocin and prostaglandins are important in the transport of sperm during ejaculation. Prostaglandins increase LH output and testosterone production and are the reason why sexual foreplay increases ejaculatory output and total number of sperm. Testosterone is of little value in the treatment of infertility, except to increase libido for 2 to 3 days after administration of low doses. Prolonged use causes testicular degeneration and a negative feedback effect on LH release.

In copulation, the male responds to the female in estrus by biting and nuzzling her neck and licking her flanks and perineal region. The male mounts and clasps the female’s hindquarters at the rear flank with his forelegs. After pelvic copulatory movement, intromission of the nonerect penis takes place, after which erection of the penis inside the vagina occurs. Ejaculation of the presperm and sperm-rich fractions of semen occurs during the most vigorous pelvic thrusting of the male, after which the male dismounts and, with the engorged penis still entrapped inside the vagina, lifts a hindleg over the rear quarters of the female and stands end to end with her in copulatory lock, or tie. During the tie, which may last from 5 to 60 minutes, the dog ejaculates the third and most voluminous fraction of semen, the prostatic fluid. Some male dogs rebreed within 2 hours of separation.

**The Intact Female Cat**

Puberty in the female cat occurs at 4 to 12 months of age. The reproductive life is 8 to 10 years. The female cat is seasonally polyestrous (January through September in the Northern Hemisphere, or continuous if 14 hours of light are available daily). Ovulation is induced by coitus or simulated coitus. Estrus occurs every 4 to 30 days. Proestrus lasts 0 to 2 days, during which pheromones increase and a very slight mucoid discharge from the Bartholin glands may occur.

_Estrus_ lasts 6 to 10 days in most queens (range, 2 to 12 days), and estrus length is not influenced by whether ovulation occurs. After nonfertile induction of ovulation, the corpora lutea last 30 to 40 days, and the cycle averages 6 weeks. The feline female has a characteristic call, rubs the head against objects with affection, purrs, crouches with forelegs, elevates rear quarters and treads, and deflects tail laterally. Ovulation occurs 24 to 50 hours after copulation (sensory nerves stimulate the hypothalamus to release gonadotropin-releasing hormone, which acts on the anterior pituitary to release a surge of LH, causing ovulation). Sperm requires 24-hour capacitation in the uterus to be fertile, and fertilization may occur up to 48 hours after ovulation.

Fertilized ova are in the oviduct for 4 days. Implantation occurs 14 days after breeding, and an endotheliochorial zonary placenta forms. Gestation length is 58 to 70 days (usually 60 to 63 days).
Postestrus occurs if the queen is not induced to ovulate. This stage is 7 to 21 days long; the ova degenerate, and then the queen returns to estrus.

Anestrus lasts 1 to 6 months, depending on photoperiod; during anestrus, the queen does not mate.

**THE INTACT MALE CAT**

Puberty occurs at about 6 months (depending on age at the beginning of the breeding season). The tom has depressed sexual activity in the fall. He has rigid territorial and behavioral habits regarding the breeding ritual, and the feline male does much calling and fighting to retain his home territory.

The male approaches the female, makes chattering sounds, and rubs his face over her shoulder and body. Foreplay is limited; the tom grasps the queen’s neck skin in his teeth and mounts.

**Additional Reading**


**UPPER RESPIRATORY TRACT EXAMINATION**

**Anatomic Limits**

The upper respiratory tract is particularly susceptible to injury and disease. Consequently, dogs and cats are commonly presented with acute-onset and chronic upper respiratory tract signs. Although there is no universal agreement stipulating where the upper respiratory tract ends and the lower respiratory tract begins, for this discussion, the term “upper respiratory tract” refers to all air-filled cavities rostral to the first cartilaginous ring of the trachea. This includes the following:

- The nasal cavity, external nares, and planum nasale
- The frontal sinuses (maxillary sinuses are not usually functional in dogs and cats)
- The nasopharynx and posterior nares (choana)
- The oral cavity (insofar as it is used for breathing and panting) and the upper dental arcade
- The oropharynx including the tonsils
- The larynx

**Localizing Clinical Signs of Upper Respiratory Tract Disease**

**Sonorous breathing:** Not a localizing sign. This term only refers to loud or noisy breathing.

**Sneezing and nasal discharge:** Localizes disease to the nasal cavity, frontal sinuses, or the upper dental arcade. In patients with epistaxis, coagulopathy must be ruled out if a nasal, sinus, or dental lesion cannot be identified. Sneezing may occur with or without evidence of nasal discharge. If discharge is present, the character (serous; mucoid, mucopurulent, or purulent; hemorrhagic [epistaxis]) should be noted. Indicate whether the discharge is bilateral or unilateral; if unilateral, designate patient’s right or left nostril.

**Sertor** (or snorting): Localizes disease to the oropharynx, the soft palate, or possibly the nasopharynx. *The patient must be examined under general anesthesia to adequately evaluate causes of stertor.*

**Stridor** (or wheezing): Localizes disease to the larynx and occasionally the cervical trachea. This is a critical sign in a dog or cat that deserves immediate attention. Restriction of airflow through the larynx is potentially life-threatening if not corrected in a timely manner. Tracheal collapse, laryngeal paralysis, laryngeal trauma, and tracheal or laryngeal tumor are possible causes that must be ruled out as soon as possible.
**Note:** Evaluation of the patient presented for chronic, persistent *stertor* or *stridor* requires examination with the patient under general anesthesia.

**Examination of the Nose and Oral Cavity**

External examination of the nose is limited to evaluation for symmetry, pain, nasal discharge (either unilateral [stipulate patient’s right or left nostril] or bilateral), and erosions or ulceration of the planum nasale. Further evaluation of the patient with a history of sonorous breathing, sneezing, or nasal discharge is indicated. Any additional studies are all special diagnostic procedures (see Section 4) that necessitate general anesthesia to achieve adequate visibility. Additional studies include the following:

- Nasal and sinus radiography
- Rhinoscopy and biopsy, where indicated
- Pharyngoscopy of both the oropharynx and the nasopharynx) and biopsy, where indicated.
- Otic examination (for nasopharyngeal polyps in cats)
- Computed tomography (CT) or magnetic resonance imaging (MRI) *(require access to a veterinary referral center, special equipment, and clinical specialists trained to perform and interpret studies)*

**Examination of the Nasopharynx and Oropharynx**

Stertor, or snorting, is the most common clinical sign associated with disease in the nasopharynx and oropharynx. Neither the nasopharynx nor the oropharynx can be properly examined in the awake or sedated patient. All dogs and cats presented because of stertor or sonorous breathing are candidates for examination under general anesthesia. Examination of the oropharynx can be conducted without the need for special equipment. Even in the anesthetized patient, visual examination of the nasopharynx is limited to what can be observed by retracting the soft palate forward (usually using a spay hook) and by digital palpation of the soft palate. Pharyngeal endoscopy (pharyngoscopy) is a valuable procedure in affected patients, as it allows the clinician to visualize the rostral-most aspect of the nasopharynx and the posterior nares, also called the *choana*.

Commonly encountered disorders include nasopharyngeal foreign bodies (anything goes!), elongated soft palate, nasopharyngeal polyps (feline only), tumor, and parasites (*Cuterebra larvae*).

**Examination of the Larynx**

Dogs and cats with clinical signs of laryngeal disease (e.g., stridor or wheezing) should be evaluated immediately for injury or disease that is causing restriction of airflow through the glottis. Assume these patients are emergency cases until proven otherwise. Failing to relieve airway obstruction at the level of the larynx may result in a fatal outcome!

External palpation of the larynx is indicated in the nonsedated patient to determine if change in the character of breathing or the pitch of the stridor can be detected. Patients that are in respiratory distress usually benefit from examination under general anesthesia. The patient should be managed as a critical patient, and the clinician should be prepared to place a tracheostomy tube if necessary to sustain anesthesia and provide oxygen to the patient. Once the patient is stable and breathing well, cervical radiographs may be helpful in elucidating certain types of foreign bodies entrapped in laryngeal tissues. Commonly encountered disorders include laryngeal paralysis (dogs and cats), laryngeal edema, laryngeal collapse or compressive trauma, foreign body (e.g., plant material, fish hook), and tumor.

**Lower Respiratory Tract Examination**

For purposes of this discussion, the lower respiratory tract extends from the first tracheal ring to the alveoli and includes the parietal and visceral pleura as well as the pleural space. When evaluating an animal with lower respiratory tract disease, it is important to (1)
carefully observe the animal while listening to the history; (2) examine the whole animal; and (3) palpate, percuss, and auscultate the thorax and neck. The completeness of the evaluation is tentative, depending on the animal’s condition. Before the animal is disturbed, determine its respiratory rate and pattern. Normally the dog breathes 10 to 30 times per minute (when not panting) and the cat breathes 20 to 60 times per minute. An increase in the respiratory rate (tachypnea) does not always mean that a respiratory disease is present. Excitement, heat, exercise, pain, shock, or anemia may cause an increased respiratory rate. A decreased respiratory rate may result from narcotic poisoning or metabolic alkalosis. An animal may have dyspnea and respiratory disease with a normal respiratory rate. The pattern or rhythm of breathing can help categorize disease.

| Dogs: 10 to 30 breaths per minute |
| Cats: 20 to 30 breaths per minute |

Labored breathing (dyspnea) can be slow, deep, and deliberate, or rapid and shallow. The dyspneic animal may assume a characteristic posture: a squatting position with abducted elbows. Dyspnea may be principally inspiratory, expiratory, or both. Inspiratory dyspnea is seen as difficulty in expanding the lungs with a relatively easy expiratory effort. Inspiratory dyspnea is observed most frequently in diseases that are restrictive. These diseases include those that restrict expansion of the lung because of disease of the pleura, chest wall, or neuromuscular apparatus or diseases with infiltrate within the lung parenchyma that displace alveolar air. Restrictive diseases are characterized by a reduced vital capacity and a small resting lung volume without an increase in airway resistance relative to lung volume. Elastic recoil is increased, and air is exhaled rapidly. The lung’s compliance is decreased. Examples of restrictive pulmonary disorders are pneumothorax, pleural effusions, or diffusely infiltrating diseases such as pneumonias or neoplasia. Inspiratory dyspnea is also observed with upper airway obstructions such as laryngeal paralysis.

Expiratory dyspnea is seen as difficulty in expelling air from the lungs. Normally, expiratory time is shorter than inspiratory time. Expiratory dyspnea is observed most frequently in obstructive lung disease. Airway obstruction caused by increased resistance to airflow can be caused by conditions inside the lumen, in the bronchial wall, or in the surrounding bronchial region. A combination of these problems can exist in disease. An airway lumen may be compromised by bronchiectasis, severe pulmonary edema, or aspiration of fluid. Contraction of bronchial smooth muscle (which occurs in bronchial asthma), hypertrophy of mucous glands, or inflammation and edema of the airway wall can cause obstruction. Obstruction outside an airway can be caused by destruction of lung parenchyma that results in loss of radial traction and narrowing, as in emphysema. Narrowing can also be caused by peribronchial edema. With obstructive disease, air can usually get into the lungs, and lung volumes are normal or even elevated. With partial airway obstruction, inspiratory forces open the airway to allow air to enter the lung, but because of dynamic compression, expiratory forces can cause the airway to collapse. Dynamic compression is the narrowing of the airways that occurs during expiration owing to increases in intrathoracic pressure and is a normal phenomenon that is exaggerated in situations of increased airway resistance and low lung volume. Inspiratory dyspnea and expiratory dyspnea may be observed together in various diseases, depending on the pulmonary changes present. Frequently, both types of dyspnea are associated with pulmonary edema.

When a dog or cat is dyspneic, the respiratory pattern is one that makes the work of breathing easiest with the least expenditure of energy. With reduced compliance or stiff lungs because of parenchyma or pleural disease, an animal will tend to take small, rapid breaths, whereas an animal with airway obstruction will take deeper, slower breaths. These patterns reflect the altered forces (elastic, viscous resistance) that must be overcome in the
breathing process. The cost of an increase in the work of breathing (oxygen consumed) will alter the ability of the animal to exercise.

Cyanosis is the appearance of a bluish tinge to the skin as a result of excess deoxyhemoglobin. For cyanosis to be apparent on physical examination, 5 g of deoxygenated hemoglobin must be present; therefore, marked hypoxemia may be present but not appreciated by observation of mucous membranes.

**Cardinal Signs of Lower Respiratory Disease**

Cough, dyspnea, production of abnormal secretions or discharges, noisy (or sonorous) breathing, and a change in characteristic airway sounds (either increased or decreased) are principal clinical signs seen in patients with lower respiratory disease. Animals with pulmonary disease may not be presented with clinical signs that direct attention to the lower respiratory tract (e.g., nasal discharge).

Determine the presence or absence of the following:

1. **Nasal discharge.** The occurrence of nasal discharge is a characteristic upper respiratory sign (see earlier) but is variably observed in patients with lower respiratory disease, especially infectious lower tract disease involving the trachea and bronchi. The discharge may be unilateral or bilateral. Determine the character (blood, mucoid, mucopurulent, purulent, or serous) and duration (acute, chronic) of the discharge.

2. **Abnormal lung sounds.** Carefully auscultate the lungs for evidence of abnormal breath sounds consistent with fluid or mucus in the lower airways.

| Note: | The absence of normal lung sounds is as important as the presence of abnormal lung sounds. |

3. **Coughing.** A true cough is characterized by the animal lowering its head and opening its mouth during expiration. The cough itself may be moist and productive, or dry, nonproductive, and paroxysmal, and it can be accentuated by collar pressure, exercise, or cold air. It should be noted whether the cough is productive or nonproductive. It is important to note that most dogs swallow expectorated secretions, so the cough may appear to be nonproductive. Examine any expelled secretions for color, consistency, cell content, foreign bodies, and parasites. Hemothysis (the presence of blood in expectorated secretions) is rare but may indicate tumor, *Paragonimus* species infection, injury from smoke inhalation, or pulmonary hypertension (especially heartworm disease).

4. **Dyspnea.** When presented for the chief complaint of intermittent “respiratory distress,” the owner should be questioned regarding when the animal has difficulty (e.g., at rest, after exercise) and how severe the distress is (e.g., open-mouth breathing, cyanosis). Animals with dyspnea at rest are critical patients. Stress and excessive handling should be avoided at all costs. In cats, dyspnea can be very subtle and may manifest only as open-mouth breathing.

5. **Noisy breathing.** Abnormal breathing sounds may be generated from the upper or lower airway. The owner should be questioned about the perceived origin of the abnormal sounds. In general, a noisy airway represents incomplete or partial airway obstruction.

**Thoracic Percussion**

**Percussion**

Percussion of the thorax entails using the fingers to strike the left and right hemithoraces with short, sharp blows as an aid in assessing the presence or absence of air versus fluid in the pleural space and lungs. Subtle changes in the sound produced may reflect excessive air or fluid in the pleural space or lungs. The technique of percussion involves placing the middle finger of the left hand firmly on the chest and using it as a pleximeter. Rap the distal phalanx abruptly with the middle finger of the right hand.
Three rules must be applied in percussion:
1. In defining boundaries, always move from the more resonant to the less resonant areas.
2. The long axis of the pleximeter (finger) must be parallel to the boundary of the edges of the organ being percussed.
3. The progression of the line of percussion should be at right angles to the edge of the organ.

Application and interpretation of percussion are more difficult in small animals than in large animals. Differences in sound are slight, and it is helpful to percuss the thorax systemically by tapping the ribs while a stethoscope is held firmly against the opposite chest wall. The percussion tones are thus greatly magnified by the instrument, and differences are more obvious.

Resonance
Resonance is increased when the pleural cavity contains air and the lung is collapsed. In this case, the musical “bell sound” may also be heard. Resonance may also be increased by emphysema (rare). Resonance is decreased when the lung is more solid than usual, as with edema, pneumonia, or tumor; when the pleura is thickened; when the pleural cavity contains fluid; or when an abdominal viscus is displaced into the thoracic space.

Thoracic Radiography
Thoracic radiography is among the most useful and important diagnostic procedures used to characterize lower respiratory tract disease in dogs and cats. Normal radiographic anatomy of the cat is depicted in Figure 2-20.

Thoracic Auscultation

The Stethoscope

All stethoscopes are not alike. Some are better designed acoustically than others. The bell transmits low-pitched sounds, and the diaphragm transmits soft, high-pitched sounds best. Electronic stethoscopes recently introduced not only include a volume control, but also have specially programmed filters designed to enhance listening to breath sounds versus heart sounds. For best results, do the following:
1. Perform the auscultation in a room as free of noise as possible.
2. Hold the stethoscope firmly against the chest.
3. Avoid hair friction and muscle noises. Wetting the subject’s hair may be helpful.
4. Listen with the animal breathing quietly, if possible.
5. Close the mouth of a panting animal; stop the subject from shivering or trembling.
6. Stop cats from purring. Gently blow short bursts of air across the nose and face or turn on a water faucet. And good luck—some cats insist on purring, no matter what!
7. Concentrate on each part of the respiratory or cardiac cycle separately. Listen intently!
8. Repeat the examination. This step is particularly important in dogs and cats suspected of having lower respiratory disease.
9. If using an electronic stethoscope, we have found maximum sound enhancement occurs when a contact medium (echocardiographic gel or EChG gel) is placed on the diaphragm of the stethoscope, thereby improving the contact between the stethoscope and the patient.

Characterization of Breath Sounds (See Table 2-4)
There is no clear consensus on the most appropriate terminology to use when describing normal versus abnormal lung sounds in animals. Auscultation of the lung sounds depends not only on the actual intensity of the sounds but also on the reflection and transmission of the sounds of the stethoscope.

Normal Breath Sounds
Normal breath sounds in dogs and cats include tracheal (very loud, high-pitched, harsh), bronchial (loud, high-pitched, tubular), and bronchovesicular (moderately loud, medium-pitched, rustling) sounds. Normal breath sounds ausculted over most of the
Peripheral lung fields are described as soft, low pitched, and having a gentle rustling quality. Normal breath sounds are usually louder in the cat, kitten, and puppy than in the dog because the transmission of the sounds to the chest wall is greater. Breath sounds may be decreased in intensity with conditions that decrease transmission, such as pleural effusion, pneumothorax, or chest wall thickness. With pleural fluid, the breath

Figure 2-20: A, Lateral radiograph of a normal cat with lung lobes identified. Cr, right and left cranial lung lobes; Cd, right and left caudal lung lobes; M, right middle lung lobe. B, Dorsoventral radiograph of a normal cat with lung lobes identified. A, Accessory lung lobe; Lcd, left caudal lung lobe; LCr, cranial lung lobe; M, right middle lung lobe; MLCr, middle portion of the left cranial lung lobe; RCd, right caudal lung lobe; RCr, right cranial lung lobe.
sounds are quiet ventrally but loud dorsally. With emphysema or complete airway obstruction, the lungs are too quiet.

**Crackles**

Previously called “rales,” crackles are discontinuous or interrupted, abnormal breath sounds usually caused by excessive fluid within the airways. This fluid could be caused by an exudate, as in pneumonia or other infections of the lung, or a transudate, as can occur in patients with pulmonary edema or decompensated congestive heart failure. Crackles are typically inspiratory and have been subcategorized as “wet” sounding or “dry.” Crackles are similar to the sound made when using a thumb and forefinger to rub one’s own hair near the ear.

<table>
<thead>
<tr>
<th>THE FOLLOWING ANATOMIC LANDMARKS SHOULD BE CONSISTENTLY IDENTIFIED AND EVALUATED WHEN REVIEWING THORACIC RADIOGRAPHS:</th>
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<tbody>
<tr>
<td>• Trachea (cervical and intrathoracic)</td>
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<tr>
<td>• Tracheal bifurcation (carina)</td>
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<tr>
<td>• Right and left cranial lung lobes</td>
</tr>
<tr>
<td>• Middle (right side only) lung lobe</td>
</tr>
<tr>
<td>• Right and left caudal lung lobes</td>
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**Wheeze**

Wheezees are characteristically described as an expiratory sound associated with forced airflow through abnormally collapsed airways with residual trapping of air. In humans, wheeze is commonly associated with asthma. However, in dogs and cats, wheezes are more likely to be associated with other causes such as airway obstruction (tumor) or obstructing foreign bodies.

> **Note:** Stridor, an audible wheeze frequently heard without the need for a stethoscope, is a critical clinical sign commonly associated with laryngeal obstruction (e.g., laryngeal paralysis). In the case of wheeze associated with laryngeal obstruction, the audible sound is likely to be best heard on inspiration.

With restrictive lung diseases, the lung volume is reduced and many airways are collapsed, and the opening of such airways produces crackles. Continuous, musical sounds that are high pitched, sibilant, or squeaky are called wheezes. Wheezing can occur during inspiration (laryngeal paralysis) or expiration (obstructive bronchial disease). It is important to realize that the absence of abnormal lung sounds does not guarantee that the lungs are normal.

**Additional Reading**


**URINARY TRACT EXAMINATION**

**Clinical History**

The history of any patient with a urinary tract disorder should include information about onset (acute or gradual), progression (improving, unchanging, or worsening), and response to previous therapy. Information about husbandry includes the animal’s immediate
environment (indoor or outdoor); use (pet, breeding, show, or working animal); geographic origin and travel history; exposure to other animals; vaccination status; diet; and previous trauma, illness, or surgery. A brief review of body systems may be conducted by determining the presence or absence of the following abnormalities: lethargy, anorexia, vomiting, diarrhea, coughing, sneezing, exercise intolerance, polyuria, polydipsia, weight loss, lameness, pruritus, alopecia, and exposure to drugs or toxins.

Questions related to the urinary tract include those about changes in water intake and the frequency or volume of urination. The owner should be questioned about pollakiuria, dysuria, or hematuria. Care must be taken to distinguish dysuria and pollakiuria from polyuria and to differentiate polyuria from urinary incontinence (see Section 3). The distinction between pollakiuria and polyuria is very important because polyuria may be a sign of upper urinary disease, whereas pollakiuria and dysuria usually indicate lower urinary tract disease. Occasionally an owner will complain that the dog is incontinent because it is urinating in the house when in reality the dog is polyuric but is not allowed outdoors frequently enough. Nocturia may be an early sign of polyuria but also can occur as a result of dysuria.

**Note:** Normal urine output ranges from 20 to 45 mL/kg/day in dogs and cats.

Information about the initiation of urination and diameter of the urine stream may be helpful because animals with partial obstruction may experience difficulty initiating urination or may have an abnormal urine stream. If hematuria is present, question the owner about its timing. Blood at the beginning of urination may indicate a disease process in the urethra or genital tract. Blood at the end of urination or throughout urination may signify a problem in either the bladder or upper urinary tract (kidneys or ureters).

Polydipsia usually is more easily detected by the owner than polyuria. Water intake should not exceed 90 mL/kg/day in dogs and 45 mL/kg/day in cats. It is helpful to describe amounts in quantitative terms familiar to the owner, such as cups (approximately 250 mL per cup) or quarts (approximately 1 L/q). Question the owner about exposure of the animal to nephrotoxins such as ethylene glycol in antifreeze (especially during fall and spring), aminoglycoside antibiotics, amphotericin B, thiacetarsamide, and nonsteroidal antiinflammatory drugs (NSAIDs). Also, determine whether the animal has received any drugs that could cause polydipsia and polyuria, such as corticosteroids or diuretics.

**Examination**

Disorders of the upper and lower urinary tract can culminate in organ failure and rapid onset systemic illness; hydration should be carefully assessed by evaluating physical parameters such as skin turgor, position of the eyes in the orbits, and moistness of mucous membranes. Pulse rate and character, CRT, and heart rate should also be recorded. The minimal amount of dehydration detectable clinically is approximately 5% of body weight; 15% dehydration is the maximal amount compatible with life. Skin elasticity and subcutaneous fat affect the reliability of skin turgor in the assessment of hydration. Obese animals may be dehydrated yet demonstrate normal skin turgor, whereas emaciated animals may have abnormal skin turgor yet not be dehydrated. Changes in weight can be used to monitor changes in hydration on an acute basis, because 1 L of fluid is equal to 1 kg of body weight. Evaluate the animal for the presence of ascites or subcutaneous edema, which may occur in patients with nephrotic syndrome.

**The Oral Cavity**

Examine the oral cavity for ulcers, which may occur in the presence of uremia, especially in dogs. Tongue-tip necrosis occasionally occurs in uremic dogs because of fibrinoid necrosis of vessels in the tongue. Examine the mucous membranes for pallor suggestive of anemia. Vascular injection may be observed in the sclera and soft palate of some uremic dogs. Examine the fundus for evidence of systemic hypertension, which can complicate renal
disease: retinal edema, retinal detachment, retinal hemorrhage, and vascular tortuosity. Young growing animals with renal failure may develop marked fibrous osteodystrophy characterized by enlargement and deformity of the maxilla and mandible, but this is uncommon in older dogs with renal failure.

### Abdominal Palpation
Both kidneys can be palpated in most cats, and the left kidney can be palpated in up to 20% of dogs. Evaluate the kidneys for size, shape, consistency, pain, and location. Unless empty, the bladder can be palpated in most dogs and cats. Note the degree of bladder distension, the presence or absence of pain, and thickness of the bladder wall. Evaluate the bladder for intramural masses (e.g., tumors) or intraluminal masses (e.g., stones, clots). In the absence of obstruction, a distended bladder in a dehydrated animal suggests abnormal renal function or administration of drugs that impair urinary concentrating ability (e.g., corticosteroids, diuretics).

### Pelvic Examination and Genitalia
Palpate the prostate gland (males) and pelvic urethra (males and females) by rectal examination. The perianal and sublumbar areas should be palpated carefully during rectal examination to determine the presence of tumors. Evaluate the prostate gland for size, symmetry, pain, and location. Exteriorize and examine the penis and palpate the testes for symmetry, consistency, masses, or pain. In the female dog, perform a vaginal examination to evaluate for abnormal discharge, masses, and the status of the urethral orifice.

### Blood Pressure Measurement
Multiple techniques for diagnostic blood pressure measurement are available. None are consistently perfect and all have advantages, as well as disadvantages. However, measurement and tracking of blood pressure in cats with renal insufficiency, and even in dogs with protein-losing nephropathy, has become an important component of care in the long-term management of these patients. In dogs and cats, blood pressure measurements center on systolic blood pressure and the prevention and management of hypertension and mitigate the risk of retinal damage (detachment) and blindness. There is little information known about the clinical significance of diastolic hypertension in dogs and cats. Therapeutic intervention (antihypertensives) is indicated in patients diagnosed with chronic renal insufficiency and systolic hypertension.

A wide variety of imaging procedures and laboratory tests are used to further assess qualitative and quantitative aspects of renal function, urine production, and micturition. Special diagnostic tests, including renal biopsy and cystoscopy, are described in detail in Section 4.

Laboratory assessment of renal function entails a variety of routine and specialized biochemical analyses, excretory (urinary) studies, and cultures. Laboratory tests and test protocols are described in detail in Section 5.

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### MAXIMUM NORMAL WATER INTAKE

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<tr>
<td><strong>Dogs:</strong></td>
<td>90 mL/kg/day</td>
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<td><strong>Cats:</strong></td>
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### NORMAL SYSTOLIC BLOOD PRESSURE

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<tr>
<td><strong>Dogs:</strong></td>
<td>160 to 180 mm Hg</td>
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<tr>
<td><strong>Cats:</strong></td>
<td>160 to 200 mm Hg</td>
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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

<table>
<thead>
<tr>
<th>DIFFERENTIAL DIAGNOSIS OF ABDOMINAL ENLARGEMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PHYSIOLOGIC ENLARGEMENT</strong></td>
</tr>
<tr>
<td>Postprandial</td>
</tr>
<tr>
<td>Pregnancy</td>
</tr>
<tr>
<td><strong>WITHOUT FLUID ACCUMULATION</strong></td>
</tr>
<tr>
<td>Organomegaly</td>
</tr>
<tr>
<td>Neoplasia</td>
</tr>
<tr>
<td>Obstructed</td>
</tr>
<tr>
<td>Gastric dilatation</td>
</tr>
<tr>
<td>Hyperadrenocorticism</td>
</tr>
<tr>
<td>Ruptured prepubic tendon</td>
</tr>
<tr>
<td>Bladder distension</td>
</tr>
<tr>
<td>Pneumoperitoneum</td>
</tr>
<tr>
<td><strong>WITH FLUID ACCUMULATION</strong></td>
</tr>
<tr>
<td>High-protein-content fluid: &gt;2.5 g/dL</td>
</tr>
<tr>
<td>Hepatic failure</td>
</tr>
<tr>
<td>Right-sided congestive heart failure</td>
</tr>
<tr>
<td>Inflammatory-infectious (e.g., feline infectious peritonitis [FIP])</td>
</tr>
<tr>
<td>Chemical or drug peritonitis</td>
</tr>
<tr>
<td>Trauma</td>
</tr>
<tr>
<td>Neoplasia</td>
</tr>
<tr>
<td>Hepatic vein thrombosis or vascular anomaly</td>
</tr>
<tr>
<td>Chyloabdomen</td>
</tr>
<tr>
<td>Low protein: &lt;2.5 g/dL</td>
</tr>
<tr>
<td>Hypoproteinemia (renal, hepatic, or gastro-intestinal cause)</td>
</tr>
<tr>
<td>Portal hypertension subsequent to primary liver disease</td>
</tr>
<tr>
<td>Neoplasia</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Aggressive Behavior in the Dog: Differential Diagnosis According to Origin</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pathophysiologically Based Aggressive Behavior</strong></td>
</tr>
<tr>
<td>Rabies</td>
</tr>
<tr>
<td>Intracranial neoplasia</td>
</tr>
<tr>
<td>Cerebral hypoxia</td>
</tr>
<tr>
<td>Seizure activity</td>
</tr>
<tr>
<td>Neuroendocrine disturbances</td>
</tr>
<tr>
<td><strong>Species-Typical Aggressive Behavior</strong>*</td>
</tr>
<tr>
<td>Dominance aggression</td>
</tr>
</tbody>
</table>


*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.

<table>
<thead>
<tr>
<th>Aggressive Behavior in the Cat: Differential Diagnosis According to Origin</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pathophysiologically Based Aggressive Behavior</strong></td>
</tr>
<tr>
<td>Rabies</td>
</tr>
<tr>
<td>Intracranial neoplasia and lesions</td>
</tr>
<tr>
<td><strong>Species-Typical Aggressive Behavior</strong>*</td>
</tr>
<tr>
<td>Intermale aggression</td>
</tr>
<tr>
<td>Predatory aggression</td>
</tr>
</tbody>
</table>

*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.
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.

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**

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.
### Differential Diagnosis of Hemoglobinuria

<table>
<thead>
<tr>
<th>Intravascular Destruction of Red Blood Cells</th>
<th>Extravascular Destruction of Red Blood Cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immune-mediated hemolytic anemia</td>
<td>Red blood cell parasites</td>
</tr>
<tr>
<td>Transfusion hemolysis</td>
<td>Immune-mediated hemolytic anemia</td>
</tr>
<tr>
<td>Sepsis</td>
<td>Pyrurate kinase deficiency (Basenji and Beagle)</td>
</tr>
<tr>
<td>Red blood cell parasites (e.g., Babesia species)</td>
<td>Congenital porphyria (cats)</td>
</tr>
<tr>
<td>Chemical-induced destruction</td>
<td>Hereditary stomatocytosis (Malamute)</td>
</tr>
<tr>
<td>Phrenothiazine</td>
<td>Microangiopathic disease (e.g., hepatic cirrhosis, hemangiosarcoma)</td>
</tr>
<tr>
<td>Acetaminophen</td>
<td></td>
</tr>
<tr>
<td>Methylene blue</td>
<td></td>
</tr>
<tr>
<td>Copper</td>
<td></td>
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<tr>
<td>Hypoosmolality</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Lysis of Red Blood Cells in Urine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hematuria combined with very dilute urine</td>
</tr>
<tr>
<td>Hematuria in stored urine</td>
</tr>
</tbody>
</table>

### Causes of Apparent or Actual Hematuria in Dogs and Cats Classified by Anatomic Site of Origin

<table>
<thead>
<tr>
<th>Site</th>
<th>Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kidney</td>
<td>Pyelonephritis, Glomerulonephopathy or glomerulonephritis, Neoplasia, Calculi, Renal cysts, Renal infarction</td>
</tr>
<tr>
<td></td>
<td>Renal trauma, Benign renal bleeding, Hematuria of Welsh Corgis, Dioctophyma renale infection, Microfilaria of Dirofilaria immitis, Chronic passive congestion</td>
</tr>
<tr>
<td>Bladder, ureter, urethra</td>
<td>Infection, inflammation, cystitis, LUTD, Cystic calculi, Neoplasia, Trauma, Thrombocytopenia, Capillaria plica infection, Cyclophosphamide therapy</td>
</tr>
<tr>
<td>Any site</td>
<td>Coagulopathy, Heat stroke, DIC</td>
</tr>
<tr>
<td>Extraurinary sources (genital tract or spurious hematuria)</td>
<td>Prostate, Neoplasia, Infection, Hypertrophy, Uterus, pyometra, Estrus, Subinvolution, Infection, Neoplasia (including TVT), Vagina, Trauma, Penis, TVT</td>
</tr>
</tbody>
</table>

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**

<table>
<thead>
<tr>
<th>Differential Diagnosis of Coma</th>
<th>Neurogenic</th>
<th>Nonneurogenic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute, nonprogressive</td>
<td>Intracranial hemorrhage</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Brain malformations</td>
<td>—</td>
</tr>
<tr>
<td>Acute, progressive</td>
<td>Metastatic lesions</td>
<td>Hypoglycemia</td>
</tr>
<tr>
<td></td>
<td>Epidural, subdural</td>
<td>Diabetic coma (hyperosmotic)</td>
</tr>
<tr>
<td></td>
<td>hemorrhage</td>
<td>Heat stroke</td>
</tr>
<tr>
<td></td>
<td>Meningoencephalitis</td>
<td>Hepatic or uremic encephalopathy</td>
</tr>
<tr>
<td></td>
<td>Cerebral edema</td>
<td>Infection</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypoxia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Thiamine deficiency (cat)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Heavy metal and drug toxicity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Carbon monoxide poisoning</td>
</tr>
<tr>
<td>Chronic, progressive</td>
<td>Hemorrhage (rare)</td>
<td>Heavy metal toxicity</td>
</tr>
<tr>
<td></td>
<td>Storage diseases</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hydrocephalus</td>
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<tr>
<td></td>
<td>Encephalitis</td>
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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.

CONSTITUTION (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 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 referrable 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

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 hypertension, especially heartworm disease

**PULMONARY PARENCHYMAL 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**

<table>
<thead>
<tr>
<th>DIFFERENTIAL DIAGNOSIS OF HEMOPTYSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CARDIOVASCULAR HEMOPTYSIS</strong></td>
</tr>
<tr>
<td>Thromboembolic disease</td>
</tr>
<tr>
<td>Heartworm disease (in the dog and cat)</td>
</tr>
<tr>
<td>Hyperadrenocorticism</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
</tr>
<tr>
<td>Renal amyloidosis (in the dog)</td>
</tr>
<tr>
<td>Idiopathic hemoptysis</td>
</tr>
<tr>
<td>Acute pulmonary edema</td>
</tr>
<tr>
<td>Arteriovenous fistula</td>
</tr>
<tr>
<td><strong>PARASITIC HEMOPTYSIS</strong></td>
</tr>
<tr>
<td>Lung flukes (e.g., <em>Paragonimus</em> species)</td>
</tr>
<tr>
<td>Lungworms (e.g., <em>Aelurostrongylus</em> species)</td>
</tr>
<tr>
<td><strong>INFLAMMATION-INDUCED HEMOPTYSIS</strong></td>
</tr>
<tr>
<td>Chronic bronchitis</td>
</tr>
<tr>
<td>Pneumonia</td>
</tr>
<tr>
<td>Mycotic lung infection</td>
</tr>
<tr>
<td>Lung abscess</td>
</tr>
<tr>
<td><strong>NEOPLASIA</strong></td>
</tr>
<tr>
<td>Either primary or metastatic neoplasia</td>
</tr>
<tr>
<td><strong>MISCELLANEOUS</strong></td>
</tr>
<tr>
<td>Coagulation disorder</td>
</tr>
<tr>
<td>Direct injury or trauma</td>
</tr>
<tr>
<td>Transtracheal aspiration (for diagnostic sampling)</td>
</tr>
</tbody>
</table>

**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**

<table>
<thead>
<tr>
<th>DIFFERENTIAL DIAGNOSIS OF DEAFNESS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ACQUIRED HEARING LOSS</strong></td>
</tr>
<tr>
<td>Degenerative causes</td>
</tr>
<tr>
<td>Neurogenic deafness in the geriatric dog and cat</td>
</tr>
<tr>
<td>Hearing loss occurring subsequent to chronic inflammatory disease (middle and inner ear structures)</td>
</tr>
<tr>
<td>Metabolic (endocrine) cause</td>
</tr>
<tr>
<td>Hypothyroidism</td>
</tr>
<tr>
<td>Neoplastic cause</td>
</tr>
<tr>
<td>Invasive tumors of the pharynx and retropharyngeal tissue</td>
</tr>
<tr>
<td>Infectious-inflammatory causes</td>
</tr>
</tbody>
</table>
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.
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).

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.
Differential Diagnosis

### Differential Diagnosis of Acute Renal Failure

<table>
<thead>
<tr>
<th><strong>Inflammatory-Infectious Causes</strong></th>
<th><strong>Nephrotoxins</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Leptospirosis</td>
<td>Heavy metals (lead, arsenic, thallium, mercury)</td>
</tr>
<tr>
<td>Pyelonephritis</td>
<td>Carbon tetrachloride</td>
</tr>
<tr>
<td>Immune complex glomerulonephropathy</td>
<td>Ethylene glycol (antifreeze)</td>
</tr>
<tr>
<td>Systemic lupus erythematosus</td>
<td>Aminoglycoside antibacterials (amikacin, gentamicin)</td>
</tr>
<tr>
<td>Heartworm disease</td>
<td>Antibiotics (cephaloridine, amphotericin B)</td>
</tr>
<tr>
<td>Pyometra</td>
<td>Hypercalcemia</td>
</tr>
<tr>
<td>Endocarditis</td>
<td>Anesthetics (adverse reaction—rare)</td>
</tr>
<tr>
<td>Feline leukemia virus infection</td>
<td></td>
</tr>
<tr>
<td>Lyme borreliosis (uncommon outside of endemic areas)</td>
<td></td>
</tr>
<tr>
<td><strong>Viral causes</strong></td>
<td></td>
</tr>
<tr>
<td>Canine distemper virus infection</td>
<td></td>
</tr>
<tr>
<td>Infectious canine hepatitis infection (rare in the United States)</td>
<td></td>
</tr>
<tr>
<td>Canine herpesvirus infection</td>
<td></td>
</tr>
<tr>
<td>(rare)</td>
<td></td>
</tr>
<tr>
<td><strong>Primary Renal Causes (Nephroves)</strong></td>
<td></td>
</tr>
<tr>
<td>Hypoperfusion (ischemia)</td>
<td></td>
</tr>
</tbody>
</table>

### 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.
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**

**INFECTIONOUS CAUSES**
- Intestinal parasites: Nematodes (e.g., ascarids, hookworms, whipworms, Strongyloides, Trichinella); protozoa (e.g., Coccidia, Giardia, Cryptosporidium, Pentatrichomonas)
- Bacterial: Escherichia coli, Salmonella, Pseudomonas, Clostridium, Campylobacter, Yersinia enterocolitica, Staphylococcus, Helicobacter (?)
- Viral: Paramyxovirus (canine distemper), parvovirus (feline and canine), adenovirus 1; coronavirus and reovirus—minor or insignificant
- Rickettsial: Salmon poisoning

**TOXIC CAUSES**
- Antimicrobials or antibiotics, parasiticides, antineoplastic agents, heavy metals, insecticides, organophosphate-containing compounds, antiinflammatory drugs

**DIETARY CAUSES**
- Dietary indiscretion, engorgement, food hypersensitivity, sudden change in diet

**BOWEL OBSTRUCTION**
- Foreign body, intussusception, volvulus, neoplasia

**EXTRAINTESTINAL CAUSES**
- 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**

<table>
<thead>
<tr>
<th>Clinical Signs</th>
<th>Small-Bowel Diarrhea</th>
<th>Large-Bowel Diarrhea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fecal volume</td>
<td>Markedly increased daily output (large quantity of bulky or watery feces with each defecation)</td>
<td>Normal or slightly increased daily output (small quantities with each defecation)</td>
</tr>
<tr>
<td>Frequency of defecation</td>
<td>Normal or slightly increased</td>
<td>Very frequent: 4-10 times per day</td>
</tr>
<tr>
<td>Urgency of tenesmus</td>
<td>Rare</td>
<td>Common</td>
</tr>
<tr>
<td>Mucus in feces</td>
<td>Rare</td>
<td>Common</td>
</tr>
<tr>
<td>Blood in feces</td>
<td>Dark black (digested)</td>
<td>Red (fresh)</td>
</tr>
<tr>
<td>Steatorrhea (malassimilation)</td>
<td>May be present</td>
<td>Absent</td>
</tr>
<tr>
<td>Weight loss and emaciation</td>
<td>Usual</td>
<td>Rare</td>
</tr>
<tr>
<td>Flatulence and emaciation</td>
<td>May be present</td>
<td>Absent</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Occasional</td>
<td>Occasional</td>
</tr>
</tbody>
</table>
### Differential Diagnosis

#### Diagnosis of Specific Chronic Diarrheal Disorders

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

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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 B<sub>12</sub> 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

<table>
<thead>
<tr>
<th>DIFFERENTIAL DIAGNOSIS FOR CYANOSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CARDIOVASCULAR CAUSES</strong></td>
</tr>
<tr>
<td>Right-to-left shunting congenital heart defect (e.g., right-to-left shunting patent ductus arteriosus)</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
</tr>
<tr>
<td>Decreased cardiac output</td>
</tr>
<tr>
<td>Arterial obstruction</td>
</tr>
<tr>
<td><strong>PULMONARY CAUSES</strong></td>
</tr>
<tr>
<td>Airway collapse or obstruction (multiple causes)</td>
</tr>
<tr>
<td>Hypoxia</td>
</tr>
<tr>
<td>Pulmonary edema</td>
</tr>
<tr>
<td>Oxygen diffusion–alveolar ventilation abnormalities</td>
</tr>
<tr>
<td>Pulmonary arterial-venous shunts or fistulas</td>
</tr>
<tr>
<td>Restrictive lung disease (e.g., hydrothorax, diaphragmatic hernia)</td>
</tr>
<tr>
<td><strong>TOXIC OR DRUG-RELATED CAUSES</strong></td>
</tr>
<tr>
<td>Paraquat poisoning</td>
</tr>
<tr>
<td>Acetaminophen (cats)</td>
</tr>
</tbody>
</table>

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.
## DIFFERENTIAL DIAGNOSES OF DOGS AND CATS PRESENTED FOR DYSPNEA

### 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
- Hemorrhax
- 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**

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

*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 (T₄) 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.

<table>
<thead>
<tr>
<th>PRIMARY CUTANEOUS CAUSES OF HAIR LOSS</th>
<th>SECONDARY CAUSES OF HAIR LOSS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infection</td>
<td>Genetic causes</td>
</tr>
<tr>
<td>Bacteria</td>
<td>Nutrition</td>
</tr>
<tr>
<td>Ectoparasites</td>
<td>Endocrine conditions (e.g., hypothyroidism, hypoadrenocorticism)</td>
</tr>
<tr>
<td>Dermatophytoses</td>
<td>Keratinization</td>
</tr>
<tr>
<td>Dermatomycoses</td>
<td>Atopic (allergic) or contact hypersensitivity</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>Drug therapy (especially corticosteroids and chemotherapeutic agents)</td>
</tr>
<tr>
<td>Keratinization</td>
<td>Environmental factors</td>
</tr>
<tr>
<td></td>
<td>Neoplasia</td>
</tr>
<tr>
<td></td>
<td>Psychogenic causes</td>
</tr>
</tbody>
</table>

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).
   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, adrenocorticotropin 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**

<table>
<thead>
<tr>
<th>DIFFERENTIAL DIAGNOSIS OF ATAXIA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CONGENITAL (SIGNS PRESENT BEFORE 3 MONTHS OF AGE)</strong></td>
</tr>
<tr>
<td>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.</td>
</tr>
<tr>
<td><strong>INFLAMMATORY</strong></td>
</tr>
<tr>
<td>Otitis interna, as an extension of otitis externa and media</td>
</tr>
<tr>
<td>Neuritis of the eighth cranial nerve (recrudescent feline herpesvirus-1 [?])</td>
</tr>
<tr>
<td>Infections</td>
</tr>
<tr>
<td><strong>TOXIC</strong></td>
</tr>
<tr>
<td>Drug-induced aminoglycoside therapy</td>
</tr>
<tr>
<td><strong>NUTRITIONAL</strong></td>
</tr>
<tr>
<td>Thiamine deficiency (cat only—rare)</td>
</tr>
<tr>
<td><strong>METABOLIC</strong></td>
</tr>
<tr>
<td>Central nervous system signs secondary to other diseases (e.g., hepatic, renal)</td>
</tr>
</tbody>
</table>

*Continued*
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]).

**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**

<table>
<thead>
<tr>
<th>POLYURIA OF RENAL ORIGIN</th>
<th>POLYURIA OF NONRENAL CAUSES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Renal failure</td>
<td>Diabetes insipidus (nephrogenic)</td>
</tr>
<tr>
<td>Glomerulonephritis</td>
<td></td>
</tr>
<tr>
<td>Tubular dysfunction</td>
<td></td>
</tr>
<tr>
<td>Renal medullary dysfunction</td>
<td></td>
</tr>
<tr>
<td>Postobstructive diuresis (e.g., feline urologic syndrome)</td>
<td></td>
</tr>
<tr>
<td>Diabetes insipidus (nephrogenic)</td>
<td></td>
</tr>
<tr>
<td>Hypercalcemic nephropathy</td>
<td></td>
</tr>
<tr>
<td>Fanconi syndrome</td>
<td></td>
</tr>
<tr>
<td>Medullary washout</td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td></td>
</tr>
<tr>
<td>Hyperadrenocorticism</td>
<td></td>
</tr>
<tr>
<td>Liver disease (nonspecific)</td>
<td></td>
</tr>
<tr>
<td>Pyometra</td>
<td></td>
</tr>
<tr>
<td>Pseudopsychogenic polydipsia</td>
<td></td>
</tr>
</tbody>
</table>

**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 T₄
   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

Apparently sick

Verify by measurement at home if necessary

CBC, biochemical profile, urinalysis

Negative

Positive

Rule out (confirm with specific tests)
Hyperthyroidism
Renal failure
Diabetes mellitus
Renal tubular glucosuria
Postobstructive diuresis
Pyometra
Hypoadrenocorticism
Hyperadrenocorticism
Hepatic failure
Polycythemia
Hypercalcemia
Hypokalemia

Nondehydrated

Dehydrated

Rehydrate

Rehydrate

Water deprivation test

Creatinine clearance test

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

<table>
<thead>
<tr>
<th>Differential Diagnosis of Pruritus (Not a Comprehensive List)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pustular dermatitis</td>
</tr>
<tr>
<td>Infectious</td>
</tr>
<tr>
<td>Puppy pyoderma</td>
</tr>
<tr>
<td>Folliculitis and furunculosis</td>
</tr>
<tr>
<td>Immune-mediated</td>
</tr>
<tr>
<td>Pemphigus foliaceus</td>
</tr>
<tr>
<td>Vesicle-forming disorders (e.g., drug eruption)</td>
</tr>
<tr>
<td>Linear immunoglobulin A (IgA) γ dermatosis</td>
</tr>
<tr>
<td>Idiopathic</td>
</tr>
<tr>
<td>Puppy “strangles”</td>
</tr>
<tr>
<td>Subcorneal pustular dermatitis</td>
</tr>
<tr>
<td>Vesicular or Bullous Eruption</td>
</tr>
<tr>
<td>Bullous dermatosis</td>
</tr>
<tr>
<td>Systemic lupus erythematosus (SLE)</td>
</tr>
<tr>
<td>Toxic epidermal necrolysis</td>
</tr>
<tr>
<td>Drug eruption</td>
</tr>
<tr>
<td>Acute contact dermatitis</td>
</tr>
<tr>
<td>Plaque Formation</td>
</tr>
<tr>
<td>Infectious dermatitis</td>
</tr>
<tr>
<td>Immune-mediated dermatitis</td>
</tr>
<tr>
<td>Neoplasia (e.g., mast cell tumor)</td>
</tr>
<tr>
<td>Papular Eruption (Dog)</td>
</tr>
<tr>
<td>Infectious</td>
</tr>
</tbody>
</table>

Folliculitis (bacterial, fungal, demodectic) |
Parasitic (Sarcoptes, Cheyletiella, lice, fleas) |
Vasculitis (Rocky Mountain spotted fever) |
Immune |
Allergy (atopy) |
Autoimmune (pemphigus foliaceus, SLE) |
Idiopathic |

Papular Eruption (Cat) |
Infectious (bacterial folliculitis) |
Dermatophytosis |
Parasitic (otodectic and notoedric mange, Cheyletiella, lice) |
Immune-mediated (hypersensitivity to food) |
Idiopathic miliary dermatitis |

Ulcerative Dermatitis |
SLE |
Leukocytoclastic vasculitis |
Erythema multiforme |
Toxic epidermal necrolysis |
Mycosis fungoides |
Epidermolysis bullosa complex |
Dermatomyositis |
Acute contact dermatitis |
Vogt-Koyanagi-Harada syndrome |
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

<table>
<thead>
<tr>
<th>ARTHROPATHIES IN THE DOG AND CAT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>NONINFLAMMATORY</strong></td>
</tr>
<tr>
<td>Degenerative joint disease (osteoarthritis, osteoarthrosis)</td>
</tr>
<tr>
<td>Primary</td>
</tr>
<tr>
<td>Secondary</td>
</tr>
<tr>
<td>As a sequela to acquired or congenital defects of the joints and supporting structures</td>
</tr>
<tr>
<td><strong>INFLAMMATORY</strong></td>
</tr>
<tr>
<td>Infectious</td>
</tr>
<tr>
<td>Traumatic</td>
</tr>
<tr>
<td>Neoplastic involvement</td>
</tr>
<tr>
<td>Drug-induced</td>
</tr>
</tbody>
</table>

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**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**

<table>
<thead>
<tr>
<th>DIFFERENTIAL DIAGNOSIS FOR LYMPHADENOMEGALY</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>GENERALIZED</strong></td>
</tr>
<tr>
<td>Lymphosarcoma</td>
</tr>
<tr>
<td>Diffuse, generalized skin disease</td>
</tr>
<tr>
<td>Infectious diseases (numerous infections are known to cause lymph node enlargement)</td>
</tr>
<tr>
<td>Parasitic (especially severe ectoparasitism, e.g., demodicosis with secondary pyoderma)</td>
</tr>
<tr>
<td>Recent (within days) vaccination</td>
</tr>
<tr>
<td><strong>LOCALIZED</strong></td>
</tr>
<tr>
<td>Any of the causes of generalized lymphadenomegaly (above)</td>
</tr>
<tr>
<td>Localized infection, especially in the skin or subcutaneous tissues</td>
</tr>
<tr>
<td>Cutaneous neoplasia other than lymphoma</td>
</tr>
</tbody>
</table>

**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).
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**

<table>
<thead>
<tr>
<th>Functional Megaesophagus*</th>
<th>Esophagitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary (or congenital)</td>
<td>Gastric reflux</td>
</tr>
<tr>
<td>Secondary (or acquired)</td>
<td>Neoplastic</td>
</tr>
<tr>
<td>Foreign body</td>
<td></td>
</tr>
<tr>
<td>Esophageal stricture</td>
<td></td>
</tr>
<tr>
<td>Esophageal diverticula</td>
<td></td>
</tr>
<tr>
<td>Neurogenic (e.g., myasthenia gravis, rabies)</td>
<td></td>
</tr>
<tr>
<td>Myopathy, smooth muscle</td>
<td></td>
</tr>
<tr>
<td>Extraesophageal compressive lesion (e.g., neoplasia)</td>
<td></td>
</tr>
<tr>
<td>Vascular anomaly</td>
<td></td>
</tr>
<tr>
<td><strong>Restrictive lesion without Megaesophagus</strong></td>
<td></td>
</tr>
<tr>
<td>Foreign body obstruction</td>
<td></td>
</tr>
<tr>
<td>Intrathoracic mass</td>
<td></td>
</tr>
<tr>
<td>Vascular ring anomaly</td>
<td></td>
</tr>
<tr>
<td>Esophageal stricture</td>
<td></td>
</tr>
</tbody>
</table>

*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**

<table>
<thead>
<tr>
<th><strong>INTRACRANIAL</strong></th>
<th><strong>EXTRACRANIAL</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital</td>
<td>Intoxication</td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>Lead</td>
</tr>
<tr>
<td>Lissencephaly</td>
<td>Organophosphates</td>
</tr>
<tr>
<td>Other malformations</td>
<td>Chlorinated hydrocarbons</td>
</tr>
<tr>
<td>Storage diseases</td>
<td>Strychnine</td>
</tr>
<tr>
<td>Vascular anomaly</td>
<td>Drugs</td>
</tr>
<tr>
<td>Traumatic</td>
<td>Garbage</td>
</tr>
<tr>
<td>Immediate</td>
<td>Metabolic</td>
</tr>
<tr>
<td>Posttrauma</td>
<td>Hypoglycemia</td>
</tr>
<tr>
<td>Inflammatory</td>
<td>Hypocalcemia</td>
</tr>
<tr>
<td>Distemper</td>
<td>Hyperkalemia</td>
</tr>
<tr>
<td>Rabies</td>
<td>Acid-base</td>
</tr>
<tr>
<td>Feline infectious peritonitis</td>
<td>Hepatic encephalopathy</td>
</tr>
<tr>
<td>Feline leukemia virus</td>
<td>Uremia</td>
</tr>
<tr>
<td>Toxoplasmosis</td>
<td>Hyperlipoproteinemia</td>
</tr>
<tr>
<td>Mycosis</td>
<td>Nutritional</td>
</tr>
<tr>
<td>Bacteria</td>
<td>Thiamine</td>
</tr>
<tr>
<td>Reticulosis</td>
<td>Parasites?</td>
</tr>
<tr>
<td>Parasites</td>
<td>Hypoxia</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>Cardiovascular disease</td>
</tr>
<tr>
<td>Primary</td>
<td>Respiratory disease</td>
</tr>
<tr>
<td>Metastatic</td>
<td>Birth</td>
</tr>
<tr>
<td>Vascular-cerebrovascular accident</td>
<td>Anesthetic accident</td>
</tr>
<tr>
<td></td>
<td>Hyperthermia</td>
</tr>
</tbody>
</table>


**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

<table>
<thead>
<tr>
<th>INTRANASAL CAUSES</th>
<th>Neoplasia (especially adenocarcinoma); bleeding is variably observed</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Serous Nasal Discharge</strong></td>
<td>Epistaxis</td>
</tr>
<tr>
<td>Acute viral upper respiratory infection (feline)</td>
<td>Acute nasal trauma</td>
</tr>
<tr>
<td>Feline chlamydioidis</td>
<td>Oronasal fistula</td>
</tr>
<tr>
<td>Intranasal parasites</td>
<td></td>
</tr>
<tr>
<td>Oronasal fistula (canine tooth)</td>
<td></td>
</tr>
<tr>
<td>Rhinosporidiosis (canine, rare)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>PURULENT NASAL DISCHARGE</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Viral upper respiratory infection with secondary bacterial infection (dog and cat)</strong></td>
<td>Bacterial rhinitis (especially <em>Bordetella bronchiseptica</em>)</td>
<td></td>
</tr>
<tr>
<td>Mycotic nasal disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Foreign body rhinitis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Traumatic rhinitis or sinusitis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cleft palate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neoplasia (several types possible)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nasopharyngeal polyps (feline, rare)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Benign nasal polyps (canine, rare)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oronasal fistula (occasionally associated with small amounts of blood)</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>MUCOID TO MUCOPURULENT NASAL DISCHARGE</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mycotic nasal disease (e.g., aspergillosis, cryptococcosis, blastomycosis)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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
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**

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

*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*
- Immune-mediated: 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 DEFCATE: 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

<table>
<thead>
<tr>
<th>DIFFERENTIAL DIAGNOSIS OF DYSCHEZIA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CONSTIPATION (see Box 3-5)</strong></td>
</tr>
<tr>
<td>Idiopathic ulcerative and inflammatory lesions</td>
</tr>
<tr>
<td>Colon (colitis)</td>
</tr>
<tr>
<td>Rectum (proctitis)</td>
</tr>
<tr>
<td>Anal glands (pain associated with inflammation or neoplasia; usually determined at surgery)</td>
</tr>
<tr>
<td>Neoplasia</td>
</tr>
<tr>
<td>Mucosa (e.g., rectal carcinoma)</td>
</tr>
<tr>
<td>Intestinal wall (e.g., carcinoma, sarcoma)</td>
</tr>
</tbody>
</table>

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.

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**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**

<table>
<thead>
<tr>
<th>DIFFERENTIAL DIAGNOSIS OF DYSURIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infectious and inflammatory causes</td>
</tr>
<tr>
<td>Bacterial cystitis</td>
</tr>
<tr>
<td>Urethritis</td>
</tr>
<tr>
<td>Prostatitis or benign prostatic hyperplasia (male dog)</td>
</tr>
<tr>
<td>Vaginitis</td>
</tr>
<tr>
<td>Feline lower urinary tract disease (FLUTD)</td>
</tr>
<tr>
<td>Cystic and urethral calculi</td>
</tr>
</tbody>
</table>

**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.
Rule out:
1. Cystitis
2. Urethritis
3. Bladder neoplasia
4. Bladder calculi
5. Vaginitis
6. Ruptured bladder, urethra

Expanded data base
1. Urine analysis and cytology
2. Urine culture (cystocentesis)
3. Double, contrast, urethral and bladder
4. Vaginal exam
5. Biopsy
6. Abdominocentesis

Normal or female — Palpate prostate — Abnormal

All cats LUTD

Empty

Distended

Male cat LUTD

(Urethral obstruction)

Abnormal — Palpate prostate — Normal or female

(Enlarged, painful)

Palpate bladder

Empty

Distended

Male cat LUTD

(Urethral obstruction)

Abnormal — Palpate prostate — Normal or female

(Enlarged, painful)

Pass catheter

Rule out urethral obstruction*

Rule out uremia due to:
1. Calculi
2. Bladder, urethral neoplasia
3. Urethral stricture

Present

1. Bacterial
2. Prostatic abscess

Expanded data base
1. Culture ejaculate
2. Lower tract radiography

Absent

1. Benign hyperplasia
2. Prostatic neoplasia
3. Prostatic cyst

Expanded data base
1. Cytology on ejaculate
2. Lower tract radiography
3. Biopsy

Expanded data base

1. BUN
2. Creatinine
3. Serum potassium
4. Total CO₂
5. Lower tract radiography
6. Biopsy

*If no obstruction exists, pursue bladder detrusor or neurologic dysfunction.

Figure 3-5: Algorithm for the differential diagnosis of dysuria. LUTD, Lower urinary tract disease.
DIFFERENTIAL DIAGNOSIS

<table>
<thead>
<tr>
<th>DIFFERENTIAL DIAGNOSIS OF PERIPHERAL EDEMA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>INCREASED CAPILLARY HYDROSTATIC PRESSURE</strong></td>
</tr>
<tr>
<td>Functional or structural obstruction to blood flow</td>
</tr>
<tr>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>Venous obstruction</td>
</tr>
<tr>
<td>Compression of a vessel by a mass lesion</td>
</tr>
<tr>
<td>Arteriovenous fistula</td>
</tr>
<tr>
<td><strong>DECREASED CAPILLARY ONCOTIC PRESSURE (Hypoalbuminemia)</strong></td>
</tr>
<tr>
<td>Protein-losing enteropathies</td>
</tr>
<tr>
<td>Protein-losing nephropathies</td>
</tr>
<tr>
<td>Decreased hepatic synthesis</td>
</tr>
<tr>
<td>Decreased dietary intake (protein malnutrition)</td>
</tr>
<tr>
<td>Chronic hemorrhage</td>
</tr>
<tr>
<td>Exudative lesion with large surface (e.g., burns, peritonitis)</td>
</tr>
<tr>
<td><strong>PERMEABILITY</strong></td>
</tr>
<tr>
<td>Chronic inflammatory disease (e.g., <em>Ehrlichia canis</em>)</td>
</tr>
<tr>
<td>Vasculitis (multiple infectious causes)</td>
</tr>
<tr>
<td>Vascular trauma</td>
</tr>
<tr>
<td>Toxins</td>
</tr>
<tr>
<td>Neurogenic, physical, or other vasoactive stimuli</td>
</tr>
<tr>
<td><strong>DECREASED LYMPHATIC DRAINAGE (Lymphedema)</strong></td>
</tr>
<tr>
<td>Congenital (primary) lymphedema—an autosomal dominant trait primarily affecting the hindlimbs by 3 to 6 months of age</td>
</tr>
<tr>
<td>Acquired (secondary) lymphedema (focal or regional)</td>
</tr>
<tr>
<td>Infectious, granulomatous, neoplastic, traumatic injury, or compression of lymphatics</td>
</tr>
<tr>
<td><strong>INCREASED INTERSTITIAL GEL MATRIX</strong></td>
</tr>
<tr>
<td>Myxedema (hypothyroidism)—rare</td>
</tr>
</tbody>
</table>

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:
   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**

<table>
<thead>
<tr>
<th>NEUROGENIC</th>
<th>NONNEUROGENIC WITHOUT DISTENDED BLADDER</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral lesions</td>
<td>Ectopic ureter(s)</td>
</tr>
<tr>
<td>Cerebellar lesions</td>
<td>Patent urachus</td>
</tr>
<tr>
<td>Brainstem lesions</td>
<td>Hormone-responsive incontinence</td>
</tr>
<tr>
<td>Spinal cord lesions</td>
<td>Nonneurogenic with Distended Bladder</td>
</tr>
<tr>
<td>Spinal nerve root lesions</td>
<td>Urethral obstruction, calculi, or neoplasia</td>
</tr>
<tr>
<td></td>
<td>Detrusor-urethral dyssynergia</td>
</tr>
<tr>
<td></td>
<td>Overflow incontinence (associated with polyuric states)</td>
</tr>
<tr>
<td></td>
<td>Urethral incompetence</td>
</tr>
<tr>
<td></td>
<td>Neoplasia</td>
</tr>
<tr>
<td></td>
<td>Reduced bladder capacity</td>
</tr>
<tr>
<td></td>
<td>Cystitis</td>
</tr>
</tbody>
</table>

**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 chorioretinitis, often with exudative retinal detachments; 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 hypophysis 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

<table>
<thead>
<tr>
<th>DIFFERENTIAL DIAGNOSIS OF VOMITING</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Infectious Causes</strong></td>
</tr>
<tr>
<td>Feline panleukopenia virus infection</td>
</tr>
<tr>
<td>Canine parvovirus infection</td>
</tr>
<tr>
<td>Canine coronavirus infection</td>
</tr>
<tr>
<td>Infectious canine hepatitis</td>
</tr>
<tr>
<td>Leptospirosis</td>
</tr>
<tr>
<td>Bacterial enteritis</td>
</tr>
<tr>
<td>Parasitic enteritis</td>
</tr>
<tr>
<td>Heartworm disease (cats)</td>
</tr>
</tbody>
</table>

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.
**Inflammatory Causes**
- Pyometra
- Prostatitis
- Peritonitis
- Acute pancreatitis
- Gastritis and enteritis
- Gastric ulcers

**Obstructive Causes**
- Intestinal foreign body
- Gastrointestinal neoplasia
- Gastric dilation-volvulus syndrome
- Pyloric stenosis
- Trichobezoar (hairballs)
- Diaphragmatic hernia

**Metabolic Causes**
- Renal failure (uremia)
- Hepatic disease

**Chemical Causes**
- Heavy metals, pesticides, solvents
- Digitalis, salicylates, mebendazole, penicillin, multiple antibiotics, morphine, antineoplastic drugs, others

**Idiopathic and Miscellaneous Causes**
- Psychogenic, vestibular (car sickness)
- Overconsumption of food, especially in puppies
- Various central nervous system diseases
- Biliary vomiting syndrome
- Autonomic epilepsy
- Constipation or obstipation
- Ileus, paralytic

**Diagnstic Plans**

1. Verification that the patient is vomiting, not gagging or retching subsequent to tracheal disease. Determine duration, precipitating causes, and current drug therapy. Assess associated signs.

2. Laboratory database, fundamental to the diagnostic plan. It must include CBC, biochemistry profile, urinalysis, and fecal flotation. Cats should also be tested for heartworm disease, FeLV, FIV, and hyperthyroidism. Perform serologic studies, as needed, to rule out systemic infections (e.g., systemic mycoses).

3. Radiographs of the thorax and abdomen; abdominal ultrasound.

4. Contrast radiographic studies of the stomach and small bowel (e.g., barium series).

5. Exploratory laparotomy, depending on patient condition.

6. Special diagnostic procedures: endoscopy, GI biopsy, double-contrast studies of the stomach and small bowel, and gastric motility studies (fluoroscopy).

**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**

<table>
<thead>
<tr>
<th>Differential Diagnosis of Hematemesis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary Gastric Disorders</strong></td>
</tr>
<tr>
<td>Gastritis</td>
</tr>
<tr>
<td>Infection (e.g., parvovirus)</td>
</tr>
<tr>
<td>Toxins</td>
</tr>
<tr>
<td>Bile reflux–bilious vomiting syndrome</td>
</tr>
<tr>
<td>Foreign body</td>
</tr>
<tr>
<td>Gastric ulcers</td>
</tr>
<tr>
<td>Drug-induced disorders (e.g., aspirin)</td>
</tr>
<tr>
<td>Idiopathic disorders</td>
</tr>
<tr>
<td>Metabolic disorders (e.g., renal failure)</td>
</tr>
<tr>
<td>Neoplastic disorders (e.g., carcinoma)</td>
</tr>
</tbody>
</table>

**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

<table>
<thead>
<tr>
<th>DIFFERENTIAL DIAGNOSIS OF WEAKNESS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CONCURRENT DRUG THERAPY</strong></td>
</tr>
<tr>
<td>Multiple drugs may be involved, especially anticonvulsants, corticosteroids, antitussives, cardiovascular drugs, and some antimicrobials.</td>
</tr>
<tr>
<td><strong>METABOLIC DISEASE</strong></td>
</tr>
<tr>
<td>Renal and hepatic diseases predominate</td>
</tr>
<tr>
<td>Electrolyte disorders (e.g., hypokalemia, hyperkalemia, hypercalcemia, hypernatremia)</td>
</tr>
<tr>
<td>Acidosis or alkalosis (pulmonary and/or renal mechanisms may be involved)</td>
</tr>
<tr>
<td>Hypertriglyceridemia (sustained)</td>
</tr>
<tr>
<td><strong>INFECTIONAL DISEASE</strong></td>
</tr>
<tr>
<td>Any acute-onset or sustained infection involving any pathogenic organism, especially in young dogs and cats</td>
</tr>
<tr>
<td><strong>HEMATOLOGIC DISORDERS</strong></td>
</tr>
<tr>
<td>Anemia predominates (acute-onset or gradual onset)</td>
</tr>
<tr>
<td>Bone marrow neoplasia (e.g., leukemia—multiple types, myeloma)</td>
</tr>
<tr>
<td><strong>ENDOCRINE DISORDERS (MULTIPLE DISORDERS)</strong></td>
</tr>
<tr>
<td>Hypothyroidism (especially in dogs)</td>
</tr>
<tr>
<td>Hyperthyroidism (feline, apathetic form)</td>
</tr>
<tr>
<td>Hyperadrenocorticism (Cushing syndrome)</td>
</tr>
<tr>
<td>Hypoadrenocorticism (Addison disease)</td>
</tr>
<tr>
<td>Parathyroid disorders</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Neoplasia (insulinoma)</td>
</tr>
</tbody>
</table>
Differential Diagnosis of Weakness—Cont’d

**Cardiovascular Disease**
Valvular disease, especially right-sided heart failure (e.g., mitral insufficiency)
  - Note: The presence of a heart murmur is not consistently associated with weakness.
  - Arrhythmia (ventricular or atrial)
  - Cardiomyopathy
  - Hypotension (includes overtreatment of hypertension)

**Respiratory Disease**
Sustained hypoxia (multiple causes)
Obstructive airway disease
Interstitial disease (pneumonitis or pneumonia; infectious and noninfectious)

**Neuromuscular Disorders**
Primary brain disease (e.g., hydrocephalus, neoplasia, encephalitis)
Nonparalyzing spinal disease (e.g., meningitis, infection [feline infectious peritonitis, FIP])
Peripheral neuropathies (especially myasthenia gravis and parasite-induced neuropathies [ticks])
Toxin or venom

**Diet and Nutritional Disorders**
Malnutrition

**Gastrointestinal Disorders**
Malabsorptive disease
Maldigestive disease
Neoplasia

Diagnostic Plans

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

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.
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.

**ASSOCIATED SIGNS**

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**DIFFERENTIAL DIAGNOSIS**

<table>
<thead>
<tr>
<th>Prehepatic (Hemolytic)</th>
<th>Hepatic (Hepatocellular)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immune-mediated hemolytic anemia (Coombs-positive anemia)</td>
<td>Cholangitis or cholangiohepatitis</td>
</tr>
<tr>
<td>Heartworm disease, especially postcaval syndrome</td>
<td>Chronic active liver disease</td>
</tr>
<tr>
<td>Hemolytic septicemia</td>
<td>Copper storage disease (Bedlington Terriers and Doberman Pinschers)</td>
</tr>
<tr>
<td>Transfusion-induced hemolysis</td>
<td>Drug-induced or vaccine-induced disease</td>
</tr>
<tr>
<td></td>
<td>Thiacetarsamide—sporadic occurrence</td>
</tr>
</tbody>
</table>
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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### Imidazole anthelmintics—sporadic occurrence
- Anticonvulsants, especially primidone
- Acetaminophen (cats)
- Hepatic fibrosis
- Septicemia
- Gram-negative bacteremia
- Leptospirosis
- Viral
- Canine viral hepatitis
- Feline leukemia
- Feline infectious hepatitis
- Neoplasia, primary or metastatic

### Posthepatic (Obstructive)
- Cholangitis or cholangiohepatitis
- Hepatic fibrosis
- Neoplasia
- Acute pancreatitis
- Extrahepatic neoplasm (by compression)
- Bile duct trauma
- Ruptured gallbladder (usually traumatic)
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Routine Procedures

Administration Techniques for Medications and Fluids—Canine

Patient Preparation
None required.

Technique
The simplest method of administering tablets or capsules to dogs is to hide the medication as bait in food. Offer small portions of unbaited cheese, meat, or some favorite food to the dog initially. Then offer one portion that includes the medication. Pill Pockets Canine Treats* is a commercially available alternative.

*Pill Pockets Treats for Dogs and Pill Pockets Treats for Cats; Greenies (www.greenies.com).
Note: Oral medication frequently is dispensed to owners without regard for the client's knowledge of how to administer a pill or tablet or without asking whether the client is even physically able to administer medications. Clear instructions that include having the client perform the technique in the hospital significantly improve compliance.

For anorexic dogs or when pills must be given without food, give medications quickly and decisively so that the process of administering the medication is accomplished before the dog realizes what has happened. With cooperative dogs, insert the thumb of one hand through the interdental space, and gently touch the hard palate. This will induce the cooperative dog to open its mouth (Figure 4-1). Using the opposite hand (the one holding the medication), gently press down on the mandible to open the mouth further (Figure 4-2).

Figure 4-1: Use of the thumb only to open a cooperative dog's mouth.

Figure 4-2: Use of the opposite hand to place a tablet or capsule on the caudal aspect of the tongue.
Quickly place the tablet or capsule onto the caudal aspect of the tongue. Quickly withdraw the hand and close the dog’s mouth. When the dog licks its nose, the medication likely has been swallowed.

Dogs that offer more resistance can be induced to open their mouths by compressing their upper lips against their teeth. As they open the mouth, roll their lips medially so that if they attempt to close the mouth, they will pinch their own lips. Alternatively, dripping water onto the nostrils or blowing into the patient’s nose sometimes encourages the patient to accept and swallow oral medications (tablets or capsules). Pilling syringes are also available and in some dogs seem to work well.

**Special Considerations**

Critical to the oral administration of medication is the ability of the owner to effectively administer the medication at home. Animals that aggressively resist oral medication should be treated by alternative methods—for example, parenteral administration of medication. It is inappropriate, and unsafe, to delegate treatment responsibilities to the owner of a dog (or cat) that might injure the individual who is attempting to treat the patient.

**Oral Administration: Tablets and Capsules—Feline**

**Patient Preparation**

None required.

**Technique**

*Caution:* Only experienced individuals should attempt this technique of administering tablets or capsules to cats. Even cooperative cats that become intolerant will bite. Therefore, this is not a technique recommended for most owners to try at home, even if specific instructions have been given.

Two methods of pill administration are used in cats. In both methods the cat's head is elevated slightly with the nose pointed upward. Success in administering pills and tablets to a cat entails a delicate balance between what works well and what works safely. In cooperative cats, it may be possible to use one hand to hold and position the head ([Figure 4-3](#)) while using the opposite hand (the one holding the medication) to open the mouth gently by depressing the proximal aspect of the mandible ([Figure 4-4](#)). Press the skin adjacent to the maxillary teeth gently between the teeth as the mouth opens, thereby discouraging the
cat from closing its mouth. With the mouth open, drop (do not push) the medication (try generously lubricating the tablet or capsule with butter) into the oral cavity. The cat can be tapped under the jaw or on the tip of the nose to facilitate swallowing if you really think this works. If the cat licks, administration was probably successful.

Alternatively, some cats will tolerate a specially designed “pilling syringe” in an attempt to administer a tablet or capsule. The pilling syringe works well as long as it is inserted cautiously and atraumatically into the cat’s mouth. However, if resistance ensues, the rigid pilling syringe may injure the hard palate during the ensuing struggle. Subsequent attempts to use the syringe may be met with increasing resistance and increasing risk of injury. Success with a pilling syringe depends largely on the cat. Pill Pockets Treats are also available for use in cats and are manufactured in chicken and fish flavors. In addition, as is the case in dogs, some cats will respond to the application of water drops on the nostrils or blowing into the nostrils to encourage swallowing.

**Special Considerations**

When dispensing oral medications for home administration to cats, do not expect clients to force a tablet or capsule into a cat’s mouth. Although some clients are remarkably capable and confident with their ability to administer oral medications to cats, the risk of injury to the client can be significant. Whenever feasible, liquid medications or pulverized tablets should be mixed with the diet or an oral treat readily accepted and consumed (see the following discussion).

**Oral Administration: Liquids**

**Without a Stomach Tube**

**Patient Preparation**

None required. Technique is appropriate for owners to perform at home.

**Technique**

Small amounts of liquid medicine can be given successfully to dogs and cats by pulling the commissure of the lip out to form a pocket (Figure 4-5). Deposit the liquid medication into the “cheek pouch,” where it subsequently flows between the teeth as the head is held slightly upward. Patience and gentleness, along with a reasonably flavored medication, contribute to the success.
Spoons are ineffective, as fluids are easily spilled. A disposable syringe can be used to measure and administer liquids orally. Depending on the liquid administered, disposable syringes can be reused several times, assuming they are rinsed after each administration. In addition, disposable syringes can be dispensed legally to clients for home administration of liquid medication. Mixing of medications in the same syringe is not recommended. However, dispensing of a separate, clearly marked syringe for each type of liquid medication prescribed for home administration is recommended.

Special Considerations
Compounding pharmacies are also available and can mix many medications into palatable flavors to help facilitate the oral administration of medications.

Dogs with swallowing disorders should not be treated at home with liquid medications because this could cause complications associated with aspiration.

With an Administration Tube

Patient Preparation
None required.

Note: This procedure is reserved for in-hospital use only. The technique should be performed only by individuals trained to perform this procedure.

Technique
Administration of medications, contrast material, and rehydrating fluids can be accomplished with the use of a well lubricated feeding tube passed through the nostrils into the stomach or distal esophagus. When a feeding tube is placed for long-term use (multiple days) and repeated use (described under Gastrointestinal Procedures later), it is generally recommended to avoid passing the tip of the tube beyond the distal esophagus. The reason for recommending nasoesophageal intubation over nasogastric intubation is based on the fact that reflex peristalsis of the esophagus against a tube passing through the cardia can result in significant mucosal ulceration within 72 hours. This is not a factor in patients receiving a single dose of medication or contrast material.
The narrow lumen of tubes passed through the nostril of small dogs and cats limits the viscosity of solutions that can be administered through a tube directly into the gastrointestinal tract. Nasoesophageal intubation can be done with a variety of tube types and sizes (Table 4-1). Newer polyurethane tubes, when coated with a lidocaine lubricating jelly, are nonirritating and may be left in place with the tip at the level of the distal esophagus. When placing the nasogastric tube, instill 4 to 5 drops of 0.5% proparacaine in the nostril of the cat or small dog; 0.5 to 1.0 mL of 2% lidocaine instilled into the nostril of a larger-breed dog may be required to achieve the level of topical anesthesia needed to pass a tube through the nostril. With the head elevated, direct the tube dorsomedially toward the alar fold (Figure 4-6). Pushing dorsally on the nasal philtrum and pushing the nostril from lateral to medially will help facilitate passage of the tube into the ventromedial nasal meatus.

**Caution:** The tip of the feeding tube can be inadvertently introduced through the glottis and into the trachea. Topical anesthetic instilled into the nose can anesthetize the arytenoid cartilages, thereby blocking a cough or gag reflex.

After inserting the tip 1 to 2 cm into the nostril, continue to advance the tube until it reaches the desired length. If the turbinates obstruct the passage of the tube, withdraw the tube by a few centimeters. Then readvance the tube, taking care to direct the tube ventrally through the nasal cavity. Occasionally it will be necessary to withdraw the tube completely.

---

**TABLE 4-1  The French Catheter Scale Equivalents**

<table>
<thead>
<tr>
<th>Scale</th>
<th>Millimeters</th>
<th>Inches</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>1</td>
<td>0.039</td>
</tr>
<tr>
<td>4</td>
<td>1.35</td>
<td>0.053</td>
</tr>
<tr>
<td>5</td>
<td>1.67</td>
<td>0.066</td>
</tr>
<tr>
<td>6</td>
<td>2</td>
<td>0.079</td>
</tr>
<tr>
<td>7</td>
<td>2.3</td>
<td>0.092</td>
</tr>
<tr>
<td>8</td>
<td>2.7</td>
<td>0.105</td>
</tr>
<tr>
<td>9</td>
<td>3</td>
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<tr>
<td>10</td>
<td>3.3</td>
<td>0.131</td>
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<td>11</td>
<td>3.7</td>
<td>0.144</td>
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<tr>
<td>12</td>
<td>4</td>
<td>0.158</td>
</tr>
<tr>
<td>13</td>
<td>4.3</td>
<td>0.170</td>
</tr>
<tr>
<td>14</td>
<td>4.7</td>
<td>0.184</td>
</tr>
<tr>
<td>15</td>
<td>5</td>
<td>0.197</td>
</tr>
<tr>
<td>16</td>
<td>5.3</td>
<td>0.210</td>
</tr>
<tr>
<td>17</td>
<td>5.7</td>
<td>0.223</td>
</tr>
<tr>
<td>18</td>
<td>6</td>
<td>0.236</td>
</tr>
<tr>
<td>19</td>
<td>6.3</td>
<td>0.249</td>
</tr>
<tr>
<td>20</td>
<td>6.7</td>
<td>0.263</td>
</tr>
<tr>
<td>22</td>
<td>7.3</td>
<td>0.288</td>
</tr>
<tr>
<td>24</td>
<td>8</td>
<td>0.315</td>
</tr>
<tr>
<td>26</td>
<td>8.7</td>
<td>0.341</td>
</tr>
<tr>
<td>28</td>
<td>9.3</td>
<td>0.367</td>
</tr>
<tr>
<td>30</td>
<td>10</td>
<td>0.393</td>
</tr>
<tr>
<td>32</td>
<td>10.7</td>
<td>0.419</td>
</tr>
<tr>
<td>34</td>
<td>11.3</td>
<td>0.445</td>
</tr>
</tbody>
</table>

*Multiple types of pediatric polyurethane nasogastric feeding tubes are available in sizes ranging from 8F to 12F that easily accommodate administration of liquids medications and fluids to kittens, cats, and small dogs.
from the nostril and repeat the procedure. In particularly small patients or patients with obstructive lesions (e.g., tumor) in the nasal cavity, it may not be possible to pass a tube. Do not force the tube against significant resistance through the nostril.

Gavage, or gastric lavage and feeding, in puppies and kittens can be accomplished by passing a soft rubber catheter or feeding tube into the mouth, tilting the puppy’s or kitten’s head, and watching it swallow the tube. Most puppies or kittens will struggle and vocalize. They usually will not vocalize if the tube has been placed into the trachea. A 12F catheter is of an adequate diameter to pass freely, but it is too large for dogs and cats less than 2 to 3 weeks of age. Mark the tube with tape or a pen at a point equal to the distance from the mouth to the last rib. Merely push the tube into the pharynx and down the esophagus to the caudal thoracic level (into the stomach). Verify the placement of the tube using the same dry syringe aspiration technique to ensure that the tube is positioned in the esophagus or stomach rather than the trachea. Attach a syringe to the flared end, and slowly inject medication or food.

Depending on the feeding tube type, the end of the tube may or may not accommodate a syringe. For example, soft, rubber urinary catheters are excellent tubes for single administration use. However, the flared end may not accommodate a syringe. To affix a syringe to the outside end of a tapered feeding tube or catheter, insert a plastic adapter (Figure 4-7) into the open end of the tube.

![Figure 4-6: Initial dorsomedial placement of a nasoesophageal tube before complete insertion.](image)

![Figure 4-7: Use of a plastic adaptor (“Christmas tree”) to affix a syringe to a nasoesophageal feeding tube.](image)
Special Considerations
Esophageal (versus intratracheal) placement of the feeding tube can be verified with a dry, empty syringe. Attach the empty syringe to the end of the feeding tube. Rather than injecting air or water in an attempt to auscultate borborygmus over the abdomen, attempt simply to aspirate air from the feeding tube. If there is no resistance during aspiration and air fills the syringe, the tube likely has been placed in the trachea. Completely remove the tube and repeat the procedure. However, if repeated attempts to aspirate are met with immediate resistance and no air enters the syringe, the tube tip is positioned properly within the esophagus. If there is any question regarding placement, a lateral survey radiograph is indicated.

Definitive confirmation of proper tube placement can be made by diluting 1 to 2 ml of an iodinated contrast agent with sterile saline, instilling the liquid into the tube, then taking a lateral thoracoabdominal radiograph to confirm entry of the contrast material into the stomach.

TOPICAL ADMINISTRATION
Ocular
Patient Preparation
None required.

Technique
The usual methods of applying medication directly to the eyes include liquid (drops) and ointments. The route and frequency of medication depend on the disease being treated. Liquids and ointments are appropriate for owner administration.

Liquid medications (usually 1 or 2 drops) can be applied directly to the cornea. It is important to instruct the owner on the proper technique and to stress that because liquids only fall downward, the patient's nose must be directed upward before one attempts to administer liquid medications onto the eye. It is still quite difficult to encourage a drop of liquid, as it is squeezed from its container, to fall horizontally, despite frequent attempts to do so. Ointment, as a ¼- or ⅛-inch strip, is typically administered directly onto the sclera (dorsally) or into the lower conjunctival cul-de-sac such that as the lids close, a film of ointment is spread across the cornea.

Special Considerations
The tip of the applicator tube for liquids and ointments should never be allowed to make contact with the eye or conjunctiva. Doing so is likely to result in contamination of the medication, especially with liquid medications.

Otic
Patient Preparation
None required.

Technique
Liquid solutions are more effective vehicles for administering medication into the external ear canal. Physically removing debris may be necessary in some patients that require topical otic medications. Occasionally, oral supplemental medication may also be required. When applying the medication, a few drops of liquid are generally sufficient. The ear should be massaged gently after instillation to facilitate the spread the medication within the external ear canal.

Special Considerations
Medicated powders generally are contraindicated in the external ear canal. Also, the application tip of liquid medications must not come in direct contact with the skin. Doing so is likely to result in contamination of the entire dispensing bottle.
Nasal

**Patient Preparation**

None required.

**Technique**

Intranasal administration of liquids in dogs and cats is usually limited to a single dose of a vaccine specifically labeled for intranasal administration. There is little indication for routine instillation of liquids into the nostrils of dogs and cats. Rarely, administration of isotonic solutions directly into the nostrils is indicated. In contrast to single-dose vaccines, lavage solutions applied intranasally are usually multiple-dose containers. Therefore, the tip of the administration device should not be allowed to directly contact the patient’s skin or nose. Doing so may result in contamination of the entire bottle. Oily drops are not advised because they may damage the nasal mucosa or may be inhaled.

The technique for intranasal administration of vaccine is straightforward and usually works quite well…the first time. Some animals, dogs more than cats, will aggressively resist intranasal administration of vaccine. Attempts to overcome this resistance include covering the eyes with a towel or otherwise distracting the patient with noise or other visual cues.

**Special Considerations**

Concerns expressed over the loss of vaccine immediately after intranasal administration are generally unfounded. Manufacturers of intranasal vaccines typically include a greater antigen (virus or bacteria) titer per dose than is necessary to induce a protective immune response. If the patient resists aggressively and the vaccine is indicated, parenteral preparations are available for all intranasal vaccines and should be considered.

Dermatologic

**Patient Preparation**

Several objectives should be considered when treating dermatologic disorders with topical medication: (1) eradication of causative agents; (2) alleviation of symptoms, such as reduction of inflammation; (3) cleansing and debridement; (4) protection; (5) restoration of hydration; and (6) reduction of scaling and callus. Many different forms of skin medications are available, but the vehicle in which they are applied is a critical factor (Box 4-1).

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**BOX 4-1 VEHICLES USED IN THE ADMINISTRATION OF TOPICAL SKIN MEDICATIONS**

- **Lotions** are suspensions of powder in water or alcohol. They are used for acute, eczematous lesions. Because they are less easily absorbed than creams and ointments, lotions need to be applied two to six times a day.
- **Pastes** are mixtures of 20% to 50% powder in ointment. In general, they are thick, heavy, and difficult to use.
- **Creams** are oil droplets dispersed in a continuous phase of water. Creams permit excellent percutaneous absorption of ingredients.
- **Ointments** are water droplets dispersed in a continuous phase of oil. They are very good for dry, scaly eruptions.
- **Propylene glycol** is a stable vehicle and spreads well. It allows good percutaneous absorption of added agents.
- **Adherent dressings** are bases that dry quickly and stick to the lesion.
- **Shampoos** are usually detergents designed to cleanse the skin. If shampoos are left in contact with the skin for a time, added medications may have specific antibacterial, antifungal, or antiparasitic effects.
Routine Procedures

Technique
In all cases, apply topical medications to a clean skin surface in a very thin film, because only the medication in contact with the skin is effective. In most cases, clipping hair from an affected area enhances the effect of medication. When dispensing medications to owners for home administration, the owner should be instructed to wear disposable examination gloves if using fingers and hands to apply the medication.

Special Considerations
With the widespread availability of compounding pharmacies, prescribing compounded medications for topical and oral administration recently has become a popular dispensing technique for dogs and cats requiring long-term, daily medication. Caution is warranted. Some compounding pharmacies that serve the veterinary profession are using inappropriate or ineffective vehicles in which the drug has been compounded, or the drug itself, purchased in bulk, is of a lower grade and possibly an ineffective product once compounded. Studies on the quality and efficacy of compounded drugs for use in veterinary patients are limited. However, of those studies that have been performed, serious questions are being raised over the bioavailability of the drug administered.

Administration by Injection (Parenteral Administration)

Patient Preparation
It would be admirable to prepare the skin surgically before making needle punctures to administer medications. Because such preparation is not practical, carefully part the hair and apply a high-quality skin antiseptic such as isopropyl alcohol. Place the needle directly on the prepared area, and thrust the needle through the skin. Although the use of antiseptics on the vial and skin is not highly effective, the procedure removes gross contamination and projects an image of professionalism. Before aspirating medications from multiple-dose vials, carefully wipe the rubber diaphragm stopper with the same antiseptic used on the skin. Observe this basic rule with all medication vials, even with modified live virus vaccines.

Subcutaneous Injection

Technique
Dogs and cats have abundant loose alveolar tissue and easily can accommodate large volumes of material in this subcutaneous space. The dorsal neck is seldom used for subcutaneous injections because the skin is somewhat more sensitive, causing some patients to move abruptly during administration. A wide surface area of skin and subcutaneous tissue over the dorsum from the shoulders to the lumbar region makes an ideal site for subcutaneous injections.

Administration of drugs, vaccines, and fluids by the subcutaneous route represents the most commonly used route of parenteral administration in dogs and cats. For small volumes (≤2 mL total), such as vaccines, a 22- to 25-gauge needle generally is used. The site most often used is the wide area of skin over the shoulders. The large subcutaneous space and the relative lack of sensitivity of skin at this location make it an ideal injection site. Cleaning of the skin with alcohol or other disinfectant generally is performed before injection. Several injection techniques are used. A common technique entails grasping a fold of skin with two fingers and the thumb of one hand. Gently lift the skin upward. Using the opposite hand, place the needle, with syringe attached, through the skin at a point below the opposite thumb. Aspiration before injection is not typically necessary when using this route of administration. After administration and on removal of the needle from the skin, gently pinch the injection site and hold it for a few seconds to prevent backflow of medication or vaccine onto the skin.

When larger volumes are to be administered—fluids in dehydrated dogs and cats—the skin directly over the shoulders is the injection site most commonly selected. Generally, only isotonic fluids are administered by the subcutaneous route. Depending on the patient’s
size, needles ranging from 16 to 22 gauge can be used. Because of the larger volumes of fluid involved, warming of the fluids before administration is recommended. Doing so can enhance significantly the patient’s tolerance for the displacement of skin during the period of administration and, in small patients, prevent hypothermia.

Depending on the rate of administration and breed of dog, relatively large volumes of fluid generally can be given in one location. Cats typically tolerate 10 to 20 mL/kg body mass in a single location. Large dogs can tolerate volumes greater than 200 mL of fluid in a single location. When administering large volumes, it is usually not necessary to use multiple injection sites for purposes of distributing the total fluid volume. Doing so actually may increase the risk of introducing cutaneous bacteria under the skin. Because the administration time required to deliver larger volumes is longer, and the injection needle will be placed in the skin for extended periods, it is appropriate to cleanse and rinse the skin carefully before actually inserting the needle. Isotonic, warmed fluids may be administered by large syringe or through an administration tube attached to a bag. Monitor skin tension and the patient’s comfort tolerance throughout the procedure.

Although fluid absorption begins almost immediately on subcutaneous administration of fluids, significant pressure caused by the bolus of fluid delivered can develop within the fluid pocket. On removal of the needle, firmly grasp the injection site with the thumb and forefinger for several seconds. The procedure is not complete until one has verified that back-leakage of fluid from the subcutaneous space onto the skin is not occurring. Depending on the patient’s hydration status and physical condition, fluid absorption may take from 6 to 8 hours.

**Note:** Not all parenteral medications can be administered safely by the subcutaneous route. When administering any compound by the subcutaneous route, verify that the product to be administered is approved for subcutaneous administration. Serious reactions, including abscess formation and tissue necrosis, can occur.

**Special Considerations**

The rate of absorption of fluid administered by the subcutaneous route largely depends on the patient’s hydration state and vascular and cardiac integrity. For that reason, the subcutaneous route is not recommended to manage patients in hypovolemic shock. Exceptions to this do exist—for example, when in a life-or-death situation access to a vein is simply not possible. Subcutaneous or intraosseous (see the following discussion) fluid administration may be the only option available.

**Implanted Subcutaneous Fluid Ports**

**Technique**

Recently, implantable subcutaneous ports* have been introduced for use in patients requiring regular administration of subcutaneous fluids at home. A 9-inch silicon tube is pre-placed under the skin and is sutured in place by a veterinarian. Objectively, this offers easy access to the subcutaneous space without the need for needle penetration. Owners simply attach a syringe or extension tube tip to the port and administer the appropriate volume of fluids at an appropriate rate and frequency.

**Special Considerations**

Because of the usual requirement for long-term placement of an implantable fluid administration tube, there is some risk of infection under the skin and around the incision site. Some cats do not tolerate the device.

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*GIF-Tube Single Implant Kit for Subcutaneous Fluid Administration, various models available. Phoenix, Arizona, [www.practivet.com](http://www.practivet.com) (owner instruction guide is also available).
**Intramuscular Injection**

*Patient Preparation*
None required.

*Technique*
Because the tightly packed muscular tissue cannot expand and accommodate large volumes of injectables without trauma, medications given by the intramuscular route should be small in volume. These medications are often depot materials that are poorly soluble, and some may be mildly irritating. Unless the animal is extremely thin, give injections into the lumbodorsal muscles on either side of the dorsal processes of the vertebral column.

After proper preparation of the skin, insert the needle through the skin at a slight angle (if the animal is thin) or perpendicularly (if the animal is obese). When injecting any medication by a route other than the intravenous one, it is imperative to retract the plunger of the syringe before injecting to be certain that a vein was not entered by mistake. This is especially crucial with oil suspension, microcrystalline suspension, or potent-dose medications.

