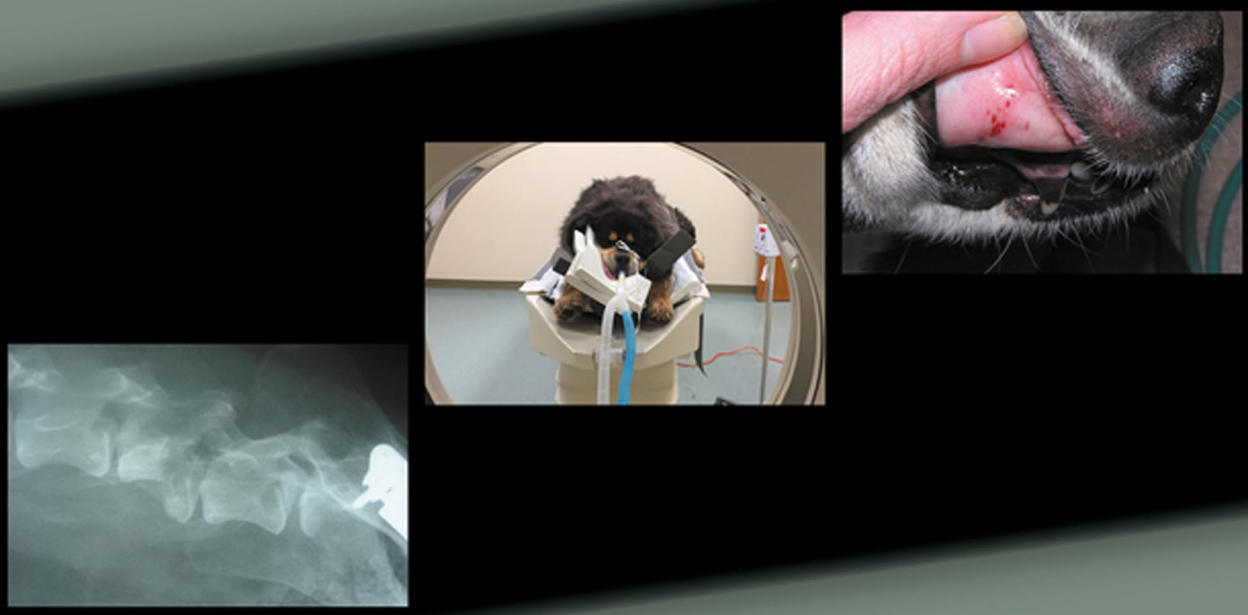
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Blackwell's Five-Minute Veterinary Consult
Clinical Companion

Small Animal Emergency and Critical Care

Second Edition



Elisa M. Mazzaferro

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WILEY Blackwell

FIVE MINUTE VETERINARY CONSULT

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Preface



Emergency and critical care medicine remains exciting, challenging, and often fast-paced. Clinicians must think on their feet to consider lists of differential diagnoses, recommend appropriate diagnostic tests, and implement therapies to save a patient's life. The Veterinary Emergency and Critical Care Society and the American College of Veterinary Emergency and Critical Care have worked together to raise the standard of care in veterinary emergency and critical care medicine for more than 25 years. Utilization of diagnostic and monitoring tools and integration of the most recent advances in patient monitoring and medicine allow us to constantly improve the probability of success in our hospitals. Together, the veterinary team of veterinarian and nurses, the pet owner, and the animal patient all benefit from constant learning and developments in the field of veterinary emergency and critical care medicine.

The second edition of this textbook offers the most up-to-date information on common clinical signs and diseases seen when an animal presents on emergency. Arranged in alphabetic order, the organization of topics allows rapid access with succinct bullet points to aid in making a rapid diagnosis and implementation of therapy in a busy emergency or daytime small animal hospital. This second edition has been expanded to include more than 15 new topics, including an extended list of the common small animal toxicities. This edition continues to provide color images and flowcharts to add to understanding the text material.

As before, I have been privileged to work with more than 50 board-certified experts who have written or updated the chapters in their respective areas of expertise. I have fortunately had to replace the first edition of this text in my emergency room more than once, as the pages became tattered from use. A tattered book is a well-used book. My hope is that your copy will be well used and loved, and help to save many a patient's life.

Most sincerely,
Elisa M. Mazzaferro

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About the Companion Website

This book is accompanied by a companion website:

www.fiveminutevet.com/emergency

The website includes:

- Client education handouts

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chapter 1

Acetaminophen Toxicity





DEFINITION/OVERVIEW

- Acetaminophen (N-acetyl-p-aminophenol) is a common OTC or prescription medication with antipyretic and analgesic properties. It is commonly known as "Tylenol," APAP, or paracetamol.
- Acetaminophen does not have antiinflammatory properties and is not considered an NSAID.
- Acetaminophen can result in accidental toxicosis in dogs, cats, and ferrets. Ingestion may be accidental or by well-intentioned pet owners who are unaware of the toxic dose or safety profile of this common medication.
- In dogs, clinical signs of toxicosis are seen at >100–150 mg/kg, while in cats and ferrets, toxic doses can be seen at 10–50 mg/kg.
- Acetaminophen toxicosis results in methemoglobinemia (cats, less commonly dogs) or hepatotoxicity (dogs, less commonly cats).
- Clinical signs of toxicosis typically include malaise, anorexia, paw or facial swelling, vomiting, respiratory distress, brown mucous membranes, and icterus.
- Unlike the majority of toxicants, acetaminophen toxicosis *does* have an antidote N-acetylcysteine (NAC), making the prognosis fair to excellent with supportive care.



ETIOLOGY/PATHOPHYSIOLOGY

- Acetaminophen is a COX-3 inhibitor.
- Acetaminophen is metabolized through two pathways: the major pathway creates inactive metabolites through conjugation to inactive glucuronide and sulfate metabolites. The other pathway metabolizes acetaminophen by the cytochrome p450 enzyme pathway to the toxic metabolite, N-acetyl-para-benzoquinoneimine (NAPQI). Toxicosis occurs when the metabolic pathways for glucuronidation and sulfation are depleted; this results in toxic metabolites building up and secondary oxidative injury to RBCs and hepatic proteins.
- Acetaminophen is rapidly absorbed from the stomach and GIT; peak blood levels are reached within 30–60 minutes.

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Systems Affected

- Gastrointestinal: vague GI signs may be seen early in acetaminophen toxicosis; more severe signs may be seen with advanced hepatic failure.
- Skin/exocrine: facial or paw swelling may be seen in both cats and dogs via an unknown mechanism; icterus with hepatotoxicity.
- Hemic/lymphatic/immune: oxidative injury to RBC and Hb molecules following glutathione depletion, resulting in MetHb and Heinz body anemia.
- Respiratory: respiratory distress secondary to the presence of MetHb and the inability to carry oxygen.
- Cardiovascular: shock secondary to anemic hypoxia.
- Hepatobiliary: hepatocellular injury and hepatic necrosis due to NAPQI.
- Nervous: hepatic encephalopathy secondary to hepatotoxicity.
- Ophthalmic: KCS has been reported with acetaminophen in dogs, even at subtoxic doses.
- Renal/urologic: rarely, large doses can result in renal tubular necrosis; this has only been reported in humans.



SIGNALMENT/HISTORY

Risk Factors

- Puppies and younger dogs appear to be overrepresented with poisoning due to their curious nature.
- Neonates, geriatric patients, or those with underlying hepatic disease may be more at risk for acetaminophen toxicosis due to abnormal or delayed metabolism.
- Cats are more susceptible to acetaminophen toxicosis, as they lack sufficient glucuronyl transferase to metabolize acetaminophen and have limited sulfate-binding capacity. Cats are also more susceptible as their hemoglobin contains eight sulfhydryl groups compared to four in other species; this makes feline RBC more prone to oxidative injury and results in MetHb developing earlier into toxicosis.
- Chronic administration.

Historical Findings

- Evidence of a tampered or chewed container or prescription bottle.
- Owner administration.
- Clinical signs consistent with acetaminophen toxicosis.



CLINICAL FEATURES

- Gastrointestinal.
  - Anorexia
  - Hypersalivation
  - Vomiting
  - Diarrhea

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  - Melena
  - Abdominal pain
- Miscellaneous.
  - Facial or paw swelling
  - Generalized malaise
  - Hypothermia
- Hemic/lymphatic/immune.
  - Brown or cyanotic mucous membranes
  - Hemoglobinemia
  - Hemoglobinuria
- Respiratory.
  - Tachypnea progressing to dyspnea
  - Brown-colored mucous membranes
  - Increased respiratory rate and effort
- Cardiovascular.
  - Tachycardia
  - Hypotension
  - Cardiovascular collapse
- Hepatobiliary.
  - Malaise
  - Icterus
  - Bruising
  - Melena
- Nervous.
  - Dull mentation
  - Generalized malaise
  - Ataxia
  - Head pressing, star gazing, or abnormal mentation
  - Tremors
  - Seizures
  - Coma
- Ophthalmic.
  - Mucopurulent discharge
  - Squinting
  - Rubbing at the eyes
  - Conjunctivitis



DIFFERENTIAL DIAGNOSIS

- In patients presenting with increased liver enzymes or evidence suggestive of hepatopathy, other hepatotoxicants (e.g., sago palm, *Amanita* mushroom, xylitol, blue-green algae, aflatoxins, etc.), metabolic causes (e.g., cholangiohepatitis, extrahepatic biliary duct obstruction, pancreatitis, etc.), neoplasia, or infectious (e.g., *Leptospira*, etc.) causes should be ruled out.

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- In patients presenting with anemia, other differential diagnoses include toxicants (such as zinc, mothballs (e.g., naphthalene), *Allium* spp. (e.g., onions, garlic, etc.), local anesthetics (e.g., benzocaine, etc.)), metabolic causes (e.g., IMHA), infectious causes (e.g., *Mycoplasma felis,* etc.), neoplasia, etc.



DIAGNOSTICS

- If an ingestion approaching a toxic dose has occurred, baseline blood work should include a CBC, biochemistry panel, and blood smear (to look for the presence of Heinz bodies) at the time of admission.
  - Common clinicopathologic findings seen with acetaminophen toxicosis include Heinz bodies, anemia, increased liver enzymes (typically seen 24–36 hours post ingestion), hyperbilirubinemia, hemoglobinemia, hemoglobinuria.
  - An extra drop of blood should be placed on a white paper towel to look for a dark or brown appearance; the presence of "dark" (deoxygenated) blood is suggestive of MetHb.
- Blood gas analysis:
  - May reveal the presence of a metabolic acidosis
  - In a patient with severe respiratory distress, an ABG can be performed to help rule out acetaminophen toxicosis; the presence of a normal PaO_2 with a low oxygen saturation is highly suspicious of toxicosis. Cooximetry can be used to measure MetHb, but is not readily available in veterinary medicine.
- Serum acetaminophen levels can be performed at a human hospital; levels are typically the most elevated 1–3 hours post ingestion. However, toxic levels in dogs and cats are not well established, and likely can only be used to confirm ingestion.
- In hospitalized patients, a daily PCV/TS and hepatic panel should be performed every 24 hours. If liver enzymes are normal after 48 hours and the patient is no longer symptomatic, the patient can be discharged after this time.
- In patients suspected of having hepatic injury (e.g., increased liver enzymes, hypoglycemic, hypocholesterolemia, etc.), a PT/PTT should be performed.
- Abdominal ultrasound + liver aspirate may be necessary in some cases to rule out other differential diagnoses.



THERAPEUTICS

- The mainstay therapy for acetaminophen toxicosis is administration of activated charcoal, oxygen therapy, intravenous (IV) fluid therapy, antidotal therapy (e.g., NAC), and hepatoprotectants (e.g., SAMe).
- Decontamination.
  - Due to the rapid absorption of acetaminophen from the stomach and GIT, emesis induction is *not* recommended. Rather, immediate administration of one dose of activated charcoal (1–5 g/kg, PO) with a cathartic (e.g., sorbitol) is warranted provided the patient is asymptomatic.

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  - As acetaminophen undergoes some enterohepatic recirculation, multiple doses of activated charcoal (with the additional doses being free of a cathartic) should ideally be administered, provided the patient is asymptomatic and parenteral administration of NAC is available. If NAC is only available orally, only one dose of charcoal should be administered, with antidotal therapy prioritized after 2 hours of administration of charcoal.
- Oxygen therapy.
  - In tachypneic or patients with severe respiratory distress, immediate oxygen therapy is warranted to help treat anemic hypoxemia.
- Fluid therapy.
  - The use of a balanced, isotonic crystalloid is warranted to help hydrate and perfuse the patient.
- Antidotal therapy.
  - The use of NAC is warranted to help act as a glutathione source and to limit formation of the toxic metabolite NAPQI. This should be implemented as soon as possible.
- Blood products.
  - In cats with severe respiratory signs, transfusion of PRBC or whole blood may be warranted, even with a normal PCV. As MetHb is unable to carry oxygen appropriately, treatment should be aimed at antidotal therapy and oxygen support; if, however, the patient fails to respond clinically, administration of blood products may be necessary to deliver hemoglobin to treat anemic hypoxia.
  - In severe cases of acute hepatic failure secondary to acetaminophen, administration of FP or FFP (10–20 mL/kg, IV) may be necessary to provide vitamin K_1-dependent coagulation factors II, VII, IX, X.
- Hepatoprotectants.
  - The use of SAMe is warranted to help reduce oxidative injury, and to act as a benign antioxidant and glutathione source.
- Miscellaneous.
  - Coagulopathic patients (secondary to liver failure) should be treated with vitamin K_1.
  - Vitamin C (ascorbic acid) can be used as a benign antioxidant, but in this author's experience does not appear to be clinically beneficial.
  - Methylene blue can be used to treat dogs with severe MetHb, and acts as an electron donor to reduce MetHb to Hb. This should not be used in cats, as it can cause Heinz body anemia.
- Gastric protectants.
  - The routine use of H_2 blockers, such as Cimetidine, is *no longer warranted or recommended* to prevent p450 enzyme interference with acetaminophen metabolism.

Drugs of Choice

- N-acetylcysteine (NAC): 140–280 mg/kg IV or PO loading dose, followed by 70 mg/kg IV or PO q6 hours × 48 hours or until clinical signs resolve.
- SAMe: 18 mg/kg PO q24 hours × 14–30 days on an empty stomach.

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- Antiemetics.
  - Maropitant (1 mg/kg SQ q24 hours; extralabel use in cats and via IV route); if evidence of hepatic failure is present, alternative antiemetics should be used.
  - Ondansetron: 0.1–0.5 mg/kg IV q12 hours.
  - Dolasetron: 0.6 mg/kg IV q24 hours.
- Vitamin K_1: 1 mg/kg PO or SQ q12–24 hours.
- Vitamin C: 30 mg/kg PO or SQ q6 hours.
- Methylene blue: 1.5 mg/kg IV 1–2×, slow (dogs only).
- KCS treatment.
  - Topical artificial tears OU, if indicated.
  - Topical cyclosporine ointment OU, if indicated.

Precautions/Interactions

- Acetaminophen is commonly combined with other ingredients such opioids or opioid-like drugs (e.g., codeine, hydrocodone, oxycodone, propoxyphene, pentazocine, tramadol, etc.), decongestants (e.g., pseudoephedrine), antihistamines (e.g., chlorpheniramine, diphenhydramine), antitussives (e.g., dextromethorphan), NSAIDS (e.g., aspirin), and stimulants (e.g., caffeine). Dual toxicosis and variable clinical signs may occur as a result.

Alternative Drugs

- Ideally, a parenteral source of NAC should be used to allow additional GIT decontamination (with multiple doses of charcoal). While extralabel, the use of inhalational NAC (Mucomyst®) can be administered IV, using sterile technique and a 0.22 micron filter. Please see a drug reference book for appropriate dosing and dilution.

COMMENTS

Client Education/Prevention/Avoidance

- Owners should be educated to appropriately pet-proof the house. Education on crate training is imperative.
- Pet owners should be educated to never give an OTC or prescription medication to their pet without consulting their veterinarian first.
- Owners and veterinary professionals should be educated to call veterinary-specific poison control centers for consultation with a veterinary toxicologist for life-saving advice as needed.

Possible Complications

- In cases where acute hepatic injury has occurred, pet owners should be educated that chronic hepatopathy may develop.

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- While acetaminophen (typically in combination with hydrocodone or oxycodone) can be used therapeutically in dogs as an alternative analgesic drug, therapeutic use in cats is *never* recommended. In dogs, even with therapeutic doses, potential adverse effects can be seen (e.g., KCS).

Expected Course and Prognosis

- Overall, the prognosis for acetaminophen toxicosis is fair to good, as the antidote NAC is readily available. However, financial limitations may preclude treatment, which often requires hospitalization for 24–72 hours.
- If clinical signs of acute hepatic failure or hepatic encephalopathy are present, the prognosis for survival is much poorer.

Synonyms

- Tylenol
- Paracetamol
- APAP
- Percoset
- Vicodin
- Any medication with the term "headache" listed
- Any medication with the term "cold and sinus" listed

Abbreviations

APAP: N-acetyl-p-aminophenol
CBC: complete blood count
COX: cyclooxygenase
FFP: fresh frozen plasma
FP: frozen plasma
GI: gastrointestinal
GIT: gastrointestinal tract
Hb: hemoglobin
IMHA: immune-mediated hemolytic anemia
IV: intravenous
MetHb: methemoglobinemia
NAC: N-acetylcysteine
NAPQI: N-acetyl-para-benzoquinoneimine
NSAID: nonsteroidal antiinflammatory drug
OTC: over the counter
OU: each eye (oculus uterque)
PCV: packed cell volume
PO: per os
PRBC: packed red blood cells

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PT: prothrombin time
PTT: partial thromboplastin time
SAMe: S-adenosyl-methionine
SQ: subcutaneous

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chapter 2

Acute Respiratory Distress Syndrome



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DEFINITION/OVERVIEW

- ARDS is a severe inflammatory disorder of the lung that can cause respiratory failure in dogs and cats.
- It is a form of noncardiogenic pulmonary edema caused by lung inflammation, cellular infiltration, and capillary leak.
- ALI is a milder form of inflammatory injury to the lungs but can progress to ARDS.



ETIOLOGY/PATHOPHYSIOLOGY

- ALI and ARDS can occur from direct pulmonary insult, or, more commonly in critically ill patients, by a generalized inflammatory response such as SIRS or sepsis.
- In SIRS or sepsis, activation of tumor necrosis factor and proinflammatory interleukins initiates inflammatory mediators and activation of neutrophils and macrophages. ARDS is a local pulmonary manifestation of SIRS.
- Pancreatitis can cause lung injury secondary to vascular endothelial damage by activated proteases and associated inflammation.
- Local pulmonary injury can trigger an inflammatory response that can become generalized within the lung parenchyma, with production of proinflammatory cytokines by inflammatory cells, lung epithelial cells, and fibroblasts.
- Clinical and histopathologic findings are similar for all etiologies.
- There are three overlapping stages. The initial exudative stage begins as a diffuse vascular leak syndrome with infiltration of erythrocytes, neutrophils, and macrophages and effusion of protein-rich fluid into the alveoli, resulting in progressive pulmonary edema and hemorrhage.
- Chemotaxis results in accumulation of inflammatory cells, particularly neutrophils, contributing to ongoing lung injury.
- Surfactant synthesis is impaired and hyaline membranes form within alveoli (organization of protein-rich fluid and cellular debris), resulting in collapse and atelectasis of alveoli.

Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Emergency and Critical Care, Second Edition. Edited by Elisa M. Mazzaferro.
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Companion website: www.fiveminutevet.com/emergency

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- The proliferative phase follows, with proliferation of type II pneumocytes.
- The fibrotic phase is characterized by interstitial fibrosis as the lung attempts to repair the damaged tissue, with inflammatory changes varying in severity and often unevenly distributed in the lung.
- In more severely affected animals, the inflammation is severe and leads to severe hypoxia and death of the patient.

Systems Affected

- Respiratory
- Cardiovascular
- Hemic/lymphatic/immune
- Renal/urologic



SIGNALMENT/HISTORY

Risk Factors/Causes

- Multiple etiologies; it may occur secondary to direct lung injury or systemic inflammation.
- Occasionally, predisposing factors cannot be identified.
- No breed, age, or sex predispositions.

Systemic Disorders

- SIRS
- Sepsis
- Shock
- Organ torsion (gastric, splenic)
- Canine parvoviral enteritis
- Pancreatitis
- Severe trauma
- Massive transfusions (reported in humans)
- Drugs and toxins

Primary Respiratory Disorders

- Aspiration or bacterial pneumonia
- Pulmonary contusions
- Smoke inhalation
- Noncardiogenic pulmonary edema secondary to strangulation, choking, or seizures
- Lung lobe torsion
- Near drowning

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Historical Findings

- Most commonly occurs in patients in the intensive care unit with other underlying diseases, but may affect other patients, causing presentation with an acute history of severe respiratory distress (increased respiratory rate and effort).
- Earliest signs often include severe respiratory distress and hypoxia.
- Usually no history of coughing, but occasionally low-grade productive cough.
- Gas exchange may be severely impaired.
- Signs may develop within hours or up to 4 days after inciting event.



CLINICAL FEATURES

- Severe respiratory distress, cyanosis.
- Auscultation – harsh lung sounds can rapidly progress to crackles.
- Dogs may cough up pink foam.
- If intubated, sanguineous fluid may drain out of the endotracheal tube in both dogs and cats.
- Often are tachycardic from poor oxygen delivery to tissues secondary to severe hypoxemia.
- Pulmonary edema in an animal with a predisposing cause of inflammatory response without evidence of heart failure.
- Evidence of other underlying or systemic disease may be present.



DIFFERENTIAL DIAGNOSIS

- Cardiogenic pulmonary edema
- Volume overload
- Pulmonary thromboembolism
- Bacterial pneumonia
- Atelectasis
- Pulmonary hemorrhage
- Neoplasia



DIAGNOSTICS

Clinical Criteria

- Acute onset of respiratory distress (<72 hours).
- Presence of one or more known risk factors.
- Inefficient gas exchange.
- Evidence of pulmonary capillary leak without increased pulmonary capillary pressure.
- ± Evidence of pulmonary inflammation.

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Thoracic Radiographs

- Early ALI – often have increased pulmonary interstitial and peribronchial markings.
- As ALI progresses to ARDS, diffuse bilateral pulmonary alveolar infiltrates develop throughout all lung fields, may be asymmetrical or patchy, and ventral lung lobes may be most severely affected (Fig. 2.1).



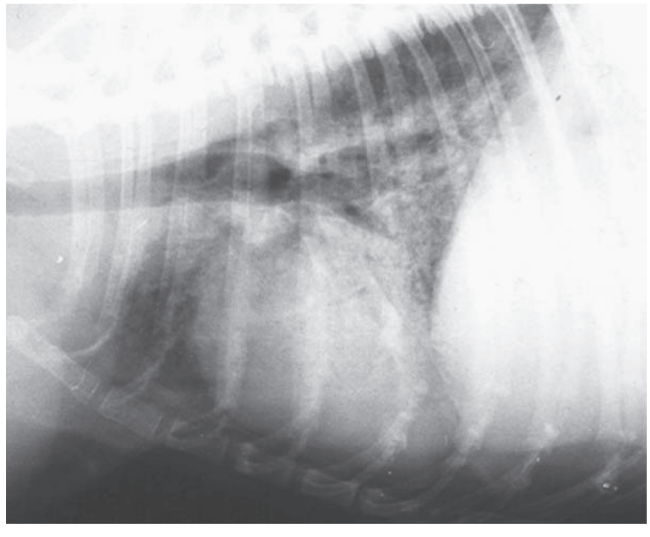


Fig. 2.1. Radiographs of a 6-year-old FS MIXB that presented with increased respiratory rate and effort. She was anesthetized for diagnostic testing, including these thoracic radiographs, which show a diffuse patchy alveolar pattern consistent with acute respiratory distress syndrome. Her oxygenation status continued to decline despite positive pressure ventilation, so she was euthanized. On necropsy, a diagnosis of severe subacute to chronic interstitial pneumonia with acute respiratory distress syndrome was made. Reprinted with permission from *The Veterinary ICU Book*, Teton NewMedia.

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- Small volume of pleural effusion may or may not be present.
- Heart and blood vessel size should be normal; otherwise left-sided congestive heart failure may be present rather than ARDS.

Arterial Blood Gases

- Arterial blood gas – severe hypoxia and usually hypocarbia as hypoxia begins to drive respiration and results in hyperventilation. If end-stage lung disease or respiratory muscle fatigue occurs, may see hypercarbia. Lactic acidosis may be present due to poor oxygen delivery and anaerobic tissue metabolism.
- PaO_2:FiO_2 ratios are also significantly low (reference range ~430–560 mmHg). Typically, PaO_2:FiO_2 ratio ≤300 is consistent with ALI, whereas PaO_2:FiO_2 ratio ≤200 is consistent with ARDS.

Complete Blood Count/Biochemical/Coagulation

- Leukopenia may be present due to sequestration of white blood cells in periphery and in lungs.
- Thrombocytopenia may be present due to platelet sequestration or consumption.
- Consumptive coagulopathy may be manifested by prolonged coagulation times and elevated fibrin degradation products or D-dimers.
- Chemistry panel usually has nonspecific changes, but hypoalbuminemia may occur due to underlying disease and exudative protein loss into the pulmonary edema fluid.

Additional Diagnostics

- Elevation of CVP or pulmonary capillary wedge pressure (>18 mmHg) may suggest congestive heart failure or fluid overload, not ALI or ARDS.
- Pulmonary function testing – poor lung compliance.
- Extremely high mean and peak airway pressures needed for PPV.
- Pulmonary hypertension may occur in severely affected patients due to obliteration of the pulmonary capillary bed.

Pathologic Findings

- Gross examination – heavy, stiff lungs.
- Histology – diffuse alveolar damage, presence of hyaline membranes, congestion, edema, neutrophil infiltration, hemorrhage, local thrombosis, and atelectasis. This is followed by type II pneumocyte proliferation (replacing dead type I pneumocytes), proliferation of fibroblasts, then finally collagen deposition in the alveolar, vascular, and interstitial beds (Fig. 2.2).

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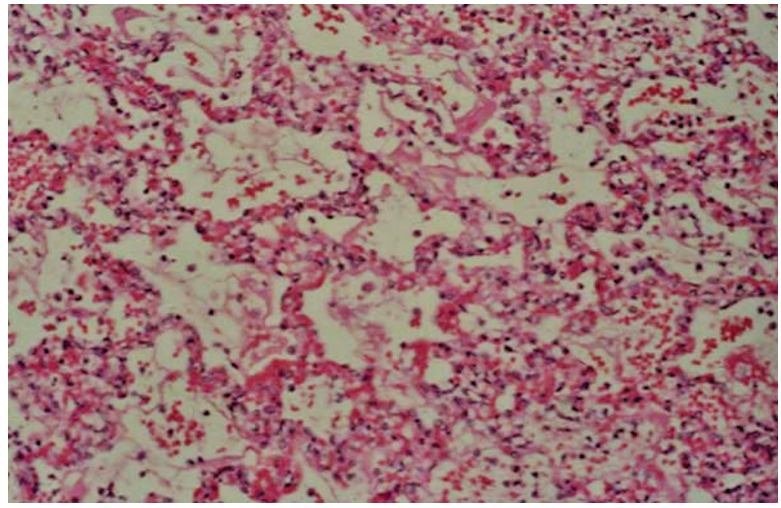


Fig. 2.2. Histopathology (20×) of the lung of a dog with acute respiratory distress syndrome, diagnosed on post-mortem examination. The capillaries are dilated and filled with red blood cells and the alveoli contain a mixture of red cells, neutrophils, macrophages, and strands of fibrin. Courtesy of Dr Tom van Winkle, Laboratory of Pathology, University of Pennsylvania. Reprinted with permission from *The Veterinary ICU Book*, Teton NewMedia.



THERAPEUTICS

- Oxygen supplementation – animals with mild ALI may respond to oxygen supplementation alone, but severe ARDS patients usually require PPV to achieve adequate gas exchange.
- Address the underlying cause of SIRS or primary lung injury to remove source of ongoing injury/prevent repeat injury, if possible.
- Carefully evaluate fluid therapy to prevent fluid overload and worsened pulmonary dysfunction. Measurement of CVP or pulmonary capillary wedge pressure to aid in fluid therapy to prevent overhydration but maintain euvolemia.
- If volume overload is present, judicious administration of diuretics such as furosemide.
- Colloid support if hypoproteinemic. Can include fresh frozen plasma (provides coagulation factors and acute phase proteins in addition to albumin), synthetic colloids, and 25% human albumin solutions.
- PPV with PEEP recruits alveoli and increases functional residual capacity, allowing ventilation at lower FiO_2 and preventing cyclical alveolar reopening and stretching with each breath. FiO_2 should be ≤0.6 to prevent oxygen toxicity and tidal volumes should be as low as possible (ideally 6–8 mL/kg) to prevent overdistension of relatively normal alveoli, shear stress, and progression of lung injury. Excessively high airway pressures (>30 cmH_2O) can cause worsening of lung permeability and also produce pneumothorax.

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Drugs of Choice

- Many treatments have been evaluated experimentally in animal models and clinically in humans with ARDS. These include corticosteroids, albumin solutions, nitric oxide, exogenous surfactant, beta-2 agonists, furosemide, N-acetylcysteine, pentoxifylline, and a variety of cyclooxygenase, thromboxane, and leukotriene inhibitors. Although some appeared promising in canine and feline models of ARDS, none has been shown to have an effect on morbidity or mortality in human clinical trials.
- Early antibiotics if appropriate for underlying disease.
- Diuretics if volume overload or cardiac disease is suspected, but will not be beneficial if pulmonary edema is due to ALI or ARDS alone. Careful if patient is hypovolemic; may worsen cardiovascular status.
- Supportive measures as required by patient: fluid therapy, pressors if indicated, and anesthesia to allow for PPV.



COMMENTS

Patient Monitoring

- Patients require intensive care monitoring with frequent arterial blood gas analysis, pulse oximetry, arterial blood pressure, urine output, temperature, ECG, thoracic radiographs, CBC, chemistry, and coagulation monitoring.

Prevention/Avoidance

- Aggressive therapy of underlying disease processes, treating any cardiovascular compromise.
- Prevention of aspiration pneumonia through careful use of analgesics and sedation and appropriate nursing care,

Possible Complications

- Respiratory failure and death.
- Progressive multiple organ dysfunction and failure (DIC, renal, gastrointestinal, and hepatic).

Expected Course and Prognosis

- Humans with ARDS have a mortality rate of 36–44% and often require mechanical ventilation for 4–6 weeks. Survival rates seem to be improving over the last 20 years, likely due to the use of protective lung ventilation and improved supportive care.
- Mortality in dogs and cats is even higher, and a grave prognosis should be given.

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Synonyms

- Shock lung
- Traumatic wet lung
- Adult hyaline membrane disease
- Capillary leak syndrome

Abbreviations

ALI: acute lung injury
ARDS: acute respiratory distress syndrome
CBC: complete blood count
CVP: central venous pressure
DIC: disseminated intravascular coagulation
ECG: electrocardiogram
PEEP: positive end-expiratory pressure
PPV: positive pressure ventilation
SIRS: systemic inflammatory response syndrome

Suggested Reading

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chapter 3

Anterior Uveitis





DEFINITION/OVERVIEW

- Uveitis is a general term for inflammation in any portion of the uveal tract, regardless of cause.
- The uveal tract is the highly vascular middle coat of the eye consisting of the iris, ciliary body, and choroid.
- Anterior uveitis is inflammation of the iris and ciliary body.
- Posterior uveitis is inflammation of the choroid, usually with concurrent inflammation of the retina (chorioretinitis) due to the close proximity of the two structures.



ETIOLOGY/PATHOPHYSIOLOGY

- Intraocular inflammation is initiated by local tissue injury (e.g., trauma, infectious agent, immune mediated, neoplasia).
- Bilateral anterior uveitis often indicates an underlying systemic disease process.
- Damaged tissue and microorganisms release tissue factors and inflammatory mediators that cause vasodilation and increased vascular permeability.
- The result is disruption of the blood–eye barrier, aqueous flare (increased protein in the anterior chamber), and inflammatory cell migration into the anterior chamber and anterior uveal tissue.
- Uveitis can be caused by trauma, can be secondary to cornea, lens or scleral disease, or can be caused by systemic or primary intraocular disease.

Dogs

- Systemic infectious causes – mycotic (blastomycosis, histoplasmosis, coccidioidomycosis, cryptococcosis); algal (protothecosis); rickettsial (*Ehrlichia* spp., *Rickettsia rickettsii*); bacterial (brucellosis, borreliosis, any bacterial septicemia); protozoal (*Toxoplasma gondii*,

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Leishmania); viral (infectious canine hepatitis); aberrant parasite migration (fly larvae, nematode larvae).
    - German shepherd dogs are predisposed to disseminated saprophytic fungal infection (*Aspergillus* spp., *Candida* spp., *Penicillium* spp., *Paecilomyces* spp.).
- Immune-mediated/presumed immune-mediated causes – exposure to lens proteins (cataract or lens capsule rupture); uveodermatologic syndrome; idiopathic; uveitis associated with uveal cysts; CAV-1 and CAV-2 vaccine reaction; scleritis.
- Neoplastic causes – primary intraocular or metastatic to eye.
    - Lymphosarcoma most commonly diagnosed.
- Metabolic causes – hyperlipidemia.
- Miscellaneous causes – systemic hypertension; hyperviscosity syndrome; lens luxation.
- Most often a cause for uveitis cannot be identified and a diagnosis of idiopathic is made.

Cats

- Systemic infectious causes – viral (FeLV, FIV, FIP, possibly FHV-1); protozoan (*Toxoplasma gondii*, *Leishmania*); bacterial (*Bartonella* spp., any cause of septicemia), fungal (blastomycosis, histoplasmosis, coccidioidomycosis, cryptococcosis).
- Immune-mediated/presumed immune-mediated causes – exposure to lens proteins (cataract or lens rupture); idiopathic.
- Neoplastic causes – primary intraocular or metastatic to eye.

Systems Affected

- Ophthalmic



SIGNALMENT/HISTORY

- Anterior uveitis can occur in any dog or cat regardless of age, breed, or sex.
- Since there are many causes for anterior uveitis, the signalment and history of the patient will vary and should be used as a tool to guide the clinician towards a diagnostic plan.

Historical Findings

- Red (scleral injection) or cloudy (corneal edema, aqueous flare) eye of variable duration.
- Ocular pain (squinting, tearing, rubbing eye).
- Decreased vision.
- Previous trauma may indicate traumatic uveitis.
- Weight loss, lethargy, or decreased appetite may indicate a systemic cause.
- Bilateral uveitis supports systemic disease as the cause.

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CLINICAL FEATURES

- Injection of conjunctival and scleral vessels.
- Aqueous flare – may be subtle and difficult to detect or severe, causing anterior chamber to appear cloudy.
- Corneal edema – may be subtle or severe.
- Inflammatory cells may or may not be detected as hypopyon (white cells settled out in AC) or keratic precipitates (white cells deposited on the corneal endothelium; Fig. 3.1).
- Fibrin or blood clots in AC.
- Miosis is seen primarily when acute, pupil is often midrange.
- Iris swelling and vessel dilation – most obvious in light-colored eyes (Fig. 3.2).
- Posterior synechia (adhesion of iris to lens capsule) – causes abnormal pupil shape, seen in chronic cases and may be minimal or extensive (Fig. 3.3).
- Iris bombé – accumulation of aqueous humor behind the iris causing it to billow forward, due to extensive posterior synechia preventing AH from entering the anterior chamber through the pupil.
- Decreased intraocular pressure unless aqueous humor outflow is obstructed; IOP can then be in normal range or elevated.
- If systemic disease is the cause of uveitis, related clinical signs may be detected.

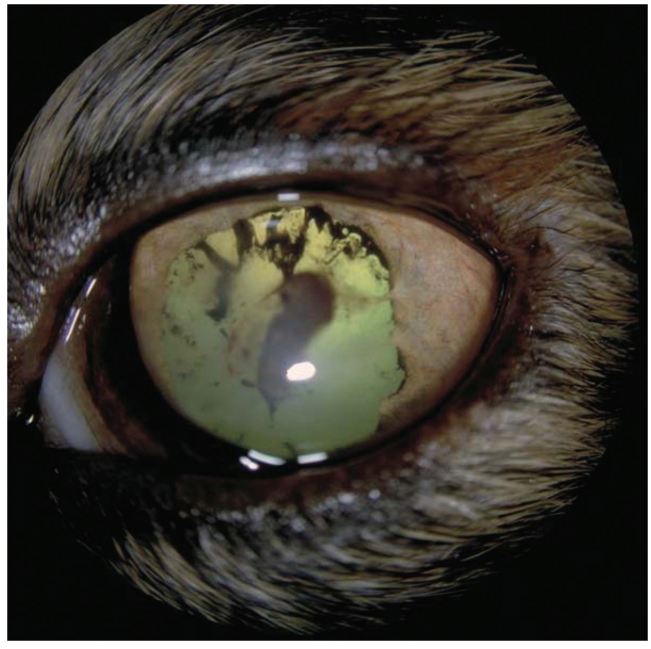
DIFFERENTIAL DIAGNOSIS

- Conjunctivitis – injection of conjunctival vessels only, ocular discharge, no intraocular changes that are associated with anterior uveitis, normal IOP, pain usually mild and relieved with topical anesthetic.

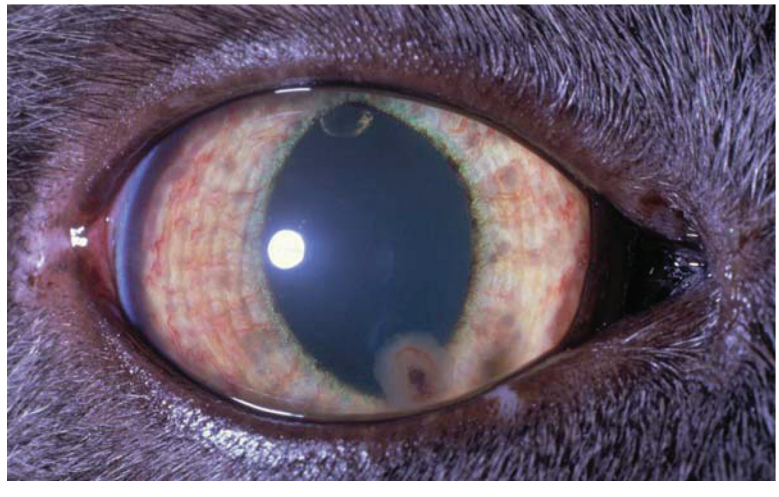


Fig. 3.1. Dog with chronic anterior uveitis, indicated by the extensive corneal neovascularization. The corneal endothelium has numerous keratic precipitates, causing the intraocular structures to be partially obscured.

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■ **Fig. 3.2.** Cat with anterior uveitis. The pupil is misshapen (dyscoria) due to extensive posterior synechia. A fibrin clot is in the central pupil and pigment from the posterior iris pigmented epithelium has been deposited onto the lens capsule.



■ **Fig. 3.3.** Cat with anterior uveitis. A small fibrin clot is partially overlying the pupil and iris ventrally. The iris is thickened and the iris vessels are injected. The circle in the dorsal pupil is a reflection.

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- Episcleritis/scleritis – conjunctival and scleral vessel injection, perilimbal corneal edema, sclera may be thickened, IOP and intraocular exam generally normal unless inflammation has extended to the uveal tract causing anterior uveitis.
- Glaucoma – elevated IOP, dilated pupil common, globe may be enlarged (buphthalmos) and cornea may have stria (streaks of corneal edema caused by breaks in Descemet's membrane).
- Any condition that causes injection of the conjunctival or scleral vessels (e.g., keratitis, Horner's syndrome).



DIAGNOSTICS

- Ocular examination including corneal fluorescein staining and intraocular pressure measurement.
- Systemic disease – general physical examination, CBC, serum chemistries, urinalysis. Further testing (serology, microbiology, imaging studies) is done based on the results of PE and initial lab work.
- Ocular ultrasound is indicated when primary ocular disease is suspected or identified and intraocular exam is not possible due to opacification of the cornea or lens.
- Examination of aqueous or vitreous humor may be required for diagnosis; ocular fluids can be used for cytology, culture and sensitivity, polymerase chain reaction, and antibody content.

Pathologic Findings

- Cornea – edema; neovascularization (if chronic); keratic precipitates (aggregates of WBCs on corneal endothelium).
- Anterior chamber – RBCs, WBCs, fibrin.
- Iris – WBC infiltration (cell type dependent on etiology); adhesions of iris to lens (posterior synechia); adhesions of iris to cornea (anterior synechia); preiridal fibrovascular membrane (if chronic).
- Ciliary body – WBC infiltration similar to iris.
- Lens – pigment migration onto capsule; posterior synechia; cataract (if chronic).



THERAPEUTICS

- Treatment goals for anterior uveitis:
  - Specific therapy for identified cause (treat corneal ulcer, infectious disease, neoplasia, luxated lens, etc.)
  - Nonspecific therapy for all cases of anterior uveitis – stop inflammation, prevent or control complications caused by inflammation (e.g., posterior synechia, glaucoma), and relieve pain.

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Drugs of Choice

- Glucocorticoids.
  - Topical.
    - Prednisolone acetate 1% suspension q1–12 hours
    - Dexamethasone 0.1% solution, 0.05% ointment q1–12 hours
    - Frequency depends on severity of inflammation
    - Taper medication as inflammation resolves
    - Contraindicated if corneal ulceration present
  - Subconjunctival injection.
    - Methylprednisolone acetate 4 mg/eye. Associated with development of subconjunctival plaque formation that could require surgical removal
    - Betamethasone 0.75 mg/eye
    - Triamcinolone 4 mg/eye
    - Rarely used to treat anterior uveitis
    - Used as a one-time injection in severe cases followed by topical therapy
    - Do not use in cats due to potential of concurrent FHV-1 infection
  - Systemic.
    - Prednisolone 0.5–2.2 mg/kg PO q12–24 hours.
    - Higher dosages for initial therapy of severe inflammation
    - Only used when systemic infectious causes have been ruled out
    - Taper medication as inflammation resolves
- Nonsteroidal antiinflammatory drugs.
  - Topical solutions.
    - Diclofenac 0.1% q6–12 hours
    - Flurbiprofen 0.03% q6–12 hours
    - Suprofen 1% q6–12 hours
    - Ketorolac 0.5% q6–12 hours
    - Frequency depends on severity of inflammation
    - Close monitoring required when corneal ulceration present due to link between topical NSAIDs and collagenolysis
  - Systemic.
    - Aspirin 10 mg/kg q12 hours (dog); 10–20 mg/kg q48–72 hours (cat) PO
    - Ketoprofen ≤2 mg/kg PO once, ≤1 mg/kg q24 hours (dog,cat)
    - Meloxicam 0.2 mg/kg PO initially, followed by 0.1 mg/kg PO (in food) once daily (dog); 0.2 mg/kg PO initially, followed by 0.1 mg/kg PO (in food) q24 hours × 2 days, then 0.25 mg/kg 2–3 times/week
    - Carprofen 2.2 mg/kg PO q12–24 hours (dog)
    - Deracoxib 1–2 mg/kg PO q24 hours or 3–4 mg/kg PO q24 hours (not to exceed 7 days at this dose)
- Topical mydriatic/cycloplegic.
  - Atropine sulfate 1% solution and ointment: q8–24 hours
  - Dilates pupil to prevent posterior synechia

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- Relieves ciliary muscle spasm to decrease pain
- Frequency of administration depends on severity of inflammation
- Should be used judiciously to effect

Precautions/Interactions

- Topical and subconjunctival glucocorticoids are contraindicated in the presence of a corneal ulcer.
- Glucocorticoids used for subconjunctival injection are in a slow-release vehicle; effects last up to 3 weeks.
- Methylprednisolone acetate may result in a subconjunctival inflammatory plaque that causes discomfort.
- Topical atropine can significantly reduce tear production.
- Continuous dilation of the pupil with atropine may obstruct aqueous humor outflow and contribute to development of secondary glaucoma.
- Use of systemic NSAIDs in cats has been associated with potentially serious side effects.
- Avoid treating with drugs that constrict the pupil (pilocarpine, latanoprost, demacarium bromide).



COMMENTS

Client Education

- The use of topical steroids is contraindicated if a corneal ulcer develops. Prevent self-trauma with a protective collar if necessary.
- Frequent follow-up examinations are essential if inflammation is moderate to severe since secondary glaucoma is common.
- Further client education based on identified underlying cause.

Patient Monitoring

- Recheck in 1–7 days depending on severity.
- Monitor IOP – IOP will increase with decreased inflammation; if IOP increases with static or worsening inflammation, this indicates aqueous outflow obstruction and impending glaucoma.
- Frequency of subsequent rechecks is dependent upon response to therapy.

Prevention/Avoidance

- Inadequate control of inflammation due to undertreating or discontinuing antiinflammatory too soon may lead to recurrent or chronic uveitis.
- Whenever possible, the cause of uveitis should be identified and eliminated to prevent recurrence.

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Possible Complications

- Posterior synechia causing abnormal pupil shape (dyscoria).
- Secondary glaucoma is common so frequent IOP measurement is important.
- Secondary cataract formation.
- Retinal detachment.
- Lens luxation.
- Systemic complications are dependent on underlying systemic disease.

Expected Course and Prognosis

- Ocular outcome varies depending on severity of and ability to control inflammation and secondary glaucoma (if present).
- Systemic outcome varies depending on disease process.

Synonyms

- Iridocyclitis
- Iritis

Abbreviations

AC: anterior chamber
AH: aqueous humor
CBC: complete blood count
FHV: feline herpesvirus
FIP: feline infectious peritonitis
FIV: feline immunodeficiency virus
IOP: intraocular pressure
NSAID: nonsteroidal antiinflammatory drug
PE: physical examination
PO: per os
RBC: red blood cell
WBC: white blood cell

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chapter 4

Anticoagulant Rodenticide Toxicity



DEFINITION/OVERVIEW

- Anticoagulant rodenticide ingestion produces an acquired coagulopathy, secondary to a depletion of vitamin K and subsequent deficiency in functional vitamin K-dependent coagulation factors.

ETIOLOGY/PATHOPHYSIOLOGY

- Vitamin K, a fat-soluble vitamin, is a crucial cofactor for hepatic posttranslational carboxylation of coagulation factors II, VII, IX, and X (and PC and protein S) (Fig. 4.1).
- Anticoagulant rodenticide ingestion inhibits normal enzymatic function of hepatic vitamin K_1 epoxide reductase, thereby preventing the recycling of the vitamin K_1 metabolite, vitamin K_1 epoxide, back to its functional form (Fig. 4.2).
- Once the hepatic vitamin K_1 stores are depleted, the production of functional vitamin K-dependent coagulation proteins ceases, causing the formation of PIVKAs.
- PIVKAs are incapable of chelating calcium, and are therefore unable to successfully partake in secondary hemostasis (soluble factor coagulation cascade).

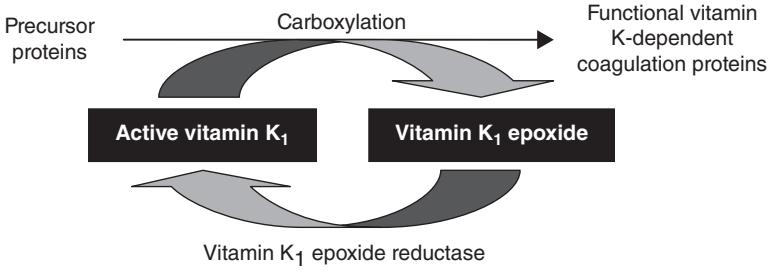
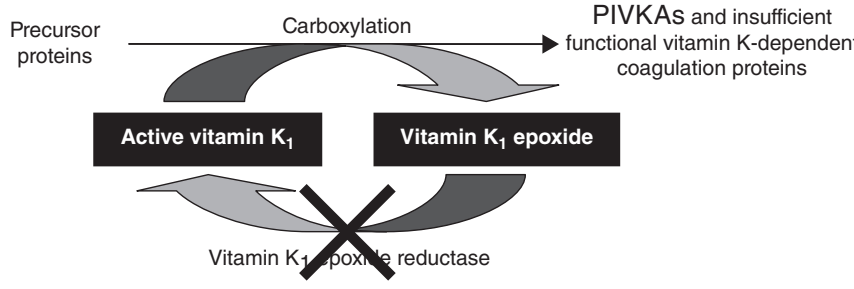


Fig. 4.1. Normal coagulation factor synthesis.

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■ **Fig. 4.2.** PIVKA formation in the presence of an anticoagulant rodenticide.

Systems Affected

- Cardiovascular: hemopericardium
- Gastrointestinal: sublingual hemorrhage or hemoabdomen
- Hemic/lymphatic/immune: active coagulation factor deficiency, anemia, hypoproteinemia
- Musculoskeletal: hemarthrosis
- Nervous: intracranial or intrathecal hemorrhage
- Respiratory: orolaryngeal hemorrhage with upper airway obstruction, peritracheal hematoma, hemothorax, and pulmonary parenchymal hemorrhage
- Skin/exocrine: SQ hemorrhage
- Ophthalmic: scleral or retrobulbar hemorrhage



SIGNALMENT/HISTORY

- No specific signalment, although intact animals may be more likely to roam and be accidentally or maliciously exposed to the toxins.

Risk Factors

- The presence of vitamin K antagonist rodenticides in the immediate environment.

Historical Findings

- May reveal possible exposure to, or ingestion of, anticoagulant rodenticides.
- Rarely, ingestion of an animal that consumed anticoagulant rodenticide (relay toxicosis).
- Vomit with green or turquoise granules in it.
- Lethargy.
- Anorexia.
- Cough, respiratory difficulty or hemorrhage/hemoptysis.

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CLINICAL FEATURES

- Clinical features are similar for dogs and cats.
- Clinically asymptomatic if ingestion occurred <48 hours previously.
- Clinical signs associated with deep tissue hemorrhage (hemoptysis, hemothorax, hemoabdomen, hemarthrosis, intracranial, perispinal, pericardial, exophthalmia, sublingual or subcutaneous hemorrhage) most frequently develop 3–6 days following consumption.
- In the acutely bleeding patient, hypoproteinemia will generally develop prior to anemia; anemia may develop simultaneously or before hypoproteinemia in the face of slow and sustained hemorrhage.

DIFFERENTIAL DIAGNOSIS

- Similar in dogs and cats, with the exception of variations in inherited coagulation factor deficiencies.
- Hepatic failure.
- Disseminated intravascular coagulation.
- Dilutional coagulopathy.
- Congenital factor deficiency.
- Massive heparin overdose.
- Spurious results (underfilling tube, polycythemia, delay in performing assay).
- Absolute vitamin K deficiency (malnourished patients receiving broad-spectrum antibiotic therapy, posthepatic biliary obstruction, or exocrine pancreatic insufficiency or intestinal malabsorption).
- Abnormal gamma-glutamyl carboxylase enzyme (Devon rex).

DIAGNOSTICS

- Diagnosis is often made with a history, clinical presentation, and/or coagulation profile that support the diagnosis, all in the face of a rapid response to vitamin K_1 therapy.
- Following ingestion, an anticoagulant effect is initially manifested as a prolonged PIVKA test (12–24 hours), then followed by a prolonged PT (24–48 hours); this initial anticoagulant effect is due solely to factor VII deficiency, which has the shortest half-life (generally 6 hours).
- Prolongation of the aPTT follows, after an additional 24–48 hours (48–96 hours post ingestion).
- Prolongation of the ACT is the last coagulation test to be abnormal, due to its lack of sensitivity to coagulation factor deficiency.
- A patient with spontaneous, active hemorrhage will have prolongation of PT, aPTT, ACT, and PIVKA test.
- Rarely is analysis of plasma or hepatic tissue needed to confirm the presence of compatible toxin.
- Point-of-care test kit available for detecting warfarin in blood of clinical patients.

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Pathologic Findings

- Presence of nonclotting blood found on aspiration of body cavity or swelling.
- Hemorrhage in the airways; uncommonly hemorrhage from mucosal surfaces.



THERAPEUTICS

- Treatment depends on timing of ingestion and the urgency of the clinical presentation.

Drugs of Choice

- Emesis induced if ingestion occurred within previous 2 hours (granules) or up to 8 hours (bar form).
- Activated charcoal administration ideal if ingestion occurred within previous 2 hours; however, this may delay efficacy of orally administered vitamin K_1.
- Vitamin K_1, 2.5–5.0 mg/kg/day PO for up to 6 weeks (dependent on agent ingested) is indicated for all patients with confirmed or even suspected ingestion; oral administration is generally more rapidly effective (<12 hours) than SQ (12–24 hours) and safer than IV (risk of anaphylaxis).
- Vitamin K_1 therapy alone is usually sufficient to reverse the anticoagulant effect in patients with elevated PIVKAs and/or prolonged PT, in the absence of hemorrhage.
- In more urgent cases (i.e., presence of hemorrhage), plasma (fresh frozen or frozen) 10–15 mL/kg and/or whole blood (fresh or stored) up to 20 mL/kg are administered, concurrently with vitamin K_1, to promptly (although temporarily) reverse anticoagulation.
- Cautious IV fluid administration to reverse shock, while minimizing exacerbation of further hemorrhage with unnecessary or excessive increases in blood pressure.
- Oxygen therapy if hypoxemia is present or in an attempt to ease respiratory distress; avoid nasal oxygen cannulation due to risk of inducing hemorrhage.

Precautions/Interactions

- Orally administered vitamin K_1 may be insufficiently absorbed if concomitant intestinal malabsorption or extrahepatic biliary obstruction is present, while hepatic dysfunction tempers the response to vitamin K_1 therapy.
- Allergic reactions may occur with SQ vitamin K_1 therapy, while anaphylaxis is a concern with IV administration.
- Vitamin K_1 administered IM may result in hematoma formation and is preferably avoided.
- Small-gauge needles should be used for SQ administration of vitamin K_1 (or any other parenteral medications), in several different sites to expedite absorption.
- Gastroenteric lavage is not indicated due to rapidly reversible nature of toxicity.

Diet

- Administration of oral vitamin K_1 with a high-fat meal (i.e., canned food) improves bioavailability.

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Activity

- With complicated toxicity, activity should be limited to avoid exacerbation of hemorrhage.

Surgical Considerations

- Thoracentesis may be indicated if the development of a hemothorax is sufficiently compromising respiration.
- Pericardiocentesis is indicated if tamponade is present.
- Preferably avoid unnecessary procedures until coagulation ability has normalized.

COMMENTS

Client Education

- Early in the course of therapy, provide extra bedding to prevent pressure-induced hemorrhage and avoid restraint with a collar to limit risk of ocular or intracranial hemorrhage.
- Herbal supplements, notably garlic, gingko, and ginseng, may exacerbate hemorrhagic tendencies.
- Concomitant vitamin E supplementation interferes with vitamin K-dependent coagulation.
- Toxins may be excreted in the milk of lactating patients.

Patient Monitoring

- For uncomplicated and complicated toxicity, monitor PT 48 and 96 hours after discontinuation of vitamin K_1 therapy to prove therapy has been administered for a sufficient duration; if prolonged, reinstitute vitamin K_1 therapy for 1–3 more weeks and repeat PT as previously instructed.
- For complicated toxicity, monitor parameters useful in detecting intravascular volume deficits from hemorrhage (tachycardia, pale mucous membranes, increase in respiratory rate, hypotension, PCV/TS, lactate, evidence of pleural space disease, etc.), as indicated.

Prevention/Avoidance

- Do not allow further access to toxin.
- Preferably avoid elective, invasive procedures until coagulation deficits have normalized.
- Nursing puppies or kittens should be changed to hand rearing and possibly supplemented with vitamin K_1 therapy.

Possible Complications

- Fatal hemorrhage – secondary to site (intracranial, myocardial, pericardial, intrapulmonary) or volume (massive).

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Expected Course and Prognosis

- In the absence of fatal hemorrhage, early and aggressive therapy should provide complete recovery with rapid cessation of hemorrhage and eventually normalization of coagulation tests (PT, aPTT, and ACT).

Abbreviations

ACT: activated clotting time
aPTT: activated partial thromboplastin time
DIC: disseminated intravascular coagulation
IM: intramuscular
IV: intravenous
PCV/TS: packed cell volume/total solids
PIVKA: **p**roteins **i**nduced by **v**itamin **K** **a**ntagonists or **a**bsence
PO: per os
PT: prothrombin time
SQ: subcutaneous

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chapter 5

Arterial Thromboembolism

DEFINITION/OVERVIEW

- ATE results from a thrombus or blood clot that is dislodged within the aorta, causing severe ischemia to the tissues served by that segment of aorta. It is one of the most devastating complications associated with myocardial diseases in cats.

ETIOLOGY/PATHOPHYSIOLOGY

- ATE is most commonly associated with myocardial disease in cats, including hypertrophic, restrictive, and dilated cardiomyopathy.
- Although the exact etiology of ATE has not been determined, it is theorized that abnormal blood flow (stasis) and a hypercoagulable state contribute to the formation of the thrombus within the left atrium. The blood clot is then embolized distally to the aorta.
- The most common site of embolization is the caudal aortic trifurcation, causing ischemic injury to both hind legs (Fig. 5.1).



Fig. 5.1. Clot in the hindlimb of a cat after arterial thromboembolism. Note the extensor rigidity of the affected limb.

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Fig. 5.2. Clot in the forelimb of a cat with arterial thromboembolism. Note the conscious proprioceptive deficits.

- Other less common sites include the front leg (Fig. 5.2), kidneys, gastrointestinal tract, or cerebrum.
- Although ATE is a well-recognized complication of myocardial disease in cats, the exact prevalence of ATE is not known in the general population of cats. In one study of cats with hypertrophic cardiomyopathy, approximately 17% presented with signs of ATE.
- Although >95% of ATE cases in cats are associated with advanced feline heart disease, another associated condition is neoplasia, typically pulmonary carcinoma.
- ATE rarely occurs in dogs. ATE in dogs typically is associated with neoplasia, sepsis, Cushing's disease, protein-losing nephropathy, or other hypercoagulable states. Severe heart disease is not often associated with ATE in the dog.

Systems Affected

- Cardiovascular: the majority of affected cats have advanced heart disease and experience left-sided heart failure.
- Nervous/musculoskeletal: severe ischemia to the muscles and nerves served by the segment of occluded aorta causes variable pain and paresis. Gait abnormalities or paralysis results in the leg or legs involved.

SIGNALMENT/HISTORY

- Typically middle-aged to older male mixed breed cat.
- Median age is typically 7–10 years (range 1–20 years).
- Males are more commonly affected than females (2:1).

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- The most common breed affected is the mixed breed cat. However, certain breeds have been overrepresented, such as Ragdolls, typically mirroring breeds that are prone to cardiomyopathies.

Risk Factors/Causes

- Although clear risk factors have not been defined, it is theorized that an enlarged left atrium or spontaneous echo contrast of the red blood cells or "smoke" observed on an echocardiographic examination may be risk factors.

Historical Findings

- Acute onset paralysis/paresis and pain are the most common owner complaints.
- Lameness or other gait abnormality may be seen.
- Tachypnea or respiratory distress is common.
- Vocalization and anxiety are common.



CLINICAL FEATURES

- Usually paraparesis or paralysis of the rear legs. Typically, both rear legs are affected equally but occasionally one leg is worse than the other. Less commonly, monoparesis of a front leg.
- Pain upon palpation of the affected legs. Gastrocnemius muscle often becomes firm several hours after embolization.
- Absent or diminished femoral pulses.
- Cyanotic or pale nail beds and footpads (Fig. 5.3).
- Affected limbs will be cooler than unaffected limbs upon palpation.
- Cardiac murmur or gallop sound may or may not be present. Despite the typical presence of severe heart disease in cats, often times a murmur or gallop may not be heard.
- Tachypnea or respiratory distress, sometimes with open mouth breathing, is often present either due to pain associated with the ischemic leg injury or due to concurrent congestive heart failure/pulmonary edema.
- Cardiac dysrhythmias may also be present. Dysrhythmias are more common during treatment and are often associated with reperfusion injury and hyperkalemia.
- Hypothermia is common in cats with ATE and is often associated with poor systemic perfusion and worse prognosis.



DIFFERENTIAL DIAGNOSIS

- Hindlimb paresis secondary to other causes such as spinal neoplasia, trauma, myelitis, fibrocartilaginous infarction, or intervertebral disk protrusion.

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Fig. 5.3. Differential cyanosis. Note the cyanotic discoloration of the footpads in the limb affected by the clot.



DIAGNOSTICS

- Typically, the diagnosis of ATE is made by physical exam alone. Many cats are in distress and empiric treatment is often initiated *prior to* diagnostic testing. In the cat, further diagnostic evaluation is helpful to better evaluate the severity and nature of the associated cardiac disease as well as systemic effects of the ATE. This information may be helpful for prognosis and treatment.
- In the dog, diagnostic evaluation is helpful to better understand the associated disease causing the hypercoagulable state.

Complete Blood Count/Biochemistry Panel/Urinalysis

Common abnormalities include the following.

- Elevated CPK, AST, ALT.
- Stress hyperglycemia.
- Azotemia with elevated BUN and creatinine as a result of possible low effective circulating volume or renal emboli.
- Electrolyte abnormalities are common (i.e., hyponatremia, hyperkalemia, hypocalcemia, and hyperphosphatemia) and are likely associated with poor renal perfusion and reperfusion injury.
- CBC and urinalysis changes are nonspecific.
- In the dog, a protein-to-creatinine ratio is advised if proteinuria is identified.

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Other Laboratory Tests

- Routine coagulation profile typically does not reveal significant abnormalities.
- In the dog, D-dimers are typically markedly elevated. Low D-dimer concentrations are sensitive that thromboembolic disease is unlikely.
- Baseline coagulation profile may be helpful to titrate heparin and possibly warfarin dosages.
- Thyroid hormone should be measured in cats over 7 years of age.

Thoracic Radiography

- Cardiomegaly is common and radiographic signs of congestive heart failure (i.e., pulmonary edema and/or pleural effusion) are seen in approximately 50–66% of cats.
- The presence of a pulmonary mass in the absence of heart disease in a cat with ATE is concerning for pulmonary carcinoma-associated embolus.

Echocardiography

- The majority of cats will have hypertrophic cardiomyopathy characterized by left ventricular hypertrophy, nondilated left ventricular lumen, and enlarged left atrium.
- Other types of heart disease are also possible, such as unclassified, restrictive, or dilated cardiomyopathy and thyrotoxic heart disease.
- Regardless of the type of myocardial disease present, the majority (>50%) have severe left atrial enlargement, (i.e., a left atrial to aortic ratio of ≥2.0).
- Occasionally, a left atrial thrombus or spontaneous echo contrast of the red blood cells (smoke) may be seen (Fig. 5.4).

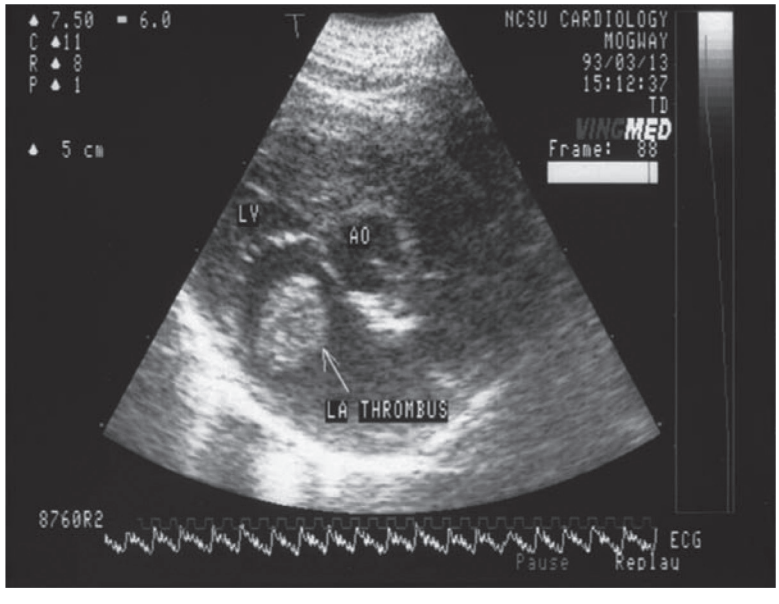


Fig. 5.4. Echocardiographic image of an enlarged left atrium that contains a thrombus.

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Abdominal Ultrasonography

- An experienced sonographer may be able to identify the thrombus in the caudal aorta. However, this imaging modality typically is not necessary to reach a diagnosis, especially in a cat.
- Abdominal ultrasound may be more useful in the dog to both identify the thrombus and look for associated diseases.

Computed Tomography Scan

- As with abdominal sonography, a multidetector CT scan is not necessary for diagnosis in a cat but could be helpful in a dog to reach a diagnosis, evaluate the extend of the thrombus, and look for associated diseases.

Pathologic Findings

- Thrombus typically is identified at the caudal aortic trifurcation.
- Occasionally, a left atrial thrombus is seen.
- Emboli of the kidneys, gastrointestinal tract, cerebrum, and other organs also may be observed.



THERAPEUTICS

The main objectives of treatment are threefold.

- First, immediate treatment of the pain associated with ischemic injury of the legs, typically with injectable opioids.
- Treatment directed at resolving the actual thrombus with anticoagulants or possibly thrombolytic agents.
- Treatment of the cat's heart disease and possible congestive heart failure.

Drugs of Choice

Pain Management

Once the diagnosis is reached, addressing the pain and distress associated with ATE is an immediate concern. If possible, intravenous opioid administration is preferred because of its rapid onset of action, bioavailability, and safety profile.

- In the cat, buprenorphine at 0.005–0.01 mg/kg IV every 6–8 hours as needed is a good initial choice. Buprenorphine can also be given in the cheek pouch or SQ if intravenous access is not obtained.
- Fentanyl (2–3 µg/kg intravenous bolus, then 1–5 µg /kg per hour IV CRI).
- Hydromorphone (0.025–0.1 mg/kg IV or SQ every 4–6 hours).
- Butorphanol (0.05–0.3 mg/kg IV or SQ every 2–6 hours as needed) has fewer analgesic effects than buprenorphine but is a good sedative. If no other opioids are available or if the cat's pain is assessed as mild, then butorphanol is a reasonable choice.

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- Cautious use of low-dose acepromazine (0.01 mg/kg IV or SQ every 8–12 hours as needed) may be helpful for additional sedation and vasodilation. Avoid acepromazine in patients with hypotension or hypothermia because of concerns about worsening systemic perfusion.

Antithrombotic Management

Cat

- Thrombolytic therapy with drugs such as streptokinase and tissue plasminogen activator is used extensively in human but is no longer used in cats. These drugs are expensive, carry a significant risk for bleeding complications, need to be administered within a few hours of ATE, and have not demonstrated significant therapeutic benefit over conservative management in veterinary medicine.
- Unfractionated heparin is the preferred drug in most clinical practices. Heparin actually has no effect on the established clot but it prevents further activation of the coagulation cascade and allows the body's endogenous fibrinolytic system to break down the clot. The initial dose typically is given IV then followed with subcutaneous administrations every 6–8 hours. The initial intravenous dose is 100–200 units/kg and the subsequent subcutaneous dose is 200–300 units/kg. Alternatively, an intravenous CRI of heparin at 600 units/kg per day could be used after the initial bolus. The dose is then ideally titrated to prolong the aPTT approximately twofold.
- In addition to heparin, the concurrent use of an antiplatelet agent is also advised. The two antiplatelet options are aspirin (5–81 mg PO every 3 days) or clopidogrel (18.75 mg (1/4 of a 75 mg tablet) PO every 24 hours). Clopidogrel requires 5–7 days to reach therapeutic concentrations, so concurrent use of aspirin is warranted in the early stage of therapy.
    - The higher dose of aspirin may be associated with more gastrointestinal adverse effects.
    - The theoretical benefit of clopidogrel is fewer gastrointestinal adverse effects and possible enhanced efficacy.
- Once signs of clinical improvement are seen, heparin therapy is gradually weaned over 1–2 days and long-term therapy is continued.

Dog

- Acute antithrombotic management considerations are similar in the dog.
- The dose of heparin in the dog is generally the same as in the cat. The doses of the antiplatelet agents are different.
    - Canine aspirin dose is 0.5–1 mg/kg PO every 24 hours.
    - The dose of clopidogrel dose in the dog is approximately 2 mg/kg PO every 24 hours. A higher loading dose of 10–11 mg/kg could be used on the first day if active clot is associated with significant ischemia.