*Special Considerations*
Never give intramuscular injections in the neck because of the fibrous sheaths there and the complications that may occur. Also, intramuscular injections into muscles of the rear legs can cause severe pain, lameness, and occasionally peroneal nerve paralysis because of local nerve involvement.

**Intradermal Injection**

*Patient Preparation*

Intracutaneous (or intradermal) injections are used for diagnostic testing purposes. Prepare the skin by carefully clipping the hair with a No. 40 clipper blade. If the skin surface is dirty, gently clean it with a moist towel. Scrubbing and disinfection are contraindicated because they may produce iatrogenic trauma and inflammation, which interfere with the test.

*Technique*
Stretch the skin by lifting a fold, and use a 25- to 27-gauge intradermal needle attached to a 1-mL tuberculin syringe. Insert the point of the needle, bevel up, in a forward lifting motion as if to pick up the skin with the needle tip. Advance the needle while pushing the syringe (levered) downward until the bevel is completely within the skin. Inject a bleb of 0.05 to 0.10 mL of fluid. If the procedure is done correctly, the small bleb will appear translucent. Intradermal injections generally are used in patients subjected to intradermal skin testing for allergenic antigens. Administration of compounds by the intradermal technique is not necessarily simple. Inadvertent administration of medications into the subcutaneous tissues is easy when attempting intradermal injection. For that reason, specific training and experience are recommended before attempting intradermal skin testing of allergic patients.

**Transdermal (Needle-Free) Administration**

*Patient Preparation*
None required.

*Technique*
Intradermal administration of vaccine and drugs in veterinary and human medicine largely has been limited to the complexities of accurately delivering the desired dose into, and not under, the skin. In 2004 a transdermal administration system* was introduced for cats (recombinant feline leukemia virus [FeLV] vaccine) that was designed after a similar device.

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*Vet-Jet Transdermal Administration System, Merial, Duluth, Georgia.*
used in human (pediatric) medicine. Recently the transdermal administration system used for administration of the recombinant FeLV vaccine has been re-designed. This same administration system is now used for the transdermal administration of the oral melanoma vaccine. The transdermal administration system consistently delivers a precise volume of vaccine into the skin, subcutaneous tissues, and muscle. Use of the transdermal administration system should only be used to administer those vaccines approved for this method of delivery.

Special Considerations
Administration of vaccine using the transdermal administration system requires training to understand proper procedure for loading and administering vaccine. At this writing, sale of the transdermal administration system for delivery of the canine oral melanoma vaccine is limited to select specialists in veterinary medicine.

INTRAVENOUS INJECTION
See Section 1.

INTRAOSSEOUS ADMINISTRATION
See Section 1.

Additional Reading

BANDAGING TECHNIQUES
See Section 1.

BLOOD PRESSURE MEASUREMENT: INDIRECT

Patient Preparation
None required.

Technique
Generally, two techniques are used. Oscillometric blood pressure (BP) measurement entails use of an automated recording system. A cuff is applied to the base of the tail or a distal limb for access to an artery. This technique generally is regarded as being most accurate in dogs. When oscillometric BP measurements are performed in dogs, the patient should be in lateral recumbency. This places the cuff at approximately the same level as the heart. In cats the patient generally remains in sternal recumbency (and minimally restrained). Most patients experience a brief acclimation period to the cuff placement. For this reason, at least three to five separate readings are obtained at 1- to 2-minute intervals. This technique can be used on awake or anesthetized patients (Figure 4-8).

The Doppler-ultrasonic flow detection system is most accurate in cats for measuring systolic BP. Again, the ventral tail base or a dorsal pedal artery (hindlimb) or the superficial palmar arterial arch (forelimb) can be used. Apply and inflate an occluding cuff. The readings are obtained by a transducer as the pressure on the cuff is reduced. Caution is recommended in interpreting results from dogs that are reported as hypertensive but have no overt clinical disease. The higher reported occurrence of falsely elevated BP in normotensive dogs measured by this method justifies additional scrutiny when interpreting Doppler BP results in dogs.

www.ajlobby.com
Clinically, the most common use of indirect BP measurement is in assessing cats for the presence (or absence) of systemic hypertension caused by renal insufficiency or hyperthyroidism (thyrotoxicosis). A common finding among untreated hypertensive cats is retinal detachment and blindness. Early detection and therapeutic intervention (e.g., enalapril and or amlodipine) is critical. In dogs, BP measurement is indicated in patients with chronic renal insufficiency and/or protein-losing nephropathy, hyperadrenocorticism, and diabetes mellitus. In veterinary medicine, interpretation of BP centers on the systolic BP reading, not the diastolic reading (Table 4-2).

**Additional Reading**

**DIAGNOSTIC SAMPLE COLLECTION TECHNIQUES**

**Bacterial Culture**
In previous editions of this book, methods of preparing and using selective culture media as well as identifying specific isolates was described. However, technologic advances in microbiology have largely replaced older methods of identifying bacterial isolates in practice. Furthermore, the diverse array of bacterial pathogens, requirements for unique culture media, the risk of sample contamination, and the need for subjective interpretation of

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**TABLE 4-2** Systolic Blood Pressure

<table>
<thead>
<tr>
<th>Normal</th>
<th>Hypertension</th>
<th>Hypotension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dog and cat</td>
<td>100-150 mm Hg</td>
<td>&gt;160 mm Hg</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt;180 mm Hg (high risk)</td>
</tr>
</tbody>
</table>

Figure 4-8: Oscillometric blood pressure measurement in a cat.
results dictate that even routine bacterial cultures and identification are best reserved for the commercial laboratory equipped to carry out these increasingly complex procedures and experienced in doing so.

What follows are fundamental methods and techniques used to properly collect diagnostic specimens and the appropriate methods for transporting samples to a laboratory in order for the best possible diagnostic result to be obtained.

**Direct Microscopic Examination**
Before actually collecting and submitting a sample to a laboratory for bacterial culture, it is appropriate (whenever feasible to do so) to prepare, stain, and examine, under direct microscopy, exudates or fluid from the suspect material or tissue. Staining the air-dried sample with a rapid Romanowsky-type stain (e.g., Diff-Quik stain) or a Gram stain may reveal evidence of neutrophilic inflammation (neutrophilia, especially with a left shift) and occasionally degenerative neutrophils with intracellular bacteria visible. These findings greatly facilitate patient management by documenting the immediate need for interventive empiric antimicrobial therapy until definitive culture and antimicrobial susceptibility results are obtained. The absence of cytologic evidence of bacterial infection does not rule out the possibility that the patient is infected or bacteremic (Table 4-3).

**Test Considerations**
Collecting diagnostic samples for bacterial culture should be attempted as early in the disease process as possible. It is also critical to accomplish the sample collection under aseptic conditions. It is appropriate, therefore, to perform a surgical scrub of the skin or tissue

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**Table 4-3: Common Bacterial Culture Results**

<table>
<thead>
<tr>
<th>Site</th>
<th>Commensals</th>
<th>Pathogens</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EXTERNAL EAR CANAL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dog</td>
<td><em>Malassezia, Clostridium,</em> <em>Staphylococcus</em> (a few), <em>Bacillus</em> (a few); never <em>Streptococcus,</em> <em>Pseudomonas,</em> or <em>Proteus</em></td>
<td>Many <em>Staphylococcus</em> and <em>Malassezia</em> together; <em>Pseudomonas,</em> <em>Proteus,</em> <em>Streptococcus,</em> <em>Escherichia coli</em></td>
</tr>
<tr>
<td>Cat</td>
<td>Not documented</td>
<td><em>Staphylococcus aureus,</em> <em>β</em>-hemolytic <em>streptococci,</em> <em>Pasteurella,</em> <em>Pseudomonas,</em> <em>Proteus,</em> <em>E. coli,</em> <em>Malassezia</em></td>
</tr>
<tr>
<td><strong>SKIN</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dog</td>
<td><em>Micrococcus,</em> <em>Clostridium,</em> diphtheroids, <em>Staphylococcus epidermidis,</em> <em>Corynebacterium,</em> <em>Malassezia</em></td>
<td><em>S. aureus</em> (coagulase positive), <em>Proteus,</em> <em>Pseudomonas,</em> <em>E. coli</em></td>
</tr>
<tr>
<td>Cat</td>
<td><em>Micrococcus,</em> <em>Streptococcus,</em> <em>S. aureus,</em> <em>S. epidermidis</em></td>
<td><em>S. aureus,</em> <em>Pasteurella multocida,</em> <em>Bacteroides,</em> <em>Fusobacterium,</em> <em>hemolytic streptococci</em></td>
</tr>
<tr>
<td>Conjunctiva</td>
<td><em>Staphylococcus,</em> <em>Streptococcus,</em> <em>Bacillus,</em> <em>Corynebacterium,</em> diphtheroids, <em>Neisseria,</em> <em>Pseudomonas</em></td>
<td><em>S. aureus,</em> <em>Bacillus,</em> <em>Pseudomonas,</em> <em>E. coli,</em> <em>Aspergillus</em></td>
</tr>
</tbody>
</table>

Continued
from which the sample is to be collected in advance. This is especially true for tissue biopsies and fluid samples collected by needle aspiration through intact skin. Failing to adequately prepare the collection site can result in significant contamination and complicate diagnostic interpretation of results.

In addition, it is recommended to collect the diagnostic sample before the administration of antibiotics in order to minimize the risk of false-negative culture results. In the event antimicrobials have been administered to a patient with a suspected infection, and that is not responding to treatment, discontinuing treatment for 48 hours before attempting sample collection is generally recommended.

Collection of an adequate amount, or volume (fluid), is equally important in obtaining meaningful result. For example, a single sterile cotton-tipped swab of contaminated tissue should be considered inadequate sampling and inappropriate for any patient. Multiple specimens are always recommended when feasible. Also, biopsy material, surgically removed tissue, and several milliliters of fluid (e.g., urine) should be collected and placed in a sterile container that can be appropriately sealed (leak-proof container) before transport. A "clean catch" of urine in a "clean cup" is not appropriate.

Inexpensive commercial containers specifically designed for the transport of infectious material are readily available today and should be used. Many containers designed to hold bacterial samples contain buffered, nonnutritive transport media to sustain the growth of pathogenic bacteria yet minimize overgrowth of bacterial contaminants during the time required to transport the sample. Most commercial laboratories provide appropriate containment devices for the transport of bacterial samples.

**Collection Technique and Sample Transport**

Because most diagnostic specimens collected for bacterial culture are submitted to commercial laboratories for bacterial isolation, identification, and antimicrobial susceptibility testing, it is important to prepare the sample properly for shipping.

Special transport media are generally not required for routine aerobic culture specimens as long as the sample can remain moist and relatively cool and the sample can

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**TABLE 4-3  Common Bacterial Culture Results—Cont’d**

<table>
<thead>
<tr>
<th>Site</th>
<th>Commensals</th>
<th>Pathogens</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vagina</td>
<td><em>Staphylococcus, Streptococcus, Enterococcus, Corynebacterium, E. coli, Haemophilus, Pseudomonas, Peptostreptococcus, Bacteroides</em></td>
<td><em>Brucella canis; pure culture of organism (especially <em>E. coli, Staphylococcus, Pseudomonas</em>) when accompanied by tissue reaction at vaginal cytology</em></td>
</tr>
<tr>
<td>Urine</td>
<td>&lt;1000* organisms per milliliter; presence of several organisms suggests contamination</td>
<td>More than 100,000* organisms per milliliter and often pure culture; <em>E. coli, enterobacteria, Klebsiella, Proteus, Pseudomonas aeruginosa, P. multocida, Staphylococcus, Streptococcus</em></td>
</tr>
</tbody>
</table>

*Absolute numbers of bacteria depend on the collection technique.
be inoculated onto culture medium within 3 to 4 hours only. For samples that must be shipped overnight to a laboratory, it is imperative that the specimen be kept cool (not frozen) and moist. Elevated temperatures during shipping contribute to bacterial overgrowth of nonpathogenic bacteria, making isolation and identification of disease-producing organisms difficult. Special transport media may be required. Contact the individual laboratory regarding information pertaining to shipping of specimens for bacterial culture.

Specimens submitted for anaerobic culture need to be inoculated onto culture media within minutes after collection. Although special anaerobic transport media are available, they may not be well suited for extended shipping times (>4 hours).

**Urine**

Among the most frequently tested fluids for bacteria, urine supports the growth of several types of bacteria. Therefore it is necessary that the genitalia be cleaned before collection of urine (free-catch specimen) or cystocentesis (preferred). Use of a urinary catheter to collect urine is likely to introduce urethral bacteria and may result in false-positive culture results. Bacteria will survive for only a limited time in urine. Samples collected must be sealed, and unless processed within 2 hours the sample must be refrigerated. Samples held for longer than 8 hours may not contain viable bacteria. If extended transportation times are required to reach a laboratory, a urine reservation tube (Vacutainer Brand Urine Transportation System, BD, Franklin Lakes, New Jersey) will allow storage for up to 48 hours at room temperature (Table 4-4).

**Exudates and Transudates**

Collection from fluid filled compartments (e.g., abscesses, seromas) requires collection with a needle and syringe. The maximum quantity possible should be collected and submitted. The skin or tissue overlying the area from which the sample is to be collected should be surgically prepared. If it becomes necessary to flush an open lesion (or perform tracheo-transtracheal aspiration or bronchoalveolar lavage [BAL]), it is recommended that a buffered solution of sterile Ringer’s lactate be used. Use of fluids that contain preservative may actually inhibit the growth of bacteria.

**Feces**

If it is necessary to submit fecal material for specific bacterial isolation, at least 2 to 3 g of feces should be submitted. A single cotton-tipped swab inserted rectally is unlikely to yield meaningful results. Multiple (up to three) samples are recommended when attempting to isolate specific pathogens (e.g., *Salmonella*). Samples should be submitted in a sealed, leak-proof container (always appreciated by the lab). The containerized sample should refrigerated if there is a significant delay (several hours) involved in submission to the laboratory.

**Blood**

Confirmation of the presence of bacteria in the blood (bacteremia) can be difficult and requires some patient preparation before collection of a series of samples. In addition, samples should be collected only in vials clearly marked for the collection of blood. Furthermore, there are several reasons for an infected patient to have a negative blood culture result—for example, prior or concurrent antimicrobial therapy, chronic (low-grade) infection, and intermittent shedding of bacteria into blood. Sample volume, numbers of samples submitted, skin preparation, and timing of collections are variables that can directly affect results.

Clip and surgically prepare the skin over the cephalic, recurrent tarsal, and/or jugular veins. Do not draw blood for culture through an indwelling intravenous or intraarterial catheter. Collection vials are available for aerobic and anaerobic bacterial culture. It is generally recommended that three blood samples be collected from separate veins over a 24-hour period. There is no advantage to collecting arterial blood. It has been suggested.
### TABLE 4-4 Interpretation of Quantitative Urine Cultures in Dogs and Cats*

<table>
<thead>
<tr>
<th>Collection Method</th>
<th>Dogs</th>
<th>Cats</th>
<th>Dogs</th>
<th>Cats</th>
<th>Dogs</th>
<th>Cats</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cystocentesis</td>
<td>≥100</td>
<td>≥100</td>
<td>100-1000</td>
<td>100-1000</td>
<td>≤100</td>
<td>≤100</td>
</tr>
<tr>
<td>Catheterization</td>
<td>≥10,000</td>
<td>≥1000</td>
<td>1000-10,000</td>
<td>100-1000</td>
<td>≤1000</td>
<td>≤100</td>
</tr>
<tr>
<td>Voluntary voiding</td>
<td>≥100,000†</td>
<td>≥10,000</td>
<td>10,000-90,000</td>
<td>1000-10,000</td>
<td>≤10,000</td>
<td>≤1000</td>
</tr>
<tr>
<td>Manual compression</td>
<td>≥100,000†</td>
<td>≥10,000</td>
<td>10,000-90,000</td>
<td>1000-10,000</td>
<td>≤10,000</td>
<td>≤1000</td>
</tr>
</tbody>
</table>

*The data represent generalities. On occasion, bacterial urinary tract infections may be detected in dogs and cats with the fewer organisms (i.e., false-negative results).

†Caution: Because contamination of midstream samples may result in colony counts of 10,000/mL or more in some dogs (i.e., false-positive results), they should not be used for routine diagnostic culture of urine from dogs.

From Osborne CA, Finco DR: *Canine and feline nephrology and urology*, Baltimore, 1995, Williams & Wilkins.
that samples collected during times when the patient is febrile may improve the likelihood of isolating bacteria. The volume of blood collected is determined by the size of the patient, the collection vials (adult, pediatric, infant) used, as well as the laboratory equipment used to propagate the culture. In addition to adult human blood culture collection vials (10 ml), pediatric blood collection vials (5 to 10 mL) and infant collection vials (0.5 to 1.0 mL) are available.

It is appropriate that sterile technique be adhered to during collection of all samples. This includes the use of sterile gloves by the individual collecting the sample. Once blood has been collected, air should not be allowed to enter the collection vial. The vial should be gently inverted (never shaken) two to four times. Vials may be maintained at room temperature (the laboratory maintains samples at 37° C).

The opportunity to submit complementary cultures (e.g., from urine) from patients in which blood cultures are being collected can help to confirm the infecting bacteria and may lead to identification of a likely source (Boxes 4-2 and 4-3).

**Additional Reading**


Osborne CA, Finco DR: Canine and feline nephrology and urology, Baltimore, 1997, Williams & Wilkins.


**Fungal Culture**

Diagnostic fungal cultures depend on selection of the most appropriate culture site and proper collection technique. Fungal cultures are mostly commonly pursued in patients suspected of having superficial fungal infections of the hair, skin, and nails (dermatophytosis).
Samples collected from patients suspected of having fungal infections of the nasal cavity (e.g., aspergillosis) or systemic (also called “deep”) mycotic infections (e.g., histoplasmosis, cryptococcosis) are usually assessed by cytopathology or serology (see Section 5) or with tissue biopsy and histopathology involving special stains.

Direct Microscopic Examination

Direct cytologic assessment of samples from patients suspected of having fungal infections is always indicated. You certainly get credit for trying! However, experience in recognizing diagnostic elements of individual fungi and spores is essential, as is the availability of special stains for wet mount (10% potassium hydroxide) cytopathology.

Collection Technique and Sample Transport

Skin and Hair

Scrapings of skin and plucked hair shafts are commonly selected for fungal culture. The area of skin and hair to be sampled should be cleaned with 70% alcohol. Iodine-based soaps and solutions should not be used. Hair shafts, particularly those immediately adjacent to the lesion, are removed from skin with a sterile hemostat. Skin scrapings can be collected with a sterile surgical blade or the edge of a clean (unused) microscope slide. Scrapings from healthy, normal-appearing skin as well as abnormal skin should be collected. Skin biopsy may be required if results of attempts to culture hair and skin scrapings are negative. Sterile cotton-tipped swabs should not be used to collect samples for fungal culture.

Hair and skin scrapings can be placed directly into a sterile, dry container without need for any type of media as long as the sample can be processed within hours. Refrigeration is generally not required. If transport times are extended, it is reasonable to place samples in a vial containing bacterial transport medium and refrigerate for up to 15 hours. Samples should never be frozen.

In-Hospital Fungal Culture

Skin and hair samples from patients suspected of having superficial fungal infections can be inoculated directly on a commercially available substrate called Dermatophyte Test Medium (DTM). Because samples can remain at room temperature and do not require special handling, the use of DTM is ideal for in-hospital use. The medium contains phenol red as a pH indicator. If a dermatophyte is present, characteristic colony morphology will be observed and the medium underlying the colonies will turn red. Vials are unreliable after 2 weeks; color change noted 2 weeks or more after inoculation of the DTM should be disregarded.

Wood Light

Ultraviolet light filtered through nickel oxide produces a beam called Wood light. If an animal is taken into a dark room and its hair and skin are exposed to a Wood lamp, fluorescence may occur for several reasons. Hair shafts affected by some species of Microsporum fluoresce a bright yellow-green (like the color of a fluorescing watch face). However, iodide medications, petroleum, soap, dyes, bacteria, and even keratin may produce purple-, blue-, or yellow-colored fluorescence. The positive fungal fluorescence is a valuable aid in selecting affected hairs for culture inoculation. However, a negative fluorescence does not preclude a possible diagnosis of fungal infection. False-negative and false-positive interpretations are common.

Additional Reading

VIRAL TESTING

Direct Microscopic Examination
Microscopic examination of fluid or tissue samples from patients suspected of having a viral infection is unlikely to contribute to the diagnosis. Because viruses are small and generally intracellular particles, neither light microscopy nor viral culture techniques are used in the practice setting. Some commercial and academic laboratories do offer electron microscopy of tissue (cells), fluids, or feces from infected patients, which may allow for direct visualization of virus particles. Results depend on the quality of the sample evaluated, the type of equipment available, and the experience of the individual performing the microscopy.

Test Considerations
Various laboratory techniques are currently available for the identification of viral infections in dogs and cats. Excellent qualitative testing platforms are commercially available for in-practice use. Molecular diagnostic tests, viral culture, histopathology, and serology, all of which are routinely available to veterinarians, require that samples be submitted to commercial laboratories for assessment.

In-Hospital Testing
Among the in-hospital test systems used to identify virus, the enzyme-linked immunosorbent assay (ELISA) is the most common testing platform used. ELISA testing can be performed quickly (minutes) with little or no patient preparation and with relatively high sensitivity and specificity. Virus (antigen) detection tests are available as point-of-care tests for FeLV antigen in blood or serum and canine parvovirus (CPV) antigen in feces. In addition, these point-of-care tests for viral infections are capable of identifying patients that have not been exposed, enabling the clinician to rule out infection and viral shedding.

Test sensitivity refers to the likelihood that a patient with known infection will have a positive test result (a test with high sensitivity is expected to have few false-positive results).

Test specificity refers to the likelihood that a patient that is free of the infection will have a negative test result (a test with high specificity is expected to have few false-negative results).

In addition, many commercial and point-of-care nonquantitative serologic assays are available that detect antibody to many of the viruses that affect dogs and cats. However, the positive predictive value of antibody tests is typically lower than that of antigen tests. For example, a positive antibody test result to a particular viral pathogen typically does not constitute a diagnosis of infection, especially in the absence of clinical signs. It may merely reflect recent vaccination (e.g., feline immunodeficiency virus). On the other hand, a negative antibody test result generally does indicate that the patient has had no prior exposure to the virus (or vaccine).

Laboratory-Based Tests
Serology refers to the use of serum to detect the concentration of antibody and is widely used in veterinary medicine. The value of antibody titers in diagnosing a viral infection is dependent on a number of factors, including the infecting virus, vaccination history, and time since exposure. Use of acute and convalescent antibody titers in a patient suspected of having an acute viral infection can be a reliable diagnostic tool if a fourfold or greater increase in titer can be demonstrated over 2 to 4 weeks. Acute and convalescent viral titers in individual patients are rarely performed in veterinary medicine.

Virus isolation, however, is a valuable diagnostic tool that is underused in veterinary medicine, perhaps because of the limited number of commercial and university laboratories that provide viral isolation services and the increased availability of molecular diagnostic testing services. Diagnosis of viral upper respiratory infection in cats (herpesvirus 1 and/or calicivirus) is perhaps among the situations for which virus isolation can be most useful, especially in cluster households where many shedding carrier cats exist and kittens may be at risk.
To obtain a sample for viral isolation from the oral cavity of a cat, quickly insert a sterile cotton swab into the oral cavity to the level of the tonsil or oropharynx. By rolling the swab across the epithelium, it is possible to harvest cells and virus from infected cats. Immediately place the swab into a virus transport medium (usually provided by the laboratory). Antibiotics added to the solution prevent bacterial overgrowth of the sample. For short-term transit (5 days or less), hold specimens for viral isolation at 4° C rather than frozen. On reaching the laboratory, the specimen will be inoculated into a suitable tissue culture. Within a few days it is usually possible to establish, based on the cytopathic effect on the tissue culture, whether a virus infection is present. Fluorescent antibody testing can be done subsequently to confirm the isolate.

Although availability is limited, direct assessment of specimens (e.g., feces for CPV or canine or feline coronavirus) can be accomplished by electron microscopy. These methods can be useful for infections in which the virus concentration in the specimen reaches $10^6$ to $10^7$ organisms per milliliter. Specimens such as feces, vesicle fluid, brain tissue, urine, or serum can be submitted for electron microscopy.

Tissue specimens and exfoliative cytologic preparations can be submitted for viral identification by histopathology, immunohistochemistry, and direct fluorescent antibody testing. Such testing has limited application in patients with active disease because of the limited availability of these types of services and the time required for samples to be processed and reviewed by a pathologist. These tests can be particularly useful in postmortem diagnostics when multiple animals are potentially at risk.

*Molecular diagnostics* refers to the use of nucleic acid–based tests for the detection of viral DNA or RNA. Polymerase chain reaction (PCR) is a laboratory technology that offers exceptional test sensitivity. Through its ability to amplify trace amounts of DNA or RNA from pathogenic organisms millions of times, PCR facilitates identification of the “target” sequence of nucleic acid and therefore the infecting organism. This technology is also available commercially for the detection of DNA from selected bacteria and rickettsiae. PCR technology is particularly useful in the very early stages of a viral infection, when the level of antibody has not yet reached levels that are detectable with conventional antibody tests. In addition, PCR testing may detect healthy virus carrier animals that pose a risk to a larger population of susceptible animals yet cannot be identified by conventional virus isolation or identification technologies. It should be noted, however, that PCR technology is still subject to false-positive and false-negative test results. Therefore such testing is not necessarily indicated as a primary or exclusive test method for an individual patient.

**Collection Technique and Sample Transport**

**Serology**

Serum, plasma, or other fluids (e.g., cerebrospinal fluid [CSF]) can be tested for the presence of antibodies to selected pathogenic viruses. Whole blood samples should be allowed to thoroughly clot and retract (or the sample should be centrifuged) before serum is collected. Samples are submitted in a leak-proof vial. Refrigeration is appropriate for samples that must be held for several hours before testing.

**Histology and Immunohistochemistry**

Samples are limited to tissue obtained during surgical biopsy. As with conventional histopathology, samples (no more than 5.0 mm thick) should be placed in 10% buffered formalin and submitted in a leak-proof vial. It is recommended that the volume of formalin used be at least 10 times greater than the tissue sample submitted.

**Fluorescent Antibody Testing**

Testing can be performed on tissues collected during surgical biopsy or from tissue impressions (exfoliative) made from tissue imprints on a clean microscope slide. It is recommended that tissue impressions on slides be fixed in alcohol or acetone before submission. Fresh tissue is submitted on wet (not dry) ice and is not subjected to formalin fixation.
**Electron Microscopy**

Small amounts of tissue suitable for electron microscopy should be no larger than 1 × 2 mm thick. Fixation in 2% to 4% glutaraldehyde for 24 hours at 20°C is required. Feces and body fluids collected for electron microscopy should be submitted fresh, not frozen or fixed in preservative. If shipping is required, feces and body fluids may be refrigerated or shipped on wet ice. Samples should be viable for 48 to 72 hours.

**Virus Isolation**

Sterile swabs may be used to collect samples for viral culture and isolation. Samples should be inoculated into a sealed vial containing viral transport medium (usually provided by the laboratory). Samples should not be frozen or fixed in preservative.

**Polymerase Chain Reaction Testing**

Laboratories offering PCR testing typically accept serum or anticoagulated (ethylenediaminetetraacetic acid [EDTA]) whole blood in leak-proof vials. Samples should be refrigerated and shipped on wet ice. Samples should not be frozen.

**Blood Collection Techniques**

In most instances, a 3- to 5-mL sample of anticoagulated whole blood is adequate for routine hematology; some laboratories will accept as little as 1 mL. For routine biochemical analyses, the volume of serum requested can vary from 1 to 2 mL, depending on the number and type of tests requested. Plan ahead which samples are required to prevent the need for further venipuncture at a later time. In small dogs and cats, using the jugular veins facilitates collection of an adequate volume of blood. If smaller samples are required, the cephalic, lateral saphenous, or medial saphenous vein can be used for sample collection. Do not use the jugular vein if a coagulopathy is suspected, as hemorrhage may be difficult to control after venipuncture.

**Patient Preparation**

For successful venipuncture, proper restraint of the animal is important. Details for the proper restraint for various venipuncture locations are discussed with each specific topic throughout this text. The patient must remain comfortable yet relatively motionless to avoid iatrogenic vessel laceration. Stretch the skin tightly over the selected vessel without causing vascular occlusion to help anchor the vessel in place during penetration by the needle.

**Technique**

The specific venipuncture will vary somewhat depending on the specific vein selected. The following sections describe venipuncture technique for each of four commonly accessed veins: the cephalic vein, jugular vein, lateral saphenous vein, and medial saphenous vein.

**Cephalic Venipuncture**

To restrain a dog or cat for venipuncture of the cephalic vein, place the dog or cat on the table, sitting or in sternal recumbency. If the right vein is to be tapped or catheterized, the assistant should stand on the left side of the animal and place the left arm or hand under the animal’s chin to immobilize the head and neck. The assistant should reach across the animal and grasp the leg just behind and distal to the right elbow joint. The assistant should use the thumb to occlude and rotate the cephalic vein laterally while the palm of the hand holds the elbow in an immobilized and extended position. Make sure that the animal stays on the table if struggling occurs. The person performing the venipuncture then grasps the leg at the metacarpal region and begins the venipuncture on the medial aspect of the leg, just adjacent to the cephalic vein proximal to the carpus.

**Jugular Venipuncture**

For a jugular venipuncture in the dog, place the patient in sternal recumbency, with the hands of the assistant placed around the patient’s muzzle to extend the neck and nose dorsally toward the ceiling. In short-coated dogs, the jugular vein usually can be seen coursing...
from the ramus of the mandible to the thoracic inlet in the jugular furrow. The vessel may be more difficult to visualize in dogs with long hair coats or if excessive subcutaneous fat or skin is present. The person performing the venipuncture should place the thumb of the nondominant hand across the jugular vein in the thoracic inlet or proximal to the thoracic inlet to occlude venous drainage from the vessel and allow it to fill. With the dominant hand, the person performing the venipuncture should insert the needle and syringe or Vacutainer (BD, Franklin Lakes, New Jersey) into the vessel at a 15- to 30-degree angle to perform the venipuncture.

For smaller and very large animals, the jugular vein also can be tapped by placing the patient in lateral recumbency. The assistant should pull the animal’s front legs caudally and extend the head and neck so that the jugular vein can be visualized. The venipuncture then can be performed as previously described. A jugular venipuncture is contraindicated in patients with thrombocytopenia or vitamin K–antagonist rodenticide intoxication.

Place cats in sternal recumbency. The assistant should stand behind the patient so that the patient cannot back away from the needle during the venipuncture. The assistant should extend the cat’s head and neck dorsally while restraining the cat’s front legs with the other hand. The cat’s fur can be clipped or moistened with isopropyl alcohol to aid in visualization of the jugular vein as it stands up in the jugular furrow. The person performing the venipuncture should occlude the vessel at the thoracic inlet and insert the needle or Vacutainer apparatus into the vessel as previously described to withdraw the blood sample. Alternately, place the cat in lateral recumbency as described in the previous paragraph.

**Lateral Saphenous Venipuncture**
To perform a lateral saphenous venipuncture, place the patient in lateral recumbency. The lateral saphenous vein can be visualized on the lateral portion of the stifle, just proximal to the tarsus. The assistant should extend the hindlimb and occlude the lateral saphenous vein just proximal and caudal to the tarsus. The person performing the venipuncture should grasp the distal portion of the patient’s limb with the nondominant hand and insert the needle or Vacutainer apparatus with the dominant hand to withdraw the blood sample.

**Medial Saphenous Venipuncture**
To perform a medial saphenous venipuncture, place the patient in lateral recumbency. Move the top hindlimb cranially or caudally to allow visualization of the medial saphenous vein on the medial aspect of the tibia and fibula. The assistant should scruff the patient, if the patient is small, or should place the forearm over the patient’s neck to prevent the patient from getting up during the procedure. With the other hand, the assistant should occlude the medial saphenous vein in the inguinal region. The person performing the medial saphenous venipuncture should grasp the paw or hock of the limb and pull the skin taut to prevent the vessel from rolling away from the needle. The fur may be clipped or moistened with isopropyl alcohol to aid in visualization of the vessel. The needle or Vacutainer apparatus can be inserted into the vessel at a 15- to 30-degree angle to withdraw the blood sample.

**Special Considerations**
Incorrect proportions of blood to anticoagulant may result in water shifts between plasma and red blood cells (RBCs). Such shifts may alter the packed cell volume (PCV), especially when small amounts of blood are added to tubes prepared with volumes of anticoagulant sufficient for much larger volumes of blood. Erroneous laboratory results also may be obtained when small volumes of blood are placed in a relatively large container. Evaporation of plasma water and adherence of the cells to the surface of the container can produce artifactual changes in hematologic results.

Refrigerate liquid blood mixed with anticoagulant after collection if the sample is to be held before being transported to a laboratory. White blood cell (WBC) and RBC counts, PCV, and hemoglobin level can be measured within 24 hours of sample
collection. Platelet counts, however, should be done within 1 hour of collection. Dried, unfixed blood smears can be stained with most conventional stains 24 to 48 hours after being made. If a considerable delay is anticipated between the time that the blood smear is made and the staining process, the blood smear should be fixed by immersion in absolute methanol for at least 5 minutes. Blood smears fixed by this method are stable indefinitely.

Never place unfixed blood smears in a refrigerator because condensation forming after the smear is removed from the refrigerator will ruin the blood smear and make it unusable for cytologic evaluation. Take care to leave unfixed blood smears face down on a countertop or in a closed box. Special stains, such as peroxidase, may require fresh blood films.

**Routine Hematologic Testing (See Also Section 5)**

The anticoagulant of choice for hematologic testing is EDTA. Heparin is especially to be avoided if blood films are to be made from blood mixed with anticoagulant because contact with whole blood will distort the morphology of cells significantly. Heparin is acceptable for most procedures requiring blood plasma. The anticoagulant effect of heparin is transitory. Specimens still may clot after 2 to 3 days.

Make blood films immediately after collection because cell morphology rapidly deteriorates after sample collection. Although blood films can be made after introducing blood to EDTA, a better practice is to make blood smears (films) immediately from the collection needle before the blood comes in contact with any anticoagulant. Never use blood exposed to heparin to make blood smears.

Incorrect proportions of blood to anticoagulant may result in water shifts between plasma and RBCs. Such shifts may alter the PCV, especially when small amounts of blood are added to tubes prepared with volumes of anticoagulant sufficient for much larger volumes of blood. Erroneous laboratory results also may be obtained when small volumes of blood are placed in a relatively large container. Evaporation of plasma water and adherence of the cells to the surface of the container can produce artifactual changes in hematologic results.

Refrigerate liquid blood mixed with anticoagulant after collection if there is a delay in making the laboratory determinations. WBC and RBC counts, PCV, and hemoglobin level can be measured within 24 hours of sample collection. Platelet counts, however, should be done within 1 hour of collection. Dried, unfixed blood smears can be stained with most conventional stains 24 to 48 hours after being made. If a considerable delay is anticipated between the time that the blood smear is made and the staining process, the blood smear should be fixed by immersion in absolute methanol for at least 5 minutes. Blood smears fixed by this method are stable indefinitely. Never place unfixed blood smears in a refrigerator because condensation forming after the smear is removed from the refrigerator will ruin the blood smear and make it unusable for cytologic evaluation. Take care to leave unfixed blood smears face down on a countertop or in a closed box. Special stains, such as peroxidase, may require fresh blood films.

**Routine Biochemistry Testing (See Also Section 5)**

**Patient Preparation**

Prepare the selected vein as described earlier.

**Technique**

Most clinical chemistry procedures are performed on serum. The serum is obtained by collecting blood without any anticoagulant and allowing the blood to clot in a clean, dry tube. Separate serum from cells within 45 minutes of sample collection (venipuncture). Special vacuum vials are available that produce a gel barrier between the clot and the serum (serum separator tubes) which avoid the need to draw off the serum into a separate vial. Clotting of the blood and retraction of the clot occur best and maximum yields of serum are obtained at room or body temperature. Refrigeration of the sample delays clot retraction. Some samples clot and retract faster than others.
Special Considerations

If a serum separator tube is not used, it is recommended to free the clot from the walls of the container by rimming with an applicator stick. After the clot is freed, allow clot retraction to occur, and then centrifuge and draw off the clear supernatant serum using a pipette or suction bulb. Allow whole blood samples to completely clot before attempting to remove serum. Failing to do so may result in a mixture of plasma and serum in the submitted sample. Serum yield is usually one third of the whole blood volume. Patients that are hypovolemic or dehydrated can have a significantly lower serum yield.

Many clinical chemistry procedures can be performed on plasma and on serum. The advantage of using plasma is that separation of cells can be accomplished immediately after centrifugation or sedimentation, without the need to wait for clot formation and retraction. The disadvantage of plasma is that the presence of the anticoagulant interferes with many of the chemistry assay procedures. Plasma is less clear than serum, which may be an additional disadvantage for colorimetric assays. Plasma and serum are virtually identical in chemical composition except that plasma has fibrinogen and the anticoagulant. For many procedures in which plasma or whole blood is to be used, heparin is the anticoagulant of choice. Heparinized blood is the only acceptable specimen for blood pH and blood gas analyses. Although blood containing EDTA is acceptable for certain chemical procedures, it cannot be used for determination of plasma electrolytes because it contributes to and sequesters them from the specimen. In addition, EDTA can interfere with alkaline phosphatase levels, decrease total carbon dioxide, and elevate blood nonprotein nitrogen.

Refer to the Tube Selection Guide in Section 5 to assure use the proper collection tube is used for the appropriate test requested.

Separate serum or plasma and remove it from the cells as soon as possible after blood is collected, because many of the constituents of plasma exist in higher concentrations in RBCs. With time, these substances leak into the plasma and cause falsely elevated values (positive interference) and falsely lower values (negative interference) (Table 4-5). Under no circumstances should whole blood be sent via the mail; serum derived from such specimens usually is hemolyzed, and results are often inaccurate. Separate serum and transfer it to a clean, dry tube for shipment.

**TABLE 4-5 Examples of Positive and Negative Interference on Biochemistry Analytes Induced by Sample Hemolysis**

<table>
<thead>
<tr>
<th>Analyte</th>
<th>Effect of Hemolysis*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alanine transaminase</td>
<td>Minimal effect</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>Increased</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>Increased</td>
</tr>
<tr>
<td>Chloride</td>
<td>Decreased</td>
</tr>
<tr>
<td>Creatinine</td>
<td>Increased</td>
</tr>
<tr>
<td>Inorganic phosphate</td>
<td>Increased</td>
</tr>
<tr>
<td>Lipase</td>
<td>Decreased</td>
</tr>
<tr>
<td>pH</td>
<td>Decreased</td>
</tr>
<tr>
<td>Potassium</td>
<td>No detectable effect</td>
</tr>
<tr>
<td>Total calcium</td>
<td>Increased</td>
</tr>
<tr>
<td>Total protein</td>
<td>Increased</td>
</tr>
<tr>
<td>Urea nitrogen</td>
<td>Increased</td>
</tr>
</tbody>
</table>

*Type and degree of interference vary among different testing modalities unique to individual laboratories or in-hospital biochemistry analyzers.
Collection of bone marrow may prove valuable in diseases of the blood in which examination of the peripheral blood reveals abnormal cells or cell counts. Conditions such as leukopenia, thrombocytopenia, nonregenerative anemia, agranulocytosis, pancytopenia, leukemias, other bone marrow cancers, and infectious diseases (e.g., histoplasmosis, ehrlichiosis) may be confirmed only by assessment of bone marrow cytology.

Bone marrow in the young animal is cellular and exists in the flat bones (sternum, ribs, pelvic bones, and vertebrae) and in the long bones (humerus and femur). As the animal ages, the cellular content of the marrow decreases, especially in the long bones. In older animals, bone marrow cells still exist in the flat bones; however, in conditions of stress in which new blood cells must be produced in large numbers, primitive cells in the bone marrow of the long bones again become active. Interpretation of the bone marrow smear may be limited by (1) technique used to obtain a bone marrow specimen or (2) the specialized knowledge necessary to interpret bone marrow cells.

Bone marrow aspiration is much underused in clinical practice. The procedure does require some degree of skill if high-quality samples are to be obtained, but the procedure is of low risk to the patient and can be highly valuable in establishing a diagnosis or prognosis.

Canine

Patient Preparation

A short-acting anesthetic occasionally may be needed, but tranquilization together with infusion of local anesthetic is usually sufficient. The site selected for aspiration or biopsy must be shaved and surgically prepared.

Technique

Bone marrow aspiration or biopsy is a percutaneous procedure conducted using sterile technique.

The techniques involved include marrow aspiration and bone marrow core biopsy alone or in combination. When aspiration biopsy fails to produce adequate cytology (as in advanced myelofibrosis, neoplasia, or marrow aplasia), a core biopsy of bone marrow is indicated. The bone marrow aspiration needle may be a 16-gauge Rosenthal needle or Illinois needle for a medium-sized dog; an 18-gauge Rosenthal needle for a small dog or a cat; or a Jamshidi (pronounced yam-she-dee) bone marrow biopsy needle, 12 gauge for most adult dogs and 14 gauge for small dogs and cats.

The selection of needles for aspiration biopsy of bone marrow is based on the biopsy site, the depth of the biopsy site, and the density of cortical bone. For bone marrow aspiration, the modified disposable Illinois sternal-iliac bone marrow aspiration needle works well (Figure 4-9). For a core biopsy of bone marrow, the Jamshidi bone marrow biopsy-aspiration needle (pediatric, 3.5 inches, 13 gauge) can be used (Figure 4-10).

The iliac crest is a commonly used site for marrow aspiration in dogs. Place the animal in lateral recumbency, and prepare the aspiration site. To aspirate marrow, have the needle enter the widest part of the iliac crest and stop the needle just after penetration of the bone. Remove the stylet, place a 12-mL syringe on the needle, and aspirate 0.2 mL of marrow. Alternatively, the head of the humerus offers easy access to abundant bone marrow. Sedation may be required. With the patient in lateral recumbency and the humerus flexed (the humerus is positioned parallel to the patient’s thorax), instill local anesthetic into the
skin and subcutaneous tissues to the level of the head of the humerus. The site of needle insertion is on the most proximal facet of the humoral head (Figure 4-1). Direct the needle into the bone toward the elbow and parallel to the humeral shaft. If the needle is positioned too far medially over the humeral head, it is easy to penetrate the joint capsule. Although this is a common occurrence, it does not pose a risk of injury to the patient (assuming the skin was surgically prepared). If joint fluid contaminates the bone marrow aspirate, the sample will be rendered useless.

Contamination of the bone marrow with peripheral blood results if (1) the marrow is not aspirated immediately after the needle enters the marrow cavity or (2) if aspiration time is sustained and a large volume of blood enters the syringe subsequent to the rupture of small blood vessels in the bone marrow.
Perhaps the least desired technique is to obtain marrow from the proximal end of the femur by insertion of the bone marrow needle into the trochanteric fossa. Make a small skin incision over the trochanteric fossa just medial to the summit of the trochanter major. Insert the bone marrow aspiration needle medial to the trochanter major, and place the long axis of the needle parallel to the long axis of the femur.

Once the site has been selected, grasp the needle firmly. Apply steady, slight pressure while alternately rotating the needle tip against the bone (fast, 180-degree clockwise and then counterclockwise movements). Begin with gentle pressure until the needle begins to seat into the bone. Gradually increase the pressure as the needle penetrates into the bone. Insert the bone marrow needle $\frac{1}{2}$ inch into the femoral canal. Remove the stylet from the needle, and aspirate using a 12- or 20-mL syringe that contains a small volume (approximately 0.1 mL) of 4% EDTA. Use significant negative pressure, for example, by withdrawing the plunger of a 12-mL syringe to the 8- or 9-mL mark.

Collection of more than 1 mL of bone marrow is unnecessary. Collection of larger volumes may cause greater amounts of peripheral blood to enter the syringe, leading to hemodilution of the sample. Once collection is complete, immediately transfer the aspirate to a watch glass containing approximately 0.25 mL of 4% EDTA. Immediately mix the sample well using the end of the syringe. This is also a good time to remove the bone marrow needle from the patient.

Prepare slides in a manner similar to that used for peripheral blood smears. Preparation of five to eight high-quality slides for submission is customary. Smears are air-dried. Slides may be stained using the same stains used for peripheral blood smears.

Bone marrow biopsy samples, usually obtained as a core, should be placed directly into 10% buffered formalin. It is generally recommended not to roll the core across a microscope slide (exfoliative cytology), as this may significantly disrupt the architecture of the sample and influence histopathologic interpretation.

**Special Considerations**

When submitting a bone marrow aspirate or bone biopsy, a complete blood count (CBC) should also be collected from that patient on the same day. The bone marrow sample and the CBC should be submitted together in order to obtain maximum diagnostic information. A thorough patient history should accompany the submitted samples.

Depending on the volume of bone marrow aspirate obtained, any additional aspirated bone marrow remaining after slides have been made can be mixed with EDTA in the same
type of tube used to collect whole blood for a CBC. Tubes may be refrigerated for short periods but never frozen. Prompt shipping and processing of liquid samples of bone marrow is encouraged, as these cells tend to rapidly undergo degeneration.

Bone marrow biopsy core samples, after fixation in 10% buffered formalin, require decalcification before processing and interpretation.

**Feline**

*Patient Preparation*

When feasible, a short-acting anesthetic administered to a cat before bone marrow aspiration or biopsy is recommended owing to the difficulty of adequately restraining a cat, even if sedated. The site selected for aspiration or biopsy must be shaved and surgically prepared. Infusion of local anesthetic at the aspiration or biopsy site is appropriate. Supplemental oxygen may be indicated.

*Technique*

Accessible sites for bone marrow sampling and biopsy in the cat are the iliac crest, the head of the humerus, and the proximal end of the femur via the trochanteric fossa. The techniques described for the dog can be used.

Smears of bone marrow are made immediately after aspiration. Extrinsic thromboplastin present in bone marrow tissue will cause the marrow to clot within 30 seconds. Unstained slides should be submitted. A core of bone marrow can be fixed in 10% buffered formalin before submission for decalcification and histologic preparation.

Another method is to aspirate the sample of bone marrow into a syringe containing 0.25 mL of 4% EDTA solution. Expel the aspirate, up to 0.5 mL, into a sterile Petri dish, from which the marrow particles can be isolated easily by aspirating an aliquot with a glass pipette, placing an appropriate volume onto several glass slides, making the appropriate number of smears.

*Special Considerations*

Slides prepared from bone marrow aspirates should be allowed to air-dry and then labeled appropriately. Slides should never be refrigerated, as moisture from condensation can alter or destroy the appearance of individual cells.

**Additional Reading**


**Cytology Collection Techniques**

(See also Section 5 for additional information on slide preparation of samples to be submitted for cytopathologic examination.)

Cytopathology involves a simple, direct, and inexpensive technique that can yield significant diagnostic information within a short time at minimal direct cost. Cytologic examination can be made of material obtained from pustules, vesicles, or the raw, ulcerated, or cut surfaces of a lesion. To make the smear, press a clean microscope slide firmly against a raw or ulcerated lesion to transfer cellular material to the slide. Exudates may be collected by sterile swab or may be aspirated into a sterile syringe. Roll the swab gently across the slide, or place a drop of fluid from the syringe onto the slide and carefully spread the fluid in a uniform film. Transfer material from a block of tissue to the slide by gently pressing the tissue onto the slide in several locations. Use various stains for different conditions.

Rapid stains such as new methylene blue or a quick Romanowsky-type stain (e.g., Diff-Quik) are useful and convenient for office procedures. Even Wright and Gram stains for evaluation of bacteria in tissues and fluids are easy to use. The presence of many bacteria,
especially mixed types, may mean only surface contamination, whereas single types of bacteria, abundant polymorphonuclear WBCs, and especially phagocytosis support the diagnosis of infection and the host response to it. A few acantholytic cells (loose epidermal cells) in the smear may be compatible with infectious processes, but large numbers, or "rafts," of acantholytic cells are highly suggestive of pemphigus and imply the need for more complex tests for positive diagnosis.

Large numbers of eosinophils sometimes are found in stained smears. Contrary to popular opinion, they usually do not mean allergy. These cells are seen most commonly with furunculosis and may be associated with the eosinophilic granulomas, eosinophilic plaques, sterile eosinophilic pustulosis, pemphigus complex, and ectoparasites. Yeasts (usually Malassezia, rarely Candida) commonly are found as budding cells in masses of wax and debris from ear smears.

Tumor cells may be recognized in some impression or aspiration samples where Giemsa is a preferred stain. Although special expertise is needed, cases of mastocytoma, histiocytoma, and lymphoma are recognized most easily. Always prepare formalin-fixed tissues for histologic diagnosis in tumor evaluations (Box 4-4).

Percutaneous Fine-Needle Aspiration

Patient Preparation

Fine-needle aspiration, the use of needle and syringe to remove cells from normal and abnormal tissue, apply them to a glass slide, stain the smear, and review the results immediately is among the most useful, cost-effective procedures available in clinical practice. In most cases there will be no specific requirements for patient preparation. Shaving hair over the aspiration site is generally not required. Surgical preparation of the site is optional.

Technique

Lymph node aspiration is a procedure that can, and should, be performed routinely in clinical practice. Follow proper technique to maximize the diagnostic utility of this procedure. Lymph node aspiration typically is indicated (1) in patients with generalized lymphadenomegaly, (2) to evaluate abnormally enlarged solitary lymph nodes, and (3) in suspected instances of tumor metastases to lymph nodes. Surgically prepare the skin over the node from which a biopsy specimen is to be taken. With one hand, localize and immobilize the lymph node; with the other hand, guide the aspiration biopsy needle into the affected node. Affix a 6-mL syringe onto a 22- to 20-gauge needle (a 25-gauge needle can be used when the site to be aspirated is particularly small), and advance the needle into the lymph node. Withdrawal of the syringe to approximately 0.5 mL before inserting it into the tissue is recommended. Doing so helps to prevent expelling material when removing the sample from the tissue. When the needle is in position in the approximate center of the node, gradually draw negative pressure on the syringe to a level of 4 to 5 mL. Hold the negative pressure in place for a few seconds. Release, and then repeat two or three times. Before removing the needle from the tissue, release the negative pressure in the syringe (this is why it is recommended to have 0.25 mL of air prepositioned inside). Do not remove the syringe from the tissue while maintaining negative pressure, because this can

<table>
<thead>
<tr>
<th>BOX 4-4 CYTOLeGIC FEATURES OF MALIGNANCY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enlargement of nucleus or nuclei larger than 10 nm</td>
</tr>
<tr>
<td>Decreased nuclear/cytoplasmic ratio</td>
</tr>
<tr>
<td>Multinucleation because of abnormal mitosis</td>
</tr>
<tr>
<td>Abnormal or frequent mitosis</td>
</tr>
<tr>
<td>Variations in size and shape of nuclei</td>
</tr>
</tbody>
</table>

www.ajlobby.com
result in the aspiration of significant amounts of blood from the skin, thereby significantly diluting the sample with peripheral blood. Eject cellular material within the needle onto clean glass slides. Handle all aspirates gently. To make slides, place two slides together and pull the slides apart to avoid shearing the cells. Do not compress or force slides together. In addition, a biopsy of the lymph node can (and usually should) be performed as a means of confirming or supporting diagnostic decisions made on aspirates. Lymph node biopsy samples can be obtained easily and safely by punch (core) techniques (e.g., 4-mm skin biopsy punch) or Tru-Cut biopsy needle.

Special Considerations
The most significant limiting factors are (1) the technical ability to prepare high-quality slides and (2) the ability to interpret the cytologic findings. Some experience is needed to obtain the skills needed to aspirate cells and make diagnostic preparations. Significant training is required to interpret the slides adequately. However, access to cytopathologists affiliated with diagnostic laboratories today makes fine-needle aspiration a highly useful diagnostic tool. The lymph node aspiration technique, described next, illustrates the finer points of the fine-needle aspiration technique.

Exfoliative Cytology
Patient Preparation
Also called “touch impression cytology,” exfoliative cytology entails preparing cytologic slides directly from the cut surface of biopsy samples. Requirements for patient preparation depend on the location of the tissue from which the sample is taken. The number and quality of cells collected are best when the procedure involves the freshly cut surface of tissue. Attempting to collect samples directly from skin lesions on the patient is much less likely to yield diagnostic cytology. Preparation, therefore, depends on the target tissue from which slides are needed. Preparation may entail local anesthesia and collection of a tissue biopsy specimen from a lesion or suspect tissue (e.g., lymph node or cutaneous tumor) or general anesthesia and an exploratory abdominal procedure (e.g., liver biopsy).

Technique
Once the tissue has been collected, a scalpel blade is used to make a full-thickness linear cut through the biopsy specimen. A fresh surface of the tissue of interest is exposed. Using forceps or a sterile needle, gently lay the tissue on a clean glass slide. Do not force the tissue onto the slide, because this can significantly damage cells. Several imprints can be made from the same surface. As needed, make new cuts to obtain a fresh surface from which to exfoliate cells. Allow the slide to air-dry completely. Apply conventional staining, and examine the specimen when it is dry. The remaining tissue, if not significantly damaged, can be submitted for histopathologic examination (recommended).

Special Considerations
Once slides have air-dried and are labeled, they may be stained and reviewed immediately or submitted for review and interpretation by a pathologist. Unstained slides should not be refrigerated, as moisture from condensation can alter the cytology of the preparation. Several slides should be submitted. Any remaining tissue may be placed in 10% buffered formalin and submitted for histopathology.

The number of diagnostic cells obtained when making slides by way of exfoliative cytology depends on the tissue. Epithelial cells (e.g., carcinoma), mast cells (cutaneous mast cell tumor), and lymph nodes readily exfoliate abundant numbers of cells. Excessively thick slides can make interpretation difficult. On the other hand, biopsy specimens of tissue composed predominantly of mesenchymal cells (e.g., granuloma, fibrosarcoma) do not readily exfoliate cells, and slides made from these types of tissues are typically hypocellular.
Contamination of the cytology specimen with peripheral blood is a common mistake that can make interpretation of the sample difficult. It may be appropriate to gently blot the cut surface of the tissue sample, thereby removing excessive blood, before making slides.

**Scrapings and Swabs**

Depending on the tissue type and lesion, it may be possible to obtain diagnostic cytologic samples from scrapings (e.g., conjunctival epithelium for virus inclusions), brushes (e.g., material obtained during endoscopy), and swabs (e.g., ear and vaginal swabs). The cells, once harvested, can be applied delicately directly to a clean glass slide by carefully rolling or even by just touching the material to the slide to create a thin layer. Allow the sample to air-dry thoroughly before staining.

**Fluids**

Cytologic examination of fluids obtained with needle and syringe from body cavities, cysts, and urine typically requires additional preparation to obtain adequate cell concentration to make diagnostic decisions. Analyze fluid specimens with respect to protein and nucleated cell count and a morphologic description of the cells. If overall cell counts are low, centrifugation will be required to concentrate cellular material for analysis. After centrifugation, remove the supernatant (and save it). Resuspend the cells in two or three drops of the supernatant. Apply a single drop of the mixture to a glass slide and allow it to air-dry. I prefer not to smear the liquid onto the slide; instead, I allow the liquid to run, by gravity, from one end of the slide to the other. After the liquid is thoroughly air-dried, it can be stained and reviewed.

**Ectoparasites**

**Skin Scrapping**

*Patient Preparation*

None required.

*Technique*

Skin scrapings frequently are obtained to find and identify microscopic parasites or fungal elements in the skin. Material required includes mineral oil in a small dropper bottle, a dull scalpel blade, glass slides, coverslips, and a microscope.

Select undisturbed, untreated skin for a scraping site. The best method is to scrape the periphery of skin lesions and avoid the excoriated or traumatized center areas. In scraping for demodectic mange, pinch a small fold of affected skin firmly and collect the surface material for examination. This procedure forces the mites out of the hair follicles and onto or near the skin surface. For sarcoptic mange, scrape large areas. Select sites on the elbows, hocks, and ear margins when searching for sarcoptic mange. Many or frequent scrapings may be necessary to demonstrate sarcoptic mange mites or their fecal pellets or eggs.

Place the accumulated material on a microscope slide and mix it with mineral oil. Examine the entire area with a ×10 objective thoroughly and carefully.

**Acetate Tape Preparation**

*Patient Preparation*

None required.

*Technique*

Acetate tape preparation is one of the simplest diagnostic procedures to perform when looking for the presence of ectoparasites, especially the nits of *Cheyletiella*. Use clear (not frosted) acetate tape. Bend the tape into a loop around the fingers with the sticky side facing out. Part the animal’s hair coat, and press the tape firmly onto the skin and hair around suspect lesions. The sticky tape picks up loose particles with which it makes contact. Cut the loop of tape and place the strip of tape sticky side down on a clean microscope slide. Use a
low-power microscope to look through the tape at the collected particles. This technique is excellent for trapping and identifying biting and sucking lice, *Otodectes* and *Cheyletiella* mites, flea dirt and larvae, fly larvae, and dandruff scales.

Acetate tape also is useful for studying hair abnormalities. Use a strong hemostat to securely clamp and quickly avulse a group of 10 to 20 hair shafts. Press the pointed distal ends onto sticky acetate tape (lined up like pickets in a fence), and cut the hair shafts off in the middle with scissors. Likewise, press the butt ends with the hair roots onto another piece of tape. Then press the tape holding the hair onto a microscope slide to allow low-power examination of the hairs through the clear tape. The tips of the hairs will be well oriented and controlled; thus, it is easy to evaluate whether the hairs are split, broken, or bitten off and whether the hair roots are in the anagen or telogen growth stage.

**Additional Reading**


**Urine Collection Techniques**

Urine can be removed from the bladder by one of four methods: (1) voided (the “free catch”), (2) manual compression of the urinary bladder (expressing the bladder), (3) catheterization, or (4) cystocentesis.

**Voiding**

For routine urinalysis, collection of urine by voiding (micturition) is satisfactory. The major disadvantage is risk of contamination of the sample with cells, bacteria, and other debris located in the genital tract and the perineal hair coat. The first portion of the stream is discarded, as it is most likely to contain debris. Voided urine samples are not recommended when bacterial cystitis is suspected.

**Manual Compression of the Bladder**

Compressing the urinary bladder is occasionally used to collect urine samples from dogs and cats. *Critical: Do not use excessive pressure*; if moderate digital pressure does not induce micturition, discontinue the technique. Excessive pressure can culminate in forcing contaminated urine (bladder) into the kidneys, or, worse, in patients with a urethral obstruction the urinary bladder can rupture. The technique is most difficult to accomplish in male dogs and male cats.

**Urinary Catheterization**

Several types of urinary catheters are currently available for use in dogs and cats. The catheter types most often used today are made of rubber, polypropylene, and latex-free silicone. Stainless steel catheters are occasionally used but unless placed with care these can cause damage to the urethra and/or urinary bladder. Generally, urinary catheters serve one of four purposes:

1. To relieve urinary retention
2. To test for residual urine
3. To obtain urine directly from the bladder for diagnostic purposes
4. To perform bladder lavage and instillation of medication or contrast material

The size of catheters (diameter) usually is calibrated in the French scale; each French unit is equivalent to roughly 0.33 mm. The openings adjacent to the catheter tips are called “eyes.” Human urethral catheters are used routinely in male and female dogs; 4F to 10F catheters are satisfactory for most dogs (*Table 4-6*). Polypropylene catheters should be individually packaged and sterilized by ethylene oxide gas.
Catheterization of the Male Dog

**Patient Preparation**

Equipment needed to catheterize a male dog includes a sterile catheter (4F to 10F; 18 inches long, with one end adapted to fit a syringe), sterile lubricating jelly, povidone-iodine soap or chlorhexidine, sterile rubber gloves or a sterile hemostat, a 20-mL sterile syringe, and an appropriate receptacle for the collection of urine.

Proper catheterization of the male dog requires two persons. Place the dog in lateral recumbency on either side. Pull the rear leg that is on top forward, and then flex it (Figure 4-12). Alternatively, long-legged dogs can be catheterized easily in a standing position.

Before catheter placement, retract the sheath of the penis and cleanse the glans penis with a solution of povidone-iodine 1% or chlorhexidine. Lubricate the distal 2 to 3 cm of the appropriate-size catheter with sterile lubricating jelly. Never entirely remove the catheter from its container while it is being passed because the container enables one to hold the catheter without contaminating it.

**Table 4-6** Recommended Urethral Catheter Sizes for Routine Use in Dogs and Cats

<table>
<thead>
<tr>
<th>Animal</th>
<th>Urethral Catheter Type</th>
<th>Size (French Units*)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cat</td>
<td>Flexible vinyl, red rubber, or</td>
<td>3.5</td>
</tr>
<tr>
<td></td>
<td>Tom Cat catheter (polyethylene)</td>
<td></td>
</tr>
<tr>
<td>Male dog (≤25 lb)</td>
<td>Flexible vinyl, red rubber, or</td>
<td>3.5 or 5</td>
</tr>
<tr>
<td></td>
<td>polyethylene</td>
<td></td>
</tr>
<tr>
<td>Male dog (≥25 lb)</td>
<td>Flexible vinyl, red rubber, or</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>polyethylene</td>
<td></td>
</tr>
<tr>
<td>Male dog (&gt;75 lb)</td>
<td>Flexible vinyl, red rubber, or</td>
<td>10 or 12</td>
</tr>
<tr>
<td></td>
<td>polyethylene</td>
<td></td>
</tr>
<tr>
<td>Female dog (≤10 lb))</td>
<td>Flexible vinyl, red rubber, or</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>polyethylene</td>
<td></td>
</tr>
<tr>
<td>Female dog (10-50 lb)</td>
<td>Flexible vinyl, red rubber, or</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>polyethylene</td>
<td></td>
</tr>
<tr>
<td>Female dog (&gt;50 lb)</td>
<td>Flexible vinyl, red rubber, or</td>
<td>10, 12, or 14</td>
</tr>
<tr>
<td></td>
<td>polyethylene</td>
<td></td>
</tr>
</tbody>
</table>


*The diameter of urinary catheters is measured on the French (F) scale. One French unit equals roughly 0.33 mm.

**Technique**

The catheter may be passed with sterile gloved hands or by using a sterile hemostat to grasp the catheter and pass it into the urethra. Alternatively, cut a 2-inch “butterfly” section from the end of the thin plastic catheter container. This section can be used as a cover for the sterile catheter, and the clinician can use the cover to grasp and advance the catheter without using gloves.

If the catheter cannot be passed into the bladder, the tip of the catheter may be caught in a mucosal fold of the urethra or there may be a stricture or block in the urethra. In small-breed dogs, the size of the groove in the os penis may limit the size of the catheter that can be passed. One also may experience difficulty in passing the catheter through the urethra where the urethra curves around the ischial arch. Occasionally a catheter of small diameter may kink and bend on being passed into the urethra. When the catheter cannot be passed...
on the first try, reevaluate the size of the catheter and gently rotate the catheter while passing it a second time. Never force the catheter through the urethral orifice.

Special Considerations
Effective catheterization is indicated by the flow of urine at the end of the catheter, and a sterile 20-mL syringe is used to aspirate the urine from the bladder. Walk the dog immediately after catheterization to encourage urination.

Catheterization of the Female Dog
Patient Preparation
Equipment needed to catheterize a female dog includes flexible urethral catheters identical to those used in the male dog. The following materials also should be on hand: a small nasal speculum, a 20-mL sterile syringe, lidocaine 0.5%, sterile lubricating jelly, a focal source of light, appropriate receptacles for urine collection, and 5 mL of povidone-iodine or a dilute chlorhexidine solution.

Use strict asepsis. Cleanse the vulva with a solution of povidone-iodine or dilute chlorhexidine. Instillation of lidocaine 0.5% into the vaginal vault helps to relieve the discomfort of catheterization. The external urethral orifice is 3 to 5 cm cranial to the ventral commissure of the vulva. In many instances the female dog may be catheterized in the standing position by passing the female catheter into the vaginal vault, despite the fact that the urethral papilla is not visualized directly.

Technique
In the spayed female dog, in which blind catheterization may be difficult, the use of a sterilized otoscope speculum and light source (Figure 4-13), vaginal speculum, or anal speculum with a light source will help to visualize the urethral tubercle on the floor of the vagina. In difficult catheterizations it may be helpful to place the animal in dorsal recumbency (Figure 4-14 and 4-15). Insertion of a speculum into the vagina almost always permits visualization of the urethral papilla and facilitates passage of the catheter. Take care to avoid attempts to pass the catheter into the fossa of the clitoris because this is a blind, possibly contaminated cul-de-sac.
Patient Preparation

Before attempting urinary bladder catheterization of the male cat, administer a short-term anesthetic (e.g., ketamine, 25 mg/kg IM), but only after a careful assessment of the cat’s physical, acid-base, and electrolyte status (see treatment of hyperkalemia in Section 1).

Figure 4-13: An otoscope speculum with attached light source provides excellent visualization of the urethral orifice in a female dog. Note the position of the otoscope handle (see Figure 4-14).

Figure 4-14: Visualization of the urethral orifice and catheterization of the urinary bladder in a female dog is accomplished using an otoscope with a sterile speculum attached. Note: The patient is in dorsal recumbency with the otoscope handle positioned upward.

Catheterization of the Male Cat

Patient Preparation

Before attempting urinary bladder catheterization of the male cat, administer a short-term anesthetic (e.g., ketamine, 25 mg/kg IM), but only after a careful assessment of the cat’s physical, acid-base, and electrolyte status (see treatment of hyperkalemia in Section 1).
In some cases, drugs to treat hyperkalemia may be required before anesthetic induction. Once the patient's electrolyte status has been evaluated and hyperkalemia, if present, addressed appropriately, anesthesia can be induced with a combination of propofol (4 to 7 mg/kg intravenously [IV]) and diazepam (0.1 mg/kg IV); then the patient is intubated and maintained on gas anesthesia.

**Technique**

Place the anesthetized patient in dorsal recumbency. Gently grasp the ventral aspect of the prepuce and move it caudally in such a manner that the penis is extruded. Withdraw the penis from the sheath and gently pull the penis backward. Keeping sterile catheters in a freezer will help them become more rigid to facilitate passage into the urethra. Pass a sterile, flexible plastic or polyethylene (PE 60 to 90) catheter or 3- to 5-inch, 3.5F urethral catheter into the urethral orifice and gently into the bladder, keeping the catheter parallel to the vertebral column of the cat.

*Caution: Never force the catheter through the urethra.* The presence of debris within the urethral lumen may require the injection of 3 to 5 mL of sterile saline to back-flush urinary “sand” or concretions so that the catheter can be passed. In some instances the presence of cystic and urethral calculi will prevent the passage of a catheter into the urethra. For this reason a lateral radiograph of the penis, with the patient's hindlimbs pulled caudally, may help document the presence of a urethral stone.

**Catheterization of the Female Cat**

**Patient Preparation**

Urinary bladder catheterization of the female cat is not a simple procedure. When indicated, and after a preanesthetic examination has been performed, attempt the technique only in the anesthetized cat. Urinary bladder catheterization can be accomplished with the use of a rubber or plastic, side-hole (blunt-ended) urinary catheter. The same catheter type used in male cats is effective in female cats. Instilling lidocaine 0.5% has been recommended as a means of decreasing sensitivity to catheter insertion in sedated (not recommended) cats. Cleanse the vulva with an appropriate antiseptic.
Technique
Catheterization can be accomplished with the cat in dorsal or ventral recumbency. Experience and size of the cat dictate which technique works best.

After cleansing of the perineum and vaginal vault, place the patient in sternal recumbency, and gently pass the catheter along the ventral floor of the vaginal vault. Conversely, if the patient is placed in dorsal recumbency, direct the catheter dorsally along the ventral vaginal floor. If a catheter cannot be placed blindly, a small otoscopic speculum can be placed into the vagina, and the catheter pushed into the urethral papilla once it is visualized directly.

Indwelling Urethral Catheter

Patient Preparation
For continuous urine drainage in the awake, ambulatory patient, use a closed collection system to help prevent urinary tract infection. A soft urethral or Foley catheter can be used, and polyvinyl chloride tubing should be connected to the catheter and to the collection bag outside the cage. The collection bag should be below the level of the animal’s urinary bladder. Place an Elizabethan collar on the animal to discourage chewing on the catheter and associated tubing.

Technique
The urinary bladder is catheterized as described previously. Despite the quality of care of the catheter, urinary tract infection still may develop in any patient fitted with an indwelling urinary catheter. Ideally, remove the catheter as soon as it is no longer necessary, or if there are clinical signs of a urinary tract infection or previously undiagnosed fever. A urinary catheter is generally changed after it has been in place for more than 48 hours.

Special Considerations
Observe the patient for development of fever, discomfort, pyuria, or other evidence of urinary tract infection. If infection is suspected, remove the catheter and submit urine for culture and sensitivity or determination of minimum inhibitory concentration (MIC). Previously, culture of the catheter tip was recommended to diagnose a catheter-induced infection. However, culture of the catheter tip is no longer recommended, as it may not accurately reflect the type of microorganisms in a urinary tract infection. The empiric use of antibiotics to help prevent catheter-induced infection is not recommended, as their use can allow colonization of resistant nosocomial bacteria in the patient’s urinary tract.

Cystocentesis

Patient Preparation
Cystocentesis is a common clinical technique used to obtain a sample of urine directly from the urinary bladder of dogs and cats when collecting a voided, or free-catch, aliquot is not preferred. The procedure is indicated when necessary to obtain bladder urine for culture purposes. Urine that is collected by free catch has passed through the urethra and may be contaminated with bacteria, thereby making interpretation of the culture results difficult. Cystocentesis also is performed as a convenience when it is desirable to obtain a small sample of urine but the patient is not ready or cooperative.

Cystocentesis involves insertion of a needle, with a 6- or 12-mL syringe attached, through the abdominal wall and bladder wall to obtain urine samples for urinalysis or bacterial culture. The technique prevents contamination of urine by urethra, genital tract, or skin and reduces the risk of obtaining a contaminated sample. Cystocentesis also may be needed to decompress a severely overdistended bladder temporarily in an animal with urethral obstruction. In these cases, cystocentesis should be performed only if urethral
catheterization is impossible. **Warning:** Penetration of a distended (obstructed) urinary bladder with a needle could result in rupture of the bladder.

**Technique**
To perform cystocentesis, palpate the ventral abdomen just cranial to the junction of the bladder with the urethra, and trap the urinary bladder between the fingers and the palm of the hand. Use one hand to hold the bladder steady within the peritoneal cavity while the other guides the needle. Next, insert the needle through the ventral abdominal wall into the bladder at a 45-degree angle (Figure 4-16). Although this procedure is relatively safe, the bladder must have a reasonable volume of urine, and the procedure should not be performed without first identifying and immobilizing the bladder. For the procedure to be performed safely and quickly, the patient must be cooperative. If collection of a urine sample by cystocentesis is absolutely necessary, sedation may be indicated to restrain the patient adequately for the procedure.

**Special Considerations**
Generally, cystocentesis is a safe procedure, assuming the patient is cooperative and the bladder can be identified and stabilized throughout the procedure. However, injury and adverse reactions can occur. In addition to laceration of the bladder with the inserted needle (patient moves abruptly), the needle can be passed completely through the bladder and into the colon, causing bacterial contamination of the bladder or peritoneal cavity. There is also risk of penetrating a major abdominal blood vessel, resulting in significant hemorrhage.

**Additional Reading**
SKIN BIOPSY

Patient Preparation

Obtaining a skin biopsy from abnormal skin only to receive a nondiagnostic result as reported from a pathologist suggests that improved biopsy technique may result in collecting a specimen with higher diagnostic value. The following guidelines apply when skin biopsies are performed:

- Consider obtaining multiple samples from multiple sites, which is especially useful when different stages of similar lesions are identifiable.
- Do not perform a surgical scrub before collecting the sample; shaving the hair away is fine, but surgically prepared skin removes superficial lesions that, had they been left in place, might have been diagnostic.
- Biopsy of lesions that are depigmenting should be done before they have turned white; the absence of color usually denotes absence of active skin lesions. Biopsies from completely depigmented skin are less likely to demonstrate active lesions.
- Biopsy of lesions associated with alopecia should be done in the center of the most alopecic area.
- Also, biopsy of lesions associated with alopecia should be done at junctional (between normal- and abnormal-appearing) skin.
- Consider submitting biopsy samples from completely unaffected, normal-appearing skin.
- Avoid biopsies of ulcerated skin areas.

Technique

Biopsy samples may be obtained with a scalpel blade (incisional or excisional) or via a dermatologic punch biopsy. Punch biopsy instruments are circular blades available in 4-mm, 6-mm, and 8-mm diameter sizes (Figure 4-17). Hold the punch perpendicular to the skin site of interest. A back-and-forth motion that rotates the circular blade cuts through the skin. When the skin no longer moves as the punch is rotated, the biopsy is complete and the skin sample may be removed (from the skin or from the biopsy instrument). Avoid grasping the dermis or epidermis of the sample with any instrument to prevent crushing of the sample and causing artifact. If the sample must be lifted, use the attached subcutaneous fat only.

Figure 4-17: Disposable skin biopsy punches: 4-, 6-, and 8-mm sizes are available.
Special Considerations
If the lesion of interest is deep, the punch biopsy technique may not be effective. In this situation, an incisional or excisional biopsy using a sterile No. 10 or No. 15 surgical blade is indicated. Biopsies of ulcerated skin and solitary nodules are best done by removing a wedge of skin (incisional biopsy). In some cases it is possible surgically to remove all visible, palpable parts of the lesion (excisional biopsy). Place each sample of skin in buffered formalin, using a volume that is at least 10 times that of the sample size. If particularly large areas of skin are harvested during biopsy, cut these into 1-cm thick pieces before placing into formalin. (Note: Placing larger tissue samples into formalin may result in inadequate or incomplete fixation of the tissue and the inability to properly prepare the tissue for examination).

Alternatively, it is possible, and in many cases important, to evaluate a biopsy specimen of skin or subcutaneous tissue at the time of collection. When the lesion of interest is suspected to be neoplastic, quickly differentiating between inflammatory cells and neoplasia may be possible by simply performing an exfoliative cytologic examination (described in this section) on one of the biopsy samples in addition to fixing a separate sample in formalin and submitting that for histopathologic examination.

Small biopsy samples that have been subjected to the additional handling required to make impressions on a glass slide are not good candidates for subsequent fixation and histopathologic examination. It is generally recommended to perform exfoliative cytologic and histopathologic examinations on separate samples.

Skin Scraping
Superficial Skin Scraping
Among the most common diagnostic procedures carried out on the skin of dogs and cats is a routine skin scraping. Yet despite the frequency with which this test is used, doing a skin scraping in such a manner that the sample recovered maximizes the opportunity to establish a diagnosis can be anything but routine. A skin scraping, properly done, does require using consistent techniques appropriate to the suspected diagnoses, and as such, superficial or deep scrapings, or both, may be indicated. Skin scraping is indicated whenever ectoparasite infestation is suspected. Superficial scrapings are appropriate for detecting mites that live on the skin surface, such as Cheyletiella species and Otodectes cynotis, as well as mites that burrow within the outermost layers of skin (stratum corneum), such as Sarcoptes species and Notoedres cati.

Patient Preparation
Because the area to be scraped is relatively large (≥2 cm²), shave dogs and cats with long hair coats before attempting the procedure, unless Cheyletiella infestation is suspected.

Technique
Make the scraping over healthy-appearing skin. Do not cleanse the skin of superficial scale or crusts. The technique for superficial skin scraping entails the use of mineral oil or pyrethrin ear drops applied to a clean scalpel blade and directly onto the area of skin to be scraped. Scraping begins as a gentle motion made in the direction of the hair coat. Gradually increase the pressure of the blade against the skin with repetitive scrapings over the same area.

Special Considerations
Take care not to lacerate the skin, although minor capillary bleeding at the site is common. Transpose material collected on the edge of the blade to a clean glass slide, cover it with a coverslip, and thoroughly examine the material under low magnification for evidence of ectoparasites. Note that for mites such as Cheyletiella or scabies, finding just one mite or one egg is diagnostic and justifies implementing treatment.
Deep Skin Scraping

Patient Preparation

Same as described for superficial skin scraping (earlier).

Technique

A slightly different technique is indicated in dogs and cats suspected of having an infestation that includes *Demodex canis* mites. The mites are known to live predominantly in sebaceous glands and hair follicles. They can survive in the skin of animals without manifesting lesions. Hair loss and skin lesions develop where overgrowth of the mite population occurs. *Demodex* infestations can be localized or generalized; infestations can occur in either dogs or cats but the most severe, generalized infestations are much more likely to occur in young dogs.

Although both superficial and deep skin scrapings may reveal the presence of mites on the skin, deep scrapings may reveal *Demodex* mites in some patients when superficial scrapings are negative. The technique for deep skin scraping targets a small area of skin (<2 cm²). It may be helpful to apply gentle pressure to the skin or actually to squeeze the area of interest between the thumb and a finger in an attempt to force mites from the deeper to the more superficial skin. In some breeds (e.g., Old English Sheepdogs and Shar-Peis) recovering mites on a skin scraping can be particularly difficult. In such cases, when *Demodex* infestation is highly suspected but the results of repeated skin scrapings are negative, a skin biopsy is appropriate.

Alternatively, a procedure called a *trichogram* that involves pulling (plucking) a few hairs from the hair follicles using a hemostat may be diagnostic. Once the hairs have been plucked from the skin, place them on a glass slide that has been preprepared with a drop of mineral oil, add a coverslip, and examine the hair shaft under low magnification. Half of all dogs with *Demodex* infestation will have a positive trichogram.

Additional Reading


EAR CLEANING: EXTERNAL EAR CANAL

Not all dogs and cats with otitis externa require comprehensive ear flushing and debridement before or as part of otic therapy. In many cases, home treatment is sufficient to resolve the problems effectively, assuming the underlying diagnosis has been established. However, in patients with chronic or particularly severe infections, topical treatment administered by the owners at home may not be sufficient. In such cases, the external ear canal requires a careful and comprehensive cleaning before administration of topical medications.

Patient Preparation

Properly performed, flushing and cleaning of the external ear canals is not a quick procedure. Anesthetize the patient. Attempting to perform a thorough ear cleansing under sedation usually will not be successful. Once the animal is anesthetized, perform a careful otoscopic (or video otoscopic) examination to establish the integrity of the ear canal, evaluating, for example, for the presence or absence of tumors or parasites. In severe cases the tympanic membrane (ear drum) may not even be visible.

Technique

With the patient in lateral recumbency, flush the ear canal (Figure 4-18) or lavage it with warm saline initially, and then aspirate the material from the canal. If this procedure is not successful in removing the debris attached to the epithelium of the ear canal, use
ceruminolytic ear solutions to facilitate breakdown and removal of this material. A 5-minute instillation and soak is recommended, followed by thorough flushing to remove debris and the ceruminolytic material. Remove hair growing inside the ear canal with forceps. A suction apparatus is recommended for removal of debris and liquid remaining.

Reintroduce an otoscope to examine the integrity of the skin in the ear canal and to look for any evidence of stenosis, foreign body, or tumor. The flushing process is not complete until it is possible to visualize the tympanic membrane. Carefully remove any remaining debris with an otologic loop (Figure 4-19), not a cotton-tipped swab.

Repeat the procedure on the opposite ear as indicated. At the conclusion of the examination, apply appropriate topical medication into the ear canal before allowing the patient to recover from anesthesia. Systemic therapy or surgical intervention may be required in some patients for complete resolution of the problem. However, a thorough examination and cleaning is critical before actually making decisions regarding medical versus surgical intervention.

Special Considerations
Use of a cotton-tipped swab to remove debris from the external ear canal, although commonly done, is not generally recommended. Repeated attempts may ultimately force debris deeper into the external ear canal. General anesthesia and otic lavage or flushing may be required to effectively correct the problem created by the use of cotton-tipped swabs.

Additional Reading

ENDOTRACHEAL INTUBATION
In selecting an appropriate endotracheal tube, consider the size of the animal and select a tube that has the largest diameter that can be inserted without force (Table 4-7). The length of tube selected must not extend beyond the bifurcation of the trachea (carina).
Patient Preparation
Always check the cuff of the endotracheal tube to ensure there are no leaks and that the cuff is working properly before intubation. Lubricate the selected endotracheal tube with sterile lubricating jelly before inducing anesthesia. After induction, place the patient in sternal recumbency and elevate the head.

Technique
The individual inserting the tube should grasp and extend the tongue with a piece of gauze. The tongue is extended to facilitate visualization of the larynx. Avoid excessive downward pressure on the tongue in order to prevent inadvertent laceration or injury from the lower incisors. If a laryngoscope is used, place the tip of scope at the base of the tongue. Gently press the tip of the laryngoscope ventrally to move the epiglottis and expose the glottis. Directly visualize the arytenoid cartilages, and then pass the tube through the arytenoid cartilages into the trachea using a slight twisting motion. If the arytenoid cartilage closes on
contact during an attempt to intubate, one or two drops of 2% lidocaine can be applied to
the arytenoid cartilages. Once the tube has been inserted into the trachea, never advance it
farther than the carina. Doing so may result in intubation of either the right or the left main
bronchus (endobronchial intubation). Once the tube is in place, secure it in place with a
loop of 1⁄4-inch white tape or muzzle gauze.

Special Considerations
Overinflation of the endotracheal tube cuff can cause tracheal ulceration, tracheitis, hemo-
rhage, tracheomalacia, fibrosis, stenosis, and subcutaneous emphysema.

Additional Reading
Mosby.

### TABLE 4-7 Recommended Sizes for Endotracheal Tubes

<table>
<thead>
<tr>
<th>Body Weight (kg)</th>
<th>Magill Size</th>
<th>French Size</th>
<th>Internal Diameter (mm)</th>
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</tr>
<tr>
<td>2</td>
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<td>38-44</td>
<td>12</td>
</tr>
<tr>
<td>Cats</td>
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<td></td>
<td></td>
</tr>
<tr>
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<td>0</td>
<td>13</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>1</td>
<td>16</td>
<td>5</td>
</tr>
<tr>
<td>4</td>
<td>1</td>
<td>20</td>
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</table>

Abdominocectesis

Patient Preparation
Abdominal paracentesis refers to the aspiration of fluid from the abdominal cavity for both
diagnostic and therapeutic purposes. Always weigh the animal before and after removing
abdominal fluid. Any subsequent gain in weight indicates a reaccumulation of abdominal fluid.
Place the animal in left lateral recumbency and restrain it in this position. Clip and
surgically prepare a 1- to 3-inch square between the bladder and the umbilicus just lateral
to the midline. If the bladder is distended, empty it before performing paracentesis. Infiltrate
the paracentesis site with lidocaine 0.5% using a 22- to 25-gauge needle. In most cases, local
anesthesia is not necessary. Abdominal puncture can be made with an 18- to 20-gauge nee-
dle (Figure 4-20).

Technique
Gently insert the needle through the skin and external abdominal oblique muscles while
simultaneously pushing and twisting, to push viscera away from the tip of the needle. Blind
abdominocectesis without the use of ultrasound to guide needle placement into a fluid pocket
can have negative results if there is less than 5 mL of fluid per kilogram within the peritoneal
cavity. When the abdominal puncture has been made, allow the animal to rest quietly to
facilitate drainage of the fluid. Some clinicians recommend tapping while the patient is in a standing position in the hope of obtaining more complete drainage. Changing the patient’s position after the tapping may result in needle-tip laceration of intraabdominal organs. Aspiration may be easier if a specially adapted needle with multiple holes drilled in the shaft is used because it is less likely to become plugged with omentum. Ideally, tap four quadrants of the abdomen. If four-quadrant abdominocentesis has been performed and no fluid has been obtained, and if there is suspicion for peritonitis, diagnostic peritoneal lavage can be performed. The abdomen is clipped and aseptically scrubbed as previously described. Next, insert an over-the-needle catheter or hypodermic needle just caudal to the umbilicus, and instill 10 mL of warmed 0.9% sterile saline or lactated Ringer’s per kilogram. After instillation of the fluid, walk the patient around or gently roll the patient from side to side to distribute the fluid throughout the abdominal cavity. Next, perform four-quadrant abdominocentesis as previously described. Only a small amount of the fluid infused will be obtained. The fluid will be diluted by the saline or lactated Ringer’s infused, so biochemical analysis will not be accurate. However, the fluid can be examined microscopically for the presence of plant material, bacteria, WBCs, or bile pigment to help diagnose various forms of peritonitis.

**Additional Reading**


**BIOPSY TECHNIQUES: ADVANCED**

Numerous biopsy techniques are available, and the selection of the appropriate technique is based on the tissue to be examined, the condition of the patient, and the skill of the examiner.
Excisional biopsy refers to the surgical removal of the entire lesion or organ with subsequent histologic examination. Excisional biopsy is used most frequently for skin lesions and cases in which an entire organ may have to be removed (such as an eye or an internal organ that has developed a tumor). Incisional biopsy refers to the surgical removal of a portion of a lesion with subsequent histologic examination. Choose a representative area of the lesion for biopsy. Include lesion margins, if possible. Needle aspiration refers to the use of needle and syringe to remove representative cells from the tissue or organ of interest. Specialized needles are available that allow removal of very small biopsy specimens that can be submitted for histopathologic examination. (See also Fine Needle Aspiration.)

**Needle Biopsy Techniques: General Considerations**

Needle biopsy or aspiration techniques refer to a variety of techniques used to obtain diagnostic tissue or cells from internal organs, including the lung, liver, spleen, pancreas, abdominal lymph nodes, and mass lesions within the abdomen and thorax. In contrast, fine-needle aspiration is a technique generally used to recover cytopathologic samples (cells only) from skin or subcutaneous tissues (e.g., superficial lymph nodes). The advantage of needle biopsy is related directly to how well the abnormal tissue has been characterized and how easily it can be identified during the procedure. In addition, depending on patient cooperation, most procedures can be performed safely with the patient sedated only. Short-term intravenous anesthesia and general anesthesia eliminate undesired patient movement during the biopsy procedure.

**Patient Preparation**

Potential lesions or abnormal tissues from which aspirate or biopsy samples are to be taken are located using palpation, radiographs, or ultrasound-guided imaging techniques. Shave the skin over the site of needle penetration and surgically prepare it. The type of sedation or anesthesia depends on the temperament of the animal and the site on which the biopsy will be performed.

**Technique**

Attach a 22-gauge needle without stylet to a 12-mL syringe prefilled with 0.5 to 1.0 mL of air. Optionally, affix a flexible extension set to the needle and connect it proximally to the syringe. Needle length may vary from 1 to 3½ inches depending on the required depth of penetration and size of the patient. Guide the needle into the tissue or organ of interest. Stabilize the tip of the needle to avoid random movements through organs, especially highly vascular tissue such as liver and spleen. Once the needle has been inserted, the aspiration technique entails withdrawing the plunger of the syringe to the 7- or 8-mL level. Hold that position for 1 to 2 seconds, and then release. Repeat the procedure. Depending on the nature of the lesion, it may not be indicated to thrust the needle into the tissue at multiple and different angles.

Neutralize the pressure in the syringe, and withdraw the needle rapidly. Expel any material within the needle onto glass slides using the air in the syringe. This same procedure can be repeated with a new needle to obtain an additional three to five samples from alternative sites. This technique allows samples to be obtained without applying negative pressure to the syringe, which may damage cells.

**Special Considerations**

Ultrasound guidance for needle aspirations from abdominal tissues greatly enhance the safety of this technique, especially when obtaining samples from smaller animals. Automatic-trigger needles such as Cook or Temno biopsy needles (14 to 18 gauge) are available for use in human beings but are seldom used in veterinary medicine. The risks associated with fine-needle aspiration include rupture of an encapsulated inflammatory process, dissemination of an infectious agent, seeding of neoplastic cells in the needle tract, and hemorrhage. Larger volumes of fluid and cells can be placed directly into a vial containing EDTA to prevent clot formation. Prepare and examine direct and sedimentation specimens.
Needle biopsy of internal organs using the Tru-Cut needle is particularly useful in patients with subcutaneous (Figure 4-21) or cutaneous masses and for localized abdominal and thoracic mass lesions and diffuse liver, kidney, and splenic disease. Serious complications, usually hemorrhage or laceration of the gallbladder (during liver biopsy), can occur when the procedure is performed blindly. Therefore ultrasound-guided needle biopsy is strongly recommended whenever a percutaneous biopsy of internal organs is performed. Additional safety factors provided by ultrasound guidance include the ability to image, and avoid, large aberrant blood vessels.

Risk of complications associated with needle aspiration of the lung is considerably higher than for most abdominal procedures. Pneumothorax can occur after a single, “clean” aspiration attempt. See Respiratory Tract Procedures for a detailed description of performing fine-needle aspiration of the lung.

Additional Reading

**SKIN BIOPSY**
Histologic examination of diseased skin can serve as a means for diagnosis of cutaneous lesions. The causative agent often is found in acute and chronic skin infections. Punch biopsy of the skin is a quick and accurate means of removing a small sample of diseased skin for histopathologic examination.

**Patient Preparation**
Select a site that is well developed but not traumatized or excoriated. The sample should include little or no normal tissue. If the lesion (pustule, vesicle) can be identified early in its development and if the biopsy sample is taken only from the lesion, one may obtain a superior specimen. It is best not to take too large a sample that contains much normal skin; by mistake, the technician might take a section that misses the lesion. Proper selection of the biopsy site is crucial to accurate diagnosis. Carefully clip the hair from the lesion. Lightly blot the skin with 70% alcohol. Avoid superficial trauma while cleaning the skin. Inject a small subcutaneous bleb of 2.0% lidocaine to deaden the area. Special equipment needed for the biopsy includes a 4-mm, 6-mm, or 8-mm biopsy punch and 10% buffered formalin solution.

**Technique**
After the area has been anesthetized with lidocaine, press and rotate the biopsy punch through the skin until the subcutaneous tissue is penetrated. Remove the biopsy specimen by "spearing" the subcutaneous fat with a fine needle. Do not grasp the specimen with a forceps. Blot the specimen gently between two paper towels. Spread the tissue out gently (like a pancake), place the specimen epidermal side up on a piece of cardboard or tongue depressor, press the specimen gently to cause adhesion, and drop the specimen into the formalin fixative. The skin defect may be closed with one or two simple interrupted sutures. If deep subcutaneous tissue or large biopsy samples are needed, a punch biopsy is inadequate. Use a small (No. 15) scalpel blade to obtain an appropriate sample. In all cases in which skin biopsies are made, take multiple samples to increase the odds that at least one will have diagnostic lesions. Specimens submitted to laboratories should be accompanied by extensive, detailed clinical information, including a differential diagnosis. Skin biopsies routinely are stained with hematoxylin-eosin; however, periodic acid-Schiff, Gomori methenamine silver, and Verhoeff stains are used for special problems.

**LIVER BIOPSY**
The diagnosis of liver disease is generally confirmed on the basis of the patient's clinical signs coupled with laboratory findings, radiography, and abdominal ultrasound. The development of a more specific diagnosis and prognosis in liver disease may be aided greatly by information obtained in a liver biopsy. Percutaneous liver biopsies are of much greater value in generalized liver disease such as cirrhosis, generalized acute hepatic necrosis, or amyloidosis than in focal hepatic disease. The major indications for performing a liver biopsy are (1) to explain an abnormal liver profile, (2) to define reasons for abnormal liver size, (3) to identify a possible liver tumor, (4) to arrive at a prognosis and rational approach to management, and (5) to identify the cause of ascites.

**Patient Preparation**
The procedures for obtaining liver tissue are numerous; however, needle biopsy of the liver, when performed properly, can be helpful. Careful physical and clinicopathologic examination should precede a liver biopsy. A normal coagulation profile should be documented on every patient undergoing liver biopsy. Detect and correct abnormalities in normal hemostatic mechanisms, if feasible, before needle biopsy of the liver. Liver biopsy should be performed only in the fasted patient and only after removal of ascitic fluid.
Technique

Percutaneous needle biopsies and fine-needle aspirations of the liver can be performed with local anesthesia in the sedated, and cooperative, patient. General anesthesia is a reasonable alternative whenever feasible. Biopsy sites in the liver can be selected best when needle biopsy techniques are used along with laparoscopy or ultrasound techniques. Blind percutaneous needle biopsies of the liver can be performed with relative safety if the liver is significantly enlarged and easily palpated. However, blind biopsies do carry the risk that the operator will be unable to determine the impact of penetrating the liver if only an abdominal radiograph and impression of abdominal palpation are available. In cases in which the liver is not palpable, blind biopsy carries significantly higher risk and should be performed only when no alternative exits.

A modified percutaneous liver biopsy can be performed by the following method. Place the animal in dorsal recumbency, and place a local block in the midline of the skin and abdomen at the caudoventral aspect of the left hepatic lobe. The incision into the peritoneal cavity should be large enough to accommodate the gloved index finger. Make a separate skin puncture site in the abdominal wall to accommodate the biopsy needle. Use the index finger manually to fix the left hepatic lobe (or other desired hepatic lobe) against the diaphragm or other adjacent structures, and insert the outer cannula and stylet through the abdominal wall in the isolated hepatic lobe. Remove the stylet, and rapidly insert the cutting prongs. If properly placed, the cutting prongs should not go through the entire hepatic lobe. Advance the outer cannula over the blades of the cutting prongs, thus entrapping the hepatic tissue material within the cutting prongs. Remove the biopsy needle. Using a wooden applicator stick, carefully place the biopsy specimen into fixative. Biopsy samples can be used to prepare slides for cytologic examination, and the biopsy needle may be cultured. Close the abdominal incision in the routine manner.