Long-Term Therapy

- Recommendations for long-term anticoagulation are variable because no one treatment modality has shown clear benefit over another.
- Factors involved in deciding which long-term therapy to use include expense, ease of oral as opposed to subcutaenous administration, need for reevaluation, and monitoring.

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- Commonly used long-term anticoagulant therapies include aspirin, clopidogrel, or a low molecular weight agent.
- Aspirin (5–81 mg every 3 days) is the least expensive option but carries a higher risk of gastrointestinal and renal adverse effects.
- Clopidogrel (18.75 mg every 24 hours in cats, 2 mg/kg PO once daily in dogs) is moderately expensive and is dosed daily but may have enhanced long-term efficacy.
- Low molecular weight heparins have also been used in the long-term management of cats surviving an ATE.
    - Dalteparin (100–200 units/kg SQ every 12–24 hours) or enoxaparin (0.8–1.5 mg/kg SQ q6–12 hours) are two commonly used low molecular weight heparins.
    - The disadvantages of these medications are expense, subcutaneous administration, and controversy over the therapeutically efficacious dose in the cat.

Congestive Heart Failure Management

- Oxygen-rich environment.
- Furosemide (1–4 mg/kg as needed, not to exceed 12 mg/kg per day) IV or SQ should provide immediate relief of respiratory distress due to concurrent congestive heart failure.
- Other cardiac therapies such as enalapril, diltiazem, or pimobendan may also be indicated based on the results of echocardiogram.

Precautions/Interactions

- Anticoagulant therapy with heparin, clopidogrel, or the thrombolytic drugs may cause severe bleeding complications.
- Reperfusion of severely ischemic legs may be associated with severe hyperkalemia. Hyperkalemia and ischemia reperfusion injury are common causes of in-hospital mortality.
- Avoid a nonselective beta-blocker such as propranolol as it may enhance peripheral vasoconstriction.

Alternative Drugs

- Warfarin, a vitamin K antagonist, is the anticoagulant most widely used in humans and could be considered in recurrent ATE.
    - The initial dose is 0.05–1 mg/kg PO every 24 hours. It should be overlapped with heparin therapy for 3 days. The dose is then adjusted to prolong the PT approximately two times its baseline value or to attain an INR of 2.0–4.0.
    - Warfarin has an unpredictable dose-to-response effect and is highly protein bound. Thus, frequent monitoring and titration of the dose are required. Warfarin also carries a more significant risk of bleeding. For this reason, warfarin therapy is no longer commonly used.

Diet

- Initially, most cats are anorexic. Tempt these cats with any type of diet. It is important to keep these cats eating to avoid hepatic lipidosis. Appetite stimulants are often used.

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Nasoesophageal tube feedings may be indicated if more than 3 days of anorexia. Chronic dietary management usually involves sodium restriction.

Activity

- Activity should be restricted. The cat should be kept quiet, stress free, and indoors only.

Surgical Considerations

- Surgical embolectomy typically is not recommended because these patients are high risks for surgery and anesthesia as a result of their heart disease.
- Rheolytic thrombectomy has recently been evaluated in the treatment of feline ATE with favorable treatment results but is not commonly available even at tertiary referral centers.

COMMENTS

- It should be emphasized that the finding of tachypnea, even open mouth breathing, upon initial examination should not presume congestive heart failure. Some cats may be tachypneic solely as a result of pain. If the tachypnea persists after appropriate analgesic therapy, or if physical examination (crackles) or radiographic findings are compatible with pulmonary edema, then furosemide therapy is indicated.
- Fluid therapy may be necessary in the initial stages if the cat is not in congestive heart failure.
- Initially, the affected legs should be minimally handled because reperfusion results; in the long term, physical therapy (passive extension and flexion of the legs) may speed full recovery.
- No venipuncture should be performed on the affected legs.
- Initially, these cats may have difficulty posturing to urinate and may need to have their bladders expressed periodically to prevent overdistension of the bladder or urine scald.

Client Education

- Owners should be aware of the poor short- and long-term prognosis. Many cats will have advanced heart disease and are at risk for reembolization. They will require lifelong medications, reevaluations, and an indoor lifestyle if they survive to discharge.
- Typically, most cats that survive an initial episode will recover complete function of the legs; however, some neurologic or musculoskeletal deficits may persist.

Patient Monitoring

- Hourly to daily examination of the legs, femoral pulses, and respiratory rate should be performed to assess clinical response to therapy.

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- Continuous ECG monitoring is helpful to identify hyperkalemia or cardiac arrhythmias/conduction disturbances associated with severe reperfusion injury.
- aPTT can also be monitored once daily to titrate the unfractionated heparin dose.
- Periodic evaluation of thoracic radiographs, electrolyte, and renal parameters is also helpful in evaluating response to therapy.

Prevention/Avoidance

- Because of the high rate of reembolization (25–75%) after surviving an initial episode, prevention with chronic aspirin, clopidogrel or low molecular weight heparin is strongly recommended. See earlier in this chapter for doses.

Possible Complications

- Death is unfortunately a common outcome due to either progression of disease or complications of therapy.
- Bleeding complications may arise with the anticoagulant therapy.
- Life-threatening hyperkalemia and arrhythmias due to reperfusion injury are complications of therapy.
- Permanent neurological deficits or muscular abnormalities in the hindlimbs may arise in some cats (~15%) with severe and prolonged ischemia.
- If a cat survives an initial episode of ATE, recurrence of ATE and congestive heart failure are common.

Expected Course and Prognosis

- Both short-term and long-term prognosis are generally poor. Most cats (>50%) are euthanized or die during their initial ATE event regardless of therapy utilized.
- Admitting rectal temperature of >99 °F, fast heart rate, only one limb affected, and presence of motor function are all associated with better short-term prognosis.
- Concurrent congestive heart failure is associated with a worse long-term prognosis. In one study, cats with concurrent congestive heart failure had a median survival time of 77 days versus those without congestive heart failure of 233 days.
- Refractory congestive heart failure or recurrence of ATE are typical terminal issues if a cat survives an initial ATE event.
- Expected course of recovery is generally days but can be weeks for return of function to the legs.
- Most cats that survive an initial episode will recover completely but approximately 15% may suffer permanent neuromuscular deficits or ischemic injuries such as loss of digits or tip of tail.

Synonyms

- Saddle thrombus

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Abbreviations

ALT: alanine transaminase
aPTT: activated partial thromboplastin time
AST: aspartate aminotransferase
ATE: arterial thromboembolism
BUN: blood urea nitrogen
CBC: complete blood count
CPK: creatine phosphokinase
CRI: constant rate infusion
CT: computed tomography
ECG: electrocardiogram
INR: international normalized ratio
IV: intravenous
PO: per os
PT: prothrombin time
SQ: subcutaneous

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chapter 6

Atrial Fibrillation and Atrial Flutter

DEFINITION/OVERVIEW

- Atrial fibrillation – rapid, irregularly irregular supraventricular rhythm.
- Two forms recognized: primary atrial fibrillation, an uncommon disease that occurs mostly in large dogs with no underlying cardiac disease, and secondary atrial fibrillation, which occurs in dogs and cats secondary to underlying cardiac disease.
- Atrial flutter is similar to atrial fibrillation, but the atrial rate is generally slower and is characterized by saw-toothed flutter waves in the baseline of the ECG. The ventricular response is generally rapid but may be regular or irregular.

Electocardiogram Features

Atrial Flutter

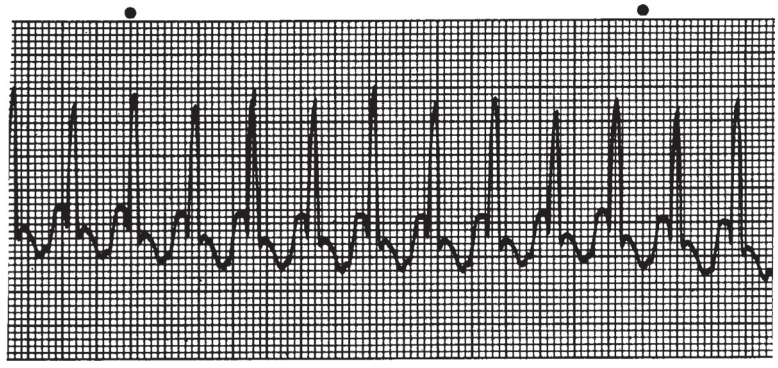
- Atrial rhythm usually regular; rate approximately 300–400 beats per minute.
- P waves usually discerned as either discrete P waves or a "saw-toothed" baseline.
- Ventricular rhythm and rate generally depend on the atrial rate and AV nodal conduction, but are generally regular or regularly irregular and rapid.
- Conduction pattern to the ventricles is variable – in some cases every other atrial depolarization produces a ventricular depolarization (2:1 conduction ratio), giving a regular ventricular rhythm; other times the conduction pattern appears random, giving an irregular ventricular rhythm that can mimic atrial fibrillation (Fig. 6.1).

Secondary Atrial Fibrillation

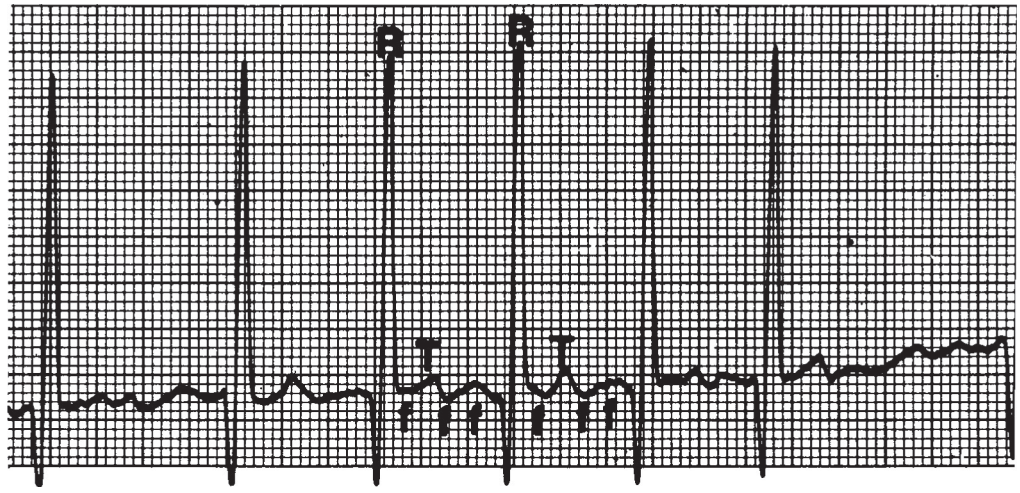
- No P waves present – baseline may be flat or may have small irregular undulations ("f" waves); some undulations may look like P waves (Fig. 6.2).
- Ventricular rate high – usually 180–240 beats per minute in dogs and >220 beats per minute in cats.
- Interval between QRS complexes is irregularly irregular; QRS complexes usually appear normal.

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■ **Fig. 6.1.** Atrial flutter with 2:1 conduction at a ventricular rate of 330/minute in a dog with an atrial septal defect. This supraventricular tachycardia was associated with a Wolff–Parkinson–White pattern. Source: Tilley 1992, with permission of John Wiley and Sons Inc.



■ **Fig. 6.2.** "Coarse" atrial fibrillation in a dog with patent ductus arteriosus. The f waves are prominent. Source: Tilley 1992, with permission of John Wiley and Sons Inc.

Primary Atrial Fibrillation

- Similar to secondary atrial fibrillation except ventricular rate usually in the normal range.

ETIOLOGY/PATHOPHYSIOLOGY

Risk Factors/Causes

- Heart disease
- Chronic valvular disease
- Cardiomyopathy

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- Congenital heart disease
- Digoxin toxicity
- Idiopathic
- Ventricular preexcitation (atrial flutter)

Atrial Fibrillation

- Caused by numerous small reentrant pathways creating a rapid (>500 depolarizations per minute) and disorganized depolarization pattern in the atria that results in cessation of atrial contraction.
- Depolarizations continuously bombard the AV nodal tissue, which acts as a filter and does not allow all depolarizations to conduct to the ventricles.
- Many atrial depolarizations activate only a part of the atria because the rapid rate renders portions of the atria refractory, and thus they cannot reach the AV junction.
- Other atrial impulses penetrate into the AV junctional tissue but are not robust enough to penetrate the entire length. Blocked impulses affect the conduction properties of the AV junctional tissue and alter conduction of subsequent electrical impulses; electrical impulses are conducted through the AV junction irregularly, producing an irregular ventricular rhythm.

Atrial Flutter

- Probably originates from one site of reentry that moves continuously throughout the atrial myocardium and frequently and regularly stimulates the AV node. When the atrial rate becomes sufficiently fast, the refractory period of the AV node exceeds the cycle length (P to P interval) of the SVT, and some atrial depolarizations are blocked from traversing the AV node (functional second-degree AV block).

Systems Affected

Cardiovascular

- Loss of atrial contraction may result in decreased stroke volume and cardiac output depending on heart rate.
- High heart rate may result in deterioration in myocardial function (tachycardia-induced myocardial failure).



SIGNALMENT/HISTORY

Species

- Dogs and cats.

Breed Predilections

- Large- and giant-breed dogs are more prone to primary atrial fibrillation.

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General Comments

- Signs generally relate to the underlying disease process or CHF rather than the arrhythmia itself, but previously stable animals may decompensate.
- Patients with primary atrial fibrillation are generally asymptomatic but may demonstrate mild exercise intolerance.

Historical Findings

- Coughing/respiratory difficulty/tachypnea.
- Exercise intolerance.
- Rarely syncope.
- Dogs with primary atrial fibrillation are typically asymptomatic.

Physical Examination Findings

- On auscultation, patients with atrial fibrillation have an erratic heart rhythm that sounds like "tennis shoes in a dryer" or "jungle drums."
- First heart sound intensity in atrial fibrillation is variable; second heart sound only heard on beats with effective ejection, not on every beat.
- Third heart sounds (gallop sounds) may be present.
- Patients with atrial fibrillation have pulse deficits and variable pulse quality.
- Signs of CHF often present (e.g., cough, respiratory distress, cyanosis).



DIFFERENTIAL DIAGNOSIS

- Frequent atrial (supraventricular) premature depolarizations
- SVT with AV block
- Multifocal atrial tachycardia (irregular)



DIAGNOSTICS

Imaging

- Echocardiography and radiography may characterize type and severity of the underlying cardiac disease; moderate to severe left atrial enlargement common.
- Typically normal in patients with primary atrial fibrillation, although mild left atrial enlargement may accompany the hemodynamic alterations imposed by the arrhythmia.

Holter Monitoring

- A baseline 24-hour Holter is recommended to determine if the arrhythmia is chronic or paroxysmal. If it is chronic, drug therapy is indicated.

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Pathologic Findings

- Depend on underlying cause.



THERAPEUTICS

Drugs of Choice

- Digoxin, beta-adrenergic blockers, and calcium channel blockers (diltiazem) are frequently used to slow conduction through the AV node; definition of an adequate heart rate response varies among clinicians, but in dogs is generally 140–160 beats per minute.
- For atrial flutter, therapy is aimed at suppressing the atrial reentry circuit using sotalol, amiodarone, or procainamide.

Dogs

- Digoxin – maintenance oral dose 0.005–0.01 mg/kg PO every 12 hours; to achieve a therapeutic serum concentration more rapidly, the maintenance dose can be doubled for the first day. If digoxin is administered alone and the heart rate remains high, check the digoxin level and adjust the dose to bring the level into the therapeutic range. If the heart rate remains high, consider adding a calcium channel blocker or a beta-adrenergic blocker.
- Diltiazem – initially administered at a dose of 0.5 mg/kg PO every 8 hours, then titrated up to a maximum of 1.5 mg/kg PO every 8 hours or until an adequate response is obtained.

Cats

- Diltiazem (1–2.5 mg/kg PO every 8 hours) or atenolol (6.25–12.5 mg PO every 12–24 hours) are the drugs of choice in most cats.
- If the heart rate is not sufficiently slowed with these drugs or if myocardial failure is present, digoxin (0.005 mg/kg PO every 24–48 hours) can be added.

Precautions/Interactions

- Calcium channel blockers and beta-adrenergic blockers, both negative inotropes, should be used cautiously in animals with myocardial failure.
- Using high-dose oral quinidine for conversion into sinus rhythm carries a risk of quinidine toxicity (e.g., hypotension, weakness, ataxia, and seizures); administration of diazepam IV controls seizures; other signs abate within several hours of discontinuing quinidine.
- Quinidine raises the digoxin level, generally necessitating a digoxin dose reduction.

Alternative Drugs

- Propranolol – initially administered at a dose of 0.1–0.2 mg/kg PO every 8 hours, then titrated upward until an adequate response is obtained. Do not exceed a dose of 0.5 mg/kg PO every 8 hours. Propranolol is poorly tolerated when used chronically and also affects beta-2 receptors and is therefore rarely used.

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Diet

- Mild-to-moderate sodium restriction if CHF.

Activity

- Restrict activity until tachycardia is controlled.

Appropriate Healthcare

- Patients with fast (secondary) atrial fibrillation are treated medically to slow the ventricular rate. Converting the atrial fibrillation to sinus rhythm would be ideal, but such attempts in patients with severe underlying heart disease or left atrial enlargement are generally futile because of a low success rate and high rate of recurrence.
- Consider electrical cardioversion to sinus rhythm for a dog with primary atrial fibrillation and only mild structural heart disease.
- Patients with primary atrial fibrillation may be converted back to normal sinus rhythm. The success rate depends on chronicity. Patients which have been in atrial fibrillation for fewer than 4 months generally have a lower success rate and a higher rate of recurrence. In these patients, rate control, if necessary, is the recommended treatment.
- Electrical (DC) cardioversion – application of a transthoracic electrical shock at a specific time in the cardiac cycle; requires special equipment, trained personnel, and general anesthesia. Using a monophasic defibrillator: start with 4J/kg; if no conversion occurs, increase dose by 50J and repeat until a maximum of 360J. Using a biphasic defibrillator: start with 1–2J/kg; if no cardioversion occurs, increase dose by 50J and repeat until a maximum of 360J.
- For atrial flutter, conversion to sinus rhythm can be done by drug therapy, electrical cardioversion, or rapid atrial pacing (transvenous pacing electrode).

Nursing Care

- As indicated for CHF.



COMMENTS

Client Education

- Secondary atrial fibrillation and atrial flutter is usually associated with severe underlying heart disease; the goal of therapy is to lower heart rate and control clinical signs.
- Sustained conversion to sinus rhythm is unlikely with secondary atrial fibrillation.

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Patient Monitoring

- Monitor heart rate and ECG closely.
- Because heart rates in the hospital and those measured on the surface ECG may be inaccurate (due to patient anxiety and other environmental factors), Holter monitoring provides a more accurate means for assessing the need for heart rate control or the efficacy of medical therapy for heart rate control.

Possible Complications

- Worsening of cardiac function with onset of arrhythmia.

Expected Course and Prognosis

- Secondary atrial fibrillation – associated with severe heart disease, so a guarded-to-poor prognosis.
- Primary atrial fibrillation with normal ultrasound findings – generally a good prognosis.

Abbreviations

AV: atrioventricular
CHF: congestive heart failure
DC: direct current
ECG: electrocardiogram
IV: intravenous
PO: per os
SVT: supraventricular tachycardia

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chapter 7

Atrial Standstill

DEFINITION/OVERVIEW

- ECG rhythm characterized by absence of P waves.
- Condition can be temporary (e.g., associated with hyperkalemia or drug induced), terminal (e.g., associated with severe hyperkalemia or dying heart), or persistent.

Electrocardiogram Features

Persistent Atrial Standstill (Fig. 7.1)

- P waves absent.
- Heart rate usually slow (<60 beats per minute).
- Rhythm regular with supraventricular type QRS complexes.
- Heart rate does not increase with atropine administration.

Hyperkalemic Atrial Standstill

- Heart rate normal or slow.
- Rhythm regular or irregular.
- QRS complexes tend to be wide and become wider as the potassium level rises; with severe hyperkalemia (potassium >10 mEq/mL), the QRS complexes are replaced by a smooth biphasic curve.
- Heart rate may increase slightly with atropine.

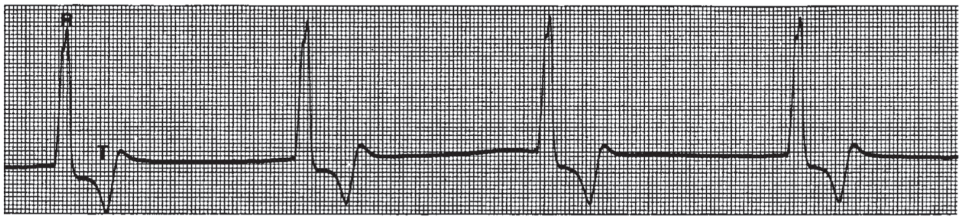


Fig. 7.1. Persistent atrial standstill in an English Springer Spaniel. No P waves are present on any of the leads (also including chest leads and intracardiac electrocardiogram, not shown here). The regular bradycardia is either junctional in origin, with pathologic involvement of the left bundle branch block (wide positive QRS complexes), or ventricular. Source: Tilley LP. Essentials of Canine and Feline Electrocardiography, 3rd edn. Baltimore, MD: Williams and Wilkins, 1992, with permission.

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ETIOLOGY/PATHOPHYSIOLOGY

Persistent Atrial Standstill

- Rare condition caused by an atrial muscular dystrophy; skeletal muscle involvement common.

Hyperkalemic Atrial Standstill

- Generally occurs with serum potassium levels >8.5 mEq/L; value influenced by serum sodium and calcium levels and acid–base status. Patients who are hyperkalemic with atrial standstill have sinus node function but impulses do not activate atrial myocytes; thus, the associated rhythm is termed a sinoventricular rhythm. Because the sinus node is functional, an irregular rhythm may be due to sinus arrhythmia.

Systems Affected

- Cardiovascular



SIGNALMENT/HISTORY

Species

- Dog and cat.

Breed Predisposition

- Persistent atrial standstill – most common in English springer spaniels; other breeds occasionally affected.

Mean Age and Range

- Most animals with persistent atrial standstill are young.
- Animals with hypoadrenocorticism are usually young to middle-aged.
- Any age if associated with uroabdomen or urethral obstruction.

Predominant Sex

- Hypoadrenocorticism more common in females (69%).

Historical Findings

- Vary with underlying cause.
- Lethargy common; syncope may occur.
- Patients with persistent atrial standstill may show signs of CHF.
- Cats with urinary obstruction may have a history of dysuria or stranguria.

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CLINICAL FEATURES

- Vary with underlying cause.
- Bradycardia common.
- Dogs with persistent atrial standstill may have skeletal muscle wasting of the antebrachium and scapula.



DIFFERENTIAL DIAGNOSIS

- Slow atrial fibrillation.
- Sinus bradycardia with small P waves lost in the baseline.



DIAGNOSTICS

Complete Blood Count/Biochemistry/Urinalysis

Persistent Atrial Standstill

- Normal

Hyperkalemic Atrial Standstill

- Hyperkalemia.
- Hyponatremia and sodium-to-potassium ratio <27 if atrial standstill secondary to hypoadrenocorticism.
- Azotemia and hyperphosphatemia with hypoadrenocorticism, renal failure, and rupture or obstruction of the urinary tract.

Other Laboratory Tests

- Baseline cortisol and/or complete ACTH stimulation test if hypoadrenocorticism suspected; if baseline cortisol is <2 µg/dL, perform a complete ACTH stimulation test to definitively diagnose hypoadrenocorticism.

Imaging

- Echocardiogram and electromyography if persistent atrial standstill suspected; cardiomegaly and depressed contractility may be seen.

Other Diagnostic Tests

- Skeletal muscle biopsy in animals with persistent atrial standstill.

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Pathologic Findings

Persistent Atrial Standstill

- Greatly enlarged and paper-thin atria; usually biatrial involvement, although one case of only left atrial involvement has been reported.
- Severe scapular and brachial muscle wasting in some dogs.
- Marked fibrosis, fibroelastosis, chronic mononuclear cell inflammation, and steatosis throughout the atria and interatrial septum.



THERAPEUTICS

Drugs of Choice

Persistent Atrial Standstill

- Treat with diuretics and ACE inhibitor (e.g., enalapril or benazepril) if CHF develops.

Hyperkalemic Atrial Standstill

- Treat the underlying cause (e.g., oliguric renal failure, hypoadrenocorticism).
- Aggressive fluid therapy with 0.9% saline and possibly sodium bicarbonate or insulin with dextrose, as will be discussed.
- Can administer sodium bicarbonate to patients with severe hyperkalemia to induce translocation of potassium into cells; if blood pH and base deficit cannot be determined, administer 1–2 mEq/kg slowly IV. To calculate bicarbonate dose more accurately:
  - Dogs: 0.3 × body weight (kg) × (21 –patient's HCO_3^-)
  - Cats: 0.3 × body weight (kg) × (19 –patient's HCO_3^-)
  - Administer half of dose and reevaluate.
- Can administer dextrose and regular insulin to patients with severe hyperkalemia to induce translocation of potassium into cells (regular insulin, 0.5 U/kg IV with 50% dextrose, 1 g/kg IV); dextrose can also be used without insulin; follow insulin and dextrose administration with 2.5% dextrose as a CRI to prevent hypoglycemia.
- For patients with life-threatening hyperkalemia, administer calcium gluconate 10% (0.5–1 mL/kg slowly IV over 10 minutes) while monitoring the ECG; calcium antagonizes the effect of potassium on the conduction system without lowering the potassium concentration.

Precautions/Interactions

- Avoid potassium-containing fluids or medications that increase potassium concentration in patients who are hyperkalemic.
- Diuretics lower preload and may worsen weakness in dogs with persistent atrial standstill and CHF unless a pacemaker has been implanted.

Activity

- Restrict activity in patients with persistent atrial standstill and signs of CHF or syncope.

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Surgical Considerations

Persistent Atrial Standstill

- Implant permanent ventricular pacemaker to regulate rate and rhythm.

Hyperkalemic Atrial Standstill

- Hyperkalemia secondary to urinary tract obstruction or rupture may require surgery.



COMMENTS

Client Education

Persistent Atrial Standstill

- Clinical signs generally improve after pacemaker implantation; signs of CHF may develop, and weakness and lethargy may persist even after heart rate and rhythm are corrected with the pacemaker.

Patient Monitoring

- Monitor ECG during treatment of hyperkalemia and periodically in animals with a permanent ventricular pacemaker.
- Monitor electrolytes in patients with hyperkalemic atrial standstill.
- Monitor patients with persistent atrial standstill for signs of CHF.

Possible Complications

- CHF in patients with persistent atrial standstill.

Expected Course and Prognosis

Persistent Atrial Standstill

- Clinical signs generally improve after pacemaker implantation. Signs of CHF may develop, and weakness and lethargy persist even after heart rate and rhythm are corrected with the pacemaker. There may be persistence of signs related to muscular dystrophy.

Hyperkalemic Atrial Standstill

- Long-term prognosis is excellent if underlying cause can be corrected and hyperkalemia reversed.

Synonyms

- Silent atria

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Abbreviations

ACE: angiotensin-converting enzyme
ACTH: adrenocorticotropic hormone
CHF: congestive heart failure
CRI: constant rate infusion
ECG: electrocardiogram
HCO_3^-: bicarbonate
IV: intravenous
pH: measure of acidity or base

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Author: Francis W.K. Smith, Jr, DVM

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chapter 8

Atrioventricular Block

DEFINITION/OVERVIEW

- Atrioventricular (AV) block refers to a delay or block of impulse conduction from the atria to the ventricles at a time when the conduction system is not refractory.
- Conduction delay or block may arise at any site between the sinoatrial node and the ventricular myocardium (i.e., intraatrial, intranodal, or subnodal block).
- While certain ECG patterns of AV block more typically reflect impaired conduction in one particular segment of the cardiac conduction system, overlap occurs, and it is often impossible to determine with certainty which part of the conduction system is responsible for the block based on the surface ECG.
- AV block is classified into three categories based on ECG characteristics.
  - First-degree AV block refers to delayed AV conduction.
    - The ECG shows a 1:1 relationship of P waves and QRS complexes with a prolonged PR interval (>0.13 s in dogs and >0.09 s in cats).
    - QRS and T waves usually appear normal (Fig. 8.1).
  - Second-degree AV block occurs when there is failure of some but not all P waves to be conducted to the ventricles; three patterns of second-degree AV block are recognized.
    - Mobitz type I (Wenckebach) second-degree AV block is characterized by gradual prolongation of PR intervals until a P wave fails to conduct (Fig. 8.2).
    - Mobitz type II second-degree AV block is characterized by abrupt failure of one or more consecutive P waves to be conducted without progressive lengthening of the PR interval prior to the blocked P wave (Fig. 8.3).

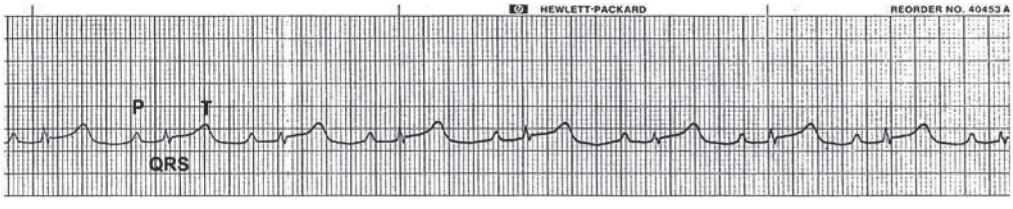


Fig. 8.1. A lead II ECG rhythm strip recorded from an 8-year-old cat. There is sinus bradycardia (120 beats/minute) and first-degree AV block. A P wave precedes each QRS complex with a constant but prolonged PR interval of 0.14 s (paper speed = 50 mm/s).

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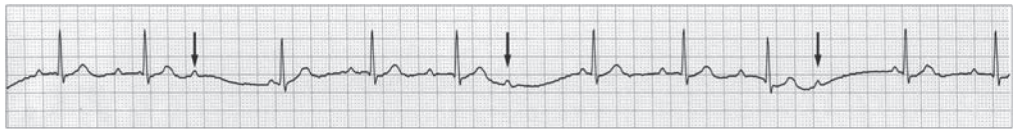


Fig. 8.2. A lead II ECG rhythm strip recorded from a dog with Mobitz type I second-degree AV block. Nonconducted P waves are indicated by arrows. The PR interval becomes progressively longer with the longest interval preceding a nonconducted P wave (paper speed = 50 mm/s).

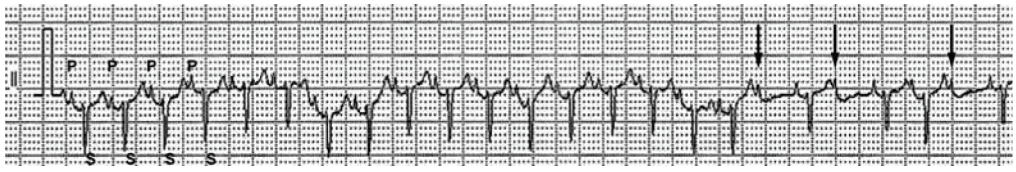


Fig. 8.3. A lead II ECG rhythm strip recorded from a cat with Mobitz type II second-degree AV block. There are intermittent nonconducted P waves (*arrows*); however, the PR interval of conducted beats is constant (0.09 s). The QRS complexes are abnormal (rS pattern) because of concomitant left anterior fascicular block (paper speed = 25 mm/s).

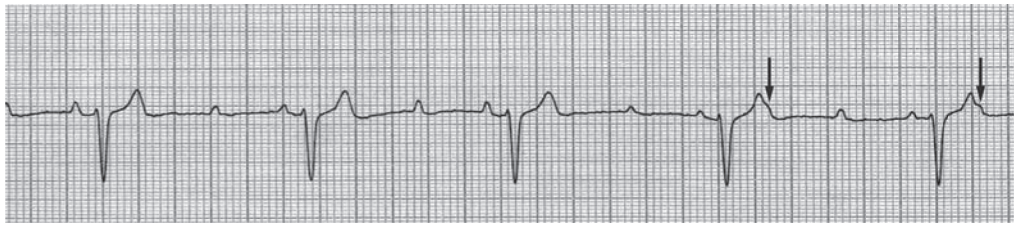
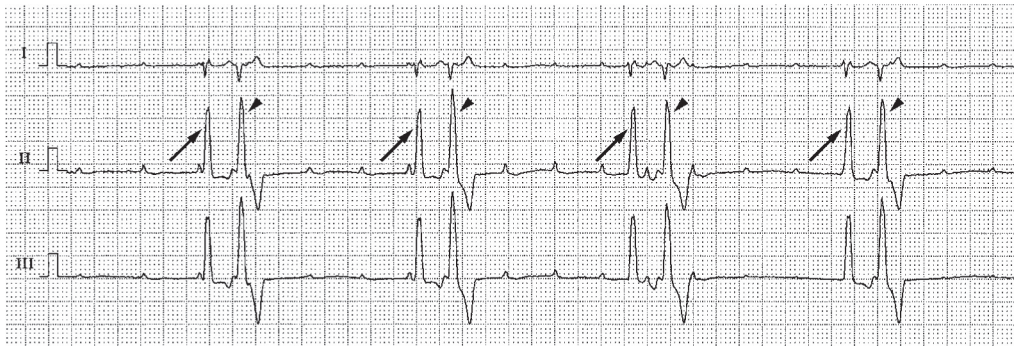


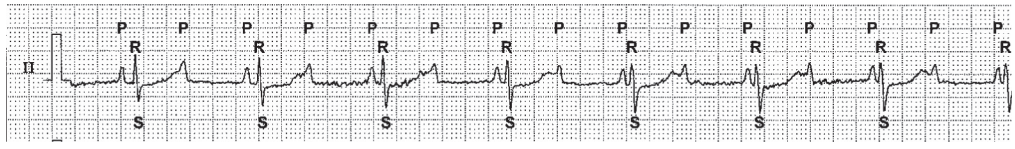
Fig. 8.4. A lead II rhythm strip recorded from a dog with bradycardia (ventricular rate = 62 beats/minute) as a result of high-grade second-degree AV block occurring in a 3:1 conduction pattern. The initial P wave of each cycle is hidden by the preceding T wave; however, these P waves can occasionally be appreciated deforming the T wave (*arrows*). The QRS complexes are abnormal (wide and negative) because the dog also has right bundle branch block (paper speed = 25 mm/s).

  - □ High-grade second-degree AV block is characterized by a fixed ratio of P waves to QRS complexes, e.g., 2:1, 3:1, 4:1 (Fig. 8.4). (This type of second-degree AV block cannot be classified as Type I or Type II.)
  - □ QRS and T waves appear normal unless there is also an intraventricular conduction abnormality.
- Third-degree (complete) AV block is the result of complete absence of AV conduction.
  - □ Characterized on ECG (in patients with sinus rhythm or other organized atrial rhythm) as a complete lack of association of P waves and QRS complexes with the ventricular rate slower than the atrial rate.

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■ **Fig. 8.5.** A simultaneous lead I, II, and III ECG recording from a dog with complete (third-degree) AV block. The atrial rate (136 beats/minute) exceeds the ventricular rate, and there is no relationship between P waves and QRS complexes. There is a ventricular escape focus depolarizing the ventricular myocardium at a regular rate of 32 beats/minute. These QRS complexes are abnormally wide (0.09 s) with a left bundle branch morphology (*arrows*). Each of the ventricular escape complexes is coupled to a premature ventricular contraction (*arrowheads*) (paper speed = 25 mm/s).



■ **Fig. 8.6.** This lead II ECG was recorded from a cat with complete (third-degree) AV block. The atrial rate (214 beats/minute) exceeds the ventricular rate (109 beats/minute), and there is no relationship between P waves and QRS complexes. The morphology of the QRS complexes is normal, indicating a junctional escape focus (paper speed = 50 mm/s).

- Because ventricular depolarization arises from escape activity of a subsidiary intraventricular pacemaker, QRS complexes are typically wide and bizarre (Fig. 8.5).
- In many cats and some dogs, the escape focus is near the origin of the His bundle (in the region defined as the AV junction), and in these animals the QRS complexes are narrow (Fig. 8.6).



ETIOLOGY/PATHOPHYSIOLOGY

- A variety of disorders or disease processes may cause impaired AV conduction, and the severity of impairment may range from first degree to complete AV block in each of these circumstances.
- AV block may be transient or permanent.
- AV block may progress from first degree to more severe block.
- AV block may result from intrinsic disease of the cardiac conduction system or from extrinsic factors adversely affecting impulse conduction.

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- Any condition that increases vagal tone may produce varying degrees of AV block.
  - Vagal stimulation is a common cause of AV conduction block in the critical care setting.
  - Second-degree AV block occurs in 64% of healthy adult dogs and 100% of healthy pups between 8 and 11 weeks of age.
  - Vagal tone is increased during sleep; therefore, AV block may disappear during waking hours and with resumption of normal activity.
  - Valsalva maneuvers may also result in transient AV block.
  - Enhanced vagal tone in dogs and cats may be associated with conditions such as upper airway disease, cervical and thoracic masses, gastrointestinal disorders, head or neck trauma, increased CSF pressure, and glaucoma.
- Electrolyte imbalances may predispose to or cause AV block (hyperkalemia directly impairs AV conduction whereas hypokalemia enhances the effect of vagal stimulation).
- Other extrinsic causes of AV conduction block include drugs or toxins such as cardiac glycosides, beta-adrenergic antagonists, calcium channel-blocking agents, propafenone, amiodarone, alpha-2-adrenergic agonists, parasympathomimetic agents, or severe procainamide or quinidine toxicity.
- Transient potentiation of AV block may be seen following administration of atropine, and bradycardic potentiation is longer in duration with subcutaneous administration.
- AV block as a result of disorders of the cardiac conduction system includes:
  - degeneration of the conduction system (AVN, bundle of His, and/or bundle branches) with or without degenerative mitral valve disease
  - inflammation affecting the AVN, atrial approaches to the AVN or Purkinje system, including bacterial endocarditis of the aortic valve and myocarditis (*Borrelia burgdorferi*, *Trypanosoma cruzi*, *Rickettsia rickettsii*, tetanus, lymphocytic-plasmacytic myocarditis)
  - infiltrative diseases (tumors, amyloid)
  - cardiomyopathy
  - congenital AV block associated with structural defects (e.g., AV septal defects) or primary congenital AV block (Pugs, Airedale Terriers)
  - severe systemic hypertension
  - blunt chest trauma
  - pheochromocytoma
  - ischemia or infarction (rare).

Systems Affected

- Cardiovascular system (bradycardia, hypoperfusion, and hypotension if AV block is high-grade second or third degree).
- Neuromuscular system (may be affected secondarily from significant bradycardia and hypoperfusion).
- Renal (azotemia may result from profound or chronic hypoperfusion).

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SIGNALMENT/HISTORY

- Both dogs and cats.
- Breed predilections: American Cocker Spaniels (degenerative AV block), Dachshunds (degenerative AV block), Doberman Pinschers, brachycephalic dogs (vagally mediated AV block), Persian cats (vagally mediated AV block).
- Young, otherwise healthy dogs as a manifestation of high vagal tone.
- Young animals with congenital heart disease.
- Older dogs and cats with degenerative conduction system disease.
- Cats of any age with hypertrophic or restrictive cardiomyopathy.

Risk Factors

- Any condition or intervention that increases vagal tone.

Historical Findings

- Many animals are asymptomatic.
- Syncope or collapse.
- Exercise intolerance.
- Occasionally seizures.
- May have a variety of clinical signs resulting from underlying cause.



CLINICAL FEATURES

Physical Exam Findings

- Normal with first-degree AV block (unless there are also abnormalities resulting from more generalized myocardial disease or noncardiac disease).
- Dogs with profound bradycardia may appear weak.
- Intermittent pauses in the cardiac rhythm with Mobitz type I or type II second-degree AV block.
- Bradycardia if high-grade second-degree or third-degree block.
- Jugular veins may show intermittent cannon A waves with third-degree block.
- First heart sound (S1) may become progressively softer, followed by a pause with Mobitz type I second-degree AV block.
- An audible S4 may be heard unaccompanied by S1 and S2 when block occurs (second- and third-degree AV block).
- A systolic ejection murmur resulting from relative aortic stenosis may be heard in patients with bradycardia.
- Secondary neurologic signs (e.g., seizures, extensor rigidity) may be noted in animals with severe bradycardia.

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- Premature beats and/or paroxysms of tachycardia secondary to myocardial hypoperfusion may be audible in animals with severe bradycardia.
- Other abnormalities reflecting the underlying etiology may be observed.



DIFFERENTIAL DIAGNOSIS

- Sinus node dysfunction (sick sinus syndrome).
- Atrial standstill.
- Accelerated idioventricular rhythm.
- P waves superimposed upon preceding T waves because of first-degree AV block should be differentiated from bifid T waves.
- Nonconducted P waves from supraventricular premature impulses or supraventricular tachycardia should be distinguished from second-degree AV block.



DIAGNOSTICS

- Standard electrocardiography.
- Continuous ECG monitoring or recording (e.g., Holter monitoring, event recording, telemetry) may be required if conduction impairment is intermittent.
- May be necessary to identify specific noncardiac causes of enhanced vagal tone.
- Atropine response test may be used to determine whether AV block is due to vagal tone (administer atropine 0.04 mg/kg IM and repeat ECG in 20–30 minutes).
- Hyperkalemia and hypokalemia may predispose to AV conduction disturbances.
- Abnormal leukogram may be noted in animals with bacterial endocarditis or myocarditis.
- Serum digoxin concentration may be high.
- High T_4 in cats if associated with thyrotoxic myocardial disease.
- Abnormally high arterial blood pressure if associated with severe hypertension or hypertensive cardiac disease.
- Positive *Borrelia*, *Rickettsia*, or *Trypanosoma cruzi* titers if associated with one of these infectious agents.
- Blood cultures may be positive in patients with bacterial endocarditis.
- Plasma cardiac troponin I (cTnI) concentration is increased in a high percentage of dogs with symptomatic AV block and may remain above normal after artificial cardiac pacing, particularly in dogs with endocarditis, myocarditis, or left ventricular dysfunction.
- Electrophysiologic studies are generally unnecessary but can be done to confirm the specific type of AV block if surface ECG findings are equivocal and to identify the specific intracardiac location of the conduction block.
- Doppler/echocardiographic examination may reveal structural heart disease (e.g., endocarditis, neoplasia, cardiomyopathy).

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Pathologic Findings

- No gross or histopathologic cardiac abnormalities if AV block is due to extrinsic causes.
- With cardiac conduction system disorders, findings may vary depending on the underlying cause.
- Older animals with degenerative change of the conduction system may have focal mineralization of the interventricular septal crest visible grossly; chondroid metaplasia of the central fibrous body, and increased fibrous connective tissue in the AV bundle noted histopathologically.



THERAPEUTICS

- The overall objectives of treatment are to eliminate or reverse the underlying cause and to provide a heart rate support when necessary.
- Temporary or permanent cardiac pacing is often the only consistently effective treatment in symptomatic patients or patients with progressive renal dysfunction due to bradyarrhythmia.

Drugs of Choice

- Medications are chosen if appropriate to manage an underlying cardiac or noncardiac condition.
- Atropine (0.02–0.04 mg/kg IV, IM) or glycopyrrolate (0.005–0.01 mg/kg IV, IM) may be used short term if positive atropine response.
- Chronic anticholinergic therapy (propantheline 0.5–2.0 mg/kg PO q8–12h or hyoscyamine 0.003–0.006 mg/kg q8h for symptomatic patients if improved AV conduction documented with atropine response test.
- Isoproterenol (0.04–0.09 µg/kg/min IV to effect) or dopamine (3–5 µg/kg/min IV to effect) may be administered in acute, life-threatening situations to enhance AV conduction and/or accelerate an escape focus.

Precautions/Interactions

- Hypokalemia – increases sensitivity to vagal tone; may potentiate AV conduction delay.
- Drugs with vagomimetic action (e.g., digoxin, bethanechol, physostigmine, pilocarpine) may cause or potentiate AV block.
- Avoid drugs likely to impair impulse conduction further or depress a ventricular escape focus (e.g., procainamide, quinidine, lidocaine, calcium channel-blocking agents, beta-adrenergic antagonists, alpha-2-adrenergic agonists, amiodarone, propafenone).
- Temporary cardiac pacing may induce pacemaker dependency by suppressing an escape focus.
- Avoid the use of isoproterenol or dopamine in patients with digoxin toxicity or ischemic heart disease.

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Diet

- Modifications or restrictions only to manage an underlying condition.

Activity

- Cage rest advised for symptomatic patients.

Surgical Considerations

- Many patients are a high anesthetic risk, and perioperative temporary pacing is advised if anesthesia is needed.
- Permanent artificial cardiac pacemaker may be required for long-term management of symptomatic patients or those with progressive renal dysfunction due to bradyarrhythmia.
- For patients needing a permanent pacemaker, an epicardial system should be considered for feline patients and for those with existing or potential hypercoaguable state, those requiring immunosuppression for a comorbid state, and those with sepsis.



COMMENTS

Client Education

- Explain that treatment is directed toward underlying cause.
- Explain that positive chronotropic agents may not be effective long term.
- Explain that cardiac pacing will not prevent progression of underlying heart disease if present.

Patient Monitoring

- Asymptomatic patients without a pacemaker must be monitored for development of clinical signs, secondary congestive heart failure, and progressive renal dysfunction.
- Monitor ECG of patients with first-degree AV block or low-grade second-degree AV block for development of higher grade conduction block.

Expected Course and Prognosis

- Depends on underlying cause.
- Prognosis for first-degree and Mobitz type I second-degree AV block is usually excellent if no significant underlying disease is present.
- AV block may be reversible if due to medications, transient ischemia, blunt trauma, hypertension, electrolyte disorders, or acute myocarditis of any cause.

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- Conditions that cause necrosis, disruption, or replacement of the cardiac conduction system cells typically result in progressive and permanent AV block (e.g., infarction, surgical disruption, radiofrequency ablation, bacterial endocarditis, neoplasia, amyloidosis, progressive muscular dystrophy, cardiomyopathy).
- With high-grade second-degree or complete AV block, sudden death may occur.

Synonyms

- Mobitz type I second-degree AV block = Wenckebach block
- Third-degree AV block = complete AV block
- PR interval = PQ interval

Abbreviations

AV: atrioventricular
AVN: atrioventricular node
CSF: cerebrospinal fluid
ECG: electrocardiogram
IM: intramuscular
IV: intravenous
PO: per os

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chapter 9

Blood Transfusion Reaction

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DEFINITION/OVERVIEW

- An adverse effect resulting from a blood or blood component transfusion.



ETIOLOGY/PATHOPHYSIOLOGY

- Transfusion reactions are either acute (within 48 hours of administration) or delayed, and can result from immune-mediated or nonimmune-mediated causes. Transfusion reactions can range from mild and subclinical to severe and life-threatening.

Acute Immune-Mediated Reactions

- Acute hemolysis (type II hypersensitivity) occurs when the recipient possesses antibodies against the RBCs in the transfusion. IgM and IgG are able to fix complement and cause intravascular hemolysis. Extravascular hemolysis occurs with removal of antibody-coated RBCs by the lymphoreticular system. Dogs require previous exposure to develop antibodies; cats possess naturally occurring alloantibodies and may develop acute hemolysis with their first transfusion.
- An allergic reaction (type I hypersensitivity) results from antigen binding with the recipient's IgE or IgG. Antigen–antibody interaction causes degranulation of mast cells, release of numerous vasoactive compounds, systemic inflammation, and vasodilation. Donor leukocytes in the transfusion may also degranulate during storage.
- A febrile nonhemolytic transfusion reaction is caused by interaction of antigens on donor leukocytes or platelets with recipient antibodies or by release of pyrogenic cytokines during storage.

Delayed Immune-Mediated Reactions

- Delayed hemolysis occurs when the recipient develops antibodies from exposure to foreign RBC antigens in the transfusion. These antibodies adhere to transfused RBCs and cause extravascular hemolysis.

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Companion website: www.fiveminutevet.com/emergency

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- Posttransfusion purpura is an immune-mediated thrombocytopenia that develops secondary to foreign platelet antigens in a transfusion.

Acute Nonimmune-Mediated Reactions

- Hemolysis of donor RBCs before transfusion is caused by improper storage (i.e., outdated product, inappropriate temperature) or administration (excessive pressure, small needle or catheter, concurrent administration of fluids other than 0.9% saline, rapid infusion, and infusion through some fluid pumps).
- Bacterial contamination from improper collection or storage, donor bacteremia, skin contamination, or contamination during administration may lead to septic shock, DIC, and MODS.
- Volume overload can occur with transfusion at an excessive rate or volume because blood products are colloidal suspensions that primarily contribute to intravascular fluid volume. Hypervolemia increases pulmonary capillary pressure and can lead to pulmonary edema.
- Citrate toxicosis may result from administration of large volumes of whole blood. Citrate anticoagulant causes hypocalcemia by chelation and depletion of the recipient's ionized calcium fraction.
- Hyperammonemia from administration of outdated blood products will cause signs of CNS toxicity and can also cause vomiting.
- Hypothermia can be caused or exacerbated by administration of blood products that are not properly warmed, especially in large-volume transfusions.

Delayed Nonimmune-Mediated Reactions

- Transmission of infectious diseases, including *Babesia* spp., *Leishmania*, *Ehrlichia canis*, FeLV, FIV, and hemotrophic mycoplasma.
- Hemosiderosis may occur with repeated or large transfusions, causing hepatic damage secondary to iron storage.

Systems Affected

- Hematologic/lymphatic/immune: hemolysis.
- Cardiovascular: tachycardia, hypotension, volume overload, dysrhythmias.
- Respiratory: tachypnea, bronchoconstriction, pulmonary edema.
- Gastrointestinal: vomiting, diarrhea.
- Skin: erythema, urticaria, pruritus, purpura, angioneurotic edema.
- Neuromuscular: tremors or tetany with citrate toxicosis.
- Neurologic: encephalopathy.
- Hepatic: hemosiderosis.
- Coagulation: hypothermia-induced coagulopathy, posttransfusion purpura, DIC.

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Incidence/Prevalence

- Reported rates of canine transfusion reactions range from 3.3% to 40%.
- Common: febrile nonhemolytic reactions.
- Rare: posttransfusion purpura, citrate toxicosis, hemosiderosis.

Geographic Distribution

- Cats with type B blood are found more commonly in Europe, Japan, Australia, and the Pacific Northwest portion of the United States.



SIGNALMENT/HISTORY

- Purebred cats, particularly British Shorthair and Devon Rex, have a higher frequency of type B blood, which carries a greater risk of incompatibility because the majority of cats in the United States are type A. Type B cats tend to react strongly to type A blood and can have a fatal reaction within hours of transfusion.

Signs

- Acute hemolysis: hemoglobinemia/hemoglobinuria with intravascular hemolysis, generally milder signs with extravascular hemolysis, vomiting, diarrhea, pyrexia, tachycardia, tachypnea, hypotension, dysrhythmias, seizures.
- Allergic reactions: pyrexia, erythema, urticaria, pruritus, angioneurotic edema, vomiting, diarrhea, and possible anaphylaxis with hypotension, tachycardia, and bronchoconstriction.
- Febrile nonhemolytic reaction: temperature increase of >1 °C, pyrexia may persist for up to 20 hours.
- Delayed hemolysis: typically mild decrease in PCV without hemoglobinuria.
- Posttransfusion purpura: petechiae, ecchymoses, spontaneous hemorrhage, usually 7–14 days post transfusion.
- Nonimmune-mediated hemolysis: typically benign but reduced efficacy of the transfusion.
- Bacterial contamination: pyrexia, vomiting, hemolysis, septic shock.
- Volume overload: tachypnea, moist cough, orthopnea, cyanosis.
- Citrate toxicosis: tremors, tetany, nausea, hypotension, dysrhythmias.
- Hyperammonemia: ataxia, dementia, head pressing, circling, seizures, vomiting.
- Hypothermia: decrease in body temperature, dysrhythmias.

Risk Factors/Causes

- Transfusion carries an inherent risk of reaction.
- Acute hemolysis: previous transfusion or pregnancy.
- Allergic reactions: worse with plasma or platelet transfusions.
- Febrile nonhemolytic reaction: risk increases with storage time of the product.
- Bacterial contamination: platelet products, which may be stored at room temperature.

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- Volume overload: cats, neonates, underlying cardiopulmonary disease, oliguric/anuric renal failure, whole blood transfusions tend to be of a larger volume.
- Citrate toxicosis: hepatic dysfunction, whole blood transfusion, rapid or large-volume transfusion.
- Hyperammonemia: hepatic dysfunction, ammonia levels in blood products increase with storage time.
- Hypothermia: large-volume transfusion, transfusion through a central line.



DIFFERENTIAL DIAGNOSIS

- Although acute transfusion reactions may appear similar clinically, correct diagnosis is necessary for appropriate treatment. If a reaction is suspected, confirm product–recipient compatibility, expiration date, and check the appearance of the product for signs of contamination. The presence of hemoglobinemia or hemoglobinuria strongly suggests hemolysis but may be absent with extravascular hemolysis.



DIAGNOSTICS

Complete Blood Count/Biochemistry/Urinalysis

- PCV: rapid decline with acute hemolysis, mild-to-marked decrease with delayed hemolysis, pink- to red-tinged plasma portion.
- Thrombocytopenia: mild to marked with posttransfusion purpura.
- Hemoglobinemia/hemoglobinuria: acute hemolysis.
- Hyperbilirubinemia: acute or delayed hemolysis.

Other Laboratory Tests

- Coombs' test: positive in delayed hemolytic reactions but may be positive from primary disease.
- Gram stain and culture: if bacterial contamination is suspected.
- Ionized calcium: low with citrate toxicosis.
- Ammonia level: high with hyperammonemia but difficult to test.

Imaging

- Thoracic radiographs: confirm volume overload, patchy interstitial to alveolar lung pattern, can develop anywhere but more common in the perihilar region.

Diagnostic Procedures

- Cross-match: confirm compatibility.
- Blood pressure: hypotension with acute hemolysis or anaphylaxis; hypertension with volume overload.

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- Pulse oximetry: hypoxemia with acute hemolysis, anaphylaxis, and volume overload.
- Central venous pressure: sharp increase with volume overload.
- ECG: bradycardia, Q-T interval prolongation, and VPCs with citrate toxicosis.



THERAPEUTICS

- The transfusion should immediately be discontinued whenever a reaction is suspected and the patient fully evaluated to determine appropriate treatment.
- Acute hemolysis: discontinue transfusion, support systemic blood pressure with intravenous fluids ± vasopressors; oxygen supplementation if hypoxemic; corticosteroids not generally indicated.
- Allergic reactions: discontinue or decrease rate of transfusion, antihistamine ± corticosteroid; epinephrine, oxygen supplementation, intravenous fluid therapy, and vasopressors may be needed with anaphylaxis.
- Febrile nonhemolytic reaction: decrease rate of transfusion, medications usually not necessary but consider antihistamines ± NSAIDs.
- Delayed hemolysis: therapy usually not indicated, additional transfusions may be required with severe anemia.
- Bacterial contamination: discontinue transfusion, broad-spectrum intravenous antibiotic therapy.
- Volume overload: discontinue or decrease rate of transfusion, oxygen supplementation, diuretic therapy.
- Citrate toxicosis: discontinue or decrease rate of transfusion, slow administration of intravenous calcium.

Drugs of Choice

- Allergic reaction: diphenhydramine (2 mg/kg IM) ± a short-acting corticosteroid if signs progress or do not improve, epinephrine (0.01–0.02 mg/kg SQ, IM, or IV) for anaphylaxis.
- Posttransfusion purpura: immunosuppressive dose of corticosteroids in severe cases.
- Volume overload: furosemide (2–4 mg/kg IV, repeat every 30 minutes until clinical signs of orthopnea resolve).
- Citrate toxicosis: calcium gluconate (94–140 mg/kg slowly IV).

Contraindications

- Avoid using corticosteroids or NSAIDs in patients with gastrointestinal hemorrhage.

Precautions/Interactions

- Administration of calcium in the same line with blood products may cause clot or precipitate formation.

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COMMENTS

Patient Monitoring

- Quick recognition allows earlier and more successful treatment of transfusion reactions. Baseline temperature, pulse, respiratory rate, and CRT at baseline and every 15 minutes for the first hour then hourly until transfusion is complete.
- Pre- and posttransfusion PCV.

Prevention/Avoidance

- Critically assess the patient's needs and avoid transfusion if not clinically warranted.
- When taking history, ask about previous transfusion and prominently note transfusion history in patient records.
- Confirm donor–recipient compatibility by typing or cross-matching.
- Choose the appropriate blood product containing only needed components with minimal antigen and volume.
- Do not administer products that are outdated or abnormal in appearance.
- Administer blood products with proper equipment.
- Leukoreduction (removal of leukocytes from a blood product) may reduce the likelihood of a febrile nonhemolytic reaction.
- Bacterial contamination can be prevented by aseptic collection technique, sterile collection equipment, and proper storage.
- Sufficiently warm blood products with an approved warming device.
- Thorough screening of donors for potential infectious diseases.
- Prophylactic administration of antihistamines or glucocorticoids is controversial but not supported by scientific evidence and is not recommended at this time.

Abbreviations

CNS: central nervous system
CRT: capillary refill time
DIC: disseminated intravascular coagulation
ECG: electrocardiogram
FeLV: feline leukemia virus
FIV: feline immunodeficiency virus
IgE: immunoglobulin E
IgG: immunoglobulin G
IgM: immunoglobulin M
IM: intramuscular
IV: intravenous
MODS: multiorgan dysfunction syndrome
NSAID: nonsteroidal antiinflammatory drug

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PCV: packed cell volume
RBC: red blood cell
SQ: subcutaneously
VPC: ventricular premature contraction

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chapter 10

Brachial Plexus Injury



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DEFINITION/OVERVIEW

- Traumatic brachial plexus injury (TBPI) is a catch-all term referring to traumatically induced neuropathy of the C6–T2 ventral spinal nerve rami.

ETIOLOGY/PATHOPHYSIOLOGY

- The brachial plexus originates from the ventral rami of the C6–T2 spinal nerves.
- Its projections comprise the suprascapular (C6–C7), subscapular (C6–C7), musculocutaneous (C6–C8), axillary (C6–C8), radial (C6–T2), median (C8–T2), ulnar (C8–T2), and lateral thoracic (C8–T1) nerves.
- Thoracic limb traction or abduction can result in injury to the brachial plexus or associated structures.
- Injury can lead to neuropraxia (focal demyelination without axonal disruption), axonotomesis (axonal injury with sparing of the endoneureum), or neurototomesis (complete severance of axons and ensheathing tissues).
- When neurototomesis occurs, it is often at the level of the nerve rootlets, resulting in brachial plexus avulsion.

SIGNALMENT/HISTORY

- Young, male, large-breed dogs with unsupervised outdoor access are likely predisposed.

CLINICAL FEATURES

- TBPI can be classified as complete, cranial (C6–C7), or caudal (C8–T2).
- Ventral rootlets and associated motor fibers are most susceptible to TBPI, although sensory projections can be affected in severe injuries.

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Cranial TBPI

- Lesions associated with the cranial brachial plexus will result in difficulty advancing the limb at the shoulder (suprascapular nerve), shoulder instability (suprascapular, subscapular nerves), and weak elbow flexion (musculocutaneous nerve).
- Animals with cranial TBPI usually have the ability to bear weight and extend the elbow as the majority of the radial distribution will be spared.
- Postural reactions may be weak, although sparing of the knuckling response is common.
- The extensor carpi radialis and triceps reflexes are likely to be preserved, whereas the biceps and flexor withdrawal reflexes may be weak.
- In severe cranial TBPI cutaneous hypalgesia or analgesia may be present. Avulsion of the C6–C8 nerve roots leads to analgesia in the autonomous zone of the axillary nerve (lateral brachium), musculocutaneous nerve (craniomedial antebrachium), and portions of the radial nerve (cranial antebrachium, dorsum of paw) (Fig. 10.1).

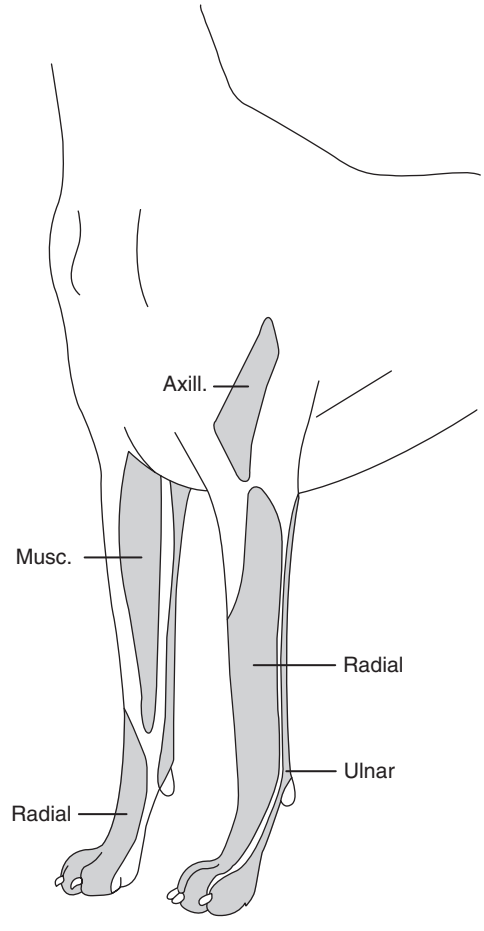


Fig. 10.1. The major autonomous zones of the thoracic limb of the dog.

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Caudal TBPI

- Caudal TBPI will lead to an inability to bear weight on the thoracic limb and loss of carpal extension.
- Proximally located lesions involving the ventral rootlets, ventral roots, or spinal nerves associated with T1–T2 may lead to ipsilateral Horner's syndrome.
- Lesions involving C8–T1 (lateral thoracic nerve) may lead to ipsilateral loss of the cutaneous trunci reflex.
- Postural reactions, such as hopping or knuckling, will be weak or absent depending on the severity of caudal TBPI.
- Reflexes, including the flexor withdrawal, extensor carpi radialis, and triceps, will be weak or absent.
- Hypalgesia or analgesia may be present in the autonomous zones of the ulnar (caudal antebrachium) and radial (cranial antebrachium, dorsum of paw) with C8–T2 TBPI.

Complete TBPI

- Complete TBPI has the features of both cranial and caudal TBPI, with analgesia to the entire limb distal to the elbow occurring with severe lesions.
- In some cases of proximally located complete TBPI, postural reaction deficits, upper motor neuron paresis, and ataxia may be present in the ipsilateral pelvic limb due to hematoma compressing the spinal cord.



DIFFERENTIAL DIAGNOSIS

- Other differential diagnoses for acute-onset monoparesis or monoplegia of a thoracic limb in dogs include:
  - Fibrocartilagenous embolic myelopathy (FCE)
  - Lateralized disk herniation
  - Brachial plexus neuritis (can be bilateral)
  - Infectious neuritis
  - Neoplasia
  - Vascular neuromyopathy (brachial artery occlusion).



DIAGNOSTICS

- Frequently TBPI is diagnosed based on clinical signs and a history of trauma.
- All animals should receive a careful physical examination, with special attention paid to intrathoracic, intraabdominal, orthopedic, and neurologic structures that may be concurrently injured.
- Animals with a history of significant trauma should have a complete blood count, serum biochemistry profile, urinalysis, chest radiograph series, and focused assessment using sonography for trauma of the thorax (TFAST) and abdomen (AFAST).

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- Those patients with discrete abnormalities in any particular system should have those investigated appropriately.
- Advanced imaging, such as CT or MRI, of the brachial plexus, spinal nerves, nerve rootlets, and spinal cord can help to exclude other etiologies.
- Electrophysiology, which can include electromyography (EMG), motor and sensory nerve conduction velocity, and cord dorsum potentials, can be performed.
- The value of electrophysiology is that it can identify specific nerve and muscle groups involved as well as separate neuropraxic injuries from those that involve the axon and may have a poorer prognosis.
- Electrophysiology is usually performed at least 8 days after injury as this is the time, in dogs with axonal damage, that it will take to develop abnormal spontaneous activity on EMG.
- Electrophysiology does not discern the etiology responsible for neuropathy.



THERAPEUTICS

- Animals with injuries to other body systems should have those addressed.
- Treatment of TBPI has traditionally consisted of physical rehabilitation and analgesia.
- Passive range of motion exercises, active weight bearing, electrical muscle stimulation, and underwater treadmill may all be of benefit.
- In the acute phase of TBPI, opioid analgesia and nonsteroidal antiinflammatory agents may be required.
- Some animals may develop neuropathic pain in the chronic phases of recovery, manifest by licking or chewing the limb. These animals may require agents such as gabapentin or tricyclic antidepressants.
- Steroids are of unknown value in the acute or chronic phases of TBPI and are not utilized by the author.
- Animals with absent nociception often do not recover function of the limb and may need limb amputation.
- Recently, techniques for nerve transposition and reimplantation have been developed that may improve functional recovery in animals with brachial plexus avulsion. Unfortunately, few veterinary surgeons or neurologists in the USA are able to perform these procedures.

Drugs of choice

Opioids

- Buprenorphine 0.005–0.02 mg/kg IV, IM or SQ BID-TID
- Fentanyl 2–5 µg/kg IV bolus; continue as CRI at 3–5 µg/kg/h
- Tramadol 3–5 mg/kg PO TID-QID
- CR morphine 1–5 mg/kg PO BID

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Nonsteroidal antiinflammatory drugs (NSAIDs)

- Carprofen 2.2 mg/kg PO BID
- Deracoxib (follow manufacturer's instructions)
- Meloxicam 0.2 mg/kg PO q24h

Drugs for neuropathic pain

- Amantadine 3–5 mg/kg PO q24h
- Amitriptyline 1–2 mg/kg PO BID
- Gabapentin 3–5 mg/kg PO TID as starting dose, with a maximum up to 50 mg/kg/day



COMMENTS

Client Education

- Recovery of function in distributions that lack nociception is unlikely.
- Physical rehabilitation may speed and enhance neurologic improvement.
- Some animals with TBPI will have chronic neuropathic pain.
- Those animals with neuropathic pain that self-mutilate or animals that have abrasions from dragging a limb will need to have wounds addressed by a veterinarian.
- Amputation is sometimes necessary if wounds cannot be medically managed.

Patient Monitoring

- Many animals with TBPI will have other body systems or regions of the nervous system that may be injured. Careful physical examination is a must.

Possible Complications

- Animals with neuropathic pain may self-mutilate.
- Wound infection.

Expected Course and Prognosis

- Prognosis is dependent on nociceptive status and whether lesions are complete or incomplete.
- Animals with absent nociception will be unlikely to recovery function in affected distributions.
- Animals with complete TBPI are likely to have a more protracted recovery than those with incomplete lesions.

Synonyms

- Brachial plexus avulsion is equivalent to complete TBPI.

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Abbreviations

AFAST: abdomen: focused assessment using sonography for trauma
BID: twice a day (bis in die)
CR: controlled release
CRI: continuous rate infusion
CT: computed tomography
EMG: electromyography
FCE: fibrocartilagenous embolic myelopathy
IM: intramuscular
IV: intravenous
MRI: magnetic resonance imaging
PO: by mouth (per os)
QID: four times a day (quater in die)
SQ: subcutaneous
TBPI: traumatic brachial plexus injury
TID: three times a day (ter in die)
TFAST: thorax: focused assessment using sonography for trauma

Suggested Reading

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Author: Jonathan M. Levine, DVM

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chapter 11

Bromethalin Rodenticide Toxicity



DEFINITION/OVERVIEW

- Bromethalin is a neurotoxic rodenticide that causes cerebral edema. It is not an anticoagulant rodenticide and should not be treated with vitamin K_1 as an antidote.
- Ingestion of toxic levels of bromethalin can result in central nervous system (CNS) signs such as CNS stimulation or depression, abnormal behavior, hyperexcitability, ataxia, hyperesthesia, tremors with secondary hyperthermia, seizures, and coma.
- Other common signs include paresis, hindlimb paralysis, anisocoria, nystagmus, and changes in the pupillary light reflex (PLR). Rarely, decreased or absent conscious proprioception, increased patella reflexes, coma, and death can be seen.