Another liver biopsy technique entails use of a Tru-Cut biopsy needle. Place the dog in dorsal recumbency. Clip a 5-cm² area over the triangle formed by the xiphoid cartilage and left costal arch, and prepare the area as for aseptic surgery. Make a small paramedian incision large enough to accommodate a sterile otoscope head 7 mm in diameter. Use a sterilized halogen-illuminated otoscope speculum to visualize the liver. Pass a Tru-Cut biopsy needle through the otoscope cone to directly obtain a biopsy specimen of the liver.

Nasal Biopsy

The technique for performing diagnostic nasal biopsies is sufficiently complex (and bloody) that it is generally recommended to refer patients in need of this procedure to a specialty or referral hospital that has rhinoscopic and/or computed tomography capabilities. Blind biopsies of dogs and cats with chronic nasal disease, especially if associated with bleeding, can be associated with significant risk, including penetration of the cranium. No one likes nasal biopsy results that indicate “normal brain.”

Renal Biopsy

Renal biopsies can be valuable in confirming or eliminating a diagnosis of renal disease that is based on history, physical examination, and radiographic and laboratory data (Box 4-5). In addition, biopsy may be a way of arriving at a prognosis in generalized renal disease and a better means of evaluating the type of treatment to be instituted. Ultrasonographic guidance can prove valuable during renal biopsy for placing the needle into the tissue desired and avoiding complications.
Patient Preparation

Before renal biopsy, the animal should have a baseline coagulation profile that includes, at the very least, an activated coagulation time and platelet count. A buccal mucosal bleeding time may be indicated if there is any history of spontaneous bleeding in a patient with a normal platelet count. Obtain biopsies from the renal cortex. Administer fluids to patients before and after biopsy.

Many patients with generalized renal disease are critically ill and debilitated, and general anesthesia is contraindicated. In these cases, a neuroleptanalgesic agent may be used for sedation. If the animal is a good anesthetic risk and renal function will permit it, use inhalation anesthesia.

Technique

When bilateral renal disease is documented, select the left kidney for biopsy because it is more accessible than the right kidney. With the anesthetized patient in right lateral recumbency, surgically prepare the skin behind and below the junction of the costal arch at the level of the second and third lumbar vertebrae. Make a 2-inch paralumbar incision parallel to, but just behind, the costal arch. Dissect muscle and fascia until the peritoneum is visible. Carefully open the peritoneal cavity. Digitally feel for and examine the caudal pole of the left kidney. Guide the needle toward the posterior pole of the kidney with the index finger. Immobilize the kidney against the body wall and insert the Tru-Cut biopsy needle, with the biopsy notch exposed into the parenchyma of the kidney. Capture the biopsy specimen by sliding the outer sleeve of the needle over the (now embedded in the kidney) biopsy notch. Remove the needle and gently lift the biopsy sample from the needle and place it into formalin. Evaluate the site for hemorrhage. Once bleeding is controlled, a second biopsy specimen may be collected. Once bleeding from the biopsy site has stopped, the incision can be closed. In dogs, renal biopsy can be performed under ultrasound guidance using probes with channels for biopsy needle insertion.

Bone Biopsy

Evaluation of bone marrow is indicated in patients with evidence of persistently diminished cell counts of any or all cell lines (WBCs, RBCs, platelets) or evidence of morphologically abnormal cells in peripheral blood. Bone marrow aspiration and bone biopsy are extremely helpful but underused diagnostic procedures. The availability of inexpensive, high-quality biopsy needles makes these procedures safe and easy to perform (once experience is gained).

Conventional practice today is to obtain a bone marrow aspirate (cytopathologic examination) and a bone biopsy specimen from the same patient during the same procedure when changes in the peripheral blood justify this level of diagnostic testing. Bone marrow aspiration technique is described earlier in this section.

Two types of bone biopsy needles are available. The most commonly described procedure involves use of the Jamshidi biopsy needle, an 11- to 13-gauge needle that ranges in length from 5 to 10 cm (see Figure 4-11). The needle contains a stylet that extends beyond the needle tip by 3 to 4 mm. Because of the size of the Jamshidi needle, its use is limited to medium and large dogs. For bone biopsies in cats and small dogs, the Illinois bone marrow aspiration needle is preferred (see Figure 4-10), which is a 15- to 18-gauge needle available in lengths ranging from 2.5 to 5.0 cm.
Patient Preparation

The patient usually is sedated or anesthetized for the procedure. Although some patients will tolerate this procedure when performed under local anesthesia only, the additional manipulation required to obtain a high-quality sample justifies sedation. In some cases the patient is sufficiently obtunded that sedation is neither indicated nor required.

Technique

The technique for bone biopsy is the same regardless of the needle used. Once the site has been selected (usually the same sites selected for bone marrow aspiration: head of the humerus, wing of the ilium, ischial tuberosity, proximal femur), clip the hair and surgically prepare the skin. Make a small stab incision in the skin over the site selected. Pass the needle, with stylet in place, through the incision and subcutaneous tissues until the needle tip makes firm contact with bone. Advance the needle using steady, increasing pressure and stable rotation. Rotation, in this case, means rotating the needle back and forth to the left 180 degrees and then to the right 180 degrees. Once the needle is situated in the bone (about 0.5 cm penetration only), stop. Carefully remove the stylet. Continue the penetration by gradually applying additional pressure and simultaneously rotating the needle.

The usual depth of penetration varies from 1 inch to as much as 3 inches. On reaching the desired depth, remove the needle by continuing to rotate as described but gradually withdrawing the needle from the bone. An obturator is provided to push the sample out of the bone. Place the core of bone directly into buffered formalin and submit it for histopathologic examination (decalcification will be required, which takes a little longer).

Special Considerations

Some authors recommend carefully rolling the bone core across a glass slide (for cytopathologic examination) before placing the bone in formalin. Most pathologists do not recommend this because additional handling of the biopsy sample can sufficiently disrupt the architecture of the tissue and compromise the quality of the biopsy (besides upsetting the pathologist). Note also that the needle can, with a little gentle manipulation, be reinserted into the hole from which the biopsy sample was obtained. Because the Illinois needle and the Jamshidi needle accommodate a syringe, it is possible to obtain (if done quickly, to prevent clotting) a bone marrow aspirate from the same site. Place that sample directly onto glass slides or (recommended) into 4% EDTA and mix it before making slides.

There are no specific requirements for postbiopsy care of the patient. Clean the blood from the skin using hydrogen peroxide; sutures generally are not required.

Prostate Biopsy

See Urinary Tract Procedures.

Additional Reading


Osborne CA, Fincio DR: Canine and feline nephrology and urology, Baltimore, 1995, Williams & Wilkins.

BLOOD GAS: ARTERIAL

The femoral and dorsal pedal arteries can be punctured to obtain an arterial blood sample for blood gas and electrolyte analyses (see Section 1 for information on indications and interpretation of results).

Patient Preparation

To obtain a sample of arterial blood gas, place the patient in lateral recumbency and restrain the patient in a manner similar to that for a medial saphenous venipuncture.

Technique

A 25-gauge needle affixed to a tuberculin syringe is preferred for arterial puncture. Prepare the tuberculin syringe by coating it with heparin and forcing all the heparin out except for that left in the hub of the needle. Pull back on the plunger of the syringe slightly to facilitate visualizing the point at which the artery is entered. Arterial blood initially will enter the syringe without the plunger being drawn back.

For collection of blood from the femoral artery, and once the patient is sufficiently restrained, the individual collecting the arterial blood sample should palpate the medial aspect of the limb over the proximal medial femur until the femoral pulse is palpated. Direct the needle at a 30- to 45-degree angle, inserting the needle slowly, watching for a flash of blood in the hub of the needle (Figure 4-22). Gradually withdraw the plunger to facilitate blood entering the syringe. Collect 0.4 to 0.5 mL and immediately submit the blood for analysis, or place it on ice until the analysis can be performed.

To obtain blood from a dorsal pedal artery, place the patient in lateral recumbency and extend the rear limb as for a medial saphenous blood sample collection. The person obtaining the blood sample should pull the paw of the down leg in the nondominant hand toward his or her body, rotating the limb slightly in a medial direction to palpate the arterial pulse. Palpate the pulse in the dorsal pedal artery on the dorsomedial aspect of the tarsus. Gently insert the needle at a 30-degree angle into the artery, watching carefully for a flash of blood into the syringe. When the necessary amount of blood has filled the syringe, remove the needle and place pressure over the site of arterial puncture for a minimum of 2 minutes.

Evacuate excess air from the syringe and needle, and cap the needle with a red rubber stopper to prevent air from entering the needle and syringe. Place the sample on ice until analysis, if arterial blood gas analyses cannot be performed immediately.

Figure 4-22: Technique for collecting arterial blood from the dorsal pedal artery of a dog.
Surgical Cutdown

In the event that percutaneous access to a peripheral artery is not possible, the femoral artery can be isolated and prepared for surgical cutdown. After appropriate aseptic skin preparation, make a 4- to 5-cm incision in the skin over the femoral artery. Find the caudal edge of the sartorius muscle by blunt dissection and then reflect it anteriorly to expose the underlying femoral artery, vein, and nerve. Taking care to avoid tearing any vessel branches, gently isolate up to 2 cm of the femoral artery from the surrounding fascia. Visually direct the needle into the artery at this point. Alternatively, catheterize the artery in the event repeated arterial samples are required. Elevate the femoral artery by preplacing two stay sutures beneath the artery and then elevating the vessel to the level of the skin. Insert a long catheter-over-the-needle system into the lumen of the artery without penetrating the deep wall. Gently insert the catheter into the vessel, remove the needle, and cap and flush the catheter. Close the incision and affix the catheter to the skin via a tape tag sutured to the skin.

Additional Reading

Cerebrospinal Fluid Collection
The collection of CSF is an important diagnostic procedure indicated for patients suspected of having significant intracranial or certain spinal diseases. However, it is our opinion that the technique to safely perform this procedure requires hands-on training and, preferably, prior experience before attempting the procedure in a clinical patient. Attempting to perform CSF collection from a written description in a text is not recommended. Although this procedure is generally safe when performed correctly, significant injury and even death are possible, despite the experience of the individual performing the procedure.

Electrocardiography
The electrocardiogram provides a fast, efficient way to obtain considerable data about a patient’s cardiovascular status. Electrocardiography is a clinical test and must be correlated with clinical findings (Box 4-6). Keep in mind that an electrocardiogram measures only electrical activity of the heart as seen on the body surface at any one instant. Electrical disorders of the myocardium can be transient or intermittent and, as such, can be missed on a single electrocardiogram.

Interpretation of the Electrocardiogram
Read each electrocardiogram using a definite system. Begin by examining the lead II rhythm strip: Is there a P wave for every QRS complex? Is there a QRS complex for every P wave? Do all the P waves look alike? Do all the QRS complexes look alike? Are the P wave and QRS complex consistently related to each other?

**Box 4-6  Indications for Performing an Electrocardiogram**
- Detect enlargement of any of the cardiac chambers
- Diagnose cardiac arrhythmia
- Identify effects of electrolyte imbalances, especially potassium
- Monitor response to and direct cardiac drug therapy
- Develop prognoses (degree of change in heart function over time)
If the answer to any of these questions is no, proceed to identify the abnormality. Next, determine the rate, rhythm, and wave character—that is, evaluate measurements of the P wave, PR interval, and QRS complex. Evaluate the ST segment, T wave, and QT interval. Use all leads to determine the axis and any miscellaneous criteria.

**Heart Rate**

Depending on the type of electrocardiographic equipment used, there are several methods for determining heart rate from the electrocardiographic tracing. Many electrocardiographs compute the heart rate and print that on the tracing. However, in patients with a significant dysrhythmia, these calculations can be flawed and should be verified manually when a question exists. Small linear lines or demarcations at the top of the electrocardiogram paper can be used to determine the heart rate. At a paper speed of 50 mm/second, the time between adjacent marks is 1.5 seconds. Counting the number of QRS complexes (or R waves) between just two of these divisions and multiplying by 20 equals the heart rate in beats per minute (Figure 4-23). For those inclined to higher mathematics, the heart rate also may be determined by counting the number of small squares between R waves (at a paper speed of 50 mm/sec) and then dividing into 3000 (Box 4-7).

**Heart Rhythm**

The normal heart rhythm is sinus in origin. For every QRS complex there is a P wave (Figure 4-24). The P waves are related to QRS complexes (P-P interval is constant). Sinus arrhythmia, sinus arrest, and wandering pacemaker are normal rhythm variations. In sinus arrhythmia, the P-P interval is irregular. The pauses are never longer than twice the usual P-P interval (Figure 4-25). A wandering pacemaker means that the P waves vary in height and may even be negative temporarily (Figure 4-26). Sinus arrest is defined as a prolongation of the P-R interval longer than twice the usual P-P interval.

![Electrocardiogram](image)

**Figure 4-23:** Using the electrocardiogram to determine heart rate. The distance between R waves is 20 small boxes: 3000/20 = 150 beats/min. (Paper speed is 50 mm/sec.)

<table>
<thead>
<tr>
<th>Box 4-7 Normal Heart Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dog</strong></td>
</tr>
<tr>
<td>- Large dogs: 60 to 100 beats/min</td>
</tr>
<tr>
<td>- Medium-sized dogs: 80 to 120 beats/min</td>
</tr>
<tr>
<td>- Small dogs: 90 to 140 beats/min</td>
</tr>
<tr>
<td><strong>Cat</strong></td>
</tr>
<tr>
<td>- Puppies: Up to 220 beats/min</td>
</tr>
<tr>
<td>- Domestic cats: 140 to 250 beats/minute</td>
</tr>
</tbody>
</table>
The normal P wave is 0.04 second × 0.4 mV (two boxes wide × four boxes tall) for the dog and 0.04 second × 0.2 mV for the cat. In P mitrale (left atrial enlargement), the P wave is wider than 0.04 second. In P pulmonale (right atrial enlargement), the P wave is taller than 0.4 mV for the dog and 0.2 mV for the cat.

**PR Interval**

The PR interval is measured from the beginning of the P wave to the beginning of the QRS complex. The normal interval is 0.06 to 0.13 second (3 to 6.5 boxes wide) for the dog and 0.06 to 0.08 second for the cat. In first-degree atrioventricular heart block, the PR interval is prolonged. The PR interval is sometimes useful in monitoring the effects of digitalis therapy.
The QRS complex duration is measured from the beginning of the Q wave to the end of the S wave. Normal duration is up to 0.04 second in cats, 0.05 second in small dogs, and 0.06 second in large dogs. A QRS complex that is too wide indicates left ventricular enlargement (Figure 4-27). An R wave that is too tall indicates left ventricular enlargement. The amplitude is measured from the baseline to the top of the R wave (Figure 4-28). The normal R wave can be up to 0.8 mV tall in cats, 2.5 mV in small dogs, and 3.0 mV in large dogs.

**Figure 4-26:** A, The wandering pacemaker in this recording is suggested by the slightly negative P waves in some of the complexes. Negative P waves of this nature result from vagal depression of the sinoatrial node and the development of a junctional atrioventricular nodal rhythm. B, Marked sinus arrhythmia and a wandering pacemaker result in a decreased heart rate (increased R-R interval) and negative P waves in the fifth complex. As the pacemaker returns to the sinoatrial node, the rate increases, and positive P waves of varying amplitude result in the sixth and seventh complexes.

**QRS Complex**

The QRS complex duration is measured from the beginning of the Q wave to the end of the S wave. Normal duration is up to 0.04 second in cats, 0.05 second in small dogs, and 0.06 second in large dogs. A QRS complex that is too wide indicates left ventricular enlargement (Figure 4-27). An R wave that is too tall indicates left ventricular enlargement. The amplitude is measured from the baseline to the top of the R wave (Figure 4-28). The normal R wave can be up to 0.8 mV tall in cats, 2.5 mV in small dogs, and 3.0 mV in large dogs.

**Figure 4-27:** In these two examples of left ventricular enlargement, the QRS complexes have normal configuration but are too wide. A, This QRS complex from a miniature poodle is 0.07 second (three boxes) wide. B, This QRS complex from a Doberman Pinscher is 0.09 second (four boxes) wide. A small dog such as the Poodle should not have a QRS complex wider than 0.05 second (wider), and the larger dog’s QRS complex should not exceed 0.06 second (three boxes). Because each dog’s QRS complex is too wide, left ventricular enlargement is diagnosed in both cases. The Doberman Pinscher has no P waves because it is in atrial fibrillation. (Paper speed is 50 mm/sec; 1 cm equals 1 mV.) (From Edwards NF: *Bolton’s handbook of canine and feline electrocardiography*, ed 2, Philadelphia, 1987, WB Saunders.)
ST Segment

The ST segment is between the end of the S wave and the beginning of the T wave. Normally the ST segment lies on the baseline and then dips into the T wave. Slurring of S into T indicates left ventricular enlargement and is seen when the S wave slurs into the T wave and no ST segment is discernible. The ST segment is elevated if it lies more than 0.1 mV (one box) above the baseline (>0.2 mV in CV_{LL} and CV_{LU}). Elevation of the ST segment may occur with hypercalcemia or myocardial hypoxia. The ST segment is depressed if it lies more than 0.1 mV (one box; >0.2 mV in CV_{LL} and CV_{LU}) below the baseline. Depression of ST may be seen with myocardial ischemia, hypoxia, or hypocalcemia.
QT Interval
The QT interval is measured from the beginning of the Q wave to the end of the T wave. The normal interval is 0.14 to 0.22 second (7 to 11 boxes wide) in dogs and up to 0.16 second in cats. A lengthened QT interval may be seen with hypokalemia or hypocalcemia. The QT interval varies with heart rate and tends to be prolonged when bradycardia occurs. A decreased QT interval may be seen with hypercalcemia.

Mean Electrical Axis
The mean electrical cardiac axis measures the direction (vector) of the cardiac ventricular impulse during depolarization. Therefore the QRS complex is examined in leads I, II, III, aVR, aVL, and aVF. These six leads determine the axis. They are arranged in a manner known as Bailey’s hexaxial lead system (Figure 4-29). The procedure is as follows:

1. Find an isoelectric lead—that is, a lead for which the total number of positive (upward) and negative (downward) deflections of the QRS complex is equal to zero (Figure 4-30). When there is no perfectly isoelectric lead, use the one that comes closest.
2. Find the lead that is perpendicular to the isoelectric lead: lead I is perpendicular to aVF; lead II is perpendicular to aVL; and lead III is perpendicular to aVR.
3. Determine whether the perpendicular lead is positive or negative on the patient’s electrocardiogram. If the perpendicular lead is negative, the axis is at the negative end of that lead (each lead has a plus and a minus pole marked). If the perpendicular lead is positive, the mean electrical axis is at the positive end of the perpendicular lead. For example, if aVL is isoelectric (normally it is), lead II is its perpendicular. If lead II is positive on the electrocardiogram, the axis is +60 degrees. If lead II is negative on the electrocardiogram, the axis is −120 degrees.

![Figure 4-29: Bailey’s hexaxial reference system.](image-url)
Normal Mean Electrical Axis

The mean electrical axis in the normal dog is +40 to +100 degrees; for the cat it is more variable, at 0 to ±180 degrees. Right axis deviation (axis more than +100) indicates right ventricular enlargement in the dog (Figure 4-31). Left axis deviation (axis 0 to +40 degrees) indicates left ventricular enlargement in the dog. When there is biventricular enlargement, the axis usually remains normal. Axis determinations are of less value in the cat because the normal range is so wide (Boxes 4-8 to 4-10).

Figure 4-30: In each of these three leads, the total of the positive and negative deflections equals zero. Each is considered an isoelectric lead.

Figure 4-31: The mean electrical axis in the frontal plane of this electrocardiogram recorded from a Wire Fox Terrier with pulmonic stenosis is approximately 165 degrees. (From Edwards NJ: Bolton’s handbook of canine and feline electrocardiography, ed 2, Philadelphia, 1987, WB Saunders.)
Inappropriate use of endoscopic equipment not only can damage expensive equipment but also can cause serious injury to the patient.

**UPPER RESPIRATORY TRACT: LARYNGOSCOPY AND PHARYNGOSCOPY**

Endoscopy of the upper respiratory tract is among the most important advanced diagnostic and therapeutic tools used in the evaluation of patients that have stertor (snorting), reverse sneeze, stridor (wheezing), and chronic cough. Laryngoscopy is of value in the diagnosis of upper airway obstructions such as eversion of the lateral ventricles, collapsed arytenoid cartilages, hyperplasia of the vocal cords, nodules on the vocal cords, elongated soft palate, collapsed proximal trachea, and traumatic injuries to the neck. Note also, however, that a careful visual examination of the larynx in the anesthetized patient (only) can be highly valuable even without the use of endoscopic equipment—for example, for assessment of laryngeal movement in patients with laryngeal paralysis. Suspected lesions inside the larynx may be difficult to visualize with or without endoscopic equipment. Examination of the trachea and main stem bronchi requires endoscopic evaluation to assess the integrity of the airway for conditions such as collapsed trachea, mediastinal tumors, hilar lymph node enlargement, parasitic nodules (*Filaroides osleri*), and foreign body aspiration. In addition, tracheobronchoscopy is a valuable technique that permits culturing and cytologic examination of material from bronchi involved in chronic respiratory disease. Upper airway obstruction that is not responsive to conservative therapy is an indication for more extensive diagnostic procedures, such as bronchoscopy.
Endoscopes of varying sizes are appropriate for use in examining the larynx and trachea. However, in cats and small dogs, examination of the trachea using equipment as small as a (human) bronchoscope may limit the examination because the endoscope nearly occludes the tracheal diameter. Additional training and/or experience is recommended for performing tracheoscopy in small patients.

One of the most important endoscopic techniques performed in dogs and cats involves examination of the nasopharynx, the upper respiratory compartment above the soft palate. Sometimes called pharyngoscopy, examination entails retroflexion of a small-diameter endoscope (e.g., bronchoscope) 170 to 180 degrees to allow visualization of the space between the posterior nares (choanae) and the larynx (Figure 4-32). This is a common location for

Figure 4-32: A, The appearance of the normal choanae (posterior nares) in a cat. B, The appearance of the choana of a cat with a posterior nasal mass diagnosed as lymphoma.
advanced procedures

foreign body entrapment and occasional tumor development in cats and dogs (Figure 4-33). Pharyngoscopy is the only effective means of examining this portion of the upper respiratory tract in patients that have a history of stertor (snorting) and so-called “reverse sneeze.”

Lower Respiratory Tract: Bronchoscopy

Endoscopic examination of the bronchi and lower airways is a highly diagnostic, occasionally therapeutic procedure indicated in patients presented with persistent cough. As in all endoscopic procedures, the patient is anesthetized for the examination. However, examination of the lower respiratory tract requires considerable attention to patient oxygenation and respiratory status during the examination. The requirement for oxygen to be administered throughout the procedure may be a significant limiting factor unless special accessories are used. In the ideal situation, the patient is a medium- to large-sized dog and the endoscope can be passed through the endoscope using a T adaptor while oxygen and anesthetic are administered simultaneously.

However, in cats and small dogs it is usually not possible to pass an endoscope through the endotracheal tube. The procedure must be done by passing the endoscope directly into the trachea to the level of the right and left main bronchi and probably not much farther. Supplemental intravenous anesthetic is likely to be required because of the time required to complete the examination. Training and/or experience is essential before performing bronchoscopy, particularly in cats and small dogs.

The greatest advantage in performing bronchoscopy is to visualize the integrity of the trachea and, to a limited extent, the lower airways. Airway collapse, not visible on conventional radiography, can be strikingly apparent. Foreign body entrapment, tumors, respiratory parasites, and airway trauma also can be identified with bronchoscopy. In addition, the bronchoscopic examination allows for collection of cytologic samples from discrete areas (airways) within the lower respiratory tract. The ability to perform BAL in patients with reactive airway disease, subclinical or clinical infections, and certain types of tumors can be highly diagnostic.

Gastrointestinal Endoscopy

Flexible fiberoptic endoscopy is a noninvasive, atraumatic means of visualizing the mucosal surfaces of the esophagus, stomach, and colon. Flexible endoscopes are available from several companies at a wide range of prices. To minimize the risk of injury to the animal and

Figure 4-33: Lateral skull radiograph of a dog depicting the proper endoscopic placement for pharyngoscopy.
to reduce the possibility of damage to the endoscope, place animals undergoing endoscopic examination under general anesthesia after routine preanesthetic preparation. A fast of 12 to 24 hours is recommended for most patients undergoing upper gastrointestinal endoscopy. However, for patients with indications of delayed gastric emptying, a longer fast (24 to 48 hours) may be needed to empty the stomach completely. In preparation for colonoscopy, a 24- to 48-hour fast is recommended. Give a high warm-water enema the evening before and again 2 to 4 hours before the procedure. Give such enemas until the return is clear.

**Esophagoscopy**
The clinical signs indicating esophageal disease and a potential benefit of esophagoscopy include repeated regurgitation, excessive drooling, ballooning of the esophagus, anorexia or dysphagia, and recurrent pneumonia. Esophagoscopy allows visualization of the mucosal lining of the esophagus and makes it possible to detect inflammation, ulcerations, dilations, diverticula, strictures, foreign bodies, tumors, and parasite infestations.

**Gastroscopy and Duodenoscopy**
Endoscopic examination of the mucosal aspect of the stomach is indicated when the clinical signs or physical findings suggest the presence of gastric disease or when there is a need for confirmation or clarification of radiographic findings. In most cases, persistent vomiting is the chief complaint. Other clinical signs suggestive of serious gastric disease include hematemesis, melena, weight loss, anemia, and abdominal pain. Gastroscopy allows visualization of the mucosal lining of the stomach and enables detection of inflammation, ulceration, foreign bodies, and tumors. In most dogs and cats the endoscope can be passed into the proximal duodenum. Depending on the patient size and length of the scope, it may be possible to evaluate as much as 12 inches or more of the proximal duodenum.

**Colonoscopy**
Colonoscopy is endoscopic examination of colon, rectum, and anus. The technique is helpful in the definitive diagnosis of lower bowel lesions, such as granulomatous colitis, foreign bodies, tumors, lacerations, and other mucosal abnormalities. The primary indication for colonoscopy is the presence of signs of large bowel disease, which typically include tenesmus and the passage of small, frequent stools containing fresh blood or excess mucus. Endoscopic examination of the colon allows direct visualization of the effects of mucosal inflammation, ulceration, mucosal polyps, malignant neoplasia, and strictures. Histologic examination of mucosal biopsy material will confirm the diagnosis of colonic disease.

The large bowel must be empty for the colonic mucosa to be visualized. The bowel can be emptied by withholding food for 24 hours and performing a colonic irrigation the evening before and again 2 hours before the examination. The material used for the enema must be nonirritating and nonoily. Mildly hypertonic saline solutions such as Fleet enemas work well if given 2 hours before examination so that gas and fluid can be passed completely. However, do not use Fleet enemas in cats or small dogs.

If the general physical condition of the animal is poor and withholding food is not possible, feeding a low-residue diet for 12 to 18 hours before colonoscopy can be helpful. This diet could consist of cooked eggs, small amounts of cooked beef or chicken, and small amounts of carbohydrates, such as a slice of toast or ¼ to ½ cup of moist kibble. Maintain good hydration. If all food is contraindicated, oral electrolyte solutions such as Gatorade (PepsiCo, Purchase, New York) can be used to maintain hydration without moving solids through the intestinal tract.

Give the animal a short-acting anesthetic and place the animal on a tilted table in lateral recumbency with the hindquarters elevated. Perform a digital examination of the rectum and pelvic cavity to ensure that there are no strictures, polyps, or other obstructions. Lubricate the proctoscope thoroughly with water-soluble jelly and pass it gently through the anal sphincter. Press the proctoscope forward slowly and carefully with a spiral motion. If any resistance is encountered, stop the motion, remove the obturator, and inspect the
bowel to determine the cause of the resistance. If possible, replace the obturator and continue forward motion until the instrument is passed its full length. Withdraw the obturator, and observe the mucosa.

The major portion of the examination is conducted as the instrument is withdrawn. To view the colonic and rectal walls completely, one must move the anterior end of the proctoscope around the circumference of a small circle while withdrawing the proctoscope. Occasional insufflation with the inflating bulb is helpful in smoothing out folds of tissue. Repeated instrumentation may produce petechiae and minor hemorrhages that are not pathologic. For examination of the terminal rectum and anus, the Hirschman anoscope provides adequate, convenient visualization.

Newer techniques for visualizing the upper and lower gastrointestinal tract are being used in dogs. The flexible fiberoptic endoscope enables one to visualize and photograph the esophagus, colon, and stomach. One is able not only to visualize lesions of the gastrointestinal tract directly but also to assess motility, take biopsies of lesions, and remove foreign bodies.

**Vaginoscopy**

The ability to visualize directly the vestibule, the vagina to the level of the cervix, and the urethral orifice in female dogs is of particular value in evaluating patients with known or suspected congenital urinary tract disorders, such as incontinence or ectopic ureters and vaginal strictures (congenital or traumatic). Numerous vaginal malformations and chronic infections cause visual changes that are identified easily during endoscopic examination. Frequently the procedure can be conducted in the standing awake patient. Sedation or general anesthesia is indicated when extensive manipulation, catheterization of the bladder, or a vaginal biopsy are indicated. Position the sedated or anesthetized patient in dorsal or ventral recumbency to facilitate orientation during the procedure. If catheterization of the urinary bladder is required during the procedure, dorsal recumbency seems to facilitate visualization of the urethral papilla and insertion of the catheter.

Vaginoscopy entails use of a relatively small, flexible endoscope 4 to 6 mm in diameter or a 2- to 3-mm rigid scope. The flexible scope offers the advantage of a larger biopsy channel and the ability to view the lateral vaginal wall easily. Vaginoscopy is considered an invasive procedure and should be conducted under sterile conditions. Before insertion of the sterilized endoscope, the vulva should be free of obvious debris, should be clipped if necessary, and should be cleaned gently with a surgical soap and rinsed. Insert the scope such that initial position of the tip of the scope is directed toward the anus. As insertion proceeds, the tip of the endoscope reaches the horizontal portion of the vestibule and vagina. When feasible, pass the scope to the level of the cervix. Slight insufflation of the vagina may be useful in dilating the vagina, greatly facilitating the examination. Conducting the examination from the level of the cervix caudally is recommended. This maximizes the ability to visualize critical anatomic features.

**Cystoscopy**

The relatively recent introduction of very small (2-mm diameter) flexible and rigid endoscopes into veterinary medicine allows visual examination of the urethra, trigone, urinary bladder, and right and left ureterovesicular junctions of female dogs and even cats. Such examinations are most useful when obstructive lesions (tumor or calculi) of the urethra or trigone are suspected. Visual examination of the interior surface of the bladder and the capability of collecting biopsy samples make this a particularly useful diagnostic tool in the hands of the experienced clinician.

**Additional Reading**


Numerous techniques are described for administering calories and nutrients to patients that are unable or unwilling to take in, chew, or swallow food. One method, intravenous hyperalimentation, is reserved for patients that are not able to tolerate any food being introduced via the gastrointestinal tract and represents a radical, and ideally transient, departure from normal. However, enteral feeding, which is always preferable to intravenous hyperalimentation, allows the clinician several options for administering food directly into the gastrointestinal tract. Consideration of several variables is critical when one is initiating enteral feeding programs, such as the patient’s diagnosis and attitude, the status of the gastrointestinal tract, and the ability of the patient to digest and absorb food once introduced. In addition, consideration of the type and constituency of the diet provided is important. Although the options available for enteral nutrition are much greater than those for intravenous hyperalimentation, the clinician must consider dietary requirements carefully when planning enteral nutritional support.

When evaluating enteral feeding for the individual patient, the clinician has four basic options: nasoesophageal tube, pharyngostomy tube (least recommended), esophagostomy tube, and percutaneous gastroscopy tube (which can be introduced using an endoscope or with the so-called “blind” technique). All techniques involve use of a polyurethane or silicone feeding tube. The nasoesophageal tube placement technique does not require general anesthesia, and the tube may be inserted using a topical anesthetic only. Each of the other techniques described requires that the patient be anesthetized to ensure proper and safe placement.

NASOESOPHAGEAL INTUBATION

For temporary, short-term feeding, nasoesophageal intubation is a simple technique that works well in cats, puppies, and adult dogs. Patients that are comatose; have severe, persistent vomiting; have esophageal disease or dysfunction; or are unable to swallow are not candidates for this procedure. The objective of the procedure is to place a small-diameter tube (8F to 10F for dogs weighing more than 15 kg and 5F to 8F for small dogs and cats) through the nasal cavity into the distal esophagus. The tube does not have to enter the stomach. When measuring the tube length, measure from the tip of the nose to the eighth or ninth rib (Figure 4-34).

Administer 3 to 5 drops of a topical ophthalmic solution (0.5% proparacaine) directly into one nostril. Hold the head gently upward for a few seconds to allow the solution to reach the back of the nasal cavity. In most patients, it is desirable to wait 1 to 2 minutes and then to repeat the instillation in the same nostril. For larger dogs, 2% lidocaine solution (0.5 to 2.0 mL) gradually instilled into the nostril is an alternative technique to achieve topical anesthesia. Lubricate the tube with a thin coat of a water-soluble lubricant, such as a 2% lidocaine lubricating gel. Pass the tube into the nasal cavity while directing the tube tip medially and ventrally into the ventral meatus. The anatomic shape of a dog’s nostril usually requires directing the tip medially but almost perpendicular to the plane of the nasal cavity to facilitate insertion. Initial resistance (pressure, not pain) usually is perceived, and the patient’s head as expected quickly retracts, leaving the operator holding the tube tip some inches away from the patient’s nose. Be persistent. Repeat the procedure, as necessary, by quickly inserting the first inch or more of the tube into the nostril. With the other hand, push the nasal philtrum up, and with a finger, push the lateral portion of the nostril medially. This will help facilitate movement of the tube into the ventromedial nasal meatus. Once started, the remainder of the technique is relatively straightforward.
As the tube reaches the caudal aspect of the nasopharynx, it should pass directly into the esophagus with little or no resistance. Affix the tube remaining outside the patient to the head or face using a “butterfly” tape, gauze, suture (Figure 4-35), or skin glue (skin glue [Superglue] generally is not recommended because this can result in loss of hair and skin pigment when the glue becomes dislodged).

**Caution:** The tip of the tube can be introduced inadvertently through the glottis and into the trachea. Topical anesthetic instilled into the nose can anesthetize the arytenoid cartilages, thereby blocking a cough or gag reflex. I prefer to check the tube placement with a dry, empty syringe. Attach the test syringe to the end of the feeding tube. Rather than inject air or water in an attempt to auscultate borborygmus over the abdomen, simply attempt to aspirate air from the feeding tube (Figure 4-36). If there is no resistance during aspiration and air fills the syringe, it is likely that the tube has been placed in the trachea. Completely
remove the tube and repeat the procedure. However, if repeated attempts to aspirate are met with immediate resistance and no air enters the syringe, the tube tip is positioned properly within the esophagus. If there is any question regarding placement, a lateral survey radiograph is indicated.

**ESOPHAGOSTOMY TUBE PLACEMENT**

*Patient Preparation*

Less invasive and not requiring endoscopy equipment, esophagostomy tube placement in dogs and cats is an alternative technique to use in patients that have long-term feeding needs. Use a 14F to 20F rubber, polyurethane, or silicone feeding tube placed at the level of the middle of the cervical esophagus to the level of the eighth rib. The technique does require general anesthesia or, in the hands of an experienced individual, short-term intravenous anesthesia. The technique has been described in detail in textbooks (see Marks SL; Additional Reading). To place an esophagostomy tube, first assemble the necessary supplies: large curved Rochester-Carmalt forceps, clipper and clean blades, antimicrobial scrub, gauze squares, red rubber tube, permanent marker, scalpel blade and handle, needle holder, suture scissors, and nonabsorbable suture (0 nonabsorbable, cutting needle).

*Technique*

After placing the patient under general anesthesia and intubating the patient, place the patient in right lateral recumbency and clip the lateral left side of the neck from the ramus of the mandible caudally to the thoracic inlet, and dorsally and ventrally to midline. Note that the left side of the neck is preferred because of the normal anatomic location of the esophagus. However, if there is injury, infection, or mass that prevents placement of the esophagostomy tube in the left lateral cervical region, the right lateral side of the neck can alternately be used.

Next, aseptically scrub the clipped area, and push the Rochester-Carmalt forceps through the mouth into the esophagus. Direct the curved tips of the instrument laterally, so the tips can be visualized under the skin. Use care to note where the external jugular vein lies, to avoid laceration of the jugular vein. Measure the tube from the proposed site of tube entrance to the mid thorax, then label the tube with a permanent marker.

Figure 4-36: Technique for verifying esophageal placement of the tip of a nasoesophageal tube in a cat.
Next, open the curved tips of the instrument, and make a stab incision through the skin, through the open tips of the instrument, into the esophagus. Push the tips of the instrument through the skin incision. Grasp the distal end of the tube with the instrument, and clamp the instrument.

Pull the tube through the skin incision and rostrally out of the front of the mouth. If the tube does not come easily, usually the hinges of the forceps are caught on tissue within the oropharynx or pieces of the endotracheal tube.

Once the distal end of the tube is through the front of the mouth, push the distal end of the tube caudally into the esophagus with a finger or the instrument. As the distal end of the tube is pushed into the esophagus, pull the proximal end of the tube, to add tension to the tube. The proximal end of the tube will flip toward the patient’s nose when the tube is situated in the esophagus. The tube can be taped in place while radiographs are taken to confirm placement.

After radiographs confirm placement in the esophagus, suture the tube in place with two sutures, one purse-string and finger-trap around the tube entrance site, and another deep suture near the atlas, with a second finger-trap.

Finally, place antimicrobial ointment over the tube entrance site, and a loose bandage around the neck. Unlike gastrostomy tubes, esophagostomy tubes can be used immediately, and removed immediately, if the patient chooses to start eating voluntarily after tube placement.

**Special Considerations**

It is important that one first observe the technique being performed by someone with experience before attempting to place an esophagostomy tube for the first time. Although post-placement complications generally are limited to local irritation or minor infection at the site of the stoma in the midcervical region, tube placement into the mediastinum or subcutaneously can occur.

**Percutaneous Gastrostomy Tube Placement**

Percutaneous gastrostomy tubes are used routinely to administer nutrients and medications orally over days or weeks to cats and dogs that cannot have nutrients administered by mouth or that will not eat (e.g., because of feline hepatic lipidosis, oropharyngeal neoplasms, maxillary or mandibular fractures, oral reconstructive surgery, esophageal masses or foreign bodies, or severe pharyngitis). The percutaneous gastrostomy tube is placed so that it extends through the skin and left cranial abdominal wall of the abdomen into the body of the stomach.

**Catheter Preparation**

Catheter preparation for percutaneous gastrostomy tube is as follows:

1. Use the French-Pezzar mushroom-tipped catheter.
2. Cut off 1.5 cm of the open (distal) end of the catheter with scissors.
3. Cut 3-mm holes on either side of the 1.5-cm piece (outer flange).
4. Cut the distal end of the catheter to form a sharp bevel point.
5. Measure the length of the tube from the mushroom tip to 2 cm below the bevel.

**Preparation of the Stomach Tube**

Stomach tube preparation for percutaneous gastrostomy tube is as follows:

1. Use a smooth-ended vinyl stomach tube.
2. Measure the length of the tube needed to reach the stomach by laying the tube along the animal’s side with the rounded end 1 to 2 cm caudal to the last rib.
3. Mark the tube with an indelible marker or adhesive tape at the tip of the muzzle and cut off the excess tube.
4. Put the tube in the freezer for 30 minutes to stiffen the tube before beginning the procedure.
Placement of the Percutaneous Gastrostomy Tube

Placement of a percutaneous gastrostomy tube is as follows:
1. Clip and surgically prepare the skin over the left abdominal wall.
2. Place the mouth speculum between the right canine teeth.
3. Place the stomach tube in the esophagus to the level of the cardia.
4. Rotate the tube counterclockwise while carefully advancing it through the cardia.
5. Turn the tube back clockwise and advance the tube until it can be visualized through the abdominal wall 1 to 2 cm caudal to the last rib (Figure 4-37).
6. Rotate the tube so that the tip lies against the stomach and abdominal wall one third of the distance between the epaxial muscles and the ventral midline.
7. Make a 2- to 3-mm skin incision directly over the lumen of the stomach tube.
8. Use a Sovereign catheter (over the needle) and puncture the abdominal and stomach walls, placing the catheter inside the lumen of the stomach tube. Remove the needle (Figure 4-38).
9. Thread a long, rigid suture through the catheter and advance it through the stomach tube until the end is observed at the mouth end of the tube (Figure 4-39).
10. Carefully remove the plastic catheter from the stomach tube opening and place a hemostat clamp at the end of the suture material.
11. Remove the stomach tube over the oral end of the stiff introduction suture line.
12. Attach the open, beveled end of the French-Pezzar catheter stomach tube to a plastic Sovereign catheter using a mattress suture (Figure 4-40).
13. Force the tip of the rubber stomach tube into the large end of the Sovereign catheter.
14. Advance the catheter tube through the mouth and esophagus into the stomach by placing traction on the abdominal end of the introduction line.
15. The catheter will emerge through the skin incision, followed by the rubber tube. Grasp the tube with forceps and pull it through the incision opening (Figure 4-41, A).
16. Remove the catheter by cutting it off 2 cm below the beveled tip. Pull the rubber tube through the abdominal wall until slight resistance is felt (Figure 4-41, B).
17. Slide the outer flange over the end of the tube down to the skin level (Figure 4-42).
18. Apply antimicrobial ointment and a sterile gauze sponge over the skin incision.
19. Bandage the gastrostomy tube in place (Figure 4-43).

Figure 4-37: Locating the end of the rigid stomach tube at the left lateral abdominal wall. (From Crow S, Walshaw S: Manual of clinical procedures in the dog, cat, and rabbit, ed 2, Philadelphia, 1997, Lippincott-Raven.)
**Figure 4-38:** Placement of the Sovereign catheter through the abdominal and stomach walls and into the lumen of the stomach tube. (From Crow S, Walshaw S: *Manual of clinical procedures in the dog, cat, and rabbit*, ed 2, Philadelphia, 1997, Lippincott-Raven.)

**Figure 4-39:** Threading the introduction line retrograde through the Sovereign catheter and stomach tube. (From Crow S, Walshaw S: *Manual of clinical procedures in the dog, cat, and rabbit*, ed 2, Philadelphia, 1997, Lippincott-Raven.)
**Figure 4-40:** Suturing the introduction line to the beveled end of the gastrostomy catheter. (From Crow S, Walshaw S: *Manual of clinical procedures in the dog, cat, and rabbit*, ed 2, Philadelphia, 1997, Lippincott-Raven.)

**Figure 4-41:** Catheter-tube assembly being pulled through the mouth and esophagus and the stomach and abdominal walls. (From Crow S, Walshaw S: *Manual of clinical procedures in the dog, cat, and rabbit*, ed 2, Philadelphia, 1997, Lippincott-Raven.)
**Figure 4-42:** Diagram showing the inner and outer flanges in place against stomach mucosa and skin, respectively. (From Crow S, Walshaw S: *Manual of clinical procedures in the dog, cat, and rabbit*, ed 2, Philadelphia, 1997, Lippincott-Raven.)

**Figure 4-43:** Full abdominal bandage showing the plugged end of the gastrostomy tube emerging dorsally. (From Crow S, Walshaw S: *Manual of clinical procedures in the dog, cat, and rabbit*, ed 2, Philadelphia, 1997, Lippincott-Raven.)

**Additional Reading**


OPHTHALMIC PROCEDURES

EVALUATION OF TEAR PRODUCTION

Tear production comes predominantly from the tarsal and conjunctival glands and from the accessory tarsal glands. The reflex tear secretors are the main lacrimal gland and the accessory lacrimal glands. The production of normal lacrimal secretions can be tested by using the Schirmer tear test, a standardized filter paper (Figure 4-44) that effectively measures the rate of tear production in millimeters per minute. Schirmer tear strips now are impregnated with a blue dye to facilitate visualization of the distance (in millimeters) that the tear migrates during the 1-minute test.

Patient Preparation

None required.

Technique

Each eye can be tested independently, or both eyes can be tested simultaneously in the cooperative patient. Carefully fold the notched end of the test strip before removing it from the plastic package. Insert the folded end into the lower conjunctival cul-de-sac (Figure 4-45) and begin the timing. Maintain the Schirmer test strip in position by gently holding the eyelids closed but not touching the paper. At the end of 1 minute, note the degree (distance) of wetting that occurred and record it in the medical record. The normal dog and cat should produce wetting over 10 to 25 mm in 1 minute for each eye. Amounts less than that are consistent with keratoconjunctivitis sicca. Amounts greater than 25 mm may be normal or may be consistent with excessive tear production, or epiphora.

FLUORESCIN STAINING OF THE CORNEA

The cornea is composed of various layers of specialized avascular epithelium and stroma. The outer layer, the corneal epithelium, is a highly sensitive, thin layer overlying the corneal stroma, the thickest layer.

Descemet’s membrane is a distinct, thin layer of tissue beneath the stroma. The innermost layer of the cornea is the endothelium. Damage to the corneal epithelium occurs frequently in dogs and cats. Clinical presentation typically is characterized by blepharospasm of the affected eye with or without a visible ocular discharge or conjunctivitis.

Whenever superficial corneal injury is suspected, assessment of the integrity of the corneal epithelium is indicated. Fluorescein dye-impregnated test strips can be used to determine whether the epithelial barrier overlying the corneal stroma has been disrupted and thus can establish the presence or absence of a corneal ulcer (Figure 4-46).
Patient Preparation

None required.

Technique

The test is simple to accomplish. Moisten the dye-impregnated tip of the test strip with a drop of balanced saline solution (or commercial ocular irrigation solution). Gently allow the tip of the test paper to touch the cornea, or sclera, of the affected eye. (In patients with particularly painful, sensitive eyes, use a topical anesthetic to moisten the test strip or apply

Figure 4-45: Placement of a Schirmer tear test strip into the lower conjunctival cul-de-sac of a dog; the test strip is held in place for 60 seconds only.

Figure 4-46: Fluorescein sodium–impregnated test strip used to enhance visualization of a corneal ulcer.
the anesthetic directly to the cornea before testing.) Immediately rinse the eye with a sterile irrigation solution to remove the excess dye (the test strip has a lot of dye; be prepared to catch the excess fluid with 2- × 2-inch gauze).

Promptly examine the eye with a direct, focal light source. Evidence of green dye uptake in the stroma indicates that an ulcer is present. The absence of staining generally indicates that the corneal integrity is intact. One exception exists. The Descemet membrane will not take up fluorescein dye. A patient with a deep corneal ulcer that penetrates through the corneal stroma and allows herniation of the Descemet membrane (descemetocoele) will not demonstrate a positive stain. Careful visualization of the cornea, however, is likely to reveal the presence of a such a serious, deep ulcer.

**Assessment of Nasolacrimal Duct Patency**

**Patient Preparation**
None required.

**Technique**
Fluorescein dye can also be used to assess patency of the nasolacrimal duct. To perform this examination, place a drop of fluorescein dye from a sterile fluorescein strip into the eye and add 1 or 2 drops of a sterile eye wash. After 2 to 5 minutes, examine the external nares with the aid of a cobalt blue filter or Wood light for the presence or absence of fluorescence. A clean, 2- × 2-inch white gauze square touched against the nasal planum also will pick up the green-colored dye if the duct is patent. If dye is present, the lacrimal excretory system is patent and functioning. If epiphora exists but the primary dye test indicates that the lacrimal excretory system is patent, hypersecretion of tear fluid may be implicated as the cause of the epiphora.

Irrigation of the nasolacrimal system is indicated if the primary dye test result is negative. In the dog the nasolacrimal puncta are located 1 to 3 mm from the medial canthus on the mucocutaneous border of the upper and lower lids. In the dog, use a 20- to 22-gauge (in the cat, a 23-gauge) nasolacrimal cannula (**Figure 4-47**). Topical anesthesia often is required. Fill a 2-mL syringe with saline, and attach the lacrimal cannula and pass it into the lacrimal puncta of the upper lid.

**Special Considerations**
Several points should be made about evaluating the nasolacrimal system. Brachycephalic-breed dogs and cats occasionally may have a negative primary dye test result, although no blockage in the nasolacrimal system exists. In flushing the nasolacrimal system of some animals, fluid may not appear at the nose; however, the animal may gag and exhibit swallowing movements, indicating that the fluid has entered the mouth and the system is patent.

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**Figure 4-47:** Lacrimal cannulas used to flush the nasolacrimal ducts.
CONJUNCTIVAL SMEARS, SCRAPINGS, AND CULTURES

Patient Preparation
None required. This procedure is preferably performed without administration of topical anesthesia. Topical anesthetics not only are bacteriostatic but also may distort the cells and compromise the cytologic examination.

Technique
In performing conjunctival scrapings, use a platinum spatula (Kimura spatula), the tip of which has been sterilized. Gently scrape the inferior conjunctival cul-de-sac (Figure 4-48). Place the material on two glass slides. Fix one slide in acetone-free 95% methanol for 5 to 10 minutes, then stain the slide with Giemsa stain. Heat-fix the other slide, and apply Gram stain.

To culture the conjunctiva, use sterile cotton-tipped applicators, fluid thioglycolate medium, and blood agar medium. Evert the palpebral conjunctiva of the lower lid, and pass one side of a sterile cotton applicator, previously moistened with sterile broth or thioglycolate medium, over the palpebral conjunctival surface. Streak the swab onto a sterile blood agar plate, then place the plate in a tube of thioglycolate broth. No topical anesthesia is used before culturing because preservatives present in anesthetics can inhibit the growth of bacteria.

TONOMETRY
Glaucoma is an increase in intraocular pressure incompatible with normal ocular and visual functions. One method used to measure intraocular pressure is tonometry, in which the tension of the outer coat of the eye is assessed by measuring the impressibility, or applanability, of the cornea. Because the measurements based on tonometry involve calculations that have a wide base of variations, tonometry readings are always approximations.

Schiotz Tonometry
The Schiotz tonometer consists of a corneal footplate, plunger, holding bracket, recording scale, and 5.5-, 7.5-, 10.0-, and 15.0-g weights. The principle of the Schiotz tonometer is that the amount that the plunger protrudes from the footplate is related to the indentability of the cornea, which in turn is related to the intraocular pressure. However, use of applanation tonometry today has virtually replaced use of the Schiotz tonometer.

Figure 4-48: Spatula used for performing conjunctival scrapings.
Applanation Tonometry

In applanation tonometry, a very small area of the cornea is flattened by a known force, usually a calibrated burst of air. The advantage of this technique over the indentation (Schiøtz) method is that the errors resulting from ocular rigidity and corneal curvature are greatly reduced. Special equipment is required to perform applanation tonometry (Figure 4-49).

Gonioscopy

The presence or absence of glaucoma, an increase in intraocular pressure, can be determined using applanation tonometry. On the other hand, gonioscopy permits one to visualize and examine the iridocorneal angle and potentially establish the cause of glaucoma. However, specific training and equipment are required not only to perform gonioscopy but also to interpret the result. This procedure is most appropriately performed by an ophthalmologist.

Additional Reading


RADIOGRAPHY: ADVANCED CONTRAST STUDIES

Gastrointestinal Studies

When considering a contrast study of the gastrointestinal tract, it is not unreasonable to question the value of doing the procedure. At issue is the fact that abdominal ultrasound and/or gastrointestinal endoscopy has largely replaced contrast radiography of the gastrointestinal tract and for good reason. Diagnostic modalities such as ultrasound (in the hands of an experienced individual) and endoscopy have a much greater diagnostic yield than the less sensitive contrast study. So why even try? Endoscopes are not available in every practice, and limited access to ultrasound equipment, much less someone who is qualified to use it, puts routine use of advanced diagnostic modalities out of reach for many practices. However, it must be appreciated that with regard to diagnostic value, a radiographic contrast study of the gastrointestinal tract is a far less sensitive diagnostic modality than abdominal ultrasound or endoscopy. The procedure for the gastrointestinal radiographic contrast study is outlined next.

Figure 4-49: Measurement of intraocular pressure using applanation tonometry.
Contrast agents available for gastrointestinal studies include barium suspension preparations or Micropaque (Guérbet, Villepinte, France), and water-soluble agents (Gastrografin [Bracco Diagnostics, Princeton, New Jersey], which is 60% meglumine and 10% sodium diatrizoate). Water-soluble agents are used if bowel perforation is suspected. Undiluted water-soluble agents are hypertonic and should be diluted at a ratio of one part Gastrografin to two parts water. No single procedure is appropriate for all gastrointestinal cases. The clinician must select procedures based on the clinical history and physical findings, apparent location of the lesion within the gastrointestinal tract, endoscopic findings, and results from other imaging studies, such as abdominal ultrasound.

**Contrast Esophagram**
The contrast esophagram also is called *barium swallow*. The decision to perform a contrast esophagram is based on physical evidence of dysphagia (difficulty or pain while attempting to swallow) and/or persistent regurgitation (reflux of swallowed food without effort).

**Patient Preparation**
The procedure necessitates that the animal fast for 12 hours before radiography. Remove all leashes from around the animal’s neck, and obtain survey radiographs of the thorax. In esophageal contrast studies, administer barium suspension contrast medium, 2 to 5 mL/kg body mass. Administration of barium as a contrast material is contraindicated if a perforation of the esophagus is suspected. When the esophagus has been coated with radiopaque material, take lateral, ventrodorsal, and right ventrodorsal oblique thoracic radiographs to visualize the esophagus.

**Technique**
Properly prepared, the barium should be relatively thick and of a pastelike consistency. Position the patient and cassette, and have the radiographic technique set. Give a tablespoonful of barium orally. Make the exposure when the animal takes its second swallow after the barium has been given.

For esophageal studies and barium swallows, sedation with acepromazine and buprenorphine (IV, IM, SQ) will produce no adverse alteration in gastrointestinal motility. For cats, ketamine 10 mg IV and midazolam 0.2 mg/kg (combined) can be administered intramuscularly (IM) with no significant effect in esophageal motility. *Caution: Patients with significant swallowing disorders have a risk of aspiration if contrast material is regurgitated. Sedation can increase that risk.*

In some cases of incomplete esophageal stricture, barium liquid will pass through the esophagus unobstructed, whereas food will not. Veterinarians should mix kibbled food with the barium in this case and allow the patient to eat the mixture just before the radiograph is taken.

**Special Considerations**
Ideally, contrast esophagrams are performed using fluoroscopy rather than conventional radiographs. In this manner it is possible not only to identify strictures and dilatations, if present, but also to obtain a dynamic study of the esophagus that provides valuable information pertaining to swallowing and esophageal motility and function and an opportunity to evaluate sphincter activity at the level of the cardia.

**Upper Gastrointestinal Tract (Stomach, Pylorus, and Small Intestine)**
Contrast studies of the upper gastrointestinal tract are used to facilitate diagnosis of persistent vomiting, hematemesis, unexplained and chronic diarrhea, suspected enteric foreign bodies, and suspected neoplasms and obstructions and for confirmation of displaced intestinal organs, as may be seen in diaphragmatic hernias.

That said, abdominal ultrasound has become sufficiently available to largely replaced the upper gastrointestinal series. With an experienced ultrasonographer, the diagnostic value of abdominal ultrasound far exceeds that derived from evaluating sequential radiographs of...
a patient after oral administration of a contrast medium such as barium. In the event that ultrasound capability is not available, a contrast study of the upper gastrointestinal tract still can be performed. However, the clinician must appreciate that a barium contrast study of the stomach, duodenum, jejunum, and ileum has a low sensitivity as a diagnostic test. That is, negative findings are not expected to correlate well with the absence of clinical disease. A negative study does not rule out disease. Likewise, a contrast study of the upper gastrointestinal tract is not recognized for its ability to confirm a diagnosis of gastrointestinal tract disease, even when disease is present. Perhaps the greatest value in performing the upper gastrointestinal series in a dog or cat today centers on the need to identify a displacement of the stomach and/or small intestine because of an extraluminal mass lesion or congenital defect in the patient. In addition, the use of a microfine barium suspension may facilitate identification of intestinal ulcers, irregularities (e.g., intraluminal neoplasia), and radiolucent foreign bodies. However, variable-diameter, solid-phase radiopaque markers called barium-impregnated polyethylene spheres (BIPS) can be used to assess gastric emptying time, gastrointestinal transit times, and, to some extent, obstructive disorders.

**Technique**

If an upper gastrointestinal study is indicated, follow the technique described:

1. Ensure that the hair of the animal is free from dirt, paint, and foreign material. Bathe the animal if necessary.
2. Withhold food for 18 to 24 hours.
3. If the colon is filled with feces, administer a cleansing enema the evening before performing the procedure. In dogs, give a second enema 3 to 5 hours before the start of the gastrointestinal series.
4. At the start of an upper gastrointestinal series, obtain survey radiographs of the abdomen. Administer a barium sulfate (micropulverized) preparation by stomach tube, or induce the animal to swallow the fluids. Flavored, prepared barium suspensions are available, but they taste bad (personal experience). Dosage levels vary, but for barium suspensions, give approximately 10 mL/kg. As an alternative to barium, use an organic iodide liquid preparation. Administer 0.5 mL/kg by stomach tube. Obtain lateral and dorsoventral radiographs of the abdomen immediately after administration of the contrast material and at 30-minute, 1-hour, and 2-hour intervals. Water-soluble contrast material passes through the gastrointestinal tract in 30 to 90 minutes. Barium suspensions take 60 to 180 minutes to traverse the intestine. The colon usually is filled with barium 6 hours after oral administration and may contain barium for 2 to 3 days after administration.

Barium contrast radiography is contraindicated if perforation of the stomach or upper gastrointestinal tract is suspected. In these cases, use water-soluble contrast media such as the oral diatrizoates because leakage into the abdomen will produce no foreign body granuloma. In addition, do not administer barium sulfate when an obstruction of the lower bowel may be present. In these cases, barium may only contribute to the obstipation.

The following radiographic views are recommended after administration of radiographic contrast material:

1. Immediately after administration of contrast material, obtain ventrodorsal, right lateral, and left lateral views. The right lateral view shows the pylorus of the stomach filled with barium, and the left lateral view shows the cardia and fundic portion filled with barium. The objective is to evaluate the distended stomach and initial gastric emptying.
2. Twenty to 30 minutes after administration of contrast material, obtain ventrodorsal and right lateral views to assess the stomach, pyloric emptying, and the proximal duodenum.
3. Sixty minutes after administration of contrast material, repeat the ventrodorsal and right lateral recumbency views to assess the small intestine.
4. Two hours after administration of contrast material, repeat the ventrodorsal and right lateral views to evaluate passage of contrast material into the colon and complete emptying of the stomach; contrast material should be in the terminal portion of the small intestine.
Guidelines for Passage of Contrast Material through the Gastrointestinal Tract

The passage of contrast material through the normal gastrointestinal tract is variable; however, the following guidelines have been suggested:

1. Contrast material is in the duodenum within 15 minutes in most patients. Excitement can delay gastric emptying time to 20 to 25 minutes.
2. Contrast material reaches the jejunum within 30 minutes and is within the jejunum and ileum at 60 minutes.
3. Contrast material reaches the ileocecal junction in 90 to 120 minutes.
4. At 3 to 5 hours after administration, contrast material has cleared the upper gastrointestinal tract and is within the ileum and the large intestine.

In evaluation of gastrointestinal contrast studies, consider the following criteria: (1) the size of the intestinal mass, (2) the contour of the mucosal surface, (3) the thickness of the bowel wall, (4) the flexibility and motility of the bowel wall, (5) the position of the small intestine, (6) the continuity of the opaque column, and (7) the transit time.

The Barium Enema

Clinical disorders for which the barium enema is indicated in dogs include ileocolic intussusception and cecal inversion (intussusception), mechanical and functional large bowel obstruction, invasive lesions of the large bowel, a mass outside the large bowel compressing the bowel, and inflammation of the lower intestinal tract. Barium sulfate enemas are contraindicated in suspected obstruction of the colon and rupture or perforation of the colon. However, these same disorders also can be identified by ultrasonic examination or colonoscopy, either of which is the preferred diagnostic modality over a barium enema.

Patient Preparation

Twenty-four hours before radiographs, administer a liquid diet only, preferably water. During the 18 to 24 hours before the radiographs, administer a mild high colonic enema or give a saline laxative orally. Do not give any irritating enemas within 12 hours of the scheduled radiographic examination; however, administer isotonic saline solution or plain water enemas before the examination to ensure that the bowel is clear. Obtain survey radiographs of the abdomen, and examine the colon to ensure that this portion of the bowel is clear. Sedation or anesthesia may be indicated.

Technique

Barium may be infused through a catheter into the colon or allowed to flow in by gravity through an enema bag. Do not force barium into the colon under pressure. Do not elevate the enema bag more than 18 inches above the animal.

Cuffed rectal catheters (Bardex cuffed rectal catheters, 24F to 38F, and the Bardex cuffed pediatric rectal catheter, 18F [C.R. Bard, Murray Hill, New Jersey]) can be used in dogs (Figure 4-50). For very small dogs and cats, use smaller catheters. A plastic catheter adapter and a three-way stopcock are needed. Various barium sulfate preparations can be used; however, the final concentration should be 15% to 20% w/v. A commercially available barium enema kit is helpful.

Place the cuffed rectal catheter so that the inflated bulb is cranial to the anal sphincter. Place the animal in right lateral recumbency and fill the colon with contrast material at a dose of 20 to 30 mL/kg. Take the radiographs after infusion of a two-thirds dose of barium. If the colon is not filled, infuse more contrast agent. Obtain radiographs in the ventrodorsal and lateral positions, and determine whether the colon is distended adequately. Remove as much of the contrast material as possible from the colon, and repeat the radiographs.

Insertion of room air at 2 mL/kg into the colon facilitates the evaluation of the colonic surface. Deflate the cuff on the catheter, and remove the catheter from the rectum. Throughout the procedure of filling the colon with contrast material or air, take care not to overdistend the colon, which may lead to rupture.

When reviewing individual radiographs, look for the following radiographic lesions: (1) irregularity of the barium-mucosal interface; (2) spasm, stricture, or occlusion of the bowel
lumen; (3) filling defects; (4) outpouching of the bowel wall caused by diverticulum or perforation; and (5) displacement of the bowel.

Additional Reading

Excretory Urography
Intravenous administration of organic iodinated compounds in high concentrations permits visualization in four phases of excretory urography: (1) the arteriogram, (2) the nephrogram, (3) the pyelogram, and (4) the cystogram (Box 4-11). The arterial phase demonstrates renal blood flow; the nephrogram demonstrates the accumulation of contrast agent in the renal tubules and is used to evaluate renal parenchyma; the pyelogram phase evaluates the urinary collecting system, including the ureters; and the cystogram reveals the collection of contrast agent in the urinary bladder.

Excretory urography does not result in any quantitative information about renal function and is not a substitute for renal function tests. The degree of visualization of contrast material within the renal excretory system depends on the concentration of iodine in the contrast medium, the technique of excretory urography performed, the state of hydration of the patient, renal blood flow, and the functional capacity of the kidneys.

Patient Preparation
An intravenous catheter is prepositioned.

<table>
<thead>
<tr>
<th>BOX 4-11 PATIENT PREPARATION FOR EXCRETORY UROGRAPHY</th>
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<tr>
<td>1. Have the patient fast for 12 to 18 hours.</td>
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<tr>
<td>2. Administer a cleansing enema or give a saline laxative orally 12 to 18 hours before radiography.</td>
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<tr>
<td>3. Ensure that the animal’s hair is free of dirt and debris.</td>
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<tr>
<td>4. Try to limit the animal’s fluid intake in the 12 hours preceding radiography.</td>
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<tr>
<td>5. Empty the animal’s bladder immediately before taking radiographs.</td>
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<tr>
<td>6. Take survey radiographs before administering contrast media.</td>
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</table>
**Technique**

The contrast medium most commonly used is a diatrizoate or iothalamate compound. Administer 850 mg/kg of an iodine compound IV by syringe. Continuous IV “push” is indicated. Obtain a ventrodorsal radiograph at 10 seconds after injection, and repeat ventrodorsal and lateral radiographs 1, 3, 5, 15, 20, and 40 minutes after injection. This method is the current standard technique. If the patient’s blood urea nitrogen level is greater than or equal to 50 mg/dL or the creatinine level is greater than 4 mg/dL, double the dose of contrast material.

Lesions that can be detected by using intravenous urography are renal mass lesions; neoplasia; renal cysts; renal and ureteral traumatic lesions; pyelonephritis; hydronephrosis; renal agenesis; hypoplasia; pelvic and ureteral obstructions (calculi, blood clots); renal parasites; ectopic ureter; and duplication of the collecting system.

**Retrograde Contrast Urethrography**

Retrograde urethrography is a diagnostic tool used to localize diseases of the lower urinary tract of dogs and cats. This method can reveal conditions such as urethral neoplasms, strictures, trauma, calculi, or other anomalies.

**Patient Preparation**

The procedure involves the injection of an aqueous iodine contrast medium into the urethra through a ureteral or balloon-tipped catheter. The radiopaque contrast material is mixed to a threefold to fivefold dilution with sterile lubricating jelly to increase the viscosity. A dilution of 1:3 contrast medium with sterile distilled water or saline also can be used. Before retrograde contrast urethrography is performed, give the animal a cleansing enema. Sedation or anesthesia may be necessary.

**Technique**

Inject 5 to 10 mL of contrast medium. Near the end of the injection, while the urethra is still under pressure, obtain a lateral radiograph. If the urinary bladder is to be distended with contrast material or air, remove urine from the bladder. In the male dog, position the catheter so that the tip of the catheter is distal to the os penis. Inject lidocaine 1 to 2 mL into the urethral lumen to anesthetize the urethra adjacent to the balloon-tipped catheter.

In male cats, retrograde contrast urethrography can aid in defining the extent of urethral damage (stricture) or the presence of urethral calculi. In male cats, use a 4F balloon catheter or a 3.5F Tomcat open-ended urethral catheter. Insert the catheter 1.5 cm into the penile urethra. If the urethra is patent, 2 to 3 mL of contrast material will enable visualization of the urethra, but increased amounts of contrast material (2 to 3 mL/lb) injected into the bladder are needed for maximum distension of the preprostatic urethra. A voiding positive contrast urethrogram is necessary to visualize the distal (penile) urethra. Apply external pressure to the bladder (using a wooden spoon or other external compression device), and radiograph the distal urethra.

**Special Considerations**

Take extreme care with the amount of fluid placed in the bladder if the urethra is occluded by a balloon catheter. Overdistension of the bladder results in hematuria, pyuria, urinary bladder rupture, and mild to severe bladder inflammation. Palpate the bladder carefully during distension, and note the backpressure on the syringe used in filling the bladder.

**Cystography**

Cystography refers to contrast radiographic procedures that facilitate visualization of the lumen and/or contents of the urinary bladder and trigone (Box 4-12). Three procedures can be used to image the urinary bladder: positive contrast cystography, negative contrast cystography (also called pneumocystography), and double-contrast cystography (combination of positive and negative cystography performed in the same patient). Note: Many of the indications for performing contrast cystography are also indications for ultrasound.
examination. Contrast cystography is most useful for characterizing congenital and acquired alterations in the normal anatomy and function of the ureters and lower urinary tract, such as ectopic ureter. Abdominal ultrasound, when available, remains the preferred method for imaging abnormalities within the bladder lumen (e.g., calculi and tumors) and changes within the bladder wall.

**Pneumocystography**

Pneumocystography, also called negative-contrast cystography, involves the insufflation of a soluble gas into the lumen of the urinary bladder to facilitate imaging of any material or tissue within the bladder lumen that otherwise would be obscured by the presence of urine or positive contrast material.

**Patient Preparation**

Prepare the patient as described previously.

**Technique**

Once a urinary catheter has been placed and the urethra is occluded, use a syringe and a three-way stopcock to inject 4 to 10 mL of carbon dioxide or nitrous oxide per kilogram. Palpate the bladder while filling it with gas to avoid overdistension or rupture. Inject air until there is pressure on the syringe barrel or leakage of air around the catheter. Replace any air that escapes during the procedure. Take lateral and ventrodorsal views of the abdomen.

**Caution:** Room air is the most accessible contrast material for pneumocystography and generally can be found in most practices. However, an increased risk of air emboli is associated with the placement of room air into the bladder under positive pressure, particularly in patients with hematuria.

**Special Considerations**

Pneumocystography is not an innocuous procedure; fatal venous air emboli have occurred in dogs and cats. This complication is seen most commonly in cases of severe hematuria. Ultrasound or positive contrast cystography is preferred over pneumocystography in such cases if a soluble gas is not available. If possible, use a gas that is readily soluble in blood (such as carbon dioxide or nitrous oxide) for bladder insufflation.

**Positive Contrast Cystography**

The injection of radiographic contrast material into the urinary bladder is referred to as contrast cystography or positive contrast cystography. When ultrasound examination is not available or not feasible, the clinical and radiographic findings noted in Box 4-13 justify the use of contrast radiography to image the bladder.

The same principles of preparation apply as for obtaining a pneumocystogram. Use a urethral catheter with a three-way valve or a small Foley catheter with an inflatable cuff. Organic iodides are the contrast material of choice and should be used in 5% to 10% concentrations.

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**BOX 4-12 INDICATIONS FOR CYSTOGRAPHY**

- Incontinence unresponsive to medical treatment, especially in young dogs
- Persistent hematuria (*Warning: Pneumocystography is contraindicated.*)
- Stranguria
- Pyuria
- Persistent crystalluria
- Significant proteinuria
- Dysuria
- Persistent or recurrent urinary tract infection

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Double-contrast Cystography

Double-contrast cystography also can be performed in patients for which a positive contrast study is not diagnostic, yet there is reasonable indication for an intraluminal lesion. In this case, the same urinary catheter as used for the contrast study, remove all remaining urine and contrast material. If necessary, inject 2 to 5 mL of an aqueous organic iodine contrast material into the bladder. Gently roll the patient over in an attempt to coat the bladder with contrast material. Then distend the bladder with air in the same manner as described for pneumocystography.

Some of the lesions routinely diagnosed with the aid of cystography are calculi (Table 4-8); neoplasia; cystitis, if proliferative changes are present; muscle hypertrophy; bladder diverticula; duplications; adhesions, especially uterine stump infection; persistent urachus; ruptures; and atonic bladder.

Additional Reading
Osborne CA, Finco DR: Canine and feline nephrology and urology, Baltimore, 1995, Williams & Wilkins.

REPRODUCTIVE TRACT: FEMALE

Vaginal examination is indicated for collection of material from the mucosal wall for culture and exfoliative cytologic examination and for vaginoscopic examination of vaginal and cervical mucosa (Box 4-14).

| TABLE 4-8 Radiopacity of Cystic Calculi on Plain Abdominal Radiographs |
|---------------------------------|-----------------|
| Calculus Composition           | Density                  |
| Calcium oxalate                | Radiopaque              |
| Calcium carbonate              | Radiopaque              |
| Triple phosphate               | Radiopaque—small calculi may be nonradiopaque |
| Cystine                        | Variable density—may have radiopaque stippling |
| Uric acid and urates           | Nonradiopaque           |
| Xanthine                       | Nonradiopaque           |
| Matrix concretions             | Nonradiopaque           |

Examination of the vagina for culture and cytologic or vaginoscopic examination occasionally can be performed in the cooperative patient without the use of sedation or anesthesia. An assistant is used to restrain the patient on an examination table. Bitches that can be restrained for other minor examinations (ears, teeth, toenails, anal sacs, and blood samples) often will tolerate vaginal examinations. Those that need further restraint may require sedation or administration of a short-acting barbiturate anesthetic.

**Patient Preparation**

Trim long perivulvar hair and cleanse the perineum with a germicidal or surgical scrub such as povidone-iodine. Water and germicidal soap usually will not control surface contamination by *Pseudomonas* and *Proteus* species, which frequently contaminate culture swabs. In dogs with long tail hair, it is appropriate to wrap the tail with gauze before the procedure to prevent bacterial contamination.

**Technique**

If vaginal culture is indicated, this procedure should be conducted first to avoid contamination induced by the general examination. Pass a sterile, warm vaginal speculum with only a thin coating of lubricating gel into the posterior vagina while an assistant spreads the vulva. Guide the speculum into the vagina by placing the speculum into the vulva just at the dorsal commissure of the vulva and applying pressure up and out against the commissure. Direct the speculum dorsally toward the rectum until meeting resistance, and then direct it horizontally into the cranial vagina. This procedure bypasses the clitoral fossa and enables visualization of the urethral opening and pelvic arch.

Take a guarded culture swab (swab covered by a protective plastic pipette) from its individual sterile bag and pass it inside the vaginal speculum to the anterior vagina or cervical area. Then expose the swab from the protective plastic tubing and rotate it against the mucosa. Retract the swab into the protective plastic tubing and carefully remove it from the vagina. The protected swab then may be placed back in its original sterile bag until it is processed for culture (30 minutes) or placed in Amies transport medium with charcoal. Amies transport medium with refrigerator packs and a Styrofoam-insulated mailing box will retain fastidious organisms for 72 to 96 hours. Process bacterial, *Mycoplasma*, and *Ureaplasma* cultures for potential infectious agents. Viral transport medium can be used for a separate sterile swab if viral agents such as the genital form of canine herpesvirus are suspected.

Immediately after the swabbing for culture, while the vaginal speculum is still in place, advance a clean or sterile swab moistened with sterile physiologic saline solution carefully into the anterior vagina to make a smear for cytologic examination. Gently scrape vaginal epithelial cells from the ceiling of the vagina at or cranial to the region of the external urethral orifice. Collect samples from the region of the clitoral fossa, which is lined by stratified squamous epithelium at all stages of the estrous cycle. Gently rub the swab on the vaginal mucosa. Remove the swab and roll it smoothly onto two or three clean glass slides.

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### BOX 4-14 EQUIPMENT TO USE FOR EXAMINATION OF THE CANINE VAGINA

<table>
<thead>
<tr>
<th>Item</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sterile vaginal speculum (e.g., adjustable spreading, stainless steel, or disposable plastic; cylindric; glass, plastic, stainless steel, or nylon)</td>
</tr>
<tr>
<td>Sterile otoscope heads of variable size for small dogs</td>
</tr>
<tr>
<td>Sterile protected culture swabs (Teigland type or other)</td>
</tr>
<tr>
<td>Sterile culture swabs (Culturettes)</td>
</tr>
<tr>
<td>Amies transport medium with charcoal</td>
</tr>
<tr>
<td>Viral transport media</td>
</tr>
<tr>
<td>Glass slides and coverslips</td>
</tr>
<tr>
<td>Sterile proctoscope (Welch Allyn, human pediatric type) or other endoscope, flexible or rigid</td>
</tr>
<tr>
<td>Sterile offset biopsy punch</td>
</tr>
</tbody>
</table>

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The smears may be fixed immediately in 95% alcohol, sprayed with a commercial fixative or hair spray, or left to dry in air.

A drop of new methylene blue stain placed on a coverslip and inverted on the smear can be used to examine a wet mount preparation immediately. This stain is not permanent and precipitates when it dries, and new methylene blue–stained smears cannot be used for comparison with other smears made later in the cycle. The Diff-Quik or Leukostat stain is a permanent stain that can be submitted for review by a pathologist. Examine the smear for stage of estrous cycle and evidence of active inflammation. Compare these findings with culture results and vaginoscopic findings to interpret evidence for an active genital tract infection, a carrier state of a potential infectious agent, or a possible contaminant at culture. A diagnostic laboratory with the ability to isolate specific infectious agents should indicate the number of organisms (few, moderate, many, or heavy) and report whether the isolates are pure or mixed and their significance.

**Examination of the Vagina**

The vagina of the bitch is long in comparison with that of other domestic animals, hence digital examination of the cervix, and in many cases the urethral orifice, is not feasible. The mucosa forms longitudinal folds. The clitoris is in a well-developed fossa in the floor of the vestibule. The vagina can be visualized completely with a small, sterile proctoscope or flexible endoscope. Lubricate the warmed, sterile instrument, and pass it to the region of the cervix. Examine first without insufflation for true color and vaginal fluids or discharge. When insufflation is performed while the vulva is compressed around the sterile proctoscope, the vagina expands and its entire wall can be viewed completely as the instrument is withdrawn.

The normal canine vagina has a uniform light pink color and longitudinal folds. During proestrus and estrus, the folds become more prominent and cross-striations give the surface a cobblestone appearance. This cobblestone appearance remains smooth when estrogen levels are high but quickly becomes angular (cobblestone appearance) when estrogen levels drop during the luteinizing hormone peak (ovulation), and progesterone levels increase. This change can be used to indicate ovulation and the ideal time for breeding. The hyperemia causes the vagina to appear reddish and congested. The pressure of air insufflation balances the mucosa. The canine vulva has a large cranial dorsal median fold that may obscure the cervix. In fact, ridges near the dorsal fold may give a false impression that this fold is the cervix. During estrogen stimulation, the cervix may be open and uterine blood may be escaping. In the management of dystocia, the vaginoscope can be used to detect puppies in the birth canal and to diagnose malpositions and aid in the correction of these conditions.

During the endoscopic examination, small tumors or polyps can be removed or large masses can be sampled with the biopsy punch. Ulcers or erosions can be cauterized, and foreign bodies can be removed.

A complete vaginal examination must include careful palpation of the vaginal wall and pelvic canal. This palpation is accomplished by digital examination through the vulva (using a sterile glove) and is assisted by palpation through the posterior abdominal wall. Incomplete hymen rings, vaginal fibrous stenotic rings, or pelvic malformation can be diagnosed. A digital rectal examination may be needed for vaginal masses or pelvic deformities.

**Estrous Cycle: Staging and Cytologic Findings—Canine**

The canine reproductive cycle begins at the age of 6 to 12 months and repeats at intervals of 4 to 12 months. In the average bitch, ovulation occurs spontaneously 1 to 3 days after the onset of estrus; in normal bitches ovulation may occur 3 days before to 11 days after the onset of estrus. Sperm live in the uterus of the estrous bitch up to 11 days, and the ovum lives up to 5 days after ovulation. The fertilized ovum takes 4 to 10 days to reach the uterus, and implantation takes place 18 to 20 days after ovulation. The gestation period from the first breeding is 57 to 72 days and from the luteinizing hormone peak is 64 to 66 days.
Anestrus
Anestrus is characterized by dryness of the mucosa and a thin vaginal wall with stratified squamous epithelial cells a few cells to several layers thick but without cornification. Noncornified epithelial cells and WBCs are present in a ratio of 1:5 in the vaginal smear. The WBCs are polymorphonuclear. The noncornified epithelial cells are 15 to 51 nm in diameter and have round free edges, granular cytoplasm, and large nuclei with distinct chromatin granules. The period of anestrus is 2 to 3 months or longer in some breeds.

Proestrus
In proestrus the vaginal wall is thicker than in anestrus, and the mucosa shows prominent cornified squamous epithelium (20 to 30 cells thick) with rete pegs. The longitudinal and transverse vaginal folds are thick, smooth, and round. The vaginal wall becomes impervious to WBCs, but there is extravasation of RBCs to the surface epithelium. The RBCs are discharged. Vaginal smears show predominantly RBCs and noncornified epithelial cells, which become cornified as proestrus progresses. WBCs are present, but their numbers decrease as estrus approaches. Debris and bacteria are abundant for 7 to 10 days.

Estrus
The vagina is thick with longitudinal and transverse folds that become angular as estrogen levels decrease and progesterone levels increase. Fluid is abundant, often tinged with blood. Noncornified epithelial cells and WBCs are absent. Cornified epithelial cells, which are polyhedral and contain pyknotic nuclei or no nuclei, are predominant; their presence seems to be related to the appearance of flirting by the bitch and acceptance of the stud. WBCs reappear about 36 to 96 hours after ovulation. Bacteria and debris are absent during estrus, but they are seen again in the smears after ovulation when WBCs reappear 7 to 10 days later.

Diestrus
The number of WBCs increases rapidly, the number of cornified epithelial cells decreases, and the number of noncornified epithelial cells increases. After 5 to 7 days, the number of WBCs may decrease to 10 to 30 per field.

After parturition, much cellular debris, WBCs, RBCs, and a few epithelial cells are present for several days, until placental sloughing is complete. The presence of masses of degenerate WBCs (and bacteria) indicates metritis or endometritis. The continued presence of blood-tinged fluids containing abundant RBCs, a few noncornified epithelial cells, and occasional WBCs (nontoxic) plus necrotic cells for months postpartum is evidence of subinvolution of placental sites.

ESTROUS CYCLE: STAGING AND CYTOLógIC FINDINGS—FELINE
Most of the characteristics just discussed that apply to bitches also pertain to queens. However, the small size of the feline vagina precludes palpation and early vaginoscopy. A sterile, warm, small-animal otoscope speculum enables fairly good visualization of the vaginal mucosa and can be used with a small, 4-mm–diameter sterile swab to obtain smears for culture procedures. Use of the speculum is easiest after parturition or during estrus.

Vaginal cells for cytologic examination can be obtained with a moistened 3-mm cotton swab (Calgiswab) inserted 2 cm into the vagina. In some cases, flushing the vagina with sterile saline injected and aspirated with a clean glass eyedropper is more successful. Use of an eyedropper may trigger ovulation, as it simulates coitus.

Unlike the bitch, the queen shows no diapedesis of RBCs during proestrus or throughout the estrous cycle. Cytologic examination of feline vaginal smears reveals the following by stage of the estrous cycle.

Anestrus or Prepuberty
Cytologic examination reveals scarce debris and numerous small, round epithelial cells with a high nuclear/cytoplasmic ratio, frequently in groups (seasonal: from September to January in the Northern Hemisphere).
Proestrus
Cytologic examination reveals increased debris and fewer but larger nucleated epithelial cells with a low nuclear/cytoplasmic ratio (0 to 2 days).

Estrus
Cytologic examination reveals markedly less debris and numerous large polyhedral cornified cells with curled edges and small dark pyknotic nuclei or loss of nuclei (6 to 8 days) after coitus or induced ovulation.

Early Diestrus
Cytologic examination reveals hazy, ragged-edged cornified cells and zero to numerous WBCs with numerous bacteria and increased debris.

Late Diestrus
Cytologic examination reveals increasing numbers of small basophilic cells with WBCs still present (total period of metestrus, 7 to 21 days). If ovulation does not occur, the smear will return to an anestrous stage with few to no WBCs.

The feline estrous cycle is continuous every 14 to 36 days if 12 to 14 hours of light are present daily. Ovulation is induced 24 to 30 hours after coitus. Sperm require 2 to 24 hours for capacitation in the uterus. Implantation is expected 13 to 14 days after coitus.

Additional Reading

Artificial Insemination: Canine
The procedure for artificial insemination in dogs includes the following steps:
1. Determine the correct time to inseminate by test-teasing with a stud, by cytologic examination of vaginal smears, or by vaginoscopic examination to determine the day when vaginal folds change from round to angular. Breed the day after the bitch first stands staunchly to accept service and “flags” her tail or during cytologic indications of estrus (complete cornification of vaginal epithelial cells) but before WBCs reappear in the smears. Breed at 48-hour intervals until the female dog goes out of heat or for three or four inseminations.
2. If the vulva is soiled, clean it thoroughly with alcohol swabs (Box 4-15).
3. Gently aspirate semen through the inseminating pipette into the warm syringe.

<table>
<thead>
<tr>
<th>BOX 4-15 MATERIALS USED FOR PERFORMING ARTIFICIAL INSEMINATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Dry, warm, sterile 5- or 10-mL syringes</td>
</tr>
<tr>
<td>• Rubber adapter tubing, ¼-inch long</td>
</tr>
<tr>
<td>• A 6- to 9-inch plastic or polypropylene inseminating pipette</td>
</tr>
<tr>
<td>• A sterile examination glove</td>
</tr>
<tr>
<td>• Alcohol</td>
</tr>
<tr>
<td>• Cotton</td>
</tr>
<tr>
<td>Do not use lubricating materials.</td>
</tr>
</tbody>
</table>

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4. Using a gloved left index finger (not lubricated) as a guide, insert the pipette through the vulva and dorsally into the vagina and forward to the cervix. Elevate the bitch’s rear quarters to a 45-degree angle by having an assistant pick up the bitch by holding the hock region so that no pressure is applied to the ventral abdomen and uterus. Eject the semen gently and slowly. Eject a bubble of air to push all the semen through the pipette. Deposit the semen in the anterior vagina.

5. Remove the pipette, and hold the bitch in an elevated position for 5 minutes. During this time, use the finger encased in a sterile glove to “feather” the ceiling of the vagina to stimulate constrictor activity. This may be important to simulate a “tie” and transport semen into the uterus.

6. Lower the bitch to the normal position, and immediately walk her for 5 minutes so that she does not sit down or jump up on a person and allow semen to run back out of the vagina.

7. For best conception, inseminate undiluted fresh semen immediately.

8. Refrigerated extended semen is best used within 24 to 48 hours if possible. However, refrigerated semen has been kept viable for up to 9 days with proper care. Skim milk has been used as an economical and adequate extender. Heat milk to 92° to 94° C for 10 minutes, cool it, and skim it at room temperature. To each milliliter, add 1000 units of crystalline penicillin. If *Pseudomonas* species affect the semen, polymyxin B may be added at 200 units/mL of extender. Dilute semen with extender at a semen/extender ratio of 1:1 to 1:4. Extend canine semen for freezing with a diluent containing 11% lactose, 4% glycerin, and 20% egg yolk. Refrigerate the 1:4 diluted semen; then pipette 0.05-mL portions into depressions in a block of dry ice and hold them for 8 minutes to freeze. Store the frozen pellets in liquid nitrogen. Frozen semen can be thawed in buffered saline at 30° to 37° C. Good semen may be stored in liquid nitrogen for many years without significant loss of motility. Conception is best when large numbers of thawed motile sperm are deposited in the cervix or uterine cavity. Conception is poor when thawed semen is placed in the anterior vagina, as done in artificial breeding with raw semen.

Additional Reading

**REPRODUCTIVE TRACT: MALE**

**SEmen Collection: Canine**

Semen is collected for examination for breeding soundness, for investigation of infertility or prostatic disease, and for artificial insemination (Box 4-16).

The following steps outline the procedure for collecting semen from a male dog:

1. Take the stud and an estrous teaser bitch (if available) to a quiet room where there will be no distractions and where there is good traction (rubber mats or rug) for mounting by the stud.

2. Hold the bitch, and allow the stud to “flirt” (become aroused) for several minutes. If the bitch is in heat, a brief period of foreplay (“foreplay” may not be the appropriate word to describe the mating behavior of dogs, but it illustrates the point) with both dogs unrestricted will help the process.

3. If necessary, have assistants restrain the muzzled bitch and control the stud by a collar and leash. Bring the stud up to the rear end of the bitch, and allow him to mount her or keep his nose in the region of her perineal area.

4. Attach the artificial vagina to the semen collection tube, and apply a scant amount of lubricant to the opening of the artificial vagina.

5. If mounting occurs, allow the stud to grasp the bitch and start to thrust his pelvis in an attempt to copulate. Gently, from the side of the sheath, grasp the penis by the prepuce and move the prepuce back over the engorged bulbus glandis; while
applying the artificial vagina to the shaft of the penis, apply pressure with the thumb and forefinger proximal to the exposed glandis. This usually can be done with one motion as the stud is thrusting. If the stud is shy and not interested, massage the penis slightly in the prepuce or in the artificial vagina to cause erection. When erection of the bulb is felt, reflect the prepuce posteriorly to free the bulbus. Apply pressure with the thumb and forefinger behind the bulbus, circling the shaft of the penis. After completion of the most rapid pelvic thrusting and ejaculation of the sperm-rich fractions of semen (1 to 3 mL), twist the penis 180 degrees backward in a horizontal plane, between the hind legs, so that the penis remains in the same plane as in the forward position, with the thumb and forefinger still applying pressure around the circumference of the penis proximal to the bulbus. The penis cannot be twisted unless the prepuce is reflected posterior or proximal to the bulbus glandis. Twisting the penis in this position simulates a natural “tie” and allows the person collecting the semen to better visualize the collection (artificial vaginas are widely available now and are much preferred because they simulate the natural pressure of the vagina). The first drops of ejaculate may be discarded, especially if any urine is present. Collect the sperm-rich fraction separately. A clear ejaculate is prostatic fluid, which may be collected separately for examination.

6. After semen collection, place the penis in the forward position, straighten out the prepuce to avoid paraphimosis, and remove the bitch from the room. Allow the stud to lick the erect penis and lose the erection. Check the stud for evidence of paraphimosis before it is released or caged. The ejaculate consists of three fractions:

First fraction: Urethral secretion (usually clear fluid)—0.1 to 2 mL within 50 seconds, pH 6.3. If evidence of urine is present, discard this fraction and do not add it to the sperm-rich fraction. In most ejaculates collected from dogs, the first and second semen fractions are collected together.

Second fraction: Sperm-containing secretion (milky opaque fluid)—0.5 to 3 mL within 1 to 2 minutes, pH 6.1.

Third fraction: Prostatic secretion (usually clear fluid)—2 to 20 mL within 30 minutes, pH 6.5. The total specimen is 0.3 to 20 mL, pH 6.4. Because the first and third fractions are clear, waterlike material and the second fraction is milky-opaque, the clinician can separate them by changing collecting tubes as each fraction is ejaculated. Collection of only enough prostatic fluid to rinse the sperm fraction into the test tube is best. Too much prostatic fluid may be detrimental to the longevity of sperm in storage. Collecting individual fractions may be important in determining the site of an inflammatory reaction, but for artificial insemination only the sperm-rich, low-volume ejaculation is needed for insemination, dilution, or freezing.

7. Return the stud to his cage. Retain the bitch until the semen is examined, if insemination is to be performed.
EVALUATION OF SEMEN

Immediately after semen collection, slowly invert the tube several times to mix the semen gently. Determine the motility of sperm by placing one drop of semen on a warmed microscope slide. Cover the slide with a coverslip, and observe the specimen under low power for progressive motility. There will be no “waves,” but general vigorous forward motion should be evident. If the sample is too concentrated for individual sperm to be found, mix one drop of semen with one drop of saline at body temperature on a warmed microscope slide. Using high power, count 10 different groups of 10 sperm, observing the numbers of motile and nonmotile sperm. Total motility for a suitable sample should be 80% or greater. Motility less than 60% is not satisfactory.

Determine the number of sperm in the total ejaculate. Sperm concentration may be determined in a hemocytometer with a 1:100 blood cell dilutor kit (Unopette), and concentration then is multiplied by volume to determine sperm numbers per ejaculate. Remember that more dilute samples will be obtained when prostatic fluid is collected, but total sperm numbers in the ejaculate will be only marginally influenced by dilution with prostatic fluid. Total sperm per ejaculate should exceed 300 million in a normal male dog and may approach 2 billion in large dogs. A minimum number of 200 million sperm per insemination is needed on average for conception.

Determine morphology. Make a smear of a drop of semen like a blood smear and allow it to air-dry. Then stain the smear with Diff-Quik stain; dip the slide into the fixative and solutions 1 and 2 for 2 to 3 minutes each. Then count 100 sperm at ×1000 magnification, noting normal and abnormal sperm. If there is any question about abnormality, examine 500 sperm cells.

Normal canine sperm are 63 nm long; the heads are 7 nm long. The percentage of abnormal sperm should be less than 20%. Differential abnormality is important, and the following abnormalities should not be exceeded in any sperm count: abnormality of the head, 10% to 12%; midpiece abnormalities, 3% to 4%; tail abnormalities, 3% to 4%; and retained protoplasmic droplets, 3% to 4%. Figure 4-51 shows abnormalities that should be counted and recorded. The presence and location of distal or proximal protoplasmic droplets, which may indicate cell immaturity, are important to note.

Defects of the cells within the testes are generally more serious than defects that occur in the sperm during epididymal transport or after ejaculation (such as fractured heads, retained protoplasmic droplets, or bent tails). Usually a biopsy should not be done on material from testes unless the testes are azoospermic. Damage produced after the sperm have left the testes may indicate epididymal disease or may be the result of cold, trauma, or osmotic or urinary contamination. When abnormalities are found, it is wise to obtain two or three semen samples within a few days for baseline evaluation and then repeat the studies in 4 to 6 weeks to determine whether there is a healing or regressing trend. There are usually 64 days from the date of sperm formation to the date of ejaculation: 54 days in the testes and 10 days in transport and maturation in the epididymis.

Normal male dogs can be used at stud once every other day indefinitely or once every day for 7 to 9 days, after which sperm numbers in the ejaculate will decline but not to less than the numbers needed to achieve conception.

Additional Reading

Baker R, Lumsden JH: The reproductive tract: vagina, uterus, prostate, and testicle. In Baker R, Lumsden JH, editors: Color atlas of the cytology of the dog and cat, St Louis, 2000, Mosby. (Note: This textbook contains exceptional color plates of normal and abnormal reproductive tract cytologic findings of the dog and cat.)


Although castration is a common first recommendation for any male dog with known or suspected prostatic disease, a number of prostatic disorders are recognized for which cytopathologic and histopathologic examination, rather than castration, is indicated. Benign prostatic hyperplasia is recognized as the most common prostatic disorder of male dogs. In half of the dog population, changes consistent with benign prostatic hyperplasia are present by 4 to 5 years of age, especially in older intact dogs. Because benign prostatic hyperplasia is androgen...
dependent, routine castration is the recommended treatment. However, at least three differential diagnoses justify additional diagnostic tests: prostatic neoplasia (usually adenocarcinoma), acute and chronic bacterial prostatitis, and prostatic cysts (septic and nonseptic).

In male dogs with prostatomegaly and associated signs (dysuria and/or dyschezia), further evaluation of the prostate is indicated. Several techniques have been described. Abdominal ultrasonography is the preferred technique for evaluating prostate size, shape, and consistency. Distension retrograde contrast urethrocystogram has been described as a means for evaluating the internal integrity of the prostate and is moderately effective in distinguishing normal from abnormal. However, this technique is not known to distinguish among various types of prostate disease.

**Patient Preparation**

Cytologic examination and quantitative bacterial culture of the ejaculate (especially the third fraction) of a male dog is recommended in any patient with prostatomegaly. However, sample collection can be difficult and is frequently not successful. In addition to lumbar radiographs and abdominal ultrasonography, performing a prostatic wash is a simple, noninvasive technique that may yield diagnostic information.

**Technique**

Using aseptic technique, place a conventional urinary catheter into the bladder and remove all urine. Lavage of the urinary bladder with up to 5 mL of sterile saline is recommended. Recover the saline and save it (sample No. 1). Subsequently, retract the catheter tip, but only to the level of the prostate gland (immediately caudal to the trigone). Position of the tip usually can be verified by tactile placement and the detection of increased resistance to catheter movement during retraction. Position can be confirmed with a lateral radiograph of the pelvis.

With the catheter in place, identify the prostate on a digital rectal examination and gently massage for approximately 1 minute to force prostatic fluids into the urethra. Infuse 5 mL of sterile saline through the catheter. The objective is to wash prostatic fluids and cells into the urinary bladder and recover the saline from the bladder (sample No. 2).

Examine fluid from both samples cytologically by distributing a drop of fluid across a glass slide, air-drying, and staining; submit a small aliquot (0.5 mL) for bacterial culture. Cytologic examination is used to detect the presence of inflammatory cells versus neoplastic cells. Low numbers of neutrophils (<5 cells per high-power field) are present in ejaculates and prostatic washes from normal dogs. Quantitative bacterial culture, with a yield of greater than 2 log_{10} of one or more bacterial species in sample No. 2 confirms bacterial prostatitis.

**Special Considerations**

Complications from this procedure are unlikely, but conceivably a patient with septic prostatitis and prostatic abscesses could become bacteremic after this procedure, which in some patients could lead to sepsis.

**Prostate Biopsy and Fine-Needle Aspiration**

**Patient Preparation**

Ultrasound examination is an important first step, when available, in assessing the size, shape, and internal integrity of the canine prostate gland and for detecting any changes in structures adjacent to the prostate. However, ultrasonography generally will not distinguish among different types of prostatic disease. Further diagnostic tests are especially indicated in castrated, middle-aged to older male dogs with evidence of prostatomegaly. Percutaneous fine-needle aspiration and/or prostatic biopsy are indicated.

**Technique**

Fine-needle aspiration of the prostate is performed through a ventral abdominal approach. Use aseptic technique, and surgically prepare the skin at the level of needle insertion. Because needle movement, once the needle has been inserted, could damage the urethra or
adjacent structures, perform the procedure in the sedated or anesthetized patient. Use an approach similar to that used for cystocentesis in a male dog with the exception that needle entry is at a point caudal to that used to enter the urinary bladder but is cranial to the pubis. The procedure can be performed with or without ultrasound guidance. In the absence of ultrasound guidance, determine needle position by tactile placement and detection of resistance as the needle enters the prostate. Multiple needle penetrations and aspirations are attempted without withdrawing the needle from the skin. Relieve negative pressure in the syringe before removing the needle. Apply any material collected to a glass slide and allow it to air-dry before staining. Any conventional stain used for peripheral blood is appropriate.

A transrectal approach to fine-needle aspiration of the prostate has been used in dogs and is performed routinely in men. However, the distance from the anus to the prostate, visualization, and the risk of infection generally are cited as reasons for not performing this technique in dogs.

Fine-needle aspiration may not be diagnostic, particularly in patients with isolated, discrete lesions (cysts or neoplastic nodules) within the prostatic parenchyma. In such cases, ultrasound-guided needle (Tru-Cut) biopsy of the prostate is indicated. Specific training and experience are indicated for performing this procedure because significant complications can result.

Special Considerations
Complications associated with prostate biopsy and fine-needle aspiration are not insignificant. Hematuria and periprostatic hemorrhage are described. Postaspiration and postbiopsy abscess also have been described. Consider the risk of urethral penetration and subsequent stricture at the site of penetration.

Additional Reading

**RESPIRATORY TRACT PROCEDURES**

**Upper Respiratory Tract**

For purposes of this discussion, the anatomic limits of the upper respiratory tract of the dog and cat extend caudally from the nasal planum to the first tracheal ring. Key anatomic structures that principally can cause clinical signs include the anterior (external) nares, nasal cavity, nasal turbinates, frontal sinuses, maxillary recesses, upper dental arcade (especially the roots of the maxillary canine teeth), choanae (posterior nares), nasopharynx, soft palate, arytenoid cartilages, glottis, larynx, and vocal folds (see Table 4-9).

<table>
<thead>
<tr>
<th>TABLE 4-9</th>
<th>Anatomic Limits of the Upper Respiratory Tract and Defining Clinical Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Compartment</strong></td>
<td><strong>Anatomic Limits</strong></td>
</tr>
<tr>
<td>I</td>
<td>Nose, nasal cavity, and paranasal sinuses</td>
</tr>
<tr>
<td>II</td>
<td>Nasopharynx, posterior nares (choanae), and soft palate</td>
</tr>
<tr>
<td>III</td>
<td>Larynx</td>
</tr>
</tbody>
</table>
Clinical signs related to the upper respiratory tract in dogs and cats are among the most common presenting complaints encountered in small animal practice and, interestingly, are frequent reasons for referral to specialty practices and veterinary teaching hospitals. The oral and nasal cavities are important portals of entry for foreign body entrapment and infectious agents. In addition to the occurrence of nasal neoplasia and trauma, it is not surprising that upper respiratory tract diseases in dogs and cats are common presentations. However, upper respiratory signs can be associated with significantly different underlying causes. Localizing the problem amid a variety of clinical signs in an anatomically complex area presents significant diagnostic and therapeutic challenges to even the most astute clinician. The presentation addresses upper respiratory disease in the dog, with specific emphasis on clearly defining the presenting clinical signs, localizing the problem, and establishing the diagnosis.

Clinical Signs
The first and most important step in establishing a diagnosis of canine upper respiratory disease is to define the presenting sign. Experience has shown that an owner's ability to describe the patient's clinical signs accurately, particularly when signs are not present at the time of examination, is usually inconsistent and inaccurate, although it can be most entertaining. The four localizing clinical signs characteristically associated with upper respiratory diseases are sneezing and/or nasal discharge, stertor, stridor, and cough. Each sign, considered independently, will focus the examination to the appropriate anatomic region of the upper respiratory tract.

Sneezing and/or Nasal Discharge
Definition of the clinical signs sneezing and nasal discharge may seem intuitive. This is the most common presenting sign in dogs with upper respiratory disease. Owners that present a dog with sneezing are likely to be accurate in their description of the problem. However, the presence or absence of a nasal discharge may be more difficult to establish. Volume, character, and frequency of the discharge ultimately determine whether the owner will have even observed this sign. The astute owner will report whether the discharge is unilateral or bilateral. In the patient that has a history of sneezing and nasal discharge, instillation of a topical nasal decongestant into each nostril occasionally will provoke sneezing and elicit the nature of any discharge that is present.

If these findings are negative, radiographs of the skull are indicated. Three views, obtained in the anesthetized patient, are indicated: lateral, ventrodorsal, and occlusal (open mouth) view. Radiographic interpretation of the nasal cavity and sinuses dictates that the clinician have a thorough understanding of the anatomy of the upper respiratory tract. Subsequently, with the patient still anesthetized, attempt a visual examination of the nasal cavity. Radiographs are always performed before visual examination of the nasal cavity. Manipulation of the tissue may result in intranasal bleeding, which will significantly complicate radiographic interpretation. A simple otoscope speculum placed into each nostril allows an adequate examination of the proximal 20% to 25% of the nasal cavity in most dogs. Visual examination of the caudal 75% of the nasal cavity can be attempted only with a small-diameter endoscope. Flexible and rigid scopes are available; each has advantages and disadvantages that will be discussed. Computed tomography and magnetic resonance
imaging are important alternative diagnostic tools; however, expense and lack of availability are significant limiting factors.

Understanding the most commonly diagnosed causes of sneezing and nasal discharge is especially helpful in patient management. In no particular order, the most common differential diagnoses for sneezing and/or nasal discharge include the following:

1. **Oronasal fistulas**: Especially common in middle-aged to older dogs, despite a history of recent dental prophylaxis. Empiric treatment with an orally administered antibiotic typically results in rapid and complete resolution of clinical signs, but only during the time the patient is receiving the antibiotic. Diagnosis is confirmed by probing the gingival sulcus of the upper canine teeth.

2. **Nasal neoplasia**: Most commonly reported in dogs 8 to 10 years of age (range: 1 to 15 years of age). No breed is predisposed, but the condition is uncommon in brachycephalic breeds. Persistent nasal discharge, sneezing, and intermittent epistaxis are common presenting signs. Nasal radiographs may demonstrate lytic bone lesions. Lysis of the vomer strongly supports neoplasia versus mycotic rhinitis. Exposure to tobacco smoke has been associated with 2.5 times greater risk in long-nosed dogs. No or minimal response of the discharge to antibiotics occurs. Eighty percent of nasal tumors are malignant. Adenocarcinoma is most common, followed by squamous cell carcinoma. Sarcomas account for small number of nasal tumors.

3. **Mycotic rhinitis**: Difficult to distinguish from neoplasia. Persistent and voluminous mucoid nasal discharge, with or without sneezing, and nasal pain are reported. Erosion of the external nares is an important physical finding. Discharge is not responsive to antimicrobial treatment. Occlusal view radiographs of the nasal cavity may demonstrate evidence of turbinate destruction and/or increased fluid density on the affected side. Forty percent of patients are 3 years of age or younger; 80% are 7 years of age or younger. The diagnosis is uncommon in brachycephalic breeds. Localized *Aspergillus fumigatus* infection is reported most commonly.

4. **Lymphoplasmacytic rhinitis**: Poorly described clinical syndrome associated with chronic sneezing and nasal discharge (bilateral or unilateral). Affected dogs are typically young to middle-aged, large-breed dogs. Signs are not usually responsive to antibiotics or steroids (topical or systemic). Diagnosis is based on ruling out other causes and nasal biopsy findings.

**Stertor**

The second most common clinical sign associated with upper respiratory disease in dogs, stertor is intermittent, yet persistent or continuous snorting, also called stertorous breathing. Paroxysms of stertor, typically called reverse sneezing, are characterized by rapid, consecutive inspiratory bursts through the nose. Seldom actually seen during examination, reverse sneezing is likely to result from the patient’s attempt to displace matter trapped in the nasopharynx and move it into the oropharynx, where it can be swallowed.

Visualization of the nasopharynx and choanae is essential in the patient that has chronic or persistent stertor. The examination can be accomplished only in the anesthetized patient. Sedation is not sufficient to conduct the examination. A flexible endoscope with the ability to flex approximately 170 to 180 degrees is recommended. Examination allows visualization of the nasopharynx and associated mucosa, the choanae (posterior nares), and the top of the soft palate (see **Figure 4-34**).

Nasopharyngeal foreign bodies are by far the most common finding. Sticks, plant material (grass and juniper twigs), peas, cotton balls, and thread are just a few examples. Neoplasia is the second most common finding. In cats, lymphoma (FeLV related) obstructing the choana most commonly is observed (see **Figure 4-35**). In dogs, neoplasia is uncommon, but (in my experience) sarcomas in young dogs have been seen most frequently.
Stridor

The least commonly encountered of the upper respiratory signs is stridor, or stridulous breathing. Stridor is audible wheezing and is associated with restriction to airflow, usually at the level of the larynx. Therefore stridor is the most critical and potentially life-threatening upper respiratory sign. This is especially true when stridor is continuous. The patient that has continuous stridor deserves immediate attention. Make every effort to discern the cause once the clinical sign is characterized. In obtaining the history, owners generally describe wheezing accurately; however, some patients actually may have severe dyspnea or orthopnea. Careful questioning of the client is indicated to determine whether wheezing is associated with the additional effort to breathe. The clinician also should make an effort to discern whether the owner has observed any change in the ability of the dog to vocalize or bark.

Simply listening to the patient breath in a quiet room is the first step in assessing stridor. A stethoscope is not required to hear wheezing but should always be used to examine the cervical trachea, the larynx, and the lungs. Any restriction to airflow in the larynx or cervical trachea can cause stridor. However, in the majority of cases the stridor will be significantly louder at the level of the larynx, indicating a restrictive lesion at that level.

If any indication of respiratory distress is reported or manifests during the examination, subject the patient to a visual examination under general anesthesia. Sedation is not sufficient to conduct the examination. Be prepared. These patients are not routine. Emergency resuscitation may be required on induction of anesthesia, including the need to perform a tracheostomy.

On induction, carefully place an endotracheal tube. If there are no complications associated with inserting the tube, once anesthesia has been effectively induced and the patient’s condition is stable, lateral and dorsoventral radiographs of the larynx and cervical trachea are indicated. Metallic objects (e.g., fish hooks) can become buried in the mucosa and may not be observed during a visual examination.

Remove the endotracheal tube in order to conduct a visual examination. A focal, hands-free light source directed into the oropharynx is strongly recommended. Carefully examine the epiglottis, arytenoid cartilages, glottis, and vocal folds using a cotton-tipped applicator. Careful observation of the symmetry and function of the arytenoid cartilages is essential. The left and right cartilages normally respond to tactile stimuli when the patient is in a light plane of anesthesia; both sides should move to the medial plane rapidly and at the same time. They may not close, depending on the depth of anesthesia. It should be possible to visualize the cartilage on the inside of the tracheal rings while looking through the glottis.

In large breed, middle-aged and older dogs, laryngeal paralysis is the most common cause of stridor. Associated signs may include exercise intolerance and collapse during exertion. Laryngeal paralysis and stridor also may be observed in young breeds as a congenital disorder (Dalmatian, Rottweiler, Bouvier des Flandres, Siberian Husky, and Bull Terrier). Foreign body penetration of the laryngeal tissues can cause serious and life-threatening obstruction because of infection and swelling. Neoplasia may cause obstructive mass lesions involving the larynx, especially squamous cell carcinoma and lymphoma. Granulomatous laryngeal disease and fungal mycetoma have been reported.

The presence of a mass lesion, assuming there is no foreign body detected, warrants biopsy of the lesion. Additional effort to control postbiopsy bleeding is important. I use a cotton-tipped applicator saturated with a 1:10,000 dilution of epinephrine held against the biopsy site for 30 to 60 seconds. This is time well spent. Postbiopsy administration of systemically effective dexamethasone has been suggested to control laryngeal swelling, but I have not found this to be effective or important.

Additional Reading


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### LOWER RESPIRATORY TRACT

**Note:** Cats and dogs with acute, severe dyspnea must be regarded as having a life-threatening condition until proved otherwise. Immediate therapeutic and diagnostic intervention is indicated. Section 1 describes appropriate interventive procedures for the management of these patients.

The following diagnostic procedures are elective and are indicated in patients with chronic disorders of the lower respiratory tract that are not considered life-threatening.

**Transtracheal Aspiration**

Transtracheal aspiration is a safe and clinically useful method for obtaining material for cytologic and bacteriologic examination from the lower respiratory tract of medium-sized to large dogs without invading the oval cavity. This procedure is not indicated in cats.

**Patient Preparation**

The technique can be performed on the unanesthetized animal, although some sedation may be indicated. The hair overlying the larynx is clipped and prepared surgically. In small dogs and cats, tracheal aspirates are collected by passing the catheter through sterile tracheal tubes. Light levels of anesthesia are used to accommodate coughing and tracheal intubation.

**Technique**

Place the animal in sternal recumbency or in the sitting position. Elevate and extend the head. Locate the cricothyroid membrane by moving the finger along the proximal trachea until the large ventral ridge of the cricoid cartilage is felt. Use a 16-gauge, 1/2-inch intravenous catheter to collect material through the trachea ([Figure 4-52](#)). Puncture the cricothyroid membrane with the 16-gauge needle, and pass the catheter into the trachea until it reaches the distal trachea or main stem bronchus. (Alternatively, in large dogs, insert the catheter between the tracheal rings at the junction of the middle third and distal third of the cervical trachea.) Withdraw the needle, and leave the catheter in place. Attach a 12-mL syringe containing sterile saline solution to the catheter. Expel 1 to 2 mL of saline from the syringe. When the animal coughs, aspirate with the syringe to collect cells and mucus for bacteriologic and cytologic examination. When material has been collected, remove the catheter and bandage the animal’s neck. Culture material present in the syringe in blood agar and in thioglycolate medium. Prepare material from aspiration for cytologic examination. Press large plugs of mucus between two clean glass slides, and stain thin smears with Wright or Giemsa stain.

**Special Considerations**

Complications of transtracheal aspiration biopsy include catheter trauma to the lower airway or needle trauma to the larynx, resulting in bleeding, subcutaneous emphysema, pneumomediastinum, pneumothorax, or airway obstruction.

**Endotracheal Wash**

In cats and small dogs and in dogs for which general anesthesia is not contraindicated, tracheal aspiration (or tracheal wash) is a relatively safe, easy-to-perform procedure that can yield excellent diagnostic cytologic and culture specimens. The procedure has some advantages over transtracheal aspiration in that it allows sample collection from airways beyond
the bifurcation of the trachea (carina) and avoids complications associated with patient discomfort and movement during the procedure. However, cough reflexes are eliminated completely, thereby decreasing potential sample yields from deep in the airway structure. In either case, transtracheal and tracheal aspirations provide the best diagnostic material from large airways, not small airways and alveoli.

Figure 4-52: A, Diagrammatic representation of anatomic structures involved with transtracheal aspiration technique. The best landmark for percutaneous puncture is the cricothyroid ligament of the larynx, although the tracheal lumen also can be entered between cervical tracheal rings. B, The needle is advanced and directed slightly caudad until the trachea is entered. Once the needle is positioned within the tracheal lumen, the catheter is advanced through the needle and down the trachea. (From Kirk RW: Current veterinary therapy VIII: Small animal practice, Philadelphia, 1983, WB Saunders.)
Patient Preparation

The anesthetized dog or cat usually is placed in sternal recumbency. Lateral recumbency (affected side down) may facilitate recovery of specimens from patients with focal or regional lung disease. Use a sterile endotracheal tube to administer the anesthetic and oxygen.

Technique

Introduce a sterile red rubber catheter (long enough to extend beyond the carina) through the endotracheal tube (Figure 4-53). (Note: Disposable adapters for use with endotracheal tubes are available that allow continuous administration of anesthetic gases while passing the rubber catheter through the tube [Figure 4-54].) Introduce the catheter blindly until resistance is met as the tube attempts to enter smaller airways.

Use aliquots of warmed, sterile saline in prepared syringes to wash and retrieve samples. Aliquots of 3 to 5 mL can be used per collection attempt in small dogs and cats, whereas volumes up to 10 and 20 mL are appropriate for larger dogs. With the catheter positioned as deep as practical in the airway, infuse the entire volume of saline. Gentle agitation (intermittent aspiration and injection) may facilitate sample collection. If a 10-mL quantity is infused, retrieval of only 1 to 2 mL as a final volume per collection attempt is not unusual. The remaining fluid is rapidly (seconds) absorbed into the pulmonary vasculature. Important: When performing this procedure, do not withdraw the rubber catheter while maintaining a high negative pressure on the syringe. Doing so actually may tear mucosa away from the airway and could lead to pneumothorax or pneumomediastinum.

Special Considerations

The procedure can be repeated safely in the same patient several times. Collection of three to five samples is routine. More samples may be indicated depending on the patient’s condition and response to the procedure. Monitoring of patients undergoing a tracheal wash procedure for oxygen saturation (pulse oximetry) throughout the procedure is recommended. In some patients with reactive airways, infusion of saline may cause significant bronchoconstriction, detected by a rapid decline in oxygen saturation.

Process collected samples immediately. Submit at least one sample of liquid (not a swab of the liquid) for bacterial culture and sensitivity or MIC. Quantitative cultures are
impractical because specimens will be diluted. If the sample appears to be highly cellular (characterized by turbidity), place aliquots into tubes containing EDTA.

**Additional Reading**


**Bronchoalveolar Lavage**

BAL is an alternative diagnostic procedure to transtracheal aspiration and endotracheal wash. BAL has the advantage of retrieving fluid samples from distal airways and alveoli. This is a highly diagnostic procedure indicated in patients with generalized lung and regional (interstitial and/or airway) disease that are not in respiratory distress. Patients suspected of having allergic or infectious respiratory disease or neoplasia are candidates for BAL. Although BAL is used as a therapeutic procedure in human beings with chronic lung disease associated with accumulations of surfactant in the alveoli, there is no therapeutic indication for BAL in dogs or cats.

**Patient Preparation**

BAL must be performed in the anesthetized patient and consequently may be contraindicated in some patients with severe respiratory disease.

**Technique**

BAL entails instilling sufficiently large volumes of fluid into the distal airways to reach, and recover, reasonable cytologic samples representative of small airways and alveoli. Several variations on the technique have been described, but all recommend blind or visual placement of a catheter or bronchoscope into an airway of a lung lobe such that the airway is occluded. Sterile, nonbacteriostatic 0.9% saline, warmed to approximately body temperature and drawn into prepared syringes, is the fluid of choice. The volume of fluid varies with the size of the patient. Defined doses of saline per kilogram of body mass have not been described. In large dogs, two 25-mL aliquots (50 mL total) can be infused into each lobe sampled. In small dogs and cats, total volumes per lobe generally are restricted to 10-mL aliquots. Recovery may be as low as 2 to 5 mL with each attempt.
For dogs undergoing BAL, particularly when reactive (allergic) airway disease is suspected, pretreatment with a bronchodilator is appropriate and is recommended. Aminophylline can be administered at 5 mg/kg (cats) or 11 mg/kg (dogs) orally 1 to 2 hours before the procedure. Alternatively, terbutaline, 0.01 mg/kg, can be administered subcutaneously to cats 30 minutes before the procedure.

Bronchoscopic BAL allows direct visualization of the airway or lobe of interest. In medium to large dogs, place the bronchoscope directly through a sterile endotracheal tube. Use of an inexpensive, disposable endotracheal tube adaptor permits simultaneous administration of oxygen and anesthetic throughout the procedure. Saline can be infused from a syringe directly through the biopsy channel of the endoscope. The bronchoscope serves as the infusion catheter. Using this technique, samples can be collected effectively from multiple lobes. Blind placement (nonbronchoscopic) BAL using a rubber end-hole catheter is required in cats and small dogs. Blind placement is also appropriately used in patients with generalized lung or airway disease when discrete placement of the bronchoscope cannot be accomplished reliably.

**Special Considerations**

As with the endotracheal wash procedure described before, gentle agitation with the syringe (intermittent aspiration and injection) may facilitate sample collection. **Do not withdraw** the bronchoscope or catheter while maintaining significant negative pressure because this may lacerate the airway, leading to pneumothorax or pneumomediastinum.

BAL is an invasive diagnostic procedure that is not without risk of injury or death. After completion of BAL, administration of 100% oxygen for 5 to 10 minutes via endotracheal tube is recommended for all patients. Evaluate the patient carefully for breathing effort and oxygen saturation (pulse oximetry) during recovery. Although significant quantities of fluid remain in the airways after BAL, most of the volume is absorbed rapidly. Residual amounts of fluid, however, can be retained for 24 to 48 hours after the procedure. During this time, some patients will manifest cough. Crackles may be auscultated.

**Additional Reading**


**Fine-Needle Aspiration of Lung**

Percutaneous aspiration needle biopsy can be helpful in establishing a diagnosis in conditions such as (1) chronic inflammatory disease of the lung—for example, granulomatous lung disease caused by mycotic organisms; (2) chronic inflammatory disease; (3) metastases to the lung; and (4) primary lung tumors. The biopsy may provide enough diagnostic information to preclude performing an exploratory thoracotomy. Lung biopsy is contraindicated in animals with hemorrhagic disease or thoracic disease that produces forceful breathing and coughing.

**Patient Preparation**

Clip and surgically prepare the biopsy site. Infiltrate the skin, subcutaneous tissue, muscle, and parietal pleura with 1% to 2% lidocaine. In patients with diffuse parenchymal lung disease, taking biopsy material from the diaphragmatic lobes is recommended. The dorsal portions of the seventh to ninth intercostal spaces are preferred for percutaneous biopsies. In diffuse lesions, take biopsy material from the right or left thorax.

**Caution:** Understanding of the risks associated with performing fine-needle aspiration of the lung is important, and these risks must be clearly communicated to owners whose pets undergo this procedure. Lung aspirates will yield only cells, fluid, and trace amounts...
of tissue, yet there is a significant risk of inducing pneumothorax with the procedure, even when performed without difficulty or complications.

**Technique**

For the procedure, a 22- to 25-gauge disposable needle (such as a 1-inch spinal needle) with stylet is preferred. Leave the stylet within the needle until the lung has been penetrated. Then quickly remove the stylet and immediately attach a sterile 6- to 12-mL syringe. The amount of air that might enter the lung between the time the stylet is removed and the syringe is attached is negligible. Holding the syringe carefully and steadily against the patient's thorax, establish negative pressure in the same manner as when obtaining an aspirate from a lymph node. As much as the patient will permit, attempt three to four aspirations without withdrawing the needle.

Alternatively, insert a conventional 25-gauge needle attached to a 6-mL syringe subcutaneously over the area of interest. Then establish significant negative pressure while the tip of the needle is still positioned in the subcutaneous tissues outside the parietal pleura. While maintaining the same amount of negative pressure in the syringe, direct the needle into the lung, leave it in place for 1 to 2 seconds, and withdraw it completely. Apply any material collected directly to glass slide. This procedure is best conducted in patients that are awake. Attempting the procedure in anesthetized dogs or cats could result in an unsuccessful aspiration, or, if the lungs were under positive pressure (ventilation or bagging), the risk of causing pneumothorax could be increased. Other reported complications include hemothorax (always exciting), lung laceration caused by patient movement during the procedure, pulmonary hemorrhage, and hemothysis. Contraindications to fine-needle aspiration include patients with a known bleeding diathesis and coagulopathy, thrombocytopenia, uncontrolled coughing, pulmonary hypertension, pulmonary cysts, and bullous emphysema.

Ultrasound-guided techniques for fine-needle aspiration or biopsy of the lung recently have been described and generally are associated with fewer procedural complications. However, additional training and experience, in addition to having access to the proper size and type of ultrasound probe, are critical.

**Additional Reading**


**Nebulization and Aerosol Therapy**

Inhalation therapy can be defined as nebulization (humidification of the inspired air) and aerosol therapy (the process whereby drugs are vaporized in a solution and delivered directly into the respiratory tract). In companion animals, inhalation therapy is most useful for humidifying air in the respiratory tract and moistening the mucous membranes (nebulization). Sustained inspiration of dry air or gases causes irritation to the respiratory epithelium, which in turn results in swelling, bronchial gland hypertrophy, goblet cell proliferation, and loss of ciliary epithelium over time. Respiratory secretions become thick and tenacious, and efficient bronchial drainage is impaired.

The objectives of inhalation therapy include the following:

1. Humidification of bronchial mucous membranes
2. Deposition of miniscule amounts of potent drugs in smaller airways to achieve optimal topical therapeutic effects with minimal systemic side effects (e.g., bronchodilators)
3. Deposition of moderate amounts of potent agents or agents that are effective only topically (e.g., antibiotics and mucolytics)
4. Deposition of relatively large quantities of bland substances that promote bronchial drainage with minimal irritation (e.g., saline, propylene glycol, glycerin, and detergents)
Nebulization is used (1) in combination with oxygen therapy; (2) in tracheostomy care; (3) in acute respiratory diseases such as tracheobronchitis, bronchiolitis, upper respiratory disease of cats, pneumonia, and postoperative atelectasis and pneumonia; and (4) in chronic respiratory diseases such as chronic bronchitis, bronchopneumonia, collapsed trachea with secondary tracheobronchitis, emphysema, and bronchiectasis.

Aerosol therapy, however, is a limited-use therapeutic technique used in dogs and cats to administer antimicrobials, bronchodilators (aminophylline, 100 mg), or corticosteroids. The advantage of doing so is to achieve relatively high levels of drug in the respiratory tract in patients with defined lower respiratory tract disease. In addition, administration of potentially toxic antimicrobials (aminoglycosides) by this route has been shown to be associated with minimal or insignificant uptake into the general circulation, thereby minimizing (or eliminating) any risk of renal toxicity.

**Drug Delivery by Aerosolization (Figure 4-55)**

Drugs that can be applied by jet nebulizer (Figures 4-56 and 4-57) include the following:

1. **Bronchodilators**: Always use bronchodilators when administering drugs that may be irritating and constricting, such as isoetharine hydrochloride 1% and phenylephrine 0.25%, 0.5 to 1.0 mL in 2 to 3 mL of saline three or four times daily.
2. **Antibiotics**: Antibiotics are poorly absorbed from the respiratory mucosa. Systemic administration of most antibiotics produces adequate pulmonary concentration for antibacterial effect. For *Bordetella* species that are located at the tips of bronchial cilia, topical contact via nebulization may be useful. Antibiotics that have been used successfully and safely include kanamycin (250 mg in 5 mL saline twice daily); gentamicin (50 mg in 5 mL saline twice daily); and polymyxin B (333,000 international units in 5 mL saline twice daily).
3. **Bland solutions**: Use these in large volume for prolonged mist effect: 0.9% sterile saline (5 to 200 mL as needed); glycerin (5% in saline); and propylene glycol (10% to 20% solution in saline).
4. **Detergents and mucolytics**: These compounds are irritating and currently are not recommended by most authors.
5. **Antifoaming agents**: Administer ethyl alcohol (70% solution, 5 to 10 mL twice daily).

![Figure 4-55: Disposable jet nebulizer used to administer humidified air and/or medication directly into the respiratory tract.](www.ajlobby.com)
Removal of uroliths from dogs and cats is a commonly performed, yet critically important, clinical procedure. Several techniques are available for removal of calculi and obstructive concretions in male and female animals. Cystotomy is performed routinely...
to remove calculi from the lumen of the bladder but, especially in male dogs, may not be an effective approach for removing obstructing urethral calculi. Advanced and expensive techniques recently have been described: laparoscopic-assisted cystotomy, use of the Ellik evacuator, use of stone “baskets” (through a cystoscope), and lithotripsy are examples. However, for removal of uroliths from dogs and cats with partial or complete urinary obstruction, urohydropulsion is among the more effective yet inexpensive techniques available.

Urohydropulsion is a therapeutic procedure for removal of foreign material, namely, uroliths, from the bladder and/or urethra of dogs. Two techniques are described: voiding urohydropulsion and retrograde urohydropulsion. Both procedures have advantages and disadvantages.

**Voiding Urohydropulsion**

The objective of voiding urohydropulsion is to induce forceful voiding of urine by manually compressing the bladder to facilitate removal of cystic uroliths in female dogs.

*Caution:* Do not perform this procedure until it can be confirmed by catheterization or cystoscopy that the urethra is patent.

With the bladder filled with urine or saline (via catheterization), lift the patient (preferably sedated or anesthetized, although this procedure can be done in the awake patient) into a position such that the tail and perineum are ventral and the head is upright. The spine should be approximately perpendicular to the working surface. Using one or both hands, gradually increase pressure on the bladder to induce and maintain a forceful stream of urine. Objectively, small uroliths will be extruded. If the procedure is only partially successful, it can be repeated as necessary. Obviously, voiding urohydropulsion has limitations and cannot be used in male dogs or in dogs with urethral obstructions or strictures.

**Retrograde Urohydropulsion**

This procedure is indicated for male dogs and cats with partial or complete urethral obstruction caused by uroliths or accumulations of “sand.” This procedure should be performed in the anesthetized patient.

*Note:* There are discrepancies in the literature regarding whether to empty the urinary bladder of urine before performing this procedure. Because patients with urethral obstructions may have a significant volume of urine in the bladder at the time of presentation, some authors recommend performing cystocentesis to relieve the internal pressure before attempting urohydropulsion. However, in patients that have had a profoundly distended bladder for several hours (even days), penetrating the urinary bladder with a needle presents significant risk of rupturing a fragile bladder. The next step, of course, is abdominal surgery. I recommend avoiding cystocentesis whenever possible. The volume of saline required to flush uroliths into the bladder is inconsequential considering the total volume already present.

With the patient positioned in lateral recumbency, retract the prepuce and expose the penis as for conventional bladder catheterization technique. Use sterile technique to pass an appropriately sized flexible catheter, which is advanced to the point of obstruction. Attach a catheter-tipped 60-mL syringe filled with warmed (my preference) sterile saline and a water-soluble lubricant mixture (approximately two parts saline to one part lubricant) to the urinary catheter. An assistant places a gloved (always preferred) finger into the rectum to identify and occlude the lumen of the pelvic urethra at the level of the pubis. Subsequently, infuse saline forcefully into the catheter to dilate the urethra proximal to the obstructing urolith. At that point, release the digital pressure on the proximal urethra while the solution continues to be infused through the catheter. Objectively, the pressure within the urethra forces small stones retrograde into the urinary bladder, thereby relieving the obstruction.
Note: The objective of this procedure is not to push the calculi into the bladder with the catheter, because this can substantially injure the urethral mucosa, nor is it to force the calculi around the catheter and move it antegrade.

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**COMMON REFERENCE RANGE VALUES**

**Note:** Reference range values listed throughout this section are for general reference only. Test results from individual patients must be compared with the reference range values for the laboratory that performs the test.

**SAMPLE HANDLING**

**SAMPLE IDENTIFICATION**

Identification of specimens is critical if the right result for a given patient is to get back to the right clinician in a timely manner. The following steps are recommended:
1. Write the animal and client names on each specimen container.
2. Write the animal and client names, species, breed, gender, and date on the test requisition form.
3. Make sure that the originating clinic name and account number are clearly identified on the form.
4. Clearly mark or write down the needed tests on the form. (*Note:* Commercial laboratories receive hundreds of samples each day with no test marked!)
5. Indicate the source, if other than a blood sample, on the form.
6. Identify the tissue or fluid source and clinic ID on all slides submitted for cytology (use a lead pencil to write on the frosted side).

**SAMPLE COLLECTION TUBES**

Most practices use a variety of glass, and occasionally plastic, vacuum tubes (Vacutainer*) to collect and submit blood, serum, or plasma from individual patients. The tubes are actually designed for collecting blood samples from humans. A variety of tube sizes, each of which maintains a predetermined negative pressure (vacuum) inside, are available. The vacuum facilitates collection of an appropriate volume of the patient’s blood to nearly fill the tube. In addition, most of the blood collection tubes contain an additive that will either accelerate or prevent clot formation.

Adult (human) tubes are available in 5-mL, 7-mL, 10-mL, and 15-mL sizes. Pediatric (human) tubes, appropriate for use in companion animal patients, are available in 2-mL, 3-mL, and 4-mL sizes. For tubes containing an additive, filling the tube with an appropriate

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*Vacutainer is the registered trademark of Becton, Dickinson and Company, Franklin Lakes, New Jersey (United States).
volume of blood is important. Underfilling any tube that contains an additive may alter the sample sufficiently that the test results are adversely affected and may not accurately represent the patient’s status.

The color of the stopper in the top of the tube indicates the type of additive, if any, and the specific type of tests that can be performed with that sample. For example, do not send serum when plasma is required! Refer to Table 5-1 as a guide for selecting the appropriate tube for the type of test desired. In addition, commercial laboratories generally provide tube selection guidelines. Interpretation of the in-office coagulation screen is outlined in Table 5-2.

**Special Considerations**

The quality and accuracy of test results are influenced by the manner in which samples are collected, stored, and shipped. For example, selecting the improper specimen container (for blood, serum, or plasma) can significantly alter test results; submission of blood in a serum-separator tube (SST) for endocrine testing can adversely affect results because of the gel additive in the tube.

In addition, whole blood submitted in ethylenediaminetetraacetic acid (EDTA) for routine hematology begins to deteriorate as soon as the blood is collected. To preserve cell morphology, slides should be made and air-dried immediately after collection of blood. Generally, slides should be submitted unstained and should not be refrigerated, as condensation can also affect cell morphology.

The preferred venous blood samples are collected from a large vein and free-flowing blood. Slow blood draws can result in hemolysis, altered cell morphology, and platelet clumping that causes altered hematology and biochemistry test results. To prevent lysis of red blood cells (RBCs), do not force clotted blood out of a syringe and into a collection tube.

When filling multiple tubes from a single syringe, always fill the red-topped tube (RTT) first to avoid contamination with liquid tube additives. Even a small amount of EDTA can significantly affect serum chemistries.

When filling a lavender-topped tube (EDTA) or light-blue–topped tube (citrate), always add the volume of blood stipulated on the tube. Overfilling or underfilling tubes affects the ratio of additive to sample and can compromise test results.

When recovering serum from whole blood by centrifugation, always allow the sample to completely clot before centrifugation. Centrifuging too early can result in a mixed sample that contains both serum and plasma (Box 5-1).

Most commercial laboratories recommend collecting a minimum volume of 2.0 mL whole blood for routine biochemical analyses; 2.0 mL of whole blood will yield close to 1.0 mL of serum. Dehydrated patients are expected to have a higher hematocrit (Hct), and therefore a larger volume of whole blood may be required in order to obtain a 1.0-mL sample of serum.

When collecting blood from a patient, it is critical to use the following:

1. The appropriately sized tubes
2. Tubes that contain the proper additive for the test(s) requested

**SAMPLE STORAGE AND TRANSPORT**

Several types of sample collection or sample submission storage tubes are available. It is critical that the type of blood collection and/or storage tube used meet the requirements of the test as defined by the laboratory.

To prepare a sample for storage and transport:

1. Stabilize serum from an SST by centrifuging the specimen before submission. If the specimen is being mailed, it is preferable to transfer the separated serum to a labeled plain RTT.

**Note:** Depending on the test requested, the tube used to collect the sample is frequently not the same tube used to submit the sample. Sample collection and sample submission requirements are provided for all tests listed in this section.
<table>
<thead>
<tr>
<th>Color of Conventional Rubber Top</th>
<th>Additive</th>
<th>Number of Tube Inversions after Collection†</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red (also called red-topped tube, or RTT)</td>
<td>Silicone coating (glass tube) Clot activator + silicone (plastic tube)</td>
<td>0 5</td>
<td>For serum determinations in chemistry. May be used for routine blood donor screening and diagnostic testing of serum for infectious disease. Tube inversions ensure mixing of clot activator with blood.</td>
</tr>
<tr>
<td>Red and dark gray (also called serum separator tube, or SST)</td>
<td>Clot activator and gel for serum separation</td>
<td>5</td>
<td>For serum determinations in chemistry. May be used for routine blood donor screening and diagnostic testing of serum for infectious disease. Tube inversions ensure mixing of clot activator with blood.</td>
</tr>
<tr>
<td>Lavender</td>
<td>Liquid K&lt;sub&gt;2&lt;/sub&gt; EDTA (glass tube) Spray-coated K&lt;sub&gt;2&lt;/sub&gt; EDTA (plastic tube)</td>
<td>8 8</td>
<td>K&lt;sub&gt;2&lt;/sub&gt; EDTA and K&lt;sub&gt;3&lt;/sub&gt; EDTA for whole blood hematology determinations. K&lt;sub&gt;2&lt;/sub&gt; EDTA may be used for routine immunohematology testing, blood donor screening, and most PCR assays. Tube inversions ensure mixing of anticoagulant (EDTA) with blood to prevent clotting.</td>
</tr>
<tr>
<td>Gray</td>
<td>Potassium oxalate, sodium fluoride Sodium fluoride, Na&lt;sub&gt;2&lt;/sub&gt; EDTA Sodium fluoride (serum tube)</td>
<td>8 8 8</td>
<td>For glucose determinations. Oxalate and EDTA anticoagulants will give plasma samples. Sodium fluoride is the antiglycolytic agent. Tube inversions ensure proper mixing of additive with blood.</td>
</tr>
<tr>
<td>Light blue</td>
<td>Buffered sodium citrate 0.105 M (approximately 3.2%) glass 0.109 M (3.2%) plastic Citrate, theophylline, adenosine, dipyridamole (CTAD)</td>
<td>3 to 4 3 to 4</td>
<td>For coagulation determinations. CTAD is added for selected platelet function assays and routine coagulation determination. Tube inversions ensure mixing of anticoagulant (citrate) to prevent clotting.</td>
</tr>
<tr>
<td>Red and light gray</td>
<td>No additive (plastic tube)</td>
<td>0</td>
<td>For use as a discard tube or secondary specimen tube.</td>
</tr>
</tbody>
</table>


†Invert tubes gently; do not shake.
Centrifuge the blood samples in a plain RTT, and transfer the serum to another RTT.

Refrigerate and transport all blood specimens, cytology fluids, tissues, viral cultures, and urine specimens for urinalysis or culture with ice packs.

Do not refrigerate unstained or unfixed slides submitted for cytologic evaluation (e.g., hematology slides, tissue impression, fine-needle aspiration [FNA]).

Keep all routine microbial cultures (except urine) and blood cultures at room temperature.

If a specimen must remain frozen for transport, dry ice is required. It is usually the responsibility of the individual practice to package frozen samples correctly. Most laboratories do not provide dry ice for shipping.

Fasting the patient for 8 to 12 hours (an overnight fast with free access to water) is often helpful to reduce the likelihood of lipemia, which may interfere with several tests by falsely increasing or decreasing the results. When applicable, comments about the presence and influence of lipemia and/or hemolysis should appear on the laboratory reports. For special tests, patient preparation may include restriction of food as well as water and certain drugs. It is important to follow the guidance provided in this section regarding patient preparation or to contact the laboratory for specific instructions.

**TABLE 5-2 Interpretation of the In-Office (or Point-of-Care) Coagulation Screen**

<table>
<thead>
<tr>
<th>Platelet (Estimate)</th>
<th>Low</th>
<th>Thrombocytopenia</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACT</td>
<td>Rapid, prolonged</td>
<td>Intrinsic or common clotting pathway defect</td>
</tr>
<tr>
<td>APTT</td>
<td>Rapid, prolonged</td>
<td>Intrinsic or common clotting pathway defect</td>
</tr>
<tr>
<td>BMBT</td>
<td>Prolonged</td>
<td>Thrombocytopenia, thrombocytopathia</td>
</tr>
</tbody>
</table>

**ACT,** Activated clotting time; **APTT,** activated partial thromboplastin time; **BMBT,** buccal mucosal bleeding time.

**BOX 5-1 COMMON MISTAKES**

Samples that clot during collection may result in:
- platelet clumping
- falsely decreased cell counts
- hemolysis, if the blood is forced into the collection tube

EDTA contamination may cause:
- falsely decreased calcium
- falsely increased potassium
- interference with various specialized tests

Underfilling the collection tube can result in an excess of anticoagulant (EDTA or citrate), causing:
- decreased RBC count and hematocrit (dilution effect)
- altered cell morphology
- inaccurate MCV, MCH, MCHC, and hemoglobin
- falsely prolonged clotting times

From *IDEXX Reference Laboratories Directory of Tests and Services—2010,* Westbrook, Maine, United States, IDEXX Laboratories.

**EDTA,** Ethylenediaminetetraacetic acid; **MCH,** mean corpuscular hemoglobin; **MCHC,** mean corpuscular hemoglobin concentration; **MCV,** mean corpuscular volume; **RBC,** red blood cell.
MINIMIZING HEMOLYSIS

Hemolysis during blood drawing can be minimized by adhering to the following recommendations. Procure a nonlipemic (fasted) sample, because lipemia can increase red cell fragility. During phlebotomy, negative pressure created by the vacuum tube or syringe may collapse the lumen of the vein against the needle, thereby crushing numerous red cells. The flutter of the lumen against the needle can be stopped by reducing the negative pressure exerted during collection and by repositioning the needle with slight rotation or deeper insertion.

**Note:** If the patient has lipemic serum or blood after an 8-hour fast, a lipid analysis should be performed on the lipemic serum. The laboratory should be instructed not to clear the sample before determining lipid levels, especially for triglyceride.

Excessive negative pressure exerted as the blood enters the vacuum tube or syringe can create hemolysis. This occurs during a slow or difficult collection, because the natural tendency is to use more negative force to enhance blood flow. More patience and “milking” the vein by alternating gentle negative pressure with a short release of all pressure usually solves the problem.

Hemolysis often occurs during the transfer of blood from a syringe into vacuum or other tubes. If a small-gauge needle is used, transfer of blood to specimen tubes is slowed, especially if small clots are present. Forcing the blood through a small-bore needle contributes to hemolysis. This problem can be avoided by removing the needle and top of the specimen tube and transferring the blood directly into the open tube. Recapping the tube and aspirating a small amount of air to reestablish negative pressure helps to avoid having caps coming off in transit.

AVOIDING CLOTS AND PLATELET CLUMPS

The presence of clots and clumped platelets in anticoagulated blood is most commonly caused by a slow blood draw and the resulting delay in mixing it with the appropriate anticoagulant. If the venipuncture was traumatic, tissue fluid (thromboplastin), activated clotting factors, and hemolysis will quickly promote clot formation. The slight transfer delay when using a syringe for collection can also contribute to this problem. To avoid the formation of clots, do the following:

1. Select a vein with good blood flow—the larger the better.
2. Minimize the trauma of venipuncture.
3. Collect blood directly into anticoagulated vacuum tubes (e.g., light blue–topped tube [citrated] or lavender-topped tube [EDTA]).
4. Mix the contents of the tube well by inverting several times immediately after filling.

If the syringe method is selected and a difficult draw is anticipated, the potential for clotting can be minimized by first rinsing the needle and syringe with a small quantity of liquid citrate (blue-topped tube [BTT]) or EDTA (lavender-topped tube). However, the anticoagulant must be emptied from the syringe before proceeding, and care must be taken to match the anticoagulant chosen with the tests to be performed. Even trace amounts of heparin or EDTA will invalidate coagulation testing, whereas EDTA or citrate will alter the accuracy of several chemistry assays. A small amount of heparin contamination is acceptable in most chemistry assays and complete blood count parameters.

Platelet clumping in samples from cats is very common and is caused by contact aggregation. An effective method to prevent this clumping has not been found. Applying fresh blood directly to the slide from the syringe and making the blood smear immediately after collection is an effective method of assessing platelet numbers in cats.

SUBMISSION REQUIREMENTS FOR RABIES SUSPECTS

Guidelines for submitting tissue from dead dogs or cats for rabies diagnostic testing vary somewhat from state to state.

Contact the State Veterinary Diagnostic Laboratory or Department of Public Health before shipping any samples. Most public health authorities require advance notification.
about impending submission of samples for rabies testing. Veterinarians should verify the address, paperwork requirements, and shipping requirements before submitting any samples for rabies testing.

Caution: Care must be taken during sample preparation to avoid direct personal contact with specimens. Preexposure rabies vaccination is recommended for persons preparing rabies specimens.

**SAMPLE SUBMISSION FOR RABIES TESTING**

1. Laboratories may limit acceptance of tissue from dead animals for rabies testing to those for which there is a documented reason for considering that animal a rabies-suspect mammal. Generally this includes animals for which there has been a reported bite, scratch, or other possible saliva or nervous tissue exposure of a human.

2. Most laboratories will accept any bat as long as there is reasonable likelihood that a human was exposed.

3. Brain tissue from a rabies-suspect mammal reported to have bitten (or otherwise had “intimate” contact with) a domestic animal will likely be acceptable (e.g., brain tissue from a stray dog or cat that bit a pet dog or cat).

4. Highly suspect surveillance specimens (with no reported human contact) may include:
   a. A rabies vector species (e.g., skunk or raccoon) showing clear signs of rabies infection
   b. A mammal not commonly recognized as a rabies vector but showing clear signs of rabies infection
   c. A domestic animal that dies or is euthanized under the care of a veterinarian for which rabies is part of the differential diagnosis of a neurologic disorder

5. Most laboratories will not accept live animals as rabies suspects. The intact head only of authorized specimens will generally be accepted. Exceptions include bats, which should be submitted whole, and livestock, for which a cross-section of the brainstem and representative sections of brain (as defined by the laboratory) may be removed by a veterinarian and submitted. Special livestock instructions may apply.

**PACKAGING REQUIREMENTS FOR AUTHORIZED SAMPLES**

In the case of a suspect dog or cat, the entire brain must be properly packaged in a standard rabies shipping container (these are often provided at county health departments). Specimens must be accompanied by a completed rabies specimen history form. Forms can often be downloaded from a website designated by the state public health authorities or the diagnostic laboratory.

**INFORMATION REQUESTED ON THE RABIES SPECIMEN HISTORY FORM**

1. Name and address of veterinarian submitting the specimen.
2. Name and address of owner (if known).
3. Indicate whether or not human exposure occurred and the type of exposure (e.g., bite, scratch). Also note whether exposure to a rabid animal is known or highly suspect.
4. Specimen:
   a. Type of specimen
   b. Age, breed, gender, pet versus stray versus wildlife
   c. Cause of death (euthanasia, killed, natural causes)
   d. Medical history of the animal (if known), including date of last rabies inoculation
   e. Health status of the animal at the time of death
5. Location: description of the geographic location (exact address) of the animal when the specimen was collected.
SUBMISSION GUIDELINES

1. Diagnostic testing of the specimen is generally performed by a designated laboratory within the state. Prior authorization to submit a rabies-suspect specimen is generally required; it is always recommended.

2. If the submission is an emergency, or made over a weekend or holiday, most laboratories will provide specific instructions to accommodate a veterinarian’s request.

3. Do not submit live animals.

4. If the suspect animal is alive, it should be humanely euthanized without damaging the head. The head must then be removed from the body and submitted intact for examination. Brain tissue that is damaged may not be accepted by the laboratory. Dead suspect bats can usually be submitted with the head intact.

5. Specimens must be preserved by refrigeration. Freezing should be avoided. Only if refrigeration is not available can the tissue be submitted frozen.

6. Tissues must not be fixed with chemical preservatives.

7. Tools, cages, and other surfaces potentially contaminated with infectious saliva or blood can be disinfected with a solution of sodium hypochlorite (1 part household bleach to 10 parts water).

8. Properly packaged specimens may be shipped directly to the rabies laboratory (verify correct address) by parcel post or commercial mail carrier. Special arrangements are likely to be required for samples arriving over weekends or holidays.

PACKING AND SHIPPING DIRECTIONS

An acceptable rabies suspect shipping set may include any of the following:

1. One preassembled shipping container, including outer cardboard box, insulated cooler, and two gallon-sized cans with lid-locking plastic seal. Packing instructions for package are printed on top inner flaps.

2. Two gel packs of refrigerant (store the pack—not the specimen—frozen until needed).

3. Two plastic bags (13 x 20 inches x 4 mil) in which the animal head, brain of livestock or other large animal, or intact bat is to be sealed before placing in can.

4. Two plastic bags (13 x 20 inches x 4 mil) in which to place the cans.

5. One large plastic bag that surrounds the closed insulated cooler.

6. Two absorbent pads to be placed in the cans, surrounding the specimen.

7. Two blank rabies history forms and directions for collection and submission of specimens.

To prepare the specimen for shipping:

1. Remove the head from the body of the animal (except bats) and place the head in a small plastic bag. Cool specimen in a refrigerator or freezer before packaging, to enhance preservation.

2. When shipping samples consisting of only cerebellum and brainstem, first place the brain tissue in a small plastic container, then place the container in the small plastic bag. If sharp objects protrude from the specimen (e.g., bone fragments, porcupine quills) wrap specimen in several layers of newspaper before putting head in the plastic bag. Wrap bagged specimen in provided absorbent material and place inside the metal can.

3. Place the lid on the metal can and secure with a mallet. Place a plastic pressure ring (provided) on the can and secure with a mallet. The plastic ring will be seated more easily if a hard surface is placed on top of the ring before using the mallet. This will allow even pressure to be applied to the ring. Caution: Infectious splashes can occur when hammering the lid in place if the groove is contaminated with blood or body fluids in the specimen.

4. Wash hands well with soap and water. Disinfect or burn all materials contaminated in specimen preparation.

5. Complete the rabies specimen history form provided with the package. Answer all questions as accurately as possible; the history form will be used to report results to the local health authority. Place form on the outside of the plastic bag that surrounds
the cooler. When shipping more than one specimen in the container (e.g., bats), be certain that each specimen is individually bagged to prevent cross-contamination, each is clearly identified, and a separate history is included for each specimen.

6. **Caution:** Do not use glass, wire, or other packaging materials capable of causing wounds or injuring skin.

**HISTOPATHOLOGY AND CYTOPATHOLOGY**

Histopathology and cytopathology are among the most important diagnostic tools available for use in clinical practice. Generally, diagnostic specimens are submitted to a commercial laboratory or university where specially trained technologists can prepare and stain the cells or tissue to be interpreted by a pathologist. One critical limiting factor in obtaining diagnostic cytology or histopathology is the quality of the specimen submitted. It is the responsibility of the practice not only to obtain but also to prepare specimens properly before submission and interpretation. This part of Section 5 describes standards for preparing and submitting specimens for cytologic or histopathologic interpretation. Sample collection techniques are described in Section 4.

**HISTOPATHOLOGY**

**Biopsy Tissue**

Tissue specimens for histology must be preserved and transported in formalin (10 parts formalin to 1 part tissue). The ideal tissue specimen is less than an inch thick. Occupational Safety and Health Administration (OSHA) and transportation safety regulations limit the size and quantity of formalin containers that can be shipped. It is strongly recommended that containers supplied by the laboratory or the Federal Aviation Administration (FAA)—approved airline be used; place the container in a Ziploc plastic bag, and then in a second outer bag that contains the requisition. Do not use sample containers that are not approved for formalin use. Samples packaged inappropriately may not be picked up by the courier. **Caution:** Do not enclose cytology samples in bags containing formalin-fixed tissues because this may alter the cytologic appearance and staining of the cells of interest.

**Very Large Specimens**

Several (preferably three or more) representative sections of large tissues or organs should be selected, preserved, and transported for histology. The remainder should be placed in a large plastic container of formalin, refrigerated, and retained in case additional samples are needed. If necessary to ship large tissue specimens to a laboratory, use only containers provided by the laboratory or submit fresh tissue in triple leakproof bags.

**Tissue Orientation and Information**

Knowing the orientation and other facts about the tissue mass is critical for the pathologist. A diagram may be included on the requisition form. Borders and areas of interest on the mass can be marked with colored or numbered sutures. State whether the entire mass has been excised, if all is being submitted, or if the tissue had to be divided into sections before submission.

**Very Small Specimens**

Tiny samples, such as endoscopic biopsy specimens, are best preserved if they are first placed in a labeled tissue cassette holder (usually available from the laboratory) and then dropped into formalin. Small biopsy specimens should not be placed in a container with large tissue, because they are easily lost. Do not submit tissue on wooden tongue depressors as the specimen tends to fall off.
CYTOPATHOLOGY

Used alone, as a diagnostic screening test for underlying disease, or in conjunction with the surgical biopsy to facilitate rapid assessment of a potentially serious lesion, cytopathology is among the most fundamental and important diagnostic tools used in clinical practice. Cytopathology is not a clinical discipline restricted to the realm of board-certified clinical pathologists. Several continuing education short courses and laboratories on diagnostic cytopathology are offered at major conferences throughout the United States. In addition, excellent textbooks, with abundant color plates, are available to facilitate cytologic interpretation of specimens collected from dogs and cats.

Cytologic preparations are perhaps most useful for distinguishing details between cell types (e.g., mesenchymal versus epithelial) and cellular activity (e.g., inflammation versus neoplasia). Detection of intracellular versus extracellular organisms can provide immediate clues, without waiting for organisms to be cultured, about the nature of the disease. Noninflammatory lesions can generally be distinguished as benign or neoplastic.

Although it is the responsibility of the individual clinician to understand personal limitations when interpreting cytopathology on individual patients, there is one special advantage that the clinician does have over the pathologist—familiarity with the patient’s health status and the nature of the lesion or disease under consideration. Described here are guidelines for preparing and submitting samples for cytologic interpretation (see Section 4 for sample collection techniques). Whether samples are sent to a commercial laboratory or a university, or are interpreted within the practice, the recommendations that follow are important when preparing a high-quality specimen.

Note: The accuracy of interpreting cytopathologic specimens is dependent on four key variables:
- Experience and training of the clinician
- Selection of the appropriate case and lesion
- Cellular quality of the specimen selected
- Techniques used to collect, prepare, and stain the sample

FINE-NEEDLE ASPIRATION

Indications
FNA involves the use of a syringe and needle to extract cells from a palpable lesion. Most commonly, FNA is performed on cutaneous and subcutaneous lesions. However, with the increasing use of ultrasound in private practice, it may not be necessary to actually “palpate” a lesion in order to extract diagnostic cytology (e.g., ultrasound-guided hepatic or splenic aspirates). Additional experience and training are essential when attempting to perform ultrasound-guided FNA.

Sample Preparation
Because sample size typically is small, the cells collected are discharged directly onto a dry, clean slide and allowed to rapidly (within 5 to 10 seconds) air-dry. It is recommended that the needle tip actually contact the slide as the aspirate is discharged rather than blowing the sample over the slide. If clear fluid is inadvertently recovered, the FNA should be reattempted from the peripheral limits of the lesion.

An extremely small harvest of cells can be sprayed directly on the slide, remain untouched, and allowed to air-dry. If the volume recovered allows placement of a formed drop onto the slide, the sample should be spread over the surface of the slide, before to air-drying, in the same way that a peripheral blood smear is prepared.

Staining Options
Once the sample has air-dried (rapidly), use of a quick Romanowsky-type (Wright) stain is appropriate. Alcohol fixation is not recommended if the specimen is to be reviewed and interpreted immediately. Alternative stains, such as new methylene blue (wet mount),
Gram stain, Giemsa stain, or Wright-Giemsa stain, can be used in practice as dictated by cytologic objectives.

FNA specimens mailed to an outside laboratory typically are air-dried and left unstained. Some laboratories recommend that the specimen be immersed in methyl alcohol for a few minutes before sending, although this additional step seems to be optional.

**Common Mistakes**

Low cell harvest, high cellular density on the slide (e.g., the result of making a “bad” slide or failing to adequately disperse the sample), and obtaining nondiagnostic material are the three most common mistakes when obtaining samples for diagnostic cytopathology. Contamination of the “wet” (not yet air-dried) sample with water, alcohol, or stain can create artifacts that will compromise the diagnostic value of the specimen. Excessive blood or tissue fluids may profoundly dilute the diagnostic sample, making interpretation difficult or impossible.

**Exfoliative Cytology (“Impression Smear”)**

**Indications**

Exfoliative cytology is made from a clean surface of exposed lesions or from the surface of tissue collected during biopsy. Preparations made from the cut surface of fresh biopsy specimens or postmortem tissues provide the greatest diagnostic yield.

**Sample Preparation**

For prevention of one of the most common mistakes, excessive tissue fluid or blood is absorbed from the cut surface (using a scalpel blade) of the specimen before the attempt to exfoliate cells on a slide. Clean, high-quality absorptive paper (such as filter paper) works well, and fragments of paper will not be left on the specimen.

Once excess fluid has been absorbed from the surface, the specimen is gently grasped and allowed to make gentle contact with a clean slide. The actual weight of the specimen is usually sufficient; it is usually not necessary to press the specimen onto the slide. After multiple contacts with the slide have been made, the sample is rapidly air-dried.

**Staining Options**

Once the sample has air-dried (rapidly), use of a quick Romanowsky-type (Wright) stain is appropriate. Alcohol fixation is not recommended if the specimen is to be reviewed and interpreted immediately. Alternative stains, such as new methylene blue and Gram stain (wet mounts), Giemsa stain, or Wright-Giemsa stain, can be used in practice as dictated by cytologic objectives.

**Common Mistakes**

Excessive or rough handling of the specimen before attempting exfoliation will compromise the quality of the specimen. In addition, excessive blood or tissue fluid on the cut surface of the tissue may effectively “dilute” the diagnostic cells in the specimen, making interpretation difficult. When additional pressure is used to exfoliate cells or the specimen is rubbed across the slide, individual cells are likely to rupture and smear, rendering the sample nondiagnostic. Failure to obtain adequate numbers of diagnostic cells is more likely to be the consequence of the type of tissue being examined than poor technique. Epithelial tissues (liver, spleen, adenoma, carcinoma) tend to exfoliate abundant numbers of cells when applied to a slide. In contrast, mesenchymal cell tissues (fibrosarcoma, chondrosarcoma) tend not to exfoliate well. Diagnostic yield of cells from mesenchymal tissue may be so low as to warrant submission of fixed tissue for histopathologic examination.

**Swabs, Scrapings, Washings, or Brushings**

**Indications**

A variety of techniques are available to collect cytologic specimens from the upper and lower respiratory tract, conjunctiva, ear canals, and vaginal mucosa. In most cases, cytologic objectives focus on the recovery and identification of infectious organisms (e.g., mites, bacteria). Section 4 describes the various techniques of sample collection from these locations.
Sample Preparation
Skin scrapings and ear swabs for diagnosis of infectious agents, and occasionally neoplasia, are perhaps the most common samples used in practice to collect diagnostic specimens. Gentle handling of the specimen once collected is the rule when attempting to exfoliate diagnostic cells or organisms. In addition, it may not be necessary to air-dry or apply a stain, depending on the samples collected (e.g., skin scrapings or ear swabs for mites).

Samples collected from washings vary considerably in the cell harvest, the consistency of the fluid recovered, and the quality of the diagnostic specimen. In some cases, fluid recovered from washings (e.g., bronchoalveolar lavage, transtracheal aspiration) will require centrifugation to acquire sufficient numbers of diagnostic cells. The supernatant (fluid portion) of the sample is discarded. The cells recovered may be resuspended in one or two drops of sterile saline or a volume of saline equal to the volume of specimen remaining in the centrifuge tube. A pipette is used to apply a sample of the fluid to a slide. The sample is distributed over the slide in the same way that a peripheral blood smear is prepared. The slide is air-dried and stained. In other cases, the sample collected from cytologic washings will be highly cellular and may be applied directly to a slide, air-dried, and stained.

Samples collected from brushings normally are obtained with specially made cytology brushes designed for use during endoscopy. Although small “pinch” biopsies are preferred, occasionally the use of a brush may be the only practical option. Cytologic specimens collected by brushing tend to be especially low in yield. Furthermore, the additional manipulation required to extract cells from the brush and onto a slide for examination tends to yield specimens of poorer quality. Cells obtained during brushing may be applied directly to a clean slide, air-dried, and stained. In other cases it may be preferable to wash the brush in a centrifuge tube containing a small volume (<1.0 mL) of sterile saline. The suspended cells may be applied directly to a slide, distributed, and then air-dried and stained. It may be necessary to centrifuge the sample (as described for washings previously) before preparing the sample.

Staining Options
Generally, the same staining options previously described apply to specimens collected from washings or brushings. Samples collected from skin scrapings typically are suspended in oil or hydrogen peroxide on the slide and examined “wet” without the use of additional stain. Swabs, especially from ears, may be stained with a quick Romanowsky-type stain or a Gram stain (wet mount) to facilitate identification of organisms.

Common Mistakes
Samples collected from skin scrapings and swabs tend to be relatively high in yield when diagnostic cells or organisms are present. Cells collected from washings and brushings are usually collected during endoscopic procedures; the yield of diagnostic cells can vary, depending on the extent of the lesion as well as the skill of the individual performing the procedure.

Body Fluids
Indications
The accumulation of fluid in either the pleural space or the abdomen, or in both, justifies attempts to remove fluid for diagnostic cytology. The volume of sample can be difficult to determine but ideally would be 2 to 3 mL of fluid collected by needle and syringe (centesis) under sterile conditions. Smaller samples of joint fluid and cerebrospinal fluid (CSF) are also collected for chemical and cytologic analysis. Any fluid recovered should be examined for color, consistency, total nucleated cell count, and protein concentration as well as for morphology of the cells recovered. Other chemistries (e.g., creatinine, amylase) can be determined depending on the nature of the fluid recovered and the patient’s condition.

Sample Preparation
Because the volume of fluid obtained may be large and the concentration of cells in the fluid recovered may be low, centrifugation is indicated to concentrate cells in small aliquots of fluid. After centrifugation and removal of the supernatant, cells can be resuspended in
1 to 2 drops of sterile saline or supernatant. Suspended cells should be distributed directly on a slide, allowed to air-dry, and stained.

When submitting fluids or washes to a commercial laboratory, it is recommended to place fluid samples into a sterile tube without clot activator (e.g., red and dark gray tube; see Table 5-6) or a (preferred) lavender-topped tube (which contains EDTA). Samples submitted in EDTA cannot be used for bacterial culture. Do not submit samples in an SST, as these tubes contain a clot activator.

Note: It is important not to delay processing of cytologic samples recovered from body fluid. The longer cells are allowed to remain in suspension, the greater the opportunity for morphologic changes of cells to occur.

Spinal fluid must be processed within 30 minutes of collection because of the fragility of cells in CSF. Furthermore, conventional centrifuges may damage any cells collected. Because of the complexities associated with processing of CSF for cytopathology, most samples are evaluated within specialty or referral hospitals.

Staining Options
Air-dried cytologic preparations can be stained in the same manner described previously.

Common Mistakes
Attempting to evaluate uncentrifuged cytologic specimens collected from body fluids can result in a low yield of diagnostic cells and may compromise the study. Allowing the cells to remain in the fluid for an extended period of time before making the cytologic preparations may significantly alter the morphology of individual cells, making interpretation difficult or impossible. Furthermore, the presence of peripheral blood in any sample collected from a body cavity must be distinguished from contamination associated with the sampling technique versus a primary bleeding disorder. When submitting slides with tissue fixed in formalin, do not package slides with the formalin vial. Even small amounts of formalin can significantly disrupt cell morphology.

Bone Marrow

Indications
Cytologic examination of a bone marrow aspirate is an especially valuable tool in the assessment of patients with persistent anemia, particularly nonregenerative anemia, abnormal numbers (either high or low) of leukocytes, thrombocytopenia, any blood dyscrasia detected in peripheral blood, and any combination of these findings. Bone marrow specimens will yield the most information if both a core biopsy and aspirate slides are submitted. The biopsy specimen should be cut first, and the core placed in a tissue-processing cassette, labeled, and dropped into a formalin container. The aspirate needle can then be placed into the same puncture site as the biopsy needle. (Bone marrow biopsy and aspiration collection techniques are described in Section 4.)

Thrombocytopenia is not necessarily a contraindication to performing bone marrow aspiration. Assuming normal platelet function, bone marrow aspiration is indicated even when platelet counts are extremely low (e.g., 5000 platelets/mm³). We have, however, observed persistent bleeding and large hematoma formation at the site of aspiration in dogs with platelets counts of less than 3000 platelets/mm³ in peripheral blood.

Sample Preparation
In most patients undergoing bone marrow aspiration, sufficient numbers of platelets will be present in the sample to justify routine use of an anticoagulant. Before collection of the sample, a few drops of 4% EDTA are placed in the center of a watch glass. The same 12-mL syringe used to draw the EDTA is used to collect the sample. This syringe will contain a small amount of EDTA. Collection of marrow is typically limited to 0.5 mL. Larger volumes may cause hemodilution of the sample, making interpretation difficult. On withdrawal of the appropriate volume, the sample is immediately added directly to the EDTA.
and mixed thoroughly. A glass pipette can be used to transfer the aspirated marrow onto a clean, dry slide. Other techniques are described in Section 4. Using the same technique to distribute peripheral blood for a differential count will suffice. The sample is allowed to air-dry.

**Staining Options**
Bone marrow staining routinely entails use of a quick Romanowsky-type stain. Alternatively, when transporting slides to a laboratory, do not apply any staining. Ensure the slides do not make contact with formalin-fixed samples; do not refrigerate slides. Special staining, usually performed by a commercial or university laboratory, may be indicated when looking for the presence of iron stores or specific types of organisms.

**Common Mistakes**
If the bone marrow contains functional platelets, failing to quickly transfer the aspirate into the EDTA can result in clot formation. The presence of clots is likely to entrap diagnostic cells, making interpretation difficult or impossible. Hemodilution and an excessive volume of EDTA are also common mistakes that can compromise the quality of the smears. Other complications usually are caused by errors in the technique of making the slide. For example, failing to adequately distribute the sample across the slide can result in an unusually thick preparation. Bone marrow aspirates taken from the head of the humerus can become contaminated with joint fluid, making the sample completely unusable.

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### BIOCHEMISTRY—ROUTINE

The ability to obtain a comprehensive biochemical profile, and to do so quickly and inexpensively, has made such testing a routine part of the clinical workup for the companion animal patient. Clearly, the biochemistry profile greatly expands the clinician’s ability to assess the patient with a history of clinical illness. In addition, it is now feasible to obtain a biochemical profile on seemingly healthy patients as part of a routine “wellness examination.”

This section discusses those analytes offered by most clinical laboratories performing companion animal (dog and cat) biochemistry profiles. Although specific analytes included on panels vary among laboratories, any individual test not discussed here is likely to be found in the portion of this section entitled Special Diagnostic Tests and Test Protocols.

The following criteria are applicable to all samples in which blood, serum, or plasma is collected and for which a routine biochemistry profile or special laboratory test is requested.

#### ANALYTE OR TEST NAME (SYNONYMS)

The name of the individual chemical analyte being measured (e.g., alkaline phosphatase) is followed in parentheses by common abbreviations used by laboratories when reporting results (e.g., SAP or alk phos). In some cases the name of the test is presented rather than the actual chemical being tested for (e.g., ACTH stimulation, in which cortisol is the actual analyte measured).

#### NORMAL

Representative reference range values for normal adult dogs and cats are listed with each analyte. In addition, Tables 5-3 to 5-7 summarize reference ranges for dogs and cats.

**Note:** Reference range values listed throughout this section are for general reference only. Test results from individual patients must be compared with the reference range values of the laboratory that performs the test.
### TABLE 5 - 3 Hematology Reference Range Values

<table>
<thead>
<tr>
<th>Test</th>
<th>Adult Canine</th>
<th>Adult Feline</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red blood cell (total)</td>
<td>5.32-7.75</td>
<td>6.68-11.8</td>
<td>×10^8 cells/mm³</td>
</tr>
<tr>
<td>Hemoglobin (Hgb)</td>
<td>13.5-19.5</td>
<td>11.0-15.8</td>
<td>grams</td>
</tr>
<tr>
<td>Hematocrit (Hct)</td>
<td>39.4-56.2</td>
<td>33.6-50.2</td>
<td>%</td>
</tr>
<tr>
<td>Mean corpuscular volume (MCV)</td>
<td>65.7-75.7</td>
<td>42.6-55.5</td>
<td>fl</td>
</tr>
<tr>
<td>Mean corpuscular hemoglobin (MCH)</td>
<td>22.57-27.0</td>
<td>13.4-18.6</td>
<td>pg</td>
</tr>
<tr>
<td>Mean corpuscular hemoglobin concentration (MCHC)</td>
<td>34.3-36.0</td>
<td>31.3-33.5</td>
<td>g/dL</td>
</tr>
<tr>
<td>Platelet count</td>
<td>194-419</td>
<td>198-405</td>
<td>×10⁴ cells/mm²</td>
</tr>
<tr>
<td>Mean platelet volume (MPV)</td>
<td>8.8-14.3</td>
<td>11.3-21.3</td>
<td>fl</td>
</tr>
<tr>
<td>White blood cell (total)</td>
<td>4.36-14.8</td>
<td>4.79-12.52</td>
<td>×10⁹ cells/mm³</td>
</tr>
<tr>
<td>Segmented neutrophils (segs)</td>
<td>3.4-9.8</td>
<td>1.6-15.6</td>
<td>×10⁹ cells/mm³</td>
</tr>
<tr>
<td>Nonsegmented neutrophils (bands or nonsegs)</td>
<td>0-0.01</td>
<td>0-0.01</td>
<td>×10⁹ cells/mm³</td>
</tr>
<tr>
<td>Lymphocytes (lymphs)</td>
<td>0.8-3.5</td>
<td>1.0-7.4</td>
<td>×10⁹ cells/mm³</td>
</tr>
<tr>
<td>Monocytes (monos)</td>
<td>0.2-1.1</td>
<td>0-0.7</td>
<td>×10⁹ cells/mm³</td>
</tr>
<tr>
<td>Eosinophils (eos)</td>
<td>0-1.9</td>
<td>0.1-2.3</td>
<td>×10⁹ cells/mm³</td>
</tr>
<tr>
<td>Basophils (basos)</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

### TABLE 5 - 4 Biochemistry Reference Range Values

<table>
<thead>
<tr>
<th>Test</th>
<th>Adult Canine</th>
<th>Adult Feline</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>73-116</td>
<td>63-150</td>
<td>mg/dL</td>
</tr>
<tr>
<td>Blood urea nitrogen (BUN)</td>
<td>8-27</td>
<td>15-35</td>
<td>mg/dL</td>
</tr>
<tr>
<td>Creatinine (Cr)</td>
<td>0.5-1.6</td>
<td>0.5-2.3</td>
<td>mg/dL</td>
</tr>
<tr>
<td>Phosphorus (P)</td>
<td>2.0-6.7</td>
<td>2.7-7.6</td>
<td>mg/dL</td>
</tr>
<tr>
<td>Calcium (Ca)</td>
<td>9.2-11.6</td>
<td>7.5-11.5</td>
<td>mg/dL</td>
</tr>
<tr>
<td>Ionized calcium (iCa)</td>
<td>1.15-1.39</td>
<td>—</td>
<td>mg/dL</td>
</tr>
<tr>
<td>Total protein (TP)</td>
<td>5.5-7.2</td>
<td>5.4-8.9</td>
<td>g/dL</td>
</tr>
<tr>
<td>Albumin (Alb)</td>
<td>2.8-4.0</td>
<td>3.0-4.2</td>
<td>g/dL</td>
</tr>
<tr>
<td>Globulin (Glob)</td>
<td>2.0-4.1</td>
<td>2.8-5.3</td>
<td>g/dL</td>
</tr>
<tr>
<td>A:G ratio</td>
<td>0.6-2.0</td>
<td>0.4-1.5</td>
<td>—</td>
</tr>
<tr>
<td>Cholesterol (Ch)</td>
<td>138-317</td>
<td>42-265</td>
<td>mg/dL</td>
</tr>
<tr>
<td>Bilirubin (Total)</td>
<td>0-0.2</td>
<td>0.1-0.5</td>
<td>mg/dL</td>
</tr>
<tr>
<td>Alkaline phosphatase (SAP or alk phos)</td>
<td>15-146</td>
<td>0-96</td>
<td>IU/L</td>
</tr>
<tr>
<td>Alanine aminotransferase (ALT)</td>
<td>16-73</td>
<td>5-134</td>
<td>IU/L</td>
</tr>
<tr>
<td>γ-Glutamyltransferase (GGT)</td>
<td>3-8</td>
<td>0-10</td>
<td>IU/L</td>
</tr>
<tr>
<td>Creatine kinase (CK; formerly CPK)</td>
<td>48-380</td>
<td>72-481</td>
<td>IU/L</td>
</tr>
<tr>
<td>Sodium (Na)</td>
<td>147-154</td>
<td>147-165</td>
<td>mEq/L</td>
</tr>
<tr>
<td>Potassium (K)</td>
<td>3.9-5.2</td>
<td>3.3-5.7</td>
<td>mEq/L</td>
</tr>
<tr>
<td>Na:K ratio</td>
<td>27.4-38.4</td>
<td>30-43</td>
<td>—</td>
</tr>
<tr>
<td>Chloride (Cl)</td>
<td>104-117</td>
<td>113-122</td>
<td>mEq/L</td>
</tr>
<tr>
<td>Bicarbonate (venous)</td>
<td>20-29</td>
<td>22-24</td>
<td>mEq/L</td>
</tr>
<tr>
<td>Anion gap</td>
<td>16.3-28.6</td>
<td>15-32</td>
<td>—</td>
</tr>
<tr>
<td>Osmolality (calculated)</td>
<td>292-310</td>
<td>290-320</td>
<td>mOsm/kg</td>
</tr>
<tr>
<td>Amylase</td>
<td>347-1104</td>
<td>489-2100</td>
<td>IU/L</td>
</tr>
<tr>
<td>Lipase</td>
<td>22-216</td>
<td>0-222</td>
<td>IU/L</td>
</tr>
<tr>
<td>Triglyceride (TG)</td>
<td>19-133</td>
<td>24-206</td>
<td>mg/dL</td>
</tr>
</tbody>
</table>
**PATIENT PREPARATION**

Any unique patient preparation parameters should be followed before the sample is collected. For routine biochemistry profiles, an 8- to 10-hour fasting period is recommended when feasible. When performing routine profiles on normal patients, it is preferable to collect samples in the morning. Owners are instructed to withhold food and water after midnight on the day the blood sample is to be collected.

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**TABLE 5 - 5  Urinalysis (Voided Sample) Reference Range Values**

<table>
<thead>
<tr>
<th>Test</th>
<th>Canine</th>
<th>Feline</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific gravity (SpGr)</td>
<td>Variable</td>
<td>Variable</td>
</tr>
<tr>
<td>Color</td>
<td>Pale to dark yellow</td>
<td>Pale to dark yellow</td>
</tr>
<tr>
<td>pH</td>
<td>5.0 to 8.5</td>
<td>5.0 to 8.5</td>
</tr>
<tr>
<td>Protein</td>
<td>Negative to +1</td>
<td>Negative to +1</td>
</tr>
<tr>
<td>Glucose</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Ketones</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>Negative to trace</td>
<td>Negative</td>
</tr>
<tr>
<td>Blood</td>
<td>Negative</td>
<td>Negative</td>
</tr>
</tbody>
</table>

**Microscopic**

- Red blood cell (RBC) count: <5 RBCs/hpf
- White blood cell (WBC) count: <3 WBCs/hpf
- Epithelial cells: Negative
- Casts: Negative
- Bacteria: Negative
- Special: Urine protein:creatinine: <0.3

**TABLE 5 - 6  Hemostasis Reference Range Values**

<table>
<thead>
<tr>
<th>Test</th>
<th>Canine</th>
<th>Feline</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelet count</td>
<td>166-600 × 10³/µL</td>
<td>230-680 × 10³/µL</td>
</tr>
<tr>
<td>Prothrombin time (PT)</td>
<td>5.1-7.9 sec</td>
<td>8.4-10.8 sec</td>
</tr>
<tr>
<td>Activated partial thromboplastin time (APTT)</td>
<td>8.6-12.9 sec</td>
<td>13.7-30.2 sec</td>
</tr>
<tr>
<td>Fibrin degradation products (FDPs)</td>
<td>&lt;10 mcg/mL</td>
<td>&lt;10 mcg/mL</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>100-245 mg/dL</td>
<td>110-370 mg/dL</td>
</tr>
<tr>
<td>Activated clotting time (ACT)</td>
<td>60-110 sec</td>
<td>50-75 sec</td>
</tr>
</tbody>
</table>

**TABLE 5 - 7  Blood Gas Analysis—Arterial Reference Range Values**

<table>
<thead>
<tr>
<th>Test</th>
<th>Canine</th>
<th>Feline</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.36-7.44</td>
<td>7.36-7.44</td>
<td>—</td>
</tr>
<tr>
<td>Po₂</td>
<td>90-100</td>
<td>90-100</td>
<td>mmHg</td>
</tr>
<tr>
<td>PcO₂</td>
<td>36-44</td>
<td>28-32</td>
<td>mmHg</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>24-26</td>
<td>20-22</td>
<td>mEq/L</td>
</tr>
<tr>
<td>TCO₂</td>
<td>25-27</td>
<td>21-23</td>
<td>mEq/L</td>
</tr>
</tbody>
</table>
COLLECT
This section stipulates the type and volume of sample to be collected, as well as the type of collection tube to be used. For routine biochemistry, collecting at least 2.0 mL of whole blood in an RTT (or SST) is required to obtain the minimum 1.0 mL of serum required for sample analysis. Dehydrated patients have a higher Hct, and a larger volume of whole blood may be required to obtain 1.0 mL of serum.

SUBMIT
This section stipulates the type and volume of sample that is to be submitted for analysis. Also, the type of vial or container in which the sample should be shipped is specified. Unless specified in the protocol (e.g., polymerase chain reaction [PCR] analyses), do not store or ship samples as whole blood; instead, separate whole blood from serum before shipping or place blood in an appropriate SST. Serum samples should be shipped in a sterile RTT. Freezing of the sample is not required for routine biochemistry profiles.

INTERPRETATION
Each analyte and test procedure is described separately. For each analyte, an interpretive summary of the significance of an abnormal (either elevated or decreased) value is provided. Test results for any analyte must be interpreted after consideration of other laboratory results (e.g., hematology, urinalysis) as well as the patient's medical history and physical examination findings.

INTERFERENCE
This section stipulates common interfering substances and factors and indicates, when known, if the interference will falsely elevate or lower test results. Samples that are lipemic, icteric, and/or hemolized may cause test interference with individual analyte assays, resulting in unreliable test results. Interference may be positive (false increased test results) or negative (false decreased test results). The degree and type of interference vary depending on the test methodology used. In the final reports, most laboratories provide details pertaining to known or potential interfering factors.

PROTOCOL
If indicated, the protocol stipulates specific test procedures or shipping requirements necessary to obtain the most valid results. For routine biochemistry profiles, other than recommended fasting of the patient, no specific test protocol is indicated. A detailed description is provided for special laboratory tests that require specific patient preparation or adherence to a unique test protocol.

ALANINE AMINOTRANSFERASE (ALT; FORMERLY SGPT)
NORMAL
16 to 73 international units/L (dog); 5 to 134 international units/L (cat)

INTERPRETATION
ALT is used in the assessment of liver disease (not a test of liver function). Increased values indicate hepatocyte injury and leakage of intracellular enzymes, such as could occur in acute hepatitis, hepatic trauma, neoplasia (occasionally), and cirrhosis. Decreased values may be noted in end-stage liver disease.
ALBUMIN

**NORMAL**
2.8 to 4.0 g/dL (dog); 3.0 to 4.2 g/dL (cat)

**INTERPRETATION**
Evaluated with total protein and globulin. This test is important in the assessment of hydration status, renal disease, gastrointestinal (GI) disease, liver function, and selected chronic infectious diseases. Increased values generally support dehydration; a commensurate increase in globulin and total protein should be expected. Decreased values suggest abnormal loss (GI tract or renal) and decreased production (protein-restricted diet, malnutrition, liver disease). Values in healthy young dogs and cats (<3 months of age) are normally lower than those in adult animals.

ALBUMIN/GLOBULIN RATIO (A:G)

**NORMAL**
0.6 to 2.0 (dog); 0.4 to 1.5 (cat)

**INTERPRETATION**
The A:G should not be interpreted without consideration of the concentration (g/dL) of both albumin and globulin. Further characterization of serum proteins can be obtained with serum protein electrophoresis. An increased A:G is considered to be clinically insignificant because it represents elevated albumin and/or decreased globulin. Alternatively, a decreased A:G indicates decreased albumin and/or increased globulin and may indicate renal or GI loss of albumin, certain neoplasms, or chronic infections.

SERUM ALKALINE PHOSPHATASE (SAP OR “ALK PHOS”)

**NORMAL**
15 to 146 international units/L (dog); 0 to 96 international units/L (cat)

**INTERPRETATION**
SAP is routinely used to assess obstructive liver and/or biliary tract disease (not a test of liver function). Increased values are normal in young dogs and cats (<3 months of age) (reflecting bone growth). In adults, increased values may indicate biliary obstruction or cholestasis, hepatitis, hepatic lipidopathy, destructive bone lesions (osteosarcoma), hyperphosphatemia, and acute pancreatitis. Corticosteroid therapy will induce SAP, causing significant elevations in the absence of cholestasis.

AMYLASE

**NORMAL**
347 to 1104 international units/L (dog); 489 to 2100 international units/L (cat)

**INTERPRETATION**
Increased value indicates pancreatitis, especially in patients with evidence of vomiting and abdominal pain. Amylase clearance is dependent on normal renal function; patients with compromised renal function (chronic renal failure) are likely to have abnormally elevated amylase not associated with pancreatic disease. Pancreatic lipase immunoreactivity (PLI) may be helpful in assessing pancreatitis in dogs and cats (see under Special Diagnostic Tests and Test Protocols).
ANION GAP

NORMAL
16.3 to 28.6 (dog); 12 to 24 (cat)

INTERPRETATION
The anion gap is a laboratory calculation (Na − [Cl + HCO₃⁻] = anion gap) used to assess quantities of unmeasured cations (Ca, Mg) and anions (proteins, sulfates, phosphates, and certain organic acids). A high anion gap suggests metabolic acidosis (ketoadidosis, lactic acidosis). Other causes of metabolic acidosis (e.g., renal tubular acidosis) may have a normal anion gap. Hypoalbuminemia is the most common cause of a low anion gap. Other causes include hypernatremia, certain gammopathies (myeloma), and severe hypercalcemia. There are numerous causes for false high and low anion gap results.

ASPARTATE AMINOTRANSFERASE (FORMERLY SGOT)

Although sometimes reported in companion animal laboratory profiles, aspartate aminotransferase (formerly SGOT) values are not considered to have clinical significance in either the dog or the cat.

BICARBONATE (HCO₃⁻)

NORMAL
24 to 26 mEq/L (dog); 22 to 24 mEq/L (cat)

INTERPRETATION
Bicarbonate measurement usually is included as a component of blood gas and/or electrolyte panel. Levels are increased with metabolic alkalosis (and with compensated respiratory acidosis) and decreased with metabolic acidosis (and with compensated respiratory alkalosis).

BILIRUBIN

NORMAL
0 to 0.2 mg/dL (dog); 0.1 to 0.5 mg/dL (cat)

INTERPRETATION
Increased value (hyperbilirubinemia) may be associated with icterus or jaundice, reflects accumulation of bilirubin in serum, and may indicate intravascular hemolysis, compromised bile excretion, biliary tract obstruction (intrahepatic or extrahepatic), and primary hepatic disease affecting bile excretion.

BLOOD UREA NITROGEN (BUN)

NORMAL
8 to 27 mg/dL (dog); 15 to 35 mg/dL (cat)

INTERPRETATION
Note: Abnormally elevated BUN (azotemia) does not define “uremia.” Increased BUN indicates decreased renal clearance of nitrogenous waste (dehydration, renal failure, urinary tract obstruction). An elevated BUN is not indicative of renal disease unless interpreted in light of other parameters (e.g., urine specific gravity, serum creatinine, history of increased water consumption or increased urination). Decreased BUN indicates increased renal excretion of nitrogenous waste (diuresis) or decreased protein intake (malnutrition, low-protein diet) or decreased production (portosystemic shunt).
CALCIUM (Ca)
NORMAL
9.2 to 11.6 mg/dL (dog); 7.5 to 11.5 mg/dL (cat)

INTERPRETATION
Warning: Levels ≤7 mg/dL dogs and cats may result in tetany; sustained levels >12 mg/dL may cause renal damage subsequent to calcium deposition. Increased levels are associated with primary hyperparathyroidism, pseudohyperparathyroidism (paraneoplastic syndrome associated with neoplasia, especially lymphosarcoma and perianal carcinoma), metastatic bone disease or primary bone tumors, hypervitaminosis D (chronic), hyperthyroidism (in cats), Addison disease (hypoadrenocorticism), and acromegaly. Hypercalcemia may be idiopathic in some animals. Decreased values are associated with any condition causing low total protein and albumin levels (most serum calcium is albumin-bound). Serum ionized calcium (iCa) is indicated in assessing any patient with significant, unexplained hypercalcemia or hypocalcemia. Other causes of decreased calcium include conditions that cause elevated phosphorus levels (e.g., renal insufficiency, hypoparathyroidism), acute pancreatitis, intravenous fluid administration, and renal tubular acidosis. See also Calcium, Ionized (iCa).

CHLORIDE (Cl)
NORMAL
104 to 117 mEq/L (dog); 113 to 122 mEq/L (cat)

INTERPRETATION
Increased Cl is associated with dehydration as well as intravenous saline administration. Decreased Cl can be associated with overhydration, Addison disease (hypoadrenocorticism), burns, metabolic alkalosis, syndrome of inappropriate secretion of antidiuretic hormone (ADH), and certain types of diuretic therapy.

CHOLESTEROL (CH)
NORMAL
138 to 317 mg/dL (dog); 42 to 265 mg/dL (cat)

INTERPRETATION
Increased CH (hypercholesterolemia) is most commonly found in hyperlipidemic patients and reflects extreme elevations of triglyceride rather than a primary underlying metabolic disorder affecting CH metabolism. In dogs, hypercholesterolemia is inconsistently associated with hypothyroidism and hyperadrenocorticism (Cushing syndrome). Hypercholesterolemia has limited diagnostic significance. Decreased CH (hypocholesterolemia) has not been found to be of diagnostic significance in the dog and cat but has been observed with hypoadrenocorticism.

CREATINE KINASE (CK; FORMERLY CPK)
NORMAL
48 to 380 international units/L (dog); 72 to 481 international units/L (cat)

INTERPRETATION
Increased CK indicates increased skeletal muscle activity or destruction (myopathy or rhabdomyolysis), inflammation or infection (myositis), or widespread muscle trauma. No diagnostic significance has been associated with a decreased CK.
CREATININE (Cr)

**NORMAL**
0.5 to 1.6 mg/dL (dog); 0.5 to 2.3 mg/dL (cat)

**INTERPRETATION**
Increased Cr is an important indicator of glomerular filtration and occurs with renal insufficiency and urinary tract obstruction; shock, severe dehydration, and untreated congestive heart failure may result in increased Cr owing to decreased renal blood flow. Rhabdomyolysis will also cause increased Cr. Pathologic causes of decreased Cr are uncommon but may occur in severe debilitation or disease that causes extreme decreases in muscle mass. Cr is less influenced by diet than by BUN.

**γ-GLUTAMYLTRANSFERASE (GGT; GAMMA GT [gGT])**

**NORMAL**
3 to 8 international units/L (dog); 0 to 10 international units/L (cat)

**INTERPRETATION**
Parameters causing increased and decreased GGT typically parallel SAP in the presence of underlying liver pathology, especially cholestasis, but not in patients with destructive bone disease. GGT is commonly elevated in cirrhosis and (obstructive) hepatic or biliary tract disease. Extreme elevations of GGT have been associated with metastatic liver disease in humans; a similar association has not been reported in animals.

**GLOBULIN**

**NORMAL**
2.0 to 4.1 g/dL (dog); 2.8 to 5.3 g/dL (cat)

**INTERPRETATION**
Globulin is a component of total protein that must be interpreted with albumin. Increased value (hyperglobulinemia) may reflect dehydration (albumin and total protein also increased), chronic inflammation, chronic infection, or myeloid neoplasia (albumin may be abnormally decreased). Serum protein electrophoresis is indicated to characterize the nature of the globulin increase. Decreased value (hypoglobulinemia) typically indicates decreased protein intake (low protein diet or malnutrition) or decreased globulin production (neoplasia).

**GLUCOSE**

**NORMAL**
73 to 116 mg/dL (dog); 63 to 150 mg/dL (cat)

**INTERPRETATION**
Increased value (hyperglycemia) indicates decreased glucose metabolism (insulin deficiency or diabetes mellitus). *Note:* Normal cats may experience transient “stress hyperglycemia” with values as high as 350 mg/dL (typically, glycosuria is absent). Decreased value (hypoglycemia) indicates excessive usage of glucose (insulin-secreting tumor) or severe illness (sepsis).

**LIPASE**

**NORMAL**
22 to 216 international units/L (dog); 0 to 222 international units/L (cat)
INTERPRETATION
Increased lipase is most commonly associated with acute pancreatitis. Certain neoplasms have been reported to cause extreme elevations of lipase in the absence of pancreatic disease. There is no clinical significance associated with decreased lipase.

PHOSPHORUS (P)
NORMAL
2.0 to 6.7 mg/dL (dog); 2.7 to 7.6 mg/dL (cat)
INTERPRETATION
Increased P is normally present in young, growing dogs and cats (associated with increase SAP activity). Abnormal elevations are most likely to occur in patients with chronic renal failure or hypoparathyroidism. Improper sample handling (hemolysis) can cause elevations in P. Decreased P is expected in patients with primary hyperparathyroidism (with increased calcium), renal tubular acidosis, and Fanconi syndrome. Several systemic illnesses may be associated with decreased P. Warning: Values of 1 mg/dL or less may be associated with neuromuscular abnormalities and cardiac arrhythmia.

POTASSIUM (K)
NORMAL
3.9 to 5.2 mEq/L (dog); 3.3 to 5.7 mEq/L (cat)
INTERPRETATION
Increased value (hyperkalemia) may indicate mineralocorticoid deficiency (Addison disease or hypoadrenocorticism) but must be interpreted with serum sodium and an adrenocorticotropic hormone (ACTH) stimulation test. Numerous causes of decreased potassium are recognized. GI and renal losses are the most common and most significant. Persistent hypokalemia warrants significant efforts to determine the underlying cause(s).
Warning: Potassium levels greater than 7.5 mEq/L may cause cardiac arrhythmias (profound bradycardia) and death. Potassium levels less than 2.5 mEq/L may cause profound weakness.

SODIUM (Na+)
NORMAL
147 to 154 mEq/L (dog); 147 to 165 mEq/L (cat)
INTERPRETATION
Increased value (hypernatremia) may result from excess dietary consumption or severe dehydration. Decreased value (hyponatremia) may indicate mineralocorticoid deficiency (Addison disease or hypoadrenocorticism) but must be interpreted in light of other tests (e.g., serum osmolality, potassium, ACTH stimulation test). Persistent diuresis caused by drugs (furosemide) or an inherent medical disorder (nephrotic syndrome) can deplete serum sodium to significantly low levels. Depending on the laboratory methodology, pseudohyponatremia may occur in patients with profoundly lipemic serum.