ETIOLOGY/PATHOPHYSIOLOGY

- Due to new EPA mandates, many anticoagulant rodenticides will be replaced by cholecalciferol and bromethalin (Fig. 11.1).
- Bromethalin works by uncoupling oxidative phosphorylation, resulting in decreased ATP production. This causes dysfunction to the ATP-dependent sodium and potassium ion channel pumps, resulting in brain electrolyte imbalances and sodium accumulation within the cell. As a result, lipid peroxidation occurs, leading to cerebral edema.
- In dogs, the LD_{50} of bromethalin is 2.4–5.6 mg/kg, with clinical signs occurring in dogs at approximately 1 mg/kg. Cats are more sensitive to the effects of bromethalin, with a significantly lower LD_{50} of 0.4–0.71 mg/kg. The minimum toxic dose reported in cats is approximately 0.3 mg/kg.
- Bromethalin is rapidly absorbed from the GIT, and is highly lipophilic.
- Bromethalin reaches peak plasma concentrations within 4 hours of ingestion, yet has a long half-life (rats: 5–6 days).
- Bromethalin undergoes enterohepatic recirculation.
- Bromethalin is typically sold as a 0.01% or 0.025% concentration. 0.01% baits contain 2.84 mg of bromethalin per ounce of bait (0.1 mg/g).
- Clinical signs are dose dependent, and the onset of clinical signs depends on the amount ingested. Typically, with acute ingestion, signs may be seen within 2–24 hours.

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■ **Fig. 11.1.** A common brand of rodenticide containing bromethalin.

Systems Affected

- Nervous/neuromuscular: CNS signs may be seen with bromethalin toxicosis, depending on the dose ingested. Both acute and delayed clinical signs have been reported.
- Ophthalmic: nystagmus, anisocoria.
- Gastrointestinal: vague GI signs can be seen.
- Respiratory: rarely, death can occur due to respiratory depression or secondary to aspiration pneumonia.



SIGNALMENT/HISTORY

Risk Factors

- Puppies and younger dogs appear to be overrepresented with poisoning due to their curious nature.
- Cats are more sensitive to the effects of bromethalin, with a significantly lower toxic dose and LD_{50}.

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Historical Findings

- Evidence of a tampered or chewed rodenticide container or bait.
- History of having green, blue, or purple bait (multiple colors available) in the feces several days prior.
- Clinical signs consistent with bromethalin.



CLINICAL FEATURES

- Nervous/neuromuscular: Agitation, sedation or decreased mentation, altered behavior, hyperexcitability, ataxia, hyperesthesia, tremors with secondary hyperthermia, paresis, hindlimb paralysis, seizures, and coma may be seen. Rarely, decreased or absent conscious proprioception, increased patella reflexes, coma, and death can be seen.
- Ophthalmic: anisocoria, abnormal PLR, nystagmus.
- Gastrointestinal: vague signs of anorexia, vomiting, and abdominal distension may be seen.
- Respiratory: rarely, respiratory depression, hypercapnia, respiratory arrest.



DIFFERENTIAL DIAGNOSIS

- In patients presenting with CNS signs, other differential diagnoses include toxicants (e.g., marijuana, prescription medications (SSRI antidepressants, amphetamines, baclofen, sleep aids, etc.), metaldehyde, lead, ethylene glycol, alcohol, etc.), structural (e.g., brain tumor, hydrocephalus), inflammatory (e.g., GME, meningitis, etc.), and infectious (e.g., *Toxoplasma*, *Neospora*, canine distemper, rabies, etc.) diseases.



DIAGNOSTICS

- No clinicopathologic changes are typically observed with bromethalin. That said, the author has noticed an increased prevalence of hypernatremia with this toxicant; this is likely due to multiple-dose charcoal administration.
    - Patients should be monitored for signs of neurotoxicity – if clinical signs develop, a serum sodium level should be checked immediately to rule out hypernatremia from activated charcoal administration. Depending on what the sodium level is, treatment for either hypernatremia or cerebral edema should be implemented (see Therapeutics).
- Hospitalized patients should have a daily PCV/TS/BG/AZO and electrolyte panel performed. Ideally, fluid therapy goals should be directed towards achieving hemodilution (e.g., PCV 35%, TS 5 g/dL).

Pathologic Findings

- Cerebral edema may be seen grossly.
- Spongy degeneration and diffuse vacuolization in the CNS white matter, along with intramyelinic edema and microgliosis, may be seen.

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THERAPEUTICS

- The mainstay therapy for bromethalin rodenticide toxicosis is early decontamination, fluid therapy, prevention of cerebral edema, appropriate monitoring, and symptomatic supportive care.
- Decontamination.
  - Decontamination should include early emesis induction, if ingestion was recent and the patient is asymptomatic.
  - With recent ingestion in an asymptomatic patient, emesis induction and administration of one dose of activated charcoal (1–5 g/kg, PO) with a cathartic (e.g., sorbitol) is warranted.
  - As bromethalin undergoes enterohepatic recirculation, the administration of multiple doses of activated charcoal (without a cathartic) is warranted (1–2 g/kg PO q6h × 24 hours).
- Fluid therapy.
  - A balanced, isotonic crystalloid at 2.5–3× maintenance should be used to hydrate and perfuse the patient. This will also help prevent iatrogenic hypernatremia secondary to activated charcoal. In patients with CNS signs, the use of 0.9% saline or hypertonic saline should be considered if necessary for treatment of cerebral edema. Note: Fluid therapy will not aid in urinary elimination of bromethalin.
  - Patients should be monitored for volume overload.
  - Goals of fluid therapy should be based on physical examination findings (e.g., moist mucous membranes, weight gain, etc.) and evidence of hemodilution (e.g., PCV/TS 35%/5 g/dL) and isosthenuria.
- Gastrointestinal support.
  - Potent antiemetics should be used for patient comfort, to prevent aspiration pneumonia, to allow for return to oral water, and to help prevent secondary complications from activated charcoal administration (e.g., emesis, aspiration of charcoal, hypernatremia, etc.).
- Neurologic support.
  - If CNS signs are seen, goals should be directed at lowering intracranial pressure (ICP). Treatment includes head elevation (15–30°), oxygen therapy, fluid therapy (to increase mean arterial pressure), muscle relaxants (for tremors), anticonvulsants (for seizures), and hyperosmolar agents (for cerebral edema).

Drugs of Choice

- Antiemetics:
  - Maropitant: 1 mg/kg SQ q24h
  - Ondansetron: 0.1–0.5 mg/kg IV q12h
  - Dolasetron: 0.6 mg/kg IV q24h
- Muscle relaxants (for tremors):
  - Methocarbamol: 55–220 mg/kg IV, to effect PRN

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- Anticonvulsants (for seizures):
  - Diazepam: 0.25–0.5 mg/kg, IV, to effect PRN
  - Phenobarbital: 4–20 mg/kg, IV, to effect PRN
  - Levetiracetam (Keppra®): 20–60 mg/kg, IV, to effect PRN
- Hyperosmolar agents (for cerebral edema):
  - Mannitol: 0.5–2 g/kg, IV, slow over 20–30 minutes; repeat PRN. Note: IV fluid should be discontinued during mannitol administration.

Precautions/Interactions

- The use of glucocorticoids has been previously cited for treatment of bromethalin-induced cerebral edema; however, this is not currently recommended based on newer evidence and known potential side effects of steroids (e.g., hyperglycemia).

Alternative Drugs

- At this time, the extra-label, experimental use of intravenous lipid emulsion (as an antidote for fat-soluble toxicants) is not recommended for bromethalin toxicosis.

Appropriate Healthcare

Monitoring

- Patients should be monitored for signs of neurotoxicity – if clinical signs develop, a serum sodium level should be checked immediately to rule out hypernatremia from activated charcoal administration. If sodium levels are normal, treatment for cerebral edema should be implemented.
- In severely affected patients (e.g., obtunded, comatose, etc.), ECG and blood pressure monitoring is imperative. Rarely, "Cushing's reflex" (e.g., acute bradycardia with concurrent severe hypertension) can be seen with severe cerebral edema; this indicates imminent herniation and prompt therapy for cerebral edema (e.g., mannitol) is warranted.

Nursing Care

- Appropriate patient care includes elevating the head at 15–30° if necessary, flipping the patient every 4–6 hours (e.g., to prevent secondary atelectasis), ophthalmic lubrication, and keeping the patient dry and clean.

COMMENTS

Client Education/Prevention/Avoidance

- Owners should be educated to appropriately pet-proof the environment. Education on crate training is imperative.
- Pet owners should be educated to never use rodenticides in a household containing pets, due to the narrow margin of safety.

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Owners and veterinary professionals should be advised to call veterinary-specific poison control centers for consultation with a veterinary toxicologist for life-saving advice as needed.

Possible Complications

- While rare, severe complications can be seen from refractory seizures including rhabdomyolysis, myoglobinuria, and DIC.
- While rare, neurologic signs can be seen for a delayed period of time.
- Rare reports of delayed CNS signs can be seen with subacute or chronic ingestions. Rarely, pet owners should be warned about the risk of clinical signs developing days after initial exposure, despite hospitalization for initial care.

Expected Course and Prognosis

- Hospitalization for the typical, uncomplicated, asymptomatic bromethalin toxicosis patient typically requires 12–18 hours of treatment for decontamination and supportive care. If no clinical signs are seen after this time, patients can be discharged with instructions to monitor carefully at home.
- Overall, the prognosis for bromethalin toxicosis varies depending on the amount ingested and the severity of clinical signs; however, prognosis is generally fair to excellent with appropriate decontamination and treatment prior to the development of clinical signs. If persistent seizures or paralytic syndrome are seen, the prognosis is poorer.

Synonyms

- Assault®
- Tomcat Mole Killer®
- Talpirid®
- Real Kill®
- Rampage®
- Clout All Weather Bait®
- Fastrac®
- Vengeance®

Abbreviations

ATP: adenosine triphosphate
AZO: blood urea nitrogen
BG: blood glucose
CBC: complete blood count
CNS: central nervous system
ECG: electrocardiogram
EPA: Environmental Protection Agency
GI: gastrointestinal

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GIT: gastrointestinal tract
GME: granulomatous meningioencephalitis
ICP: intracranial pressure
ILE: intravenous lipid emulsion
IM: intramuscular
IV: intravenous
PCV: packed cell volume
PLR: pupillary light response
PO: by mouth (per os)
PRN: as needed (pro re nata)
SQ: subcutaneous
TS: total solids

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Author: Justine A. Lee, DVM

chapter 12

Bundle Branch Block – Left

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DEFINITION/OVERVIEW

- Conduction delay or block in both the left posterior and left anterior fascicles of the left bundle.
- A supraventricular impulse activates the right ventricle first through the right bundle branch; the left ventricle is activated late, causing the QRS to become wide and bizarre.

Electrocardiogram Features

- QRS prolonged; dogs, >0.08 s, cats, >0.06 s.
- QRS wide and positive in leads I, II, III, and aVF.
- Block can be intermittent or constant.

ETIOLOGY/PATHOPHYSIOLOGY

- Because the left bundle branch is thick and extensive, the lesion causing the block must be large.
- Usually an incidental ECG finding – does not cause hemodynamic abnormalities.

Risk Factors/Causes

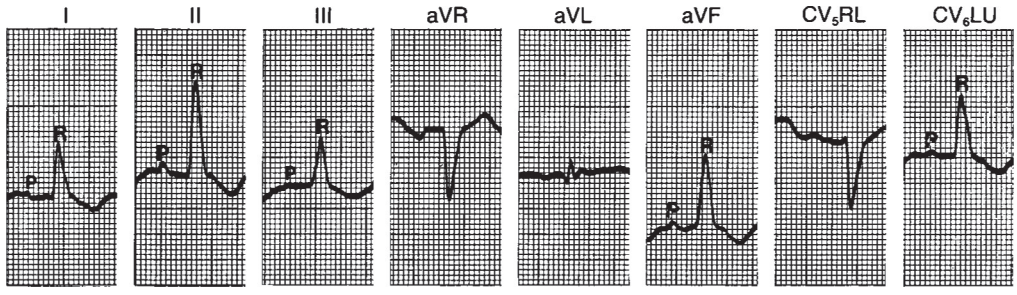
- Cardiomyopathy
- Direct or indirect cardiac trauma (e.g., hit by car, cardiac needle puncture)
- Neoplasia
- Subvalvular aortic stenosis
- Fibrosis
- Ischemic cardiomyopathy (e.g., arteriosclerosis of the coronary arteries, myocardial infarction, and myocardial hypertrophy that obstructs coronary arteries)

Systems Affected

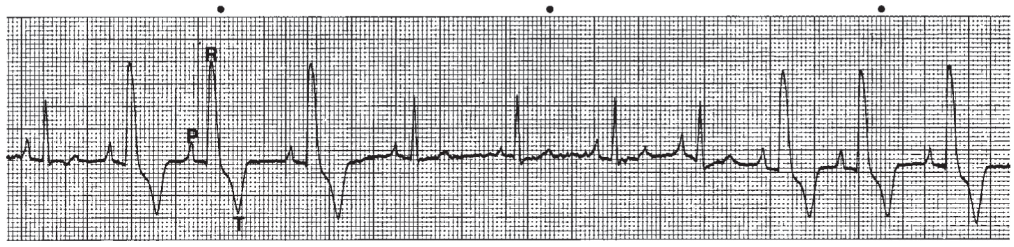
- Cardiovascular (Figs 12.1 and 12.2)

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■ **Fig. 12.1.** Left bundle branch block in a cat with hypertrophic cardiomyopathy. The QRS complex is of 0.07 s duration and is positive in leads I, II, III, and aVF. Neither a Q wave nor an S wave occurs in these leads. The QRS complex is inverted in lead aVR. Source: Tilley 1992, reproduced with permission of John Wiley and Sons Inc.



■ **Fig. 12.2.** Intermittent left bundle branch block in a Chihuahua. QRS complexes are wider (0.07–0.08 s) in the second, third, and fourth complexes and in the last three complexes. Consistent PR interval confirms a sinus origin for the abnormal-appearing QRS complexes (lead II, 50 mm/s, 1 cm 5 1 mV). Source: Tilley 1992, reproduced with permission of John Wiley and Sons Inc.

SIGNALMENT/HISTORY

Species

- Cats and dogs.

Historical Findings

- Usually an incidental ECG finding; does not cause hemodynamic abnormalities.
- Signs usually associated with the underlying condition.

CLINICAL FEATURES

- Does not cause signs or hemodynamic compromise.

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DIFFERENTIAL DIAGNOSIS

- Left ventricular enlargement.
- No left ventricular enlargement on thoracic radiograph or cardiac ultrasound studies supports diagnosis of isolated left bundle branch block.
- Can also be confused with ventricular ectopic beats, but the PR interval is usually constant and left bundle branch block has no pulse deficits.



DIAGNOSTICS

Imaging

- Echocardiography may reveal structural heart disease; absence of left-sided heart enlargement supports a diagnosis of left bundle branch block.
- Thoracic and abdominal radiographs may show masses or pulmonary metastatic lesions.
- Traumatic injuries could result in localized or diffuse pulmonary densities.

Diagnostic Procedures

- Electrocardiography.
- Long-term ambulatory monitoring (Holter) may reveal intermittent left bundle branch block.

Pathologic Findings

- Possible lesions or scarring on endocardial surface in the path of the bundle branches; applying Lugol's iodine to the endocardial surface within 2 hours postmortem enables clear visualization of the conduction system.



THERAPEUTICS

Drugs of Choice

- None, unless required for management of underlying condition.

Diet

- No modifications unless required for management of underlying condition.

Activity

- Unrestricted unless required for management of underlying condition.

Appropriate Healthcare

- Directed toward the underlying cause.

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Nursing Care

- Generally not necessary.

COMMENTS

Client Education

- Left bundle branch block per se does not cause hemodynamic abnormalities.
- Lesion causing the block could progress, leading to more serious arrhythmias or complete heart block.

Patient Monitoring

- Serial ECG may show clearing or progression to complete heart block.

Possible Complications

- Causative lesion could progress, leading to a more serious arrhythmia or complete heart block.
- First- or second-degree AV block may indicate involvement of the right bundle branch.

Expected Course and Prognosis

- No hemodynamic compromise.

Abbreviations

AV: atrioventricular
ECG: electrocardiogram

See Also

Atrioventricular Block

Suggested Reading

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Author: Larry P. Tilley, DVM

chapter 13

Bundle Branch Block – Right

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DEFINITION/OVERVIEW

- Conduction delay or block in the right bundle branch resulting in late activation of the right ventricle.
- The block can be complete or incomplete.

Electrocardiogram Features

- A right axis deviation and wide QRS (≥0.08 s in dogs; ≥0.06 s in cats) in most patients.
- Large, wide S waves in leads I, II, III, and aVF (Figs 13.1, 13.2).

ETIOLOGY/PATHOPHYSIOLOGY

- The right bundle branch is anatomically vulnerable to injury because it is a thin strand of tissue and has a long undivided course.
- No hemodynamic compromise.

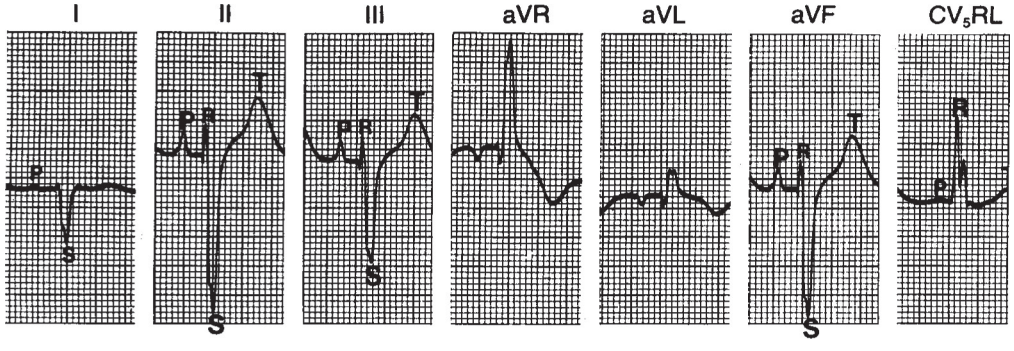


Fig. 13.1. Right bundle branch block in a dog. The electrocardiographic features include QRS duration of 0.08 s; positive QRS complex in aVR, aVL, and CV_5RL (M shaped); and large wide S waves in leads I, II, III, and aVF. There is a right axis deviation (approximately –110°) (50 mm/s, 1 cm 5 1 mV). Source: Tilley 1992, reproduced with permission of John Wiley and Sons Inc.

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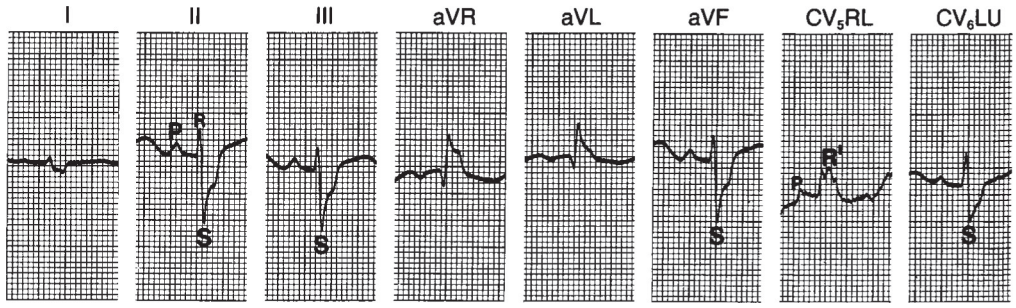


Fig. 13.2. Right bundle branch block in a cat with the dilated form of cardiomyopathy. The QRS duration is 0.08 s (4 boxes). Large and wide S waves are present in leads I, II, III, aVF, and CV_6LU. The QRS in CV_5RL has a wide R wave (M shaped). There is a marked axis deviation (approximately –90°). Source: Tilley 1992, reproduced with permission of John Wiley and Sons Inc.

Risk Factors/Causes

- Occasionally seen in normal and healthy dogs and cats
- Congenital heart disease
- Chronic valvular fibrosis
- After surgical correction of a cardiac defect
- Trauma caused by cardiac needle puncture to obtain blood sample
- Trauma from other causes
- Chronic infection with *Trypanosoma cruzi* (Chagas' disease)
- Neoplasia
- Heartworm disease
- Acute thromboembolism
- Cardiomyopathy
- Hyperkalemia (most commonly in cats with urethral obstruction)

Systems Affected

- Cardiovascular



SIGNALMENT/HISTORY

Species

- Dogs and cats.

Breed Predilections

- In beagles, incomplete right bundle branch block can result from a genetically determined localized variation in right ventricular wall thickness.

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Historical Findings

- Usually an incidental ECG finding – does not cause hemodynamic abnormalities.
- Observed signs are usually associated with the underlying condition.



CLINICAL FEATURES

Physical Examination Findings

- Splitting of heart sounds because of asynchronous activation of ventricles in some patients.
- Does not cause signs of hemodynamic compromise.



DIFFERENTIAL DIAGNOSIS

- Right ventricular enlargement – absence of right ventricular enlargement on thoracic radiographs or echocardiogram supports a diagnosis of right bundle branch block.
- It can also be confused with ventricular ectopic beats (especially if the block is intermittent), but PR intervals are consistent and no pulse deficits with right bundle branch block.



DIAGNOSTICS

Complete Blood Count/Biochemistry/Urinalysis

- None specific.
- Serum potassium may be extremely high in cats with urethral obstruction.

Other Laboratory Tests

- Occult heartworm test may be positive in dogs or cats.
- Chagas' indirect fluorescent antibody test, direct hemagglutination, and complement fixation test may be positive in dogs.

Imaging

- Echocardiogram may show structural heart disease; absence of right-sided heart enlargement supports the diagnosis.
- Thoracic and abdominal radiographs may show masses or pulmonary metastatic lesions; traumatic injuries could cause localized or diffuse pulmonary densities.

Diagnostic Procedures

- Electrocardiography
- Echocardiography

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Pathologic Findings

- Possible lesions or scarring on endocardial surface in the path of the bundle branches; applying Lugol's iodine to the endocardial surface within 2 hours postmortem gives clear visualization of the conduction system.



THERAPEUTICS

Drugs of Choice

- None, unless needed to manage underlying condition.

Diet

- No modifications unless required to manage underlying condition.

Appropriate Healthcare

- Direct treatment toward the underlying cause.



COMMENTS

Client Education

- Does not cause hemodynamic abnormalities itself.
- The lesion causing the block could progress, leading to more serious arrhythmias or complete heart block.

Patient Monitoring

- Serial ECG may show resolution of the lesion or progression to complete heart block.

Possible Complications

- The causative lesion could progress, leading to a more serious dysrhythmia or complete heart block.
- First- or second-degree AV block may indicate involvement of the left bundle branch.

Expected Course and Prognosis

- No hemodynamic compromise.

Abbreviations

AV: atrioventricular
ECG: electrocardiogram

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Author: Larry P. Tilley, DVM

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chapter 14

Canine Distemper



DEFINITION/OVERVIEW

- Canine distemper virus (CDV) infection is among the most serious, contagious, systemic infections affecting dogs worldwide.
- In nature, the virus is found in a multitude of species. Because of its presence through many species, opportunity for eradication is minimal.
- Vaccination is highly effective and recommended for all dogs, beginning at 6–8 weeks of age.



ETIOLOGY/PATHOPHYSIOLOGY

- Caused by an RNA virus of the genus *Morbillivirus*.
- Found in a wide range of carnivores; dogs serve as the principal reservoir for CDV and may be an important source of virus transmitted to wildlife.
- Viral shedding begins about 7 days post infection and may be sustained for up to 60–90 days.
- Transmission occurs most commonly through infectious respiratory secretions but can be found in most tissues and other secretions of infected animals, including urine.
- Spontaneous infections are most likely to occur in puppies between 3 and 6 months of age.
- Subclinical infection is relatively common; therefore the prevalence of infection is higher than that of clinical disease. As a result, risk for infection in highest among puppies that (a) have contact with other young dogs, (b) do not receive an initial vaccine series, and (c) are colostrum deprived (i.e., fail to receive maternally derived antibody). (NOTE: protection afforded by maternally derived canine distemper antibody persists until about 9–12 weeks of age in puppies with a normal nursing history. Depending on the antibody titer of the bitch, the duration of protective immunity may be more or less than expected.)
- CDV can be isolated from most tissues/fluids of infected dogs, although epithelium and CNS tissues are preferentially targeted.

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- Virus is widely disseminated throughout lymphoid tissues by 6 days post infection; virus shedding occurs by 14 days post infection, even in dogs with subclinical infection.
- Clinical consequences of CDV infection are largely defined by the degree and type of immune response the patient is able to mount.
- By 9–14 days post infection, dogs that mount a poor immune response experience widespread dissemination of virus that involves the intestinal epithelium, exocrine and endocrine glands, respiratory tract, genitourinary tract, and even skin.
- Acute CDV encephalitis can follow hematogenous spread of virus into the CNS and is most likely to occur in young or immunosuppressed dogs.
- Acute CDV encephalitis is associated with a poor prognosis for recovery.
- CDV encephalitis also occurs as a subacute or chronic infection and is associated with reduced viral replication in the CNS, a relatively strong humoral immune response, and lack of lymphoid cell depletion.
- The ability of virus to persist in the CNS may be associated with what is now described as an uncommon consequence of infection, "old dog encephalitis" (ODE) or "old dog distemper."
- Old dog encephalitis is a noncontagious variant of CDV infection that is associated with progressive inflammatory disease involving gray matter of the cerebral hemispheres and brainstem.
- Minor genetic variations of CDV have been recognized that may explain the variation in virulence seen in dogs. For example, the Snyder Hill strain of CDV is neurotropic and causes polioencephalomyelitis. Despite this variation, CDV isolates remain serologically homogeneous.



SIGNALMENT/HISTORY

- There is no age, breed, or gender predilection for CDV infection following exposure.
- Most infections do occur in puppies, particularly those that have not nursed, have an inadequate vaccination history, or have been group-housed with other dogs (e.g., shelters or pet stores).
- Although transmission typically occurs following exposure to infectious respiratory secretions, neonatal infections can result following transplacental transmission of CDV.
- Affected puppies may be aborted, stillborn, or weak at birth.
- Neurologic signs develop in affected neonates following transplacental exposure between 4 and 6 weeks of age, despite mild to inapparent signs in the bitch.

Historical Findings

- Owners of puppies with clinical signs caused by CDV report rapid onset of deterioration (days) in a previously healthy dog.
- Clinical signs of CDV can include lethargy, loss of appetite, vomiting, diarrhea, and thick (purulent) nasal/ocular discharge.

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CLINICAL FEATURES

- Clinical signs of CDV infection are seldom pathognomonic during initial physical examination.
- Systemic signs range from mild (lethargy) to severe (seizures).
- The spectrum of acute-onset clinical signs includes fever, dehydration, purulent nasal and ocular discharge (Fig. 14.1), cough, respiratory distress, conjunctivitis, vomiting, and diarrhea, which may be liquid in consistency and may contain frank blood or mucus.
- Dogs that survive acute systemic disease can develop a variety of clinical signs.
- Skin lesions include pustular dermatitis in puppies, nasal and digital ("hard pad") hyperkeratosis, and juvenile cellulitis.
- Neurologic signs may develop as early as 1–3 weeks following recovery from systemic illness; onset of neurologic signs may be delayed for several weeks or months.
- Depending on the area of the CNS affected, signs include hyperesthesia or cervical rigidity (meningitis), seizures, vestibular signs, ataxia, paresis, and myoclonus (involuntary muscle twitching).
- Bone lesions have been associated with CDV infection in large-breed dogs between 3 and 6 months of age. Long bone lesions consistent with HOD have been associated with CDV infection and with administration of modified-live virus vaccine.
- Ocular lesions associated with CDV infection may develop after recovery from acute systemic disease and include sudden blindness and persistent mydriasis (optic neuritis), retinal detachment, inactive, circumscribed areas of retinal hyperreflectivity ("gold medallion lesions"), and keratoconjunctivitis sicca ("dry eye").



Fig. 14.1. Conjunctivitis, rhinitis, and facial dermatitis in a 2-year-old, unvaccinated, moribund dog at presentation; acute canine distemper was confirmed.

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Fig. 14.2. Enamel hypoplasia in a young dog that recovered from canine distemper virus infection.

- Enamel hypoplasia, occasionally seen in adult dogs, is evidence of prior CDV infection (Fig. 14.2).

DIFFERENTIAL DIAGNOSIS

- Because of the wide spectrum of tissues affected, dogs with CDV infection are difficult to identify on the basis of clinical signs alone.
- Canine infectious tracheobronchitis is a common differential diagnosis for CDV infection in dogs of any age, particularly in nonvaccinates.
- Because of the prevalence of neurologic signs associated with CDV infection and the predilection for disease to develop in nonvaccinated puppies, rabies must be considered a principal differential diagnosis.

DIAGNOSTICS

- Clinical signs of systemic illness consistent with distemper can lead to a presumptive diagnosis in puppies 3–6 months of age.
- Confirmation of CDV infection is more problematic, especially in adult dogs.
- Routine hematology and biochemistry panels may reflect the degree of systemic illness but are not diagnostic for CDV infection.
- Examination of peripheral blood smears or exfoliative cytology (e.g., conjunctival scrapings) for intracellular distemper inclusion bodies is highly diagnostic, particularly in the early stages of infection (Fig. 14.3). (NOTE: use of a quick Romanowsky-type stain is recommended over conventional Wright–Giemsa stains for elucidating distemper inclusions.)

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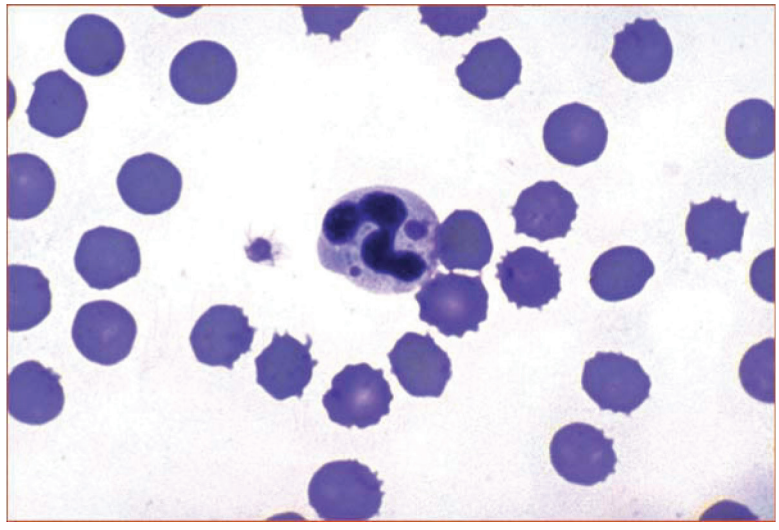


Fig. 14.3. Dark blue-staining distemper virus inclusion body within the cytoplasm of a neutrophil (at the 2 o'clock position) of an unvaccinated dog with acute distemper; the slide was stained with a commercial Diff-Quik stain (recommended) to elucidate inclusion bodies.

- Detection of elevated levels of distemper-specific IgG in CSF is diagnostic of infection. Distemper vaccination does not cause increased levels of CSF antibody. Detection of elevated levels of distemper-specific IgG in serum (virus neutralization), however, is indicative of protection but is not diagnostic of infection.
- Although availability is limited, advanced diagnostic techniques such as immunohistochemistry and detection of CDV RNA in whole blood, serum, and CSF can be used for antemortem diagnosis of distemper.

Pathologic Findings

- A wide range of postmortem findings are described in dogs that die from CDV infection.
- Common gross lesions include pneumonia, catarrhal enteritis, and evidence of rhinitis, conjunctivitis, and tracheobronchitis. Gross CNS lesions are uncommon.
- Histopathology of CNS tissue demonstrates encephalitis and neuronal demyelination with or without perivascular inflammation.
- CDV inclusions are typically found in epithelium of mucous membranes and urinary bladder, leukocytes, and neurons from 5 to 6 weeks post infection.

THERAPEUTICS

- Treatment is limited to supportive therapy intended to reduce mortality.
- Dogs with advanced CNS signs have a poor prognosis for recovery; recommendation for euthanasia may be justified.

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- Supportive care typically includes oral or intravenous antimicrobials, fluid replacement, and analgesics.
- Treatment options for neurologic signs are limited and less likely to be effective.



COMMENTS

Client Education

- Clients who have experienced canine distemper infection in a pet should understand the importance of administering distemper vaccination prior to bringing a new dog into the environment, especially if it has been less than 1 month from the time of diagnosis or death.
- CDV does not reside in the environment for extended periods and common disinfectants are effective in killing virus.
- Dogs that recover from the acute systemic illness may shed CDV for 1–2 weeks and therefore pose a significant risk to susceptible dogs.

Prevention/Avoidance

- Vaccination of all dogs against CDV is recommended.
- Two vaccine types are available: MLV and rCDV. There are no killed CDV vaccines.
- Current (revised) vaccination recommendations stipulate that puppies should receive a dose of CDV (usually in combination with parvovirus and adenovirus-2) at 3–4-week intervals beginning as early as 6 weeks of age and ending not earlier than 15–16 weeks of age, regardless of the product used.
- All dogs should be revaccinated 1 year following administration of the last dose in the initial series. All commercial vaccines provide sustained immunity that lasts several years; vaccination of adult dogs every 3 years is recommended.
- In high-exposure risk environments and in the face of an outbreak (e.g., shelters), early vaccination beginning at 3 weeks of age, and every 2 weeks thereafter, is indicated until 15–16 weeks of age.
- Maternal antibody interference of vaccine is the most common cause of immunization failure when using MLV CDV vaccine.
- Recombinant distemper (rDistemper) vaccine has been shown to immunize puppies in the face of maternal antibody and may provide a significant advantage over MLV vaccines in high-risk environments.

Abbreviations

CDV: canine distemper virus
CNS: central nervous system
CSF: cerebrospinal fluid
HOD: hypertrophic osteodystrophy
IgG: immunoglobulin G

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MLV: modified-live virus
rCDV: recombinant canine distemper virus
RNA: ribonucleic acid

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Acknowledgment to the author of the original chapter in *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Emergency and Critical Care, First Edition:* Richard B. Ford.

chapter 15

Cardiopulmonary Arrest and Cardiopulmonary Resuscitation

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DEFINITION/OVERVIEW

- Cessation of effective perfusion and ventilation due to the absence of coordinated cardiac and respiratory function. In veterinary patients, respiratory arrest is most common. Cardiac arrest invariably follows respiratory arrest if not recognized and corrected.
- Cardiopulmonary resuscitation (CPR) and cardiopulmonary cerebral resuscitation (CPCR) are used interchangeably. Although CPCR is a more descriptive term, CPR is more widely recognized.