TOTAL PROTEIN (TP)
NORMAL
5.5 to 7.2 g/dL (dog); 5.4 to 8.9 g/dL (cat)
INTERPRETATION
TP must be evaluated with constituent proteins albumin and globulin. Increased TP (hypoproteinemia) may indicate dehydration (elevated albumin and globulin) or extreme elevations in globulin (chronic inflammation, infection, neoplasia, especially myeloma). Decreased TP may indicate increased protein loss (especially albumin), chronic malassimilation or maldigestion, starvation, or chronic illnesses (tumor cachexia).
TRIGLYCERIDE (TG)

**Normal**

19 to 133 mg/dL (dog); 24 to 206 mg/dL (cat)

**Interpretation**

TG is normally increased in any animal during the postprandial state (with 6 hours after a meal). TG is the cause of gross lipemia when concentrations exceed approximately 500 mg/dL. Increased TG (in the fasted patient) is associated with familial hypertriglyceridemia, a condition most often reported in Miniature Schnauzers (other breeds and mixed breeds may be affected) born in the United States (the condition has not been described in Miniature Schnauzers in Europe or the United Kingdom) and certain lines of mixed-breed cats. There is no clinical significance associated with decreased TG in either the dog or the cat.

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**SPECIAL DIAGNOSTIC TESTS AND TEST PROTOCOLS**

This section includes advanced biochemical laboratory tests not typically included in routine companion animal medicine laboratory profiles. These tests are selected on the basis of abnormal findings revealed during routine physical examination and laboratory profiling. Additional special laboratory tests and test procedures can be found in the organ system-specific sections that follow.

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**Note:** Throughout the Special Diagnostic Tests and Test Protocols section, the following information is provided, where appropriate, for each laboratory test described:

- Test or analyte name (abbreviations or common names)
- Normal (representative reference range value for normal adult dogs and cats)
- Patient preparation (includes any special requirements before sample collection)
- Sample (type of sample and recommended minimum volume to be collected)
- Submit (component of sample to submit for analysis, store, or mail)
- Interpretation (basic interpretation of test results that are outside the reference range)
- Interference (variables that may falsely elevate or decrease test results)
- Protocol (as applicable, accepted procedures for performing the test are outlined)

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**ACETYLCHOLINE (ACH) RECEPTOR ANTIBODY**

See Immunology.

**ADRENOCORTICOTROPIC HORMONE (ACTH), ENDOGENOUS**

See Endocrinology.

**ADRENOCORTICOTROPIC HORMONE (ACTH) STIMULATION TEST**

See Endocrinology.

**ALDOSTERONE**

See Endocrinology.

**AMMONIA (NH₃) (FASTING AMMONIA)**

**Normal**

45 to 120 mcg/dL (dog); 30 to 100 mcg/dL (cat)

**Patient Preparation**

Overnight fast
COLLECT
Whole blood, 2.0 mL minimum, in EDTA (purple-topped tube) or in heparin

SUBMIT
Plasma, 1.0 mL minimum

INTERPRETATION
Decreased levels of ammonia are not considered clinically significant. Elevated ammonia levels support the diagnosis of underlying, significant liver disease. This test generally is considered a liver function test and usually is performed to support a diagnosis of hepatic encephalopathy. Fasting ammonia and ammonia tolerance tests are uncommonly performed today because of sample instability and specimen handling requirements. These tests have largely been replaced by pre- and post-bile acid assay.

INTERFERENCE
Hemolysis; elevated BUN; glucose values greater than 600 mg/dL. NH₃ is unstable if not frozen at – 20° C.

LIMITING FACTORS
Ideally, blood should be collected in a sealed, cold glass collection tube, centrifuged immediately, and plasma analyzed within 20 minutes of collection. Alternatively, plasma can be stored for up to 48 hours if frozen immediately after collection and kept frozen until time of analysis.

AMMONIA TOLERANCE TEST

NORMAL RESTING VALUES
45 to 120 mcg/dL (dog); 30 to 100 mcg/dL (cat)

Note: Minimal change should be detected after oral challenge because clearance is nearly 100% after a single pass through the liver.

PATIENT PREPARATION
Overnight fast

COLLECT
Whole blood, 2.0 mL minimum, in EDTA (purple-topped tube) or in heparin

SUBMIT
Plasma, 1.0 mL minimum, for each prechallenge and postchallenge sample

INTERPRETATION
Elevated ammonia levels support the diagnosis of underlying, significant liver disease. This generally is considered a liver function test and is usually performed to support a diagnosis of hepatic encephalopathy. Fasting ammonia and ammonia tolerance tests are uncommonly performed today because of sample instability and specimen handling requirements. These tests have largely been replaced by pre- and post-bile acid assay.

INTERFERENCE
Hemolysis; elevated BUN; glucose values greater than 600 mg/dL. NH₃ is unstable if not frozen at – 20° C.

LIMITING FACTORS
Ideally, blood should be collected in a sealed, cold glass collection tube, centrifuged immediately, and plasma analyzed within 20 minutes of collection. Alternatively, plasma can be stored for up to 48 hours if frozen immediately after collection and kept frozen until time of analysis.
Protocol
Two plasma samples are required. The first is a baseline sample. The second sample is collected 30 to 45 minutes after administration of ammonium chloride (NH₄Cl) at 100 mg/kg body weight (not to exceed 3 grams) as an oral 5% solution in approximately 20 to 50 mL of saline. NH₄Cl is also available as a powder that can be administered orally, at the same dose, which lowers the risk of vomiting or aspiration.

Antinuclear Antibody (ANA)
See Immunology.

Bile Acids
Normal (Dog and Cat)
Prefeeding, ≤7 µmol/L; postfeeding, ≤15 µmol/L.

Patient Preparation
12-hour or overnight fast before collection of the prefeeding sample

Collect
Whole blood, 2.0 mL minimum, in RTT for each sample collected

Submit
Serum, 1.0 mL minimum, for each sample submitted

Interpretation
Bile acids are indicated for the assessment of hepatobiliary disease in nonicteric patients. There is no value in performing this test in patients that are icteric. Hepatobiliary disease (e.g., portosystemic shunt) is supported with either a prefeeding sample of more than 7 µmol/L or a postfeeding sample of more than 15 µmol/L. Note: Reference range values may vary among different laboratories.

Interference
Lipemia; icterus; hemolysis. Results in patients that vomit the meal before collection of the 2-hour postfeeding sample cannot be expected to be reliable. Individual variations in gastric emptying and absorption can result in discordant results (e.g., the prefeeding sample is higher than the postfeeding sample). Such results are not reliable, and the test should be repeated.

Protocol
1. The prefeeding (or fasting) blood sample is collected after a 12-hour fast. Label the tube accordingly.
2. Feed a relatively high-fat meal (to stimulate gallbladder contraction). A protein-restricted diet with corn oil added is appropriate for those patients with protein intolerance and signs of hepatic encephalopathy.
3. Two hours after consumption of the meal, collect the postfeeding sample. Label the tube accordingly.

Blood Gases (Arterial and Venous)
The values represented in the following table are expected from patients breathing room air.
NORMAL

<table>
<thead>
<tr>
<th>Value</th>
<th>Arterial</th>
<th></th>
<th>Venous</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Dog</td>
<td>Cat</td>
<td>Dog</td>
<td>Cat</td>
</tr>
<tr>
<td>pH</td>
<td>7.36-7.44</td>
<td>7.36-7.44</td>
<td>7.34-7.46</td>
<td>7.33-7.41</td>
</tr>
<tr>
<td>Pco₂</td>
<td>36-44</td>
<td>28-32</td>
<td>32-49</td>
<td>34-38</td>
</tr>
<tr>
<td>Po₂</td>
<td>90-100</td>
<td>90-100</td>
<td>24-48</td>
<td>35-45</td>
</tr>
<tr>
<td>Tco₂</td>
<td>25-27</td>
<td>21-23</td>
<td>21-31</td>
<td>27-31</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>24-26</td>
<td>20-22</td>
<td>20-29</td>
<td>22-24</td>
</tr>
</tbody>
</table>

PATIENT PREPARATION

Patients breathing 100% oxygen at the time of sample collection are expected to have different results from those of patients that are breathing room air during sample collection. Note the conditions under which the sample was collected.

COLLECT

Whole blood, either arterial or venous, depending on the assessment required.

SUBMIT

Sample cannot be stored. Immediate testing is required for reliable results to be obtained.

INTERPRETATION

Tco₂ is synonymous with HCO₃⁻ in patients breathing room air. The overall interpretation of venous and/or arterial blood gas results will vary considerably depending on the patient’s health status. Several variations in test results are possible. The clinician should consult appropriate references to interpret results from individual patients (see Section 1).

INTERFERENCE

The test should be performed immediately after collection of the sample. Delays could cause significant abnormalities in actual results. Exposure of the sample to room air (bubbles within the sample) may cause Pco₂ to decrease, whereas pH and Po₂ may increase.

BLOOD TYPING

See page 593 in this section.

BODY FLUIDS (SUBMITTED FOR CHEMISTRY ANALYSIS)

NORMAL

Not applicable.

PATIENT PREPARATION

When feasible, the skin over the site selected for centesis should be shaved and surgically prepared before sample collection is attempted, to avoid contamination of either the sample or the body cavity from which the sample is collected.

COLLECT

1 to 2 mL, minimum, by direct centesis of body cavity or fluid-filled compartment.

SUBMIT

Centrifugation of whole blood contamination is indicated to remove particulate material (e.g., blood cells, cellular debris) when prompt evaluation of a specimen is not possible.
INTERPRETATION
Any biochemical analyte determined in serum or plasma may be assayed in body fluid—for example, amylase, lipase (pancreatitis), urea nitrogen, creatinine (ruptured bladder), glucose, lactate.

INTERFERENCE
Blood and blood components, bilirubin, bile, and urine may significantly interfere with test results. Centrifugation of the sample (blood contamination) may be necessary before any biochemistry test is performed.

BRAIN NATRIURETIC PEPTIDE (BNP, proBNP, NT proBNP)

NORMAL
<800 pmol/L (dog); <50 pmol/L (cat)
Recommendations for the normal reference ranges of proBNP are subject to change as new information is published on this assay. Refer to the reference laboratory for the latest recommendations on interpreting test results for the dog and cat.

PATIENT PREPARATION
None

COLLECT
Anticoagulated whole blood, 2.0 to 3.0 mL; use a lavender-topped tube. The sample must be centrifuged and plasma separated within 30 minutes after collection. Plasma sample should be inverted several times after separation.

SUBMIT
1.0 mL plasma; must be submitted in a special transport tube provided by the laboratory (IDEXX Laboratories, Westbrook, Maine).

INTERPRETATION
Current studies do show, in both dogs and cats, that elevated levels of proBNP correlate well with existing heart disease, particularly cardiomyopathy. Test results must be considered with information derived from a clinical evaluation and cardiac examination, including electrocardiography and echocardiography. Ongoing studies are evaluating the value of proBNP levels in monitoring individual response to treatment for various heart diseases.

INTERFERENCE
Not stipulated

CALCIUM, IONIZED (iCa)

NORMAL
1.12 to 1.42 mmol/L (dog); 1.12 to 1.42 mmol/L (cat)

PATIENT PREPARATION
None

COLLECT
Whole blood, 2.0 mL

SUBMIT
Serum, 1.0 mL
INTERPRETATION
Results reflect the concentration of the biologically active, ionized fraction of calcium without the influence of plasma proteins (e.g., albumin).

INTERFERENCE
The reported values of iCa can vary with patient’s blood pH; iCa decreases as pH increases.

CEREBROSPINAL FLUID (CSF)

<table>
<thead>
<tr>
<th></th>
<th>Dog</th>
<th>Cat</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Normal</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WBCs (×10³/L)</td>
<td>≤3</td>
<td>≤2</td>
</tr>
<tr>
<td>RBCs (×10⁶/L)</td>
<td>≤30</td>
<td>≤30</td>
</tr>
<tr>
<td>Protein (mg/dL)</td>
<td>≤33</td>
<td>≤36</td>
</tr>
<tr>
<td><strong>Cytology (%)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Monocytes</td>
<td>87</td>
<td>69-100</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>4</td>
<td>0-27</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>3</td>
<td>0-9</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Macrophages</td>
<td>6</td>
<td>0-3</td>
</tr>
</tbody>
</table>

PATIENT PREPARATION
General anesthesia is required. Specific training and/or experience is strongly recommended before collection of CSF from the cisterna magna (between the head and C1). Fatalities can result from improper technique.

COLLECT
Usually, two 0.5- to 1.0-mL samples are collected in RTTs (no additives).

SUBMIT
Samples collected

INTERPRETATION
If one sample contains excessive numbers of neutrophils, the second sample is submitted for culture and sensitivity; treatment recommendations should include use of an antibiotic (preferably intravenous) that will penetrate the blood-brain barrier.

INTERFERENCE
Blood contamination is the most common interfering factor. An RBC count greater than 30 × 10⁶/L is consistent with peripheral blood contamination. Immediate analysis is recommended. It is not recommended to submit CSF via mail for assessment.

PROTOCOL
Proper patient preparation and collection technique are critical (see Section 4).

COBALAMIN (VITAMIN B₁₂)

NORMAL
Results vary considerably among laboratories; consult individual laboratory.

PATIENT PREPARATION
Fasted
**COLLECT**
Whole blood, 2.0 mL minimum (RTT)

**SUBMIT**
Serum, 1.0 mL

**INTERPRETATION**
Test usually is performed with folate and trypsin-like immunoreactivity (TLI). It is used in the assessment of chronic small bowel diarrhea with associated weight loss. Significantly decreased cobalamin levels support the need to measure TLI (exocrine insufficiency), support a finding of mucosal disease, and may be indicative (in cats) of hepatic disease (hepatic lipidosis).

**INTERFERENCE**
Hemolysis; lipemia

**ETHYLENE GLYCOL**

**NORMAL**
Negative. “Trace” amounts may be detected in normal patients.

**PATIENT PREPARATION**
None

**COLLECT**
Urine (within 3 to 6 hours of ingestion), whole blood, or serum. Collect volume of sample in accordance with manufacturer’s directions.

**SUBMIT**
Not applicable. Procedure is an in-hospital test kit for emergency use.

**INTERPRETATION**
Values greater than 50 mg/dL indicate exposure to ethylene glycol. Immediate treatment is indicated. Supporting laboratory documentation of ethylene glycol exposure is based on results of serum osmolality (increased), demonstration of an osmolar gap, and anion gap (increased). Blood gas analysis may reveal severe metabolic acidosis. In addition, examination of urine under polarizing light microscopy may detect calcium oxalate crystals if performed within 3 to 6 hours after ingestion.

**INTERFERENCE**
Some drugs (pentobarbital and diazepam) will cause false elevations of ethylene glycol in the test kit results but will not induce calcium oxalate crystalluria.

**PROTOCOL**
Follow manufacturer’s recommendations for use of the test kit.

**FECAL FAT, 72-HOUR QUANTITATIVE COLLECTION**
No one wants to either collect or analyze a pound of feces. Better tests are available. See the discussion of TLI in this section.

*Ethylene Glycol Test Kit, PRN Pharmacal, Pensacola, Florida.*
Fecal Occult Blood

Normal
Negative for blood (dog and cat)

Patient Preparation
Discontinue all red meat and orally administered drugs at least 3 days before collection of the sample for analysis (see Interference).

Collect
About 1 g of fresh feces

Submit
One gram of fresh feces is sufficient. Sample may be stored for up to 4 days at 2° to 8° C.

Interpretation
Guaiac test methodology is used to detect the presence of occult blood. Animals with two positive consecutive test results 48 hours apart are likely to have a primary lesion in the GI tract. Benign ulcerative lesion and neoplasia are the two principal rule-outs.

Interference
Thrombocytopenia; known platelet disorder; recent aspirin administration; corticosteroid therapy (oral or parenteral); oral iron supplementation; diet containing red meat.

Folate

Normal
Results vary considerably among laboratories; consult individual laboratory.

Patient Preparation
Overnight fast

Collect
Whole blood, 4.0 mL (RTT); separate serum from cells immediately.

Submit
Serum, 2.0 mL

Interpretation
Test usually is performed in conjunction with TLI and serum cobalamin (vitamin B₁₂). Decreased levels of folate support the diagnosis of small intestinal mucosal disease. Increased levels of folate support exocrine pancreatic insufficiency and/or small intestinal bacterial overgrowth.

Interference
Hemolysis; lipemia

Fructosamine

Normal
225 to 375 µmol/L (dog and cat)
Consult individual laboratory because test results may vary.

Patient Preparation
None
**COLLECT**
Whole blood, 2.0 mL (RTT)

**SUBMIT**
Serum, 1.0 mL; sample must be frozen and shipped on cold packs for overnight delivery.

**INTERPRETATION**
This is a single-sample test representing mean blood glucose over the prior 1 to 3 weeks. Increased fructosamine indicates poor glycemic control (hyperglycemia); declining fructosamine indicates improved or adequate glycemic control. Values greater than 500 µmol/L suggest inadequate glycemic control over the previous 1 to 3 weeks. Values less than the lowest reference range value suggest that the patient sustained significant periods of hypoglycemia over the previous 1 to 3 weeks. Values less than 400 µmol/L and clinical signs of polyuria and polydipsia (PU/PD) and polyphagia are suggestive of a Somogyi phenomenon. Fructosamine levels should not be used to make specific adjustments in daily insulin therapy.

**INTERFERENCE**
The assay is a colorimetric procedure; therefore, significant hemolysis or icterus could affect results. Hypoproteinemia and/or hypoalbuminemia will cause falsely low values. Hyperlipidemia and azotemia may also alter results similarly.

**GLYCOSYLATED HEMOGLOBIN (GLYCATED HEMOGLOBIN; GLY Hb)**

**NORMAL**
1.7% to 4.9% (dog and cat)
Consult individual laboratory because test results may vary.

**PATIENT PREPARATION**
None

**COLLECT**
Whole blood, 2.0 mL in EDTA (purple-topped tube)

**SUBMIT**
Plasma, 1.0 mL; separate plasma and refrigerate until assayed.

**INTERPRETATION**
This is a single-sample test representing mean blood glucose over the lifespan of RBCs (approximately 3 to 4 months). This test is used less in veterinary medicine than the fructosamine assay. In dogs, values consistently ranging from 4% to 6% are associated with adequate glycemic control and owner satisfaction.

**INTERFERENCE**
Storage at room temperature and for longer than 7 days will decrease values; patients with Hct less than 35% may have lower than expected values. Note: Laboratories must use an assay that has been validated for dogs and for cats. Human assays performed on animal plasma may not be valid.

**IRON**

**NORMAL**
Consult individual laboratory because test results may vary.

**PATIENT PREPARATION**
None
COLLECT
Whole blood, 2.0 mL (RTT)

SUBMIT
Serum, 1.0 mL

INTERPRETATION
Results should be interpreted with total iron-binding capacity (TIBC) and ferritin. Decreased values reflect chronic, not acute, blood loss (e.g., hookworms, intestinal ulceration, bleeding from neoplasia). In cases of iron deficiency, expect TIBC to be normal or high, whereas serum ferritin will be low. Patients with anemia associated with chronic inflammatory disease are expected to have normal to low TIBC, whereas serum ferritin will be normal to high.

INTERFERENCE
Hemolysis; lipemia

LACTIC ACID (LACTATE)

NORMAL
2 to 13 mg/dL (0.22 to 1.44 mmol/L) (dog); results not reported for cats

PATIENT PREPARATION
Avoid venous stasis when collecting sample. Clean venipuncture and rapid draw of sample are important.

COLLECT
Whole blood, 2.0 mL, in lithium heparin plasma or in iodoacetate tubes. Some laboratories will accept samples collected in fluoride tubes.

SUBMIT
Plasma, which should be rapidly separated from blood. If this is not possible, the sample may be refrigerated immediately at 4° C, but only for 2 hours, at which time the plasma must be separated from blood.

INTERPRETATION
Resting values greater than 6.0 mmol/L indicate severe acidosis and a poor prognosis. Test is also used to assess metabolic myopathies, especially in Labrador Retrievers.

INTERFERENCE
Aspirin, phenobarbital, and epinephrine may alter lactate values. Also, allowing the sample to sit at room temperature will result in an increased level of lactate.

PROTOCOL
To diagnose metabolic myopathy in Labrador Retrievers, two samples are recommended. The first blood sample is collected at rest. A second sample is collected after 10 to 15 minutes of brisk walking or running.

LEAD, BLOOD

NORMAL
Results vary considerably among laboratories; consult individual laboratory. Usually, values of less than 0.05 ppm in whole blood (or less than 3 ppm in liver or kidney) are within the range of normal.

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**Patient Preparation**
None

**Collect**
Whole blood, 2.0 mL, in EDTA (lavender-topped tube) or heparin

**Submit**
Entire sample

**Interpretation**
Refer to the individual laboratory for specific interpretation of the values reported. Values greater than 0.3 ppm suggest exposure. Values greater than 0.4 ppm are generally considered diagnostic of toxicosis.

**Interference**
Incorrect tube used for collection or storage of whole blood

**Lipoprotein Electrophoresis**

**Normal**
Normal values have not been established for the dog and cat

**Patient Preparation**
12-hour fast

**Collect**
Whole blood, 1.0 mL (RTT)

**Submit**
Serum, 0.5 mL

**Interpretation**
Test consists of electrophoretic separation of various lipoprotein categories in serum. It may qualitatively identify various categories of lipoproteins, including chylomicrons, very-low-density lipoproteins (VLDLs), low-density lipoproteins (LDLs), and high-density lipoproteins (HDLs). Standards have not been established for the dog or cat.

**Interference**
Lipemia is not an interfering factor because electrophoresis will separate various lipid fractions.

**Magnesium (MG)**

**Normal**
1.5 to 2.5 mg/dL (dog and cat)

**Patient Preparation**
None

**Collect**
Whole blood, 2.0 mL, in RTT

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SUBMIT
Serum, 1.0 mL

INTERPRETATION
Increased Mg may reflect renal failure or insufficiency. Decreased Mg is observed in many GI disorders (malabsorption, pancreatitis, chronic diarrhea), renal disease (glomerulonephritis, diuresis, tubular necrosis), and multiple endocrine diseases, as well as with sepsis, blood transfusion, and parenteral nutrition.

INTERFERENCE
Mg-containing drugs (oral antacids and laxatives) will falsely elevate test results. Some intravenous fluids contain Mg, which also may falsely elevate test results. Falsely decreased values may result from diuretic therapy or intravenous fluid therapy–induced diuresis.

OSMOLALITY, ESTIMATED (SERUM)
NORMAL
290 to 310 mOsm/kg (dog); 308 to 335 mOsm/kg (cat)

PATIENT PREPARATION
None

COLLECT
Whole venous blood, 2.0 mL, in an RTT or SST

SUBMIT
Serum, 1.0 mL

INTERPRETATION
Osmolality of extracellular fluid (ECF) is determined predominantly by electrolytes, especially sodium, and small molecules (glucose and urea) and is reflective of fluid shifts between the vascular space and the interstitium. Increased ECF osmolality (>350 mOsm/L), or hyperosmolality, is likely to be associated with clinical signs (especially neurologic) because of the shift of water from the interstitial space into the vascular space.

Note: Direct laboratory measurement of serum osmolality can be performed but is expensive. Serum osmolality is usually calculated according to the following formula:

\[
\text{mOsm/kg} = 1.86 (\text{Na}^+ + \text{K}^+) + (\text{glucose} \div 18) + (\text{BUN} \div 2.8) + 9
\]

PANCREATIC LIPASE (PL; FORMERLY PLI) (ALSO CANINE: cPL AND FELINE: fPL)
NORMAL
2.2 to 102.1 mcg/L (dog—cPL); 2.0 to 6.8 mcg/L (cat—fPL)

PATIENT PREPARATION
Fasted for 12 hours before collection of blood

COLLECT
Whole blood, 3.0 mL minimum in RTT or SST

SUBMIT
Serum, 1.0 mL minimum. Immediately separate serum from clot. Ship serum only. Do not ship whole blood.
INTERPRETATION
PL is species specific; samples must be labeled “DOG” (cPL) or “CAT” (fPL). Elevated values are considered to be highly specific for a diagnosis of pancreatitis in both the cat and the dog.

INTERFERENCE
Anticoagulant, hemolysis; moderate or greater lipemia

PROTOCOL
Serum should be separated immediately after clot formation and retraction

PROTEIN ELECTROPHORESIS (SERUM)

<table>
<thead>
<tr>
<th>Value</th>
<th>Dog</th>
<th>Cat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total protein (g/dL)</td>
<td>6.0-7.6</td>
<td>7.3-7.8</td>
</tr>
<tr>
<td>Albumin (g/dL)</td>
<td>2.7-3.7</td>
<td>2.8-4.2</td>
</tr>
<tr>
<td>α₁-Globulin (g/dL)</td>
<td>0.25-0.60</td>
<td>0.3-0.65</td>
</tr>
<tr>
<td>α₂-Globulin (g/dL)</td>
<td>0.72-1.40</td>
<td>0.40-0.68</td>
</tr>
<tr>
<td>β₁-Globulin (g/dL)</td>
<td>0.63-0.89</td>
<td>0.77-1.25</td>
</tr>
<tr>
<td>β₂-Globulin (g/dL)</td>
<td>0.60-1.0</td>
<td>0.35-0.50</td>
</tr>
<tr>
<td>γ₁-Globulin (g/dL)</td>
<td>0.50-0.83</td>
<td>1.39-2.22</td>
</tr>
<tr>
<td>A:G ratio</td>
<td>0.8-1.0</td>
<td>0.63-1.15</td>
</tr>
</tbody>
</table>

PATIENT PREPARATION
Fasted for 12 hours (overnight) to prevent postprandial lipemia

COLLECT
Whole blood, 2.0 mL (RTT)

SUBMIT
Serum, 1.0 mL. Most laboratories will accept a volume of serum from 0.5 to 1.0 mL.

INTERPRETATION
Multiple interpretations are possible, depending on the patient’s condition. Test usually is performed to assess degree of loss of albumin or increases in one or more globulin fractions (e.g., hypergammaglobulinemia associated with feline infectious peritonitis (FIP), canine ehrlichiosis, multiple myeloma). The test is not used to confirm a diagnosis.

Most clinical assessments are made from the shape of the curve in a densitometer tracing of the electrophoresis rather than specific numbers. When requesting serum protein electrophoresis, it is important to request a copy of the curve as well as the quantitated results for each protein fraction.

INTERFERENCE
Lipemia; hemolysis

TRYPSIN-LIKE IMMUNOREACTIVITY, CANINE (CANINE TLI)

<table>
<thead>
<tr>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.0 to 35.0 mcg/L</td>
</tr>
</tbody>
</table>

PATIENT PREPARATION
Fasted
COLLECT
Whole blood, 2.0 mL in RTT

SUBMIT
Serum, 1.0 mL. Separate serum from clot. Ship serum only. Do not ship whole blood.

INTERPRETATION
TLI is species specific; samples must be labeled “DOG.” It is a sensitive and specific test for the diagnosis of exocrine pancreatic insufficiency in dogs and cats. Values less than 2.5 mcg/L, in the presence of clinical signs, support the diagnosis. Values greater than 50 mcg/L have been used to diagnose pancreatitis in dogs. However, TLI has been replaced by the canine pancreatic lipase immunoreactivity (cPLI) assay to diagnose pancreatitis in dogs.

INTERFERENCE
Hemolysis; moderate or greater lipemia

TRYSIN-LIKE IMMUNOREACTIVITY, FELINE (FELINE TLI)
NORMAL
12 to 82 mcg/L

PATIENT PREPARATION
Fasted

COLLECT
Whole blood, 2.0 mL (RTT)

SUBMIT
Serum, 1.0 mL. Separate serum from clot. Ship serum only. Do not ship whole blood.

INTERPRETATION
TLI is species specific; samples must be labeled “CAT.” This is a sensitive and specific test for the diagnosis of exocrine pancreatic insufficiency in dogs and cats. Values less than 2.5 mcg/L, in the presence of clinical signs support the diagnosis. Values greater than 100 mcg/L have been used to diagnose pancreatitis in cats. However, TLI has been replaced by the feline pancreatic lipase immunoreactivity (fPLI) assay to diagnose pancreatitis in cats.

INTERFERENCE
Hemolysis; moderate or greater lipemia

Serum for PLI and TLI assays may be submitted to the Gastrointestinal Laboratory Department of Small Animal Medicine and Surgery, Texas A&M University, 4474 TAMU, College Station, TX 77843-4474.

HEMOSTASIS AND COAGULATION

Tests of hemostasis are directed at determining platelet numbers and function, activation and abnormalities of the intrinsic and extrinsic clotting cascade, and quantitation of breakdown products of thrombosis and fibrinolysis. Obtain blood samples for evaluation of coagulation abnormalities by careful venipuncture, and insert samples into plastic or silicone-coated glass syringes.

Because tissue thromboplastin can activate the clotting cascade, some authors advocate using two syringes and two needles to obtain blood for coagulation tests. First, carefully insert the needle into the vein and withdraw 1 mL of blood. Leave the needle in the vessel, and remove the first syringe. Attach a second syringe and obtain the appropriate volume
of blood; then remove the needle from the vessel. Rapidly replace the needle on the second syringe with a fresh needle and then inject the blood sample into the appropriate tubes for later analyses.

Platelet tests should be performed on fresh samples within 2 hours of collection. Plasma samples can be spun down and frozen at −20° C for several days, and at −40° C for several months to a year for later analyses.

**INITIAL IN-OFFICE SCREENING TESTS**

The initial in-office screening tests for coagulation defects include Hct, peripheral blood smear, activated coagulation test (activated clotting time [ACT]) or activated partial thromboplastin time (APTT), prothrombin time (PT), and, if indicated, buccal mucosal bleeding time (BMBT) assay.

**Hematocrit**

The patient’s Hct and total protein should be evaluated to determine whether anemia is present. The color of the plasma in the spun-down microhematocrit tube can aid in making a diagnosis if intravascular hemolysis (red) or icterus (yellow) is present. The buffy coat from a microhematocrit tube can be evaluated microscopically for the presence of microfilaria in heartworm disease or mast cells in systemic mastocytosis.

**Peripheral Blood Smear**

The peripheral blood smear should be evaluated for RBC morphology, RBC fragments (schizocytes), platelet count, large platelets, WBC count and morphology, and blood parasites.

**Platelet Count**

One of the most simple cage-side tests when determining the cause of a coagulopathy is the platelet count. To perform this test:

1. Obtain an anticoagulated sample of peripheral blood (trisodium citrate or sodium oxalate are the anticoagulants of choice for platelet and coagulation testing), and make a stained blood smear.
2. Scan the slide, including the peripheral edge, for platelets and platelet clumps. If platelet clumps are present, the platelet estimate cannot be accurately measured; also, it is unlikely that thrombocytopenia is the cause of the patient’s hemorrhage.
3. If no platelet clumps are present in the feathered edge of the blood smear, scan multiple areas of the slide on 100× (oil) magnification. Count the number of platelets per high-power field (hpf) and then multiply the value by 15,000 to arrive at an approximate estimation of platelet number.

Hemorrhage secondary to thrombocytopenia occurs when platelet numbers decrease to less than 40,000/µL (less than three platelets per high-power field). If there are signs of superficial hemorrhage and more than five platelets per high-power field, a thrombocytopathia (platelet function problem) such as von Willebrand disease, disseminated intravascular coagulation (DIC), or aspirin-induced coagulopathy may be present.

**Activated Coagulation (Clotting) Time (ACT)**

ACT is a measure of the function of the intrinsic and common coagulation pathway (factors II, V, VIII, IX, X, XI, and XII). The ACT can be used reliably to screen for disorders of secondary hemostasis. Severe thrombocytopenia (<10,000 to 20,000 platelets/µL) and decreased fibrinogen, in addition to decreases in activated clotting factors listed previously, can cause prolongation in the ACT. An ACT tube contains diatomaceous earth that stimulates blood clotting on contact.

To measure the ACT:

1. Warm the ACT tube to 37° C in a heating block or water bath.
2. Use a 3-mL syringe without any anticoagulant to obtain 3.0 mL of blood. The venipuncture should be atraumatic. Because tissue factor stimulates the clotting cascade, quickly change the needle and push 2.0 mL of the blood sample into the ACT tube.
inverting the tube several times to mix the contents, and then place the tube in the water bath or heat source. Start counting the time at the moment you inject the blood into the tube. (The remaining blood can be used to fill microhematocrit tubes and make peripheral blood smears.)

3. To check the tube for clots, quickly invert the tube and then return it to the heat source at 60 seconds and then every 5 seconds thereafter. Record the time that the first sign of a clot (gel) is observed.

Normal ACT is 90 to 120 seconds for dogs and 80 to 100 seconds for cats.

APTT is another, more sensitive test to detect defects in the intrinsic clotting cascade. It is more sensitive than the ACT in that it will become prolonged earlier than the ACT. Point-of-care coagulation analyzers (SCA-2000, Symbiotics, San Diego, California) are available that require less blood than an ACT and therefore may be the preferred test.

Prothrombin Time (PT)
PT is a test to determine abnormalities in the extrinsic (factor VII) coagulation pathway. Because factor VII is the most labile clotting factor and has the shortest half-life, PT will become prolonged before any changes in ACT or APTT (intrinsic pathway) occur. The prothrombin–complex clotting factors are II, VII, and X; these factors interact with factor V and fibrinogen in the presence of tissue thromboplastin and calcium chloride.

Buccal Mucosal Bleeding Time (BMBT)
The BMBT is the time required for platelets to become activated and interact with damaged vascular endothelium to form a primary platelet plug. It is a test of primary hemostasis. The BMBT becomes prolonged with thrombocytopenia (<100,000/µL) and platelet dysfunction syndromes such as von Willebrand disease. The BMBT is usually performed without any sedation in dogs and with ketamine in cats.

To measure the BMBT:
1. When obtaining the BMBT in dogs, place a loose tie of gauze around the dog's muzzle to lift the lip so that the buccal mucosa is exposed and the veins are slightly engorged. It is important to not tie the gauze too tightly, as vasoconstriction can artifactually change test results.

2. Use a BMBT template (Simplate R) to make two small nick incisions in the buccal mucosa. Gently wick the blood away from the site with a piece of filter paper (if you don't have filter paper, a coffee filter works well). Allow the blood to wick into the filter paper without touching the incisions or the clot.

3. Note the time from making the initial incision to the time that hemorrhage stops (i.e., a platelet plug has formed). Normal BMBT is less than 3 minutes in dogs and cats.

If the BMBT is prolonged, von Willebrand disease, nonsteroidal antiinflammatory drug (NSAID) influence, congenital thrombopathies (Bassett Hounds and Otterhounds), and systemic illness (azotemia, hepatic failure, malignancy) should be ruled out. If the BMBT is normal in the face of a normal platelet count and clinical bleeding, tests of the coagulation cascade (APTT, PT, ACT) should be considered.

Ancillary Tests of Hemostasis

Thrombin Time
The thrombin time is a measure of the amount of functional fibrinogen in plasma. The test is used in the diagnosis of DIC when fibrinogen levels are low. Fibrinogen levels may also be normal in DIC, but thrombin time will still be altered because of in vivo fibrinolysis. This test is now rarely used, because more sensitive and specific tests such as D-dimer concentration are available for the diagnosis of DIC.

Fibrinogen
Fibrinogen levels are used in the detection of DIC. In DIC, fibrinogen levels can decrease as a result of the activation of thrombin and fibrin formation and the activation of plasmin, which causes degradation of fibrin and fibrinogen. Fibrinogen levels can be decreased,
normal, or increased in cases of chronic DIC owing to compensatory overproduction. Because of the variability in fibrinogen levels, this test alone is not conclusive to make a diagnosis of DIC.

**Fibrinogen Degradation Products (FDPs)**

Fibrinogen degradation products (FDPs; also called fibrin split products [FSPs]) are formed when the enzyme plasmin acts on fibrin monomers, cross-linked fibrin, and fibrinogen. Because fibrinogen can increase during periods of inflammation without DIC, the presence of FDPs alone does not allow a diagnosis of DIC. FDPs are cleared by the hepatic reticuloendothelial system. In cases of hepatic insufficiency or hepatic failure, FDPs can be elevated without concurrent DIC.

**D-Dimers**

D-dimers are used in the diagnosis of DIC. D-dimers are released as a result of the breakdown of cross-linked fibrin by plasmin. Because D-dimers occur as a result of a stable fibrin clot, elevated levels are more sensitive and specific for a diagnosis of DIC.

**The PIVKA Test**

The PIVKA (proteins induced by vitamin K absence or antagonism) test is most useful in diagnosing vitamin K deficiencies. Moderate deficiencies in Vitamin K–dependent coagulation factors (II, VII, IX, and X) will cause abnormal PIVKA test results. The PIVKA test result becomes prolonged 12 to 24 hours after the PT becomes prolonged.

**Saline Agglutination**

The saline agglutination test is simple to perform in house and aids in the diagnosis of immune-mediated hemolytic anemia (IMHA). To perform a saline agglutination test:

1. Place one drop of 0.9% saline on a microscope slide. Mix the drop of saline with one drop of the patient’s anticoagulated blood and observe for the presence of agglutination under the microscope.
2. If agglutination is present, mix a second drop of saline with the blood-saline mixture on the slide and review under the microscope a second time.

If the “agglutination” disperses, it is likely caused by rouleaux secondary to inflammation. If the agglutination remains, autoagglutination of RBCs is occurring owing to interaction with antibodies directed against glycoprotein moieties on the surface of the RBC membranes.

**Note:** Management of patients with a confirmed coagulopathy involves correcting any underlying cause, replenishing oxygen-carrying capacity in the form of RBCs or purified hemoglobin, replacing clotting factors and antithrombin in the form of fresh frozen plasma, and maintaining end-organ perfusion. The management of specific conditions and coagulopathies is listed under their subheadings. A more thorough approach to transfusion management is described in Section 1.

**Submission of Samples for Coagulation Testing**

1. Draw blood sample into a BTT that contains sodium citrate. Fill the BTT to at least 75%, but preferably 90% or more, because results will be affected by excess citrate anticoagulant.
2. Centrifugation and separation of plasma from cells is strongly recommended if transportation to the laboratory may require more than 12 hours.
3. Use a plastic pipette or small syringe to transfer the plasma to a clean plastic tube. Cap the tube and keep cold or freeze at −20° C or lower. Freezing the plasma is not necessary unless testing will be delayed for more than 24 hours, but it should always stay cold. Repeated freezing and thawing of plasma denatures coagulation proteins.
4. If samples will be mailed, ship overnight with frozen cold packs.

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ACTIVATED COAGULATION (OR CLOTTING) TIME (ACT)

NORMAL
90 to 120 seconds (dog); 80 to 100 seconds (cat)

PATIENT PREPARATION
Direct penetration of the vein is important.

COLLECT
Venous blood in an ACT Vacutainer tube. Fill to maximum allowed by vacuum.

SUBMIT
Blood sample in collection tube per protocol

INTERPRETATION
ACT is a convenient in-hospital screening test that evaluates both the intrinsic and common coagulation pathways. Prolonged clotting time implies coagulation factor deficiency. A specific coagulation factor deficiency must be less than 5% to increase the ACT. Note: hemophiliac patients may have factor VIII or IX activity at only 40% to 60% of normal and yet would have a normal ACT (and a normal APTT).

INTERFERENCE
The presence of tissue thromboplastin in sample (e.g., failing to obtain blood from a “clean” venipuncture) will activate the extrinsic pathway.

PROTOCOL
A two-tube technique is recommended to eliminate any chance of tissue thromboplastin contaminating the sample. Fill two tubes from the same draw. Use the second tube only. Prewarm the tubes in a water bath or heating block (37° C). Place the filled sample tubes in the water bath or heating block and begin timing. Incubate the sample in the collection tube for 60 seconds for dogs, and 45 seconds for cats. Invert sample every 5 seconds to assess for evidence of clot formation. Stop procedure at first sign of clot formation.

ACTIVATED PARTIAL THROMBOPLASTIN TIME (APTT)

NORMAL
8.6 to 12.9 seconds (dog); 13.7 to 30.2 seconds (cat)

PATIENT PREPARATION
None (atraumatic venipuncture is recommended)

COLLECT
Venous blood in citrate (BTT); fill Vacutainer tube to the maximum allowed by the vacuum.

SUBMIT
Citrated plasma only (plasma must be separated from cells) in RTT

INTERPRETATION
APTT is the most sensitive and specific test of coagulation factor activity. Prolonged APTT implies anticoagulant therapy (heparin) or specific coagulation factor deficiency.

INTERFERENCE
Clotted sample, failure to use citrate as the anticoagulant; incorrect ratio of citrate to whole blood.
PROTOCOL
On collection of blood, invert tube several times to ensure adequate mixing of sample and anticoagulant. Centrifuge immediately. Transfer plasma to RTT and label as “Citrated Plasma.”

ANTIPLATELET ANTIBODY
See Immunology.

BLOOD TYPING, FELINE
NORMAL
Results reported as positive or negative for blood type A, B, or AB

PATIENT PREPARATION
None

COLLECT
Venous blood, 1.0 mL, in EDTA (lavender-topped tube)

SUBMIT
Entire sample

INTERPRETATION
The majority of blood donors should be type A. However, blood typing and cross-matching blood before transfusion in cats is highly recommended, because some type B cats are present in the United States. It has been reported that as little as 1.0 mL of type A blood transfused into a type B cat is fatal.

BLOOD TYPING FOR COMPLETE DOG ERYTHROCYTE ANTIGEN (DEA)
NORMAL
Results are reported as positive or negative for DEA 1.1, DEA 1.2, DEA 3, DEA 4, DEA 5, and DEA 7.

PATIENT PREPARATION
None

COLLECT
Venous blood, 1.0 mL, in EDTA (lavender-topped tube)

SUBMIT
Entire sample

INTERPRETATION
Universal or A-blood donors should be negative for DEA 1.1, DEA 1.2, and DEA 1.7.

BUCCAL MUCOSAL BLEEDING TIME (BMBT)
NORMAL
2.6 ± 0.48 minutes (dog); results not reported in cats.

PATIENT PREPARATION
None
COLLECT
Not applicable

SUBMIT
Not applicable. This is an in-hospital screening test for platelet function.

INTERPRETATION
BMBT is a sensitive and specific test of platelet function. Prolonged BMBT is expected in patients with von Willebrand disease and uremia. Test is not generally recommended for thrombocytopenic patients.

INTERFERENCE
Improper technique and patients with thrombocytopenia

PROTOCOL
The test entails a standardized cut into the buccal mucosa with subsequent “capture” of blood onto filter paper until bleeding ceases.

CLOT RETRACTION TEST
Not generally recommended
  Because of the insensitivity of this test, the clot retraction test is not recommended for the assessment of patients with suspected disorders of hemostasis.

COAGULATION FACTOR ACTIVITY (FACTOR ASSAY)
The following inherited coagulation factor deficiencies have been reported in dogs and cats:
  Hemophilia A (factor VIII deficiency)—the most common factor deficiency
  Hemophilia B (factor IX deficiency)
  Factor XII deficiency (Hageman trait)—of minor significance in affected cats
  Vitamin K–dependent factor deficiency—occurs in Devon Rex cats, with severe bleeding
  Other, rare deficiencies have been reported.

DIAGNOSIS OF COAGULATION FACTOR DEFICIENCY
Coagulation factor deficiency usually is suspected in the individual dog or cat on the basis of initial test results from routine coagulation profiles (see ACT, APTT, and PT in this section). Occasionally it is possible to measure activity of specific factors in individual patients. Specialized laboratories experienced in performing these assays should be consulted regarding sample, sample size, submission requirements, and interpretation.

CROSS-MATCH: MAJOR AND MINOR

NORMAL
Results (in dogs and cats) are reported as “compatible” (“no agglutination”) or “incompatible” (“agglutination and/or hemolysis”) in either major or minor cross-match tubes.

PATIENT PREPARATION
None

DONOR PREPARATION
None

COLLECT (PATIENT)
Venous blood, 2 mL, in RTT plus anticoagulated venous blood, 2.0 mL, in lavender-topped tube
Collect (Donor)
The same (this is where it's important to label the tubes!)

Submit (Patient)
Serum, 1 mL, plus anticoagulated whole blood, 1.0 mL

Submit (Donor)
The same

Interpretation
No agglutination and/or hemolysis in either tube indicates that the match is compatible and the donor’s blood may be used.

The presence of agglutination and/or hemolysis in the major cross-match tube indicates that the donor’s blood should not be used.

The presence of agglutination and/or hemolysis in the minor cross-match tube suggests that the compatibility is not ideal; if another donor cannot be found, the blood can be used—although with caution.

The presence of agglutination and/or hemolysis in the donor control (donor cells mixed with donor serum) suggests incompatibility; the donor’s blood should not be used.

The presence of agglutination and/or hemolysis in the patient control (patient cells mixed with patient serum) likely reflects the patient’s diagnosis. Transfusion is indicated.

Interference
In vitro hemolysis associated with difficulty collecting a sample or inappropriate handling of the blood; profound lipemia (lactescence)

Protocol
1. Wash RBCs from patient and donor in 0.9% saline solution three times; add 4.8 mL of saline to 0.2 mL of RBCs from patient and donor. Mix accordingly:
   a. Major cross-match: Mix 0.1 mL (2 drops) of donor RBCs + 0.1 mL (2 drops) of patient serum
   b. Minor cross-match: Mix 0.1 mL (2 drops) of patient RBCs + 0.1 mL (2 drops) of donor serum
   c. Patient control: Mix 0.1 mL (2 drops) of patient RBCs + 0.1 mL (2 drops) of patient serum
   d. Donor control: Mix 0.1 mL (2 drops) of donor RBCs + 0.1 mL (2 drops) of donor serum
2. Incubate for 15 minutes at 37° C. Centrifuge for 1 minute.
3. Observe the supernatant in all tubes for evidence of hemolysis in the test samples. Examine the suspension of RBCs for agglutination (macroscopically and microscopically).

D-Dimer (Fragment D-Dimer; Fibrin Degradation Fragment)
Normal
Consult laboratory reference range (dog); studies in cats are lacking.

Patient Preparation
None

Collect
Anticoagulated venous blood, 2 mL, in EDTA or heparin

Submit
Plasma, 1.0 mL
**Interpretation**

D-dimer is the proteolytic fragment of fibrinogen degradation. D-dimer concentration is used in the assessment of DIC in dogs. Elevated levels represent a marker of clot lysis and therefore support a diagnosis of DIC; a negative test result has a high negative predictive value and reliably rules out a diagnosis of DIC. The test also has the potential to identify patients with pulmonary thromboembolic disease, although results are not reliably predictive.

**Interference**

None reported

**Fibrinogen, Qualitative (Estimated)**

**Normal**

Refer to laboratory reference range (dog and cat).

**Patient Preparation**

None

**Collect**

Whole venous blood, 2.0 mL, in EDTA (lavender-topped tube)

**Submit**

Plasma, 1.0 mL

**Interpretation**

Fibrinogen levels can be estimated as the difference between plasma protein concentrations before and after heating. An increased value correlates with clot lysis and supports the diagnosis of DIC.

**Interference**

Clots in sample

**Protocol**

Invert tube several times to ensure adequate mixing of venous blood and anticoagulant.

**Fibrinogen, Quantitative**

**Normal**

100 to 245 mg/dL (dog); 110 to 370 mg/dL (cat)

**Patient Preparation**

None

**Collect**

Completely fill a citrated tube (BTT) with whole blood. Mix thoroughly. Centrifuge immediately. Transfer plasma to an RTT.

**Submit**

Plasma, 1.0 mL, in an RTT; label as “Citrated Plasma.”

**Interpretation**

Increased concentration is associated with DIC. However, there is no single test for the diagnosis of DIC. The clinician must also assess FDPs (increased), APTT (prolonged), PT (prolonged), and platelet count (decreased).
**Interference**
Incorrect ratio of citrate (anticoagulant) to whole blood; clots in sample; use of anticoagu-
lants other than citrate

**Protocol**
On collection of blood, invert tube several times to ensure adequate mixing of sample and anti-
coagulant. Centrifuge immediately. Transfer plasma to an RTT; label as “Citrated Plasma.”
Sample is stable for only 24 hours if held at 2° to 8° C; for extended storage, sample must
be frozen.

**Fibrin Degradation Products (FDPs; Fibrin Split Products [FSPs])**

**Normal**
<10 mcg/mL (dog); <10 mcg/mL (cat)

**Patient Preparation**
None

**Collect**
Venous blood, 2.0 mL, in EDTA or in an RTT
Alternatively, sample may be submitted as clotted whole blood placed into a special FDP
tube provided by the laboratory (contact the laboratory for additional information).

**Submit**
1.0 mL serum or plasma; or, as indicated by the laboratory, clotted whole blood contained in
a special FDP tube provided. Sample should be refrigerated.

**Interpretation**
Assay is used to document breakdown of fibrin clots. Increased concentration is associated
with DIC (see also D-Dimer). However, there is no single test for DIC diagnosis. The clini-
cian must also assess fibrinogen (increased), APTT (prolonged), PT (prolonged), and plate-
let count (decreased).

**Interference**
Clots in sample, unless sample submitted is clotted whole blood

**Partial Thromboplastin Time (PTT)**
See Activated Partial Thromboplastin Time (APTT).

**PIVKA Test (Proteins Induced by Vitamin K Antagonism Test; Also “Thrombotest”)**

**Normal**
Refer to laboratory reference range (dog and cat).

**Patient Preparation**
Atraumatic venipuncture is recommended.

**Collect**
Completely fill a citrated tube (BTT) with whole blood. Mix thoroughly. Centrifuge imme-
diately. Transfer plasma to an RTT.

**Submit**
Plasma, 1.0 mL, in RTT; label as “Citrated Plasma.”
**INTERPRETATION**
Test is used in conjunction with PT in the assessment of patients suspected of having warfarin toxicosis.

**INTERFERENCE**
Incorrect ratio of citrate (anticoagulant) to whole blood; clots in sample; use of anticoagulants other than citrate

**PROTOCOL**
On collection of blood, invert tube several times to ensure adequate mixing of sample and anticoagulant. Centrifuge *immediately*. Transfer plasma to an RTT; label as “Citrated Plasma.” Sample is stable for only 24 hours if held at 2° to 8° C; for extended storage, sample must be frozen.

**PLATELET COUNT**

**NORMAL**
166 to 600 × 10^3/µL (dog); 230 to 680 × 10^3/µL (cat)

**PATIENT PREPARATION**
Atraumatic collection is recommended.

**COLLECT**
Venous blood, 1.0 mL, in EDTA (lavender-topped tube)

**SUBMIT**
Entire sample

**INTERPRETATION**
Decreased platelet count is indicative of many disorders, including immune-mediated thrombocytopenia (extremely low platelet count), infection, sepsis, and DIC and therefore must be assessed in light of other physical, hematologic, and biochemical parameters. Elevated platelet counts (up to 1 million cells/µL) can be normal for some patients. Cats with extreme thrombocytosis should be tested for feline leukemia virus.

**INTERFERENCE**
Slow draw of blood from the vein, transfer of blood from syringe to tube, and traumatic venipuncture may falsely decrease platelet count.

**PROTHROMBIN TIME (PT)**

**NORMAL**
5.1 to 7.9 seconds (dog); 8.4 to 10.8 seconds (cat)

**PATIENT PREPARATION**
None

**COLLECT**
Collect whole blood in a citrated tube (BTT). Fill the tube to the capacity allowed by the vacuum. Invert immediately to mix. Centrifuge immediately and collect plasma. Transfer to a sterile plastic tube, using plastic pipette. Freeze. *Note*: Label as “Citrated Plasma.”

**SUBMIT**
Citrated plasma, 1.0 mL. Ship sample with dry ice. Store frozen.
**Interpretation**
PT is used to assess extrinsic and common coagulation pathways. Prolonged PT is used to assess patients with suspected vitamin K antagonism (warfarin toxicosis).

**Interference**
Incorrect ratio of citrate to whole blood; clots in specimen; use of a noncitrated anticoagulant (e.g., EDTA in lavender-topped tube).

If the citrated plasma sample contacts glass, clotting factor activation may occur.

**Protocol**
On collection of blood, invert tube several times to ensure adequate mixing of sample and anticoagulant. Centrifuge immediately. Transfer plasma to an RTT. (Label as “Citrated Plasma.”) Sample is stable for only 24 hours if held at 2° to 8° C; for extended storage, sample must be frozen.

**Von Willebrand Factor (vWF)**

**Normal**
Results reported are specific for the laboratory performing the test.

**Patient Preparation**
None

**Collect**
Collect whole blood in a citrated tube (BTT). Fill the tube to the capacity allowed by the vacuum. Invert immediately to mix. Centrifuge immediately and collect plasma. Transfer to a sterile plastic tube, using a plastic pipette. Freeze. Label tube as “Citrated Plasma.” Although EDTA (lavender-topped tube) may be accepted by some laboratories, sodium citrated samples are preferred when samples are submitted for vWF testing.

**Submit**
Citrated plasma, 1.0 mL. Ship sample with dry ice. If storing longer than 24 hours, store frozen.

**Interpretation**
Von Willebrand disease is the most common inherited hemostatic disorder reported in dogs. This test is usually performed to confirm the diagnosis of von Willebrand disease, in conjunction with the BMBT. Although the condition is inherited, variable degrees of expression are recognized. Dogs with vWF levels ≤30% have a tendency to bleed spontaneously (e.g., epistaxis).

**Interference**
Recent transfusion may falsely elevate vWF levels. Incorrect ratio of citrate to whole blood, a clotted specimen, and use of a noncitrated anticoagulant (e.g., EDTA in lavender-topped tube) can also affect results. Do not use a glass pipette or glass tube. If the citrated plasma sample contacts glass, clotting factor activation may occur.

**Adrenocorticotrophic Hormone (ACTH), Endogenous**

**Normal**
10 to 70 pg/mL (dog); results not reported for cat

**Patient Preparation**
Patient should be hospitalized overnight.
COLLECT
Whole blood, 2.0 mL, in EDTA (chilled, lavender-topped tube). Immediately transfer plasma to plastic tube (ACTH adheres to glass) and freeze. Samples should be stored frozen until assayed. Maximum storage time: 1 month at −20° C.

Contact laboratory directly before collecting samples for ACTH testing. Some laboratories request plasma samples be submitted in aprotinin and will provide specially prepared tubes for this purpose. Aprotinin (protease inhibitor) is added to a lavender-topped tube to stabilize ACTH. Freezing the sample is not necessary. The treated plasma should be separated immediately by centrifugation, transferred to a plastic tube, capped, and refrigerated. Transport sample to the lab with cold packs.

SUBMIT
Plasma, 1.0 mL; sample should not be allowed to sit at room temperature even for a short period.

INTERPRETATION
Adrenal tumors and iatrogenic Cushing syndrome are expected to suppress ACTH secretion; pituitary-dependent Cushing syndrome is characterized by excessive plasma concentration of ACTH.

INTERFERENCE
Recent or current corticosteroid administration; “stress” at or around the time of blood collection.

Samples must be handled quickly because ACTH disappears quickly from whole fresh blood.

PROTOCOL
After overnight hospitalization, the sample is collected between 8 and 9 AM the following morning.

ACTH STIMULATION TEST (ACTH “STIM”)

NORMAL CORTISOL LEVELS

<table>
<thead>
<tr>
<th></th>
<th>Dog</th>
<th>Cat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pretest</td>
<td>0.5-6.0 mcg/dL</td>
<td>0.5-5.0 mcg/dL</td>
</tr>
<tr>
<td>Posttest</td>
<td>6-17 mcg/dL</td>
<td>≤13 mcg/dL</td>
</tr>
</tbody>
</table>

Note: A level of 17 to 22 mcg/dL is considered “borderline” for dogs; a level of 13 to 16 mcg/dL is considered “borderline” for cats.

PATIENT PREPARATION

No prior treatment with corticosteroids for at least 5 to 7 days before testing

COLLECT

Heparinized whole blood, 2.0 mL (green-topped tube). Do not submit blood collected in EDTA.

SUBMIT

Plasma, 0.5 mL minimum for each sample submitted; sample should be refrigerated if shipped. Sample is assayed for cortisol.
**Interpretation**

The test measures endogenous cortisol before and after stimulation with ACTH. This is the most commonly used screening test for hyperadrenocorticism in dogs and cats. Patients with pituitary-dependent hyperadrenocorticism or adrenal tumor are expected to have an exaggerated cortisol response after stimulation with ACTH, assuming that the adrenal glands have retained ACTH responsiveness. Poststimulation values of 22 mcg/dL or greater are considered diagnostic for hyperadrenocorticism in dogs (16 mcg/dL or greater in cats) in the presence of clinical signs (especially PD) and supporting laboratory data and abdominal ultrasound findings. *Note:* ACTH stimulation does not differentiate between pituitary-dependent hyperadrenocorticism and adrenal tumor. An alternative test to use when screening for canine Cushing syndrome is the low-dose dexamethasone test (see following entry).

The ACTH stimulation test is the only reliable test for monitoring patients undergoing o,p′-DDD (Lysodren) treatment of pituitary-dependent hyperadrenocorticism. Dogs with adequate pharmacologic suppression of adrenal function should have unchanged prestimulation and poststimulation values (typically <2.0 mcg/dL for both).

**Interference**

Concurrent or recent treatment with corticosteroids. Anticonvulsant medications may adversely affect test results.

**Protocol**

Several protocols are available; the following are representative.

Collect pretest sample; then administer ACTH gel at 2.2 international units/kg intramuscularly (IM) (dog). Collect posttest sample 2 hours after ACTH administration.

Or

Collect pretest sample; then administer synthetic (expensive) ACTH (tetracosactrin, cosynotropin [Cortrosyn]) at 5 mcg/kg IV (not to exceed 250 mcgs) (dog) or 0.125 mg (cat) IM or intravenously (IV). Collect posttest sample 1 hour after ACTH administration (dogs).

Or

Collect pretest sample; then administer ACTH at 125 mcg IM (cat). Collect two posttest samples 30 minutes and 60 minutes after ACTH administration (cats).

**Aldosterone, Serum**

**Normal**

Pretest, 49 pg/mL (mean); posttest, 306 pg/mL (mean); reported range, 146 to 519 pg/mL (dogs). Results not reported for cats.

**Patient Preparation**

None

**Collect**

Venous blood, 2.0 mL, in EDTA (lavender-topped tube) as baseline (pretest); and repeat in 1 hour.

**Submit**

Plasma, 1.0 mL, for each of the two samples collected

**Interpretation**

Low baseline and minimal or no increase in aldosterone levels support a diagnosis of hypoaldosteronism. The test is designed to distinguish dogs with primary hypoaldrenocorticism from those with secondary hypoaldrenocorticism. However, the sensitivity of the test in dogs is such that the positive predictive value is relatively low.
INTERFERENCE
Clots in sample

PROTOCOL
In dogs, the pretest and posttest samples are collected at 1-hour intervals after administration of ACTH. Follow the same protocol used to test for hyperadrenocorticism (see ACTH Stimulation Test).

COBALAMIN (VITAMIN B₁₂)
See also Trypsin-Like Immunoreactivity and Folate.

NORMAL
249 to 733 ng/L (dog); 290 to 1500 ng/L (cat)

PATIENT PREPARATION
Overnight fast

COLLECT
Whole blood, 4.0 mL (RTT); separate serum from cells immediately

SUBMIT
Serum, 2.0 mL

INTERPRETATION
The test usually is performed in conjunction with TLI and serum folate. Decreased levels of cobalamin (vitamin B₁₂) support the diagnosis of small intestinal mucosal disease, small intestinal bacterial overgrowth, and exocrine pancreatic insufficiency. There is no significance attached to levels above the reported reference range.

INTERFERENCE
Hemolysis; lipemia

CORTISOL, RESTING (BASAL)
See also ACTH Stimulation Test.

NORMAL
0.5 to 6.0 mcg/dL (dog); 0.5 to 5.0 mcg/dL (cat)

Resting plasma cortisol in dogs is not routinely recommended because of the wide range of values in healthy animals. Although dogs with hyperadrenocorticism are expected to have increased values, reported values may still be within the limits of the reference range listed for normal dogs.

DEXAMETHASONE SUPPRESSION TEST, LOW-DOSE (LDDS TEST; DEXAMETHASONE SCREENING TEST)
NORMAL Cortisol Levels

<table>
<thead>
<tr>
<th></th>
<th>Dog</th>
<th>Cat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pretest</td>
<td>0.5-6.0 mcg/dL</td>
<td>0.5-5.0 mcg/dL</td>
</tr>
<tr>
<td>Posttest, 4 hr</td>
<td>Usually &lt;1.0 mcg/dL</td>
<td>Same</td>
</tr>
<tr>
<td>Posttest, 8 hr</td>
<td>Usually &lt;1.0 mcg/dL</td>
<td>Same</td>
</tr>
</tbody>
</table>
**Patient Preparation**

No treatment with corticosteroids for at least 5 to 7 days before testing.

**Collect**

Heparinized whole blood, 1.0 mL to 2.0 mL at each collection (green-topped tube). Do not collect blood in EDTA.

**Submit**

Plasma, 0.5 mL minimum for each sample submitted; sample should be refrigerated if shipped. Sample is assayed for cortisol.

**Interpretation**

The test measures endogenous cortisol before and after corticosteroid-induced suppression of cortisol production. This is a screening test for hyperadrenocorticism in dogs and cats. Administration of dexamethasone decreases plasma cortisol to less than 1.0 or 1.4 mcg/dL (depending on the laboratory) within 2 to 3 hours in normal dogs. An 8-hr posttest sample showing less than 1.4 mcg/dL is consistent with Cushing syndrome in dogs and cats with clinical signs (especially PD) and supporting laboratory data and abdominal ultrasound findings. Note: LLDS does not differentiate between pituitary-dependent hyperadrenocorticism and adrenal tumor.

The 4-hr posttest sample is not interpreted as part of the screening test but is considered an aid in differentiating pituitary-dependent hyperadrenocorticism from adrenal-dependent disease. Demonstrating a transient cortisol suppression (4-hour posttest sample) supports pituitary-dependent disease and rules out adrenal-dependent disease.

**Interference**

Recent or concurrent corticosteroid administration. Anticonvulsant medications may adversely affect test results.

**Protocol**

Collect pretest sample of plasma; administer dexamethasone (either in sodium phosphate or polyethylene glycol) at 0.01 mg/kg IV in dogs, or 0.1 mg/kg IV in cats; then collect a 4-hr posttest plasma sample, followed by an 8-hr posttest plasma sample. Submit the three plasma samples. (Note the higher dose of dexamethasone used in cats versus dogs.)

**Dexamethasone Suppression Test, High-Dose (HDDS Test)**

**Normal Cortisol Levels**

<table>
<thead>
<tr>
<th></th>
<th>Dog</th>
<th>Cat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pretest</td>
<td>0.5–6.0 mcg/dL</td>
<td>Same</td>
</tr>
<tr>
<td>Posttest, 4 hr</td>
<td>Usually &lt;1.0 mcg/dL</td>
<td>Same</td>
</tr>
<tr>
<td>Posttest, 8 hr</td>
<td>Usually &lt;1.0 mcg/dL</td>
<td>Same</td>
</tr>
</tbody>
</table>

**Patient Preparation**

No treatment with corticosteroids for at least 5 to 7 days before testing.

**Collect**

Heparinized whole blood, 1.0 mL to 2.0 mL at each collection (green-topped tube). Do not collect blood in EDTA.

**Submit**

Plasma, 0.5 mL minimum for each sample submitted; sample should be refrigerated if shipped; sample is assayed for cortisol.
INTERPRETATION
The test measures endogenous cortisol before and after corticosteroid-induced suppression of cortisol production. The HDDS test is used in dogs with abnormal ACTH stimulation or LDSS test results to distinguish between pituitary-dependent disease and adrenal tumor. Administration of dexamethasone decreases plasma cortisol to less than 1.0 or 1.4 mcg/dL (depending on the laboratory) within 2 to 3 hours in normal dogs. Dogs with an adrenal tumor or pituitary-dependent hyperadrenocorticism are not expected to demonstrate suppression of cortisol after administration of dexamethasone at the dose prescribed.

Note: “Suppression” is defined as:
Plasma cortisol concentration less than 50% of baseline at 4 hours or at 8 hours after dexamethasone administration
or
Plasma cortisol concentration <1.4 mcg/dL at 4 hours or at 8 hours after dexamethasone administration

INTERFERENCE
Recent or concurrent corticosteroid administration. Anticonvulsant medications may adversely affect test results.

PROTOCOL
Collect pretest sample of plasma; administer dexamethasone (either in sodium phosphate or in polyethylene glycol) at 0.1 mg/kg IV in dogs, or 1.0 mg/kg IV in cats; then collect a 4-hour posttest plasma sample, followed by an 8-hour posttest plasma sample. Submit the three plasma samples. (Note the higher dose of dexamethasone used in cats versus dogs.)

ESTRADIOL (BASELINE)
NORMAL
Not normally detectable (dog and cat)

PATIENT PREPARATION
None

COLLECT
Venous blood, 2.0 mL, in an RTT

SUBMIT
Serum, 1.0 mL

INTERPRETATION
This assay is not commonly requested for dogs and cats. Elevated levels have been used to detect testicular tumors and ovarian remnant syndrome. However, better tests are available.

INTERFERENCE
Variations in results occur with different methodologies used.

FOLATE
See also Trypsin-Like Immunoreactivity and Cobalamin.

NORMAL
6.5 to 11.5 mcg/L (dog); 9.7 to 21.6 mcg/L (cat)

PATIENT PREPARATION
Overnight fast
**COLLECT**
Whole blood, 4.0 mL (RTT); separate serum from cells immediately.

**SUBMIT**
Serum, 2.0 mL

**INTERPRETATION**
Assay usually is performed in conjunction with TLI and serum cobalamin. Elevated levels of folate support the diagnosis of small intestinal bacterial overgrowth in the upper small intestine. Values below the reference range support the diagnosis of proximal small intestinal disease.

**INTERFERENCE**
Hemolysis; lipemia

**FRUCTOSAMINE**

**NORMAL**
225 to 375 µmol/L (dog and cat)
Refer to laboratory reference range; ranges vary depending on methodology used.

**PATIENT PREPARATION**
Fasted

**COLLECT**
Whole blood, 2.0 mL, in an RTT (serum), lavender-topped tube (plasma in EDTA) or green-topped tube (plasma in heparin). Caution: Sample must be nonhemolyzed.

**SUBMIT**
Serum or plasma, 1.0 mL

**INTERPRETATION**
Test results reflects glycemic levels over the preceding 1 to 3 weeks; test generally is used in assessing quality of glycemic control in patients with diabetes mellitus.

**INTERFERENCE**
Hemolysis; icterus

**GASTRIN**

**NORMAL**
Varies according to individual laboratory (dog); results not established for cat.

**PATIENT PREPARATION**
None

**COLLECT**
Venous blood, 2.0 mL, in RTT

**SUBMIT**
Serum, 1.0 mL; sample should be kept frozen until assayed.

**INTERPRETATION**
Test is not commonly performed. Levels will be elevated in patients with functional gastrinoma, pyloric obstruction, renal failure, and gastric ulcers. There is no significance associated with decreased values.
**INTERFERENCE**
Concurrent administration of histamine-2 (H₂) antagonist drugs (e.g., cimetidine)

**GLUCAGON STIMULATION, INTRAVENOUS (IVGS TEST)**
Intravenous glucagon stimulation is a complex test protocol to perform and yields results that are not generally reliable in distinguishing patients with type 1 diabetes from those with type 2 diabetes. It has been used to diagnose patients with insulin-secreting tumor. However, risk is associated with performing this test in patients with insulin-secreting tumor. Administration of glucagon will elevate serum glucose, which, in turn, promotes secretion of excessive amounts of insulin; subsequent hypoglycemic crisis is a potential consequence.

**GLUCOSE CURVE, 12-HOUR**

**NORMAL**
Glucose concentration ranges between 100 mg/dL and 250 mg/dL for the entire sampling period (dog and cat).

**PATIENT PREPARATION**
Ideally, a venous catheter should be placed 1 hour before serial collections are started. Patient attitude during testing is important, because stressed or unusually aggressive animals may not be appropriate subjects for this test.

**COLLECT**
Venous blood, approximately 1 mL per collection; plan on drawing as many as seven samples.

**SUBMIT**
Serum from each sample for routine glucose determination

**INTERPRETATION**
This test is used to evaluate glucose levels in diabetic patients receiving insulin, particularly those who may experience recurrence of clinical signs as a result of undertreatment. Objectively, sufficient numbers of samples should be collected to establish a true nadir (lowest point) during the day. For example, if the nadir is greater than 450 mg/dL, each dose of insulin might be increased by 1 to 2 units. When increasing insulin dose, it is appropriate to increase the dose by the same number of units for each administration throughout the day.

**INTERFERENCE**
Stress. Also, use of portable glucose meters to measure serial glucose levels in individual patients may result in falsely lower values.

**PROTOCOL**
The patient is given the usual dose of insulin and fed at home in the morning. On arrival at the hospital, a short intravenous catheter is placed in a suitable vein and secured appropriately. Serial samples are collected at 2-hour intervals over a 10- to 12-hour period. At the conclusion of the sampling period, the patient is usually fed and given a second daily dose of insulin, as appropriate. Then you, and the patient, can go home.

**GLUCOSE TOLERANCE TEST, INTRAVENOUS (IVGT TEST)**
Not generally recommended
NORMAL
By 60 minutes postinjection, serum insulin should be within 1 standard deviation of the baseline, and serum glucose should be within normal reference range for both dogs and cats.

PATIENT PREPARATION
24-hour fast. Preplacement of an intravenous catheter is recommended.

COLLECT
Whole blood, 2.0 mL in an RTT, for each sample submitted

SUBMIT
Serum, 1.0 mL, for each sample submitted

INTERPRETATION
Uncommonly performed, the IVGT test is an “insulin secretagogue test” used to distinguish type 1 diabetes from type 2 in cats. (Note: It is appropriate to consider all diabetic dogs as having type 1 [insulin-dependent] diabetes.) Patients with a mean serum insulin level greater than 15 mcg/mL by 60 minutes after injection are likely to have type 2 (non-insulin-dependent) diabetes. However, in cats, results are inconsistent and rarely diagnostic—another reason why this is not a popular test.

INTERFERENCE
Hemolysis. Prolonged contact of serum with RBCs will cause a false decrease in glucose concentration. (Note: Do not use gray-topped tubes to collect samples.) The IVGT test can be adversely influenced by diet, certain drugs (steroids, insulin), stage of estrus, underlying illness or infection (sepsis), and stress.

PROTOCOL
1. Fast the patient overnight.
2. Place an intravenous catheter.
3. Collect venous blood in an RTT. Submit 1.0 mL serum for a baseline glucose.
4. Administer 0.5 g/kg of 50% glucose solution IV over 30 seconds.
5. Collect approximately 2.0 mL of whole blood at 1 minute, 5 minutes, 15 minutes, 25 minutes, 35 minutes, 45 minutes, 1 hour, and 2 hours after administration of glucose. (Times may vary slightly depending on author and/or reference used.)
6. Submit 0.5 to 1.0 mL of serum for each sample. Note: Centrifuge and separate each serum sample as soon as practical after clot formation.
   Each sample is submitted for both insulin and glucose determination.

GLUCOSE TOLERANCE TEST, ORAL (OGT TEST)
Not generally recommended
   The OGT test, although commonly performed in humans, is rarely performed in dogs and cats because of the difficulty associated with reliably administering the required volume of glucose orally.
   An oral glucose absorption test has previously been described in the literature as a means of assessing patients with malabsorptive GI disorders. Today, considering that superior tests are available, this test is no longer recommended for the assessment of malassimilation in dogs and cats.

INSULIN
NORMAL
5 to 20 µU/mL (dog and cat)
**Patient Preparation**
Overnight fast

**Collect**
Whole blood, 2.0 mL, in RTT

**Submit**
Serum, 1.0 mL

**Interpretation**
Test is indicated for the diagnostic assessment of patients suspected of having an insulin-secreting tumor (e.g., insulinoma). If the patient has profound hypoglycemia at the time the test sample is collected, test results for insulin may be reported as normal. Simultaneous testing of serum glucose is recommended. Low glucose (<60 mg/dL) and an insulin level of greater than 20 µU/mL are consistent with insulin-secreting tumor.

**Interference**
Hemolysis; blood collected in EDTA (plasma)

**Protocol**
Most laboratories recommend that the patient’s insulin level be determined in conjunction with blood glucose. Profound hypoglycemia may result in a normal insulin level being reported. A recent meal as well as several drugs can influence insulin concentrations.

**Parathyroid Hormone (PTH)**

**Normal**
2 to 13 pmol/L (dog and cat). Results vary among laboratories.

**Patient Preparation**
12-hour fast

**Collect**
Whole blood, 2.0 mL, in RTT. Serum should be separated from cells within 1 hour after collection; serum should be frozen and shipped on ice. Deliver to laboratory via overnight delivery. Keep frozen.

**Submit**
Serum (*frozen*), 1.0 mL, in sterile plastic tube. Do not ship in an SST.

**Interpretation**
PTH levels will be increased in patients with primary hyperparathyroidism, secondary renal or nutritional hyperparathyroidism, and other disorders causing hypocalcemia. No measurable PTH level is consistent with primary hypoparathyroidism. PTH testing should always include iCa assay.

**Interference**
Hemolysis; thawing of sample for extended periods.

**Parathyroid Hormone-Related Protein (PTHrP)**

**Normal**
Refer to laboratory reference range values (dog and cat).
**Patient Preparation**

12-hour fast

**Collect**

Whole blood, 2.0 mL, in RTT. Serum should be separated from cells within 1 hour after collection; serum should be frozen and shipped with ice in plastic tube. Deliver to laboratory via overnight delivery. Keep frozen.

**Submit**

Serum (*frozen*), 1.0 mL, in sterile plastic tube

**Interpretation**

Interpretation of PTHrP entails simultaneous testing for calcium (or iCa) and PTH. PTHrP levels are low to undetectable in patients with primary hyperparathyroidism. Patients with hypercalcemia associated with lymphosarcoma or chronic renal insufficiency will have increased levels of PTHrP.

**Test Interference**

Hemolysis; thawing of sample for extended periods

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**T₃ (TRIIODOTHYRONINE)**

**Normal**

0.8 to 1.5 mg/dL by radioimmunoassay (RIA) (dog); 0.8 to 1.5 ng/mL by RIA (cat)

Results will vary among different laboratories.

**Patient Preparation**

None (patient should not be receiving exogenous thyroid hormone supplementation).

**Collect**

Whole blood, 1 to 2 mL, in RTT

**Submit**

Serum, 0.5 mL minimum. Storage and shipment of samples in plastic, rather than glass, containers is recommended. Sample should be frozen and shipped with cold packs.

**Interpretation**

T₃ is a poor indicator of thyroid function and generally provides little reliable diagnostic information pertaining to thyroid-related disease; baseline T₃ does not reliably distinguish between hypothyroid and euthyroid states. Test results for T₃ include both free T₃ (fT₃) and protein-bound T₃. RIA is the preferred test method.

**Interference**

Patients receiving exogenous thyroid supplementation can have positive or negative test interference, depending on the dose of drug administered and the time the last dose was given. T₃ autoantibody, if present, may falsely lower test results. Note: Storage of serum or plasma in glass can cause a significant false increase in serum T₃ concentration.

**Reverse T₃ (rT₃; REVERSE TRIIODOTHYRONINE)**

There are currently no established diagnostic guidelines associated with baseline reverse T₃ values in dogs and cats.

**T₃ Suppression**

**Normal**

Suppression of T₄ to 1.5 mcg/dL after seven doses of synthetic T₃ (in cats)
Patient Preparation
None

Collect
Whole blood, 3.0 mL, in RTT, for each sample (pretest and posttest)

Submit
Serum, 1.0 mL minimum for each sample

Interpretation
This test measures $T_4$ and $T_3$ after sequential administration of seven doses of synthetic $T_3$; it may distinguish between euthyroid and slightly hyperthyroid cats. Hyperthyroid cats demonstrate minimal or no decrease in serum $T_4$, which remains at 2.0 mcg/dL or more. $T_4$ values of 1.5 to 2.0 g/dL are nondiagnostic. $T_3$ values should increase in all cats (normal as well as hyperthyroid). If $T_3$ values do not increase, test results are considered invalid.

Interference
Hemolysis; lipemia; icterus; blood collected in EDTA

Protocol
Pretest sample is collected (to be submitted for $T_3$ and $T_4$ testing).

Free $T_4$ ($fT_4$)

Normal
0.8 to 3.5 ng/dL (dog); 1.0 to 4.0 ng/dL (cat)

Patient Preparation
None

Collect
Venous blood, 2.0 mL, in RTT

Submit
Serum, 1.0 mL minimum; freeze and store in plastic tubes rather than glass; ship samples with cold packs to arrive for analysis within 5 days of collection.

Note: Conventional RIA technology has largely been replaced by the equilibrium dialysis (ED) technology in performing $fT_4$ assays. Recently, IDEXX Laboratories announced the introduction of a new technology for determining $fT_4$ that is faster yet provides results comparable to those achieved with ED technology.

Interpretation
$fT_4$ is used in preference to conventional $T_4$ to confirm hypothyroidism in dogs and hyperthyroidism in cats. Results less than 0.8 ng/dL (and especially less than 0.5 ng/dL) are consistent with a diagnosis of hypothyroidism in dogs. As with conventional $T_4$ levels, $fT_4$ levels in cats that exceed 4.0 ng/dL are consistent with the diagnosis of hyperthyroidism.

Interference
Storing and shipping serum in glass containers may alter test results; circulating thyroid autoantibody does not interfere with test results. Results determined by RIA alone may be significantly lower than those determined by the ED method. Severe illness may cause low $fT_4$ values in dogs with normal thyroid function (sick, euthyroid). $T_4$ autoantibody does not interfere with the assay for $fT_4$. 

www.ajlobby.com
TOTAL T\textsubscript{4} (THYROXINE OR TETRAIODOTHYRONINE)

**NORMAL**
1.5 to 3.5 mcg/dL (dog); 1.0 to 4.0 mcg/dL (cat)

| Note: | A point-of-care enzyme-linked immunosorbent assay (ELISA) test kit for in-hospital assessment of T\textsubscript{4} is available. However, it is recommended that ELISA test results be confirmed by RIA. |

**PATIENT PREPARATION**
None (patient should not be receiving exogenous thyroid hormone supplementation).

**COLLECT**
Whole blood, 1 to 2 mL, in RTT

**SUBMIT**
Serum, 0.5 mL minimum. Storage and shipment of samples in plastic, rather than glass, containers is recommended. Sample should be frozen and shipped with cold packs.

**INTERPRETATION**
T\textsubscript{4} is produced within the thyroid gland, and therefore the total T\textsubscript{4} is the preferred test of thyroid function. The test combines measurement of fT\textsubscript{4} plus protein-bound T\textsubscript{4}. Dogs with T\textsubscript{4} levels less than 2.0 mcg/dL are likely to have hypothyroidism (if associated clinical signs are present); cats with T\textsubscript{4} levels less than 4.0 mcg/dL are likely to have hyperthyroidism.

**Dogs**
Decreased values suggest hypothyroidism (dogs); however, dogs with underlying illness not related to abnormal thyroid function may still have abnormally decreased T\textsubscript{4} concentration (sick, euthyroid). A comprehensive physical examination and laboratory profile are indicated in establishing a diagnosis of hypothyroidism in dogs.

**Cats**
In middle-aged and old cats, hyperthyroidism becomes an important differential diagnosis when T\textsubscript{4} levels exceed 4.0 mcg/dL in the presence of clinical signs. Most cases are caused by a functional multinodular adenoma. Less than 5% of cases are associated with thyroid adenocarcinoma.

**INTERFERENCE**
Patients receiving exogenous thyroid supplementation can have positive or negative test interference, depending on the dose of drug administered and the time the last dose was given. Underlying illness and T\textsubscript{4} autoantibody, if present, may falsely lower test results. **Note:** Storage of serum or plasma in glass can cause a significant false increase in serum T\textsubscript{4} concentration.

THYROTROPIN, CANINE (THYROID-STIMULATING HORMONE [TSH]; BASELINE TSH)

**NORMAL**
Up to 0.6 ng/mL (dog); values not established for cats.

| Note: | Lower limits of normal (approximately 0.1 ng/mL) for methodologies used are below the sensitivity of the assay. |

**PATIENT PREPARATION**
None, if patient is not receiving exogenous thyroid hormone supplementation

www.ajlobby.com
Collect
Venous blood, 1 to 2 mL, in RTT

Submit
Serum, 0.5 mL minimum
  Storage and shipment of samples in plastic, rather than in glass, containers is recommended. Sample should be frozen and shipped with cold packs.

Interpretation
This is a reasonable test for the assessment of hypothyroidism in dogs; however, TSH fluctuations can produce normal results in 20% to 40% of hypothyroid dogs. TSH should not be interpreted without having same-sample results for \( T_4 \) or \( fT_4 \). A low \( T_4 \) or \( fT_4 \) and increased TSH in a dog are consistent with the diagnosis of hypothyroidism. Normal \( T_4 \) or \( fT_4 \) and TSH effectively rule out hypothyroidism. Clinical signs and a routine laboratory profile must be part of the diagnostic assessment of any patient suspected of having thyroid disease.

Interference
The same interfering factors that influence \( T_4 \) assays are likely to affect TSH.

TSH or Thyroid-Stimulating Hormone, Canine
See Thyrotropin, Canine.

Thyrotropin Response (TSH Response Test)
Not generally recommended
  Initially believed to be useful in diagnosing hyperthyroidism, the TSH response test has been shown in subsequent studies to be limited in the ability of abnormal thyroid tissue to respond to stimulation. Other test limitations, including the removal of bovine TSH from the market, have resulted in the current recommendation against its use.

ImmunoLogic

Acetylcholine (ACh) Receptor Antibody for Myasthenia Gravis
Normal
<0.6 nmol/L (dog); <0.3 nmol/L (cat)

Patient Preparation
None.

Collect
2.0 to 4.0 mL whole blood

Submit
1.0 to 2.0 mL serum (may be submitted in an SST). Ship with a cold gel pack during summer months. Do not submit whole blood.

Interpretation
ACh receptor antibody levels exceeding the reference range for the laboratory is highly indicative of acquired myasthenia gravis in dogs and cats. Patients with congenital myasthenia gravis are not expected to have elevated levels. Cranial mediastinal masses are common in cats with elevated levels. \textit{Note:} The ACh receptor antibody test is significantly
more sensitive (fewer false negatives) than the “Tensilon test” for diagnosis of myasthenia gravis.

**INTERFERENCE**
Significant lipemia (lactescence). Another sample should be collected after a significant fasting period. If the patient is still lipemic after a fast, the clinician should attempt to determine the underlying cause, as this could be a risk for acute pancreatitis or other GI disorders.

**ALLERGEN-SPECIFIC IMMUNOGLOBULIN E (IgE) ANTIBODY TEST (RADIOALLERGOSORBENT TEST [RAST]; ALLERGY SCREEN)**

**NORMAL**
Refer to laboratory for interpretation of results reported.

**PATIENT PREPARATION**
None

**COLLECT**
Venous blood, 2.0 mL, in RTT

**SUBMIT**
Serum, 1.0 mL

**INTERPRETATION**
This in vitro assay is used to identify causative allergens in atopic animals. The RAST has also been suggested for evaluation of patients with suspected food-related hypersensitivity. At this time, results are inconclusive.

**INTERFERENCE**
Concurrent corticoid therapy

**ANTIBODY TITERS FOR INFECTIOUS DISEASE DIAGNOSIS**
See Infectious Disease Serology and Microbiology.

**ANTINUCLEAR ANTIBODY (ANA)**

**NORMAL**
Results are reported as a titer (ratio); refer to the laboratory reference range (dog and cat).

**PATIENT PREPARATION**
None

**COLLECT**
Venous blood, 2.0 mL, in RTT

**SUBMIT**
Serum, 1.0 mL

**INTERPRETATION**
This is an adjunctive (arguably the most important) test in the assessment of patients suspected of having systemic lupus erythematosus (SLE). Results must be interpreted after considering other underlying disorders in the individual patient. Low positive titers will be reported in patients having any of several disorders, including inflammatory disease, neoplasia, and infectious diseases. A high positive titer, in the presence of associated clinical and laboratory findings, supports a diagnosis of SLE.
INTERFERENCE
Concurrent illness or infection

ANTIPLATELET ANTIBODY
No commercially available test
To date, a sensitive and specific test for the diagnosis of immune-mediated thrombocytopenia (IMT) by determination of antiplatelet antibody has not been developed. Generally, extreme thrombocytopenia (<30,000 platelets/mm³) is managed with immunosuppressive doses of corticosteroids on the assumption that the condition is immune mediated.

COOMBS TEST (DIRECT COOMBS TEST; DIRECT ANTIGLOBULIN TEST [DAT])

NORMAL
Negative (dog and cat)

PATIENT PREPARATION
None

COLLECT
Anticoagulated venous blood, 1.0 mL, in EDTA (lavender-topped tube)

SUBMIT
Entire sample of anticoagulated venous blood

INTERPRETATION
The Coombs test detects presence of antibody and/or complement on the surface of RBCs and supports the diagnosis of IMHA. It is generally reported by degree of positivity: +1 to +4. Strength of the reaction does not predict severity of the disease or prognosis. A negative test result does not rule out the diagnosis of IMHA. The test is reported to be positive only 60% to 70% of the time.

INTERFERENCE
Concurrent steroid therapy; severe autoagglutination

RHEUMATOID FACTOR, CANINE

NORMAL
Negative (dog); values not established for cats

PATIENT PREPARATION
None

COLLECT
Venous blood, 2.0 mL, in RTT

SUBMIT
Serum, 1.0 mL

INTERPRETATION
This assay detects the presence of circulating autoantibody directed against IgG. It is an adjunctive test used in the diagnostic assessment of patients suspected of having rheumatoid arthritis or SLE. Results are reported as “positive” or “negative.” A positive test result does not confirm a diagnosis of rheumatoid arthritis. Several other immune-mediated disorders, especially if chronic, can cause positive test results.
ANAPLASMA PHAGOCYTOPHILUM ANTIBODY

NORMAL
Negative (dog)

PATIENT PREPARATION
None

COLLECT
Venous blood, 2.0 mL, in RTT

SUBMIT
Serum, 1.0 mL

INTERPRETATION
Limited studies are available about antibody responses to infection with Anaplasma phagocytophilum; refer to the laboratory test results for information on interpretation.

INTERFERENCE
Cross-reactivity with A. platys is expected.

ASPERGILLUS SPECIES ANTIBODY TITER (NONAVIAN)

Not generally recommended in dogs and cats

The high rate of false-positive and false-negative test results (depending on test methodology) limits the value of serology in establishing a diagnosis of aspergillosis in dogs without clinical signs.

NORMAL
Negative (dog and cat)

PATIENT PREPARATION
None

COLLECT
Venous blood, 2 to 3 mL, in RTT

SUBMIT
Serum, 1.0 mL minimum

INTERPRETATION
It is recommended to concurrently request Penicillium species titer. A positive antibody titer in a dog that is not responsive to empiric antibiotic therapy and with persistent nasal discharge, masseter muscle atrophy, and erosions of the nasal planum is highly suggestive of aspergillosis.

INTERFERENCE
A positive test result may simply denote exposure.
**BABESIA ANTIBODY TITER, CANINE**

**Normal**

*Babesia canis*, <80; *B. gibsoni*, <320

**Patient Preparation**

None

**Collect**

Venous blood, 2 to 3 mL, in RTT

**Submit**

Serum, 1.0 mL minimum

**Interpretation**

Titers greater than 80 for *B. Canis* or greater than 320 for *B. gibsoni* are consistent with the diagnosis of infection in patients with corresponding clinical symptoms.

**Interference**

There can be considerable cross-reactivity between serologic assays for *B. Canis* and *B. gibsoni*. Negative results in patients suspected of being infected should be followed with a convalescent sample 4 weeks after the initial test.

**BARTONELLA SPECIES (BARTONELLA HENSELAE TITER)**

**Normal**

Negative (cat)

**Patient Preparation**

None

**Collect**

Venous blood, 2.0 mL, in RTT

**Submit**

Serum, 1.0 mL

**Interpretation**

Different test methodologies are used commercially, including immunofluorescent antibody (IFA) assay, ELISA, and Western blot analysis. Although there is cross-reactivity with other *Bartonella* species, the test is reported to be relatively sensitive and specific for infection in cats. At issue, however, is whether all cats that test positive are, in fact, clinically ill and whether treatment is indicated on the basis of one positive test result. Results on positive cats may be reported as “Serum, +1 to +4.”

**Interference**

None reported

**BLASTOMYCOSIS ANTIBODY TITER**

**Normal**

Negative (dog and cat)

**Patient Preparation**

None
**INFECTIOUS DISEASE SEROLOGY AND MICROBIOLOGY**

**COLLECT**
Venous blood, 2.0 mL, in RTT

**SUBMIT**
Serum, 1.0 mL minimum

**INTERPRETATION**
A positive serologic test result in a dog with clinical signs consistent with blastomycosis does correlate with infection. Many cats with known blastomycosis infection, however, will have negative serologic results.

**INTERFERENCE**
None reported

**BLOOD CULTURE (BACTERIA)**

**NORMAL**
Negative ("no growth") after 10 or more days of incubation (dog and cat)

**PATIENT PREPARATION**
Ideally, sample should be collected while patient is febrile. The peripheral vein must be surgically prepared before venipuncture. Use at least two veins. Do not collect blood via a catheter.

**COLLECT**
Venous blood, 6 to 10 mL, in a syringe (with no anticoagulant added)

**SUBMIT**
Transfer blood directly to a suitable (commercially prepared) vial containing a blood culture medium. **Note:** Special media designed to remove certain antibiotics are available for patients that are concurrently receiving antibacterial therapy at the time of sample collection.

**INTERPRETATION**
The laboratory will report identification of any growth and minimum inhibitory concentration (MIC) susceptibility test results.

**INTERFERENCE**
Contaminating bacteria obtained during the collection process

**PROTOCOL**
Samples from a separate vein, when feasible, should be collected. Generally, three samples are submitted from the same patient, taken at approximately 1-hour blood intervals, collected by venipuncture (syringe and needle) from different sites.

**BORRELLIA BURGDORFERI**
See Lyme Borreliosis.

**BRUCELLA CANIS ANTIBODY**

**PRELIMINARY ASSESSMENT BY RSAT OR TAT**
RSAT is rapid slide agglutination test; TAT is tube agglutination test.

**NORMAL**
Negative (dog)
Patient Preparation
None

Collect
Venous blood, 2.0 mL, in RTT

Submit
Serum, 1.0 mL

Interpretation
Dogs with a negative test result are likely not to be infected. Follow-up testing is recommended for dogs with a negative test result but high likelihood of infection. Dogs with a positive test result should be retested by agar gel immunodiffusion (AGID) (see following entry) to confirm infection.

Interference
Because of the nature of the screening tests, the frequency of false-positive test results can be high.

Protocol
Both the RSAT and the TAT should be performed with 2-mercaptoethanol (2-ME) to eliminate interference caused by heterologous IgM (responsible for most false-positive reactions).

Note: Optional testing by IFA is commercially available. Consider using IFA to compare with the RSAT and TAT.

Confirmatory Test by AGID (Agar Gel Immunodiffusion)
Normal
Titers less than 50 are considered negative (dog).

Patient Preparation
None

Collect
Venous blood, 2.0 mL, in RTT

Submit
Serum, 1.0 mL

Interpretation
Titers greater than 200 are generally consistent with positive results on blood culture.

Interference
None reported

Canine Distemper Antibody

In Cerebral Spinal Fluid (IgG or IgM)
Normal
Negative (refer to laboratory reference range).

Patient Preparation
Fluid should be collected from the cisterna magna; sample is collected with the patient under general anesthesia and an endotracheal tube placed; sterile technique is required.
Collect
CSF

Submit
CSF, 1.0 mL

Interpretation
The presence of any titer for canine distemper virus (CDV) antibody (IgG or IgM) in CSF is consistent with infection, provided there is no contamination of the sample with blood or plasma. CSF titers should be assessed in conjunction with serum antibody titer.

Interference
Blood or plasma contamination of sample during collection may cause false-positive results in vaccinated dogs. Vaccine-induced antibody is not expected to cross into CSF.

Note: When individual patients are being assessed for distemper antibody, various laboratory methods are used to perform serology. The virus neutralization (VN) test method for CDV antibody is recommended.

In Serum (IgG or IgM)
Normal
Any patient with a “positive” titer is considered to have protective immunity. Titer results do not distinguish prior exposure and recovery from vaccination. Uninfected dogs have no evidence of a rising titer when results of the acute and convalescent titers are compared. A single IgM titer is expected to be negative in patients that have a “positive” titer subsequent to vaccination. Vaccination will affect results. Methodology (e.g., VN, IFA) will affect values reported. Refer to laboratory reference range.

Patient Preparation
None

Collect
Whole blood, 2.0 mL

Submit
Serum, 1.0 mL

Interpretation
Actual test results may vary from one laboratory to another. Individual laboratories will provide interpretation information.

COCCIDIOIDOMYCOSIS ANTIBODY TITER
Normal
Negative (dog and cat)

Patient Preparation
None

Collect
Whole blood, 2.0 mL, in RTT
Submit
Serum, 1.0 mL

Interpretation
Recent advances in testing have resulted in the use of various test methods by different laboratories. Although AGID has been largely used, latex agglutination and ELISA test methodologies are also available. In cats, serotesting is more likely to be performed for tube precipitin (TP, which is predominantly IgM) and complement fixation (CF, which is predominantly IgG) antibodies. In dogs and cats, false-negative test results can occur.

Interference
Cross-reactivity in patients with histoplasmosis or blastomycosis can occur with all test methods used to detect Coccidioides immitis antibody.

Cryptococcal Antigen (Serum or CSF)

Normal
Negative (dog and cat)

Patient Preparation
None

Collect
Venous blood, 2.0 mL, in RTT; CSF, 0.5 mL

Submit
Serum, 1.0 mL; CSF, 0.5 mL

Interpretation
Any titer to Cryptococcus neoformans is consistent with infection and justifies treatment. Antibody titers for cryptococcosis are not valid.

Interference
None reported

Ehrlichia canis Antibody

Normal
Refer to laboratory reference range (dog and cat).

Patient Preparation
None

Collect
Venous blood, 2.0 mL, in RTT

Submit
Serum, 1.0 mL

Interpretation
Interpretation varies among laboratories and methodologies used to detect antibody. The clinician must consider test results with clinical signs and results of routine laboratory profiles when making the decision to treat a patient with a positive titer. Dogs with confirmed
infections may continue to have a positive antibody titer for *E. canis* for several months after recovery and despite several weeks of antibiotic therapy.

**Note:** Correlation between antibody titer and active infection is poor with all available antibody tests on the market today.

**INTERFERENCE**
None reported

**FELINE CORONAVIRUS ANTIBODY (FeCoV Ab)**
Inappropriately called the *FIP Ab test*; not generally recommended

Coronavirus titer is not a diagnostic test for FIP. In fact, at this time there is no diagnostic test for FIP. The only value in submitting serum for a coronavirus titer is to identify cats that have a truly negative antibody titer (titered to zero). A negative antibody titer, although exceptionally rare in domestic cats, may denote no prior exposure to coronavirus (FIP).

**EHRlichia SPECIES (BY PCR)**

**NORMAL**
Negative (dog and cat)

**PATIENT PREPARATION**
None

**COLLECT**
Venous blood, 2.0 mL, in EDTA (lavender-topped tube)

**SUBMIT**
Entire sample

**INTERPRETATION**
Reported as positive or negative; positive samples should be subjected to further testing in order to confirm infection.

**INTERFERENCE**
PCR tests are subject to false-positive results because of minute traces of cross-reacting DNA in the sample.

**FELINE INFECTIOUS ANEMIA**
See *Haemobartonella*.

**FELINE CORONAVIRUS (BY RT-PCR)**

**NORMAL**
Negative for coronavirus RNA

**PATIENT PREPARATION**
None

**COLLECT**
RT-PCR can be performed on body fluids (including blood, serum, and plasma), frozen tissue, tissue imbedded in paraffin (for histopathology), tissue aspirates, CSF, and feces. Pleural and/or abdominal fluids are preferred.
**Submit**
Specimens should be submitted in a sterile tube or RTT. Whole blood specimens should be submitted as anticoagulated blood collected in EDTA (lavender-topped tube).

**Interpretation**
RT-PCR does not distinguish between benign FeCoV and the coronavirus known to cause FIP; therefore false-positive test results are possible. Results must be correlated with laboratory and clinical findings. The presence of FeCoV in abdominal or pleural effusions correlates well with active infection.

**Interference**
Not reported.

**Feline Coronavirus Antibody (FeCoV Ab)**
Inappropriately called the FIP Ab test; not generally recommended as a valid diagnostic test for cats suspected of having FIP. The presence of FeCoV antibody, regardless of level, is not diagnostic for FIP. The only value in submitting serum for a coronavirus titer is to identify cats that have a truly negative antibody titer (titered to zero) and therefore have not been exposed to FeCoV. A negative antibody titer, although uncommon among domestic cats, may denote no prior exposure to coronavirus (FIP).

**Feline Leukemia Virus Antigen (FeLV Ag; p27 Ag TEST)**
*All* commercial and in-hospital FeLV tests detect antigen, not antibody.

**Normal**
Negative (denotes absence of virus)

**Patient Preparation**
None

**Collect**
**IFA**
Whole blood, 1.0 mL, in EDTA (lavender-topped tube)

**ELISA**
Whole blood, 2.0 mL, in RTT

**Submit**
**IFA**
Buffy coat smear or 1.0 mL anticoagulated whole blood collected in EDTA. *Note:* IFA is the preferred method for assessing bone marrow aspiration samples for FeLV Ag.

**ELISA**
Serum, 1.0 mL

**Interpretation**
Both IFA and the ELISA detect the presence of the core protein p27.

**IFA**
A positive test result identifies the presence of FeLV cell-associated antigen (in WBCs and/or platelets) and defines “persistent infection,” especially in cats with clinical and/or laboratory signs consistent with FeLV infection.
ELISA
A positive test result identifies the presence of soluble, circulating FeLV antigen; healthy cats with a positive test result should be retested in 1 to 2 months to reassess virus status or should be subjected to corroborative testing by IFA.

Note
The American Association of Feline Practitioners (AAFP) and the Academy of Feline Medicine (AFM) AAFP/AFM Task Force on Feline Retrovirus Testing stresses that healthy cats with positive test results may have false-positive test results (by either method). Corroborative testing using a different test method is indicated. A positive test result in a cat with clinical signs suggestive of chronic illness, lymphoid neoplasia, or significant hematologic abnormalities is highly indicative of infection. Negative test results are highly accurate.

INTERFERENCE
FeLV vaccination will not interfere with test results, regardless of test method used.

IFA
Thrombocytopenia and/or leukopenia may cause false-negative test results. Poor slide quality, eosinophilia, and hemolysis may influence ability to accurately read stained slides.

ELISA
Hemolysis

Note
The 2008 AAFP Feline Retrovirus Management Guidelines make no stipulation that the IFA for FeLV Ag is the “confirmatory test” for cats with positive ELISA results. In fact, ELISA is more sensitive than IFA in detecting the presence of FeLV antigen.

FELINE IMMUNODEFICIENCY VIRUS ANTIBODY (FIV Ab)

NORMAL
Negative (negative test results indicate no prior FIV exposure).

PATIENT PREPARATION
None

COLLECT
Whole blood, 2.0 mL, in RTT

SUBMIT
Serum, 1.0 mL each for IFA (or ELISA) and Western blot analysis

INTERPRETATION
Cats with a positive test result by IFA (or ELISA) should be subjected to confirmatory testing by Western blot analysis.

INTERFERENCE
Any cat having received at least one inoculation with the FIV vaccine (killed, adjuvanted product) will produce interfering antibody that is detected by all commercially available FIV Ab tests (IFA, ELISA, Western blot analysis). False-positive test results are expected to persist for at least 1 year after vaccination. Currently, there is no test that will reliably and consistently distinguish between infected and vaccinated cats, including PCR. Also, kittens that have nursed from vaccinated queens are expected to have a false-positive test result for FIV antibody associated with maternally derived antibody. Duration of the false-positive results is unknown.

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Kittens younger than 6 months of age are expected to have maternally derived antibody if the queen is infected and may have a false-positive test result when tested by IFA or ELISA. Subsequent testing of positive kittens at 6 months of age or older is indicated to determine true infection status.

**Note:** The 2008 AAFP Feline Retrovirus Management Guidelines (available at www.catvets.com) stresses that healthy cats with a positive test result (by IFA or ELISA) may have false-positive test results, especially in populations in which the prevalence of infection is low. Confirmatory testing is indicated using the Western blot analysis. Negative test results are highly accurate.

**GIARDIA ANTIGEN**

**Normal**

Negative for antigen (dog and cat)

**Patient Preparation**

None

**Collect**

2 to 5 g of fresh feces

**Submit**

Entire sample in a sterile container; sample can be stored for 24 hours at 2° to 8° C. Frozen feces may be stored for slightly longer periods.

**Interpretation**

Test results are reported as either positive or negative for antigen; positive test results are expected in dogs or cats during active cyst and trophozoite shedding. The zoonotic potential of canine and feline giardiasis is controversial.

**Interference**

Extended or improper storage of the sample could result in false-negative results.

**HEARTWORM ANTIBODY, FELINE**

See also Heartworm Antigen, Feline.

**Normal**

Negative

**Patient Preparation**

None

**Collect**

Whole blood, 1.0 mL, in RTT

**Submit**

Serum, 1.0 mL

**Interpretation**

Test results should be interpreted in conjunction with a heartworm antigen test performed on the same sample. Because diagnostic confirmation of heartworm infection in cats is problematic (for several reasons), serologic test results must be considered in light of other laboratory and radiographic assessments.
A negative heartworm antibody (HWAb) test result suggests that there has been no exposure to *Dirofilaria immitis*. A negative result typically is used to rule out feline heartworm infection.

A positive HWAb test result only supports prior exposure. It does *not* confirm infection. Cats that are positive for HWAb should be subsequently tested for heartworm antigen (see later).

**INTERFERENCE**
Marked hemolysis or lipemia

**HEARTWORM ANTIGEN, FELINE**
See also *Heartworm Antibody, Feline.*

**NORMAL**
Negative

**PATIENT PREPARATION**
None

**COLLECT**
Whole blood, 2.0 mL, in an RTT

**SUBMIT**
Serum, 1.0 mL

**INTERPRETATION**
Test results should be interpreted in conjunction with an HWAb test performed on the same sample. Because diagnostic confirmation of heartworm infection in cats is problematic (for several reasons), serologic test results must be considered in light of other laboratory and radiographic assessments.

A negative heartworm antigen (HWAg) test result is not diagnostically useful; heartworm infection is still possible. A positive HWAg test result is highly specific; infection is likely.

**INTERFERENCE**
Marked hemolysis may cause a false-positive test result. A cat with only male heartworm infection will not have a positive result. Low worm burdens (common) may result in false-negative test results.

**HEARTWORM ANTIGEN, CANINE**

**NORMAL**
Negative

**PATIENT PREPARATION**
None

**COLLECT**
Whole blood, 2.0 mL, in RTT

**SUBMIT**
Serum, 1.0 mL

**INTERPRETATION**
A negative (HWAg) test result implies no infection; a positive HWAg test result strongly supports active infection.
Interference
Marked hemolysis may cause a false-positive test result. A dog with a low worm burden may have a false-negative test result. Note: The canine HWAg test may remain positive for up to 16 weeks after adulticide therapy.

Haemobartonella (feline infectious anemia; Mycoplasma haemofelis, Mycoplasma haemominutum)

Normal
Negative

Patient Preparation
None

Collect
Whole blood, 1.0 mL in EDTA (lavender-topped tube)

Submit
Submit entire sample

Interpretation
A positive test result is supportive of the diagnosis of infection; a negative test result implies no exposure.

Interference
Sample contamination; improper handing; extended storage times. Samples are stable for 48 hours if refrigerated at 2° to 8° C.

Leptospirosis Antibody Titer by Microscopic Agglutination Test (MAT)

Normal
Negative

Patient Preparation
None

Collect
Whole blood, 4.0 mL, in RTT

Submit
Serum, 2.0 mL

Interpretation
For dogs (and cats), laboratories in the United States typically provide titers for at least the following serogroups: Leptospira canicola, Leptospira icterohaemorrhagiae, Leptospira grippotyphosa, Leptospira pomona, Leptospira bratislava, and Leptospira autumnalis. A positive test result may indicate infection (high titers in the presence of clinical signs), prior exposure, or recent vaccination. The serogroup having the highest antibody MAT titer is generally considered to be the infecting serogroup (see Interference). A negative titer indicates no recent exposure or vaccination. Caution: Interpretation of Ab titer results for leptospirosis on the basis of a single serum sample in previously vaccinated dogs is difficult and may not be indicative of infection. A single positive antibody titer for any serovar in a healthy, recently vaccinated dog is not diagnostic for infection. Documentation of a rising titer, based on test results of two samples 3 to 4 weeks apart, is often recommended to confirm a diagnosis.
**INTERFERENCE**
Recent vaccination, regardless of the titer and the number of vaccine serovars administered, can result in higher-than-expected titers for multiple serogroups. Cross-reactivity within the MAT can be significant, leading to “positive” titer results for multiple serogroups on the same sample. In dogs with active leptospirosis, the serogroup having the highest MAT titer may not, in fact, be the infecting serogroup.

**Note:** When submitting samples for leptospirosis serology, it is important to provide information regarding date of last vaccination (if known), key clinical signs, and known laboratory abnormalities.

**LEPTOSPIROSIS BY RT-PCR**

**NORMAL**
Negative (dog)

**PATIENT PREPARATION**
None

**COLLECT**
4 mL fresh urine in a sterile container

**SUBMIT**
4 mL urine. Keep refrigerated. Urine specimen should be shipped promptly in a sealed container. Contact the laboratory regarding shipping instructions.

**INTERPRETATION**
Urine specimens for PCR for leptospirosis should be submitted at the same time as serum submitted for corresponding MAT. Test results will be interpreted by the laboratory.

**INTERFERENCE**
Sample contamination and test-related sensitivity or specificity can result in false-positive or false-negative results.

**LYME BORRELIOSIS (BORRELLIA BURGDORFERI)**

**QUALITATIVE C6 ANTIBODY BY ELISA (SNAP 3DX OR SNAP 4DX TEST)**

**NORMAL**
Negative (dog)

**Patient Preparation**
None

**Collect**
Venous blood, 1.0 mL, in a syringe or RTT (for submission)

**Submit**
Use collected sample for the point-of-care (SNAP) test. Submit a minimum of 0.5 mL of serum if test is being sent to a commercial laboratory.

**Interpretation**
A positive test result denotes exposure to *B. burgdorferi*; the presence of C6 antibody has a high correlation with infection. In dogs, infection is not always associated with clinical signs. The decision to treat or not to treat a healthy dog with a positive test result is based on the clinician’s assessment of the individual patient and supporting laboratory data.
Occasionally a dog will have a negative test result subsequent to treatment. However, this is an inconsistent finding. Use of the quantitative C6 antibody test to monitor response to treatment is recommended.

**Interference**
None; prior vaccination (regardless of vaccine used) will *not* cause false-positive test results.

**Protocol**
Sample may be submitted to a commercial laboratory or can be rapidly assessed in the hospital with a point-of-care (SNAP) test; follow manufacturer’s procedure outline.

*Note:* PCR testing of blood or serum for Lyme borreliosis is not recommended owing to the ability of spirochetes to reside, undetected, in tissue, resulting in a high number of false-negative test results.

**Quantitative C6 Antibody Test for Canine Lyme Disease**

**Normal**
Usually less than 30 antibody units (dog); refer to the laboratory reference range.

**Patient Preparation**
None

**Collect**
Venous blood, 2.0 mL, in RTT

**Submit**
Serum, 1.0 mL

**Interpretation**
Patients with antibody levels greater than 30 antibody units by the quantitative assay may be at risk of developing clinical disease. Patients with an initial positive test result that undergo treatment for Lyme borreliosis infection can be monitored for response (decline in antibody level) to treatment over time.

**Interference**
None; prior vaccination (regardless of vaccine used) will *not* cause false-positive test results.

**Protocol**
The quantitative test generally is indicated for patients that (1) have tested positive by the SNAP test and/or (2) are undergoing treatment for Lyme borreliosis.

**Borrelia Burgdorferi Antibody (IFA and Western Blot Analysis)**
The sensitivity and specificity data on C6 antibody support the recommendation that routine laboratory testing of patients suspected of having Lyme borreliosis be based on either the C6 antibody (SNAP test) or the quantitative C6 antibody test.

**Indirect Fluorescent Antibody (IFA); Also, Immunofluorescent Antibody**

**Normal**
Values vary among laboratories. A negative titer is normal and indicates no exposure to *B. burgdorferi* or recent vaccination.

*IDEXX Laboratories, Westbrook, Maine, United States.*
Patient Preparation
None

Collect
Whole blood, 2.0 mL, in RTT

Submit
Serum, 1.0 mL

Interpretation
Titers determined by IFA may not distinguish between vaccinated and infected dogs. Lyme borreliosis titers determined by IFA are indicated only in dogs that have not been vaccinated against Lyme disease. Lyme borreliosis vaccination can result in a positive titer. Dogs with a positive test result should be subjected to either the Western blot analysis or the quantitative C6 antibody test (preferred).

Interference
Prior vaccination against Lyme disease can result in a positive test result.

Western Blot Analysis
Normal
Results vary among laboratories. A negative test result is normal and indicates no exposure to B. burgdorferi.

Patient Preparation
None

Collect
Whole blood, 2.0 mL, in RTT

Submit
Serum, 1.0 mL

Interpretation
Titers may distinguish between vaccinated and infected dogs. Western blot analysis is indicated for dogs that have a positive IFA titer, as an alternative test.

Interference
Prior vaccination with a whole-cell, killed B. burgdorferi vaccine may complicate interpretation of the Western blot analysis.

Parvovirus Antibody IgG (Canine and Feline)

In Serum (IgG or IgM)
Normal
Any patient with a “positive” titer is considered to have protective immunity. Titer results do not distinguish prior exposure and recovery from vaccination. Uninfected dogs and cats have no evidence of a rising titer when results of the acute and convalescent titers are compared. Methodology (e.g., hemagglutination inhibition [HI], IFA) will affect actual values reported. Refer to laboratory reference range.

Patient Preparation
None
Collect
Whole blood, 2.0 mL

Submit
Serum, 1.0 mL

Interpretation
Actual test results may vary from one laboratory to another. Individual laboratories will provide interpretation information.

RABIES TITER BY FAVN (FLUORESCENT ANTIBODY VIRUS NEUTRALIZATION)

Samples may be submitted directly to:
Rabies Laboratory
Kansas State University
2005 Research Park Circle
Manhattan, KS 66502
USA.
785-532-4483
www.vet.ksu.edu/rabies

Normal
Provided by the Rabies Laboratory. Results determine whether or not a dog or cat has responded to rabies vaccination. Titer results are not generally acceptable by states as an index of immunity and cannot be used in lieu of local or state rabies vaccination requirements.

Patient Preparation
Rabies vaccine should not be administered less than 3 weeks before collection of blood to ensure maximal postvaccination response. Microchip identification number is requested when submitting samples.

Collect
Venous blood, 2.0 to 4.0 mL; allow blood to completely clot.

Submit
Serum (clear) 1.0 to 2.0 mL. Sample should be sent to the laboratory in a sealed, leakproof tube then placed inside a Ziploc bag. Ship in a padded box with dry ice or gel packs.

Important: An “FAVN Report Form,” provided online by the Rabies Laboratory, must accompany the sample. Entries on the form cannot be changed once the form has been submitted; check carefully for errors before shipping. Although overnight shipping is recommended, samples may be refrigerated and stored for up to 7 days. Do not ship to arrive on a weekend or holiday.

Interference
Gross hemolysis; lipemia; samples other than serum (e.g., plasma is not acceptable). Other causes for sample to be rejected include insufficient quantity of serum, bacterial contamination of sample, and unlabeled sample.

Note: FAVN rabies antibody results for dogs and cats are required by some rabies-free countries and regions before entry. Positive test results may be required before a dog or cat can leave the United States. Antibody titers determined by the RFFIT (see later), which measures rabies virus neutralizing antibody (RVNA), are not acceptable when dogs or cats are being exported.
RABBIES TITER BY RFFIT (RAPID FLUORESCENT FOCUS INHIBITION TEST FOR RABBIES VIRUS NEUTRALIZING ANTIBODY)

NORMAL

Rabies antibody titers determined by RFFIT cannot be interpreted as an index of immunity and are not used when exporting animals to rabies-free countries that require testing before exportation.

PATIENT PREPARATION

None

COLLECT

Whole blood, 4.0 mL, in RTT

SUBMIT

Serum, 2.0 mL (minimum is 500 µL), in a leakproof container (e.g., with screw-on cap). Place sample container inside a second container with gel packs or dry ice. Overnight shipping is recommended.

Note: Values for a “protective titer” in animals have not been established and will not be reported by the laboratory. The presence of RVNA in serum is indicative of an immune response to rabies but does not distinguish between antibody from vaccination and rabies virus exposure. RVNA levels are not to be used in place of current vaccination for either management of rabies exposure or for determination of booster vaccinations for animals.

INTERFERENCE

Gross hemolysis; lipemia; samples other than serum (e.g., plasma is not acceptable). Other causes for sample to be rejected include insufficient quantity of serum, bacterial contamination of sample, and unlabeled sample.

ROCKY MOUNTAIN SPOTTED FEVER (RMSF)

NORMAL

Negative (dog and cat)

PATIENT PREPARATION

None

COLLECT

Whole blood, 2.0 mL, in RTT

SUBMIT

Serum, 1.0 mL

INTERPRETATION

Generally, two samples are recommended (“acute” and “convalescent”), obtained 2 to 3 weeks apart. Titers reported vary among laboratories. The laboratory performing the titer will provide recommendations for interpreting results.

INTERFERENCE

None

TOXOPLASMOSIS TITERS (IgG AND IgM)

NORMAL

See Interpretation
Patient Preparation
None

Collect
Whole blood, 2.0 mL, in RTT

Submit
Serum, 1.0 mL

Interpretation
A positive titer denotes exposure, not active infection. IgG and IgM titers typically are reported individually. A titer greater than 1:256 for IgM is consistent with active infection in patients with clinical signs (e.g., pneumonia in cats, myositis in dogs). It is recommended that two samples for IgG titer be submitted; samples should be collected 2 to 3 weeks apart. A fourfold or greater rise in titer within 2 to 3 weeks is supportive of the diagnosis of active infection. Cats seropositive on a single titer are unlikely to be shedding oocysts.

Interference
Hemolysis; lipemia

Vaccine Titers
See listing under specific pathogen.

Laboratories providing vaccine titers typically limit these services to canine parvovirus, canine distemper, and feline panleukopenia. A limited number of laboratories offer titers for feline herpesvirus-1 and feline calicivirus.

Several university and commercial laboratories now provide antibody titers for selected canine and feline viruses as a means of assessing immunity derived from prior vaccination.

National laboratory standards for determining serum antibody titers against these pathogens have not been established. Because methods for performing titers vary among laboratories, titer results and ranges also can vary dramatically.

It is recommended that samples be analyzed by laboratories using the VN test (for canine distemper) and HI (for canine parvovirus and feline panleukopenia).

Note: A “positive” antibody titer usually will equate to “protective immunity.” A “negative” antibody titer does not necessarily equate to “susceptibility.”

Urine
An RTT is the preferred collection tube for urinalysis. A Copan swab can be used for urine culture, but this precludes quantitation of bacteria, if present. Urine for culture is best collected by cystocentesis and transported with a cold pack to prevent bacterial overgrowth.

Cortisol, Urine
See Urine Cortisol:Creatinine Ratio (UC:CR; Urinary C:C Ratio).

Microalbuminuria Test (Early Renal Disease [ERD] In-Hospital Test Kit)

Normal
Negative test strip indication (dog and cat)

Patient Preparation
None

Collect
2-mL (minimum) aliquot of urine in a clean container
Submit
Same

Interpretation
Test strip indicator grades the approximate degree of microalbuminuria. The manufacturer of the test kit provides recommendations for interpreting test results. However, it should be noted that a positive test result in clinically normal dogs is not known to be predictive of impending renal disease. In various studies, it has been shown that a significant percentage of healthy dogs and certain breeds (soft-coated Wheaten Terriers) will have positive test results. Until more information is available about the clinical utility of this test, its use should be restricted to monitoring urine protein loss in patients with known or suspected glomerular disease.

Interference
Blood contamination of urine sample

Urine Cortisol:Creatinine Ratio (UC:Cr, Urinary C:C Ratio)

Normal
Varies according to individual laboratory and test methodology used (dog and cat)

Patient Preparation
Owner should collect urine at home on the day (morning is preferable) that the test is submitted, thereby reducing stress-induced artifact.

Collect
3.0- to 5.0-mL aliquot of pooled urine in a sterile container

Submit
Same; sample should be refrigerated during transport to the laboratory.

Interpretation
The UC:Cr is reported to have high sensitivity (negative predictive value) and therefore has been recommended to rule out the diagnosis of hyperadrenocorticism in dogs.

Controversy exists regarding the diagnostic value of the UC:Cr to diagnose canine Cushing syndrome. Reference values for the cat are not reported. The test currently is not recommended as a single diagnostic test. In serial UC:Cr studies performed in hyperthyroid cats, elevated ratios were observed; successful treatment (medical and surgical) did result in a significant decrease in UC:Cr in cats.

Interference
The effect of urine collected from hospitalized dogs (stress) versus urine collected from dogs at home remains an arguable variable. Owners should be advised to collect urine at home on the scheduled day of examination and testing.

Protocol
Instruct the owner to collect urine in a single, clean container over 2 consecutive hours on the same day that the urine sample is to be submitted to the laboratory. A 3.0- to 5.0-mL aliquot of pooled urine is submitted for analysis. Note: Not all commercial laboratories offer this test. Check before submitting.

Urine Protein-Creatinine Ratio (UP:Cr; P:Cr; UPC)

Normal
Ratio <0.3 (dog); ratio <0.6 (cat)

Patient Preparation
None
**Collect**
2- to 3-mL aliquot of randomly collected urine in a clean container

**Submit**
Same

**Interpretation**
UP:Cr greater than 1.0 is consistent with the diagnosis of pathologic proteinuria. The ratio does not confirm the source of the protein loss. However, in patients with consistent hypoalbuminemia and significantly elevated urine P:Cr, loss of protein through the glomerulus is likely (e.g., glomerulonephritis).

**Interference**
Blood contamination (cystitis, cystocentesis)
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<thead>
<tr>
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<th>Phone Number and Web Address</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Need</strong></td>
<td><strong>Agency</strong></td>
</tr>
<tr>
<td>To obtain information regarding the treatment of a known or suspected poisoning or toxicosis case</td>
<td>American Society for the Prevention of Cruelty to Animals (ASPCA) Animal Poison Control Center. A $65 fee for service may apply.</td>
</tr>
<tr>
<td>To obtain information regarding the treatment of a known or suspected poisoning or toxicosis case</td>
<td>Pet Poison Helpline Available 24 hours; a $35 fee is charged per case.</td>
</tr>
</tbody>
</table>

Continued
<table>
<thead>
<tr>
<th>Need</th>
<th>Agency</th>
<th>Phone Number and Web Address</th>
</tr>
</thead>
<tbody>
<tr>
<td>To report known or suspected adverse drug (not vaccine) reactions</td>
<td>Food and Drug Administration (FDA), Center for Veterinary Medicine (CVM)</td>
<td>888-332-8387 (voice messages accepted)</td>
</tr>
<tr>
<td></td>
<td></td>
<td><a href="http://www.fda.gov">www.fda.gov</a> Search: animal and veterinary</td>
</tr>
<tr>
<td>To report shortages of medically necessary veterinary drugs</td>
<td>Food and Drug Administration (FDA), Center for Veterinary Medicine (CVM)</td>
<td>240-276-9239</td>
</tr>
<tr>
<td>To report known or suspected adverse vaccine reactions</td>
<td>U.S. Dept of Agriculture (USDA), Center for Veterinary Biologics It is recommended to contact the vaccine manufacturer directly before contacting the USDA. Note: This is for reporting purposes only.</td>
<td>800-752-6255 <a href="http://www.aphis.usda.gov">www.aphis.usda.gov</a> Search: vaccine adverse event</td>
</tr>
<tr>
<td>For inquiries regarding transfusion medicine</td>
<td>Animal Blood Bank hotline (no charge to caller)</td>
<td>800-243-5759 (24-hour) <a href="http://www.animalbloodbank.com">www.animalbloodbank.com</a></td>
</tr>
<tr>
<td>For inquiries regarding transfusion medicine and purchase of blood and blood components</td>
<td>Eastern Veterinary Blood Bank</td>
<td>800-949-3822 (24-hour) <a href="http://www.evbb.com">www.evbb.com</a></td>
</tr>
<tr>
<td>For inquiries regarding transfusion medicine—a full-service, nonprofit blood bank and educational network for animal treatment</td>
<td>HEMOPET</td>
<td>714-891-2022 (24-hour) <a href="http://www.hemopet.org">www.hemopet.org</a></td>
</tr>
<tr>
<td>Access to a commercial blood bank and purchase of blood and blood components</td>
<td>Veterinarians’ Blood Bank</td>
<td>812-358-8500 <a href="http://www.vetbloodbank.com">www.vetbloodbank.com</a></td>
</tr>
<tr>
<td>For inquiries regarding pesticides, pesticide products, poisonings, and toxicities</td>
<td>National Pesticide Information Center</td>
<td>800-858-7378</td>
</tr>
<tr>
<td>To contact the Office of Diversion Control of the DEA</td>
<td>Drug Enforcement Administration (DEA)</td>
<td>800-882-9539</td>
</tr>
<tr>
<td></td>
<td></td>
<td><a href="http://www.dea.gov">www.dea.gov</a></td>
</tr>
</tbody>
</table>

www.ajlobby.com
The American Kennel Club (AKC) currently recognizes 150 dog breeds, each of which is assigned to one of seven breed groups. The AKC maintains an excellent website that offers considerable information on individual breeds (www.akc.org/breeds/index.cfm).

**Sporting group**
- American Water Spaniel
- Boykin Spaniel
- Brittany
- Chesapeake Bay Retriever
- Clumber Spaniel
- Cocker Spaniel
- Curly-Coated Retriever
- English Cocker Spaniel
- English Setter
- English Springer Spaniel
- Field Spaniel
- Flat-Coated Retriever
- German Shorthaired Pointer
- German Wirehaired Pointer
- Golden Retriever
- Gordon Setter
- Irish Red and White Setter
- Irish Setter
- Irish Water Spaniel
- Labrador Retriever
- Nova Scotia Duck Tolling Retriever
- Pointer
- Spinone Italiano
- Sussex Spaniel
- Vizsla
- Weimaraner
- Welsh Springer Spaniel
- Wirehaired Pointing Griffon

**Hound group**
- Afghan Hound
- American Foxhound
- Basenji
- Basset Hound
- Beagle
- Black and Tan Coonhound
- Bloodhound
- Bluetick Coonhound
- Borzoi
- Dachshund
- English Foxhound
- Greyhound
- Harrier
- Ibizan Hound
- Irish Wolfhound
- Norwegian Elkhound
- Otterhound
- Petit Basset Griffon Vendéen
- Pharaoh Hound
- Plott
- Redbone Coonhound
- Rhodesian Ridgeback
- Saluki
- Scottish Deerhound
- Whippet

**Working group**
- Akita
- Alaskan Malamute
- Anatolian Shepherd Dog
- Bernese Mountain Dog
- Black Russian Terrier
- Boxer
- Bullmastiff
- Cane Corso
- Doberman Pinscher
- Dogue de Bordeaux
- German Pinscher
- Giant Schnauzer
- Great Dane
- Great Pyrenees
- Greater Swiss Mountain Dog
- Komondor
- Kuvasz
- Leonberger
- Mastiff
- Neapolitan Mastiff
- Newfoundland
- Portuguese Water Dog
- Rottweiler
- Saint Bernard
- Samoyed
- Siberian Husky
- Standard Schnauzer
- Tibetan Mastiff

**Terrier group**
- Airedale Terrier
- American Staffordshire Terrier
- Australian Terrier
- Bedlington Terrier
- Border Terrier
- Bull Terrier
- Cairn Terrier
- Dandie Dinmont Terrier
- Glen of Imaal Terrier
- Irish Terrier
- Kerry Blue Terrier
- Lakeland Terrier
- Manchester Terrier
- Miniature Bull Terrier
- Miniature Schnauzer
- Norfolk Terrier

---

Continued
### Table 6-2: Dog Breeds Recognized by The American Kennel Club (AKC)—Cont’d

<table>
<thead>
<tr>
<th>Toy Group</th>
<th>Herding Group</th>
<th>Miscellaneous Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>Norwich Terrier</td>
<td>Norwegian Lundehund</td>
<td>American Eskimo Dog</td>
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<tr>
<td>Parson Russell Terrier</td>
<td>Poodle</td>
<td>Bergamasco</td>
</tr>
<tr>
<td>Scottish Terrier</td>
<td>Schipperke</td>
<td>Boerboel</td>
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<tr>
<td>Sealyham Terrier</td>
<td>Shiba Inu</td>
<td>Cesky Terrier</td>
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<tr>
<td>Skye Terrier</td>
<td>Tibetan Spaniel</td>
<td>Chinook</td>
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<tr>
<td>Smooth Fox Terrier</td>
<td>Tibetan Terrier</td>
<td>Dogo Argentino</td>
</tr>
<tr>
<td>Soft Coated Wheaten Terrier</td>
<td>Xoloitzcuintli</td>
<td>Finnish Lapphund</td>
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<tr>
<td>Staffordshire Bull Terrier</td>
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<td>Peruvian Inca</td>
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<tr>
<td>Welsh Terrier</td>
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<td>Rat Terrier</td>
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<tr>
<td>West Highland White Terrier</td>
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<td>Russell Terrier</td>
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<tr>
<td>Wire Fox Terrier</td>
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<td>Sloughi</td>
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<tr>
<td></td>
<td><strong>Toy Group</strong></td>
<td><strong>Treeing Walker Coonhound</strong></td>
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<tr>
<td></td>
<td></td>
<td><strong>Wirehaired Vizsla</strong></td>
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<tr>
<td>Affenpinscher</td>
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<tr>
<td>Brussels Griffon</td>
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<td>Cavalier King Charles Spaniel</td>
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<td>Chihuahua</td>
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<td>Chinese Crested</td>
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<td>English Toy Spaniel</td>
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<td>Italian Greyhound</td>
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<td>Japanese Chin</td>
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<tr>
<td>Maltese</td>
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<tr>
<td>Manchester Terrier (Toy)</td>
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<td>Miniature Pinscher</td>
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<td>Papillon</td>
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<td>Pekingese</td>
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<td>Pomeranian</td>
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<td>Poodle</td>
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<tr>
<td>Pug</td>
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<tr>
<td>Shih Tzu</td>
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<tr>
<td>Silky Terrier</td>
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<tr>
<td>Toy Fox Terrier</td>
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<td>Yorkshire Terrier</td>
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<td></td>
<td><strong>Herding Group</strong></td>
<td><strong>American English Coonhound</strong></td>
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<tr>
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<td><strong>Bergamasco</strong></td>
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<td><strong>Boerboel</strong></td>
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<td><strong>Cesky Terrier</strong></td>
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<td><strong>Chinook</strong></td>
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<td><strong>Dogo Argentino</strong></td>
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<td><strong>Finnish Lapphund</strong></td>
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<td><strong>Peruvian Inca</strong></td>
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<td><strong>Rat Terrier</strong></td>
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<td><strong>Russell Terrier</strong></td>
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<td><strong>Sloughi</strong></td>
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<td></td>
<td><strong>Treeing Walker Coonhound</strong></td>
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<td><strong>Wirehaired Vizsla</strong></td>
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</table>
The Cat Fanciers’ Association (CFA) presently recognizes 41 pedigreed breeds for showing in the Championship Class. For additional information on individual breeds, see the CFA website, www.cfa.org (Search: Breeds)

**CHAMPIONSHIP CLASS**

<table>
<thead>
<tr>
<th>Breed</th>
<th>Breed</th>
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<tbody>
<tr>
<td>Abyssinian</td>
<td>LaPerm</td>
</tr>
<tr>
<td>American Bobtail</td>
<td>Maine Coon</td>
</tr>
<tr>
<td>American Curl</td>
<td>Manx</td>
</tr>
<tr>
<td>American Shorthair</td>
<td>Norwegian Forest Cat</td>
</tr>
<tr>
<td>American Wirehair</td>
<td>Oicat</td>
</tr>
<tr>
<td>Balinese (including Javanese)</td>
<td>Oriental</td>
</tr>
<tr>
<td>Birman</td>
<td>Persian</td>
</tr>
<tr>
<td>Bombay</td>
<td>RagaMuffin</td>
</tr>
<tr>
<td>British Shorthair</td>
<td>Ragdoll</td>
</tr>
<tr>
<td>Burmese</td>
<td>Russian Blue</td>
</tr>
<tr>
<td>Chartreux</td>
<td>Scottish Fold</td>
</tr>
<tr>
<td>Chinese Li Hua</td>
<td>Selkirk Rex</td>
</tr>
<tr>
<td>Colorpoint Shorthair</td>
<td>Siamese</td>
</tr>
<tr>
<td>Cornish Rex</td>
<td>Siberian</td>
</tr>
<tr>
<td>Devon Rex</td>
<td>Singapura</td>
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<tr>
<td>Egyptian Mau</td>
<td>Somali</td>
</tr>
<tr>
<td>European Burmese</td>
<td>Sphynx</td>
</tr>
<tr>
<td>Exotic</td>
<td>Tonkinese</td>
</tr>
<tr>
<td>Havana Brown</td>
<td>Turkish Angora</td>
</tr>
<tr>
<td>Japanese Bobtail</td>
<td>Turkish Van</td>
</tr>
<tr>
<td>Korat</td>
<td></td>
</tr>
<tr>
<td>TABLE 6-4</td>
<td>Useful Information for Rodents and Rabbits</td>
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<tr>
<td>-----------</td>
<td>-------------------------------------------</td>
</tr>
<tr>
<td></td>
<td>Hamster</td>
</tr>
<tr>
<td>Weight at birth</td>
<td>2 g (F) 28-31 days (M) 45 days (best to breed at 70 days)</td>
</tr>
<tr>
<td>Puberty</td>
<td>(F) 28-31 days (M) 45 days (best to breed at 70 days)</td>
</tr>
<tr>
<td>Duration of estrous cycle*</td>
<td>4 days</td>
</tr>
<tr>
<td>Gestation (days)</td>
<td>16</td>
</tr>
<tr>
<td>Separation of adults during parturition and weaning</td>
<td>Yes</td>
</tr>
<tr>
<td>Number per litter</td>
<td>4-10</td>
</tr>
<tr>
<td>Eyes open</td>
<td>15 days</td>
</tr>
<tr>
<td>Wean at</td>
<td>25 days</td>
</tr>
<tr>
<td>Postpartum estrus</td>
<td>Within 24 hr</td>
</tr>
<tr>
<td>Breeding life</td>
<td>11-18 mo</td>
</tr>
<tr>
<td>Adult weight</td>
<td>(F) 120 g (M) 108 g</td>
</tr>
<tr>
<td>Life span (yr)</td>
<td>2-3</td>
</tr>
<tr>
<td>Body temperature</td>
<td>97°-101° F (36.1°-38.3° C)</td>
</tr>
<tr>
<td>Daily adult water consumption</td>
<td>8-12 mL/day</td>
</tr>
<tr>
<td>------------------------------</td>
<td>-------------</td>
</tr>
<tr>
<td>Daily adult food consumption (varies with age and condition)</td>
<td>7-12 g/day</td>
</tr>
<tr>
<td>Diet</td>
<td>Commercial rat, mouse, or hamster chow supplemented with kale,† cabbage,† apples, milk</td>
</tr>
<tr>
<td>Room temperature</td>
<td>65°-75° F (18.3°-24° C)</td>
</tr>
<tr>
<td>Humidity (%)</td>
<td>50</td>
</tr>
</tbody>
</table>


†All species listed except rabbits are seasonally polyestrous.

†Better source of vitamin C than lettuce.
## Table 6-5: Determination of the Sex of Mature and Immature Rodents and Rabbits

<table>
<thead>
<tr>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mature hamsters, mice, rats, guinea pigs, and gerbils</strong></td>
<td></td>
</tr>
<tr>
<td>Anogenital distance longer in the male.</td>
<td></td>
</tr>
<tr>
<td>Manipulate “genital papilla” (prepuce) to the protrude penis.</td>
<td></td>
</tr>
<tr>
<td>Palpate for testicles either in a scrotal sac (if present) or subcutaneous in inguinal region.</td>
<td></td>
</tr>
<tr>
<td>Males have only two external openings in the inguinal area:</td>
<td></td>
</tr>
<tr>
<td>a. Anus</td>
<td></td>
</tr>
<tr>
<td>b. Urethral orifice at tip of penis In very fat males there may be a depression between the penis and anus. This depression can be obliterated by manipulating the skin in this area.</td>
<td></td>
</tr>
<tr>
<td>Anogenital distance shorter in the female.</td>
<td></td>
</tr>
<tr>
<td>Look for the three external openings in inguinal area:</td>
<td></td>
</tr>
<tr>
<td>a. Anus (most caudal opening)</td>
<td></td>
</tr>
<tr>
<td>b. Vaginal orifice (middle opening)—look carefully</td>
<td></td>
</tr>
<tr>
<td>c. Urethral orifice at tip of urethral papilla (most anterior opening). In these animals the urethral papilla is located outside the vagina (unlike in dogs and cats). In very fat or young females, the vaginal orifice may be either hidden by folds of skin (the former) or sealed (latter). Gentle manipulation of the skin in this area will divulge the orifice.</td>
<td></td>
</tr>
<tr>
<td><strong>Mature rabbits</strong></td>
<td></td>
</tr>
<tr>
<td>Protrude penis by manipulating skin of prepuce.</td>
<td></td>
</tr>
<tr>
<td>Palpate for testicles.</td>
<td></td>
</tr>
<tr>
<td>Anogenital distance is longer in males.</td>
<td></td>
</tr>
<tr>
<td>There is a common orifice for both the vagina and urethra (as in dogs and cats).</td>
<td></td>
</tr>
<tr>
<td>No structure like a “penis” can be protruded from the urogenital orifice.</td>
<td></td>
</tr>
<tr>
<td>Anogenital distance is shorter in females.</td>
<td></td>
</tr>
</tbody>
</table>

**Table 6-6** Blood Values and Serum Chemical Constituents for Rodents and Rabbits

<table>
<thead>
<tr>
<th>Laboratory Test</th>
<th>Rats</th>
<th>Mice</th>
<th>Hamsters</th>
<th>Guinea Pigs</th>
<th>Rabbits</th>
<th>Mongolian Gerbils</th>
</tr>
</thead>
<tbody>
<tr>
<td>AST (Sigma-Frankel units)</td>
<td>25-42</td>
<td>32-41</td>
<td>22-36</td>
<td>10-25</td>
<td>14-27</td>
<td>—</td>
</tr>
<tr>
<td>Alkaline phosphatase (Bodansky units)</td>
<td>4.1-8.6</td>
<td>2.4-4.0</td>
<td>2.0-3.5</td>
<td>1.5-8.1</td>
<td>2.1-3.2</td>
<td>—</td>
</tr>
<tr>
<td>BUN (mg/dL)</td>
<td>10-20</td>
<td>8-30</td>
<td>10-40</td>
<td>8-20</td>
<td>5-30</td>
<td>18-24</td>
</tr>
<tr>
<td>Sodium (mEq/L)</td>
<td>144</td>
<td>114-154</td>
<td>106-185</td>
<td>120-155</td>
<td>100-145</td>
<td>144-158</td>
</tr>
<tr>
<td>Potassium (mEq/L)</td>
<td>5.9</td>
<td>3.0-9.6</td>
<td>2.3-9.8</td>
<td>6.5-8.2</td>
<td>3.0-7.0</td>
<td>3.8-5.2</td>
</tr>
<tr>
<td>Bilirubin, total (mg/dL)</td>
<td>0.42</td>
<td>0.18-0.54</td>
<td>0.3-0.4</td>
<td>0.24-0.30</td>
<td>0.15-0.20</td>
<td>—</td>
</tr>
<tr>
<td>Blood glucose (mg/dL)</td>
<td>50-115</td>
<td>108-192</td>
<td>32.6-118.4</td>
<td>60-125</td>
<td>50-140</td>
<td>69-119</td>
</tr>
<tr>
<td>RBCs (10⁶ cells/mm³)</td>
<td>7.2-9.6</td>
<td>9.3-10.5</td>
<td>4.0-9.3</td>
<td>4.5-7.0</td>
<td>3.2-7.5</td>
<td>8.3-9.3</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>14.8</td>
<td>12-14.9</td>
<td>9.7-16.8</td>
<td>11-15</td>
<td>10-15</td>
<td>10-16</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>40-50</td>
<td>35-50</td>
<td>40-52</td>
<td>35-50</td>
<td>35-45</td>
<td>35-45</td>
</tr>
<tr>
<td>WBCs (10³ cells/mm³)</td>
<td>8-14</td>
<td>8-14</td>
<td>7-15</td>
<td>5-12</td>
<td>8-10</td>
<td>9-14</td>
</tr>
<tr>
<td>Segmented (%)</td>
<td>30</td>
<td>26</td>
<td>16-28</td>
<td>42</td>
<td>30-50</td>
<td>10-20</td>
</tr>
<tr>
<td>Nonsegmented (%)</td>
<td>0</td>
<td>0</td>
<td>8</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Lymphocytes (%)</td>
<td>65-77</td>
<td>55-80</td>
<td>64-78</td>
<td>45-81</td>
<td>30-50</td>
<td>70-89</td>
</tr>
<tr>
<td>Eosinophils (%)</td>
<td>1</td>
<td>3</td>
<td>1</td>
<td>5</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Monocytes (%)</td>
<td>4</td>
<td>5</td>
<td>2</td>
<td>8</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>Basophils (%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>


AST, Aspartate aminotransferase; BUN, blood urea nitrogen; RBCs, red blood cells; WBCs, white blood cells.

*These are values found in healthy-appearing animals and can be used as guides but should not be interpreted as physiologic norms for the species listed.
### TABLE 6-7  Ferrets—Physiologic, Anatomic, and Reproductive Data

<table>
<thead>
<tr>
<th>Data</th>
<th>Range or Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Physiologic data</strong></td>
<td></td>
</tr>
<tr>
<td>Life span</td>
<td>5-9 yr (average 5-7)</td>
</tr>
<tr>
<td>Commercial breeding life</td>
<td>2-5 yr</td>
</tr>
<tr>
<td>Body temperature</td>
<td>101°-104° F (38°-40° C)</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>32-36 breaths/min</td>
</tr>
<tr>
<td>Heart rate</td>
<td>220-250 beats/min (average 240)</td>
</tr>
<tr>
<td>Water consumption</td>
<td>75-100 mL/day</td>
</tr>
<tr>
<td>Chromosome number</td>
<td>2n = 40</td>
</tr>
<tr>
<td><strong>Anatomic data</strong></td>
<td></td>
</tr>
<tr>
<td>Dental formula</td>
<td>2 (I3/3, C1/1, P3/4, M1/2)</td>
</tr>
<tr>
<td>Vertebral formula</td>
<td>C-7, T-14, L-6, S-3, Cd-14–Cd-18</td>
</tr>
<tr>
<td><strong>Reproductive data</strong></td>
<td></td>
</tr>
<tr>
<td>Gestation</td>
<td>39-46 days (average 42)</td>
</tr>
<tr>
<td>Litter size</td>
<td>2-17 kits (average 8)</td>
</tr>
<tr>
<td>False pregnancy</td>
<td>40-42 days</td>
</tr>
<tr>
<td>Placentation</td>
<td>Zonal</td>
</tr>
<tr>
<td>Implantation time</td>
<td>12-31 days</td>
</tr>
<tr>
<td>Weaning</td>
<td>5-6 wk</td>
</tr>
<tr>
<td>Ovulation</td>
<td>30-40 hr postcoitus</td>
</tr>
</tbody>
</table>


### TABLE 6-8  Hematologic Values for Normal Ferrets*  

<table>
<thead>
<tr>
<th>Laboratory Test</th>
<th>Mean</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hematocrit (%)</td>
<td>52.3</td>
<td>42-61</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>17.0</td>
<td>15-18</td>
</tr>
<tr>
<td>RBCs (10⁶ cells/mm³)</td>
<td>9.17</td>
<td>6.8-12.2</td>
</tr>
<tr>
<td>WBCs (10³ cells/mm³)</td>
<td>10.1</td>
<td>4.0-19</td>
</tr>
</tbody>
</table>

**WBCs**

<table>
<thead>
<tr>
<th>Lab Test</th>
<th>Mean</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphocytes (%)</td>
<td>34.5</td>
<td>12-54</td>
</tr>
<tr>
<td>Neutrophils (%)</td>
<td>58.3</td>
<td>11-84</td>
</tr>
<tr>
<td>Monocytes (%)</td>
<td>4.4</td>
<td>0-9.0</td>
</tr>
<tr>
<td>Eosinophils (%)</td>
<td>2.5</td>
<td>0-7.0</td>
</tr>
<tr>
<td>Basophils (%)</td>
<td>0.1</td>
<td>0-2.0</td>
</tr>
<tr>
<td>Reticulocytes (%)</td>
<td>4.6</td>
<td>1-14</td>
</tr>
<tr>
<td>Platelets (10³ cells/mm³)</td>
<td>499</td>
<td>297-910</td>
</tr>
<tr>
<td>Total protein (g/dL)</td>
<td>6.0</td>
<td>5.1-7.4</td>
</tr>
</tbody>
</table>


*RBCs, Red blood cells; WBCs, white blood cells.

*Values are for both sexes.*
**TABLE 6 - 9  Serum Chemistry Values for Normal Ferrets**

<table>
<thead>
<tr>
<th>Analyte</th>
<th>Unit</th>
<th>Mean</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>mg/dL</td>
<td>136</td>
<td>94-207</td>
</tr>
<tr>
<td>BUN</td>
<td>mg/dL</td>
<td>22</td>
<td>10-45</td>
</tr>
<tr>
<td>Albumin</td>
<td>mg/dL</td>
<td>3.2</td>
<td>2.3-3.8</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>International units/L</td>
<td>23</td>
<td>9-84</td>
</tr>
<tr>
<td>AST</td>
<td>International units/L</td>
<td>65</td>
<td>28-120</td>
</tr>
<tr>
<td>Total bilirubin</td>
<td>mg/dL</td>
<td>&lt;1.0</td>
<td></td>
</tr>
<tr>
<td>Cholesterol</td>
<td>mg/dL</td>
<td>165</td>
<td>64-296</td>
</tr>
<tr>
<td>Creatinine</td>
<td>mg/dL</td>
<td>0.6</td>
<td>0.4-0.9</td>
</tr>
<tr>
<td>Sodium</td>
<td>mEq/L</td>
<td>148</td>
<td>137-162</td>
</tr>
<tr>
<td>Potassium</td>
<td>mEq/L</td>
<td>5.9</td>
<td>4.5-7.7</td>
</tr>
<tr>
<td>Chloride</td>
<td>mEq/L</td>
<td>116</td>
<td>106-125</td>
</tr>
<tr>
<td>Calcium</td>
<td>mg/dL</td>
<td>9.2</td>
<td>8.0-11.8</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>mg/dL</td>
<td>5.9</td>
<td>4.0-9.1</td>
</tr>
</tbody>
</table>


AST, Aspartate aminotransferase; BUN, blood urea nitrogen.

*Values for both sexes.

**TABLE 6 - 10  Electrocardiographic Data for Normal Ferrets**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate rhythm</td>
<td>224 ± 51</td>
<td>150-340 bpm</td>
</tr>
<tr>
<td><strong>Measurements</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P wave</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Width</td>
<td>0.03 ± 0.009</td>
<td>0.015-0.04 s</td>
</tr>
<tr>
<td>Height</td>
<td>0.106 ± 0.03</td>
<td>0.05-0.20 mV</td>
</tr>
<tr>
<td>PR interval</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Width</td>
<td>0.05 ± 0.01</td>
<td>0.04-0.08 s</td>
</tr>
<tr>
<td>QRS complex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q wave</td>
<td>Usually none</td>
<td></td>
</tr>
<tr>
<td>R wave</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Width</td>
<td>0.049 ± 0.008</td>
<td>0.04-0.06 s</td>
</tr>
<tr>
<td>Height</td>
<td>1.59 ± 0.63</td>
<td>0.6-3.15 mV</td>
</tr>
<tr>
<td>S wave</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height</td>
<td>0.166 ± 0.101</td>
<td>0.1-0.25 mV</td>
</tr>
<tr>
<td>ST segment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Width</td>
<td>0.030 ± 0.016</td>
<td>0.01-0.06 s</td>
</tr>
<tr>
<td>QT interval</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Width</td>
<td>0.13 ± 0.027</td>
<td>0.10-0.18 s</td>
</tr>
<tr>
<td>T wave</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Width</td>
<td>0.06 ± 0.01</td>
<td>0.03-0.1 s</td>
</tr>
<tr>
<td>Height</td>
<td>0.24 ± 0.12</td>
<td>0.10-0.45 mV</td>
</tr>
<tr>
<td>Mean electrical axis (frontal plane)</td>
<td></td>
<td>+65-100 degrees</td>
</tr>
</tbody>
</table>

*Ferrets in right lateral recumbency; sedation with ketamine and xylazine. bpm, Beats per minute; s, seconds; mV, millivolts.
### Table 6-11: Conversion of Body Weight in Kilograms to Body Surface Area in Square Meters for Dogs

<table>
<thead>
<tr>
<th>Kilograms</th>
<th>Square Meters</th>
<th>Kilograms</th>
<th>Square Meters</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.50</td>
<td>0.06</td>
<td>26.00</td>
<td>0.88</td>
</tr>
<tr>
<td>1.00</td>
<td>0.10</td>
<td>27.00</td>
<td>0.90</td>
</tr>
<tr>
<td>2.00</td>
<td>0.15</td>
<td>28.00</td>
<td>0.92</td>
</tr>
<tr>
<td>3.00</td>
<td>0.20</td>
<td>29.00</td>
<td>0.94</td>
</tr>
<tr>
<td>4.00</td>
<td>0.25</td>
<td>30.00</td>
<td>0.96</td>
</tr>
<tr>
<td>5.00</td>
<td>0.29</td>
<td>31.00</td>
<td>0.99</td>
</tr>
<tr>
<td>6.00</td>
<td>0.33</td>
<td>32.00</td>
<td>1.01</td>
</tr>
<tr>
<td>7.00</td>
<td>0.36</td>
<td>33.00</td>
<td>1.03</td>
</tr>
<tr>
<td>8.00</td>
<td>0.40</td>
<td>34.00</td>
<td>1.05</td>
</tr>
<tr>
<td>9.00</td>
<td>0.43</td>
<td>35.00</td>
<td>1.07</td>
</tr>
<tr>
<td>10.00</td>
<td>0.46</td>
<td>36.00</td>
<td>1.09</td>
</tr>
<tr>
<td>11.00</td>
<td>0.49</td>
<td>37.00</td>
<td>1.11</td>
</tr>
<tr>
<td>12.00</td>
<td>0.52</td>
<td>38.00</td>
<td>1.13</td>
</tr>
<tr>
<td>13.00</td>
<td>0.55</td>
<td>39.00</td>
<td>1.15</td>
</tr>
<tr>
<td>14.00</td>
<td>0.58</td>
<td>40.00</td>
<td>1.17</td>
</tr>
<tr>
<td>15.00</td>
<td>0.60</td>
<td>41.00</td>
<td>1.19</td>
</tr>
<tr>
<td>16.00</td>
<td>0.63</td>
<td>42.00</td>
<td>1.21</td>
</tr>
<tr>
<td>17.00</td>
<td>0.66</td>
<td>43.00</td>
<td>1.23</td>
</tr>
<tr>
<td>18.00</td>
<td>0.69</td>
<td>44.00</td>
<td>1.25</td>
</tr>
<tr>
<td>19.00</td>
<td>0.71</td>
<td>45.00</td>
<td>1.26</td>
</tr>
<tr>
<td>20.00</td>
<td>0.74</td>
<td>46.00</td>
<td>1.28</td>
</tr>
<tr>
<td>21.00</td>
<td>0.76</td>
<td>47.00</td>
<td>1.30</td>
</tr>
<tr>
<td>22.00</td>
<td>0.78</td>
<td>48.00</td>
<td>1.32</td>
</tr>
<tr>
<td>23.00</td>
<td>0.81</td>
<td>49.00</td>
<td>1.34</td>
</tr>
<tr>
<td>24.00</td>
<td>0.83</td>
<td>50.00</td>
<td>1.36</td>
</tr>
<tr>
<td>25.00</td>
<td>0.85</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 6-12: Conversion of Body Weight in Kilograms to Body Surface Area in Square Meters for Cats

<table>
<thead>
<tr>
<th>Kilograms</th>
<th>Square Meters</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.50</td>
<td>0.06</td>
</tr>
<tr>
<td>1.00</td>
<td>0.10</td>
</tr>
<tr>
<td>1.50</td>
<td>0.12</td>
</tr>
<tr>
<td>2.00</td>
<td>0.15</td>
</tr>
<tr>
<td>2.50</td>
<td>0.17</td>
</tr>
<tr>
<td>3.00</td>
<td>0.20</td>
</tr>
<tr>
<td>3.50</td>
<td>0.22</td>
</tr>
<tr>
<td>4.00</td>
<td>0.24</td>
</tr>
<tr>
<td>4.50</td>
<td>0.26</td>
</tr>
<tr>
<td>5.00</td>
<td>0.28</td>
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<td>0.39</td>
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<tr>
<td>9.00</td>
<td>0.41</td>
</tr>
<tr>
<td>9.50</td>
<td>0.42</td>
</tr>
<tr>
<td>10.00</td>
<td>0.44</td>
</tr>
</tbody>
</table>
The standard French, or Charrière, scale (abbreviated F or Fr) is generally used in the size calibration of catheters and other tubular instruments. It is based on the metric system, with each unit being approximately 0.33 mm, with a difference of 0.33 mm in diameter between consecutive sizes. Example: 27F indicates a diameter of 9 mm; 30F, a diameter of 10 mm. A convenient conversion table from the French scale to the English and American scales that is sometimes used for certain instruments is given here.

<table>
<thead>
<tr>
<th>1 mm</th>
<th>2 mm</th>
<th>3 mm</th>
<th>4 mm</th>
<th>5 mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>8</td>
<td>9</td>
<td>10</td>
<td>11</td>
<td>12</td>
</tr>
<tr>
<td>13</td>
<td>14</td>
<td>15</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6 mm</th>
<th>8 mm</th>
<th>10 mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>16</td>
<td>17</td>
<td>18</td>
</tr>
<tr>
<td>19</td>
<td>20</td>
<td>22</td>
</tr>
<tr>
<td>24</td>
<td>26</td>
<td>28</td>
</tr>
<tr>
<td>30</td>
<td>32</td>
<td>34</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>12 mm</th>
<th>15 mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>38</td>
<td>36</td>
</tr>
<tr>
<td></td>
<td>45</td>
</tr>
<tr>
<td></td>
<td>40</td>
</tr>
</tbody>
</table>
### Table 6-14 International System of Units (SI) Conversion Guide*

<table>
<thead>
<tr>
<th>Analyte</th>
<th>Fluid</th>
<th>Traditional Units</th>
<th>Conversion Factor</th>
<th>SI Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACTH (adrenocorticotropic; corticotropin)</td>
<td>Plasma</td>
<td>pg/mL</td>
<td>0.2202</td>
<td>pmol/L</td>
</tr>
<tr>
<td>ALT (alanine aminotransferase; SGPT)</td>
<td>Serum</td>
<td>mg/dL</td>
<td>1</td>
<td>units/L</td>
</tr>
<tr>
<td>Albumin</td>
<td>Serum</td>
<td>g/dL</td>
<td>10</td>
<td>g/L</td>
</tr>
<tr>
<td>Aldosterone</td>
<td>Serum</td>
<td>ng/dL</td>
<td>27.74</td>
<td>pmol/L</td>
</tr>
<tr>
<td>Ammonia (NH₃)</td>
<td>Plasma</td>
<td>mcg/dL</td>
<td>0.5872</td>
<td>µmol/L</td>
</tr>
<tr>
<td>Ammonium (NH₄⁺)</td>
<td>Plasma</td>
<td>mcg/dL</td>
<td>0.5543</td>
<td>µmol/L</td>
</tr>
<tr>
<td>Amylase</td>
<td>Serum</td>
<td>units/L</td>
<td>1</td>
<td>units/L</td>
</tr>
<tr>
<td>Antibodies</td>
<td>Serum</td>
<td>Highest possible dilution</td>
<td>1</td>
<td>Highest possible dilution</td>
</tr>
<tr>
<td>AST (aspartate aminotransferase; SGOT)</td>
<td>Serum</td>
<td>units/L</td>
<td>1</td>
<td>units/L</td>
</tr>
<tr>
<td>Bile acids (total)</td>
<td>Serum</td>
<td>mcg/mL</td>
<td>2.547</td>
<td>µmol/L</td>
</tr>
<tr>
<td>Bilirubin (total)</td>
<td>Serum</td>
<td>mg/dL</td>
<td>17.1</td>
<td>µmol/L</td>
</tr>
<tr>
<td>Blood gases</td>
<td>Arterial blood</td>
<td>mm Hg</td>
<td>0.1333</td>
<td>kPa</td>
</tr>
<tr>
<td></td>
<td></td>
<td>pH units</td>
<td>1</td>
<td>pH units</td>
</tr>
<tr>
<td></td>
<td></td>
<td>mm Hg</td>
<td>0.1333</td>
<td>kPa</td>
</tr>
<tr>
<td>BUN (blood urea nitrogen)</td>
<td>Serum</td>
<td>mg/dL</td>
<td>0.357</td>
<td>mmol/L of urea</td>
</tr>
<tr>
<td>Calcium</td>
<td>Serum</td>
<td>mg/dL</td>
<td>0.250</td>
<td>mmol/L</td>
</tr>
<tr>
<td>Calcium, ionized (iCa)</td>
<td>Serum, plasma</td>
<td>mEq/L</td>
<td>0.500</td>
<td>mmol/L</td>
</tr>
<tr>
<td>CBC (complete blood count):</td>
<td>Whole blood</td>
<td>%</td>
<td>0.01</td>
<td>as a fraction of 1</td>
</tr>
<tr>
<td>Hematocrit</td>
<td></td>
<td>g/dL</td>
<td>10</td>
<td>g/L</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td></td>
<td>pg</td>
<td>1</td>
<td>pg</td>
</tr>
<tr>
<td>MCH (mean corpuscular hemoglobin)</td>
<td></td>
<td>g/dL</td>
<td>10</td>
<td>g/L</td>
</tr>
<tr>
<td>MCHC (mean corpuscular hemoglobin concentration)</td>
<td></td>
<td>1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Presented in alphabetical order.
<table>
<thead>
<tr>
<th>Test</th>
<th>Unit</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCV (mean corpuscular volume)</td>
<td>µm³</td>
<td>1 µm³</td>
</tr>
<tr>
<td>Platelet count</td>
<td>103/mm³</td>
<td>1 109/L</td>
</tr>
<tr>
<td>Reticulocyte count</td>
<td>No. per 1000 red blood cells (RBCs)</td>
<td>0.001 as a fraction of 1</td>
</tr>
<tr>
<td>Reticulocyte count</td>
<td>As a %</td>
<td>0.01 as a fraction of 1</td>
</tr>
<tr>
<td>Differential cell count</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neutrophils (segmented)</td>
<td>cells/mm³ (mL)</td>
<td>1 106 cells/L</td>
</tr>
<tr>
<td>Neutrophils (band)</td>
<td>cells/mm³</td>
<td>1</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>cells/mm³</td>
<td>1</td>
</tr>
<tr>
<td>Monocytes</td>
<td>cells/mm³</td>
<td>1</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>cells/mm³</td>
<td>1</td>
</tr>
<tr>
<td>Basophils</td>
<td>cells/mm³</td>
<td>1</td>
</tr>
<tr>
<td>Cholesterol (total)</td>
<td>Serum</td>
<td>mg/dL</td>
</tr>
<tr>
<td>CK (creatinine kinase)</td>
<td>Serum</td>
<td>units/L</td>
</tr>
<tr>
<td>Cortisol</td>
<td>Serum, plasma</td>
<td>mcg/dL</td>
</tr>
<tr>
<td>Cortisol (free)</td>
<td>Urine</td>
<td>mcg/24 hr</td>
</tr>
<tr>
<td>Creatinine</td>
<td>Serum</td>
<td>mg/dL</td>
</tr>
<tr>
<td>Electrolytes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chloride</td>
<td>Serum</td>
<td>mEq/L</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>Whole blood</td>
<td>mEq/L</td>
</tr>
<tr>
<td>Potassium</td>
<td>Serum</td>
<td>mEq/L</td>
</tr>
<tr>
<td>Sodium</td>
<td>Serum</td>
<td>mEq/L</td>
</tr>
<tr>
<td>Fibrinogen (coagulation factor I)</td>
<td>Plasma</td>
<td>g/dL</td>
</tr>
<tr>
<td>Fibrin (fibrin degradation products)</td>
<td>Serum</td>
<td>mcg/mL</td>
</tr>
<tr>
<td>GGT (γ-glutamyltransferase)</td>
<td>Serum</td>
<td>units/L</td>
</tr>
<tr>
<td>Glucose</td>
<td>Serum</td>
<td>mg/dL</td>
</tr>
<tr>
<td>Insulin</td>
<td>Serum</td>
<td>µU/mL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>µU/L</td>
</tr>
<tr>
<td></td>
<td></td>
<td>mcg/L</td>
</tr>
</tbody>
</table>

Continued
<table>
<thead>
<tr>
<th>Analyte</th>
<th>Fluid</th>
<th>Traditional Units</th>
<th>Conversion Factor</th>
<th>SI Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lead</td>
<td>Plasma</td>
<td>mcg/dL</td>
<td>0.04826</td>
<td>µmol/L</td>
</tr>
<tr>
<td></td>
<td></td>
<td>mg/dL</td>
<td>48.26</td>
<td>µmol/L</td>
</tr>
<tr>
<td>Lipase</td>
<td>Serum</td>
<td>units/L</td>
<td>1</td>
<td>units/L</td>
</tr>
<tr>
<td>Magnesium</td>
<td>Serum</td>
<td>mg/dL</td>
<td>0.4114</td>
<td>mmol/L</td>
</tr>
<tr>
<td></td>
<td></td>
<td>mEq/L</td>
<td>0.500</td>
<td>mmol/L</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>Serum</td>
<td>mg/dL</td>
<td>0.3229</td>
<td>mmol/L</td>
</tr>
<tr>
<td>Plasminogen</td>
<td>Plasma</td>
<td>%</td>
<td>0.01</td>
<td>as a fraction of 1</td>
</tr>
<tr>
<td>Protein (total)</td>
<td>Serum</td>
<td>g/dL</td>
<td>10</td>
<td>g/L</td>
</tr>
<tr>
<td>Protein (spinal fluid)</td>
<td>Cerebrospinal fluid (CSF)</td>
<td>mg/dL</td>
<td>0.01</td>
<td>g/L</td>
</tr>
<tr>
<td>PT (prothrombin time)</td>
<td>Plasma</td>
<td>seconds</td>
<td>1</td>
<td>seconds</td>
</tr>
<tr>
<td>PTTT (partial thromboplastin time)</td>
<td>Plasma</td>
<td>seconds</td>
<td>1</td>
<td>seconds</td>
</tr>
<tr>
<td>Thyroid tests</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TSH (thyroid-stimulating hormone)</td>
<td>Serum</td>
<td>µU/mL</td>
<td>1</td>
<td>mU/L</td>
</tr>
<tr>
<td>T&lt;sub&gt;4&lt;/sub&gt; (thyroxine)</td>
<td>Serum</td>
<td>mcg/dL</td>
<td>12.87</td>
<td>nmol/L</td>
</tr>
<tr>
<td>Thyroxine, free T&lt;sub&gt;4&lt;/sub&gt;</td>
<td>Serum</td>
<td>ng/dL</td>
<td>12.87</td>
<td>pmol/L</td>
</tr>
<tr>
<td>T&lt;sub&gt;3&lt;/sub&gt; (triiodothyronine)</td>
<td>Serum</td>
<td>ng/dL</td>
<td>0.01536</td>
<td>nmol/L</td>
</tr>
<tr>
<td>Prefix</td>
<td>Multiply by</td>
<td>Factor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>---------</td>
<td>----------------</td>
<td>---------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>milli-</td>
<td>0.001 (1/1000)</td>
<td>$\times 10^{-3}$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>centi-</td>
<td>0.01 (1/1000)</td>
<td>$\times 10^{-2}$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>deci-</td>
<td>0.1 (1/10)</td>
<td>$\times 10^{-1}$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>deka-</td>
<td>10</td>
<td>$\times 10$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>hecto-</td>
<td>100</td>
<td>$\times 10^{3}$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>kilo-</td>
<td>1000</td>
<td>$\times 10^{5}$</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Unit</th>
<th>Abbreviations</th>
</tr>
</thead>
<tbody>
<tr>
<td>The standard unit of volume in the metric system is the liter.</td>
<td>1 milliliter = 0.001 liter</td>
<td>1 milliliter = 1 mL = 1 cc*</td>
</tr>
<tr>
<td></td>
<td>1 centiliter = 0.01 liter</td>
<td>1 centiliter = 1 cL</td>
</tr>
<tr>
<td></td>
<td>1 deciliter = 0.1 liter</td>
<td>1 deciliter = 1 dL</td>
</tr>
<tr>
<td></td>
<td>1 liter</td>
<td>1 liter = 1 L</td>
</tr>
<tr>
<td></td>
<td>1 kiloliter = 1000 liters</td>
<td>1 kiloliter = 1 kL</td>
</tr>
</tbody>
</table>

| The standard unit of mass in the metric system is the gram. | 1 nanogram = 10^-9 gram | 1 nanogram = 1 ng |
|                                                            | 1 microgram = 10^-6 gram | 1 microgram = 1 mcg or 1 µg |
|                                                            | 1 milligram = 0.001 gram | 1 milligram = 1 mg |
|                                                            | 1 centigram = 0.01 gram | 1 centigram = 1 cg |
|                                                            | 1 decigram = 0.1 gram | 1 decigram = 1 dg |
|                                                            | 1 gram | 1 gram = 1 g |
|                                                            | 1 kilogram = 1000 grams | 1 kilogram = 1 kg |

| The standard unit of length in the metric system is the meter. | 1 millimeter = 0.001 meter | 1 millimeter = 1 mm |
|                                                              | 1 centimeter = 0.01 meter | 1 centimeter = 1 cm |
|                                                              | 1 meter | 1 meter = 1 m |
|                                                              | 1 decimeter = 0.1 meter | 1 decimeter = 1 dm |
|                                                              | 1 kilometer = 1000 meters | 1 kilometer = 1 km |

*1 cc (or cubic centimeter) = 1 cm³ = 1 mL.
<table>
<thead>
<tr>
<th>Vaccine Type</th>
<th>Core versus</th>
<th>Recommended Vaccination Interval for Administration of Booster Inoculations</th>
<th>Minimum Duration of Immunity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distemper: modified live (parenteral)</td>
<td>Core</td>
<td>3 years</td>
<td>5+ to 7+ years (depending on strain)</td>
</tr>
<tr>
<td>Recombinant distemper (parenteral)</td>
<td>Core</td>
<td>3 years</td>
<td>5+ years</td>
</tr>
<tr>
<td>Measles virus: modified live (parenteral)</td>
<td>Noncore</td>
<td>Not indicated</td>
<td>Not applicable</td>
</tr>
<tr>
<td>Available only in combination with modified live distemper + adenovirus-2 + parainfluenza vaccine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parvovirus: modified live (parenteral)</td>
<td>Core</td>
<td>3 years</td>
<td>7+ years</td>
</tr>
<tr>
<td>Coronavirus: modified live (parenteral)</td>
<td>NR</td>
<td>NR</td>
<td>Cannot be determined</td>
</tr>
<tr>
<td>Coronavirus: killed (parenteral)</td>
<td>NR</td>
<td>NR</td>
<td>Cannot be determined</td>
</tr>
<tr>
<td>Canine adenovirus-2: modified live (parenteral)</td>
<td>Core</td>
<td>3 years</td>
<td>7+ years</td>
</tr>
<tr>
<td>Canine adenovirus-2: modified live (intranasal)</td>
<td>Core</td>
<td>3 years</td>
<td>3+ years</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td><em>Note:</em> the intranasal vaccine is not intended for the prevention of canine hepatitis virus infection.</td>
</tr>
<tr>
<td>Canine adenovirus-2: killed (parenteral)</td>
<td>Noncore</td>
<td>Annual</td>
<td>Unknown</td>
</tr>
<tr>
<td>Parainfluenza virus: modified live (parenteral)</td>
<td>Noncore</td>
<td>3 years</td>
<td>3+ years</td>
</tr>
<tr>
<td>Parainfluenza virus: modified live (intranasal)</td>
<td>Noncore</td>
<td>3 years</td>
<td>3+ years (preferred)</td>
</tr>
<tr>
<td><em>Bordetella bronchiseptica:</em> avirulent live (intranasal)</td>
<td>Noncore</td>
<td>Annual</td>
<td>12 months</td>
</tr>
<tr>
<td><em>B. bronchiseptica:</em> antigen extract (parenteral)</td>
<td>Noncore</td>
<td>Annual</td>
<td>Not established</td>
</tr>
<tr>
<td>Canine influenza virus: killed (parenteral)</td>
<td>Noncore</td>
<td>Annual</td>
<td>Not established</td>
</tr>
<tr>
<td>Vaccine Type</td>
<td>Core/Non-Core</td>
<td>Frequency</td>
<td>Duration</td>
</tr>
<tr>
<td>--------------------------------------</td>
<td>---------------</td>
<td>-----------</td>
<td>----------</td>
</tr>
<tr>
<td>Leptospira var canicola</td>
<td>Non-core</td>
<td>Annual</td>
<td>12 months</td>
</tr>
<tr>
<td>Leptospira var icterhemorrhagiae</td>
<td>Non-core</td>
<td>Annual</td>
<td></td>
</tr>
<tr>
<td>Leptospira var pomona</td>
<td>Non-core</td>
<td>Annual</td>
<td></td>
</tr>
<tr>
<td>Leptospira var grippotyphosa</td>
<td>Non-core</td>
<td>Annual</td>
<td></td>
</tr>
<tr>
<td>Recombinant Lyme (parenteral)</td>
<td>Noncore</td>
<td>Annual</td>
<td>1 year</td>
</tr>
<tr>
<td>Lyme: killed (parenteral)</td>
<td>Noncore</td>
<td>Annual</td>
<td>1 year</td>
</tr>
<tr>
<td>Crotalus atrox (rattlesnake vaccine)</td>
<td>Noncore</td>
<td>Annual or seasonally as recommended by manufacturer</td>
<td>Unknown (license is conditional at this writing—challenge studies in dogs have not been performed)</td>
</tr>
<tr>
<td>Rabies, 1-year: killed (parenteral)</td>
<td>Core</td>
<td>As defined by local and state law</td>
<td>3+ years</td>
</tr>
<tr>
<td>Rabies, 3-year: killed (parenteral)</td>
<td>Core</td>
<td>As defined by local and state law</td>
<td>3+ years</td>
</tr>
</tbody>
</table>

NR, Not generally recommended.
<table>
<thead>
<tr>
<th>Vaccine Type</th>
<th>Adjuvanted versus Nonadjuvanted</th>
<th>Core versus Noncore</th>
<th>Recommended Vaccination Interval for Administration of Booster Inoculations</th>
<th>Minimum Duration of Immunity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Panleukopenia: modified live (parenteral)</td>
<td>Nonadjuvanted</td>
<td>Core</td>
<td>3 years</td>
<td>7 + years</td>
</tr>
<tr>
<td>Panleukopenia: killed (parenteral)</td>
<td>Adjuvanted</td>
<td>Noncore</td>
<td>Annual</td>
<td>5 + years</td>
</tr>
<tr>
<td>Panleukopenia: modified live (intranasal)</td>
<td>Nonadjuvanted</td>
<td>Noncore</td>
<td>3 years</td>
<td>Not established</td>
</tr>
<tr>
<td>Herpesvirus-calicivirus: modified live (parenteral)</td>
<td>Nonadjuvanted</td>
<td>Core</td>
<td>3 years (annual vaccination may be recommended in high-risk settings)</td>
<td>5 + years</td>
</tr>
<tr>
<td>Herpesvirus-calicivirus: killed (parenteral)</td>
<td>Adjuvanted</td>
<td>Noncore</td>
<td>Annual</td>
<td>5 + years</td>
</tr>
<tr>
<td>Herpesvirus-calicivirus: modified live (intranasal)</td>
<td>Nonadjuvanted</td>
<td>Noncore</td>
<td>Annual</td>
<td>Not established</td>
</tr>
<tr>
<td>Chlamydophilia felis: killed</td>
<td>Adjuvanted</td>
<td>Noncore</td>
<td>Annual</td>
<td>1 year (maximum)</td>
</tr>
<tr>
<td>C. felis: live, avirulent</td>
<td>Nonadjuvated</td>
<td>Noncore</td>
<td>Annual</td>
<td></td>
</tr>
<tr>
<td>Recombinant feline leukemia virus (transdermal in the United States; parenteral in Canada, the United Kingdom, and Europe)</td>
<td>Nonadjuvanted</td>
<td>Noncore*</td>
<td>Annual where risk is sustained</td>
<td>1 year</td>
</tr>
<tr>
<td>Feline leukemia virus: killed (parenteral)</td>
<td>Adjuvanted</td>
<td>Noncore*</td>
<td>Annual where risk is sustained</td>
<td>1 year</td>
</tr>
<tr>
<td>Feline immunodeficiency virus: killed (parenteral)</td>
<td>Adjuvanted</td>
<td>Non-core*</td>
<td>Annual</td>
<td>1 year</td>
</tr>
<tr>
<td>Feline infectious peritonitis: modified live (intranasal)</td>
<td>Nonadjuvanted</td>
<td>NR</td>
<td>NR</td>
<td>Not established</td>
</tr>
<tr>
<td>Bordetella bronchiseptica: modified live (intranasal)</td>
<td>Nonadjuvanted</td>
<td>Noncore</td>
<td>Annual</td>
<td>1 year</td>
</tr>
<tr>
<td>Vaccination Type</td>
<td>Adjuvanted</td>
<td>Core</td>
<td>Annual</td>
<td>Time Required</td>
</tr>
<tr>
<td>--------------------------------------------------------------------------------</td>
<td>------------</td>
<td>---------</td>
<td>-----------------</td>
<td>--------------------------------------------</td>
</tr>
<tr>
<td>Virulent systemic feline calicivirus: killed (parenteral)</td>
<td>Adjuvanted</td>
<td>Noncore</td>
<td>Annual</td>
<td>1 year</td>
</tr>
<tr>
<td>Recombinant rabies (parenteral)</td>
<td>Nonadjuvanted</td>
<td>Core</td>
<td>Annual</td>
<td>1+ years (must be administered in accordance with state and local requirements)</td>
</tr>
<tr>
<td>Rabies, 1-year: killed (parenteral)</td>
<td>Adjuvanted</td>
<td>Core</td>
<td>Annual</td>
<td>3+ years (must be administered in accordance with state and local requirements)</td>
</tr>
<tr>
<td>Rabies, 3-year: killed (parenteral)</td>
<td>Adjuvanted</td>
<td>Core</td>
<td>3 years (as required by law)</td>
<td>3+ years (must be administered in accordance with state and local requirements)</td>
</tr>
</tbody>
</table>

NR, Not generally recommended.

*Because of the high susceptibility of kittens to infection, current guidelines state that feline leukemia virus (FeLV) vaccination is "highly recommended" for all kittens and cats. Consequently, FeLV vaccination becomes noncore thereafter.
<table>
<thead>
<tr>
<th>TABLE 6-18 Canine Vaccination Recommendations—Initial Puppy Series</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Core vaccines</strong></td>
</tr>
<tr>
<td>MLV or rDistemper + MLV Parovirus + MLV Adenovirus-2</td>
</tr>
<tr>
<td>Option:</td>
</tr>
<tr>
<td>Parainfluenza virus is often administered in combination with the above vaccines.</td>
</tr>
<tr>
<td>Rabies, 1-year (killed)</td>
</tr>
<tr>
<td><strong>Noncore vaccines</strong></td>
</tr>
<tr>
<td>B. bronchiseptica + parainfluenza virus (intranasal)</td>
</tr>
<tr>
<td>Leptospira (killed) four-serovar [two-way Leptospira vaccines are no longer recommended]</td>
</tr>
<tr>
<td>Note: Do not administer the first dose before 12 wk of age.</td>
</tr>
<tr>
<td>Also: In small-breed dogs, delay initial administration until 2 to 4 wk after completion of the initial core series.</td>
</tr>
<tr>
<td>Lyme disease (recombinant or killed)</td>
</tr>
<tr>
<td>Note: Do not administer the first dose before 12 wk of age.</td>
</tr>
<tr>
<td>Also: In small-breed dogs, delay initial administration until 2 to 4 wk after completion of the initial core series.</td>
</tr>
<tr>
<td>Canine influenza virus (killed)</td>
</tr>
<tr>
<td>Note: Do not administer the first dose before 12 wk of age.</td>
</tr>
<tr>
<td>Also: In small-breed dogs, delay initial administration until after completion of the initial core series.</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Crotalus atrox</td>
</tr>
<tr>
<td>Administration recommendations may vary depending on the level of risk and patient size.</td>
</tr>
</tbody>
</table>

*MLV, Modified live virus.*
### Table 6-19 Canine Vaccination Recommendations—Adult

<table>
<thead>
<tr>
<th><strong>Canine core vaccines</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>• A single booster dose of combination distemper + parvovirus + adenovirus-2 vaccine is recommended every 3 years.</td>
</tr>
<tr>
<td>• Rabies vaccine is administered in accordance with state or local requirements. All states currently recognize the 3-year rabies vaccine for dogs. Some municipalities or counties may require rabies vaccine to be administered more frequently than every 3 years. Some States allow discretionary use of a labeled 3-year rabies in place of a labeled 1-year rabies vaccine.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Canine non-core vaccines</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Administer a single dose annually to patients with sustained exposure risk.</td>
</tr>
<tr>
<td>• Patients that have not been vaccinated within the past 2 years should receive two doses of a noncore vaccine, 2 to 6 weeks apart. The exception is the intranasal <em>Bordetella bronchiseptica</em> + parainfluenza virus (+ adenovirus-2) vaccines, in which a single dose is considered sufficient to induce a protective immune response regardless of the time since administration of the last vaccine.</td>
</tr>
<tr>
<td><strong>TABLE 6-20</strong> Feline Vaccination Recommendations—Initial Kitten Series</td>
</tr>
<tr>
<td>-------------------------------------------------</td>
</tr>
<tr>
<td><strong>Core vaccines</strong></td>
</tr>
<tr>
<td>MLV panleukopenia + MLV herpesvirus + MLV calicivirus</td>
</tr>
<tr>
<td>When feasible, avoid the use of killed (adjuvanted) vaccines in cats.</td>
</tr>
<tr>
<td>Rabies (recombinant [the only nonadjuvanted rabies vaccine])</td>
</tr>
<tr>
<td>Alternative: rabies (killed-adjuvanted)</td>
</tr>
<tr>
<td><strong>Noncore vaccines</strong></td>
</tr>
<tr>
<td>Feline leukemia virus (FeLV) Recombinant-nonadjuvanted (also available as killed-adjuvanted)</td>
</tr>
<tr>
<td>Feline immunodeficiency virus (FIV) Killed-adjuvanted</td>
</tr>
<tr>
<td>Feline <em>Bordetella bronchiseptica</em> Attenuated live intranasal</td>
</tr>
<tr>
<td>Feline <em>Chlamydophila felis</em> (formerly <em>Chlamydia psittaci</em>)</td>
</tr>
<tr>
<td>Virulent systemic (VS) calicivirus Killed-adjuvanted</td>
</tr>
</tbody>
</table>

*MLV*, Modified live virus.

*Note:* The Feline Infectious Peritonitis (FIP) vaccine is deemed “not generally recommended” by the AAFP Vaccine Advisory Panel.
**Table 6-21** Feline Vaccination Recommendations—Adult

<table>
<thead>
<tr>
<th>Vaccine</th>
<th>Administration</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Core vaccines</strong></td>
<td></td>
</tr>
<tr>
<td>MLV panleukopenia + MLV herpesvirus + MLV calicivirus</td>
<td>Administer a single dose every 3 years after completion of the initial kitten series and the first booster.</td>
</tr>
<tr>
<td>When feasible, avoid the use killed (adjuvanted) vaccines in cats.</td>
<td></td>
</tr>
<tr>
<td>Rabies (recombinant [the only nonadjuvanted rabies vaccine]) or Rabies (killed-adjuvanted)</td>
<td>Administer a single dose annually in accordance with State or local law.</td>
</tr>
</tbody>
</table>

| **Noncore vaccines** | |
| Feline leukemia virus (FeLV) Recombinant-nonadjuvanted (also available as killed-adjuvanted) | FeLV vaccination recommended annually only if risk is sustained (e.g., outdoor cats with reasonable risk of encounter with other cats). The recombinant FeLV vaccine (administered transdermally) is not adjuvanted; all other FeLV vaccines contain adjuvant. |

*MLV, Modified live virus.

*Other noncore vaccines are seldom administered and should be considered only after assessing and defining a clear risk of exposure. All other noncore vaccines are recommended for annual administration as long as the risk of exposure persists.

**Table 6-22** Compendium of Animal Rabies Prevention and Control, 2005,* National Association of State Public Health Veterinarians (NASPHV)†

Rabies is a fatal viral zoonosis and a serious public health problem.† The recommendations in this compendium serve as the basis for animal rabies prevention and control programs throughout the United States and facilitate standardization of procedures among jurisdictions, thereby contributing to an effective national rabies-control program. This document is reviewed annually and revised as necessary. Principles of rabies prevention and control are detailed in Part I; Part II contains recommendations for parenteral vaccination procedures. All animal rabies vaccines licensed by the U.S. Department of Agriculture (USDA) and marketed in the United States are listed in Part III.

**Part I: Rabies prevention and control**

A. Principles of rabies prevention and control

1. Rabies exposure. Rabies is transmitted only when the virus is introduced into bite wounds, into open cuts in skin, or onto mucous membranes from saliva or other potentially infectious material such as neural tissue. Questions about possible exposures should be directed to state or local health authorities.

2. Human rabies prevention. Rabies in humans can be prevented either by eliminating exposures to rabid animals or by providing exposed persons with prompt local treatment of wounds combined with the administration of human rabies immune globulin and vaccine. The rationale for recommending preexposure and postexposure rabies prophylaxis and details of their administration can be found in the current recommendations of the Advisory Committee on Immunization Practices (ACIP). These recommendations, along with information concerning the current local and regional epidemiology of animal rabies and the availability of human rabies biologics, are available from state health departments.

*Continued*
3. Domestic animals. Local governments should initiate and maintain effective programs
to ensure vaccination of all dogs, cats, and ferrets and to remove strays and unwanted
animals. Such procedures in the United States have reduced laboratory-confirmed
cases of rabies in dogs from 6949 in 1947 to 117 in 2003. Because more rabies cases
are reported annually involving cats (321 in 2003) than dogs, vaccination of cats
should be required. Animal shelters and animal control authorities should establish
policies to ensure that adopted animals are vaccinated against rabies. The
recommended vaccination procedures and the licensed animal vaccines are specified
in Parts II and III of the compendium.

4. Rabies in vaccinated animals. Rabies is rare in vaccinated animals. If such
an event is suspected, it should be reported to state public health officials;
the vaccine manufacturer; and USDA, Animal and Plant Health Inspection
Service, Center for Veterinary Biologics (www.aphis.usda.gov/vs/cvb/ic/
adverseeventreport.htm; telephone 800-752-6255; or e-mail CVB@usda.gov).
The laboratory diagnosis should be confirmed and the virus characterized by a
rabies reference laboratory. A thorough epidemiologic investigation should be
conducted.

5. Rabies in wildlife. The control of rabies among wildlife reservoirs is difficult.
Vaccination of free-ranging wildlife or selective population reduction might be
useful in some situations, but the success of such procedures depends on the
circumstances surrounding each rabies outbreak (see Part I. C. Prevention and
Control Methods Related to Wildlife). Because of the risk of rabies in wild animals
(especially raccoons, skunks, coyotes, foxes, and bats), AVMA, NASPHV, and CSTE
strongly recommend the enactment and enforcement of state laws prohibiting their
importation, distribution, and relocation.

6. Rabies surveillance. Laboratory-based rabies surveillance is an essential component
of rabies control and prevention programs. Accurate and timely information is
necessary to guide human postexposure prophylaxis decisions, determine the
management of potentially exposed animals, aid in emerging pathogen discovery,
describe the epidemiology of the disease, and assess the need for and effectiveness of
oral vaccination programs for wildlife.

7. Rabies diagnosis. Rabies testing should be performed by a qualified laboratory
that has been designated by the local or state health department in accordance
with the established national standardized protocol for rabies testing
(www.cdc.gov/ncidod/dvrd/rabies/Professional/publications/DFA_diagnosis/
DFA_protocol-b.htm). Euthanasia should be accomplished in such a way as
to maintain the integrity of the brain so that the laboratory can recognize the
anatomic parts. Except in the case of very small animals, such as bats, only the
head or brain (including brainstem) should be submitted to the laboratory.
Any animal or animal specimen being submitted for testing should be kept
under refrigeration (not frozen or chemically fixed) during storage and
shipping.

8. Rabies serology. Some “rabies-free” jurisdictions may require evidence of vaccination
and rabies antibodies for importation purposes. Rabies antibody titers are indicative
of an animal’s response to vaccine or infection. Titers do not directly correlate with
protection because other immunologic factors also play a role in preventing rabies,
and our abilities to measure and interpret those other factors are not well developed.
Therefore, evidence of circulating rabies virus antibodies should not be used as a
substitute for current vaccination in managing rabies exposures or determining the
need for booster vaccinations in animals.

Continued
B. Prevention and control methods in domestic and confined animals

1. Preexposure vaccination and management. Parenteral animal rabies vaccines should be administered only by or under the direct supervision of a veterinarian. Rabies vaccinations may also be administered under the supervision of a veterinarian to animals held in animal control shelters before release. Any veterinarian signing a rabies certificate must ensure that the person administering vaccine is identified on the certificate and is appropriately trained in vaccine storage, handling, and administration and in the management of adverse events. This practice ensures that a qualified and responsible person can be held accountable to ensure that the animal has been properly vaccinated.

Within 28 days after primary vaccination, a peak rabies antibody titer is reached and the animal can be considered immunized. An animal is currently vaccinated and is considered immunized if the primary vaccination was administered at least 28 days previously and vaccinations have been administered in accordance with this compendium.

Regardless of the age of the animal at initial vaccination, a booster vaccination should be administered 1 year later (see Parts II and III for vaccines and procedures). No laboratory or epidemiologic data exist to support the annual or biennial administration of 3-year vaccines following the initial series. Because a rapid anamnestic response is expected, an animal is considered currently vaccinated immediately after a booster vaccination.

a. Dogs, cats, and ferrets. All dogs, cats, and ferrets should be vaccinated against rabies and revaccinated in accordance with Part III of this compendium. If a previously vaccinated animal is overdue for a booster, it should be revaccinated. Immediately after the booster, the animal is considered currently vaccinated and should be placed on an annual or triennial schedule depending on the type of vaccine used.

b. Livestock. Consideration should be given to vaccinating livestock that are particularly valuable or that might have frequent contact with humans (e.g., in petting zoos, fairs, and other public exhibitions). Horses traveling interstate should be currently vaccinated against rabies.

c. Confined animals.

1) Wild. No parenteral rabies vaccines are licensed for use in wild animals or hybrids (the offspring of wild animals crossbred to domestic animals). Wild animals or hybrids should not be kept as pets.

2) Maintained in exhibits and in zoologic parks. Captive mammals that are not completely excluded from all contact with rabies vectors can become infected. Moreover, wild animals might be incubating rabies when initially captured; therefore, wild-caught animals susceptible to rabies should be quarantined for a minimum of 6 months before being exhibited. Employees who work with animals at such facilities should receive preexposure rabies vaccination. The use of preexposure or postexposure rabies vaccinations for employees who work with animals at such facilities might reduce the need for euthanasia of captive animals. Carnivores and bats should be housed in a manner that precludes direct contact with the public.

3) Stray animals. Stray dogs, cats, and ferrets should be removed from the community. Local health departments and animal control officials can enforce the removal of strays more effectively if owned animals have identification and are confined or kept on leash. Strays should be impounded for at least 3 business days to determine if human exposure has occurred and to give owners sufficient time to reclaim animals.

Continued
2. Importation and interstate movement of animals.
   a. International. The CDC regulates the importation of dogs and cats into the United States. Importers of dogs must comply with rabies vaccination requirements (42 CFR, Part 71.51[c] [www.cdc.gov/ncidod/dq/animal.htm]) and complete CDC form 75.37 (www.cdc.gov/ncidod/dq/pdf/cdc7537-05-24-04.pdf). The appropriate health official of the state of destination should be notified within 72 hours of the arrival into his or her jurisdiction of any imported dog required to be placed in confinement under the CDC regulation. Failure to comply with these requirements should be promptly reported to the Division of Global Migration and Quarantine, CDC (telephone 404-498-1670).

   Federal regulations alone are insufficient to prevent the introduction of rabid animals into the country. All imported dogs and cats are subject to state and local laws governing rabies and should be currently vaccinated against rabies in accordance with this compendium. Failure to comply with state or local requirements should be referred to the appropriate state or local official.

   b. Interstate. Before interstate (including commonwealths and territories) movement, dogs, cats, ferrets, and horses should be currently vaccinated against rabies in accordance with the compendium’s recommendations (see Part I. B.1. Preexposure Vaccination and Management). Animals in transit should be accompanied by a currently valid NASPHV Form 51, Rabies Vaccination Certificate (www.nasphv.org/83416/106001.html). When an interstate health certificate or certificate of veterinary inspection is required, it should contain the same rabies vaccination information as Form 51.

   c. Areas with dog-to-dog rabies transmission. The movement of dogs from areas with dog-to-dog rabies transmission for the purpose of adoption or sale should be eliminated. Rabid dogs have been introduced into the United States from areas with dog-to-dog rabies transmission. This practice poses the risk of introducing canine-transmitted rabies to areas where it does not currently exist.