PATHOPHYSIOLOGY/ETIOLOGY

- Generalized or cellular hypoxia may be the cause or effect of sudden death.
- After 1–4 minutes of airway obstruction, breathing efforts stop while circulation remains intact.
- If obstruction continues for 6–9 minutes, severe hypotension and bradycardia lead to dilated pupils, absence of heart sounds, and lack of palpable pulse.
- After 6–9 minutes, myocardial contractions cease even though the ECG may look normal (PEA; also known as EMD).
- Ventricular fibrillation, ventricular asystole, and PEA are rhythms indicating cessation of myocardial function.

Systems Affected

- All systems are affected, but those requiring the greatest supply of oxygen and nutrients are affected first.
    - Cardiovascular
    - Renal
    - Neurologic

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SIGNALMENT/HISTORY

- Any age, breed, or sex.
- Risk of cardiopulmonary arrest is directly related to underlying disease present in patient.

Signs

- Loss of consciousness
- Dilated pupils
- Cyanosis
- Agonal gasping or absence of ventilation
- Absence of peripheral pulses
- Hypothermia
- Absence of auscultable heart sounds
- Absence of palpable heartbeat
- Lack of response to stimulation

Risk Factors/Causes

- Cardiovascular disease
- Respiratory disease
- Trauma
- Anesthesia
- Sepsis
- Endotoxemia
- Ventricular dysrhythmias (ventricular tachycardia, R on T phenomenon, multiform)
- Increased parasympathetic tone (gastrointestinal disease, respiratory disease, manipulation of eyes, larynx, or abdominal viscera)
- Prolonged seizures
- Invasive cardiovascular manipulation (pericardiocentesis, surgery, angiography)
- Hypoxemia caused by ventilation-perfusion mismatch, diffusion impairment, hypoventilation, or shunting
- Poor oxygen delivery due to anemia or vasoconstriction
- Myocardial disease: infectious, inflammatory, infiltrative, traumatic, neoplastic, or embolic
- Acid–base abnormalities
- Electrolyte derangements: hyperkalemia, hypocalcemia, and hypomagnesemia
- Hypovolemia
- Shock
- Anesthetic agents
- CNS trauma
- Electrical shock

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DIFFERENTIAL DIAGNOSIS

- Severe hypovolemia and absence of palpable pulses.
- Pericardial effusion, decreased cardiac output, and muffled heart sounds.
- Pleural effusion with respiratory arrest.
- Respiratory arrest can be confused with CPA.
- Upper airway obstruction can rapidly progress to CPA.
- Sudden cardiovascular collapse associated with inadequate cardiac output can lead to severe consequences.



DIAGNOSTICS

- Quick assessment and diagnosis of CPA is critical.
- Assess the ABCs: airway, breathing, and circulation.

Complete Blood Count/Biochemistry/Urinalysis

- May be beneficial in identifying an underlying or contributing cause to CPA but should not be part of initial triage of patient.

Other Laboratory Tests

- Blood gas evaluation may be useful during or after resuscitative procedures but again is not part of initial emergency management.
- Venous blood gas analysis provides an excellent emergency metabolic profile which includes electrolyte and lactate concentrations.
- Venous blood gas analysis may be more beneficial during resuscitation than arterial blood gas.

Imaging

- Thoracic radiographs may help identify underlying disease processes but should only be considered when the patient has been stabilized.
- FAST (TFAST and AFAST) ultrasound will be helpful in assessing underlying disease.
- Echocardiography may confirm pericardial effusion or underlying myocardial disease but should not interfere with resuscitative procedures.

Diagnostic Procedures

- Once CPA has developed, continuous ECG, blood pressure monitoring, pulse oximetry, and capnography should be implemented; thoracic auscultation may be useful in monitoring effectiveness of resuscitative procedures.

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THERAPEUTICS

- CPR should be instituted immediately upon diagnosing CPA. CPR can be divided into basic life support and advanced life support.

Basic Life Support

- Recommendations from the American Heart Association (2010 guidelines) have suggested chest compressions be started immediately following CPA. The rationale is based on the importance of perfusion during the initial phases of CPA compared to oxygenation/ventilation.
- While theoretically this may not be applicable to veterinary patients as respiratory arrest is usually the initiating event, veterinary RECOVER guidelines recommend prompt and immediate intervention with thoracic compressions.

A: Airway

- Assessment: the airway should be visualized by extending the patient's head and neck and pulling the tongue forward. Any debris such as secretions, blood, or vomitus should be cleared manually or with suction.
- Establish: an airway should be established either via per oral endotracheal intubation or, if complete obstruction exists, emergency tracheotomy.

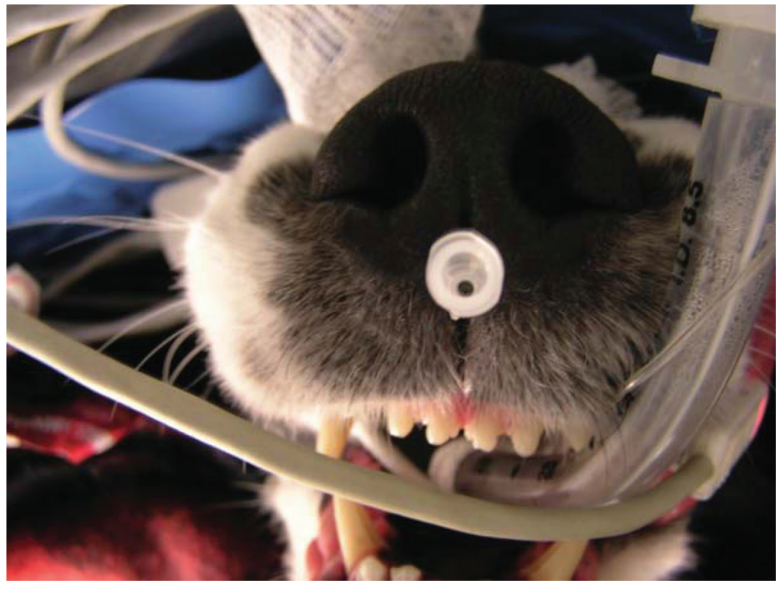
B: Breathing

- Assessment: confirm animal is not breathing.
- Institute manual/artificial ventilation: administer two short breaths approximately 2 seconds duration each and reassess. If spontaneous respiration does not occur, continue ventilations at a rate of 10 breaths/minute. Techniques for ventilation include mouth to mouth, mouth to nose, or mouth to endotracheal tube. These techniques provide approximately 16% oxygen. The use of an Ambu bag and room air provides 21% oxygen.
- The preferred technique is endotracheal intubation and ventilation with 100% oxygen using an Ambu bag or anesthesia machine. The suggested rate of oxygen administration is 150 mL/kg/min.
- The "Gen Chung" maneuver involves placement of a needle through the point of the nasal philtrum to the level of the periosteum and twisting. The stimulus can aid in reestablishing spontaneous respiration (Fig. 15.1).

C: Circulation

- Assessment: palpate peripheral pulses and auscultate heart to confirm CPA. Significant time should not be used if patient is nonresponsive and apneic.
- External cardiac massage has been shown to provide, at best, about 30% of normal cardiac output. Internal cardiac massage has been shown to be 2–3 times more effective in improving cerebral and coronary perfusion.
- Hemodynamic studies in animal models suggest that several different mechanisms exist for the generation of blood flow (artificial systole) during chest compressions. During

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■ **Fig. 15.1.** Placement of a needle through the nasal philtrum to the level of the periosteum in an attempt to stimulate spontaneous respiration.

external cardiac massage, the cardiac pump theory takes advantage of direct compression of the heart in animals less than 7 kg. In animals larger than 7 kg, the thoracic pump theory is used. This technique utilizes increases in intrathoracic pressures to increase cardiac output via the major arteries.

Compression/Ventilation Techniques

- Chest compressions should be performed rapidly at a rate of between 100 and 120 compressions per minute. The chest should be displaced approximately 30%.
- Compressions performed to the music "Staying Alive" provide a compression rate of approximately 100 compressions per minute.
- Hemodynamic studies in animal models suggest that several different mechanisms exist for the generation of blood flow (artifical systole) during chest compressions.
- In animals less than 7 kg body weight, the cardiac pump mechanism should be utilized. This technique takes advantage of direct mechanical compression of the heart. With the animal in right lateral recumbency, compressions are performed directly over the heart (intercostal spaces 3–5). This can be done using one or two hands.
- In animals larger than 7 kg body weight, the thoracic pump mechanism should be utilized. This technique takes advantage of increased intrathoracic pressure to increase blood flow via the great vessels. With the animal in right lateral, thoracic compressions are applied at the widest portion of the thorax (Fig. 15.2).
- If using asynchronous ventilation and compression, one breath is given following every five compressions.
- If using synchronous ventilation and compression, one breath is given simultaneously with every fourth to fifth compression.

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Fig. 15.2. Demonstration of the thoracic pump, in which compressions are performed over the widest portion of the thorax. In this image, an assistant is also performing interposed abdominal compressions to help increase venous return during the time when the thorax is not compressed.

- Interposed abdominal compressions between chest compressions have been shown to increase venous return and enhance cerebral and coronary blood flow by increasing aortic diastolic pressure.

Open Chest Cardiopulmonary Resuscitation

- Indicated if closed chest CPR is ineffective or preexisting conditions such as flail chest, obesity, diaphragmatic hernia, pericardial effusion, etc. preclude effective closed chest techniques.
- Performed through a left thoracotomy at the fourth to fifth intercostal space (Fig. 15.3).

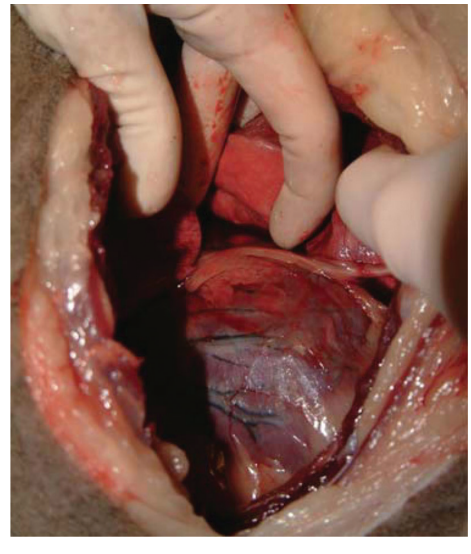


Fig. 15.3. The heart within the pericardial sac during open chest cardiopulmonary chest resuscitation. Visualize the phrenic nerve over the dorsal portion of the pericardium and avoid damage to the phrenic nerve by incising the pericardium at the apex of the heart, then ripping the pericardium with your fingers until the heart can be extracted from the pericardial sac for direct cardiac compressions.

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- A pericardectomy should be performed.
- The palmar surface of the fingers and thumb are used to milk the ventricular blood toward the great vessels. Digital compression of the descending aorta may help perfusion of the cerebral and coronary arteries.

Advanced Life Support

D: Drugs

- Drug selection is based on the arrhythmia that is present.
- Atropine and epinephrine are most often the correct selections.

E: Electrocardiogram

- Accurate ECG interpretation is imperative.
- Try to minimize cessation of thoracic compressions when evaluating ECG.
- Most common arrhythmias are asystole and PEA.

F: Fibrillation Controls and Fluids

- Defibrillation is time dependent and must be performed immediately.
- Fluids should be administered cautiously.
- Aggressive volume resuscitation should not be used unless hypovolemia is thought to be a contributing factor in CPA.
- Volume overload can lead to decreased coronary artery perfusion; therefore, intravenous fluids are contraindicated unless relative (i.e., sepsis or anesthetic gases) or absolute (i.e., hemorrhage, severe volume loss due to vomiting, diarrhea, or wound exudates) hypovolemia is present.

Drugs of Choice

- The selection of drugs is based on the arrhythmia present.
- Epinephrine: (low dose) 0.01 mg/kg IV repeated every 3–5 minutes.
    - High dose (not recommended) 1 mL (1 mg/mL) undiluted per 10 kg body weight can be used in refractory cases.
    - Low dose (recommended) 1 mL (1 mg/mL) diluted 1:10 per 10 kg body weight.
- Atropine: 0.04 mg/kg IV repeated every 3–5 minutes for maximum of three doses.
- Vasopressin: 0.8 U/kg IV; may be used in refractory cases.
- Drugs may be administered via central vein, intratracheal, intraosseous, or peripheral vein in descending order of preference. Intratracheal administration requires double the calculated volume to be delivered. Sodium bicarbonate should *never* be administered into the trachea.
- Acronym for drugs that can safely administered via the intratracheal route is:
  NAVLE – Naloxone, Atropine, Vasopressin, Lidocaine, Epinephrine.
- Blind intracardiac injection is *contraindicated*, as injection directly into the myocardium or damage to the coronary arteries can occur.

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COMMENTS

Comparative Cardiopulmonary Resuscitation

- Current recommendations of the American Heart Association include defibrillation as a component of basic life support. This is unlikely to be useful for veterinary patients unless ventricular fibrillation is confirmed. The most common rhythm associated with cardiac arrest in humans is ventricular fibrillation, whereas the most common rhythm in veterinary patients that leads to cardiac arrest is PEA or asystole.
- If a defibrillator is not available, and ventricular fibrillation is observed, a precordial thump, a rapid compression or bump with a fist on the patient's sternum, can be performed.

Postresuscitation Patient Monitoring

- Maintain heart rate and blood pressure with fluids and inotropic agents.
- Monitor arterial blood pressure.
- Monitor central venous pressure.
- Blood gas analysis.
- Support respiration with artificial ventilation and supplemental oxygen.
- Continued neurological assessment. If signs of increased intracranial pressure, consider mannitol and furosemide.
- Continued monitoring of ECG.
- Monitor urine output.
- Monitor body temperature.
- Radiograph thorax to assess resuscitative injury such as rib fractures or pulmonary contusions. Noncardiogenic pulmonary edema can occur secondary to airway obstruction.
- Diagnose and correct factors that lead to initial CPA.

Possible Complications

- Vomiting
- Aspiration pneumonia
- Fractured ribs or sternebrae
- Pulmonary contusions and edema
- Pneumothorax
- Acute renal failure
- Neurologic deficits
- Cardiac dysrhythmias

Expected Course and Prognosis

- Prognosis is dependent on the underlying disease process.
- Rapid return to spontaneous cardiac and respiratory function improves prognosis.
- Overall prognosis is poor with less than 10% of patients being discharged.

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Synonyms

- Cardiac arrest
- Heart attack

Abbreviations

AFAST: abdominal cavity FAST
CNS: central nervous system
CPA: cardiopulmonary arrest
CPCR: cardiopulmonary cerebral resuscitation
CPR: cardiopulmonary resuscitation
ECG: electrocardiogram
EMD: electrical-mechanical dissociation
FAST: focused assessment with sonography in trauma
IV: intravenous
PEA: pulseless electrical activity
TFAST: thoracic cavity FAST

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chapter 16

Cholecalciferol – Rodenticide Toxicity



DEFINITION/OVERVIEW

- Cholecalciferol, the chemical name for vitamin D_3, is one of the most deadly toxicants to pets due to its narrow margin of safety. Cholecalciferol is expensive to treat (typically requiring 2–5 days of hospitalization) and can predispose the patient to long-term effects of chronic renal failure (CRF).
- Sources of vitamin D3 include over-the-counter (OTC) or prescription vitamins (typically found in a calcium/vitamin D3 combination), psoriasis creams (in the form of calcipotriene), inappropriate supplementation in pet food, and rodenticides.
- Ingestion of toxic levels of cholecalciferol can result in severe hypercalcemia and hyperphosphatemia, with secondary acute kidney injury (AKI) developing as a result of dystrophic mineralization to the soft tissue and kidneys.
- The toxic dose of cholecalciferol is 0.1–0.5 mg/kg. One international unit (IU) of vitamin D_3 is equivalent to 0.025 µg of cholecalciferol; alternatively, 40 IU is the equivalent of 1 µg of vitamin D_3.
- Clinical signs and clinicopathologic findings seen with cholecalciferol toxicosis include generalized malaise, anorexia, vomiting, polyuria, polydipsia, and dehydration. As the toxicosis progresses, hypercalcemia, hyperphosphatemia, azotemia, uremic halitosis, hemorrhagic diarrhea, melena, weight loss, and chronic wasting can occur.



ETIOLOGY/PATHOPHYSIOLOGY

- Due to new EPA mandates, many anticoagulant rodenticides will be replaced by cholecalciferol and bromethalin (see Bromethalin Rodenticide Toxicity for more information).
- Cholecalciferol-containing rodenticides have a very narrow margin of safety. In dogs, cholecalciferol has an LD_{50} of 88 mg/kg (based on the rodenticide concentration of 0.075%). Doses of vitamin D_3 >0.1–0.5 mg/kg can result in clinical signs and hypercalcemia, respectively.

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- Cholecalciferol results in increased calcium absorption from the GIT, reduced renal excretion of calcium, increased calcium absorption in the renal distal tubules, and increased resorption of calcium from the bone.
- When Ca × P >70, soft tissue mineralization occurs to the kidneys, GIT, cardiac muscle, skeletal muscle, blood vessels, and ligaments, resulting in decreased functional capacity or structural damage.
- Cholecalciferol is rapidly absorbed following oral exposure.
- Cholecalciferol and its metabolites are fat soluble and have a wide volume of distribution. The half-life is 19–25 hours, with the terminal half-life lasting for weeks to months.
- Cholecalciferol undergoes enterohepatic recirculation.
- Clinical signs of cholecalciferol toxicosis can be seen within 4–36 hours, and can last for weeks due to the slow release from adipose tissue.

Systems Affected

- Gastrointestinal: vague GI signs can be seen acutely within several hours after ingestion.
- Renal/urologic: AKI secondary to dystrophic mineralization in the kidneys can be seen acutely within 12–48 hours after ingestion.
- Cardiovascular: arrhythmias can develop due to dystrophic mineralization of the myocardium or changes in the ratio of intracellular to extracellular ion concentrations and a change in the depolarization threshold.
- Nervous: with severe electrolyte abnormalities, seizures can be seen.
- Respiratory: decreased lung compliance due to pulmonary calcification.



SIGNALMENT/HISTORY

Risk Factors

- Puppies and younger dogs appear to be overrepresented with poisoning due to their curious nature.
- Puppies are more sensitive to cholecalciferol toxicosis than adults.
- Cats are more sensitive to cholecalciferol toxicosis than dogs.
- Neonates, geriatric patients, or those with underlying disease (e.g., renal disease, hyperparathyroidism, hypercalcemia of malignancy, etc.) are more at risk with cholecalciferol toxicosis.

Historical Findings

- Evidence of a rodenticide container or bait that has been tampered with or chewed.
- History of having green or blue coloring (multiple colors available) in the feces several days before.
- Clinical signs consistent with cholecalciferol toxicosis (see Clinical Features).
- Clinicopathologic findings of azotemia, hypercalcemia, and hyperphosphatemia.

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CLINICAL FEATURES

- Gastrointestinal: initial clinical signs include anorexia, hypersalivation, and vomiting. As AKI develops, more profound signs of uremic ulcers, uremic halitosis, hematemesis, diarrhea, hematochezia, melena, and abdominal pain may be seen.
- Renal/urologic.
  - Electrolyte abnormalities can be seen as early as 12 hours post ingestion. Typically, hyperphosphatemia is seen first, followed by hypercalcemia.
  - AKI is typically seen 24–48 hours post ingestion, but may be seen as early as 12 hours.
  - GI signs, along with polyuria, polydipsia, oliguria/anuria, and renomegaly, may be seen.
- Cardiovascular.
  - Bradyarrhythymias
  - Tachyarrhythmias
  - ECG changes (e.g., prolonged PR interval, shortened QT interval)
- Nervous.
  - Lethargy
  - Depression
  - Seizures
- Respiratory.
  - Tachypnea
  - Compensatory respiratory alkalosis secondary to uremic metabolic acidosis
  - Panting



DIFFERENTIAL DIAGNOSIS

- In patients presenting with AKI, other nephrotoxicants such as ethylene glycol, *Vitus* spp. (e.g., grapes, raisins), NSAIDs, etc. should be ruled out.
- In patients presenting with hypercalcemia, other differential diagnoses such as primary hyperparathyroidism, hypoadrenocorticism, CRF, hypercalcemia of malignancy, idiopathic hypercalcemia of cats, osteolytic bone disease, granulomatous disease, juvenile hypercalcemia, or inappropriate supplementation should be ruled out.



DIAGNOSTICS

- Due to the narrow margin of safety seen with cholecalciferol rodenticide, all ingestions should generally be considered toxic. If an ingestion approaching a hypercalcemic dose has occurred, baseline blood work should include the following.
  - CBC
  - Biochemistry panel

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  - Urinalysis (including pretreatment urine specific gravity)
  - Venous blood gas
  - Ionized calcium
- Clinicopathologic findings may include hemoconcentration secondary to dehydration, anemia (secondary to anemia of chronic disease, lack of erythropoietin, GI bleeding), isosthenuria, azotemia, electrolyte disturbances, and the presence of a metabolic acidosis.
- In hospitalized patients, a PCV/TS, electrolytes, and renal panel (e.g., BUN, creatinine) should be performed every 12–24 hours (typically for 2–4 days), then every 2–3 days thereafter for the next 2–4 weeks (depending on the severity of hypercalcemia). The fluid therapy plan and duration of hospitalization may need to be adjusted based on results of clinicopathologic testing.
- Less common: if rodenticide exposure is not confirmed, additional testing to rule out primary hyperparathyroidism, hypercalcemia of malignancy, or other differential diagnoses (e.g., PTH-rP level, radiographs, ultrasound, etc.) may be necessary.

Pathologic Findings

- Soft tissue mineralization to the kidneys, GIT, cardiac and skeletal muscles may be seen, along with secondary changes from azotemia (e.g., oral or GIT ulceration, gastrointestinal hemorrhage).
- Proteinaceous casts and microscopic renal lesions may be present.



THERAPEUTICS

- The mainstay therapy for cholecalciferol toxicosis is decontamination, aggressive IV fluid therapy (e.g., 0.9% NaCl), treatment for hypercalcemia (e.g., pamidronate, calcitonin), promotion of calciuresis (e.g., prednisone, furosemide), GI support (e.g., antiemetics, H_2 blockers, sucralfate, phosphate binders, etc.), clinicopathologic monitoring, and symptomatic supportive care.
- Treatment is often expensive and requires hospitalization for an extended period of time (typically 2–5 days). Patients should ideally be hospitalized at a 24-hour facility during treatment for this potentially life-threatening toxicant.
- Hypercalcemic patients should be continued on oral furosemide and prednisone for several weeks, following discharge from the hospital. Blood work should be monitored every 2–3 days for 2–3 weeks to allow appropriate titration of the prednisone and furosemide therapy, and to ensure that the patient does not develop secondary AKI or CRF.
- Decontamination.
  - Decontamination should include emesis induction, if ingestion was recent and the patient is asymptomatic. Alternatively, due to the narrow margin of safety, gastric lavage under sedation may be necessary.
  - With recent ingestion in an asymptomatic patient, emesis induction and administration of one dose of activated charcoal (1–5 g/kg, PO) with a cathartic (e.g., sorbitol) are warranted.

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  - As cholecalciferol undergoes enterohepatic recirculation, the administration of multiple doses of activated charcoal (without a cathartic) is warranted (1–2 g/kg PO q6h × 24 hours).
- Fluid therapy.
  - Aggressive fluid rates (8–10 mL/kg/h) of 0.9% saline is the mainstay therapy for cholecalciferol toxicosis. The use of a high sodium-containing crystalloid is necessary to promote calciuresis.
  - Goals of fluid therapy should be based on physical examination findings (e.g., moist mucous membrane, weight gain, etc.) and evidence of hemodilution (e.g., PCV/TS 35%/5 g/dL) and isosthenuria.
  - Patients with underlying cardiopulmonary disease should be monitored appropriately for volume overload.
  - In patients presenting with hypercalcemia + AKI secondary to cholecalciferol toxicosis, severe polyuria and polydipsia may be seen. Fluid therapy should be adjusted based on calculated "ins and outs" and urine output (UOP).
- Treatment for hypercalcemia: drugs such as pamidronate or salmon calcitonin should be implemented in hypercalcemic patients failing to respond to saline diuresis alone.
- Promotion of calciuresis.
  - Once the patient has been *adequately hydrated*, the use of loop diuretics (e.g., furosemide) and corticosteroids (e.g., prednisone) should be implemented in hypercalcemic patients to help promote calciuresis.
  - Due to the multiple doses of activated charcoal administered, oral medications should not be used for at least 24 hours; parenteral dosing is imperative initially.
- Gastrointestinal support.
  - Potent antiemetics should be used for patient comfort, to prevent aspiration pneumonia, to help prevent secondary complications from activated charcoal administration (e.g., emesis, aspiration of charcoal, hypernatremia, etc.), and to allow return to oral intake as soon as possible.
  - Phosphate binders should be used to help treat hyperphosphatemia.
  - Gastroprotectants are necessary, especially in patients that are azotemic and may have uremic ulcers throughout the GIT.

Drugs of Choice

- Bisphosphonates (to inhibit bone resorption and treat hypercalcemia): Pamidronate disodium (1.3–2 mg/kg IV diluted in saline, slow over 2 hours × 1 dose). If calcium and phosphorus levels do not decrease or rebound, a second dose can be given 3–7 days after the first dose.
- Calcitonin (to inhibit osteoclastic bone resorption and treat hypercalcemia): 4–6 U/kg, SQ q8–12h, PRN.
- Furosemide (to promote calciuresis): 2–4 mg/kg IV, SQ, PO q8–12h.
- Corticosteroids (to promote calciuresis).
  - Dexamethasone 0.2–mg/kg IV q12h.
  - Prednisone 1–2 mg/kg PO q12h.

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- Antiemetics.
  - Maropitant (1 mg/kg SQ q24h; extra-label use in cats and via IV route).
  - Ondansetron (0.1–0.5 mg/kg IV q12h).
  - Dolasetron (0.6 mg/kg IV q24h).
- Phosphate binders: aluminum hydroxide 30–100 mg/kg/day PO q8–12h.
- Gastric protectants.
  - H_2 blockers (famotidine 0.5–1 mg/kg SQ, IV, PO q12–24h).
  - Proton pump inhibitors (omeprazole 0.7–1 mg/kg PO q12–24h; pantoprazole, 0.7–1 mg/kg IV q12–24h).
  - Sucralfate (0.25–1 g PO q6–8h).

Precautions/Interactions

- Salmon calcitonin can cause an anaphylactic reaction in animals hypersensitive to it. Prior to use, this author recommends administering a small, diluted amount intradermally to evaluate for the presence of erythema or a wheal.

Alternative Drugs

- Intravenous lipid emulsion (ILE) as an antidote (for fat-soluble toxicants) can be used for the treatment of cholecalciferol toxicosis.

Diet

- A low-calcium, low-phosphorus diet should be fed to patients with AKI or CRF.
- Water (e.g., "baited" food) should be added to dry kibble in patients with AKI or CRF to help increase oral water intake, if possible.

COMMENTS

Client Education/Prevention/Avoidance

- Owners should be educated to appropriately pet-proof the house. Education on crate training is imperative.
- Pet owners should be educated to never use vitamin D rodenticides in a household containing pets, due to the narrow margin of safety.
- Pet owners should be educated about other sources of vitamin D throughout the household (e.g., multivitamins, psoriasis creams, etc.).
- Owners and veterinary professionals should be advised to call veterinary-specific poison control centers for consultation with a veterinary toxicologist for life-saving advice as needed.

Possible Complications

- Patients with AKI secondary to cholecalciferol toxicosis are at increased risk for CRF. Pet owners should be cautioned about long-term effects and chronic management and monitoring.

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- Puppies are often overrepresented with cholecalciferol toxicosis; clinicopathologic monitoring may be difficult due to juvenile hypercalcemia.

Expected Course and Prognosis

- Overall, the prognosis for cholecalciferol toxicosis is fair to good in patients that respond well to decontamination, aggressive fluid therapy, treatment for hypercalcemia, promotion of calciuresis, and symptomatic supportive care. If hypercalcemia does not develop within 48–72 hours of hospitalization, the prognosis is good.
- In situations where financial limitations may preclude treatment, the prognosis is poor to grave.
- In patients developing hypercalcemia and secondary AKI, the prognosis for long-term outcome is fair to poor.

Synonyms

- Calcipotriene
- Dovonex
- Calcitriol
- Tomcat Vitamin D_3®
- Quintox®
- Mouse-B-Gone®
- Rampage®

Abbreviations

AKI:	acute kidney injury
BUN:	blood urea nitrogen
CBC:	complete blood count
CRF:	chronic renal failure
ECG:	electrocardiogram
GI:	gastrointestinal
GIT:	gastrointestinal tract
IM:	intramuscular
IU:	international units
IV:	intravenous
NSAID:	nonsteroidal antiinflammatory drug
OTC:	over the counter
PCV:	packed cell volume
PO:	by mouth (per os)
PTH:	parathyroid hormone
PTH-rP:	parathyroid hormone-related polypeptide
SQ:	subcutaneous
TS:	total solids
UOP:	urine output

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chapter 17

Chylothorax



DEFINITION/OVERVIEW

- The accumulation of chyle in the pleural space.
- Chyle is fluid that is absorbed by lacteals from the intestines. It is composed of mainly lymph and triglycerides.
- Thoracic lymphangiectasia – dilated lymphatics. Occurs in many animals with chylothorax.
- Fibrosing pleuritis – condition that can be caused by chronic irritation of the pleurae by chyle. It results in inflammation and thickening of the pleurae, which causes decreased ventilatory capacity.



ETIOLOGY/PATHOPHYSIOLOGY

- Chyle flows from lymphatics in the mesentery to the cisterna chyli in the abdomen, which then continues in the thorax as the thoracic duct. The thoracic duct empties into the venous system.
- In most cases of chylothorax, abnormal flow or pressures in the thoracic duct are thought to result in the exudation of chyle from dilated thoracic lymphatic vessels into the pleural space.
- Lymphangiectasia is believed to result from increased lymphatic flow (due to increased lymph formation), decreased lymphatic drainage (due to high venous pressures), or the simultaneous occurrence of both processes.
- May be caused by any disease that increases systemic venous pressures (right-sided heart failure, mediastinal neoplasia, cranial vena cava thrombi, or fungal granulomas).
- Thoracic duct rupture due to trauma is an uncommon cause of chylothorax in dogs and cats.

Incidence/Prevalence

- Unknown.

Geographic Distribution

- Worldwide.

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Species

- Dogs and cats.

Breed Predilection

- Dogs – Afghan hounds and Shiba Inus
- Cats – Asian breeds (e.g., Siamese and Himalayan)

Mean Age and Range

- Any age can be affected.
- Cats – may be more common in older cats (possibly associated with neoplasia).
- Afghan hounds – middle-aged.
- Shiba Inus – young (less than 1–2 years old).

Systems Affected

- Respiratory: chylous effusion or fibrosing pleuritis interferes with lung expansion.
- Cardiovascular: decreased intravascular fluid volume due to third spacing of fluid.
- Acid–base, electrolytes: can contribute to electrolyte derangements that include hyponatremia and hyperkalemia due to third spacing of fluid.



SIGNALMENT/HISTORY

Historical Findings

- Most common presenting signs are respiratory difficulty or cough.
- Sometimes duration of cough is months prior to examination.
- Tachypnea.
- Depression.
- Anorexia.
- Weight loss.
- Exercise intolerance.

Physical Examination Findings

- Muffled heart and lung sounds
- Increased bronchovesicular sounds in dorsal lung fields
- Cyanosis
- Pale mucous membranes
- Decreased anterior thoracic compressibility in cats with cranial mediastinal mass and pleural effusion

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- If heart disease is present:
  - Dysrhythmia
  - Murmur
  - Jugular pulses associated with right-sided heart failure

Risk Factors/Causes

- Cranial mediastinal masses (i.e., mediastinal lymphosarcoma, thymoma)
- Heart disease (i.e., cardiomyopathy, pericardial effusion, heartworm infection, tetralogy of Fallot, tricuspid dysplasia, cor triatriatum dexter)
- Trauma
- Fungal granuloma
- Venous thrombus
- Congenital abnormality of thoracic duct
- Lung lobe torsion
- Diaphragmatic hernia
- Idiopathic (most common cause)



DIFFERENTIAL DIAGNOSIS

- Any cause of respiratory distress or coughing.
- Differential diagnoses for exudative pleural effusion include pyothorax, effusion from FIP, neoplastic effusion, pseudochylous effusion, and chylous effusion.
- Pseudochylous effusion is effusion that has a milky appearance due to a high cellularity, but has a low triglyceride level and a high cholesterol level compared to serum.



DIAGNOSTICS

Complete Blood Count/Chemistry/Urinalysis

- Often normal.
- May find lymphopenia and hypoalbuminemia.

Other Laboratory Tests

Pleural Fluid Analysis

- Characteristics: milky and opaque, usually white in color but can range from yellow to pink; may not appear milky in animals with long-term inappetance/anorexia (Fig. 17.1).
- Protein content: inaccurate measurement on refractometer due to high lipid content.
- Total nucleated count usually less than 10 000 cells/μL.

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■ **Fig. 17.1.** Thoracocentesis and chylous effusion in a cat with restrictive cardiomyopathy.

Fluid Cytology

- Mostly small lymphocytes or neutrophils.
- Nondegenerative neutrophils may be predominant cell type with chronic chylothorax due to prolonged loss of lymphocytes or when multiple thoracocenteses result in inflammation.
- If abnormal lymphocytes are present, there may be underlying neoplasia.

Additional Tests

- Fluid triglyceride concentration of chylous effusion is higher than serum triglyceride level.
- Fluid cholesterol level is lower than or equal to serum cholesterol level.
- Sudan III stain for lipid droplets.
- Ether clearance test of fluid.
- Occult heartworm test.
- Viral serology (FeLV and FIV) in cats.

Imaging

Thoracic Radiography (Fig. 17.2)

- Repeat thoracic radiographs after thoracocentesis is performed to remove most of pleural fluid.
- If atelectatic or consolidated lung lobes are noted after pleural fluid is removed, underlying pulmonary parenchymal disease (e.g., lung lobe torsion, pneumonia, neoplasia) or pleural disease (e.g., fibrosing pleuritis) should be suspected.

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■ **Fig. 17.2.** Ventrodorsal thoracic radiograph of a cat with chylous pleural effusion and a cranial mediastinal mass. Note the widening of the mediastinum cranial to the cardiac silhouette.