3. Adjunct procedures. Methods or procedures that enhance rabies control include the following:
   a. Identification. Dogs, cats, and ferrets should be identified (e.g., metal or plastic tags or microchips) to allow for verification of rabies vaccination status.

   b. Licensure. Registration or licensure of all dogs, cats, and ferrets may be used to aid in rabies control. A fee is frequently charged for such licensure, and revenues collected are used to maintain rabies- or animal-control programs. Evidence of current vaccination is an essential prerequisite to licensure.

   c. Canvassing. House-to-house canvassing by animal control officials facilitates enforcement of vaccination and licensure requirements.

   d. Citations. Citations are legal summonses issued to owners for violations, including the failure to vaccinate or license their animals. The authority for officers to issue citations should be an integral part of each animal-control program.

   e. Animal control. All communities should incorporate stray animal control, leash laws, and training of personnel in their programs.

4. Postexposure management. Any animal potentially exposed to rabies virus (see Part I. A.1. Rabies Exposure) by a wild, carnivorous mammal or a bat that is not available for testing should be regarded as having been exposed to rabies.
   a. Dogs, cats, and ferrets. Unvaccinated dogs, cats, and ferrets exposed to a rabid animal should be euthanized immediately. If the owner is unwilling to have this done, the animal should be placed in strict isolation for 6 months.

Continued
Rabies vaccine should be administered on entry into isolation or 1 month before release to comply with preexposure vaccination recommendations (see Part I. B.1.a.). Protocols for the postexposure vaccination of previously unvaccinated domestic animals have not been validated, and evidence exists that the use of vaccine alone will not prevent the disease. Animals with expired vaccinations need to be evaluated on a case-by-case basis. Dogs, cats, and ferrets that are currently vaccinated should be revaccinated immediately, kept under the owner’s control, and observed for 45 days. Any illness in an isolated or confined animal should be reported immediately to the local health department.

b. Livestock. All species of livestock are susceptible to rabies; cattle and horses are among the most frequently infected. Livestock exposed to a rabid animal and currently vaccinated with a vaccine approved by USDA for that species should be revaccinated immediately and observed for 45 days. Unvaccinated livestock should be slaughtered immediately. If the owner is unwilling to have this done, the animal should be kept under close observation for 6 months. Any illness in an animal under observation should be reported immediately to the local health department.

The following are recommendations for owners of livestock exposed to rabid animals:

1) If the animal is slaughtered within 7 days of being bitten, its tissues may be eaten without risk of infection, provided that liberal portions of the exposed area are discarded. Federal guidelines for meat inspectors require that any animal known to have been exposed to rabies within 8 months be rejected for slaughter.

2) Neither tissues nor milk from a rabid animal should be used for human or animal consumption. Pasteurization temperatures will inactivate rabies virus; therefore, drinking pasteurized milk or eating cooked meat does not constitute a rabies exposure.

3) Having more than one rabid animal in a herd or having herbivore-to-herbivore transmission is uncommon; therefore restricting the rest of the herd if a single animal has been exposed to or infected by rabies might not be necessary.

c. Other animals. Other mammals bitten by a rabid animal should be euthanized immediately. Animals maintained in USDA-licensed research facilities or accredited zoologic parks should be evaluated on a case-by-case basis.

5. Management of animals that bite humans.

a. Dogs, cats, and ferrets. Rabies virus may be excreted in the saliva of infected dogs, cats, and ferrets during illness and/or for only a few days before illness or death. A healthy dog, cat, or ferret that bites a person should be confined and observed daily for 10 days; administration of rabies vaccine to the animal is not recommended during the observation period to avoid confusing signs of rabies with possible side effects of vaccine administration.

Such animals should be evaluated by a veterinarian at the first sign of illness during confinement. Any illness in the animal should be reported immediately to the local health department. If signs suggestive of rabies develop, the animal should be euthanized and the head shipped for testing as described in Part I. A.7. Any stray or unwanted dog, cat, or ferret that bites a person may be euthanized immediately and the head submitted for rabies examination.

Continued
b. Other biting animals. Other biting animals that might have exposed a person to rabies should be reported immediately to the local health department. Management of animals other than dogs, cats, and ferrets depends on the species, the circumstances of the bite, the epidemiology of rabies in the area, and the biting animal’s history, current health status, and potential for exposure to rabies. Prior vaccination of these animals may not preclude the necessity of euthanasia and testing.

C. Prevention and control methods related to wildlife.

The public should be warned not to handle or feed wild mammals. Wild mammals and hybrids that bite or otherwise expose persons, pets, or livestock should be considered for euthanasia and rabies examination. A person bitten by any wild mammal should immediately report the incident to a physician who can evaluate the need for antirabies treatment (see current rabies prophylaxis recommendations of the ACIP). State-regulated wildlife rehabilitators may play a role in a comprehensive rabies control program. Minimum standards for persons who rehabilitate wild mammals should include rabies vaccination, appropriate training, and continuing education. Translocation of infected wildlife has contributed to the spread of rabies; therefore the translocation of known terrestrial rabies reservoir species should be prohibited.

1. Terrestrial mammals. The use of licensed oral vaccines for the mass vaccination of free-ranging wildlife should be considered in selected situations, with the approval of the state agency responsible for animal rabies control. The distribution of oral rabies vaccine should be based on scientific assessments of the target species and followed by timely and appropriate analysis of surveillance data; such results should be provided to all stakeholders. In addition, parenteral vaccination (trap-vaccinate-release) of wildlife rabies reservoirs may be integrated into coordinated oral rabies vaccination programs to enhance their effectiveness. Continuous and persistent programs for trapping or poisoning wildlife are not effective in reducing wildlife rabies reservoirs on a statewide basis. However, limited population control in high-contact areas (e.g., picnic grounds, camps, suburban areas) may be indicated for the removal of selected high-risk species of wildlife. State agriculture, public health, and wildlife agencies should be consulted for planning, coordination, and evaluation of vaccination or population-reduction programs.

2. Bats. Indigenous rabid bats have been reported from every state except Hawaii and have caused rabies in at least 40 humans in the United States. Bats should be excluded from houses, public buildings, and adjacent structures to prevent direct association with humans. Such structures should then be made bat-proof by sealing entrances used by bats. Controlling rabies in bats through programs designed to reduce bat populations is neither feasible nor desirable.

### Part II: Recommendations for Parenteral Rabies Vaccination Procedures

A. Vaccine administration

All animal rabies vaccines should be restricted to use by, or under the direct supervision of, a veterinarian except as recommended in Part I.B.1. All vaccines must be administered in accordance with the specifications of the product label or package insert.

B. Vaccine selection

Part III lists all vaccines licensed by USDA and marketed in the United States at the time of publication. New vaccine approvals or changes in label specifications made subsequent to publication should be considered as part of this list. Any of the listed vaccines can be used for revaccination, even if the product is not the same brand...
previously administered. Vaccines used in state and local rabies control programs should have a 3-year duration of immunity. This constitutes the most effective method of increasing the proportion of immunized dogs and cats in any population. No laboratory or epidemiologic data exist to support the annual or biennial administration of 3-year vaccines following the initial series.

C. Adverse events
Currently, no epidemiologic association exists between a particular licensed vaccine product and adverse events, including vaccine failure. Adverse events should be reported to the vaccine manufacturer and to USDA, Animal and Plant Health Inspection Service, Center for Veterinary Biologics (www.aphis.usda.gov/vs/cvb/ic/adverseeventreport.htm; telephone 800-752-6255; or e-mail CVB@usda.gov).

D. Wildlife and hybrid animal vaccination
The safety and efficacy of parenteral rabies vaccination of wildlife and hybrids have not been established, and no rabies vaccines are licensed for these animals. Parenteral vaccination (trap-vaccinate-release) of wildlife rabies reservoirs may be integrated into coordinated oral rabies vaccination programs as described in Part I. C.1. to enhance their effectiveness. Zoos or research institutions may establish vaccination programs, which attempt to protect valuable animals, but these should not replace appropriate public health activities that protect humans.

E. Accidental human exposure to vaccine
Human exposure to parenteral animal rabies vaccines listed in Part III does not constitute a risk for rabies infection. However, human exposure to vaccinia-vectored oral rabies vaccines should be reported to state health officials.

F. Rabies certificate
All agencies and veterinarians should use NASPHV Form 51, Rabies Vaccination Certificate, which can be obtained from vaccine manufacturers or from NASPHV (www.nasphv.org). It is also available from CDC (www.cdc.gov/ncidod/dvrd/rabies/professional/profesi.htm). The form must be completed in full and signed by the administering or supervising veterinarian. Computer-generated forms containing the same information are also acceptable.

*The material in this report originated at the National Center for Infectious Diseases (Anne Schuchat, MD, Acting Director) and the Division of Viral and Rickettsial Diseases (James W. LeDuc, PhD, Director). The NASPHV Committee: Suzanne R. Jenkins, VMD, MPH, Co-Chair; Mira J. Leslie, DVM, MPH, Co-Chair; Michael Auslander, DVM, MSPH; Lisa Conti, DVM, MPH; Paul Ettestad, DVM, MS; Faye E. Sorhage, VMD, MPH; and Ben Sun, DVM, MPVM. Consultants to the Committee: Donna M. Gatewood, DVM, MS, Center for Veterinary Biologics, U.S. Department of Agriculture (USDA); Ellen Mangione, MD, MPH, Council of State and Territorial Epidemiologists (CSTE); Lorraine Moule, National Animal Control Association (NACA); Greg Pruitt, Animal Health Institute; Charles E. Rupprecht, VMD, MS, PhD, CDC; John Schiltz, DVM, American Veterinary Medical Association (AVMA); Charles V. Trimarchi, MS, New York State Health Department; and Dennis Slate, PhD, Wildlife Services, USDA. This compendium has been endorsed by AVMA, CDC, CSTE, and NACA. Corresponding author: Mira J. Leslie, DVM, MPH, Washington Department of Health, Communicable Disease Epidemiology, 1610 NE 150th Street, MS K17-9, Shoreline, WA 98155-9701.
References


**Table 6-23** Prescription Writing Reference: Do’s and Don’ts

<table>
<thead>
<tr>
<th>Veterinarian Information</th>
<th>Owner Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prescribing veterinarian’s name</td>
<td>Patient’s name (in “quotes”)</td>
</tr>
<tr>
<td>Practice address</td>
<td>Patient’s age or date of birth</td>
</tr>
<tr>
<td>Practice telephone number</td>
<td>Owner’s name (or that of an owner representative)</td>
</tr>
<tr>
<td>DEA # (if written for a controlled substance)</td>
<td>Owner’s address</td>
</tr>
<tr>
<td>Current date</td>
<td>Owner’s phone number</td>
</tr>
</tbody>
</table>

**Prescription**
- Drug name: Print full brand name or generic name; never abbreviate
- Dosage form: Specify tablet, capsule, suspension, other
- Strength (e.g., mg, g, mcg) or concentration (mg/mL); use metric units
- Total quantity (# 10 [for 10 tablets]; 60 mL)
- Sig: Include the following: dose (individual); route; frequency; duration; indication or use
- Number of refills: Define the number permitted
- Designate whether or not generic substitution is permissible
- Signature

**Common prescription writing errors**
- Always use metric units: for example, g (gram) for solids; mL (milliliter) for liquids.
- Use per instead of a slash (/), which can be interpreted as the number 1.
- Use units instead of the abbreviation u, which can be interpreted as 0 or 4 or µ.
- Use once daily instead of sid, which has been interpreted as 5/d, or 5 per day! (Note: “sid” is not a conventional prescription abbreviation.)
- Use three times daily instead of tid, and four times daily instead of qid.
- Use every other day instead of qod.
- Remember: Abbreviations such as qd, qid, and qod are easily confused with one another.
- When writing numbers:
  - Use a leading zero with decimals: for example, use 0.5 mL rather than .5 mL.
  - Avoid using a trailing zero: for example, use 3 rather than 3.0.
  - And finally—When in doubt, spell it out.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Proprietary Names</th>
<th>Action and Use</th>
<th>Formulation</th>
<th>Recommended Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acepromazine</td>
<td>Many generic products</td>
<td>Tranquilizer and antiemetic</td>
<td>5-, 10-, and 25-mg tablets and 10-mg/mL injection</td>
<td>Dog: 0.56-1.13 mg/kg IM, SQ, IV; 0.56-2.25 mg/kg PO q6-8h Cat: 1.13-2.25 mg/kg IM, SQ, IV</td>
</tr>
<tr>
<td>Acetaminophen</td>
<td>Tylenol and other generic brands</td>
<td>NSAID; analgesia</td>
<td>120-, 160-, 325-, and 500-mg tablets</td>
<td>Dog: 15 mg/kg PO q8h Cat: Do not use</td>
</tr>
<tr>
<td>Acetaminophen with codeine</td>
<td>Tylenol with codeine, other generic products</td>
<td>NSAID + opioid; analgesia</td>
<td>Oral solution and tablets Many forms (e.g., 300 mg acetaminophen plus 15, 30, or 60 mg codeine)</td>
<td>Follow dosing recommendations for codeine Dog: (analgesia) 0.5-1 mg/kg PO q6-8h Cat: Do not use</td>
</tr>
<tr>
<td>Acetazolamide</td>
<td>Diamox</td>
<td>Diuretic; management of glaucoma</td>
<td>125- and 250-mg tablets</td>
<td>Glaucoma: 5-10 mg/kg PO q8-12h Diuretic: 4-8 mg/kg PO q8-12h</td>
</tr>
<tr>
<td>§ Acetylcysteine</td>
<td>Mucomyst</td>
<td>Antidote; acetaminophen toxicosis in cats</td>
<td>20% solution (200 mg/mL)</td>
<td>Cat (acetaminophen toxicosis): 140 mg/kg (initial loading dose); then 70 mg/kg PO or IV q4h for five doses</td>
</tr>
<tr>
<td>ACTH gel</td>
<td>See Corticotropin</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Activated charcoal</td>
<td>ActaChar, Charcodote, ToxiBan, and generic</td>
<td>GI adsorbent</td>
<td>Oral suspension</td>
<td>1-4 g/kg PO (granules) 6-12 mL/kg (suspension)</td>
</tr>
<tr>
<td>Albendazole</td>
<td>Valbazen</td>
<td>Antiparasitic, especially respiratory parasites and Giardia species</td>
<td>113.6-mg/mL suspension and 300-mg/mL paste</td>
<td>General antiparasitic: 25-50 mg/kg PO q12h for 3–5–days Respiratory parasites: 50 mg/kg, q24h PO for 10-14 days Giardia: 25 mg/kg q12h for 2–5–days; 2 to 5 puffs four times daily</td>
</tr>
<tr>
<td>Albuterol</td>
<td>Proventil, Ventolin</td>
<td>Bronchodilator</td>
<td>2-, 4-, and 5-mg tablets; 2 mg/5 mL syrup; aerosol (metered inhaler at 90 mcg/dose)</td>
<td>20-50 mcg/kg four times per day; up to maximum of 100 mcg/kg four times per day</td>
</tr>
</tbody>
</table>

*Listings preceded by § are for rapid reference and denote drug or dose used in the emergency or critical care setting.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Proprietary Names</th>
<th>Action and Use</th>
<th>Formulation</th>
<th>Recommended Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allopurinol</td>
<td>Lopurin, Zyloprim</td>
<td>Antiinflammatory; adjunct therapy for leishmaniasis; urolith prevention</td>
<td>100- and 300-mg tablets</td>
<td>Urolith prevention: 10 mg/kg q8h; then reduce to 10 mg/kg q24h</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Leishmaniasis: 10 mg/kg q12h PO for 4 months or more</td>
</tr>
<tr>
<td>Aluminum carbonate gel</td>
<td>Basaljel</td>
<td>Antacid, GI phosphate binder ( uncommonly used today)</td>
<td>Capsules (equivalent to 500 mg aluminum hydroxide)</td>
<td>10-30 mg/kg PO q8h (with meals)</td>
</tr>
<tr>
<td>Aluminum hydroxide gel</td>
<td>Amphojet</td>
<td>Antacid, GI phosphate binder ( uncommonly used today)</td>
<td>64 mg/mL oral suspension; 600-mg tablet</td>
<td>10-90 mg/kg PO q8h (with meals)</td>
</tr>
<tr>
<td>Amikacin</td>
<td>Amiglyde-V (veterinary), Amikin (human)</td>
<td>Antibacterial</td>
<td>50- and 250-mg/mL injection</td>
<td>Dog: 15-30 mg/kg, IV, IM, SQ, q24h</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cat: 10-14 mg/kg, IV, IM, SQ, q24h</td>
</tr>
<tr>
<td>Aminophylline</td>
<td>Many generic brands</td>
<td>Bronchodilator; chronic bronchitis and asthma</td>
<td>100- and 200-mg tablets; 25 mg/mL injection</td>
<td>Dog: 5-11 mg/kg PO, IM, IV q8-12h</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cat: 6.6 mg/kg PO q12h</td>
</tr>
<tr>
<td>§ Amiodarone</td>
<td>Cordarone</td>
<td>Antiarrhythmic; life-threatening arrhythmias</td>
<td>200-mg tablet and 50 mg/mL injection</td>
<td>Dog: 10-15 mg/kg PO q12h, up to 1 wk; then 5-7.5 mg/kg PO q12h for 2 wk; then 7.5 mg/kg q24h as maintenance</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cat: no dosage recommendation</td>
</tr>
<tr>
<td>Amitraz</td>
<td>Mitaban</td>
<td>Antiparasitic, especially ectoparasites <em>Demodex</em> and <em>Sarcoptes</em></td>
<td>10.6 mL concentrated dip (19.9%)</td>
<td>10.6 mL per 7.5 L water (0.025% solution); apply three to six topical treatments every 2 weeks for refractory cases; this dose has been exceeded to produce increased efficacy. Doses used have included 0.025%, 0.05%, and 0.1% concentration applied twice a week and 0.125% solution applied to one-half body every day for 4 weeks to 5 months.</td>
</tr>
<tr>
<td><strong>Amitriptyline</strong> (Elavil)</td>
<td>**Behavior modifier; separation anxiety and (in cats) chronic idiopathic cystitis</td>
<td>**10-, 25-, 50-, 75-, 100-, and 150-mg tablets; 10 mg/mL injection</td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
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<td></td>
<td></td>
</tr>
<tr>
<td><strong>Dog:</strong> 1-2 mg/kg PO q12-24h (range 0.25-4 mg/kg q12-24h)</td>
<td><strong>Cat:</strong> 0.5-2 mg/kg q12-24h or approximately 5-10 mg per cat per day PO</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Amlodipine</strong> (Norvasc)</th>
<th><strong>Calcium channel blocker, vasodilator for systemic hypertension</strong></th>
<th><strong>2.5-, 5-, and 10-mg tablets</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dog:</strong> 2.5 mg/dog or 0.1-0.4 mg/kg PO q12-24h</td>
<td><strong>Cat:</strong> 0.625 mg/cat/day PO initially; then increase if needed to 1.25 mg/cat/day (average is 0.18 mg/kg once daily)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Ammonium chloride</strong></th>
<th><strong>Urinary acidifier; acidifies urine and treats metabolic alkalosis</strong></th>
<th><strong>Available as crystals</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dog:</strong> 200 mg/kg/day divided TID-QID</td>
<td><strong>Cat:</strong> 800 mg/cat (approximately 1/3 to 1/4 tsp) mixed with food daily</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Amoxicillin</strong> trihydrate</th>
<th><strong>Amoxi-Tabs, Amoxi-Drops, Amoxil, others</strong></th>
<th><strong>Broad-spectrum antibacterial</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>50-, 100-, 200-, and 400-mg tablets; 50 mg/mL oral suspension</strong></td>
<td><strong>6-20 mg/kg PO q8-12h</strong></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Amoxicillin-clavulanate</strong> (Clavamox)</th>
<th><strong>Broad-spectrum antibacterial</strong></th>
<th><strong>62.5-, 125-, 250-, and 375-mg tablets; 62.5 mg/mL suspension</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dog:</strong> 12.5-15 mg/kg PO q12h</td>
<td><strong>Cat:</strong> 62.5 mg/cat PO q12h; consider administering these doses q8h for gram-negative infections</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Amphotericin B</strong></th>
<th><strong>AmBisome (new formulation; less toxic but expensive)</strong></th>
<th><strong>Antifungal (liposomal formulation); deep, systemic fungal infection and leishmaniasis</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>50-mg injectable vial</strong></td>
<td><strong>3-5 mg/kg/day IV administered over 3-5 hours.</strong></td>
<td><strong>Caution:</strong> Use with care. Consider giving in 1/2 to 3-hour divided TID-QID doses. <strong>Note:</strong> Overdose may cause death.</td>
</tr>
<tr>
<td><strong>Fungizone (traditional formulation)</strong></td>
<td><strong>Antifungal; deep systemic fungal infection and leishmaniasis</strong></td>
<td><strong>50-mg injectable vial</strong></td>
</tr>
<tr>
<td><strong>0.5 mg/kg IV (slow infusion) q48h; cumulative dose is 4-8 mg/kg.</strong></td>
<td><strong>Caution:</strong> Use with care. Consider giving in 1/2 to 3-hour divided TID-QID doses. <strong>Note:</strong> Overdose may cause death.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Ampicillin</strong></th>
<th><strong>Omnipen, Principen, others</strong></th>
<th><strong>Broad-spectrum antibacterial</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>250- and 500-mg capsules; 125-, 250-, and 500-mg vials of ampicillin sodium</strong></td>
<td><strong>10-20 mg/kg IV, IM, SQ q6-8h (ampicillin sodium); 20-40 mg/kg PO q8h</strong></td>
<td></td>
</tr>
</tbody>
</table>

*Listings preceded by $ are for rapid reference and denote drugs of low or does used in the emergency or critical care setting.*
<table>
<thead>
<tr>
<th>Drug</th>
<th>Proprietary Names</th>
<th>Action and Use</th>
<th>Formulation</th>
<th>Recommended Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>§ Ampicillin + sulbactam</td>
<td>Unasyn</td>
<td>Broad-spectrum antibacterial</td>
<td>1.5- and 3-g vials in 2:1 combination for injection</td>
<td>20-50 mg/kg (total combined) IV, IM q8h</td>
</tr>
<tr>
<td>Ampicillin trihydrate</td>
<td>Polyflex</td>
<td>Broad-spectrum antibacterial</td>
<td>10- and 25-mg vials for injection</td>
<td>6.5-22 mg/kg IM, SQ q12h</td>
</tr>
<tr>
<td>Amprolium</td>
<td>Amprol, Corid</td>
<td>Thiamine analogue; treatment for coccidia</td>
<td>9.6% (9.6 g/dL) oral solution; soluble powder</td>
<td>1.25 g of 20% amprolium powder to daily feed, or 30 mL of 9.6% amprolium solution to 3.8 L of drinking water for 7 days</td>
</tr>
<tr>
<td>§ Antiserum, snakebite</td>
<td>Antivenin</td>
<td>Antivenin, concentrated serum globulin from horses immunized with multiple types of venom</td>
<td>10-mL vials</td>
<td>Dose varies from 10 to 50 mL (1 to 5 vials) initially; additional doses may be administered q2h after initial treatment</td>
</tr>
<tr>
<td>§ Apomorphine</td>
<td>Generic</td>
<td>Emetic (potent)</td>
<td>6-mg tablets</td>
<td>0.02-0.04 mg/kg IV, IM, 0.1 mg/kg SQ or instill 0.25 mg in conjunctiva of eye (dissolve 6-mg tablet in 1-2 mL of saline)</td>
</tr>
<tr>
<td>Ascorbic acid</td>
<td>Vitamin C</td>
<td>Vitamin supplement</td>
<td>Various forms</td>
<td>100-500 mg/animal/day (diet supplement) or 100 mg/animal q8h (urine acidification)</td>
</tr>
<tr>
<td>L-Asparaginase</td>
<td>Elspar</td>
<td>Antineoplastic; lymphoid malignancies</td>
<td>10,000 units per vial for injection</td>
<td>Dog: 10,000 to 20,000 international units/m² IV once weekly Cat: 400 units/kg SQ or IM (as part of a protocol) Pretreatment with antihistamine (diphenhydramine), 2 mg/kg (dog) and 1 mg/kg (cat) 30 minutes earlier is recommended</td>
</tr>
</tbody>
</table>

www.ajlobby.com
<table>
<thead>
<tr>
<th>Drug</th>
<th>Brand Name(s)</th>
<th>Type(s)</th>
<th>Formulations</th>
<th>Dog:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>Many generic and brand name products (e.g., Bufferin, Ascriptin)</td>
<td>NSAID; anticoagulant</td>
<td>81- and 325-mg tablets</td>
<td>Mild analgesia: 10-25 mg/kg q8-12h Antiinflammatory: 20-25 mg/kg q8-12h Antiplatelet: 5-10 mg/kg q24-48h To prevent thromboembolism (IMHA): 0.5 mg/kg/day Cat: 10-20 mg/kg q48h Antiplatelet: 80 mg q48h</td>
</tr>
<tr>
<td>Atenolol</td>
<td>Tenormin</td>
<td>β-blocker; hypertension and tachyarrhythmias</td>
<td>25-, 50-, and 100-mg tablets; 25 mg/mL oral suspension; and 0.5 mg/mL ampule for injection</td>
<td>Dog: 6.25-12.5 mg/dog q12h (or 0.25-1.0 mg/kg q12-24h) Cat: 6.25-12.5 mg/cat q12h (approximately 3 mg/kg)</td>
</tr>
<tr>
<td>§ Atipamezole</td>
<td>Antisedan</td>
<td>Treatment of amitraz toxicity</td>
<td>5.0 mg/mL, injection only</td>
<td>50 mcg/kg IM</td>
</tr>
<tr>
<td>Atracurium</td>
<td>Tracrium</td>
<td>Neuromuscular blocking agent; adjunct to general anesthesia for muscle relaxation</td>
<td>10-mg/mL injection</td>
<td>0.2 mg/kg IV initially; then 0.15 mg/kg q30min (or intravenous infusion at 3-8 mcg/kg/min)</td>
</tr>
<tr>
<td>§ Atropine</td>
<td>Many generic products</td>
<td>Antimuscarinic-anticholinergic; preanesthetic agent, treatment of some bradyarrhythmias</td>
<td>400-, 500-, and 540 mcg/mL injection; 15 mg/mL injection</td>
<td>0.02-0.04 mg/kg IV, IM, SQ q6-8h or 0.2-0.5 mg/kg (as needed) for organophosphate and carbamate toxicosis</td>
</tr>
<tr>
<td>Auranofin (triethylphosphine gold)</td>
<td>Ridaura</td>
<td>Gold compound; immune-mediated skin disease</td>
<td>3-mg capsule</td>
<td>0.1-0.2 mg/kg PO q12h</td>
</tr>
</tbody>
</table>

Listings preceded by § are for rapid reference and denote drug or dose used in the emergency or critical care setting.

Continued
<table>
<thead>
<tr>
<th>Drug</th>
<th>Proprietary Names</th>
<th>Action and Use</th>
<th>Formulation</th>
<th>Recommended Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aurothioglucose</td>
<td>Solganal</td>
<td>Gold compound; immune-mediated skin disease</td>
<td>50 mg/mL injection</td>
<td>Dog &lt;10 kg: 1 mg IM 1st wk, 2 mg IM 2nd wk, then 1 mg/kg/wk maintenance; &gt;10 kg: 5 mg IM 1st wk, 10 mg 2nd wk, and then 1 mg/kg/wk maintenance Cat: 0.5-1 mg/cat IM q7days</td>
</tr>
<tr>
<td>Azathioprine</td>
<td>Imuran</td>
<td>Purine antagonist; immunosuppressive agent</td>
<td>50-mg tablet; 10 mg/mL injection</td>
<td>Dog: 2 mg/kg PO q24h initially; then 0.5-1 mg/kg q48h Cat (use cautiously): 1 mg/kg PO q48h Monitoring patient CBC is indicated during therapy.</td>
</tr>
<tr>
<td>Azithromycin</td>
<td>Zithromax</td>
<td>Antibacterial, broad-spectrum activity with extended half-life in tissue</td>
<td>250-mg capsule; 250- and 600-mg tablets; 20 mg/mL oral suspension</td>
<td>Dog: 5-10 mg/kg PO once daily for 3-5 days or 5 mg/kg PO once daily for 2 days, then every 3-5 days for a total of 5 doses Cat: 5-10 mg/kg PO daily for 3-5 days</td>
</tr>
<tr>
<td>BAL</td>
<td>See Dimercaprol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Benazepril</td>
<td>Lotensin</td>
<td>ACE inhibitor; chronic heart failure, hypertension, first choice in treating protein-losing nephropathies</td>
<td>5-, 10-, 20-, and 40-mg tablets</td>
<td>Dog: Heart failure: 0.25-0.5 mg/kg PO q24h Hypertension: 0.25 mg/kg PO q12h Cat: Heart failure: 0.25-0.5 mg/kg PO once or twice daily Hypertension: 0.25-1.0 mg/kg PO once or twice daily</td>
</tr>
<tr>
<td>Betamethasone</td>
<td>Celestone</td>
<td>Potent glucocorticoid and antiinflammatory; immune-mediated disease</td>
<td>600-mcg (0.6-mg) tablet; 3 mg/mL sodium phosphate injection</td>
<td>Dog and cat: Antiinflammatory: 0.1-0.2 mg/kg PO q12-24h Immunosuppressive: 0.2-0.5 mg/kg once or twice daily</td>
</tr>
<tr>
<td>Drug</td>
<td>Generic Name</td>
<td>Description</td>
<td>Dosage</td>
<td></td>
</tr>
<tr>
<td>-------------------------------</td>
<td>--------------------</td>
<td>-----------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Bethanechol</td>
<td>Urecholine</td>
<td>Muscarinic-cholinergic; enhances urinary bladder contraction</td>
<td>Dog: 5-15 mg/dog PO q8h Cat: 1.25-5 mg/cat PO q8h</td>
<td></td>
</tr>
<tr>
<td>Bisacodyl</td>
<td>Dulcolax</td>
<td>Stimulant laxative</td>
<td>5 mg/animal PO q8-24h</td>
<td></td>
</tr>
<tr>
<td>Bismuth subsalicylate</td>
<td>Pepto-Bismol</td>
<td>GI protectant; treatment of simple (uncomplicated) diarrhea</td>
<td>0.25 mL/kg PO q4-6h up to 2 mL/kg q6-8h</td>
<td></td>
</tr>
<tr>
<td>Bleomycin</td>
<td>Blenoxane</td>
<td>Antineoplastic; used in multiple cancer protocols</td>
<td>Dog: 10 units/m² IV or SQ once daily for 3 days; then 10 units/m² weekly (maximum cumulative dose 200 units/m²)</td>
<td></td>
</tr>
<tr>
<td>Bromide</td>
<td>See Potassium bromide</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Bromocriptine mesylate        | Parlodel           | Dopamine agonist and prolactin inhibitor; pregnancy termination or pseudopregnancy (pseudocyesis) in dogs | Pseudocyesis: 10-100 mcg/kg PO once daily for 10 days, or 30 mcg/kg PO once daily for 16 days  
Pregnancy termination: 50-100 mcg/kg PO twice daily for 4-7 days; begin treatment from day 35-45 after LH surge  
Caution: Vomiting is a common side effect. |
| Bunamidine                    | Scolaban           | Antiparasitic; tape worms                                                   | 20-50 mg/kg PO per treatment                                                                                                          |
| Bupivicaine                   | Marcaine and generic | Local anesthetic (parenteral)                                               | 1 mL of 0.5% solution/10 cm for an epidural                                                                                          |
| Buprenorphine                 | Buprenex           | Partial opiate agonist analgesic                                            | Dog: 0.005-0.02 mg/kg IV, IM, SQ q6-12h  
Cat: 0.005-0.01 mg/kg IV, IM q6-12h  
Buccal administration is well tolerated in cats, and effect lasts approximately 6 hours.  
Dog: 1 mg/kg PO q8-12h (useful in social and panic disorders) |
| Buspirone                     | Buspar             | Nonbenzodiazepine anxiolytic; control of urine spraying                     | Cat: 2.5-5 mg/cat PO daily (may be increased to twice daily for some cats)                                                            |

Listings preceded by § are for rapid reference and denote drug or dose used in the emergency or critical care setting.
<table>
<thead>
<tr>
<th><strong>Drug</strong></th>
<th><strong>Proprietary Names</strong></th>
<th><strong>Action and Use</strong></th>
<th><strong>Formulation</strong></th>
<th><strong>Recommended Dosage</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Busulfan</td>
<td>Myleran</td>
<td>Oral antineoplastic; chronic granulocytic leukemia</td>
<td>2-mg tablet</td>
<td>3-4 mg/m² PO q24h</td>
</tr>
<tr>
<td>Butorphanol</td>
<td>Torbutrol, Torbugesic</td>
<td>Opioid analgesic; perioperative analgesia</td>
<td>1-, 5-, and 10-mg tablets; 0.5 or 10 mg/mL injection</td>
<td>Dog: Antitussive: 0.055 mg/kg SQ q6-12h or 0.55 mg/kg PO Preanesthetic: 0.2-0.4 mg/kg IV, IM, SQ (with acepromazine) Analgesic: 0.2-0.4 mg/kg IV, IM, SQ q2-4h or 0.55-1.1 mg/kg PO q6-12h Cat: Analgesic: 0.2-0.8 mg/kg IV, SQ q2-6h or 1.5 mg/kg PO q4-8h</td>
</tr>
<tr>
<td>Calcitriol</td>
<td>Rocaltrol, Calcijex</td>
<td>Calcium supplement; increases calcium absorption in the GI tract; used in management of hypoparathyroidism</td>
<td>Available as injection (Calcijex) and capsules (Rocaltrol): 0.25- and 0.5-mcg capsules; 1- or 2-mcg/mL injection</td>
<td>Dog: 2.5-3.5 mg/kg PO once daily Cat: 1.65-3.63 mg/kg PO daily</td>
</tr>
<tr>
<td>Calcium carbonate</td>
<td>Generic and many brand name products (e.g., Tums)</td>
<td>Calcium supplement</td>
<td>Many tablets or oral suspension (e.g., 650-mg tablet contains 260 mg calcium ion)</td>
<td>70-185 mg/kg/day PO in divided doses For phosphate binder: 60-100 mg/kg/day PO in divided doses</td>
</tr>
<tr>
<td>Calcium chloride</td>
<td>Generic</td>
<td>Calcium supplement (IV)</td>
<td>10% (100 mg/mL) solution</td>
<td>0.1-0.3 mL/kg IV (slowly)</td>
</tr>
<tr>
<td>Calcium citrate (OTC)</td>
<td>Citracal</td>
<td>Calcium supplement</td>
<td>950-mg tablet (contains 200 mg calcium ion)</td>
<td>Dog: 20 mg/kg/day PO (with meals) Cat: 10-30 mg/kg q6h PO (with meals)</td>
</tr>
<tr>
<td>Calcium gluconate</td>
<td>Kalcinate and generic</td>
<td>Calcium supplement (IV)</td>
<td>10% (100 mg/mL) injection</td>
<td>0.5-1.5 mL/kg IV (slowly)</td>
</tr>
<tr>
<td>Calcium lactate (OTC)</td>
<td>Generic</td>
<td>Calcium supplement</td>
<td>Available as a powder and various-sized tablets</td>
<td>Dog: 0.5-2.0 g/dog/day PO (in divided doses) Cat: 0.2-0.5 g/cat/day PO (in divided doses)</td>
</tr>
<tr>
<td>Drug</td>
<td>Brand Name</td>
<td>Class</td>
<td>Formulations</td>
<td>Dosages</td>
</tr>
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<td>-------------------------------</td>
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<td>------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Captopril</td>
<td>Capoten</td>
<td>ACE inhibitor (vasodilator); hypertension and congestive heart failure</td>
<td>25-mg tablet</td>
<td>Dog: 0.5-2 mg/kg PO q8-12h  Cat: 3.12-6.25 mg/cat PO q8h</td>
</tr>
<tr>
<td>Carbenicillin</td>
<td>Geopen, Pyopen</td>
<td>Antibacterial</td>
<td>1-, 2-, 5-, 10-, and 30-g vials for injection</td>
<td>No longer available in the US</td>
</tr>
<tr>
<td>Carbenicillin indanyl sodium</td>
<td>Geocillin</td>
<td>Antibacterial</td>
<td>500-mg tablet</td>
<td>22-33 mg/kg PO q8h for 7-10 days</td>
</tr>
<tr>
<td>Carboplatin</td>
<td>Paraplatin</td>
<td>Antineoplastic; multiple tumor types</td>
<td>50- and 150-mg vials for injection</td>
<td>Dog: 300 mg/m² IV q3-4 wk  Cat: 200 mg/m² IV q4wk</td>
</tr>
<tr>
<td>$ Carprofen</td>
<td>Rimadyl</td>
<td>NSAID</td>
<td>25-, 75-, and 100-mg tablets 50 mg/mL in 20-mL vials for injection</td>
<td>Dog: 2.2 mg/kg PO or SQ q12h; or 4.4 mg/kg PO or SQ once daily  Cat: Not approved for use in cats</td>
</tr>
<tr>
<td>Cascara sagrada (OTC)</td>
<td>Many brand name products</td>
<td>Laxative</td>
<td>100- and 325-mg tablets</td>
<td>Dog: 1-5 mg/kg/day PO  Cat: 1-2 mg/cat/day</td>
</tr>
<tr>
<td>Castor oil (OTC)</td>
<td>Generic</td>
<td>Laxative</td>
<td>Oral liquid (100%)</td>
<td>Dog: 8-30 mL/day PO  Cat: 4-10 mL/day PO</td>
</tr>
<tr>
<td>Cefaclor</td>
<td>Cefclor</td>
<td>Antibacterial</td>
<td>250- and 500-mg capsules and 25 mg/mL oral suspension</td>
<td>7-13 mg/kg PO q8h for 14-21 days</td>
</tr>
<tr>
<td>§ Cefadroxil</td>
<td>Cefa-Tabs, Cefa-Drops</td>
<td>Antibacterial</td>
<td>50 mg/mL oral suspension; 50-, 100-, 200-, and 1000-mg tablets for injection</td>
<td>Dog: 22-30 mg/kg PO q12h  Cat: 22 mg/kg PO q24h</td>
</tr>
<tr>
<td>Cefepime</td>
<td>Maxipime</td>
<td>Antibacterial</td>
<td>500-mg, 1-g, and 2-g vials for injection</td>
<td>40 mg/kg IV q6h</td>
</tr>
<tr>
<td>Cefixime</td>
<td>Suprax</td>
<td>Antibacterial</td>
<td>20 mg/mL oral suspension; 200- and 400-mg tablets</td>
<td>10 mg/kg PO q12h  For cystitis: 5 mg/kg PO q12-24h</td>
</tr>
<tr>
<td>Cefotaxime</td>
<td>Claforan</td>
<td>Antibacterial</td>
<td>500-mg and 1-, 2-, and 10-g vials for injection</td>
<td>Dog: 50 mg/kg IV, IM, SQ q12h  Cat: 20-80 mg/kg IV, IM q6h</td>
</tr>
<tr>
<td>Cefotetan</td>
<td>Cefotan</td>
<td>Antibacterial</td>
<td>1-, 2-, and 10-g vials for injection</td>
<td>30 mg/kg IV, SQ q8h</td>
</tr>
<tr>
<td>§ Cefoxitin</td>
<td>Mefoxin</td>
<td>Antibacterial</td>
<td>1-, 2-, and 10-g vials for injection</td>
<td>30 mg/kg IV q6-8h</td>
</tr>
</tbody>
</table>

Listings preceded by $ are for rapid reference and denote drug or dose used in the emergency or critical care setting.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Proprietary Names</th>
<th>Action and Use</th>
<th>Formulation</th>
<th>Recommended Dosage</th>
</tr>
</thead>
</table>
| **Ceftazidime**             | Fortaz, Ceptaz, Tazicef     | Antibacterial                                           | 0.5-, 1-, 2-, and 6-g vials reconstituted to 280 mg/mL | Dog and cat: 30 mg/kg IV, IM q6h  
CRI: Loading dose 1.2 mg/kg; then 1.56 mg/kg/hr with IV fluids |
| **Ceftiofur**               | Naxcel (ceftiofur sodium), Excenel (ceftiofur hydrochloride) | Antibacterial                                           | 50 mg/mL injection                               | Dog: 30 mg/kg, SQ, q4-6h  
2.2-4.4 mg/kg SQ q24h (for urinary tract infections) |
| **Cephalexin**              | Keflex and generic          | Antibacterial, especially skin, urinary, respiratory tract infections | 250- and 500-mg capsules; 250- and 500-mg tablets; 100 mg/mL or 125 and 250 mg/5 mL oral suspension | 10-30 mg/kg PO q6-12h  
Pyoderma: 22-35 mg/kg PO q12h |
| **Cephalothin sodium**      | Keflin                      | Antibacterial                                           | 1- and 2-g vials for injection                    | 10-30 mg/kg IV, IM q4-8h  |
| **Cephapirin**              | Cefadyl                     | Antibacterial                                           | 500-mg and 1-, 2-, and 4-g vials for injection    | 10-30 mg/kg IV, IM q4-8h  |
| **Charcoal, activated**     | Acta-Char, Charcodote, Toxiban, and generic | GI adsorbent                                           | Oral suspension                                   | 1-4 g/kg PO (granules) 6-12 mg/kg (suspension) |
| **Chlorambucil**            | Leukeran                    | Antineoplastic; has also been used to treat eosinophilic granuloma complex in cats | 2-mg tablet                                       | Dog: 2-6 mg/m² q24h initially; then q48h PO  
Cat: 0.1-0.2 mg/kg q48h initially; then q48h PO |
| **Chloramphenicol and chloramphenicol palmitate** | Chloromycetin and generic | Antibacterial                                           | 30 mg/mL oral suspension (palmitate); 250-mg capsule; and 100-, 250-, and 500-mg tablets | Dog: 45-60 mg/kg PO q8h  
Cat: 25-50 mg/kg PO q12h |
<table>
<thead>
<tr>
<th>Drug Name</th>
<th>Trade Name</th>
<th>Description</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chloramphenicol sodium succinate</td>
<td>Chloromycetin and generic</td>
<td>Antibacterial</td>
<td>100 mg/mL injection</td>
</tr>
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<td></td>
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<td></td>
<td>Dog: 40-50 mg/kg IV, IM q6-8h</td>
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<td></td>
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<td></td>
<td>Cat: 12.5-20 mg/cat IV, IM q12h</td>
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</tr>
<tr>
<td>Chlorothiazide</td>
<td>Diuril</td>
<td>Diuretic; also used as an antihypertensive</td>
<td>20-40 mg/kg PO q12h</td>
</tr>
<tr>
<td>Chlorpheniramine maleate (OTC)</td>
<td>Chlor-Trimeton, Phenetron, others</td>
<td>Antihistamine (H&lt;sub&gt;1&lt;/sub&gt;-blocker), weak antipruritic agent in allergic animals</td>
<td>4- and 8-mg tablets</td>
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<td>Dog: 4-8 mg/dog PO q12h (up to maximum of 0.5 mg/kg q12h)</td>
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<td></td>
<td></td>
<td>Cat: 2 mg/cat PO q12h</td>
</tr>
<tr>
<td>Chlorpromazine</td>
<td>Thorazine</td>
<td>Tranquilizer, antiemetic</td>
<td>0.5 mg/kg IM, SQ q6-8h</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>Before cancer chemotherapy: 2 mg/kg SQ q3h</td>
</tr>
<tr>
<td>Chlortetracycline</td>
<td>Generic</td>
<td>Antibacterial</td>
<td>Powdered feed additive</td>
</tr>
<tr>
<td>Chorionic gonadotropin</td>
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</tr>
<tr>
<td>Chlorothiazide</td>
<td>Diuril</td>
<td>Diuretic; also used as an antihypertensive</td>
<td>20-40 mg/kg PO q12h</td>
</tr>
<tr>
<td>Cimetidine</td>
<td>Tagamet (available OTC and prescription)</td>
<td>Antihistamine (H&lt;sub&gt;2&lt;/sub&gt;-blocker); treatment and prevention of gastric ulcer</td>
<td>10 mg/kg IV, IM, PO q6-8h</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>In renal failure: 2.5-5 mg/kg IV, PO q12h</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>Cipro (generic also available)</td>
<td>Antibacterial</td>
<td>5-15 mg/kg PO, IV q12h</td>
</tr>
<tr>
<td>Cisapride</td>
<td>Propulsid</td>
<td>Prokinetic; stimulates GI tract motility</td>
<td>Dog: 0.1-0.5 mg/kg PO q8-12h (doses as high as 0.5-1.0 mg/kg have been used in some dogs)</td>
</tr>
<tr>
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<td></td>
<td>Cat: 2.5-5 mg/cat PO q8-12h (doses as high as 1 mg/kg q8h have been used in cats)</td>
</tr>
<tr>
<td>Drug</td>
<td>Proprietary Names</td>
<td>Action and Use</td>
<td>Formulation</td>
</tr>
<tr>
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<td>------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Cisplatin</td>
<td>Platinol</td>
<td>Antineoplastic; multiple tumor types</td>
<td>1-mg/mL injection; 50-mg vial</td>
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<tr>
<td>Clemastine</td>
<td>Tavist, Contac 12</td>
<td>Antihistamine (H₁-blocker), antipruritic in allergic dogs</td>
<td>1.34-mg tablet (OTC); 2.64-mg tablet (prescription); 0.134 mg/mL syrup</td>
</tr>
<tr>
<td></td>
<td>Hour, and generic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$ Clindamycin</td>
<td>Antirobe, Cleocin</td>
<td>Antibacterial, especially gram-positive infections; recommended for toxoplasmosis (controversial)</td>
<td>25 mg/mL oral liquid; 25-, 75-, and 150-mg capsules; and 150-mg/mL injection (Cleocin)</td>
</tr>
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</tr>
<tr>
<td>Clofazimine</td>
<td>Lamprene</td>
<td>Antibacterial</td>
<td>50- and 100-mg capsules</td>
</tr>
<tr>
<td>Clomipramine</td>
<td>Anafranil (human);</td>
<td>Tricyclic antidepressant; behavior modification</td>
<td>10-, 25-, and 50-mg tablets (human); 5-, 20-, and 80-mg tablets (veterinary)</td>
</tr>
<tr>
<td></td>
<td>Clomicalm (veterinary)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clonazepam</td>
<td>Klonopin</td>
<td>Anticonvulsant; also used to manage certain types of behavior disorders</td>
<td>0.5-, 1-, and 2-mg tablets</td>
</tr>
<tr>
<td>Clorazepate</td>
<td>Tranxene</td>
<td>Anticonvulsant; also used to manage certain types of behavior disorders</td>
<td>3.75-, 7.5-, 11.25-, 15-, and 22.5-mg tablets</td>
</tr>
<tr>
<td>Clotrimazole</td>
<td>Many generic products,</td>
<td>Antifungal (topical only); nasal aspergillosis</td>
<td>1% topical solution in 30 mL</td>
</tr>
<tr>
<td></td>
<td>including lotrimazole</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>topical solution, USP 1%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note: Patient preparation is required.*
<table>
<thead>
<tr>
<th>Drug</th>
<th>Formulation</th>
<th>Category</th>
<th>Dose</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cloxacillin</td>
<td>Capsules; oral solution</td>
<td>Antibacterial</td>
<td>250- and 500-mg capsules; 25 mg/mL oral solution</td>
<td>20-40 mg/kg PO q8h</td>
</tr>
<tr>
<td>Codeine</td>
<td>Tablets; oral solution</td>
<td>Opioid analgesic</td>
<td>15-, 30-, and 60-mg tablets; 5 mg/mL syrup; 3 mg/mL oral solution</td>
<td>Analgesia: 0.5-2 mg/kg PO q6-8h Antitussive: 0.1-0.3 mg/kg PO q6-8h</td>
</tr>
<tr>
<td>Colchicine</td>
<td>Tablets; ampule</td>
<td>Antiinflammatory; hepatic failure</td>
<td>500- and 600-mcg tablets; 500 mcg/mL ampule injection</td>
<td>0.01-0.03 mg/kg PO q24h</td>
</tr>
<tr>
<td>Colony-stimulating factor</td>
<td>Injection</td>
<td>Hormone; stimulates granulocyte production in bone marrow</td>
<td>300 mcg/mL injection</td>
<td>5 mcg/kg SQ q24h for 5 days at a time (pulse therapy)</td>
</tr>
<tr>
<td>§ Corticotropin (ACTH gel)</td>
<td>Injection</td>
<td>Hormone; diagnostic test drug for the diagnosis of hyperadrenocorticism and hypoadrenocorticism</td>
<td>5 mL (multiple dose) 80 USP units/mL</td>
<td>Response test: Collect pre-ACTH sample and inject 2.2 international units/kg IM; Dog: Collect post-ACTH sample in 2 hr Cat: Collect post-ACTH samples at 1 and 2 hr</td>
</tr>
<tr>
<td>§ Cosyntropin (Cortrosyn)</td>
<td>Vial</td>
<td>Hormone; diagnostic test drug for the diagnosis of hyperadrenocorticism and hypoadrenocorticism</td>
<td>250 mcg per vial (can be stored in freezer for 6 months)</td>
<td>Response test: Dog: Collect presample and inject 5 mcg/kg IV Cat: Collect presample and inject 0.125 mg IV Dog and cat: Collect postsample 1 hr postadministration</td>
</tr>
<tr>
<td>Cyanocobalamin</td>
<td>See Vitamin B₁₂</td>
<td>Antineoplastic; multiple tumor types, and adjunctive in immune-mediated disorders</td>
<td>25 mg/mL injection; 25- and 50-mg tablets</td>
<td>Anticancer: 50 mg/m² PO once daily 4 days/wk or 150-300 mg/m² IV and repeat in 21 days Immunosuppressive therapy: 50 mg/m² (approx 2.2 mg/kg) PO q48h or 2.2 mg/kg once daily for 4 days/wk Cat: 6.25-12.5 mg/cat once daily 4 days/wk</td>
</tr>
<tr>
<td>Cyclophosphamide</td>
<td>Tablets; injection</td>
<td>Antineoplastic; multiple tumor types, and adjunctive in immune-mediated disorders</td>
<td>25 mg/mL injection; 25- and 50-mg tablets</td>
<td>Anticancer: 50 mg/m² PO once daily 4 days/wk or 150-300 mg/m² IV and repeat in 21 days Immunosuppressive therapy: 50 mg/m² (approx 2.2 mg/kg) PO q48h or 2.2 mg/kg once daily for 4 days/wk Cat: 6.25-12.5 mg/cat once daily 4 days/wk</td>
</tr>
<tr>
<td>Drug</td>
<td>Proprietary Names</td>
<td>Action and Use</td>
<td>Formulation</td>
<td>Recommended Dosage</td>
</tr>
<tr>
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</tr>
<tr>
<td>Cyclosporine (cyclosporin A)</td>
<td>Neoral, Sandimmune, Optimmune (ophthalmic)</td>
<td>Immunosuppressant (CMI); multiple uses ranging from atopic dermatitis to hemolytic anemia to perianal fistulas. Consult additional references before prescribing</td>
<td>Neoral: 25-mg and 100-mg microemulsion capsules; 100-mg/mL oral solution (for microemulsion) Sandimmune: 100-mg/mL oral solution; 25- and 100-mg capsules Optimmune: 0.2% ointment</td>
<td>Dog: 3-7 mg/kg PO q12-24h (adjust dose based on condition being treated and by monitoring blood levels) Hemolytic anemia: Up to 10 mg/kg PO q12h (as adjunctive therapy) Cat: 4-6 mg/kg PO q12h Note: Multiple products are available but all are not bioequivalent.</td>
</tr>
<tr>
<td>Cyproheptadine</td>
<td>Periactin</td>
<td>Antihistamine; appetite stimulant in cats</td>
<td>4-mg tablet; 2 mg/5 mL syrup</td>
<td>Antihistamine: 1.1 mg/kg PO q8-12h Appetite stimulant: 2 mg/cat PO</td>
</tr>
<tr>
<td>Cytarabine (cytosine arabinoside)</td>
<td>Cytosar-U</td>
<td>Antineoplastic; lymphoma and leukemia</td>
<td>100-mg vial</td>
<td>Dog (lymphoma): 100 mg/m² IV, SQ once daily or twice daily for 4 days Cat: 100 mg/m² once daily for 2 days</td>
</tr>
<tr>
<td>Dacarbazine</td>
<td>DTIC</td>
<td>Antineoplastic; lymphoreticular neoplasms and soft tissue sarcomas</td>
<td>200-mg vial for injection</td>
<td>200 mg/m² IV for 5 days q3wk; or 800-1000 mg/m² IV q3wk</td>
</tr>
<tr>
<td>Dalteparin</td>
<td>Fragmin</td>
<td>Low–molecular-weight heparin; management of thromboembolic disease</td>
<td>Multiple injectable preparations</td>
<td>Prophylaxis: 70 units/kg SQ q24h Treatment: Dog: 100-150 units/kg SQ q24h Cat: 180 units/kg SQ q24h</td>
</tr>
<tr>
<td>Danazol</td>
<td>Danocrine</td>
<td>Anabolic steroid; adjunctive therapy for immune-mediated disease</td>
<td>50-, 100-, and 200-mg capsules</td>
<td>5-10 mg/kg PO q12h</td>
</tr>
<tr>
<td>Dantrolene</td>
<td>Dantrium</td>
<td>Muscle relaxant; urethral obstruction, and prevention of malignant hyperthermia</td>
<td>100-mg capsules; 0.33 mg/mL injection</td>
<td>Malignant hyperthermia: 2-3 mg/kg IV Muscle relaxation: Dog: 1-5 mg/kg PO q8h Cat: 0.5-2 mg/kg PO q12h</td>
</tr>
</tbody>
</table>
Desferal

See Selegiline

§ Deferoxamine

Deprenyl
(l-deprenyl)
§ Desmopressin
acetate

www.ajlobby.com

Benylin and others

D5W

Dextromethor­phan

§ Dextrose
solution 5% in
water

Antitussive, weak cough
suppressant
Replacement fluid

Mineralocorticoid;
hypoadrenocorticism
Glucocorticoid; multiple
uses as
antiinflammatory and
immunosuppressive
agent; also used in the
diagnosis of
hyperadrenocorticism

Hormone; used in the
clinical management of
patients with DI and
patients with von
Willebrand disease

Antibacterial;
Mycobacterium species
Antidote, iron toxicosis

Available in syrup, capsule, and
tablet; many OTC products
Fluid solution for IV
administration

40-50 mL/kg IV q24h

Continued

Antiinflammatory: Dog: 0.5-1 mg IV or
IM q24h for 3-5 days or 0.25-1.25 mg
PO q24h
Cat: 0.125-0.5 mg IV or IM for 3-5 days
or 0.125-0.5 mg PO q24h
For shock, spinal injury: 2.2-4.4 mg/kg IV
(of sodium phosphate form) For
diagnostic testing: See Dexamethasone
Suppression Test in Section 5
0.5-2 mg/kg PO q6-8h

DI: 1-4 drops q12-24h in the conjunctival
sac or 2-5 mcg SQ q12-24h animal oral
dose not established, but dose
extrapolated from humans is 0.05 mg/
animal q12h PO with increase to 0.1 or
0.2 mg/animal as needed von Willebrand
disease: 1 mcg/kg SQ, IV diluted in
20 mL saline administered over 10 min
1.5-2.2 mg/kg IM q25days

100 mcg/mL injection and
desmopressin acetate nasal
solution (0.01% metered spray);
0.1- and 0.2-mg tablets

25 mg/mL suspension for
injection
Azium solution, 2 mg/mL;
sodium phosphate forms are
3.33 mg/mL; 0.25-, 0.5-, 1-,
1.5-, 2-, 4-, and 6-mg tablets.

10 mg/kg IV, IM q2h for two doses; then
10 mg/kg q8h for 24 hr

1.1 mg/kg PO q8-12h

500-mg vial for injection

25- and 100-mg tablets

Listings preceded by § are for rapid reference and denote drug or dose used in the emergency or critical care setting.

Percorten-V, DOCP,
DOCA pivalate
Azium solution in
polyethylene glycol;
sodium phosphate
forms include
DexaJect SP, Dexavet,
and Dexasone; tablets
include Decadron and
generic

Desoxycorticost­
erone pivalate
Dexamethasone
(dexamethasone
solution and
dexamethasone
sodium
phos­phate)

DDAVP

Generic

Dapsone

Charts and Tables
683

6


<table>
<thead>
<tr>
<th>Drug</th>
<th>Proprietary Names</th>
<th>Action and Use</th>
<th>Formulation</th>
<th>Recommended Dosage</th>
</tr>
</thead>
</table>
| $ Diazeplan         | Valium and generic| Anticonvulsant; multiple neurotropic effects ranging from behavior disorders to seizure control | 2- and 5-mg tablets; 5 mg/mL solution for injection | Preanesthetic: 0.1 mg/kg IV slowly  
Status epilepticus: 0.5 mg/kg IV, 1.0 mg/kg rectal; repeat if necessary  
Appetite stimulant (cat): 0.05-0.4 mg/kg IV, IM or PO |
| $ Dichlorphenamide  | Daranide          | Diuretic; management of glaucoma              | 50-mg tablet                     | 3-5 mg/kg PO q8-12h                                                              |
| Dichlorvos          | Task              | Antiparasitic; roundworms, hookworms, whipworms | 10- and 25-mg tablets           | Dog: 26.4-33 mg/kg PO  
Cat: 11 mg/kg PO |
| Dicloxacillin       | Dynapen           | Antibacterial                                 | 125-, 250-, and 500-mg capsules; 12.5 mg/mL oral suspension | 25 mg/kg PO q6h  
Oral doses not absorbed |
| Diethylcarbamazine (DEC) | Caricide, Filaribits | Antiparasitic; prevention of heartworm disease in dogs; treatment of ascarids in cats | Chewable tablets; 50-, 60-, 180-, 200-, and 400-mg tablets | Heartworm prophylaxis: 6.6 mg/kg PO q24h  
Cat (for ascarids): 55-110 mg/kg PO once |
| Diethylstilbestrol (DES) | Limited availability; compounding required | Hormone; estrogen replacement and urinary incontinence; induces abortion in dogs | Tablets (prepared through compounding pharmacies) | Dog: 0.1-1.0 mg/dog PO q24h for 5 days, then 1 mg PO once weekly  
Cat: 0.1-1 mg/cat PO q24h for 5 days, then 1 mg PO once weekly |
<p>| Difloxacin          | Dicural           | Antibacterial                                 | 11.4-, 45.4-, and 136-mg tablets | 5-10 mg/kg/day PO                                                               |
| Digitoxin           | Crystodigin       | Cardiac inotrope; congestive heart failure and management of various tachyarrhythmias | 0.05- and 0.1-mg tablets        | 0.02-0.03 mg/kg PO q8h |</p>
<table>
<thead>
<tr>
<th>Drug</th>
<th>Trade Name(s)</th>
<th>Description</th>
<th>Dosage</th>
<th>Dosage Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digoxin</td>
<td>Lanoxin, Cardoxin</td>
<td>Cardiac inotrope; congestive heart failure and management of various tachyarrhythmias</td>
<td>0.0625-, 0.125-, 0.25-mg tablets; 0.05 and 0.15 mg/mL elixir</td>
<td>Dog: &lt;20 kg, 0.01 mg/kg q12h; &gt;20 kg, 0.22 mg/m² PO q12h (subtract 10% for elixir)</td>
</tr>
<tr>
<td></td>
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<td>Dog (rapid digitalization): 0.0055-0.011 mg/kg IV q1h to effect</td>
<td>Cat: 0.008-0.01 mg PO q48h (approximately ⅈ of a 0.125-mg tablet/cat)</td>
</tr>
<tr>
<td>Dihydrota-</td>
<td>See Vitamin D analogue</td>
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<tr>
<td>chysterol (DHT)</td>
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<tr>
<td>§ Diltiazem</td>
<td>Cardizem, Dilacor</td>
<td>Calcium channel blocker; hypertension; also supraventricular tachycardia and hypertrophic cardiomyopathy</td>
<td>30-, 60-, 90-, and 120-mg tablets; 50 mg/mL injection</td>
<td>Dog: 0.5-1.5 mg/kg PO q8h; 0.25 mg/kg over 2 min IV (repeat if necessary)</td>
</tr>
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<td></td>
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<td></td>
<td>Cat: 1.75-2.4 mg/kg PO q8h; for Dilacor XR or Cardizem CD, dose is 10 mg/kg PO once daily</td>
<td></td>
</tr>
<tr>
<td>§ Dimenhy-</td>
<td>Dramamine</td>
<td>Antihistamine; prevention of motion sickness</td>
<td>50-mg tablets; 50 mg/mL injection</td>
<td>Dog: 4-8 mg/kg PO, IM, IV q8h; 12.5 mg/cat IV, IM, PO q8h</td>
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<td>drinate</td>
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<tr>
<td>Dimercaprol (BAL)</td>
<td>BAL in oil</td>
<td>Chelating agent; binds heavy metals (lead, mercury) and arsenicals</td>
<td>100 mg/mL injection</td>
<td>2.5-5 mg/kg IM q4h for 2 days, then q8h for 1 day, then q12h for next 10 days</td>
</tr>
<tr>
<td>Dinoprost trometam</td>
<td>See Prostaglandin F2-alpha</td>
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<tr>
<td>Dioctyl calcium</td>
<td>See Docusate calcium</td>
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<tr>
<td>sulfo succinate</td>
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<tr>
<td>Dioctyl sodium</td>
<td>See Docusate sodium</td>
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</tr>
</tbody>
</table>

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<tr>
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<th>Action and Use</th>
<th>Formulation</th>
<th>Recommended Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>$ Diphenhydramine</td>
<td>Benadryl</td>
<td>Antihistamine; weak sedative, prevents motion sickness</td>
<td>Available OTC: 2.5 mg/mL elixir; 25- and 50-mg capsules and tablets; 50 mg/mL injection</td>
<td>2-4 mg/kg PO q6-8h or 1 mg/kg IM, IV (for dogs, administer 25-50 mg/dog IV, IM, PO q8h)</td>
</tr>
<tr>
<td>Diphenoxylate</td>
<td>Lomotil</td>
<td>Meperidine congener; treatment of diarrhea</td>
<td>2.5-mg tablet</td>
<td>Dog: 0.1-0.2 mg/kg PO q8-12h</td>
</tr>
<tr>
<td>Diphenylhydantoin</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Diphosphonate disodium</td>
<td>See Etidronate disodium</td>
<td></td>
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</tr>
<tr>
<td>Dipyridamole</td>
<td>Persantine</td>
<td>Anticoagulant; prevention of thromboembolism</td>
<td>25-, 50-, 75-mg tablets; 5 mg/mL injection</td>
<td>4-10 mg/kg PO q24h</td>
</tr>
<tr>
<td>Disopyramide</td>
<td>Norpace</td>
<td>Antiarrhythmic in dogs; oral treatment or prevention of ventricular arrhythmias (dog only)</td>
<td>100- and 150-mg capsules</td>
<td>Dog: 11-22 mg/kg, PO, q8h</td>
</tr>
<tr>
<td>Divalproex sodium</td>
<td>See Valproic acid</td>
<td></td>
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</tr>
<tr>
<td>$ Dobutamine</td>
<td>Dobutrex</td>
<td>Rapid acting cardiac inotrope (β-agonist); short-term treatment of heart failure</td>
<td>250 mg/20 mL vial for injection (12.5 mg/mL)</td>
<td>Dog: 5-20 mcg/kg/min intravenous infusion Cat 0.5-2 mcg/kg/min intravenous infusion Warning: may induce arrhythmias, facial twitching, or seizure (cats)</td>
</tr>
<tr>
<td>Docusate calcium</td>
<td>Surfak, Doxidan</td>
<td>Stool softener</td>
<td>60-mg tablet (and many others)</td>
<td>Dog: 50-100 mg/dog PO q12-24h Cat 50 mg/cat PO q12-24h</td>
</tr>
<tr>
<td>Docusate sodium</td>
<td>Colace, Doxan, Doss, many OTC products</td>
<td>Stool softener</td>
<td>50- and 100-mg capsules; 10 mg/mL liquid</td>
<td>Dog: 50-200 mg/dog PO q8-12h Cat 50 mg/cat PO q12-24h</td>
</tr>
<tr>
<td>$ Dolasetron</td>
<td>Anzemet</td>
<td>5-HT3 receptor antagonist, antiemetic</td>
<td>50- and 100-mg tablets; 20 mg/mL injection</td>
<td>Prevention: 0.6 mg/kg PO or IV q24h Treatment: 1 mg/kg PO or IV q24h</td>
</tr>
<tr>
<td>Drug</td>
<td>Brand Name</td>
<td>Description</td>
<td>Dosage</td>
<td>Notes</td>
</tr>
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<td>------------</td>
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</tr>
<tr>
<td>1-Dopa</td>
<td>See Levodopa</td>
<td>Cardiac inotrope (β-agonist); vasodilation (lower doses); adjunctive treatment of acute heart failure and oliguric renal failure</td>
<td>40-, 80-, or 160-mg/mL injection</td>
<td>2-10 mcg/kg/min by intravenous infusion; treatment limited to the critical care setting</td>
</tr>
<tr>
<td>Doxapram</td>
<td>Dopram</td>
<td>CNS stimulant; stimulates respiration, especially in neonates</td>
<td>20-mg/mL injection</td>
<td>5-10 mg/kg IV</td>
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<td>Neonate: 1-5 mg SQ, sublingually, or via umbilical vein</td>
</tr>
<tr>
<td>Doxepin</td>
<td>Sinequan</td>
<td>Tricyclic antidepressant; psychogenic dermatoses</td>
<td>Various capsules; 10 mg/mL oral solution</td>
<td>Dog: 3-5 mg/kg PO q12h (especially lick granuloma)</td>
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<tr>
<td></td>
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<td></td>
<td>Cat: 0.5-1 mg/kg PO q12h</td>
</tr>
<tr>
<td>Doxorubicin</td>
<td>Adriamycin</td>
<td>Antineoplastic (antibiotic); used in treatment protocols for multiple tumor types</td>
<td>2 mg/mL injection</td>
<td>30 mg/m² IV q21days; or &gt;20 kg, 30 mg/m² &lt;20 kg, 1 mg/kg</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>Cat: 1 mg/kg IV</td>
</tr>
<tr>
<td>Doxycycline</td>
<td>Vibramycin and generic</td>
<td>Antibacterial</td>
<td>10 mg/mL oral suspension; 100-mg tablet; 100-mg injection vial</td>
<td>3-5 mg/kg PO, IV q12h; or 10 mg/kg PO q24h</td>
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<tr>
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<td></td>
<td></td>
<td>For Rickettsia in dogs: 5 mg/kg q12h</td>
</tr>
<tr>
<td>Edrophonium</td>
<td>Tensilon, others</td>
<td>Short-acting cholinergic; administered as a test agent for myasthenia gravis</td>
<td>10 mg/mL injection</td>
<td>Dog: 0.11-0.22 mg/kg IV</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td>Cat: 0.25-0.5 mg/cat IV</td>
</tr>
<tr>
<td>EDTA</td>
<td>Calcium disodium versenate</td>
<td>Chelates heavy metals; treatment of lead or zinc toxicosis</td>
<td>20 mg/mL injection</td>
<td>25 mg/kg SQ, IM, IV q6h for 2-5 days</td>
</tr>
</tbody>
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<tr>
<td>Enalapril</td>
<td>Enacard</td>
<td>ACE inhibitor; vasodilator used in the treatment of heart failure and/or hypertension; also used in the treatment of patients with protein-losing nephropathies and chronic renal failure</td>
<td>2.5-, 5-, 10-, and 20-mg tablets</td>
<td>Dog: 0.5 mg/kg PO q12-24h</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cat: 0.25-0.5 mg/kg PO q12-24h</td>
</tr>
<tr>
<td>Enflurane</td>
<td>Ethane</td>
<td>Inhalation anesthetic</td>
<td>Available as solution for inhalation</td>
<td>Induction: 2%-3%</td>
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<tr>
<td></td>
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<td>Maintenance: 1.5%-3%</td>
</tr>
<tr>
<td>Enilconazole</td>
<td>Imaverol, Clinafarm-EC</td>
<td>Antifungal (topical only); infusion for treatment of nasal aspergillosis and topical uses in certain dermatophytoses</td>
<td>10% or 13.8% emulsifiable concentrate</td>
<td>Nasal aspergillosis: 10 mg/kg q12h instilled into nasal sinus via surgically implanted tubes for 14 days (10% solution diluted 50/50 with water)—this is nasty!</td>
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<td>Note: Generally replaced by clotrimazole soak (see Clotrimazole)</td>
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<td></td>
<td>Dermatophytes: Dilute 10% solution to 0.2% and wash lesion with solution four times at 3- to 4-day intervals</td>
</tr>
<tr>
<td>$ Enoxaparin</td>
<td>Lovenox</td>
<td>Low–molecular-weight heparin; thromboembolic disease</td>
<td>Multiple preparations</td>
<td>Dog: 0.8 mg/kg SQ q12h</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cat: 0.5-1 mg/kg SQ q6h</td>
</tr>
<tr>
<td>§ Enrofloxacin</td>
<td>Baytril</td>
<td>Antibacterial</td>
<td>68-, 22.7-mg, and 5.7-mg tablets; Taste Tabs are 22.7 and 68 mg; 22.7 mg/mL injection</td>
<td></td>
</tr>
<tr>
<td>Dog: 5-20 mg/kg PO or IM once daily or divided twice daily</td>
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<tr>
<td>Cat: 2.5-5 mg/kg PO, SQ, IM once to twice daily</td>
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<tr>
<td>Parenteral solution for intramuscular use has been administered by the intravenous route—administer slowly if indicated.</td>
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<tr>
<td>Warning: Doses of 10 mg/kg and higher are not recommended in cats because of risk of drug-induced retinal damage and blindness.</td>
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</tbody>
</table>

| § Ephedrine | Ephedrine sulfate | Sympathomimetic; primarily for urinary incontinence |
| Emergency use: Hypotension associated with anesthesia |
| 25-mg capsules and 50 mg/mL in 1-mL ampules for injection |
| Urinary incontinence: |
| Dog: 4 mg/kg, or 12.5-50 mg/dog (total) PO q8-12h; Also, 1-2 mg/kg PO q8h, or 5 to 15 mg/dog (total) q8h |
| Cat: 2-4 mg/kg, PO q8-12h |
| Hypotension: 0.03-0.1 mg/kg intravenous bolus |
| Note: Dilute 5 mg in 10 mL saline; give the lower dose first; may repeat in 5 minutes after first dose if hypotension does not improve. |

| § Epinephrine | Adrenalin and generic products (adrenaline) | α- and β-Adrenergic agonist; anaphylaxis and cardiac arrest |
| 1 mg/mL (1:1,000) injection solution |
| Cardiac arrest: 10-20 mcg/kg IV or 200 mcg/kg intratracheal (may be diluted in saline) |
| Anaphylaxis: 2.5-5 mcg/kg IV or 50 mcg/kg intratracheal (may be diluted in saline) |

| Epsiprantel | Cestex | Oral cesticide; tapeworms |
| Coated tablet |
| Dog: 5.5 mg/kg PO given once |
| Cat: 2.75 mg/kg PO given once |

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<tbody>
<tr>
<td>Ergocalciferol</td>
<td>See Vitamin D&lt;sub&gt;2&lt;/sub&gt;</td>
<td>Antibacterial; also used as a prokinetic (increases gastric emptying in dogs and cats)</td>
<td>250-mg capsule or tablet</td>
<td>Antibacterial dose: 10-20 mg/kg PO q8-12h</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>Many brand name and generic products</td>
<td></td>
<td></td>
<td>Prokinetic dose: 0.5-1.0 mg/kg PO q8h</td>
</tr>
<tr>
<td>Erythropoietin, human recombinant (rHuEPO); also, EPO</td>
<td>Epogen, Eprex, Procrit</td>
<td>Hormone; induction of erythropoiesis in anemia associated with chronic renal failure</td>
<td>Various preparation as units per milliliter in single-dose and multidose vials for injection</td>
<td>Doses range from 35 or 50 units/kg three times per week to 400 units/kg/wk IV, SQ (adjust dose to hematocrit of 0.30-0.34)</td>
</tr>
<tr>
<td>Esmolol</td>
<td>Brevibloc</td>
<td>Ultra-short-acting β&lt;sub&gt;1&lt;/sub&gt; blocker; short-term treatment of cardiac arrhythmias, especially supraventricular tachycardia</td>
<td>10 mg/mL injection</td>
<td>500 mcg/kg IV, which may be given as 0.05-0.1 mg/kg slowly every 5 minutes or 50-200 mcg/kg/min infusion</td>
</tr>
<tr>
<td>Estradiol cypionate (ECP)</td>
<td>DEPO-Estradiol, generic</td>
<td>Hormone; previously used to prevent pregnancy after an unplanned breeding</td>
<td>2 mg/mL injection</td>
<td>Pregnancy avoidance:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Warning: Not recommended for use as an abortifacient in dogs or cats</td>
<td></td>
<td>Dog: 22-44 mcg/kg IM (total dose not to exceed 1.0 mg)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Warning: May cause bone marrow suppression; in some cases, may cause aplastic anemia</td>
<td></td>
<td>Cat: 250 mcg/cat IM between 40 hr and 5 days of mating</td>
</tr>
<tr>
<td>Etidronate</td>
<td>Didronel</td>
<td>Bisphosphonate; reduced calcium resorption from bone in hypercalcemic patients</td>
<td>200- and 400-mg tablets; 50 mg/mL injection</td>
<td>Dog: 5 mg/kg/day PO</td>
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<td></td>
<td></td>
<td>Cat: 10 mg/kg/day PO</td>
</tr>
<tr>
<td>Brand Name</td>
<td>Generic Name</td>
<td>Description</td>
<td>Dosage</td>
<td>Dog:</td>
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<tr>
<td>Etodolac</td>
<td>EtoGesic</td>
<td>Oral NSAID; pain management in dogs</td>
<td>150- and 300-mg tablets</td>
<td>10-15 mg/kg PO once daily</td>
</tr>
<tr>
<td>§ Famotidine</td>
<td>Pepcid</td>
<td>H₂-receptor antagonist; reduces gastric acid production, used to treat or prevent gastric ulcer</td>
<td>10-mg tablet; 10 mg/mL injection</td>
<td>0.5 mg/kg IM, SQ, IV, or PO q12-24h</td>
</tr>
<tr>
<td>Felbamate</td>
<td>Felbatol</td>
<td>Dicarbamate anticonvulsant; management of seizures in dog only</td>
<td>400- and 600-mg tablets; 120 mg/mL flavored oral suspension</td>
<td>Dog: Start with 15 mg/kg PO q8h and increase gradually to maximum of 65 mg/kg q8h</td>
</tr>
<tr>
<td>Fenbendazole</td>
<td>Safe-Guard, Panacur</td>
<td>Anthelmintic; effective against a variety of internal parasites</td>
<td>Panacur granules 22.2% (222 mg/kg); 100 mg/mL liquid</td>
<td>25 to 50 mg/kg/day PO for 3 days</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>Sublimaze, generic</td>
<td>Analgesic (opiate); parenteral pain control</td>
<td>250 mg/5 mL injection</td>
<td>0.02-0.04 mg/kg IV, IM, SQ q2h; or 0.01 mg/kg IV, IM, SQ (with acetylpromazine or diazepam)</td>
</tr>
<tr>
<td>Fentanyl transdermal</td>
<td>Duragesic</td>
<td>Analgesic (opiate); transdermal pain control</td>
<td>25-, 50-, 75-, and 100-mcg/hr patches</td>
<td>Dog: 10-20 kg, 50 mcg/hr patch q72h</td>
</tr>
<tr>
<td>§ Ferrous sulfate (OTC)</td>
<td>Generic</td>
<td>Oral iron supplement; iron deficiency anemia</td>
<td>Many oral preparations available</td>
<td>Dog: 100-300 mg/dog PO q24h</td>
</tr>
<tr>
<td>Finasteride</td>
<td>Proscar</td>
<td>5α-reductase inhibitor; benign prostatic hyperplasia in dogs</td>
<td>5-mg tablet</td>
<td>Dog: 0.1 mg/kg PO q24h or 5 mg/10- to 50-kg dog PO q24h</td>
</tr>
</tbody>
</table>

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<tbody>
<tr>
<td>Fipronil</td>
<td>Frontline</td>
<td>GABA-regulated chloride channel inhibitor; topical control of ticks and fleas</td>
<td>Topical solution only</td>
<td>Applied topically once each month as recommended by the manufacturer; approved for use in both dogs and cats</td>
</tr>
<tr>
<td>Firocoxib</td>
<td>Previcox</td>
<td>NSAID; management of inflammation and pain associated with osteoarthritis in dogs</td>
<td>57-mg and 227-mg chewable tablets</td>
<td>Dogs: 5 mg/kg, PO, once daily Cat: 1.5 mg/kg administered as a single dose Long-term safety has not been established in cats.</td>
</tr>
<tr>
<td>Florfenicol</td>
<td>Nuflor</td>
<td>Antibacterial (primarily used in cattle)</td>
<td>300 mg/mL (available only as a cattle preparation)</td>
<td>Dog: 25-50 mg/kg q8h SQ or IM Cat: 25-50 mg/kg q12h SQ or IM</td>
</tr>
<tr>
<td>Fluconazole</td>
<td>Diflucan</td>
<td>Antifungal, oral (dog and cat) or parenteral (dog only); treatment for systemic deep mycoses or nasal fungal infection</td>
<td>50-, 100-, 150-, and 200-mg tablets; 10 or 40 mg/mL oral suspension; 2 mg/mL intravenous injection</td>
<td>Dog: 2.5-5.0 mg/kg once daily PO or IV Cat: 2.5-10 mg/cat PO q12h; or 25 mg/cat/day PO</td>
</tr>
<tr>
<td>Flucytosine</td>
<td>Ancobon</td>
<td>Antifungal; treatment of systemic mycoses</td>
<td>250-mg capsule; 75 mg/mL oral suspension</td>
<td>25-50 mg/kg PO q6-8h (up to a maximum dose of 100 mg/kg PO q12h)</td>
</tr>
<tr>
<td>Fludrocortisone</td>
<td>Florinef</td>
<td>Mineralocorticoid; treatment of hypoadrenocorticism</td>
<td>100-mcg (0.1-mg) tablet</td>
<td>Dog: 0.2-0.8 mg/dog or 0.02 mg/kg PO q24h (13-23 mcg/kg) Cat: 0.1-0.2 mg/cat PO q24h</td>
</tr>
<tr>
<td>§ Flumazenil</td>
<td>Romazicon</td>
<td>Benzodiazepine antagonist; antidote to reverse therapeutic effects or overdose</td>
<td>100 mcg/mL (0.1 mg/mL) injection</td>
<td>0.01-0.02 mg (total dose) IV as needed Caution: May cause significant hypotension</td>
</tr>
<tr>
<td>Flumethasone</td>
<td>Flucort</td>
<td>Oral glucocorticoid; antiinflammatory</td>
<td>0.5 mg/mL injection</td>
<td>Dog: 0.0625-0.25 mg/day in divided doses IV, IM, SQ Cat: 0.03-0.125 mg/day IV, IM, SQ</td>
</tr>
</tbody>
</table>
### Flunixin meglumine
**Banamine**
NSAID; pain management
250-mg packet granules; 10 and 50 mg/mL injection
PO 3 days/wk
Ophthalmic: 0.5 mg/kg IV once

### 5-Fluorouracil (5-FU)
**Fluorouracil**
Antineoplastic; used in treatment protocols for multiple tumor types
50-mg/mL vial
Dog: 150 mg/m² IV once/week
Cat: Do not use

### Fluoxetine
**Prozac**
SSRI; treatment of behavior disorders
10- and 20-mg capsules; 4 mg/mL oral solution
Dog: 0.5 mg/kg day initially PO; then increase to 1 mg/kg/day PO (10-20 mg/dog)
Cat: 0.5-4 mg/cat PO q24h

### Fluvoxamine
**Luvox**
SSRI; treatment and diagnosis of behavior disorders
25-, 50-, and 100-mg tablets
Dog: 0.5-2.0 mg/kg, PO, bid
Cat: 0.25-0.5 mg/kg PO once daily

### § Fomepizole (4-methylpyrazole; 4-MP)
**Antizol-Vet**
Antidote; ethylene glycol poisoning
1.5-mL single-use vial; reconstitute in 30 mL of 0.9% NaCl for a 5% solution (50 mg/mL)
20 mg/kg IV initially within 8 hr of ingestion; then 15 mg/kg IV at 12- and 24-hr intervals; then 5 mg/kg IV at 36 hr
Note: Cats require 7× the dose used in dogs; agent is effective only if administered within 3 hr after ingestion of ethylene glycol.

### Furazolidone
**Furoxone**
Antibacterial and antiprotozoal; generally a second-choice drug
100-mg tablet
4 mg/kg PO q12h for 7-10 days

### § Furosemide
**Lasix, generic**
Diuretic; multiple uses; commonly used to treat congestive heart failure and pulmonary edema
12.5-, 20-, and 50-mg tablets; 10 mg/mL oral solution; 50 mg/mL injection
Dog: 2-6 mg/kg IV, IM, SQ, PO q8-12h (or as needed); 0.6-1.0 mg/kg/h IV
Cat: 1-4 mg/kg IV, IM, SQ, PO q8-24h

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<tr>
<td>Gemfibrozil</td>
<td>Lopid</td>
<td>Antilipemic; treatment of hypertriglyceridemia in patients that do not respond to dietary fat restriction</td>
<td>300-mg capsule; 600-mg tablet</td>
<td>7.5 mg/kg PO q12h</td>
</tr>
</tbody>
</table>
| Gentamicin          | Gentocin          | Antibacterial (aminoglycoside) | 50 and 100 mg/mL solution for injection | Dog: 2-4 mg/kg q6-8h or 6-10 mg/kg IV, 1M, SQ q24h  
Cat: 3 mg/kg q8h or 9 mg/kg IV, 1M, SQ q24h  
**Warning:** Do not administer to patients that are dehydrated or acidic; can cause acute renal failure. |
| Glipizide           | Glucotrol         | Oral hypoglycemic; variably effective control of type 2 diabetes in cats | 5- and 10-mg tablets | 2.5-7.5 mg/cat PO q12h; usual dose is 2.5 mg/cat initially; then increase to 5 mg/cat q12h |
| § Glucagon          | Glucagon Emergency Kit (Lilly), GlucaGen (products are rDNA origin) | Parenteral hyperglycemic; treatment of acute hypoglycemia and/or insulin overdose | 1 mg (1.0 unit) per vial | Dilute 1 mg glucagon in 1 L of 0.9% sterile saline. Resulting concentration: 1000 mg/mL  
Administer 50 mg/kg (or 0.05 mL/kg of diluted solution) by intravenous bolus, then 10 to 15 mg/kg/min IV by CRI as needed to correct hypoglycemia. |
| Glucosamine + chondroitin sulfate | Cosequin and other brands | Neutraceutical; adjunctive treatment of nonseptic arthritis; may be useful in treating cats with lower urinary tract disease (FLUTD) | Regular-strength (RS) and double-strength (DS) capsules | Dog: 1 or 2 RS capsules per day (2-4 capsules of DS for large dogs)  
Cat: 1 RS capsule daily |
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<tr>
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<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glyburide</td>
<td>Diabeta, Micronase, Glynase</td>
<td>Oral hypoglycemic; variably effective control of type 2 diabetes in cats</td>
<td>1.25-, 2.5-, and 5-mg tablets</td>
<td>0.625 mg per cat once daily (represents one half of 1.25-mg tablet)</td>
</tr>
<tr>
<td>Glycerin (OTC)</td>
<td>Generic</td>
<td>Oral osmotic; reduces intraocular (and CSF) pressure</td>
<td>Oral solution</td>
<td>1-2 mL/kg PO q8h</td>
</tr>
<tr>
<td>Glycopyrrolate</td>
<td>Robinul-V</td>
<td>Antimuscarinic; multiple uses—preanesthetic medication, antidote</td>
<td>0.2 mg/mL injection</td>
<td>0.005-0.011 mg/kg IV, IM, SQ</td>
</tr>
<tr>
<td>Gold sodium thiomalate</td>
<td>Myochrysine</td>
<td>Gold salt; treatment of immune-mediated skin disorders</td>
<td>Injection</td>
<td>1-5 mg IM first wk, then 2-10 mg IM second wk, then 1 mg/kg IM once/wk maintenance</td>
</tr>
<tr>
<td>Gold therapy</td>
<td>See Aurothioglucose</td>
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<td></td>
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<tr>
<td>GoLYTELY</td>
<td>See Polyethylene glycol electrolyte solution</td>
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</tr>
<tr>
<td>Gonadorelin</td>
<td>Factrel</td>
<td>Hormone; diagnosis and treatment of various reproductive disorders</td>
<td>50 mcg/mL injection</td>
<td>Therapeutic doses: Dog: 50-100 mcg/dog SQ, IV, IM q24-48h × 2 doses Cat: 25 mcg/cat IM once</td>
</tr>
<tr>
<td>Gonadotropin, human chorionic (hCG)</td>
<td>Profasi, Pregnyl, APL, and generic</td>
<td>Hormone; induces luteinization</td>
<td>5000-, 10,000-, and 20,000-unit injection</td>
<td>Dog: 22 units/kg IM q24-48h or 44 units IM once Cat: 250 units/cat IM once Warning: Do not use in pregnant animals.</td>
</tr>
<tr>
<td>Gonadotropin-releasing hormone</td>
<td>See Gonadorelin</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Granisetron</td>
<td>Kytril</td>
<td>Antiemetic; prevents emesis associated with chemotherapy</td>
<td>1 mg/mL injection; 1-mg tablet</td>
<td>0.01 mg/kg (10 mcg/kg) IV</td>
</tr>
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<tbody>
<tr>
<td>Griseofulvin (microsize)</td>
<td>Fulvicin U/F</td>
<td>Antifungal (fungistatic antibiotic); treatment of dermatophytes (especially <em>Microsporum canis</em>)</td>
<td>125-, 250-, and 500-mg tablets; 25 mg/mL oral suspension; 125 mg/mL oral syrup</td>
<td>50 mg/kg PO q24h (up to a maximum dose of 110-132 mg/kg/day in divided treatments)</td>
</tr>
<tr>
<td>Growth hormone (hGH)</td>
<td>Humatrope, Nutropin, Protropin, Somatropin, Somatrem</td>
<td>Hormone replacement hormone in patients with confirmed deficiency</td>
<td>5- and 10-mg vials</td>
<td>0.1 unit/kg SQ, IM three times per week for 4-6 wk <em>Warning</em>: Is diabetogenic</td>
</tr>
<tr>
<td>Halothane</td>
<td>Fluothane</td>
<td>Inhalation anesthetic</td>
<td>250-mL liquid</td>
<td>Induction: 3% Maintenance: 0.5%-1.5%</td>
</tr>
<tr>
<td>$ Heparin sodium</td>
<td>Liquaemin</td>
<td>Anticoagulant; treatment of DIC, treatment and prevention of thromboembolic disease</td>
<td>1000 and 10,000 units/mL injection</td>
<td>100-200 units/kg IV loading dose, then 100-300 units/kg SQ q6-8h Low-dose prophylaxis (dog and cat): 70 units/kg SQ q8-12h</td>
</tr>
<tr>
<td>$ Hydralazine</td>
<td>Apresoline</td>
<td>Vasodilator; hypertension and adjunctive treatment of heart failure</td>
<td>10-mg tablet; 20 mg/mL injection</td>
<td>Dog: 0.5 mg/kg (initial dose); titrate to 0.5-2 mg/kg PO q12h Cat: 2.5 mg/cat PO q12-24h</td>
</tr>
<tr>
<td>Hydrochlorothiazide</td>
<td>HydroDIURIL, generic</td>
<td>Diuretic; hypertension, congestive heart failure, and nephrogenic (ADH-resistant) DI</td>
<td>10 and 100 mg/mL oral solution; 25-, 50-, and 100-mg tablets</td>
<td>2-4 mg/kg PO q12h</td>
</tr>
<tr>
<td>$ Hydrocodone bitartrate</td>
<td>Hycodan (contains atropine)</td>
<td>Analgesic (opiate); pain management</td>
<td>5-mg tablet</td>
<td>Dog: 0.22 mg/kg PO q4-8h Cat: no dose available</td>
</tr>
<tr>
<td>Drug</td>
<td>Manufacturer/Trade Name</td>
<td>Type/Effects</td>
<td>Dosage/Use</td>
<td></td>
</tr>
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<td></td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>Cortef and generic</td>
<td>Glucocorticoid; antiinflammatory and replacement therapy in adrenal insufficient conditions</td>
<td>5-, 10-, and 20-mg tablets</td>
<td>Replacement therapy: 0.2-0.5 mg/kg PO q24h Antiinflammatory: 1.5-5 mg/kg PO q12h</td>
</tr>
<tr>
<td>§ Hydrocortisone sodium succinate</td>
<td>Solu-Cortef and shock treatment</td>
<td>Glucocorticoid; antiinflammatory injection</td>
<td>Various size vials for antiinflammatory: 5 mg/kg IV q12h</td>
<td>Shock: 50-150 mg/kg IV</td>
</tr>
<tr>
<td>§ Hydromorphone</td>
<td>Dilaudid</td>
<td>Analgesic (opiate); pain management and restraint</td>
<td>Tablets, oral solution, and injectable forms available</td>
<td>Dog: 0.22 mg/kg, IM, SQ, q4-6h as needed for pain</td>
</tr>
<tr>
<td>§ Hetastarch, Hydroxyethyl starch (HES)</td>
<td>Hespan, Hextend</td>
<td>Volume expander; used when colloidal therapy is indicated</td>
<td>Injection</td>
<td>10-20 mL/kg IV to effect, 20-30 mL/kg/day</td>
</tr>
<tr>
<td>Hydroxyurea</td>
<td>Hydrea</td>
<td>Antineoplastic; polycythemia vera, mastocytoma, leukemias</td>
<td>500-mg capsule</td>
<td>Dog: 50 mg/kg PO once daily, 3 days/wk Cat: 25 mg/kg PO once daily, 3 days/wk</td>
</tr>
<tr>
<td>Hydroxyzine</td>
<td>Atarax</td>
<td>Antihistamine; antipruritic and sedative effects, especially in atopic patients</td>
<td>10-, 25-, and 50-mg tablets; 2 mg/mL oral solution</td>
<td>Dog: 1-2 mg/kg q6-8h IM, PO Cat: 5-10 mg/cat PO q8-12h</td>
</tr>
<tr>
<td>Ifosfamide</td>
<td>Ifex</td>
<td>Antineoplastic; lymphomas and other sarcomas</td>
<td>1 g powder for intravenous infusion in single-dose vials</td>
<td>Dogs and cats: Dose ranges from 300 to 500 mg/m² IV Caution: Consult treatment protocol before administering.</td>
</tr>
<tr>
<td>Imidacloprid</td>
<td>Advantage</td>
<td>Topical flea treatment for dogs and cats</td>
<td>Topical solution</td>
<td>Apply topically once monthly as directed by the manufacturer for the treatment of fleas.</td>
</tr>
<tr>
<td>Imidacloprid + permethrin</td>
<td>K9 Advantix</td>
<td>Topical flea treatment and tick repellent for dogs only</td>
<td>Topical solution</td>
<td>Apply topically once monthly as directed by the manufacturer for the treatment of fleas. Cat: Do not use; contains permethrin.</td>
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| Imidocarb dipropionate        | Imizol            | Antiprotozoal; treatment of babesiosis, ehrlichiosis | Parenteral solution for injection IM or SQ; 120 mg/mL in 10-mL multidose vial | Dog: 5 mg/kg IM or SQ once; repeat in 2 wk  
For babesiosis: 6.6 mg/kg IM or SQ once; repeat in 2 wk  
Cat: (cytauxzoonosis) 5 mg/kg IM q2wk as needed |
| Imipenem + cilastatin        | Primaxin          | Antibacterial                                       | 250- and 500-mg vials for injection       | 5-10 mg/kg IV, IM q6-8h; has been administered to dogs at 10 mg/kg q8h SQ        |
| Imipramine                   | Tofranil          | Tricyclic antidepressant; treatment of behavior disorders | 10-, 25-, and 50-mg tablets               | 2-4 mg/kg PO q12-24h                                                              |
| Indomethacin                  | Indocin           |                                                     | Safe dose has not been established.      |                                                                                   |
| Interferon (interferon alfa-2a, HuIFN-alpha) | Roferon           | Cytokine; immunomodulation in cats with FeLV and/or FIV infection (clinical value of treatment is not established) | 3 million–unit pre-filled syringe         | Cat: 30 units/cat/day PO or high dose: 10,000-1,000,000 units/cat SQ q24h  
IM or SQ once daily for 7 days and repeated every other wk |
| Ipecac syrup (OTC)            | Ipecac            | Oral emetic                                         | No longer recommended: can cause fatal arrhythmias |                                                                                   |
| Ipodate                       | Bilivist, Oragrafin | Organic iodine; treatment of hyperthyroidism (especially in cats) | 500-mg capsule (should be formulated for cats as 50-mg ampule) | Dog: 15 mg/kg PO q12h  
Cat: 100-200 mg (total dose)/cat once daily; dose may be reduced if the 2-wk response is judged satisfactory |
| Iron                          | See Ferrous sulfate | Inhalation anesthetic                               | 100-mL bottle                             | Induction: 5%  
Maintenance: 1.5%-2.5%                                                           |
| § Isoproterenol | Isuprel | β-Agonist; uncommonly used to treat acute bronchoconstriction and certain cardiac arrhythmias | 0.2 mg/mL ampule for injection | 10 mcg/kg IM, SQ q6h; or dilute 1 mg in 500 mL of 5% dextrose or Ringer’s solution and infuse IV 0.5-1 mL/min (1-2 mcg/min) or to effect |
| § Isosorbide dinitrate | Isordil, Isorbid, Sorbitrate | Vasodilator; congestive heart failure | 2.5-, 5-, 10-, 20-, 30-, and 40-mg tablets; 40-mg capsules | 2.5-5 mg/animal PO q12h (or 0.22-1.1 mg/kg PO q12h) |
| § Isosorbide mononitrate | Monoket | Vasodilator; congestive heart failure | 10- and 20-mg tablets | 5 mg/dog PO, two doses/day 7 hr apart |
| Isotretinoin | Accutane | Synthetic retinoid; treatment of dermatologic diseases associated with epithelial cell proliferation (e.g., ichthyosis, cutaneous lymphoma) | 10-, 20-, and 40-mg capsules | 1-3 mg/kg/day (up to maximum recommended dose of 3-4 mg/kg/day PO) |
| Itraconazole | Sporanox | Antifungal; treatment of systemic mycoses | 100-mg capsule | Dog: 2.5 mg/kg PO q12h or 5 mg/kg PO q24h Cat: 1.5-3.0 mg/kg PO up to 10 mg/kg PO q24h |
| Ivermectin | Heartgard, Ivomec, Eqvalan liquid | Antiparasiticide; multiple applications | 1% (10 mg/mL) injectable solution; 10 mg/mL oral solution; 18.7 mg/mL oral paste; 68-, 136-, and 272-mcg tablets | Heartworm preventative: Dog: 6 mcg/kg (range: 3 to 12 mcg/kg) PO q30days Cat: 24 mcg/kg PO q30days Microfilaricide: 50 mcg/kg PO 2 wk after adulticide therapy Ectoparasite therapy (dog and cat): 200-300 mcg/kg IM, SQ, PO Endoparasites (dog and cat): 200-400 mcg/kg SQ, PO weekly Demodex therapy: start at 100 mcg/kg/day PO q24h, increase by 100 mcg/kg increments weekly to biweekly until target of 600 mcg/kg is reached |

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<tbody>
<tr>
<td>Kanamycin</td>
<td>Kantrim</td>
<td>Antibacterial</td>
<td>200 and 500mg/mL injection</td>
<td>10 mg/kg IV, IM, SQ q6-8h</td>
</tr>
<tr>
<td>§</td>
<td>Kaopectate (kaolin + pectin) (OTC)</td>
<td>GI adsorbent; management of acute, simple diarrheal disorders, especially result of dietary indiscretion</td>
<td>12-oz oral suspension</td>
<td>1-2 mL/kg PO q2-6h</td>
</tr>
<tr>
<td>§ Ketamine</td>
<td>Ketalar, Ketavet, Vetalar</td>
<td>Dissociative anesthetic</td>
<td>100 mg/mL injection solution</td>
<td>Dog: 5.5-22 mg/kg IV, IM (recommend adjunctive sedative or tranquilizer treatment) Cat: 2-25 mg/kg IV, IM (recommend adjunctive sedative or tranquilizer treatment)</td>
</tr>
<tr>
<td>Ketoconazole</td>
<td>Nizoral</td>
<td>Antifungal; systemic mycoses, <em>Malassezia canis</em> infection; limited application in the treatment of canine hyperadrenocorticism</td>
<td>200-mg tablet; 100 mg/mL oral suspension (available only in Canada)</td>
<td>Dog: 10-15 mg/kg PO q8-12h <em>Malassezia canis</em>: 10 mg/kg PO q24h or 5 mg/kg PO q12h Cat: 5-10 mg/kg PO q8-12h Hyperadrenocorticism: Dog: 15 mg/kg PO q12h</td>
</tr>
<tr>
<td>§ Ketoprofen</td>
<td>Orudis KT (OTC), Ketofen</td>
<td>NSAID; pain management</td>
<td>12.5-mg tablet (OTC); 100 mg/mL injection</td>
<td>Dog and cat: 1 mg/kg PO q24h for up to 5 days or 2.0 mg/kg IV, IM, SQ for one dose</td>
</tr>
<tr>
<td>Ketorolac tromethamine</td>
<td>Toradol</td>
<td>NSAID; pain management</td>
<td>10-mg tablet; 15 and 30 mg/mL injection in 10% alcohol</td>
<td>Dog: 0.5 mg/kg PO, IM, IV q12h for not more than two doses Cat: 0.25 mg/kg IM q8-12h for 1-2 doses</td>
</tr>
<tr>
<td>§ Lactated Ringer’s solution</td>
<td>Generic</td>
<td>Fluid replacement</td>
<td>250-, 500-, and 1000-mL bags</td>
<td>Maintenance: 40-50 mL/kg/day IV Shock therapy: Dog: 90 mL/kg IV Cat: 60-70 mL/kg IV</td>
</tr>
<tr>
<td>Drug</td>
<td>Brand Name, Form</td>
<td>Description</td>
<td>Dosage</td>
<td>Comments</td>
</tr>
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</tr>
</tbody>
</table>
| Lactulose                     | Chronulac, generic | Disaccharide laxative; limits bowel absorption of protein and facilitates lowering of blood ammonia levels in patients with hepatic encephalopathy | 10 g/15 mL                                     | Constipation: 1 mL/4.5 kg PO q8h (to effect)  
Hepatic encephalopathy:  
Dog: 0.5 mL/kg PO q8h  
Cat: 2.5-5 mL/kg PO q8h |
| Leucovorin (folinic acid)     | Wellcovorin, generic | Antidote; folic acid antagonism; application in dogs and cats is not established | 5-, 10-, 15-, and 25-mg tablets; 3 and 5 mg/mL injection | With methotrexate administration: 3 mg/m² IV, IM, PO  
Antidote for pyrimethamine toxicosis: 1 mg/kg PO q24h |
| Levamisole                    | Levasole, Tramisol injectable | Antiparasitic; treatment of nematode infection; also proposed to be a nonspecific immunostimulant | 0.184-g bolus; 11.7-g/13-g packet; 50-mg tablet | Dog: Hookworms: 5-8 mg/kg PO once (up to 10 mg/kg PO for 2 days)  
Microfilaricide: 10 mg/kg PO q24h for 6-10 days  
Immunostimulant: 0.5-2 mg/kg PO 3 times/wk  
Cat: 4.4 mg/kg once PO (for lungworms: 20-40 mg/kg PO q48h for five treatments) |
| Levetiracetam                 | Keppra           | Oral anticonvulsant                              | 250-, 500-, and 750-mg tablets                 | Dog: initially 20 mg/kg PO q8h, then gradually increase as needed to control seizures  
Cat: 30 mg/kg PO q12h (look for an underlying cause of seizure activity) |
| L-dopa, Levodopa              | Larodopa, (multiple products available) | Dopamine agonist; hepatic encephalopathy          | 100-, 250-, and 500-mg tablets or capsules   | Hepatic encephalopathy: 6.8 mg/kg initially, then 1.4 mg/kg q6h |
| Levothyroxine sodium (T₄)     | Soloxine, Thyro-Tabs, Synthroid | Hormone; hypothyroidism                          | 0.1- to 0.8-mg tablets (in 0.1-mg increments) | Dog: 18-22 mcg/kg PO q12h (adjust dose via monitoring T₄ levels)  
Cat: 10-20 mcg/kg/day, PO (adjust dose via monitoring T₄ levels) |

Listings preceded by § are for rapid reference and denote drug or dose used in the emergency or critical care setting.
<table>
<thead>
<tr>
<th>Drug</th>
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<th>Formulation</th>
<th>Recommended Dosage</th>
</tr>
</thead>
</table>
| Lidocaine (without epinephrine) | Xylocaine, generic | Anesthetic and antiarrhythmic; ventricular arrhythmias; also local and regional anesthetic; has been used systemically for pain | 5, 10, 15, and 20 mg/mL injection | Dog (antiarrhythmic): 2-4 mg/kg IV (to a maximum dose of 8 mg/kg over 10-min period); 25-75 mcg/kg/min intravenous infusion  
Cat (antiarrhythmic): 0.25-0.75 mg/kg IV slowly  
For epidural (dog and cat): 4.4 mg of 2% solution per kilogram |
| Lincomycin | Lincocin | Antibacterial | 100-, 200-, and 500-mg tablets | 15-25 mg/kg PO q12h  
For pyoderma: Doses as low as 10 mg/kg q12h have been used |
| Liothyronine (T\(_3\)) | Cytomel | Hormone (active form of T\(_3\)); replacement therapy in patients with hypothyroidism that fail to respond to T\(_4\) | 60-mcg tablet | 4.4 mcg/kg PO q8h  
For T\(_3\) suppression test (cats): Collect presample for T\(_4\) and T\(_3\); administer 25 mcg q8h for 7 doses; then collect postsamples for T\(_3\) and T\(_4\) after last dose |
| Lisinopril | Prinivil, Zestril | ACE inhibitor; vasodilator for treatment of hypertension or heart failure | 2.5-, 5-, 10-, 20-, and 40-mg tablets | Dog: 0.5 mg/kg PO q24h  
Cat: 0.25-0.5 mg/kg PO q24h |
| Lithium carbonate | Lithotabs | Nonspecific immunostimulant; adjunctive treatment to increase neutrophil counts in patients with chemotherapy-induced neutropenia | 150-, 300-, and 600-mg capsules; 300-mg tablet; 300 mg/5 mL syrup | Dog: 10 mg/kg PO q12h  
Cat: Not recommended |
| Loperamide | Imodium, generic | Analgesic (opiate); nonspecific management of diarrhea | 2-mg tablet; 0.2 mg/mL oral liquid | Dog: 0.1 mg/kg PO q8-12h  
Cat: 0.08-0.16 mg/kg PO q12h |
|-----------|----------------|------------------------------------------------------|-----------------------------------|-----------------------------|
| Lufenuron | Program | Antiparasitic; flea control | 45-, 90-, 135-, 204.9-, and 409.8-mg tablets; 135- and 270-mg suspension per unit pack | Dog: 10 mg/kg PO q30days  
Cat: 30 mg/kg PO q30days; 10 mg/kg SQ q6mo |
| Lufenuron + milbemycin oxime | Sentinel tablets, Flavor Tabs | Antiparasitic; flea control plus heartworm preventative effective against certain intestinal parasites | Milbemycin-lufenuron ratio is as follows: 2.3/46 mg, Sentinel tablets; 5.75/115, 11.5/230, and 23/460 mg, Flavor Tabs | Dog: Administer 1 tablet q30days as recommended by manufacturer (each tablet formulated for size of dog)  
Cat: *Do not use* |
| Luteinizing hormone | See Gonadorelin |
| L-lysine (OTC) | Multiple preparations | Amino acid; prevention of feline herpesvirus-1 recrudescence | 250-500-mg capsules | Cat (empiric dose): Mix 250 to 500 mg with food once daily. Kittens receive approximately 250 mg daily, with food.  
*Note:* Efficacy studies have not been performed; agent has no known effect on feline calicivirus carrier cats. |
| $ Magnesium chloride | Generic | Elemental salt; ventricular dysrhythmias, refractory hypokalemia, and ventricular fibrillation | 200 mg/mL in 50-mL vials for injection | 0.15-0.3 mEq/kg IV over 2-10 min; or 0.75 mEq/kg/day IV by CRI |
| Magnesium citrate | Citroma, Citro-Nesia (Citro-Mag in Canada) | Laxative | Oral solution | 2-4 mL/kg PO |
| Magnesium hydroxide (OTC) | Multiple products available | Laxative | Oral liquid | Antacid: 5-10 mL/kg PO q4-6h  
Cathartic:  
Dog: 15-50 mL/kg PO  
Cat: 2-6 mL/cat PO q24h |

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*Continued*
<table>
<thead>
<tr>
<th>Drug</th>
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<th>Formulation</th>
<th>Recommended Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Magnesium sulfate (OTC)</td>
<td>Multiple products available</td>
<td>Laxative; also used for oral magnesium supplementation</td>
<td>Crystals; many generic preparations</td>
<td>Dog: 8-25 g/dog PO q24h</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cat: 2-5 g/cat PO q24h</td>
</tr>
<tr>
<td>§ Mannitol</td>
<td>Osmirol</td>
<td>Diuretic (osmotic); management of anuric and/or oliguric renal failure; applications in management of glaucoma (repeat in 6 hr if necessary) and cerebral edema</td>
<td>5%-25% solution for injection</td>
<td>Diuretic: 1 g/kg 5%-25% solution IV to maintain urine flow</td>
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<tr>
<td></td>
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<td></td>
<td>Glaucoma or CNS edema: 0.25-2 g/kg</td>
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<tr>
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<td></td>
<td>15%-25% solution IV over 30-60 min</td>
</tr>
<tr>
<td>Marbofloxacin</td>
<td>Zeniquin</td>
<td>Antibacterial</td>
<td>25-, 50-, 100-, 200-mg tablets</td>
<td>Dog: 2.75-5.55 mg/kg PO q24h</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cat: Dose not established</td>
</tr>
<tr>
<td>Maropitant</td>
<td>Cerenia</td>
<td>Antiemetic (parenteral and oral); oral administration to prevent motion sickness in dogs</td>
<td>10 mg/mL solution for injection; 16-, 24-, 60-, and 160-mg tablets</td>
<td>Dog: 1 mg/kg SQ once daily for up to 5 days; 2 mg/kg PO once daily for up to 5 days</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>For motion sickness: 8 mg/kg PO once daily for up to 2 days</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cat: Dose not established</td>
</tr>
<tr>
<td>MCT (medium chain triglyceride) oil</td>
<td>Multiple OTC products available</td>
<td>Medium-chain triglyceride; lipid supplement used in patients with GI absorptive disorders</td>
<td>Oral liquid</td>
<td>1-2 mL/kg/day in food</td>
</tr>
<tr>
<td>Mebendazole</td>
<td>Telmintic</td>
<td>Antiparasitic; multiple applications for treatment of endoparasites</td>
<td>40 mg/powder</td>
<td>22 mg/kg (with food) q24h for 3 days</td>
</tr>
<tr>
<td>Drug</td>
<td>Common Trade Name(s)</td>
<td>Description</td>
<td>Dosage Form</td>
<td>Veterinary Use</td>
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</tr>
</tbody>
</table>
| $ Meclizine | Antivert, generic | Antihistamine, antiemetic, especially when nausea is associated with vertigo | 12.5-, 25-, and 50-mg tablets | Dog: 25 mg PO q24h (for motion sickness, administer 1 hr before traveling)  
Cat: 12.5 mg PO q24h |
| Meclofenamate | Arquel, Meclomen | NSAID; pain management | 50- and 100-mg capsules | Dog: 1 mg/kg/day PO for up to 5 days |
| $ Medetomidine | Domitor | Analgesic (parenteral); adjunct for anesthesia, restraint | 1.0 mg/mL injection | 750 mcg/m² IV or 1000 mcg/m² IM |
| Medium-chain triglycerides | See MCT oil |
| Medroxyprogesterone acetate | Depo-Provera (injection), Provera (tablets) | Hormone; management of certain dermatologic and behavior disorders, including urine spraying in cats; benign prostatic hyperplasia | 150 and 400 mg/mL suspension injection; 2.5-, 5-, and 10-mg tablets | 1.1-2.2 mg/kg IM q7days  
Behavior disorders: 10-20 mg/kg SQ or IM q3 months (dogs and cats)  
Prostatic hyperplasia: 3-5 mg/kg SQ, IM |
| Megestrol acetate | Ovaban, Megace | Hormone; management of certain dermatologic and behavior disorders, including urine spraying in cats | 5-mg tablet | Dog: Proestrus: 2 mg/kg PO q24h for 8 days  
Anestrus: 0.5 mg/kg PO q24h for 30 days  
Behavior disorders: 2-4 mg/kg q24h for 8 days (reduce dose for maintenance)  
Cat (Note: Any use in cats is extra-label):  
Dermatologic therapy or urine spraying: 2.5-5 mg/cat PO q24h for 1 wk, then reduce to 5 mg once or twice per week  
Estrus suppression: 5 mg/cat/day for 3 days, then 2.5-5 mg once/wk for 10 wk |

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Continued
<table>
<thead>
<tr>
<th>Drug</th>
<th>Proprietary Names</th>
<th>Action and Use</th>
<th>Formulation</th>
<th>Recommended Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Melarsomine</td>
<td>Immiticide</td>
<td>Antiparasitic (arsenical); treatment of canine heartworm disease</td>
<td>25 mg/mL injection; after reconstitution, retains potency for 24 hr</td>
<td>Administer via deep intramuscular injection</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td>Class 1-2 dogs: 2.5 mg/kg/day for 2 consecutive days</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Class 3 dogs: 2.5 mg/kg once, then in 1 mo two additional doses 24 hr apart</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cat: <em>Do not use</em></td>
</tr>
<tr>
<td>Meloxicam</td>
<td>Metacam</td>
<td>NSAID; pain management</td>
<td>1.5 mg/mL oral solution</td>
<td>0.2 mg/kg PO initial loading dose; then 0.1 mg/kg PO q12h</td>
</tr>
<tr>
<td>Melphalan</td>
<td>Alkeran</td>
<td>Antineoplastic; used in treatment protocols for multiple tumor types</td>
<td>2-mg tablet</td>
<td>1.5 mg/m² or 0.1-0.2 mg/kg PO q24h for 7-10 days; repeat every 3 wk</td>
</tr>
<tr>
<td>Meperidine</td>
<td>Demerol</td>
<td>Analgesic (opiate); pain management</td>
<td>50- and 100-mg tablets; 10 mg/ml syrup; 25, 50, 75, and 100 mg/mL injection</td>
<td>Dog: 5-10 mg/kg IV, IM as often as q2-3h (or as needed)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cat: 3-5 mg/kg IV, IM q2-4h (or as needed)</td>
</tr>
<tr>
<td>Mepivacaine</td>
<td>Carbocaine-V</td>
<td>Local anesthetic</td>
<td>2% (20 mg/mL) injection</td>
<td>Variable dose for local infiltration</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>For epidural, 0.5 mg of 2% solution q30sec until reflexes are absent</td>
</tr>
<tr>
<td>6-Mercaptopurine</td>
<td>Purinethol</td>
<td>Antineoplastic; used in treatment protocols for multiple tumor types</td>
<td>50-mg tablet</td>
<td>50 mg/m² PO q24h</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*Caution: Consult treatment protocol before administering.</td>
</tr>
<tr>
<td>Meropenem</td>
<td>Merrem</td>
<td>Antibacterial; especially in treating resistant infections caused by <em>Pseudomonas</em>, <em>Escherichia coli</em>, and <em>Klebsiella</em></td>
<td>500 mg in 20-mL vial, or 1 g in 30-mL vial for injection</td>
<td>20 mg/kg IV q8h</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>For meningitis: 40 mg/kg IV q8h</td>
</tr>
<tr>
<td>Drug Name</td>
<td>Brand Name(s)</td>
<td>Category/Action</td>
<td>Dose/Usage</td>
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<td>--------------------------------------------</td>
<td>-----------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Metaproterenol</td>
<td>Alupent, Metaprel</td>
<td>β-Agonist; bronchodilator therapy</td>
<td>10- and 20-mg tablets; 5 mg/mL syrup; inhalers</td>
<td></td>
</tr>
<tr>
<td>Metformin</td>
<td>Glucophage</td>
<td>Oral hypoglycemic; management of type 2 diabetes in cats</td>
<td>500- and 800-mg tablets</td>
<td></td>
</tr>
<tr>
<td>Methazolamide</td>
<td>Neptazane</td>
<td>Carbonic anhydrase inhibitor; treatment of open-angle glaucoma</td>
<td>25- and 50-mg tablets</td>
<td></td>
</tr>
<tr>
<td>Methenamine hippurate</td>
<td>Hiprex, Urex</td>
<td>Urinary antiseptic (of questionable value)</td>
<td>1-g tablet</td>
<td></td>
</tr>
<tr>
<td>Methenamine mandelate</td>
<td>Mandelamine, generic</td>
<td>Urinary antiseptic (of questionable value)</td>
<td>1-g tablet; granules for oral solution; 50 and 100 mg/mL oral suspension</td>
<td></td>
</tr>
<tr>
<td>Methimazole</td>
<td>Tapazole</td>
<td>Antithyroidal; management of feline hyperthyroidism</td>
<td>5- and 10-mg tablets</td>
<td></td>
</tr>
<tr>
<td>Methionine (dl)</td>
<td>Uroeze; dl-methionine powder</td>
<td>Urinary acidifier</td>
<td>500-mg tablets and powders added to animal's food; 75 mg/5 mL pediatric oral solution; 200-mg capsule</td>
<td></td>
</tr>
<tr>
<td>Methionine (3-adenosyl)</td>
<td>See SAMe</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$ Methocarbamol</td>
<td>Robaxin-V</td>
<td>Muscle relaxant; adjunctive therapy for trauma, acute inflammation of skeletal muscle, and/or tremorgenic toxins</td>
<td>500- and 750-mg tablets; 100 mg/mL injection</td>
<td></td>
</tr>
<tr>
<td>Methohexital</td>
<td>Brevital</td>
<td>Ultra-short-acting barbiturate; anesthetic induction</td>
<td>0.5-, 2.5-, and 5-g vials for injection</td>
<td></td>
</tr>
</tbody>
</table>

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<tr>
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</tr>
</thead>
</table>
| Methotrexate          | MTX, Mexate, Folex, Rheumatrex, generic | Antineoplastic; used in treatment protocols for multiple tumor types, especially lymphomas | 2.5-mg tablet; 2.5 or 25 mg/mL injection | 2.5-5 mg/m² PO q48h (dose depends on specific protocol) or:  
Dog: 0.3-0.5 mg/kg IV once/wk  
Cat: 0.8 mg/kg IV q2-3wk |
| § Methoxamine         | Vasoxyl                    | Vasopressor; used in critical care setting to increase blood pressure         | 20 mg/mL injection         | 200-250 mcg/kg IM or 40-80 mcg/kg IV                                               |
| § Methylene blue 0.1% | Generic; also called new methylene blue | Antidote; emergency treatment of methemoglobinemia                         | 1% solution (10 mg/mL)    | 1.5 mg/kg IV slowly; use once                                                       |
| Methylprednisolone    | Medrol                     | Glucocorticoid; antiinflammatory and immunosuppressive                       | 1-, 2-, 4-, 8-, 18-, and 32-mg tablets | In cats, use with caution: 0.22-0.44 mg/kg PO q12-24h  
Note: Methylprednisolone is 1.25 times more potent than prednisolone. |
| Methylprednisolone acetate | Depo-Medrol               | Repository glucocorticoid; antiinflammatory (extended duration of activity) | 20 and 40 mg/mL suspension for injection | Dog: 1 mg/kg (or 20-40 mg/dog) IM q1-3wk  
Cat: 10-20 mg/cat IM q1-3wk  
Note: Actual dose may vary, depending on use and effect. |
| § Methylprednisolone sodium succinate | Solu-Medrol            | Glucocorticoid; adjunctive treatment for patients in shock or with spinal cord trauma or swelling | 1- and 2-g and 125- and 500-mg vials for injection | For emergency use: 30 mg/kg IV; repeat at 15 mg/kg IV in 2-6 hr  
For replacement therapy or antiinflammatory therapy; see also Prednisolone |

§ 4-Methylpyrazole (4-MP) See *Fomepizole*
<table>
<thead>
<tr>
<th>Drug Name</th>
<th>Trade Name(s)</th>
<th>Description</th>
<th>Dosage Formulations</th>
<th>Dosages and Administration</th>
</tr>
</thead>
</table>
| Methyltestosterone | Android, generic   | Hormone; replacement therapy; also an anabolic agent used to induce erythropoiesis      | 10- and 25-mg tablets                                                                 | Dog: 5-25 mg/dog PO q24-48h  
Cat: 2.5-5 mg/cat PO q24-48h |
| § Metoclopramide | Reglan, Maxolon, others | Antiemetic, especially in patients with vomiting associated with gastroparesis | 5- and 10-mg tablets; 1 mg/mL oral solution; 5 mg/mL injection | 0.2-0.5 mg/kg IV, IM, PO q6-8h; or 1-2 mg/kg/day IV by CRI (approximately 0.01-0.02 mg/kg/hr) |
| Metoprolol     | Lopressor             | β-Blocker; management of tachycardia                                            | 50- and 100-mg tablets; 1 mg/mL injection                                           | Dog: 5-50 mg/dog (0.5-1.0 mg/kg) PO q8h  
Cat: 2-15 mg/cat PO q8h |
| § Metronidazole | Flagyl, generic      | Antiparasitic and antibacterial; effective against anaerobic bacteria; somewhat effective against Giardia (fenbendazole is preferred) | 250- and 500-mg tablets; 50 mg/mL suspension; 5 mg/mL injection | Anaerobic infection:  
Dog: 15 mg/kg PO q12h or 12 mg/kg q8h  
Cat: 10-25 mg/kg PO q12h  
Giardia: Dog: 12-15 mg/kg PO q12h for 8 days  
Cat: 25 mg/kg q12h for 8 days |
| § Mexiletine   | Mexitil               | Antiarrhythmic; ventricular arrhythmias                                         | 150-, 200-, and 250-mg capsules                                                    | Dog: 5-8 mg/kg PO q8-12h (Use cautiously.)  
Cat: No dose established |
| Mibolerone     | Cheque Drops          | Hormone (androgenic); suppression of estrus and treatment of false pregnancy (pseudocyesis) | 55 mcg/mL oral solution                                                            | Dog: 0.45-11.3 kg, 30 mcg; 11.8-22.7 kg, 60 mcg; 23-43.3 kg, 120 mcg; >45.8 kg, 180 mcg; or approximately 2.6-5 mcg/kg/day PO  
Cat: Do not use  
Warning: Multiple adverse effects are possible when used in prepubertal females. |
| § Midazolam    | Versed                | Benzodiazepine; preanesthetic medication                                        | 5 mg/mL injection                                                                  | 0.1-0.25 mg/kg IV, IM (or 0.1-0.3 mg/kg/hr intravenous infusion)  
*Note:* May cause excitement in cats |

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Continued
### TABLE 6-24 Common Drug Indications and Dosages—Cont'd

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<tr>
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<th>Formulation</th>
<th>Recommended Dosage</th>
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</thead>
<tbody>
<tr>
<td>Milbemycin oxime</td>
<td>Interceptor; Interceptor</td>
<td>GABA inhibitor; prevention of canine heartworm disease, microfilaricide; also</td>
<td>23-, 11.5-, 5.75-,</td>
<td>Dog: Microfilaricide: 0.5 mg/kg Demodex: 2 mg/kg PO q24h for 60-120 days Heartworm prevention: 0.5-0.99 mg/kg PO q30 days</td>
</tr>
<tr>
<td></td>
<td>Flavor Tabs</td>
<td>used to treat demodicosis</td>
<td>and 2.3-mg tablets</td>
<td></td>
</tr>
<tr>
<td>Milk of magnesia</td>
<td>See Magnesium hydroxide</td>
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<tr>
<td>(OTC)</td>
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<tr>
<td>Mineral oil (OTC)</td>
<td>Generic</td>
<td>Laxative (lubricant)</td>
<td>Oral liquid</td>
<td>Dog: 10-50 mL/dog PO q12h Cat: 10-25 mL/cat PO q12h</td>
</tr>
<tr>
<td>Minocycline</td>
<td>Minocin</td>
<td>Antibacterial</td>
<td>50- and 100-mg</td>
<td>5-12.5 mg/kg PO q12h</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>tablets; 10 mg/mL</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>oral suspension</td>
<td></td>
</tr>
<tr>
<td>§ Misoprostol</td>
<td>Cytotec</td>
<td>Prostaglandin E₁ analogue; treatment of gastric ulcers, especially those</td>
<td>0.1-mg (100-mcg) and</td>
<td>Dog: 2-5 mcg/kg PO q6-8h Cat: Dose not established</td>
</tr>
<tr>
<td></td>
<td></td>
<td>associated with NSAID use</td>
<td>0.2-mg (200-mcg)</td>
<td></td>
</tr>
<tr>
<td>Mitotane (o,p'-DDD)</td>
<td>Lysodren</td>
<td>Cytotoxic agent; treatment of hyperadrenocorticism associated with adrenal</td>
<td>500-mg tablet</td>
<td>Dog: Pituitary-dependent hyperadrenocorticism: 50 mg/kg/day (divided doses) PO for 7-10 days, then 25 mg/kg/wk PO Adrenal neoplasia: 50-75 mg/kg PO q12h for 10 days, then 75-100 mg/kg PO divided doses q12h</td>
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<tr>
<td></td>
<td></td>
<td>hyperplasia; less effective if treating adrenal gland neoplasia</td>
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<td></td>
</tr>
<tr>
<td>Mitoxantrone</td>
<td>Novantrone</td>
<td>Antineoplastic; used in treatment protocols for multiple tumor types</td>
<td>2 mg/mL injection</td>
<td>Dog: 6 mg/m² IV q21days Cat: 6.5 mg/m² IV q21days</td>
</tr>
</tbody>
</table>

**Note:** OTC = Over-the-counter, PO = by mouth, IV = by injection
<table>
<thead>
<tr>
<th>Drug</th>
<th>Formulation</th>
<th>Indications</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Morphine</strong></td>
<td>Generic</td>
<td>Analgesic (opiate); pain management</td>
<td>1 and 15 mg/mL injection; 30- and 60-mg delayed-release tablets</td>
</tr>
<tr>
<td><strong>Naloxone</strong></td>
<td>Narcan</td>
<td>Opiate antagonist; opiate reversal</td>
<td>20 and 400 mcg/mL injection</td>
</tr>
<tr>
<td><strong>Naltrexone</strong></td>
<td>Trexan</td>
<td>Opiate antagonist; management of certain behavioral disorders (e.g., tail chasing, self-mutilation)</td>
<td>50-mg tablet</td>
</tr>
<tr>
<td><strong>Nandrolone decanoate</strong></td>
<td>Deca-Durabolin</td>
<td>Anabolic steroid; appetite stimulant; also used to stimulate erythropoiesis</td>
<td>50, 100, and 200 mg/mL injection</td>
</tr>
<tr>
<td><strong>Naproxen</strong></td>
<td>Naprosyn, Naxen, Aleve (naproxen sodium)</td>
<td>NSAID; pain management</td>
<td>220-mg tablet (OTC); 25 mg/mL suspension liquid; 250-, 375-, and 500-mg tablets (prescription)</td>
</tr>
<tr>
<td><strong>Neomycin</strong></td>
<td>Biosol</td>
<td>Antibacterial; management of hepatic encephalopathy (gut &quot;sterilization&quot;)</td>
<td>500-mg bolus; 200 mg/mL oral liquid</td>
</tr>
<tr>
<td><strong>Neostigmine bromide and neostigmine methylsulfate</strong></td>
<td>Prostigmin, Stiglyn</td>
<td>Anticholinesterase; diagnosis of myasthenia gravis; antidote for anticholinergic intoxication and massiveivermectin overdose in cats</td>
<td>15-mg tablet (neostigmine bromide); 0.25 and 0.5 mg/mL injection (neostigmine methylsulfate)</td>
</tr>
</tbody>
</table>

Listings preceded by § are for rapid reference and denote drug or dose used in the emergency or critical care setting.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Proprietary Names</th>
<th>Action and Use</th>
<th>Formulation</th>
<th>Recommended Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitrofurantoin</td>
<td>Macrodantin, Furalan, Furatoin, Furadantin, and generic</td>
<td>Antibacterial, especially in susceptible urinary tract infections</td>
<td>Macrodantin and generic: 25-, 50-, and 100-mg capsules; Furalan, Furatoin, and generic: 50- and 100-mg tablets; Furadantin: 5 mg/mL oral suspension</td>
<td>Susceptible UTI = 4 mg/kg PO every 6 hours; Prophylactic dose = 3-4 mg/kg PO q24 hours (at night immediately before bedtime)</td>
</tr>
<tr>
<td>§ Nitroprusside</td>
<td>Nitropress</td>
<td>Vascular and smooth muscle relaxant; acute hypertension, acute heart failure secondary to mitral regurgitation</td>
<td>50-mg vial for injection</td>
<td>Hypertensive crisis: initiate dose at 1-2 mcg/kg/min IV, increase dose incrementally every 3-5 min until target BP is attained; Adjunctive rx for heart failure: 0.5-10 mcg/kg/min IV at a low fluid rate (&lt; = 2 mL/kg/hr)</td>
</tr>
<tr>
<td>Nizatidine</td>
<td>Axid</td>
<td>H₂-receptor antagonist; reduction of gastric acid production and prevention of gastric ulcers</td>
<td>150- and 300-mg capsules</td>
<td>2.5-5.0 mg/kg PO once daily</td>
</tr>
<tr>
<td>Norfloxacin</td>
<td>Noroxin</td>
<td>Antibacterial</td>
<td>400-mg tablet</td>
<td>22 mg/kg PO q12h</td>
</tr>
<tr>
<td>Olsalazine</td>
<td>Dipentum</td>
<td>Antidiarrheal; alternative drug to sulfasalazine for management of colitis in dogs (expensive)</td>
<td>500-mg tablet</td>
<td>Dosage in animals is not established. Dog: 5-10 mg/kg PO q8h is recommended</td>
</tr>
<tr>
<td>Omeprazole</td>
<td>Prilosec (former Losec), Gastrogard (equine paste)</td>
<td>Proton pump inhibitor; gastric ulceration and erosion</td>
<td>20-mg capsule</td>
<td>Dog: 20 mg PO once daily (if &lt;20 kg use 0.7 mg/kg q24h); Cat: 0.7 mg/kg PO once daily</td>
</tr>
<tr>
<td>§ Ondansetron</td>
<td>Zofran</td>
<td>5-HT3 receptor antagonist; antiemetic for patients with severe vomiting</td>
<td>4- and 8-mg tablets; 2 mg/mL injection</td>
<td>0.1-1.0 mg/kg PO 30 min before cancer chemotherapy; For intractable vomiting: 0.11 to 0.176 mg/kg IV, slow push</td>
</tr>
</tbody>
</table>

Note: Is well tolerated in dogs
<table>
<thead>
<tr>
<th>Name</th>
<th>Generic Name</th>
<th>Category</th>
<th>Description</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orbifloxacin</td>
<td>Orbax</td>
<td>Antibacterial</td>
<td>5.7-, 22.7-, and 68-mg tablets</td>
<td>2.5 to 7.5 mg/kg PO once daily</td>
</tr>
<tr>
<td>Ormetoprim + sulfadimethoxine</td>
<td>Primor</td>
<td>Antibacterial</td>
<td>Combination tablet: 120-, 250-, 600-, and 1200-mg tablets</td>
<td>Initially 55 mg/kg (combined drug) PO on the first day of therapy, then 27.5 mg/kg PO once daily for at least 2 days after remission of clinical signs (not approved for treatment &gt;21 days)</td>
</tr>
<tr>
<td>Oxacillin</td>
<td>Prostaphlin, generic</td>
<td>Antibacterial</td>
<td>250- and 500-mg capsules; 50 mg/mL oral solution</td>
<td>22-40 mg/kg PO q8h</td>
</tr>
<tr>
<td>Oxazepam</td>
<td>Serax</td>
<td>Benzodiazepine; appetite stimulant</td>
<td>15-mg tablet</td>
<td>Cat: Appetite stimulant: 2.5 mg/cat PO</td>
</tr>
<tr>
<td>Oxtriphylline</td>
<td>Choledyl SA</td>
<td>Bronchodilator; chronic bronchitis (feline asthma?)</td>
<td>400- and 600-mg tablets (oral solutions and syrup available in Canada but not United States)</td>
<td>Dog: 47 mg/kg (equivalent to 30 mg of theophylline per kilogram) PO q12h Cat: Dose not available</td>
</tr>
<tr>
<td>Oxybutynin</td>
<td>Ditropan</td>
<td>Urinary antispasmodic; adjunctive treatment of detrusor hyperreflexia (includes FeLV-positive cats)</td>
<td>5-mg tablet</td>
<td>Dog: 0.2 mg/kg PO q8-12h (or 1.25-3.75 mg/dog q12h) Cat: 0.5-1.0 mg/kg (total dose) PO q8-12h</td>
</tr>
<tr>
<td>Oxymetholone</td>
<td>Anadrol</td>
<td>Hormone (anabolic steroid); may stimulate erythropoiesis</td>
<td>50-mg tablet</td>
<td>1-5 mg/kg/day PO</td>
</tr>
<tr>
<td>Oxymorphone</td>
<td>Numorphan</td>
<td>Analgesic (opiate); pain management</td>
<td>1.5 and 1 mg/mL injection</td>
<td>Analgesia: 0.1-0.2 mg/kg IV, SQ, IM (as needed); re-treat with 0.05-0.1 mg/kg q1-2h. Preanesthetic: 0.025-0.05 mg/kg IM, SQ</td>
</tr>
<tr>
<td>Oxytetracycline</td>
<td>Terramycin</td>
<td>Antibacterial</td>
<td>250-mg tablets; 100 and 200 mg/mL injection</td>
<td>7.5-10 mg/kg IV q12h; 20 mg/kg PO q12h</td>
</tr>
<tr>
<td>Oxytocin</td>
<td>Pitocin, Syntocinon</td>
<td>Hormone; induction of labor or parturition</td>
<td>10 and 20 units/mL injection; 40 units/mL nasal solution</td>
<td>Dog: 5-20 units/dog SQ, IM (repeat every 30 min for primary inertia) Cat: 0.25-1 units SC or IM every 30-60 min</td>
</tr>
</tbody>
</table>

Listings preceded by § are for rapid reference and denote drug or dose used in the emergency or critical care setting. Continued
<table>
<thead>
<tr>
<th>Drug</th>
<th>Proprietary Names</th>
<th>Action and Use</th>
<th>Formulation</th>
<th>Recommended Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>2-PAM</td>
<td>See Pralidoxime chloride</td>
<td>Digestive enzymes; management of exocrine insufficiency</td>
<td>16,800 units lipase, 70,000 units protease, and 70,000 units amylase per 0.7 g; also capsules and tablets</td>
<td>Mix 2 tsp powder with food per 20 kg; body weight or 1-3 tsp/0.45 kg of food 20 min before feeding</td>
</tr>
<tr>
<td>Pancreatic enzymes</td>
<td>Viokase</td>
<td>Neuromuscular blocker; muscle relaxation as an adjunct to anesthesia</td>
<td>1 and 2 mg/mL injection</td>
<td>0.1 mg/kg IV, or start with 0.01 mg/kg and additional doses of 0.01 mg/kg q30min</td>
</tr>
<tr>
<td>Pancuronium bromide</td>
<td>Pavulon</td>
<td>Antidiarrheal; management of simple diarrhea</td>
<td>2 mg morphine per 5 mL paregoric</td>
<td>0.05-0.06 mg/kg PO q12h</td>
</tr>
<tr>
<td>Paregoric</td>
<td>Corrective mixture</td>
<td>Antiparasitic; cryptosporidiosis in cats</td>
<td>250-mg capsule</td>
<td>Cat: 125-165 mg/kg PO q12h for 7 days. Warning: Toxicity and renal damage have been reported at these doses.</td>
</tr>
<tr>
<td>Paromomycin</td>
<td>Humatin</td>
<td>Chelating agent; treatment of lead poisoning; also for cystine urolithiasis</td>
<td>125- and 250-mg capsules and 250-mg tablet</td>
<td>Dog: 1 mg/kg up to 3 mg/kg PO once daily Cat: 2.5-5 mg total dose PO once daily</td>
</tr>
<tr>
<td>Paroxetine</td>
<td>Paxil</td>
<td>SSRI; management of behavior disorders</td>
<td>10-, 20-, 30-, and 40-mg tablets</td>
<td>10-15 mg/kg PO q12h</td>
</tr>
<tr>
<td>d-Penicillamine</td>
<td>Cuprimine, Depen</td>
<td>Antibacterial</td>
<td>150,000 units/mL, combined with 150,000 units/mL procaine, penicillin G</td>
<td>40,000 IU/kg IM q5 days</td>
</tr>
<tr>
<td>Penicillin G benzathine</td>
<td>Benza-Pen, others</td>
<td>Antibacterial</td>
<td>5 million– to 20 million–unit vials</td>
<td>20,000-40,000 units/kg IV, IM q6-8h</td>
</tr>
<tr>
<td>Penicillin G potassium; penicillin G sodium</td>
<td>Multiple</td>
<td>Antibacterial</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drug</td>
<td>Generic</td>
<td>Indication</td>
<td>剂型</td>
<td>Dosage</td>
</tr>
<tr>
<td>--------------------</td>
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<td>---------------------------------------------</td>
</tr>
<tr>
<td>Penicillin G</td>
<td>Procaine</td>
<td>Antibacterial</td>
<td>300,000 units/mL suspension</td>
<td>20,000-40,000 units/kg SC, IM q12-24h</td>
</tr>
<tr>
<td>Penicillin V</td>
<td>Pen-Vee</td>
<td>Antibacterial</td>
<td>250- and 500-mg tablets</td>
<td>10 mg/kg PO q8h</td>
</tr>
<tr>
<td>Pentazocine</td>
<td>Talwin-V</td>
<td>Analgesic (opiate); pain management</td>
<td>30 mg/mL injection</td>
<td>Dog: 1.65-3.3 mg/kg IM q4h Cat: 2.2-3.3 mg/kg IV, IM, SQ q4h (due to dysphoria, pentazocine is not recommended in cats)</td>
</tr>
<tr>
<td>§ Pentobarbital</td>
<td>Nembutal</td>
<td>Anesthetic; sedative or injectable anesthetic</td>
<td>50 mg/mL Note: This formulation is not to be used for euthanasia.</td>
<td>Anesthesia: 10-30 mg/kg IV to effect</td>
</tr>
<tr>
<td>Pentoxifylline</td>
<td>Trental</td>
<td>Antiinflammatory effects; has been used to treat immune-mediated skin disorders (e.g., associated with vasculitis) in dogs</td>
<td>400-mg tablet</td>
<td>Dog: For use in canine dermatology and for systemic or regional vasculitis, 10 mg/kg PO q12h</td>
</tr>
<tr>
<td>§ Phenobarbital</td>
<td>Luminal</td>
<td>Barbiturate; sedation and anticonvulsant</td>
<td>15-, 30-, 60-, and 100-mg tablets; 30, 60, 65, and 130 mg/mL injection; 4 mg/mL oral elixir solution</td>
<td>Dog: 2-8 mg/kg PO q12h Cat: 2-4 mg kg PO q12h Dog and cat: Adjust dose by monitoring plasma concentration Status epilepticus: administer in increments of 10-20 mg/kg IV to effect</td>
</tr>
<tr>
<td>Phenoxybenzamine</td>
<td>Dibenzyline</td>
<td>α-Adrenergic blocker; reduces internal urethral sphincter tone associated with detrusor areflexia; also hypertension associated with pheochromocytoma</td>
<td>10-mg capsule</td>
<td>Dog: Urinary: 0.25 mg/kg PO 12-24h or 0.5 mg/kg q24h Hypertension: 0.2-1.5 mg/kg, PO bid for 10-14 days before surgery Cat: 2.5 mg/cat q8-12h or 0.5 mg/cat PO q12h Note: In cats, doses as high as 0.5 mg/kg IV have been used to relax urethral smooth muscle</td>
</tr>
<tr>
<td>Phentolamine</td>
<td>Regitine</td>
<td>Vasodilator; hypertension</td>
<td>5-mg vial for injection</td>
<td>0.02-0.1 mg/kg IV</td>
</tr>
</tbody>
</table>

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<tr>
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<th>Formulation</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Phenylbutazone</td>
<td>Butazolidin, generic</td>
<td>NSAID</td>
<td>100-, 200-, 400-mg and 1-g tablets; 200 mg/mL injection</td>
<td><em>Not recommended for use in dogs and cats (better drugs are available)</em></td>
</tr>
<tr>
<td>§ Phenylephrine</td>
<td>Neo-Synephrine</td>
<td>α-Adrenergic; treatment of hypotension in the critical care setting; also used topically intranasally before rhinoscopy</td>
<td>10 mg/mL injection; 1% nasal solution</td>
<td>Dog and cat: 1-3 mcg/kg/min CRI in 0.9% saline or D5W; 0.1 mg/kg IM, SQ q15min Topical: 3-5 drops intranasally to effect to induce local vasoconstriction</td>
</tr>
<tr>
<td>Phenylpropanolamine</td>
<td>Dexatrim, Propagest, others</td>
<td>Adrenergic agonist; urinary incontinence associated with urethral sphincter hypotonus</td>
<td>15-, 25-, 30-, and 50-mg tablets</td>
<td>Dog: 12.5-50 mg (total) PO q8h or 1.5-2 mg/kg PO q12h Cat: 12.5 mg (total) PO q8h or 1.5 mg/kg PO q8h</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>Dilantin</td>
<td>Anticonvulsant; not generally recommended; limited application in digoxin-induced arrhythmias</td>
<td>30 and 1250 mg/mL oral suspension; 30- and 100-mg capsules; 50 mg/mL injection</td>
<td>Antiepileptic (dog): 20-34 mg/kg q8h Digoxin-induced antiarrhythmia: 30 mg/kg PO q8h or 10 mg/kg IV over 5 min</td>
</tr>
<tr>
<td>§ Phenytoin + pentobarbital</td>
<td>Beuthanasia-D Special, Euthasol</td>
<td>Euthanasia solution</td>
<td>100-mL multiple-dose vials</td>
<td>1 mL/10 lb body weight IV. *Note: Alternative routes (at the same dosage) for sodium administration can be used in profoundly debilitated patients (e.g., intraperitoneal, intracardiac).</td>
</tr>
<tr>
<td>Physostigmine</td>
<td>Antilirium</td>
<td>Cholinesterase inhibitor/limited application; may be of use in promoting micturition in patients with urinary retention (postoperatively)</td>
<td>1 mg/mL injection</td>
<td>0.02 mg/kg IV q12h</td>
</tr>
<tr>
<td>Drug</td>
<td>Formulation</td>
<td>Indication</td>
<td>Dosage/Frequency</td>
<td></td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-------------</td>
<td>-----------------------------------------------------------------------------</td>
<td>-------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Phytomenadione</td>
<td>See Vitamin K₁</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phytanadione</td>
<td>See Vitamin K₁</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Piperacillin</td>
<td>Pipracil</td>
<td>Antibacterial</td>
<td>2-, 3-, 4-, and 40-g vials for injection</td>
<td>40 mg/kg IV or IM q6h</td>
</tr>
<tr>
<td>Piperazine</td>
<td>Many</td>
<td>Antiparasitic; roundworms</td>
<td>860 mg powder; 140-mg capsule, 170, 340, and 800 mg/mL oral solution</td>
<td>44-66 mg/kg PO administered once</td>
</tr>
<tr>
<td>Piroxicam</td>
<td>Feldene, generic</td>
<td>NSAID; has antitumor effects (indirect) in patients with transitional cell carcinoma (palliative treatment)</td>
<td>10-mg capsule</td>
<td>Dog: 0.3 mg/kg PO once daily</td>
</tr>
<tr>
<td>Pitressin (ADH)</td>
<td>See Vasopressin and Desmopressin acetate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plicamycin</td>
<td>Mithracin</td>
<td>Antineoplastic; adjunctive treatment in carcinoma protocols; also used to decrease calcium levels in hypercalcemic cancer patients</td>
<td>2.5 mg/mL injection</td>
<td>Dog: Antineoplastic: 25-30 mcg/kg/day IV (slow infusion) for 8-10 days</td>
</tr>
<tr>
<td>Polyethylene glycol electrolyte solution</td>
<td>GoLYTELY</td>
<td>Laxative</td>
<td>Oral solution</td>
<td>25 mL/kg PO; repeat in 2-4 hr</td>
</tr>
<tr>
<td>Polysulfated glycosaminoglycan (PSGAG)</td>
<td>Adequan Canine</td>
<td>Antiarthritic; long-term management of osteoarthritis</td>
<td>100 mg/mL injection in 5-mL vial (250 mg/mL vials for horses)</td>
<td>4.4 mg/kg IM twice weekly for up to 4 wk</td>
</tr>
</tbody>
</table>

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<th>Recommended Dosage</th>
</tr>
</thead>
</table>
| Potassium bromide (KBr)       | No commercial formulation | Anticonvulsant; long-term antiepileptic therapy   | Usually prepared as oral solution              | Dog and Cat: 30-40 mg/kg PO q24h  
Note: If administered without phenobarbital, higher doses of up to 40-50 mg/kg may be needed; adjust doses by monitoring plasma concentrations; loading doses of 400 mg/kg divided over 3 days have been administered. |
| $ Potassium chloride (KCl)    | Generic           | Potassium salt; replacement therapy               | Various concentrations for injection (usually 2 mEq/mL); oral suspension and oral solution | 0.5 mEq potassium/kg/day; or supplement 10-40 mEq/500 mL of fluids, depending on serum potassium |
| Potassium citrate             | Urocit-K and generic | Potassium salt; replacement therapy               | 5-mEq tablet; some forms are in combination with potassium chloride | 2.2 mEq/100 kcal of energy/day PO; or 0.5 mEq/kg per day PO |
| Potassium gluconate           | Kaon; Tumil-K; generic | Potassium source; replacement therapy            | 2-mEq tablet; 500-mg tablet; Kaon elixir is 20 mg/15 mL elixir | Dog: 0.5 mEq/kg PO 12-24h  
Cat: 2-8 mEq/day PO divided twice daily |
| $ Pralidoxime chloride (2-PAM) | Protopam Chloride | Cholinesterase reactivator; adjunctive treatment in patients with organophosphate toxicosis | 50 mg/mL injection                              | 20 mg/kg q8-12h (initial dose) IV slowly or IM |
| Praziquantel                  | Droncit           | Antiparasitic; treatment of cestodes (tapeworms)  | 23- and 34-mg tablets; 56.8 mg/mL injection     | Dog IM/SC dosing: ≤5 lbs, 17 mg IM/SC; 6-10 lbs, 28.4 mg IM/SC; 11-25 lbs, 56.8 mg IM/SC  
Dog PO dosing: ≤5 lbs, 17 mg PO; 6-10 lbs, 34 mg PO; 11-15 lbs, 51 mg PO; 16-30 lbs, 68 mg PO; 31-45 lbs, 102 mg PO; 46-60 lbs, 136 mg PO; ≥60 lbs, 170 mg  
Cat IM/SC dosing: ≤5 lbs, 11.4 mg IM/SC; 5-10 lbs, 22.7 mg IM/SC; ≥11 lbs, 34.1 mg IM/SC  
Cat PO dosing: ≤4 lbs, 11.5 mg PO; 5-11 lbs, 23 mg PO; ≥11 lbs, 34.5 mg PO  
for Paragonimus: 23-25 mg/kg PO q8h for 3 days |
<table>
<thead>
<tr>
<th>$ Prazosin</th>
<th>Minipress</th>
<th>$ α_1$-Blocker; adjunctive treatment of congestive heart failure; also hypertension and pulmonary hypertension (e.g., heartworm disease)</th>
<th>1-, 2-, and 5-mg capsules</th>
<th>0.5- and 2-mg/animal (1 mg/15 kg) PO q8-12h</th>
</tr>
</thead>
<tbody>
<tr>
<td>$ Prednisolone</td>
<td>Delta-Cortef; many others</td>
<td>Glucocorticoid; antiinflammatory and immunosuppressive</td>
<td>5- and 20-mg tablets</td>
<td>Dog (cat often requires 2 × dog dose): Antiinflammatory: 0.5-1 mg/kg IV, IM, PO q12-24h initially, then taper to q48h Immunosuppressive: 2.2-6.6 mg/kg/day IV, IM, PO initially, then taper to 2-4 mg/kg q48h Replacement therapy: 0.2-0.3 mg/kg/day PO Shock, spinal trauma: See Prednisolone sodium succinate</td>
</tr>
<tr>
<td>$ Prednisolone sodium succinate</td>
<td>Solu-Delta-Cortef</td>
<td>Glucocorticoid; adjunctive therapy for endotoxic or septic shock</td>
<td>100- and 200-mg vials for injection (10 and 50 mg/mL)</td>
<td>Shock: 5.5-11 mg/kg IV (repeat in 1, 3, 6, or 10 hr) CNS trauma: 15-30 mg/kg IV, then taper to 1-2 mg/kg q12h</td>
</tr>
<tr>
<td>$ Prednisone</td>
<td>Deltasone and generic; Meticorten for injection</td>
<td>Glucocorticoid; antiinflammatory and immunosuppressive</td>
<td>1-, 2.5-, 5-, 10-, 20-, 25-, and 50-mg tablets; 1 mg/mL syrup (Liquid-Pred in 5% alcohol); 1 mg/mL oral solution (in 5% alcohol); 10 and 40 mg/mL prednisone suspension for injection</td>
<td>Same as for prednisolone</td>
</tr>
</tbody>
</table>

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### Table 6-24: Common Drug Indications and Dosages—Cont’d

<table>
<thead>
<tr>
<th>Drug</th>
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<th>Formulation</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Primidone</td>
<td>Mylepsin; Neurosyn</td>
<td>Anticonvulsant; idiopathic epilepsy (not generally recommended)</td>
<td>50- and 250-mg tablets</td>
<td>8-10 mg/kg PO q8-12h as initial dose, then adjust via monitoring to 10-15 mg/kg q8h</td>
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<td></td>
<td><strong>Warning:</strong> May cause irreversible liver disease with prolonged administration</td>
</tr>
<tr>
<td>Procainamide</td>
<td>Pronestyl and generic</td>
<td>Antiarrhythmic; ventricular premature contractions (e.g., ventricular tachycardia)</td>
<td>250, 375, 500 mg/mL injection</td>
<td>Dog: 10-30 mg/kg PO q6h (up to maximum dose of 40 mg/kg); 8-20 mg/kg IV, IM; 25-50 mcg/kg/min IV infusion</td>
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<tr>
<td></td>
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<td></td>
<td>Cat: 3-8 mg/kg IM, PO q6-8h</td>
</tr>
<tr>
<td>Procarbazine</td>
<td>Matulane, Natulan, Natulanar</td>
<td>Antineoplastic; component drug used in lymphoma protocols</td>
<td>50-mg capsule</td>
<td>Used in combination with mechlorethamine and prednisolone; consult latest information on protocols for precise dose</td>
</tr>
<tr>
<td>Prochlorperazine</td>
<td>Compazine</td>
<td>Phenothiazine; antiemetic</td>
<td>5-, 10-, and 25-mg tablets (maleate); 5 mg/mL injection (edisylate)</td>
<td>0.1-0.5 mg/kg IM, SQ q6-8h</td>
</tr>
<tr>
<td>Progesterone, repositol</td>
<td>See Medroxyprogesterone acetate</td>
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<td></td>
</tr>
<tr>
<td>Promethazine</td>
<td>Phenergan</td>
<td>Phenothiazine; antiemetic</td>
<td>6.25 and 25 mg/5 mL syrup; 12.5-, 25-, and 50-mg tablets; 25 and 50 mg/mL injection</td>
<td>Antiemetic: 2 mg/kg IM/PO once daily</td>
</tr>
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<td></td>
<td></td>
<td>Antihistamine: 0.2-0.4 mg/kg IV/IM/PO q6-8h (up to max dose of 1 mg/kg)</td>
</tr>
<tr>
<td>Propantheline</td>
<td>Pro-Banthine</td>
<td>Antimuscarinic, antidiarrheal; also used to treat urge incontinence associated with detrusor hyperreflexia; oral antiemetic effect</td>
<td>7.5- and 15-mg tablets</td>
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</tbody>
</table>
|               |            | Dog: Urge incontinence: 0.2 mg/kg PO q6-8h  
Diarrhea: 0.25 mg/kg PO three times daily for 2-3 days max  
Cat: Urge incontinence: 0.25-0.5 mg/kg PO once or twice daily  
Chronic colitis: 0.5 mg/kg PO two to three times daily |

**Propionibacterium acnes** (injection)  
**ImmunoRegulin**  
Nonspecific immunostimulant used as adjunctive therapy in dogs with pyoderma  
5 mL vial  
Dog: Manufacturer’s dosing recommendations:  
- up to 15 pounds: 0.25 mL IV  
- 16-45 pounds: 0.50 mL IV  
- 46-75 pounds: 1.0 mL IV  
- over 75 pounds: 2.0 mL IV  
Product is not approved for use in cats.

**Propiopromazine**  
**Tranvet, Largon**  
Antiemetic, tranquilizer; sedation, parenteral antiemetic  
20 mg/mL injection  
1.1-4.4 mg/kg q12-24h PO or 0.1-1.1 mg/kg IV or IM (range depends on sedation needed)

§ **Propofol**  
**Rapinovet, PropoFlo**  
Short-acting injectable anesthetic (hypnotic); induction or restraint for short-term procedures  
1% (10 mg/mL) injection in 20-mL ampules  
6.6 mg/kg IV slowly over 60 sec (CRIs have been used at 2 mg/kg/hr)

§ **Propranolol**  
**Inderal (other products are available)**  
β-Blocker, antiarrhythmic  
10-, 20-, 40-, 60-, 80-, and 90-mg tablets; 1 mg/mL injection; 4 and 8 mg/mL oral solution  
Dog: 20-60 mcg/kg over 5-10 min IV; 0.2-1 mg/kg PO q8h (titrate dose to effect)  
Cat: 0.4-1.2 mg/kg (2.5-5 mg/cat) PO q8h

**Propylthiouracil**  
**Propyl-Thyracil, generic**  
Antithyroid; alternative drug used in the management of feline hyperthyroidism  
50- and 100-mg tablets  
Cat: 11 mg/kg PO q12h

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<tr>
<td>Prostaglandin F₂, alpha (dinoprost)</td>
<td>Lutalyse</td>
<td>Prostaglandin; open pyometra; pregnancy termination in dogs</td>
<td>5 mg/mL solution for injection</td>
<td>Note: Any use of this drug in dogs and cats is extra-label. Open pyometra: Dog: 0.1-0.2 mg/kg SQ once daily for 5 days Cat: 0.1-0.25 mg/kg SQ twice daily for 5 days Note: Concurrent antibiotic therapy is recommended. Surgery is always preferred! Abortion (within 30 days of the last unwanted breeding): Dog: 0.1 mg/kg SQ q8h for 2 days, then 0.2 mg/kg SQ q8h until abortion is confirmed by ultrasound</td>
</tr>
<tr>
<td>Pseudoephedrine</td>
<td>Sudafed, many others (some formulations have other ingredients)</td>
<td>Adrenergic agonist; urinary incontinence (generally only used when phenylpropanolamine is not available)</td>
<td>30- and 60-mg tablets; 120-mg capsule; 6 mg/mL syrup</td>
<td>0.2-0.4 mg/kg (or 15-60 mg/dog) PO (OTC) q8-12h</td>
</tr>
<tr>
<td>Psyllium</td>
<td>Metamucil, others</td>
<td>Laxative, stool softener</td>
<td>Available as powder</td>
<td>1 tsp/5-10 kg (added to each meal)</td>
</tr>
<tr>
<td>Pyrantel pamoate and tartrate</td>
<td>Nemex, Strongid</td>
<td>Antiparasitic; treatment of ascarids and hookworms</td>
<td>180 mg/mL paste and 50 mg/mL suspension</td>
<td>Dog: 5 mg/kg PO once; repeat in 7-10 days Cat: 20 mg/kg PO once</td>
</tr>
<tr>
<td>Pyridostigmine bromide</td>
<td>Mestinon, Regonol</td>
<td>Cholinesterase inhibitor; management of myasthenia gravis</td>
<td>12 mg/mL oral syrup; 60-mg tablet; 5 mg/mL injection</td>
<td>Antimyasthenic: 0.02-0.04 mg/kg IV q2h, or 0.5-3 mg/kg PO q8-12h Antidote (nondepolarizing muscle relaxant): 0.15-0.3 mg/kg IM, IV</td>
</tr>
<tr>
<td>Drug Name</td>
<td>Trade Name</td>
<td>Description</td>
<td>Dosage</td>
<td>Notes</td>
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</tr>
<tr>
<td>Pyrimethamine</td>
<td>Daraprim</td>
<td>Folic acid inhibitor; treatment of toxoplasmosis and neosporosis</td>
<td>25-mg tablet</td>
<td>Dog: 1 mg/kg PO q24h for 14-21 days (5 days for <em>Neospora caninum</em>)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cat: 0.5-1 mg/kg PO q24h for 14-28 days</td>
</tr>
<tr>
<td>Quinacrine</td>
<td>Limited availability in the United States</td>
<td>Antiprotozoal; may be useful in management (not cure) of <em>Giardia</em> infections, leishmaniasis, and coccidiosis</td>
<td>100-mg tablet</td>
<td>Dog: 6.6 mg/kg PO q12h for 5 days</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Cat: 11 mg/kg PO q24h for 5 days</td>
</tr>
<tr>
<td>Quinidine gluconate</td>
<td>Quiniglute, Duraquin</td>
<td>Antiarrhythmic; ventricular arrhythmias</td>
<td>324-mg tablets; 80 mg/mL injection</td>
<td>Dog: 6-20 mg/kg IM q6h; 6-20 mg/kg PO q6-8h (of base)</td>
</tr>
<tr>
<td>Quinidine polygalacturonate</td>
<td>Cardioquin</td>
<td>Antiarrhythmic; ventricular arrhythmias</td>
<td>275-mg tablet</td>
<td></td>
</tr>
<tr>
<td>Quinidine sulfate</td>
<td>Cin-Quin, Quinora</td>
<td>Antiarrhythmic; ventricular arrhythmias</td>
<td>100-, 200-, and 300-mg tablets; 200- and 300-mg capsules; 20 mg/mL injection</td>
<td>Dog: 6-20 mg/kg PO q6-8h (of base); 5-10 mg/kg IV</td>
</tr>
<tr>
<td>§ Ranitidine</td>
<td>Zantac</td>
<td>H\textsubscript{2}-receptor antagonist; treatment and prevention of gastric and duodenal ulcers</td>
<td>75-, 150-, and 300-mg tablets; 150- and 300-mg capsules; 25 mg/mL injection</td>
<td>Dog: 2 mg/kg IV; PO q8h</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cat: 2.5 mg/kg IV q12h; 3.5 mg/kg PO q12h</td>
</tr>
<tr>
<td>Retinol</td>
<td>See Vitamin A</td>
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<tr>
<td>Riboflavin</td>
<td>See Vitamin B\textsubscript{2}</td>
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<tr>
<td>Rifampin</td>
<td>Rifadin</td>
<td>Antibacterial (reported to have limited antifungal and antiviral activity)</td>
<td>150- and 300-mg capsules</td>
<td>10-20 mg/kg PO q8-12h</td>
</tr>
<tr>
<td>§ Ringer’s solution</td>
<td>Generic</td>
<td>Fluid replacement</td>
<td>250-, 500-, and 1000-mL bags for infusion</td>
<td>55-65 mL/kg/day (2.5 mL/kg/hr IV, SQ, IP maintenance)</td>
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<td></td>
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<td></td>
<td>15-30 mL/kg/hr IV for moderate dehydration</td>
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<td></td>
<td></td>
<td></td>
<td>50 mL/kg/hr IV for severe dehydration</td>
</tr>
<tr>
<td>Salicylate</td>
<td>See Aspirin</td>
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<tr>
<td>SAMe (S-adenosyl-methionine)</td>
<td>Denosyl-SD4</td>
<td>Nucleotide-like molecule derived from the amino acid methionine; adjunctive therapy in patients with chronic liver disease</td>
<td>Enteric-coated tablets</td>
<td>20 mg/kg PO daily</td>
</tr>
<tr>
<td>Selamectin</td>
<td>Revolution</td>
<td>Antiparasitic (ivermectin); multiple applications in dogs and cats</td>
<td>Various sizes of topical solutions available for dogs and cats</td>
<td>See manufacturer’s dosage instructions for the specific condition being treated</td>
</tr>
<tr>
<td>Selegiline</td>
<td>Anipryl (also known as deprenyl and L-deprenyl)</td>
<td>MAO-B inhibitor; canine cognitive dysfunction; reported use in treatment of canine hyperadrenocorticism (use in canine Cushing is currently not recommended)</td>
<td>2-, 5-, 10-, 15-, and 30-mg tablets</td>
<td>Dog: Begin with 1 mg/kg PO q24h; if no response within 2 mo, increase dose to maximum of 2 mg/kg PO q24h Cat: Dose not established.</td>
</tr>
<tr>
<td>Senna</td>
<td>Senokot</td>
<td>Laxative; feline constipation</td>
<td>Granules in concentrate, or syrup</td>
<td>Cat: syrup: 5 mL/cat q24h; granules ½ teaspoon/cat q24h (with food)</td>
</tr>
<tr>
<td>Sertraline</td>
<td>Zoloft, Altruline, Anilar, others</td>
<td>SSRI; management of certain behavior disorders in dogs</td>
<td>25-, 50-, and 100-mg tablets; 20 mg/mL injectable in 60-mL vials</td>
<td>Dog: 0.5-4.0 mg/kg q24h Cat: 0.5-1.0 mg/kg q24h</td>
</tr>
<tr>
<td>§ Sodium bicarbonate (NaHCO₃) (OTC)</td>
<td>Generic (e.g., baking soda, soda mint)</td>
<td>Alkalinizing agent; management of acidosis and renal failure; also used to alkalinize urine when indicated</td>
<td>325-, 520-, and 650-mg tablets; injection of various strengths (4.2% to 8.4%), and 1 mEq/mL</td>
<td>Acidosis: 0.5-1 mEq/kg IV Renal failure: 10 mg/kg PO q8-12h Alkalization of urine: 50 mg/kg PO q8-12h (1 tsp is approximately 2 g)</td>
</tr>
<tr>
<td>Drug</td>
<td>Use</td>
<td>Dosage</td>
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<tr>
<td><strong>Sodium chloride 0.9%</strong></td>
<td>Fluid replacement</td>
<td>500- and 1000-mL infusion</td>
<td></td>
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<tr>
<td>§</td>
<td></td>
<td>Moderate dehydration: 15-30 mL/kg/hr IV, severe dehydration 50 mL/kg/hr IV</td>
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</tr>
<tr>
<td><strong>Sodium chloride 7.2%</strong></td>
<td>Fluid replacement</td>
<td>Infusion</td>
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<tr>
<td>§</td>
<td></td>
<td>2-8 mL/kg IV</td>
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<tr>
<td><strong>Sodium iodide 20%</strong></td>
<td>Iodine replacement; replacement for confirmed deficiencies</td>
<td>100 mcg elemental iodide (118 mcg sodium iodide) per milliliter injection</td>
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<tr>
<td><strong>Sotalol</strong></td>
<td>Nonselective β-blocker</td>
<td>80-, 160-, 240-mg tablets</td>
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<tr>
<td>§</td>
<td>(antiarrhythmic); ventricular tachycardia</td>
<td>Dog: 1-2 mg/kg PO q12h (start with 40 mg/dog q12h, then increase to 80 mg if no response)</td>
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<tr>
<td>§</td>
<td></td>
<td>Cat: 1-2 mg/kg PO q12h</td>
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</tr>
<tr>
<td><strong>Spironolactone</strong></td>
<td>Aldosterone antagonist; K-sparing diuretic used in the treatment of congestive heart failure; generally used in patients that do not respond to furosemide and ACE inhibitors</td>
<td>25-, 50-, and 100-mg tablets</td>
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<tr>
<td>§</td>
<td></td>
<td>2-4 mg/kg/day (or 1-2 mg/kg PO q12h)</td>
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<tr>
<td><strong>Stanozolol</strong></td>
<td>Anabolic steroid; adjunctive therapy for no one really knows what; has been used to treat anemia of chronic disease</td>
<td>50 mg/mL injection; 2-mg tablet</td>
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<tr>
<td>§</td>
<td></td>
<td>Dog: 2 mg/dog (or range of 1-4 mg/dog) PO q12h; 25-50 mg/dog/wk IM</td>
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<td></td>
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<tr>
<td>§</td>
<td></td>
<td>Cat: 1 mg/cat PO q12h; 25 mg/cat/wk IM</td>
<td></td>
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<tr>
<td>§</td>
<td></td>
<td><strong>Caution:</strong> Use in anorexic patients can cause weight loss (catabolic effect?).</td>
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</tr>
<tr>
<td><strong>Succimer</strong></td>
<td>Heavy metal chelator; treatment of lead poisoning</td>
<td>100-mg capsule</td>
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<tr>
<td>§</td>
<td></td>
<td>10 mg/kg PO q8h for 5 days, then 10 mg/kg PO q12h for 2 more wk</td>
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</table>
| § Sucralfate       | Carafate          | Antiulcer treatment; treatment of gastric and duodenal ulcers (may have preventive effect) | 1-g tablet; 200 mg/mL oral suspension | Dog: 0.5-1 g/dog PO q8-12h  
Cat: 0.25 g/cat PO q8-12h |
| Sufentanil         | Sufenta           | Analgesic (potent opiate); adjunct to anesthesia or epidural anesthesia       | 50 mcg/mL injection               | Dogs: 3 mcg/kg IV (maximum dose is 5 mcg/kg IV)  
Cat: 0.1-0.5 mcg/kg IV |
| Sulfadiazine       | Generic combined with trimethoprim in Tribrissen | Antibacterial | 500-mg tablet | 100 mg/kg IV PO (loading dose), followed by 50 mg/kg IV PO q12h (see also Trimethoprim + sulfonamide) |
| Sulfadimethoxine   | Albon, Bactrovet, generic | Antibacterial | 125-, 250-, and 500-mg tablets; 400 mg/mL injection; 50 mg/mL suspension | 55 mg/kg PO (loading dose), followed by 27.5 mg/kg PO once daily (see Ormetoprim + sulfadimethoxine) |
| Sulfamethazine     | Many brand name products (e.g., Sulmet) | Antibacterial | 30-g bolus | 100 mg/kg PO (loading dose), followed by 50 mg/kg PO q12h |
| Sulfamethoxazole   | Gantanol          | Antibacterial | 50-mg tablet | 100 mg/kg PO (loading dose), followed by 50 mg/kg PO q12h |
| Sulfasalazine      | Azulfidine        | Antibacterial and antiinflammatory activity; ulcerative colitis and other forms of inflammatory bowel disease in dogs | 500-mg tablets; pediatric suspension | Dog: 10-30 mg/kg PO q8-12h  
Warning: Has been reported to cause keratoconjunctivitis sicca in dogs |
| Sulfisoxazole      | Gantrisin         | Antibacterial | 500-mg tablet; 500 mg/5 mL syrup | 50 mg/kg PO q8h (urinary tract infections) |
| Taurine            | Generic           | Amino acid; taurine deficiency cardiomyopathies | Available in powder | Dog: 500 mg PO q12h  
Cat: 250 mg/cat PO q12-24h |
| Drug | Trade Name(s) | Use | Dosage *
|------|--------------|-----|-------
| Tepoxalin | Zubrin | NSAID; management of pain associated with osteoarthritis in dogs | 30-, 50-, 100-, and 200-mg tablets | Dog: 10-20 mg/kg PO on the first day; then 10 mg/kg PO once daily; thereafter as needed |
| § Terbutaline | Brethine, Bricanyl | β-agonist; bronchodilator; use includes feline asthma | 2.5- and 5-mg tablets; 1 mg/mL injection (equivalent to 0.82 mg/mL) | Dog: 1.25-5 mg/dog PO q8h Cat: 0.1-0.2 mg/kg PO q12h (or 0.625 mg/cat, 1/4 of 2.5-mg tablet) |
| Testosterone cypionate ester | Andro-Cyp, Andronate, Depo-Testosterone, others | Hormone; replacement therapy; most commonly used for testosterone-responsive urinary incontinence in neutered male dogs and cats | 100 and 200 mg/mL injection | 1-2 mg/kg IM q2-4 wk (see also Methyltestosterone) |
| Testosterone propionate ester | Testex | Hormone; replacement therapy; most commonly used for testosterone-responsive urinary incontinence in neutered male dogs and cats | 100 mg/mL injection | 0.5-1 mg/kg IM 2-3 times/wk |
| Tetracycline | Panmycin | Antibacterial | 250- and 500-mg capsules; 100 mg/mL suspension | 15-20 mg/kg PO q8h; or 4.4-11 mg/kg IV, IM q8h |
| Thenium closylate | Canopar | Antiparasitic; hookworms | 500-mg tablet | Dog: >4.5 kg, 500 mg PO once and repeat in 2-3 wk; 2.5-4.5 kg, 250 mg q12h for 1 day and repeat in 2-3 wk |
| Theophylline | Many brand name and generic products | Bronchodilator; chronic bronchitis and feline asthma | 100-, 125-, 200-, 250-, and 300-mg tablets; 27 mg/5 mL oral solution or elixir; injection in 5% dextrose | Dog: 9 mg/kg PO q6-8h Cat: 4 mg/kg PO q8-12h (see also Aminophylline) |

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<tbody>
<tr>
<td>Theophylline, sustained-release</td>
<td>Theo-Dur, Slo-Bid Gyrocaps</td>
<td>Bronchodilator; chronic bronchitis and feline asthma</td>
<td>100-, 200-, 300-, and 450-mg tablets (Theo-Dur); 50- to 200-mg capsules (Slo-Bid)</td>
<td>Dog: 10 mg/kg PO q12h, if adequate but no side effects increase to 15 mg/kg PO q12h Cat: 20 mg/kg PO q24h (at night) for theo-pur and 25 mg/kg PO q24h for slo-bid</td>
</tr>
<tr>
<td>Thiabendazole</td>
<td>Omnizole, Equizole, Tresaderm (topical-otic)</td>
<td>Antiparasitic; multiple applications for parasitic infections</td>
<td>2 or 4 g/oz (30 mL) suspension or liquid</td>
<td>Dog: 50 mg/kg q24h for 3 days and repeat in 1 mo; Respiratory parasites: 30-70 mg/kg PO q12h Cat: Strongyloides: 125 mg/kg q24h for 3 days</td>
</tr>
<tr>
<td>Thiacetarsamide sodium</td>
<td>Caparsolate</td>
<td>Arsenical; formerly used to treat canine heartworm disease</td>
<td>Not commercially available</td>
<td></td>
</tr>
<tr>
<td>Thiamine</td>
<td>See Vitamin B&lt;sub&gt;1&lt;/sub&gt;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thioguainine (6-TG)</td>
<td>Generic</td>
<td>Antineoplastic; lymphocytic or granulocytic leukemia</td>
<td>40-mg tablet</td>
<td>Dog: 40 mg/m&lt;sup&gt;2&lt;/sup&gt; PO q24h Cat: 25 mg/m&lt;sup&gt;2&lt;/sup&gt; PO q24h for 1-5 days</td>
</tr>
<tr>
<td>Thiopental sodium</td>
<td>Pentothal</td>
<td>Short-acting injectable anesthetic; anesthesia induction or restraint for short procedures</td>
<td>Various size vials from 250 mg to 10 g (mix to desired concentration)</td>
<td>Dog: 10-25 mg/kg IV (to effect) Cat: 5-10 mg/kg IV (to effect)</td>
</tr>
<tr>
<td>Thiotepa</td>
<td>Generic</td>
<td>Antineoplastic; lymphocytic or granulocytic leukemia</td>
<td>15-mg injection (usually in solution of 10 mg/mL)</td>
<td>0.2-0.5 mg/m&lt;sup&gt;2&lt;/sup&gt;/wk, or daily for 5-10 days (IM, intracavitary, or intratumor)</td>
</tr>
<tr>
<td>Thyroid hormone</td>
<td>See Levothyroxine sodium (&lt;i&gt;T&lt;/i&gt;&lt;sub&gt;4&lt;/sub&gt;) and Liothyronine (&lt;i&gt;T&lt;/i&gt;&lt;sub&gt;3&lt;/sub&gt;)</td>
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</tbody>
</table>
**Thyrotropin (thyroid-stimulating hormone [TSH])**

Thyrotropin; used to test for hypothyroidism (primarily in dogs)

10-unit vial

Dog: Collect baseline sample, followed by 0.1 unit/kg IV (maximum dose is 5 U); collect post-TSH sample at 6 hr
Cat: Collect baseline sample, followed by 2.5 units/cat IM; collect a post-TSH sample 6 hr later

**Ticarcillin**

Ticar, Ticillin

Antibacterial

6 g/50 mL vial; vials containing 1, 3, 6, 20, and 30 g

33-50 mg/kg IV, IM q4-6h

**Ticarcillin + clavulanate**

Timentin

Antibacterial

3-g vial for injection

33-50 mg/kg IV, IM q4-6h

**Tiletamine + zolazepam**

Telazol, Zoletil

General anesthetic; indicated for restraint and minor procedures of short duration in healthy dogs and cats

Sterile vial to which 5 mL of sterile water is added; provides the equivalent of 50 mg of tiletamine per milliliter

*Caution:* Limited shelf life after reconstitution

Dosage is based on combined milligrams of each drug. Administer by deep intramuscular injection.

Dog: 6.6-10 mg/kg IM (restraint); 10-13 mg/kg IM deep (minor surgical procedures)

Do not exceed 26.4 mg/kg IM total dose.

Cat: 9.7-11.9 mg/kg IM (restraint), 10.6-12.5 mg/kg IM (minor surgical procedures), 14.3-15.8 mg/kg IM (anesthesia)

Do not exceed 72 mg/kg IM total dose.

**Tobramycin**

Nebcin

Antibacterial

40 mg/mL injection

2-4 mg/kg IV, IM, SQ q8h

§ **Tocainide**

Tonocard

Oral antiarrhythmic; used to manage patients with ventricular arrhythmias

400- and 600-mg tablets

Dog: 15-20 mg/kg PO q8h

Cat: Dose not established

**Tolazoline**

Tolazine

α-Adrenergic blocker; reversal agent for xylazine

100 mg/mL in 100-mL multidose vials

4 mg/kg slowly IV (approx 1 mL/sec)

**Triamcinolone**

Vetalog, Trimtabs, Aristocort, generic

Glucocorticoid; antiinflammatory (not generally used in the treatment of immune-mediated disease)

Veterinary (Vetalog): 0.5- and 1.5-mg tablets

Human form: 1-, 2-, 4-, 8-, and 16-mg tablets; 10 mg/mL injection

Antiinflammatory: 0.05-0.11 mg/kg two to three times daily, within 2 weeks reduce dose to 0.028-0.055 mg/kg/day (however, manufacturer recommends doses of 0.11-0.22 mg/kg/day)

Listings preceded by § are for rapid reference and denote drug or dose used in the emergency or critical care setting.

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<tbody>
<tr>
<td>Triamcinolone acetonide</td>
<td>Vetalog</td>
<td>Glucocorticoid; antiinflammatory (not generally used in the treatment of immune-mediated disease)</td>
<td>2 and 6 mg/mL suspension injection; 0.5- and 1.5-mg tablets</td>
<td>0.1-0.2 mg/kg IM, SQ; repeat in 7-10 days Intralesional: 1.2-1.8 mg, or 1 mg for every cm diameter of tumor q2wk</td>
</tr>
<tr>
<td>Triamterene</td>
<td>Dyrenium</td>
<td>Diuretic; K-sparing diuretic used as an alternative to spironolactone</td>
<td>50- and 100-mg capsules</td>
<td>1-2 mg/kg PO q12h</td>
</tr>
<tr>
<td>Trientine hydrochloride</td>
<td>Syprine</td>
<td>Oral copper chelating agent; copper-associated hepatopathy; indicated in dogs that cannot tolerate penicillamine</td>
<td>250-mg capsules</td>
<td>Dog: 10-15 mg/kg PO q12h</td>
</tr>
<tr>
<td>Trifluoperazine</td>
<td>Stelazine</td>
<td>Phenothiazine; antiemetic</td>
<td>10 mg/mL oral solution; also as 1-, 2-, 5-, and 10-mg tablets; 2.0 mg/mL injection</td>
<td>0.03 mg/kg IM q12h</td>
</tr>
<tr>
<td>Triflupromazine</td>
<td>Vesprin</td>
<td>Phenothiazine; antiemetic</td>
<td>10 and 20 mg/mL injection</td>
<td>0.1-0.3 mg/kg IM, PO q8-12h</td>
</tr>
<tr>
<td>Triiodothyronine</td>
<td>See Liothyronine ($T_3$)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triolostane</td>
<td>Vetoryl</td>
<td>Oral treatment of canine hyperadrenocorticism (pituitary-dependent and adrenal tumor)</td>
<td>10-, 30-, 60-mg capsules (available in the United States)</td>
<td>3.9 to 9.2 mg/kg/day PO Commonly used dose in dogs is 6.1 mg/kg/day PO Dose adjustments are made on the basis of routine cortisol testing.</td>
</tr>
<tr>
<td>Trimeprazine tartrate with prednisolone</td>
<td>Temaril-P</td>
<td>Phenothiazine antihistamine + glucocorticoid combination; antitussive and antipruritic; not generally recommended today</td>
<td>5 mg trimeprazine + 2-mg prednisolone (combined) tablet</td>
<td>Dog: See manufacturer’s recommendations regarding indications and dose.</td>
</tr>
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</tr>
<tr>
<td>Trimethoprim + sulfonamide (sulfadiazine or sulfamethoxazole)</td>
<td>Tribrissen, others</td>
<td>Antibacterial</td>
<td>30-, 120-, 240-, 480-, and 960-mg tablets</td>
<td>15 mg/kg PO q12h, or 30 mg/kg PO q12-24h For Toxoplasma: 30 mg/kg PO q12h</td>
</tr>
<tr>
<td>TSH (thyroid-stimulating hormone)</td>
<td>Tribrissen, others</td>
<td>Antibacterial</td>
<td>Available as soluble powder with 2.2 g tylosin per teaspoon (tablets available for dogs in Canada)</td>
<td>Dog and cat: 7-15 mg/kg PO q12-24h Dog (for colitis): 11 mg/kg q8h with food</td>
</tr>
<tr>
<td>Tylosin tartrate</td>
<td>Tylocine, Tylan</td>
<td>Antibacterial; has antiinflammatory effects in the bowel and is sometimes used to treat inflammatory bowel disease and chronic colitis</td>
<td>Available as soluble powder with 2.2 g tylosin per teaspoon (tablets available for dogs in Canada)</td>
<td>Dog and cat: 7-15 mg/kg PO q12-24h Dog (for colitis): 11 mg/kg q8h with food</td>
</tr>
<tr>
<td>Ursodiol (ursodeoxycholic acid)</td>
<td>Actigall</td>
<td>Bile acid; adjunctive therapy in patients with chronic liver disease</td>
<td>300-mg capsule</td>
<td>10-15 mg/kg PO q24h</td>
</tr>
<tr>
<td>Valproic acid</td>
<td>Depakene (valproic acid), Depakote (divalproex)</td>
<td>Anticonvulsant; uncommonly used alternative to conventional anticonvulsant therapy</td>
<td>Depakote: 125-, 250-, and 500-mg tablets Depakene: 250-mg capsule; 50 mg/mL syrup</td>
<td>Dog: 60-200 mg/kg PO q8h; or divalproex 25-105 mg/kg/day PO when administered with phenobarbital Cat: Do not use</td>
</tr>
</tbody>
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</thead>
</table>
| Vancomycin                  | Vancocin; Vancoled | Antibacterial                                       | Vials for injection (0.5 to 10 g)                | Dog: 15 mg/kg q6-8h IV by CRI  
Cat: 12-15 mg/kg q8h IV by CRI  
For diagnostic purposes:  
Dog: Test protocol: administer 2 mcg IM to dogs < 15 kg body weight; administer 4 mcg IM to dogs > 15 kg  
Note: Diabetes insipidus test protocol entails patient preparation in advance of administration of vasopressin. |
| Vasopressin (ADH)           | Pitressin         | Hormone; diagnostic test agent to distinguish central diabetes insipidus from nephrogenic diabetes insipidus (not routinely recommended); see also Desmopressin acetate for treatment of diabetes insipidus. | 20 (pressor) units/mL in 0.5-, 1.0-, and 10-mL vials (aqueous only) and 1-mL ampules |                                                                                                                                                      |
| § Verapamil                 | Calan, Isoptin    | Calcium channel blocker; supraventricular tachycardia and hypertension | 40-, 80-, and 120-mg tablets; 2.5 mg/mL injection | Dog: 0.05 mg/kg, IV slowly (can repeat every 5 min) to a maximum cumulative dose of 0.15-0.2 mg/kg  
For hypertension: 1-5 mg/kg PO q8h  
Cat: 0.025 mg/kg IV slowly (can repeat every 5 minutes) to a maximum cumulative dose of 0.15-0.2 mg/kg  
Note: Diabetes insipidus test protocol entails patient preparation in advance of administration of vasopressin. |
| Vinblastine                 | Velban            | Vinca alkaloid, antineoplastic                      | 1 mg/mL injection                                | 2 mg/m² IV (slow infusion) q7-14 days                                                                                                                                                      |
| § Vincristine               | Oncovin, Vincasar, generic | Vinca alkaloid, antineoplastic; also for the treatment of thrombocytopenia | 1 mg/mL injection                                | Antitumor: 0.5-0.75 mg/m² IV q7-14 days (q7 days in cats, depending on protocol); for thrombocytopenia: 0.02 mg/kg IV, once weekly (alternatively, 0.5-0.7 mg/m² as an infusion over 4-6 hr) each week |
| Viokase                     | See Pancreatic Enzymes |                                                     |                                                  |                                                                                                                                                      |
| Vitamin A (retinoids)       | Aquasol A         | Vitamin; nutritional supplementation                | Oral solution: 5000 units per 0.1 mL and 10,000-, 25,000-, and 50,000-unit tablets | 625-800 units/kg PO q24h                                                                                                                                                                      |
| Vitamin B<sub>1</sub>       | Thiamine                  | Vitamin; nutritional supplementation | 250 mcg/5 mL elixir; tablets of various sizes from 5 mg to 500 mg; 100 and 500 mg/mL injection | Dog: 10-100 mg/dog/day PO  
Cat: 5-30 mg/cat/day PO (up to maximum dose of 50 mg/cat/day) |
|---------------------------|---------------------------|--------------------------------------|-------------------------------------------------------------------------------------------------|-----------------------------------------------------------------|
| Vitamin B<sub>2</sub>     | Riboflavin                | Vitamin; nutritional supplementation  | Tablets of various sizes in increments of 10 to 250 mg; 100 mcg/mL injection                 | Dog: 10-20 mg/day PO  
Cat: 5-10 mg/day PO                                                  |
| Vitamin B<sub>12</sub>    | Cyanocobalamin            | Vitamin; nutritional supplementation | 100 mcg/mL injection                                                                            | Dog: 100-200 mcg/day PO  
Cat: 50-100 mcg/day PO                                                  |
| Vitamin C                 | Ascorbic acid             | Tablets of various sizes and injection |                                                                                                | 100-500 mg/day                                                   |
| Vitamin D analogue        | Dihydrotachysterol (DHT), Hytakerol | Vitamin; management of hypocalcemia associated with hypoparathyroidism or parathyroid gland surgery | 0.125-mg tablet; 0.5 mg/mL oral liquid                                                                | 0.01 mg/kg/day PO  
Acute treatment: 0.02 mg/kg initially; then 0.01-0.03 mg/kg PO q24-48h thereafter |
| Vitamin D<sub>2</sub>     | Ergocalciferol, Calciferol, Drisdol | Vitamin; management of hypocalcemia associated with hypoparathyroidism or parathyroid gland surgery | 400-unit tablet (OTC); 50,000-unit tablet (1.25 mg); 500,000 units/mL (12.5 mg/mL) injection | 4000 to 6000 units/kg/day PO (initial); 1000 to 2000 units/kg/day PO (maintenance) |
| Vitamin D<sub>3</sub>     | 1-25, dihydroxy-vitamin D<sub>3</sub> | Vitamin, also considered a hormone; management of hypocalcemia associated with hypoparathyroidism or parathyroid gland surgery; also used to supplement hypocalcemia of chronic renal failure | See *Vitamin D analogue*                                                                 | Hypocalcemia: 0.030-0.06 mcg/kg PO once daily  
Chronic renal failure: 0.025 mcg/kg PO once daily                      |

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<tbody>
<tr>
<td>Vitamin E (may be combined with selenium)</td>
<td>alpha-tocopherol, Aquasol E, generic</td>
<td>Vitamin; nutritional supplementation and adjunctive therapy in patients with chronic liver disease; may be combined with selenium as adjunctive therapy for immune-mediated skin disease in dogs; efficacy in management of arthritic dogs is questionable</td>
<td>Wide variety of capsules, tablets, oral solution available (e.g., 1000 units per capsule)</td>
<td>See manufacturer’s recommendations for treatment indications and dose</td>
</tr>
<tr>
<td>§ Vitamin K&lt;sub&gt;1&lt;/sub&gt;</td>
<td>Phytonadione, phytomenadione, Aqua-MEPHYTON (injection), Mephyton (tablets), Veta-K1 (capsules)</td>
<td>Antidote; anticoagulant, rodenticide toxicosis and in any disorder affecting formation of vitamin K–dependent coagulation factors</td>
<td>2 and 10 mg/mL injection; 5-mg tablet (Mephyton); 25-mg capsule (Veta-K1)</td>
<td>Rodenticide toxicosis: 2.5-5.0 mg/kg/day PO (preferred) for up to 6 weeks depending on the agent ingested. Acute intoxication: 5 mg/kg SQ in multiple locations with 25-gauge needle</td>
</tr>
<tr>
<td>Warfarin</td>
<td>Coumadin and generic</td>
<td>Anticoagulant; adjunctive treatment for and prevention of thromboemboli</td>
<td>1-, 2-, 2.5-, 4-, 5-, 7.5- and 10-mg tablets</td>
<td>Dog: 0.22 mg/kg PO q12h to prolong PT by 1.25 to 1.5 times normal Pulmonary thromboemboli: 0.2 mg/kg PO daily to prolong PT by 1.5 to 2.5 times normal Cat: Chronic treatment: 0.1-0.2 mg/kg PO once daily to prolong PT by 2 to 2.5 times normal Aortic embolus: 0.06-0.1 mg/kg PO once daily</td>
</tr>
<tr>
<td>Drug</td>
<td>Use</td>
<td>Dose/Injection</td>
<td>Comments</td>
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</tr>
<tr>
<td>Xylazine</td>
<td>α₂-Adrenergic agonist; sedative and analgesic (sometimes used as an emetic in cats)</td>
<td>20 and 100 mg/mL injection</td>
<td>Dog and cat: 1.1 mg/kg IV; or 1.1 to 2.2 mg/kg IM or SQ</td>
<td></td>
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<tr>
<td></td>
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<td></td>
<td>Cat (to induce emesis): 0.4-0.5 mg/kg IV</td>
<td></td>
</tr>
<tr>
<td>Yohimbine</td>
<td>α₂-Adrenergic antagonist; reverses xylazine (and possibly amitraz)</td>
<td>2 mg/mL injection in 20-mL vials</td>
<td>0.11 mg/kg IV slowly</td>
<td></td>
</tr>
<tr>
<td>Zidovudine (AZT)</td>
<td>Antiretroviral agent; adjunctive treatment of FeLV- and FIV-positive cats</td>
<td>300-mg tablets; 100-mg capsules; 10 mg/mL syrup</td>
<td>Cat: 5-15 mg/kg PO q12h; or 5 mg/kg PO q8h for 5 wk and then rest for 4 wk</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>10 mg/mL injection</td>
<td>Caution: Significant bone marrow suppression (usually reversible with cessation of therapy) is expected; monitor CBC during therapy.</td>
<td></td>
</tr>
</tbody>
</table>

**Zolazepam**

See Tiletamine + zolazepam

**Abbreviations:**

ACE, Angiotensin-converting enzyme; ACTH, adrenocorticotropic hormone; ADH, antidiuretic hormone; CBC, complete blood count; CMI, cell-mediated immunity; CNS, central nervous system; CRI, constant-rate infusion; CSF, cerebrospinal fluid; D5W, dextrose 5% in water; DI, diabetes insipidus; DIC, disseminated intravascular coagulation; FeLV, feline leukemia virus; FIV, feline immunodeficiency virus; GA BA, γ-aminobutyric acid; GnRH, gonadotropin-releasing hormone; H₂, histamine; IM, intramuscularly; IMHA, immune-mediated hemolytic anemia; IP, intraperitoneally; IV, intravenously; LH, luteinizing hormone; LHRH, luteinizing-hormone-releasing hormone; MOA, monoamine oxidase; NSAID, nonsteroidal antiinflammatory drug; OTC, over the counter (prescription not required); PO, orally; PT, prothrombin time; SQ, subcutaneously; SSRI, selective serotonin reuptake inhibitor; USP, United States Pharmacopeia.

*Doses listed in this table are based on best available evidence at the time of table preparation; although considerable effort has been made to verify all doses listed, it is prudent to verify treatment protocols and drug dosages whenever using a product for the first time. Adverse effects may be possible from virtually any of the drugs listed in this table. High-risk warnings and precautionary statements are listed. Veterinarians using this table are encouraged to check current literature, product label, and manufacturer's disclosure for information regarding reported changes in efficacy or safety as well as any new treatment contraindications not identified at the time of preparation of these tables. When dose listed does not stipulate dog or cat, drug may be administered to both dogs and cats at the dose listed. Listings preceded by § are for rapid reference and denote drug or dose used in the emergency or critical care setting.*
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