Ultrasonography

- Should be performed before removing fluid if possible as fluid can enhance visualization of thoracic structures.
- Used to detect underlying cardiac disease, pericardial disease, and mediastinal masses.

Diagnostic Procedures

- Thoracocentesis allows characterization of fluid.

THERAPEUTICS

Drugs of Choice

- Rutin: 50–100 mg/kg PO every 8 hours.
  - One study shows complete resolution of chylous effusion within 2 months of therapy in at least 25% of patients. Since chylothorax resolves spontaneously in some animals, more studies need to be performed to see what percentage of chylothorax cases would have resolved without any treatment.

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Contraindications

- Severe fibrosing pleuritis is associated with a grave prognosis; medical or surgical treatment is unlikely to be beneficial. This occurs more commonly in cats than in dogs.

Medical Management

- Immediate thoracocentesis for patients with respiratory difficulty and suspected pleural effusion.
- Treat underlying cause if possible.
- Medical management with intermittent thoracocentesis when respiratory difficulty is noted.
- Perform thoracocentesis under aseptic conditions to reduce risk of iatrogenic infection.
- Chest tubes only in patients with chylothorax due to trauma (rare), patients with rapid fluid accumulation, or postoperative patients.
- Rutin.
- Low-fat diet; may decrease fat content in pleural effusion, which may improve fluid reabsorption from thoracic cavity and decrease chyle accumulation.
- Some cases of chylothorax may resolve spontaneously in several weeks or months.
- Most cases of chylothorax due to traumatic rupture of the thoracic duct resolve without surgery within a few weeks.

Surgical Management

- Recommended in patients who do not respond to medical management after 2–3 months.
- Thoracic duct ligation.
    - The theory is that after the thoracic duct is ligated, abdominal lymphaticovenous anastomoses form for the transport of chyle to the venous system, bypassing the thoracic duct.
    - The thoracic duct has multiple branches in the caudal thorax; all of these branches need to be occluded. A mesenteric lymphatic should be catheterized for lymphangiography – facilitates visualization and complete occlusion of all branches of thoracic duct.
    - Success rate reported to be approximately 50% in dogs.
    - High incidence of continued or recurrent chylous or nonchylous effusion postoperatively.
- Thoracic duct ligation and pericardectomy.
    - Thoracic duct ligation alone may fail due to thickening of pericardium from chronic irritation by chyle resulting in increased right-sided venous pressures, which would impede the drainage of chyle through lymphaticovenous communications. A pericardectomy may help reduce the occurrence of this.
    - Success rates variable, but reported to be as high as 100%.
- Thoracic duct ligation and cisterna chyli ablation.
    - Thoracic duct ligation alone may lead to caudal thoracic duct and cisterna chyli hypertension, which could be a stimulus for development of collateral lymphatics that causes recurrence of chylothorax after thoracic duct ligation.

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  - Cisterna chyli ablation may force disrupted lymphatic channels to form new drainage connections within the abdominal cavity.
  - This procedure resulted in resolution of chylous pleural effusion in 88% of dogs in one study.
- Thoracic duct ligation, cistern chyli ablation, and pericardectomy.
  - Recommended by some surgeons.

Other

- If medical management and surgical management fail, there are palliative options to drain the pleural space.
  - Placement of a subcutaneous vascular access port connected to an intrathoracic drain or Pleuralport allows for chronic chest drainage through a subcutaneous injection port.
  - Historically, other devices and procedures have been tried for palliative treatment of chylothorax, including pleuroperitoneal shunts, pleurovenous shunts, and pleurodesis. These options are no longer commonly practiced.

COMMENTS

Miscellaneous

- Diffuse lymphatic abnormalities (i.e., intestinal lymphangiectasia, hepatic lymphangiectasia, pulmonary lymphangiectasia, and chylous ascites) may be present. These findings may be associated with a worse prognosis.
- Young patients with chylothorax may have a better prognosis than older animals due to increased incidence of neoplasia in older patients.

Patient Monitoring

- Monitor closely for respiratory difficulty – thoracocentesis as needed.
- Resolution (spontaneously or post surgery); periodically reevaluate for several years to detect recurrence.

Possible Complications

- Fibrosing pleuritis– most common serious complication of chronic chylothorax.
- Immunosuppression – may develop in patients undergoing repeated and frequent thoracocentesis due to lymphocyte depletion.
- Hyponatremia and hyperkalemia in dogs undergoing multiple thoracocenteses.
- Iatrogenic infection from repeated thoracocenteses.

Expected Course and Prognosis

- May resolve spontaneously or after surgery.
- Chronic disease may result in severe fibrosing pleuritis and persistent respiratory difficulty.
- Euthanasia frequently performed in patients that do not respond to therapy.

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Abbreviations

FeLV: feline leukemia virus
FIP: feline infectious peritonitis
FIV: feline immunodeficiency virus

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chapter 18

Coonhound Paralysis





DEFINITION/OVERVIEW

- Acute inflammation of multiple nerve roots and peripheral nerves in dogs, with or without a previous history of contact with a raccoon.
- Proposed animal model for GBS in humans.



ETIOLOGY/PATHOPHYSIOLOGY

- Largely unknown, although coonhound paralysis is closely linked with raccoon contact, specifically with raccoon saliva.
- Suspected immune-mediated disease for both coonhound paralysis and ACIP.
- ACIP has been suspected to be associated with previous vaccination and possibly respiratory or gastrointestinal viral or bacterial infections.
- Most commonly recognized polyneuropathy in dogs in North America, although the incidence is low.
- The distribution of coonhound paralysis is relative to the distribution of raccoons (North and Central America, as well as parts of South America).
- ACIP is found worldwide.

Systems Affected

- Nervous: the peripheral nervous system, with the most severe involvement being in the ventral nerve roots and ventral root components of the spinal nerves.
- Cranial nerves are affected in some patients, with the most commonly affected nerves being VII and X.
- Respiratory paralysis may occur in severe cases, secondary to intercostal and phrenic nerve involvement.

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SIGNALMENT/HISTORY

- In coonhound paralysis, there is a predilection for coonhounds and other raccoon hunting breeds, but any breed in contact with raccoons is susceptible.
- Previous disease does not confer immunity and may actually increase the risk of disease recurrence.
- Multiple bouts of coonhound paralysis are not uncommon in the same dog.
- There are no breed predilections with ACIP.

Historical Findings

- Neurologic signs appear 7–14 days after contact with a raccoon or after other antecedent precipitating events (i.e., vaccinations).
- Owners initially note a stiff-stilted gait in all four limbs.
- There is a rapid progression to a flaccid lower motor neuron tetraparesis to tetraplegia.
- Appetite and water consumption usually remain normal, although dogs may not be able to voluntarily reach food or water.
- Urination and defecation also are usually reported as being normal.
- Initial progression of disease usually occurs over 4–5 days, although occasionally progression can occur up to 10 days.



CLINICAL FEATURES

Neurologic Examination Findings

- Usually neurologic signs are symmetrical.
- There is a generalized hyporeflexia to areflexia, hypotonia to atonia, and severe neurogenic muscle atrophy (more chronic change).
- Pelvic limbs are usually more severely affected than thoracic limbs.
- Respiration may be labored in severely affected dogs with occasional progression to respiratory paralysis.
- Aphonia or dysphonia is common.
- Facial paresis, if present, will be bilateral with incomplete palpebral closure in many patients.
- Pain sensation is intact, although many dogs initially demonstrate a hyperesthesia because of variable dorsal nerve root inflammatory involvement.
- Motor dysfunction always predominates.
- Tail wag is invariably intact.



DIFFERENTIAL DIAGNOSIS

- Other acute polyneuropathy
- Distal denervating disease

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- Botulism
- Tick paralysis
- Fulminant myasthenia gravis
- Generalized (diffuse) or multifocal myelopathy (involving both cervical and lumbosacral intumescences)



DIAGNOSTICS

- Serum immunoglobulin analysis reveals a high serum IgG but not IgM in some patients.
- There is a serum reaction to raccoon saliva on ELISA. Dogs with coonhound paralysis have a strong positive reaction that decreases in intensity over time. Dogs without clinical disease but with recent raccoon contact also have a strong positive reaction. Dogs with ACIP but with no raccoon contact have a negative reaction.

Cerebrospinal Fluid Analysis

- Lumbar CSF analysis will reveal a high protein without an increase in leukocytes at all stages of disease.
- Cerebellomedullary CSF will only reveal a mildly elevated protein in severely affected patients in the acute stages of disease.
- CSF protein elevation is due to albumin leakage across a disrupted blood–brain and blood–nerve barrier.
- Most patients have no intrathecal production of immunoglobulin.

Electrodiagnostics

- Generalized spontaneous activity is seen on EMG, the severity of which depends on the time of examination after disease onset and the severity of neurologic signs.
- There will be markedly low compound muscle action potential amplitudes after motor nerve stimulation.
- Motor nerve root studies (F waves) will reveal an increased minimum latency.
- Motor nerve conduction velocities may be within normal range in mildly to moderately affected dogs, whereas severely affected patients may have mildly low values.
- Sensory nerve function is usually normal.

Pathologic Findings

- Ventral nerve roots and the ventral root components of the spinal nerves develop the most severe lesions, consisting of various degrees of axonal degeneration, paranodal and segmental demyelination, and leukocyte infiltration (predominantly monocytes and macrophages, with scattered groups of lymphocytes and plasma cells).
- Peripheral motor and mixed nerves are similarly affected, although to a lesser degree.
- Dorsal nerve roots are much less severely affected.

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THERAPEUTICS

- Patients should be closely monitored in the progressive stage of the disease (especially during the first 4 days) for respiratory problems.
- If severe respiratory compromise develops, intensive care and ventilatory support may be required.
- Intravenous fluid therapy would only be necessary if the patient is dehydrated due to an inability to reach water.
- Excellent nursing care is essential.
- Patients are able to eat and drink if they can reach food and water but often must be hand fed because of difficulty in moving their limbs and raising their head and neck.
- Intensive physiotherapy is important to decrease muscle atrophy.
- Frequent turning and excellent padding are essential to prevent pressure sores.
- Encourage the patient to try to move as much as possible.

Drugs of Choice

- None have been proven in dogs to be effective in improving clinical signs or shortening the course of disease.
- Human IVIG at 1 g/kg IV daily for 2 consecutive days or 0.4 g/kg IV daily for 4–5 consecutive days; given early, has been shown to decrease the severity or shorten recovery time of GBS in humans.
- A report studying human IVIG administered to dogs (0.5 g/kg IV given daily for 3 consecutive days) documented a trend toward faster recovery time in dogs with ACIP.
- Corticosteroids are contraindicated. They do not improve clinical signs or shorten the course of disease. They have been shown to reduce survival in humans with GBS.

COMMENTS

Client Education

- Inform the client that good nursing care is essential.
- Discuss the importance of preventing pressure sores and urine scalding and of limiting the degree of muscle atrophy by diligent physiotherapy (e.g., passive limb movement, massage, electrical stimulation, and swimming as the patient's strength begins to improve).
- Inform the client that the patient needs soft, resilient bedding (straw or fleeces are excellent) that must be kept clean and free of urine and feces.
- Frequent turning (every 3–4 hours), frequent bathing, and adequate nutrition also are important.

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Patient Monitoring

- It is important to keep in close contact with the client regarding complications or changes in the patient's condition.
- Perform urinalyses periodically to check for cystitis in tetraplegic or severely tetraparetic patients.
- Ideally, reevaluate the patient at least every 2–3 weeks initially.

Prevention/Avoidance

- In dogs with coonhound paralysis, the best prevention for future episodes is to avoid further contact with raccoons. This is often not feasible because of the dog's environment and primary use as raccoon hunters.
- Because raccoons are very common in suburban areas across the country, avoiding contact between household dogs and raccoons also can be challenging.
- Avoid future vaccination if ACIP was definitively linked to a specific vaccine.

Possible Complications

- Respiratory paralysis in the progressive stage of the disease.
- Pressure sores, urine scalding, and cystitis are common in chronically recumbent dogs.

Expected Course and Prognosis

- Most affected dogs recover fully.
- Mild residual neurologic deficits may remain.
- The disease duration is usually several weeks (6–8) in mildly to moderately affected dogs.
- Severely affected dogs can show signs for 3–4 months.
- The order of improvement in signs usually follows the order of initial dysfunction.

Abbreviations

ACIP: acute canine idiopathic polyradiculoneuritis
CSF: cerebrospinal fluid
ELISA: enzyme-linked immunosorbent assay
EMG: electromyography
GBS: Guillain–Barré syndrome
IgG: immunoglobulin G
IgM: immunoglobulin M
IV: intravenous
IVIG: intravenous immunoglobulin

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chapter 19

Diabetic Ketoacidosis





DEFINITION/OVERVIEW

- Severe form of unregulated diabetes mellitus in which by-products of fat metabolism cause accumulation of ketoacids that result in clinical signs of lethargy, inappetance, vomiting, and dehydration.
- Characteristic bloodwork abnormalities include hyperglycemia, metabolic acidosis, presence of ketonemia, and electrolyte abnormalities that can include hyponatremia, hyper- or hypokalemia, and hypomagnesemia.



ETIOLOGY/PATHOPHYSIOLOGY

- Diabetes mellitus is caused by the absolute or relative deficiency of insulin production and release from the pancreas.
- States of prolonged obesity or states of insulin resistance (inflammation, infection, neoplasia) can result in the acute development of diabetes mellitus and diabetic ketoacidosis, or can result in an animal that has been previously diagnosed with diabetes mellitus becoming unregulated or starting to fail insulin therapy.
- The deficiency of insulin or the presence of glucocounterregulatory hormones (cortisol, epinephrine, glucagon, and growth hormone) released during states of stress (inflammation, infection, and neoplasia) can result in an inability to utilize carbohydrate sources for energy, leading to unregulated lipogenolysis.
- By-products of lipolysis are the ketoacids acetoacetate, acetate, and beta-hydroxybutyrate.
- Ketoacidosis stimulates the chemoreceptor trigger zone to induce nausea and vomiting.
- Profound hyperglycemia and osmotic diuresis further contribute to water loss and dehydration.

Systems Affected

- Endocrine/metabolic.
  - Hyperglycemia
  - Hyponatremia

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  - Metabolic acidosis
  - Hyper- or hypokalemia
  - Hypomagnesemia
  - Hypophosphatemia
- Gastrointestinal.
  - Nausea
  - Inappetance
  - Vomiting
- Hematologic.
  - Hypophosphatemia can result in intravascular hemolysis after onset of insulin therapy.



SIGNALMENT/HISTORY

- Middle-aged to older dogs (females predisposed more than males).
- Cats: can occur in any age of cat, but tends to be found in cats older than 10 years (male cats predisposed over females).

Breed Predisposition

- Dogs: miniature poodle, dachshund
- Cats: none



CLINICAL FEATURES

- Polydipsia
- Polyuria
- Polyphagia to inappetance
- Anorexia
- Lethargy
- Depressed mentation/mental dullness
- Weakness
- Plantigrade stance
- Muscle wasting/cachexia
- Weight loss
- Lack of grooming/unkempt or matted haircoat
- Clay litter adhering to paws (from glucosuria)
- Vomiting
- Dehydration
- Fruity odor of ketones on breath
- Cranial organomegaly/hepatomegaly
- Possible cranial abdominal discomfort or pain if pancreatitis is also present

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Bloodwork

- CBC.
  - Neutrophilic leukocytosis
  - Anemia (cats)
- Biochemical panel.
  - Hyperglycemia BG >250 mg/dL
  - Azotemia: elevated BUN and creatinine
  - Elevated cholestatic and hepatocellular liver enzyme activities
  - Hypercholesterolemia
  - Lipemia
  - Hyperosmolarity >330 mOsm/kg
- Venous blood gas and electrolytes.
  - Metabolic acidosis (high anion gap)
  - Hyponatremia
  - Hyper- to hypokalemia
  - Hypochloridemia
  - Hypophosphatemia
  - Hypomagnesemia
- Urinalysis.
  - Hyposthenuria to isosthenuria.
  - Urine sediment may or may not show WBCs/pyuria – culture is still necessary to rule out bacterial cystitis and/or pyelonephritis.
  - Ketonuria: the urine ketone sticks measure acetate and acetoacetate, but not beta-hydroxybutyrate, so may appear negative even if ketonuria is present.



DIFFERENTIAL DIAGNOSIS

- Hypoglycemia
- Intracranial disease
- Primary gastrointestinal disease
- Renal disease
- Hepatic disease
- Sepsis



DIAGNOSTICS

- Diagnostic imaging should include thoracic radiographs to rule out pulmonary parenchymal and pleural space disease that could contribute to insulin resistance.
- Abdominal ultrasound should be performed to rule out obvious causes of insulin resistance.

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THERAPEUTICS

- Patients who are not very dehydrated, and are not vomiting or inappetant, can sometimes be started on long-acting insulin therapy without need for hospitalization.
- The mainstay of therapy for an animal with diabetic ketoacidosis is to rehydrate the patient with intravenous fluids and administer insulin, along with a substrate for which insulin can act.
- First, the patient's overall dehydration deficit should be calculated and added to maintenance fluid therapy, with a goal of the patient becoming rehydrated over 24 hours.
- Short-acting, regular insulin can be administered either IM or IV (this author prefers IV administration as a constant rate infusion), with the goal of dropping the patient's BG by 50–70 mg/dL/h.

Insulin Administration

IM Regular Insulin

- 0.2 U/kg IM once as an initial dose
- 0.1–0.2 U/kg IM every 2–4 hours, depending on the patient's BG

IV Regular Insulin as CRI

- Take 2.2 U/kg (1.1–2.2 units/kg for cats) regular insulin, and add to a 250 mL bag of 0.9% NaCl, then allow 50 mL of the solution to run through the length of IV tubing.
- Administer at the following rate, according to BG level.

Patient BG (mg/dL)	Insulin Solution Rate (mL/h)
>250	10
200 < BG < 249	7
150 < BG < 199	5
100 < BG < 149	5
BG < 100	0

Dextrose Supplementation

Patient BG (mg/dL)	% Dextrose Supplementation
>250	0
200 < BG < 249	2.5
150 < BG < 199	2.5
100 < BG < 149	5
BG < 100	0

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Potassium Supplementation

- Patients with diabetes mellitus and diabetic ketoacidosis have a whole-body potassium deficit due to osmotic diuresis and urinary losses. With the administration of insulin and carbohydrates, potassium moves intracellularly, and the patient can rapidly become hypokalemic.
- The potassium should be checked every 4–6 hours initially, then every 6–12 hours, depending on the patient's response to therapy.

Serum Potassium (mEq/L)	Potassium Supplementation (mEq/L)
<2	80
2.1–2.5	60
2.6–3.0	40
3.1–3.5	30
3.6–5.0	20
>6.0	None

* Total potassium rate should not exceed 0.5 mEq/kg/h

Magnesium Supplementation

- Even when magnesium cannot be measured, animals with diabetes mellitus and diabetic ketoacidosis often have a whole-body magnesium deficit.
- If an animal experiences refractory hypokalemia despite rehydration and maximal potassium supplementation, magnesium supplementation should be considered, as magnesium is required as a cofactor for potassium scavenging.
- Magnesium dose: 0.75–1.0 mEq/kg/day as a constant rate infusion.
- Use care in animals with renal compromise; can cause prolonged PR intervals and AV block.

Phosphorus Supplementation

- An animal's phosphorus may be within normal reference ranges on initial bloodwork.
- As carbohydrate sources are utilized for the production of ATP in the presence of insulin, phosphorus can rapidly become depleted.
- If serum phosphorus <2.0 mg/dL, intravascular hemolysis can occur, as phosphorus is required for the maintenance of red blood cell membrane integrity.
- Phosphorus dose: 0.03–0.05 mmol/kg/h.
    - NOTE: If phosphorus is administered as potassium phosphate, use care to account for the added potassium administration, so as not to exceed a maximal rate of >0.5 mEq/kg/h.

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Bicarbonate Supplementation

- A patient's bicarbonate deficit can be calculated from the following formula:

$$\text{Bicarbonate deficit (meq/L)} = (\text{base deficit} \times 0.4 \times \text{kg body weight})$$

- The administration of bicarbonate should be considered for patients whose serum pH is less than 7.0, but its use is controversial, as the by-product of ketoacid metabolism is bicarbonate.
- With the administration of intravenous fluids for rehydration, as well as insulin and dextrose, the patient's pH will slowly rise due to the metabolism of ketoacids in circulation.

Patient Monitoring

- Initially BG should be evaluated every 2 hours.
- Venous blood gas and electrolytes should be monitored every 4–24 hours, depending on patient's response to therapy.
- Serum and urine glucose and ketone should be monitored daily.
- PCV/TS should be monitored at least twice a day; if the plasma appears hemolyzed, intravascular hemolysis from hypophosphatemia may be occurring.

Long-Term Therapy

- Once a patient is rehydrated, mentally alert, and eating and drinking, transition to a longer-acting insulin can be started.

Prevention/Avoidance

- An animal with DKA that requires either a CRI or q2h insulin administration should be monitored at a facility with 24-hour care, whenever possible, to avoid iatrogenic complications of therapy.

Possible Complications

- Hypokalemia
- Hyponatremia
- Hypoglycemia/seizures
- Hypophosphatemia
- Intravascular hemolysis and anemia
- AKI
- Intravascular volume overload/pulmonary edema
- Cerebral edema/seizures

Synonyms

- DKA

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Abbreviations

AKI: acute kidney injury/insult
ATP: adenosine triphosphate
AV: atrioventricular
BG: blood glucose
BUN: blood urea nitrogen
CBC: complete blood count
CRI: constant rate infusion
IM: intramuscular
IV: intravenous
mEq: milliequivalents
mmol: millimoles
mOsm: milliosmoles
NaCl: sodium chloride
PCV/TS: packed cell volume/total solids
WBC: white blood cell count

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chapter 20

Diaphragmatic Hernia

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DEFINITION/OVERVIEW

- A diaphragmatic hernia (DH) is disruption of the diaphragm that results in displacement of the abdominal viscera into the thoracic cavity.
- A pleuroperitoneal DH involves displacement of abdominal viscera into the pleural cavity and is typically acquired due to trauma, but may rarely be congenital.
- A peritoneopericardial DH (PPDH) is congenital in origin and occurs due to failure of the diaphragm to fuse separately from the pericardial sac and may result in displacement of abdominal viscera into the pericardial space.

ETIOLOGY/PATHOPHYSIOLOGY

- Traumatic DH results from an excessive pressure differential between the abdominal and thoracic cavities.
- When the abdomen is compressed due to trauma (e.g., automobile accident, fall from height), there is increased intraabdominal pressure that is transmitted across the diaphragm to the thoracic cavity.
- If the glottis is open, the air within the lungs is allowed to escape to allow decreased intrathoracic pressure. The pressure differential allows the diaphragm to tear, creating the hernia.
- With traumatic hernias, many factors may contribute to respiratory and cardiovascular compromise.
- Fluid or air accumulation and organ entrapment within the thorax result in lung compression and hypoventilation.
- Pulmonary contusions may further limit lung expansion, and increased capillary permeability due to ongoing inflammation can result in pulmonary edema. Pulmonary contusions result in diffusion impairment and hypoxemia.
- Myocardial contusions may result in cardiac dysrhythmias and decreased cardiac output.

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Systems Affected

- Cardiovascular: myocardial contusions may occur due to trauma and cause cardiac dysrhythmias. Dysrhythmias usually develop within 24–48 hours of the trauma and will typically resolve within approximately 5 days. Ventricular dysrhythmias are most common but other dysrhythmias may also occur. Cardiac output can also be decreased due to hypovolemic shock if hemorrhage has occurred.
- Gastrointestinal: the stomach and intestines are commonly displaced into the thoracic cavity with both congenital or traumatic DH; gastrointestinal displacement in the thorax can cause vomiting, inappetence, and weight loss. Gas distension of the stomach and intestines can occur and compromise lung expansion. If the tear or rent in the diaphragm is small, the intestines can become devitalized due to strangulation, volvulus, or entrapment.
- Hepatobiliary: the liver can be displaced into the thorax and its blood circulation or biliary flow can be disrupted.
- Musculoskeletal: trauma to the thoracic wall can occur and cause decreased chest wall excursion. Other orthopedic injuries can occur secondary to trauma.
- Respiratory: respiratory compromise occurs due to loss of negative thoracic pressure, trauma to the chest wall, fluid or air accumulation within the thoracic cavity, pulmonary contusions, and secondary pulmonary edema.



SIGNALMENT/HISTORY

- Congenital: breed predilection for Weimaraners and cocker spaniel dogs and Himalayan and Persian cats for PPDH.
- Congenital hernias can be present at birth, but may not become clinically apparent for several years.
- Traumatic: no breed predilection.
- More common in younger, male animals due to higher potential for roaming.

Risk Factors/Causes

- Free roaming increases risk for trauma.

Historical Findings

- Mild-to-severe respiratory distress
- Exercise intolerance
- Gastrointestinal signs
- Difficulty lying down
- Weight loss with chronicity

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- Icterus with liver entrapment
- History of trauma with acquired DH; however, some hernias may not be diagnosed for weeks to years after traumatic event.



CLINICAL FEATURES

- Can be incidental finding on thoracic radiographs.
- Respiratory difficulty/orthopnea.
- Muffled heart and lung sounds; intestinal sounds or borborygmi can be ausculted within thorax.
- Cardiac dysrhythmias can occur secondary to myocardial contusions or shock.
- Signs of hypovolemic shock: pale mucous membranes, weak and thready pulses, cool peripheral extremities, hypothermia, or hypotension.
- Empty abdomen on palpation.
- Icterus may be noted with chronic hepatic entrapment.
- Congenital PPDH may coexist with other congenital defects including umbilical and supraumbilical hernias, sternal malformations, and cardiac defects.



DIFFERENTIAL DIAGNOSIS

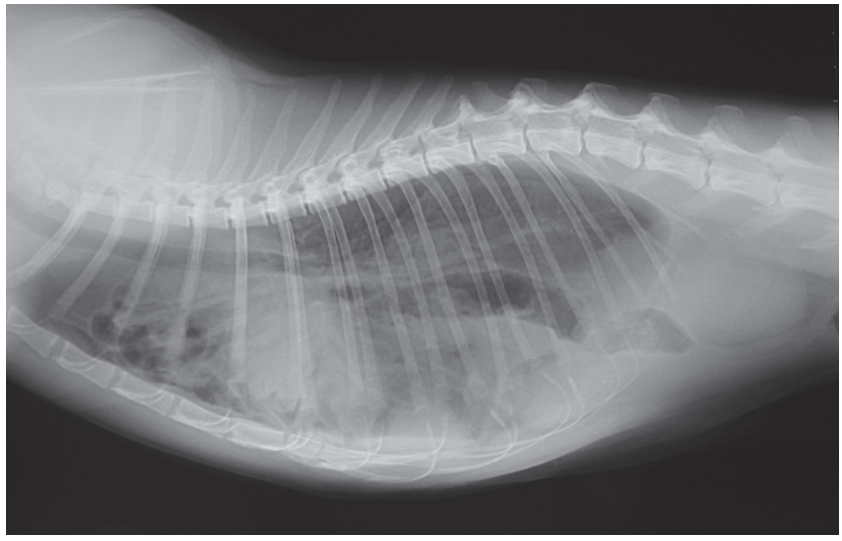
- Pulmonary contusions, pleural effusion, acute respiratory distress syndrome, pulmonary thromboembolism, thoracic neoplasia, and lung lobe torsion.
- Pericardial effusion and dilated cardiomyopathy are differentials for enlarged cardiac silhouette associated with PPDH.



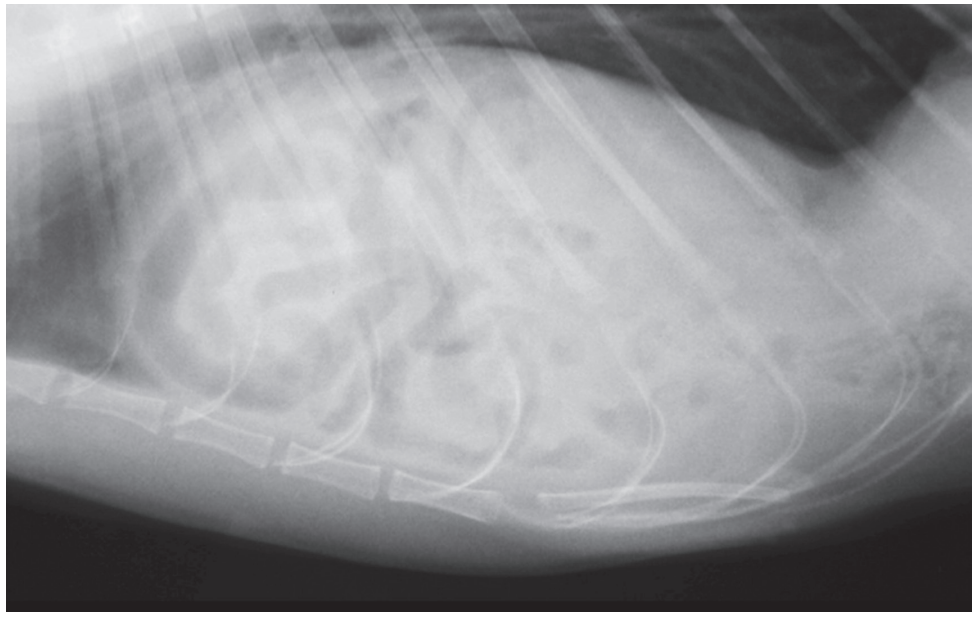
DIAGNOSTICS

- Thoracic radiographs: loss of continuity of diaphragm, soft tissue opacities or gas-filled intestinal loops within thoracic cavity (Figs 20.1, 20.2). Pleural effusion, pulmonary contusions, and rib fractures may also be seen. Plain radiographs will diagnose approximately 66% of diaphragmatic hernias.
- If pleural effusion obscures the diaphragm, performing thoracocentesis and then repeating radiographs can be helpful in making a diagnosis.
- With PPDH, an enlarged cardiac silhouette, dorsal displacement of the trachea, and gas patterns overlying the cardiac silhouette may be seen as well as loss of diaphragmatic continuity ventrally.
- Ultrasonography: allows evaluation of diaphragm continuity and visualization of abdominal viscera within the thorax.
- Upper GI barium study: allows visualization of stomach or intestines within the thoracic cavity, but there is potential for aspiration of barium into the lungs; false negatives can occur if the herniated viscera does not include any portion of the upper gastrointestinal tract.
- Positive contrast peritoneography: limited diagnostic value.

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■ **Fig. 20.1.** Lateral thoracic radiograph of a cat with a traumatic diaphragmatic hernia. The diaphragmatic outline is not visible. Note the appearance of small and large intestinal loops within the thoracic cavity.



■ **Fig. 20.2.** Lateral thoracic radiograph of a cat with a peritoneopericardial diaphragmatic hernia. Note the enlarged cardiac silhouette with dorsal displacement of the trachea. There is loss of continuity of the diaphragm and evidence of small intestinal loops within the cardiac silhouette.

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Pathologic Findings

- Diaphragmatic rent.
- Abdominal viscera displaced into thorax.
  - Compromise to blood supply may result in nonviable tissue.
  - Gas distension of the stomach or intestines may be noted.
- Adhesions if hernia is chronic.
- Thoracic fluid may be noted (i.e., blood, serosanguineous fluid, or chyle).
- Pulmonary contusions or edema and lung atelectasis may be present.



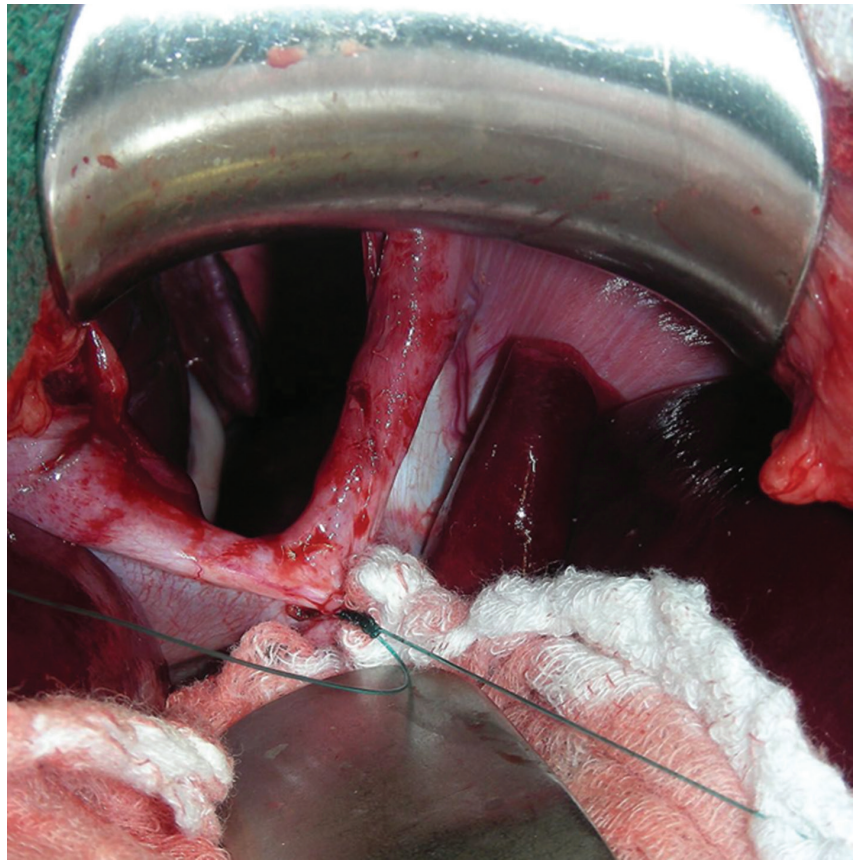
THERAPEUTICS

- Patient stabilization: administer oxygen, intravenous fluids, analgesia, and antiarrhythmics as indicated. With gas distension of the stomach within the thorax, emergency decompression should be performed by passing an orogastric or nasogastric tube or by percutaneous trocharization of the stomach.
- Surgical repair of the hernia once the patient is stable. Use a balanced anesthetic protocol that consists of a premedication, induction drug(s), and inhalant or injectable anesthetic drugs. Premedicate patient to reduce anxiety and decrease the amount of general anesthesia needed. Preoxygenate. Rapid induction (diazepam 0.5 mg/kg IV with propofol 2–4 mg/kg IV, etomidate 1–2 mg/kg IV, or fentanyl 10 µg/kg IV with 0.5 mg/kg diazepam) and intubation. Control positive pressure ventilation once the patient is intubated. Administer perioperative antibiotics. Clip and aseptically scrub both the ventral abdomen and the lateral and ventral thorax in case the incision needs to be extended cranially. The surgical approach should be through a ventral midline abdominal incision. Inspect the herniated abdominal contents for viability and gently replace them into the abdomen. If necessary, enlarge the diaphragmatic defect to aid gentle reduction of herniated organs. Gently break down any adhesions, then resect and remove any devitalized tissue. Close the diaphragmatic rent using absorbable monofilament suture (e.g., 2-0 to 3-0 PDS™ or Maxon™), working from the least accessible portion of the rent toward the surgeon (Fig. 20.3). Restore negative intrathoracic pressure by placing a thoracostomy tube or by percutaneous or transdiaphragmatic thoracocentesis. Close the abdomen in a routine manner.
- Postoperatively, supplemental oxygen, intravenous fluid therapy, and analgesia should be administered. If a continued pneumothorax is present, continuous negative suction of thoracostomy tube may be required. Postoperative monitoring should include pulse oximetry or arterial blood gas analysis, electrolytes, glucose, packed cell volume, total protein, ECG, and blood pressure. Urine output should be monitored if there is concurrent urinary tract trauma.

Precautions/Interactions

- Nitrous oxide is contraindicated with anesthesia for diaphragmatic hernias because it diffuses into the chest cavity, reexpands the gas space within the pleural cavity, and inhibits lung expansion.

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■ **Fig. 20.3.** Intraoperative photograph of a diaphragmatic tear in a dog. The herniated abdominal viscera has been replaced into the abdominal cavity, and the surgical closure has been begun in the most dorsal, least accessible point of the rent.

- Chamber/mask induction is not recommended due to length of time and depth of anesthesia required before intubation and positive pressure ventilation can be initiated.

Activity

- Limited preoperatively due to respiratory compromise; limited for at least 2 weeks postoperatively for healing and recovery.

Surgical Considerations

- In the past, delaying surgery for at least 24 hours was recommended to reduce mortality, but recent studies indicate no difference in mortality with early versus delayed surgical intervention. However, the cardiovascular system should ideally be stabilized prior to anesthesia and surgery.

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- Animals with gastric herniation, evidence of visceral or vascular compromise, or with severe respiratory compromise associated with herniation of abdominal contents should undergo surgery as soon as possible.
- Congenital diaphragmatic hernias should be repaired as soon as they are recognized to avoid the potential for significant complications in the future (e.g., adhesions, organ entrapment, cardiac tamponade).
- Atelectatic lung should be slowly reinflated to avoid any concern about contributing to the development of reexpansion pulmonary edema, a rare but often fatal complication.
- Strangulated viscera should be resected without allowing reestablishment of normal circulation to avoid release of oxygen radicals and bacterial toxins into the general circulation.



COMMENTS

Client Education

- Advise owner of cost and prognosis.

Patient Monitoring

- Respiratory rate and quality, SpO_2
- Cardiac rate and rhythm
- Blood pressure
- Pain

Prevention/Avoidance

- Avoid allowing animals to roam free.

Possible Complications

- Pneumothorax
- Reexpansion pulmonary edema
- Reperfusion injury
- Cardiac arrhythmias

Expected Course and Prognosis

- Mortality rates reported range from 10% to 35%; deaths are most often a result of concurrent injury.
- Good to excellent long-term prognosis if patient survives first 12–24 hours following surgery.

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Abbreviations

DH: diaphragmatic hernia
ECG: electrocardiogram
GI: gastrointestinal
IV: intravenous
PPDH: peritoneopericardial diaphragmatic hernia

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chapter 21

Disorders of Chloride

DEFINITION/OVERVIEW

- Disorders of chloride include hyperchloridemia and hypochloridemia.
- Derangements of chloride can result in acid–base disturbances and alterations in free water balance.

ETIOLOGY/PATHOPHYSIOLOGY

- Chloride ions account for roughly two-thirds of the anions within the extracellular fluid, including plasma.
- Chloride is the major anion that is filtered at the glomerulus and reabsorbed by the renal tubules.
- The chloride ion concentration within cells is much lower than the plasma concentration. The intracellular concentration varies in different types of cells and is dependent on the resting membrane potential of the cell.
    - For example, the intracellular concentration of chloride in red blood cells is, on average, 60 mEq/L, whereas the intracellular concentration of chloride in muscle cells is only 2–4 mEq/L. The higher intracellular concentration of chloride within the red blood cells allows for the effective movement of chloride in and out of the red blood. This ready movement of chloride is the basis for the "chloride shift."
- Chloride is the most prevalent anion found in gastric fluid as well as small and large intestinal fluid.
- Chloride ions are both reabsorbed and secreted in the gastrointestinal tract. The colon is extremely efficient at chloride reabsorption.
- Chloride is the second most prevalent ion found in the glomerular ultrafiltrate.
- Fifty to 60% of the filtered load of chloride is reabsorbed by the proximal tubule of the nephron.
- Chloride reabsorption also occurs in the thick ascending limb of the loop of Henle. Loop diuretics such as furosemide act in the loop of Henle by competing for the chloride site of the Na^+-K^+-$2Cl^-$ carrier.

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- Metabolic acidosis is classically divided into hyperchloremic and normochloremic disturbances based upon the anion gap and the chloride concentration.
  - The anion gap represents the difference between measured cations (sodium and potassium) and measured anions (chloride and bicarbonate). Physiologically, electroneutrality must be maintained so the anion gap represents a clinically useful oversimplification.
  - Metabolic acidosis is seen when there is an increase in the concentration of "strong" anions. A strong anion is one which dissociates completely at body pH. Examples include chloride, lactate, and the ketoanions.
  - When chloride concentration is increased, the sum of the measured anions is unchanged, resulting in a hyperchloremic or normal anion gap acidosis.
  - If the strong anion, which is increased, is unmeasured then the chloride concentration remains normal and the bicarbonate concentration decreases. The sum of the measured anions decreases, resulting in a high anion gap metabolic acidosis.
- In metabolic acidosis and chronic respiratory acidosis, there is an increase in ammonium chloride excretion. It is common to see hypochloremia in chronic respiratory acidosis.
- The roles of hypochloremia and volume depletion in metabolic alkalosis are topics of debate. There is no debate however, that when lost fluid (i.e., vomitus) contains an excess of chloride relative to bicarbonate, a metabolic alkalosis will occur.
- Hyperchloremia is seen with chronic respiratory alkalosis to decrease renal ammonium chloride excretion and increase chloride reabsorption.



SIGNALMENT/HISTORY

- Hypochloremia has been documented in racing greyhounds.



CLINICAL FEATURES

- Clinical signs are related to underlying disease and acid–base abnormalities.



DIFFERENTIAL DIAGNOSIS

Corrected Hypochloremia

- Lipemia (pseudohypochloremia)
- Loss of chloride-rich fluid (i.e., vomitus, diarrhea)
- Administration of diuretics (i.e., furosemide, thiazides) or sodium bicarbonate
- Administration of sodium penicillin (extremely high dose)
- Exercise induced (racing greyhounds)
- Chronic respiratory acidosis
- Hyperadrenocorticism

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- Gastrointestinal disease that mimics hypoadrenocorticism (hyponatremia and hyperkalemia)
- In cats:
  - Acute tumor lysis syndrome
  - Primary hypoadrenocorticism
  - Anemia
  - Hemorrhagic pleural effusion
  - Diabetic ketoacidosis

Corrected Hyperchloremia

- Lipemia (pseudohyperchloremia)
- Potassium bromide therapy (pseudohyperchloremia)
- Diarrhea
- Total parenteral nutrition
- Chloride salt therapy (i.e., potassium chloride or ammonium chloride)
- Fluid therapy (0.9% NaCl, hypertonic saline)
- Salt poisoning
- Renal failure
- Renal tubular acidosis
- Hypoadrenocorticism
- Diabetes mellitus
- Chronic respiratory alkalosis
- Administration of acetazolamide or spironolactone



DIAGNOSTICS

- Prior to evaluation, chloride concentration can be "corrected" to account for changes in sodium concentration.
- Canine:

$$Cl^{-}(\text{corrected}) = Cl^{-} \times 146 / Na^{+}$$

- Feline:

$$Cl^{-}(\text{corrected}) = Cl^{-} \times 156 / Na^{+}$$



THERAPEUTICS

Hypochloremia

- Therapy is directed at correcting the chloride deficit. This is usually achieved by administering 0.9% NaCl.
- When hypokalemia is also present, potassium chloride can be added to the fluid therapy.

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- In the rare instance where volume expansion is not required, therapy with potassium chloride or ammonium chloride can be given.
- The use of sodium chloride or potassium chloride requires intact renal function and the use of ammonium chloride requires both renal and hepatic function.

Hyperchloremia

- Therapy is directed at correcting the underlying disease.
- It is important to consider the chloride content of commonly administered fluids as they often have chloride concentrations in excess of normal plasma.
- In severe metabolic acidosis (pH <7.2), ammonium chloride therapy may be considered.



COMMENTS

Abbreviations

NaCl: sodium chloride
pH: acid–base balance

Suggested Reading

Autran de Morais HS. Chloride ion in small animal practice: the forgotten ion. J Vet Emerg Crit Care 1992; 2:11–24.

Author: Teresa M. Rieser, VMD

chapter 22

Disorders of Phosphorus

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DEFINITION/OVERVIEW

- Phosphorus plays an essential role in cellular structure and function.
- Without phosphorus, vital cellular metabolic processes cannot occur efficiently.
- Hypophosphatemia and hyperphosphatemia can be seen in conjunction with a variety of diseases and have important consequences for the affected animal.

ETIOLOGY/PATHOPHYSIOLOGY

- Phosphorus exists in organic and inorganic forms within the body.
- Clinical laboratories measure inorganic phosphate.
  - Ten to 20% of the inorganic phosphate is protein bound.
  - The remainder exists as a free anion or complexed with sodium, magnesium, or calcium.
- Phosphate is the major intracellular anion.
- Movement into or out of the intracellular compartment can rapidly change the concentration of serum phosphorus.
- The majority of total body phosphorus is found in bone or soft tissue. Less than 1% of the total body phosphorus is found within the extracellular fluid compartment.
- Dogs younger than 1 year may have hyperphosphatemia, which is considered normal. The effect of age is less pronounced in cats, but juvenile cats may tend toward higher serum concentrations.
- Carbohydrate meals or infusions will decrease serum phosphorus due to intracellular shifting.
- Protein intake will increase serum phosphorus concentration.

Hypophosphatemia

- Hemic/lymphatic/immune.
  - Hypophosphatemia increases red blood cell fragility and results in hemolysis.
  - Reduces 2,3-DPG concentrations, and thus impairs tissue oxygen delivery.

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  - Impairs bacterial killing, phagocytosis, and chemotaxis by leukocytes.
  - Thrombocytes have a shorter survival time.
  - Clot retraction is impaired and thrombocytopenia may be seen.
- Neuromuscular.
  - Rhabdomyolysis
  - Intestinal ileus
  - Generalized muscle weakness
- Cardiovascular.
  - Decreased cardiac contractility
- Respiratory.
  - Respiratory failure has been seen in humans with acute, severe hypophosphatemia.
- Nervous.
  - Decreased CNS glucose utilization leading to metabolic encephalopathy
- Renal/urologic.
  - Proximal tubule bicarbonate wasting
  - Decreased titratable acidity
  - Decreased renal ammoniagenesis
- Musculoskeletal.
  - Bone demineralization

Hyperphosphatemia

- Endocrine/metabolic.
  - Decreased serum calcium
- Musculoskeletal.
  - Soft tissue mineralization



SIGNALMENT/HISTORY

- Dogs <1 year of age may have hyperphosphatemia that is considered appropriate (up to 10.8 mg/dL) for the age.
- Effect of age is less pronounced in cats, but immature cats may tend toward high normal serum phosphorus.

Risk Factors/Causes

Hypophosphatemia

- Carbohydrate load or insulin administration can result in hypophosphatemia.
- Diabetic patients, especially patients with diabetic ketoacidosis, are at risk.
- Malnourished patients receiving total parenteral nutrition.
- Both antacid and magnesium administration have been associated with hypophosphatemia in humans.
- Tachypnea and respiratory alkalosis can cause hypophosphatemia.

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Hyperphosphatemia

- Phosphate enema administration.
  - Vitamin D intoxication
- Parenteral phosphate administration.



CLINICAL FEATURES

- Phosphorus derangements most commonly occur as a result of other underlying disease. Clinical features reflect the underlying condition.



DIFFERENTIAL DIAGNOSIS

Hypophosphatemia

- Cellular translocation.
  - Carbohydrate load
  - Insulin administration
  - Respiratory alkalosis
  - Total parenteral nutrition
  - Hypothermia
- Increased renal loss.
  - Hyperparathyroidism
  - Renal tubular defects (Fanconi's syndrome)
  - Proximal acting diuretics (carbonic anhydrase inhibitor)
  - Eclampsia
  - Hyperadrenocorticism
- Decreased intestinal absorption.
  - Dietary deficiency
  - Vomiting
  - Malabsorption
  - Phosphate binder administration
  - Vitamin D deficiency

Hyperphosphatemia

- Cellular translocation.
  - Tumor cell lysis
  - Trauma/rhabdomyolysis
  - Hemolysis
  - Metabolic acidosis

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- Increased intake.
  - Phosphate enema administration
  - Vitamin D intoxication
  - Parenteral phosphate administration
- Decreased excretion.
  - Renal failure
  - Uroabdomen
  - Urethral obstruction
  - Hypoparathyroidism
  - Hyperthyroidism
  - Acromegaly
- Physiologic (young growing animal).
- Laboratory error.
  - Lipemia
  - Hyperproteinemia



DIAGNOSTICS

- Serum phosphorus.
  - Due to the effects of fasting and other factors, serial phosphorus measurements are recommended.
- Serum phosphorus <2.0 mg/dL is considered severe hypophosphatemia.



THERAPEUTICS

Hypophosphatemia

- Animals that are asymptomatic for hypophosphatemia but at risk for worsening hypophosphatemia may be supplemented with phosphate.
- Animals that are symptomatic due to hypophosphatemia should be treated with parenteral phosphate supplementation.

Drugs of Choice

- Potassium phosphate or sodium phosphate.
- Give 0.01–0.06 mmol/kg per hour IV; recheck serum phosphorus every 6–8 hours. (Note: when supplementing with potassium phosphate, remember that additional potassium is also being administered.)

Hyperphosphatemia

- Intravenous fluid therapy with 0.9% NaCl enhances phosphorus excretion.
- Administration of glucose or insulin will temporarily decrease serum phosphorus. This is rarely indicated.

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Drugs of Choice

- Aluminum hydroxide 30–90 mg/kg per day. Recheck serum phosphorus in 10–14 days and adjust dose accordingly.

Diet

- Phosphorus restriction is accomplished via protein restriction.
- Oral phosphate binders may be indicated in chronic renal failure where dietary restriction of phosphorus is insufficient.



COMMENTS

Abbreviations

CNS:	central nervous systems
IV:	intravenous
NaCl:	sodium chloride
2,3-DPG:	2,3 diphosphoglycerate

Suggested Reading

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Author: Teresa M. Rieser, VMD

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chapter 23

Disorders of Sodium



DEFINITION/OVERVIEW

- Disorders of sodium include hypernatremic and hyponatremic states.
- Sodium disorders reflect derangements of water balance. Therefore, it is imperative to understand water balance when approaching sodium disorders.



ETIOLOGY/PATHOPHYSIOLOGY

- Hypernatremia occurs through three different mechanisms.
  - Pure water loss in which either water is lost through the kidneys (diabetes insipidus) or water is not consumed appropriately to meet insensible loss (hypodipsia).
  - Hypotonic fluid loss in which the loss of free water occurs in excess of the loss of solute. This may occur from either renal or extrarenal causes.
  - Impermeant solute gain is a true increase in the solute concentration.

Systems Affected

Hypernatremia

- Cardiovascular: in the loss of hypotonic fluid or pure water deficits, the lost volume can contribute to interstitial dehydration and compromise of the effective circulating volume.
- Nervous: increases in osmolality result in the osmotic movement of water out of the brain cells. Rapid changes can result in cerebral hemorrhage.
- Respiratory: with a true gain of sodium, volume overload and pulmonary edema may be seen.

Hyponatremia

- Hyponatremia is usually associated with hypoosmolality. The approach to hyponatremia therefore centers around the plasma osmolality.
  - Hyponatremia (normal plasma osmolality) occurs due to laboratory error (i.e., hyperlipemia or hyperproteinemia).
  - Hyponatremia (high plasma osmolality >310 mOsm/kg) occurs with the addition of an impermeant solute to the ECF. If this solute is not sodium, there is a relative

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decrease in the serum sodium concentration, but the osmolality is increased. An example would be the intravenous infusion of mannitol.
    - Hyponatremia (low plasma osmolality <290 mOsm/kg) occurs when the relative concentration of sodium is less than normal compared to the volume of the ECF. This can occur in hypervolemia, normovolemia, or hypovolemia.
- Cardiovascular: as with hypernatremia, some hyponatremic animals have compromise of the effective circulating volume.
- Nervous: impairment again is related to rapidity of changes in osmolality.
- With chronic hyperosmolality, the brain will produce idiogenic osmoles, which prevent dehydration of the brain and allow the animal to be relatively asymptomatic.



SIGNALMENT/HISTORY

Risk Factors/Causes

- In the case of pure water deficits, animals without access to water are at risk.
- Animals whose primary access to water is salt water (playing on the beach) may be at risk for true salt gain.

Historical Findings

- Historical findings in acute hypernatremia may include failure to provide the animal access to water or the compromise of the water supply.
- In the case of acute hyponatremia, historical findings may include the ingestion of a large volume of water prior to the onset of clinical signs.



CLINICAL FEATURES

- Often the clinical features are nonspecific but may include anorexia, lethargy, vomiting, muscle weakness, behavioral changes, disorientation, ataxia, seizures, coma, and death.
- Clinical signs may also be referable to the underlying cause.



DIFFERENTIAL DIAGNOSIS

Hypernatremia

- Pure water loss.
    - Primary hypodipsia
    - Diabetes insipidus (central or nephrogenic)
    - High environmental temperature
    - Fever/hyperthermia
    - Inadequate access to water

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- Hypotonic fluid loss.
  - Gastrointestinal loss (i.e., vomiting/diarrhea)
  - Third space loss
    - Pancreatitis
    - Peritonitis
  - Cutaneous burns
  - Renal loss
    - Osmotic diuresis (i.e., mannitol, hyperglycemia)
    - Diuretic administration (i.e., furosemide)
    - Chronic renal failure
    - Polyuric acute renal failure
    - Postobstructive diuresis
- Impermeant solute gain.
  - Salt poisoning (e.g., homemade play dough, salt water ingestion)
  - Hyperaldosteronism
  - Hypertonic fluid administration (i.e., hypertonic saline)

Hyponatremia

- Normal plasma osmolality.
  - Hyperlipemia
  - Hyperproteinemia
- High plasma osmolality.
  - Hyperglycemia
  - Mannitol administration
- Low plasma osmolality.
  - Volume status: hypervolemia
    - Severe liver disease
    - Congestive heart failure
    - Nephrotic syndrome
    - Renal failure
  - Volume status: normovolemia
    - Psychogenic polydipsia
    - SIADH
    - Myxedema coma
    - Hypotonic fluid administration
    - Drugs that stimulate or prolong the renal effects of vasopressin
  - Volume status: hypovolemia
    - Vomiting
    - Diarrhea
    - Third space loss
    - Pancreatitis

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- Peritonitis
- Uroabdomen
- Pleural effusion
- Peritoneal effusion

- Cutaneous burns
- Hypoadrenocorticism
- Diuretic administration



DIAGNOSTICS

- Serum sodium.
- Calculated plasma osmolality:

$$\text{Calc Osmolality: } 2Na + BUN/2.8 + glucose/18$$

- Measured plasma osmolality.
- Urinalysis.
- Additional testing is guided by the suspected underlying cause of the sodium disturbance.



THERAPEUTICS

Hypernatremia

- Pure water loss.
  - Therapy is directed at replacing the free water deficit.

$$Water\ deficit = Weight\,(kg) \times \left(\frac{P_{Na(present)}}{P_{Na(previuos)}} - 1 \right)$$

  - If the sodium prior to water loss is unknown, assume a normal serum sodium.
  - This equation provides the total water deficit in liters.
  - When replacing a water deficit, intravenous sources of water include 5% dextrose in water or 0.45% NaCl.
  - When using 5% dextrose in water, the entire volume administered will be free water. Half of the volume of 0.45% NaCl will be free water.
  - Correct water deficits slowly (0.5 mEq/h maximum). Overenthusiastic corrections can result in rapid changes in osmolality, myelinolysis, and cerebral edema.
- Hypotonic fluid loss.
  - Hypernatremia in this situation is complicated by more severe extracellular volume contraction than pure water loss alone.
  - Initial fluid resuscitation should be an isotonic replacement fluid (Normosol-R, 0.9% NaCl) to correct volume deficits.

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  - Once the animal is volume replete, free water deficits can be corrected using 0.45% NaCl or 5% dextrose in water.
- Gain of impermeant solute (salt poisoning).
  - These animals are often normovolemic to hypervolemic so fluid therapy should be undertaken cautiously.
  - In an animal with normal cardiac and renal function, the administration of 5% dextrose in water will promote diuresis and natriuresis.
  - Animals with cardiac or renal disease may develop pulmonary edema if additional fluids are administered.
  - Diuretics such as furosemide may promote natriuresis and speed the return of the ECF to normal.

Hyponatremia

- Acute, symptomatic hyponatremia is treated with isotonic, crystalloid therapy (0.9% NaCl, Normosol-R). Hypertonic solutions (3% NaCl) are not recommended.
- Chronic hyponatremia is usually asymptomatic due to hypoosmolality and therapy should be directed at correcting the underlying cause.
- With some of the underlying diseases, therapy may involve restriction of water intake, sodium restriction, or diuretic therapy. It is important to diagnose the underlying disease before advocating water restriction.



COMMENTS

- One key to successful treatment of sodium disturbances is to correct them slowly. Rapid correction of long-standing derangements can be fatal.

Expected Course and Prognosis

- Largely dependent on the underlying cause, severity and duration of hyper- or hyponatremia.
- Hyper- and hyponatremic patients have increased risk of nonsurvival.

Abbreviations

BUN: blood urea nitrogen
ECF: extracellular fluid
NaCl: sodium chloride
SIADH: syndrome of inappropriate antidiuretic hormone

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Suggested Reading

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Author: Teresa M. Rieser, VMD

chapter 24

Drowning and Submersion Injury

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DEFINITION/OVERVIEW

- Drowning: death from asphyxia within 24 hours of submersion in water. Alternate definition is the process of experiencing respiratory impairment from submersion or immersion in liquid, and the outcome is classified as death, morbidity or no morbidity.
- Near drowning: water submersion followed by survival for at least 24 hours. This term is discouraged in human medicine.
- Further classification may include very cold water (≤5 °C), cold water (5–20 °C), or warm water (>20 °C) submersion injuries.
- Secondary drowning: death from complications of a submersion event greater than 24 hours after the injury.
- Immersion syndrome: sudden death due to cardiac arrest after sudden immersion in cold water.
- Submersion injury: any of the above events.

ETIOLOGY/PATHOPHYSIOLOGY

- Following submersion, the rise in carbon dioxide levels in the bloodstream stimulates respiration and the animal subsequently aspirates water. In rare cases (approximately 7–10% of people), laryngospasm or hyperventilation prior to submersion prevents aspiration of water ("dry drowning").
- In experimental dogs, the four phases of drowning include: (1) breath holding and swimming motions, (2) aspiration of water, choking, and struggling, (3) vomition, and (4) cessation of movement and subsequent death. Similar observations have been made in cats. The mammalian diving reflex may lead to bradycardia, apnea, and vasoconstriction of nonessential capillary beds.
- Typically, large volumes of water are not aspirated (less than 4 mL/kg in human drowning victims), although aspiration of 1–3 mL/kg of fluid leads to impaired gas exchange in the lungs. Fluid aspirated into the lungs produces a vagally mediated pulmonary vasoconstriction and hypertension.

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- Aspiration of sea water typically leads to more severe hypoxemia than aspiration of an equal volume of fresh water.
- Regardless of the fluid type, the initial cause of hypoxemia after near-drowning is due to ventilation-to-perfusion mismatch and shunting of blood through perfused, but not ventilated, alveoli.
- Following fresh water aspiration, the water moves quickly across the alveolar-capillary membrane and into the microcirculation. The animal's pulmonary surfactant is diluted, leading to alveolar instability and collapse which prevents normal ventilation of affected alveoli.
- Hypertonic seawater aspiration leads to surfactant washout from the aspirated fluid and diffusion of interstitial water into the alveoli, thus preventing ventilation.
- Up to 75% of blood flow may circulate through hypoventilated lung units following either type of near-drowning injury.
- The submersion time, water temperature (see later in this chapter), and type of water (fresh versus salt versus chemical water) determine the development and severity of organ damage. In some patients, aspiration of vomitus, sand, silt, and sewage may cause bronchial and bronchiolar occlusion, bronchospasm, pneumonia, and inflammatory changes to the alveolar-capillary membranes. In addition, postobstructive pulmonary edema may occur in patients with laryngeal spasm.
- Swimming pools may contain chlorine which can irritate the lungs; newer pools with salt-cleaning systems can also lead to irritation of the airways. Fresh-water lakes contain bacteria, algae, and/or protozoa; ocean water frequently has algae, sand, bacteria, and other particulate matter that can lead to pneumonia if aspirated.
- Water temperature can significantly affect the outcome of animals following near-drowning. At temperatures below 15°C, there is a rapid loss of body heat, causing a more rapid onset of fatigue. In addition, the ability to swim is impaired and cutaneous receptors lead to hyperventilation and sensation of respiratory distress as soon as the animal enters the water.

Systems Affected

- Respiratory, nervous, cardiovascular, gastrointestinal, hemic, musculoskeletal, renal, hepatic



SIGNALMENT/HISTORY

- Dogs and cats <4 months of age are at greatest risk.
- Clinical signs may include respiratory distress, cyanosis, coughing, apnea, auscultable crackles or wheezes over chest, tachycardia or bradycardia, vomiting, obtunded to comatose mental state or asystole.

Risk Factors

- Drowning and near-drownings frequently occur in areas where swimming pools, lakes, rivers, canals, oceans, or ponds are present, although indoor drownings are also common (buckets, bathtubs). More drownings may occur in the summer months.

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- Inadequate safety precautions, owner negligence, or purposeful harm are the most common causes of drowning in small animals.
- Dogs and cats <4 months of age are at greatest risk.
- Animals that are in or near water at the time of a seizure, head trauma, hypoglycemic event, cardiac arrhythmia, or syncopal episode are also at risk for drowning.

Historical Findings

- Animal is often observed thrashing/gasping in water or is found motionless in water.



CLINICAL FEATURES

Dogs and Cats

- Cyanosis, coughing +/- clear to frothy red sputum, apnea, dyspnea, auscultable crackles or wheezes over chest, tachycardia or bradycardia, vomiting, obtunded to comatose, +/-hypothermia, asystole.



DIFFERENTIAL DIAGNOSIS

- Hypothermia, neck trauma, and meningitis should be ruled out.
- In the event of drowning secondary to a seizure, neuromuscular disease, head trauma, hypoglycemic event, cardiac arrhythmia, or syncopal episode, appropriate diagnostics should be performed (i.e., labwork, blood pressure monitoring, electrocardiogram, spinal radiographs, cerebrospinal fluid analysis, and brain imaging).
- The history at the time of presentation may be informative.



DIAGNOSTICS

CBC/Biochemistry/Urinalysis

- Inhalation or ingestion of large amounts of fresh water can lead to hemodilution (decrease in hematocrit and total protein), hemolysis, and a decrease in sodium, chloride, and urine specific gravity. Inhalation or ingestion of hypertonic salt water can lead to hemoconcentration (an increase in hematocrit and total protein) and an increase in sodium, chloride, and urine specific gravity.
- Acute renal dysfunction or failure may occur in near-drowning patients. Therefore renal values and urine output should be closely monitored.
- Increased liver enzyme activity has been reported in dogs following fresh-water submersion.
- Disseminated intravascular coagulation may develop following a submersion injury and the resultant hypoxia, hypotension, +/- hypothermia. Therefore, coagulation times,

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platelet counts, and fibrin(ogen) degradation products and/or D-dimer levels should be monitored. Thromboelastography and/or antithrombin levels may also be useful.
- Rhabdomyolysis has been reported in people following a submersion injury and will cause an increase in creatine kinase levels.

Other Laboratory Tests

- Arterial blood gas reveals hypoxemia (PaO_2 <80 mmHg), hypoventilation ($PaCO_2$ >50 mmHg), hypoglycemia, and acid–base derangements such as a respiratory or metabolic acidosis.

Thoracic Radiography

- Radiographic changes may not be detectable for 24–48 hours following the near-drowning. A reduction in cardiovascular dimensions may be seen upon presentation if the patient is hypovolemic.
- Focal or diffuse alveolar pattern may be present due to aspiration pneumonia or noncardiogenic pulmonary edema (typically affecting dorsocaudal lung fields primarily) (Figs 24.1 and 24.2). Mixed bronchial, alveolar, and interstitial patterns may be present, and a radiopaque material filling the airways ("sand bronchogram") has been described.
- Foreign body inhalation may produce segmental atelectasis.

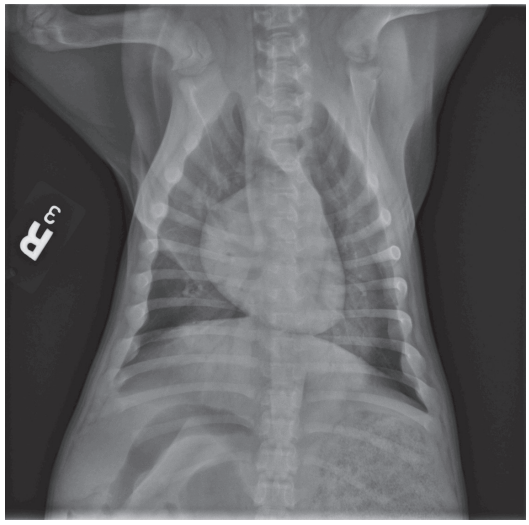


Fig. 24.1. VD thoracic radiograph from a dog that sustained a submersion injury 6 hours before. Note the alveolar lung pattern in the caudal fields, typical of noncardiogenic pulmonary edema. Source: University of Pennsylvania Veterinary Medical Teaching Hospital.

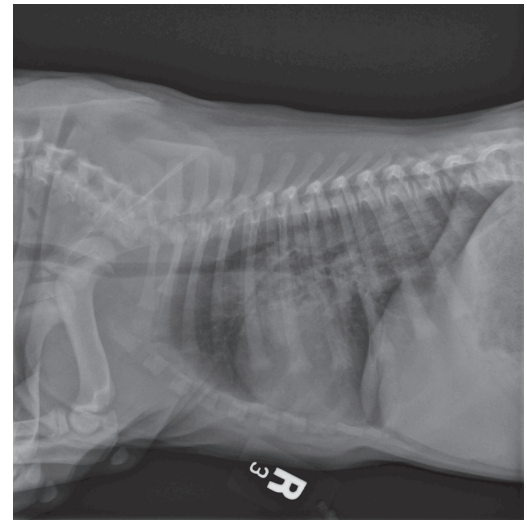


Fig. 24.2. Lateral thoracic radiograph from a dog that sustained a submersion injury 6 hours before. Note the alveolar lung pattern in the caudodorsal lung fields, typical of noncardiogenic pulmonary edema. Source: University of Pennsylvania Veterinary Medical Teaching Hospital.

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- Progression of pulmonary injury to acute respiratory distress syndrome is possible and may appear as bilateral, diffuse, symmetrical alveolar infiltrates.

Diagnostic Procedures

- Endotracheal or transtracheal wash, cytologic evaluation and culture and susceptibility testing are indicated.
- Bronchoscopy may be required for removal of aspirated debris or foreign bodies.
- Electrocardiographic monitoring, continuous pulse oximetry, cervical radiographs or CT, MRI or CT of brain, and BAER may be helpful in select cases.

Pathologic Findings

- The lungs of animals that have drowned are often heavy, sink in formalin, and contain fluid +/-debris that was aspirated within the airways.
- The stomach may also contain ingested water and debris.
- Rhabdomyolysis may be present in some patients.
- Animals that survive for more than 24–48 hours may have evidence of pneumonia, acute lung injury/vasculitis, and/or end-organ damage (i.e., kidneys) secondary to hypoxemia.
- Cerebral edema may be present grossly and histopathologically in some animals.



THERAPEUTICS

- Mouth-to-muzzle resuscitation should be initiated on site. Airway clearance, if obstructed, is the first priority. Cardiopulmonary resuscitation may be necessary.
- Near-drowning victims require emergent, inpatient care.
- Oxygen supplementation should be provided.
- Intubation and mechanical ventilation with positive end-expiratory pressure (PEEP) may be required in animals with severe hypoxemia, hypercapnia, or imminent respiratory fatigue.
- Gravitational drainage or abdominal thrusts (the Heimlich maneuver) are not recommended in the absence of airway obstruction due to the high risk of regurgitation and subsequent aspiration of stomach contents.
- Fluid therapy and acid–base/electrolyte management are crucial.
- Gradual rewarming (over 2–3 hours) in hypothermic animals should be performed.
- Prolonged parenteral nutrition may be required in animals with severe neurologic or pulmonary injury.

Drugs of Choice

- Mannitol therapy, 0.5 g/kg of 25% solution IV over 30 minutes may be beneficial in animals with suspected cerebral edema and elevated intracranial pressures.
- Broad-spectrum antibiotics (ampicillin 22 mg/kg IV q8h and enrofloxacin 15 mg/kg IV q24h in the dog and 5 mg/kg q24h in the cat) may be necessary if aspiration pneumonia develops, but prophylactic therapy is not recommended and may predispose to the development of antibiotic-resistant pathogens.

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- Beta-2-agonists may help animals with suspected bronchospasm.
- Pentoxifylline decreased incidence of lung injury in dogs with experimental fresh-water aspiration.

Precautions/Interactions

- Corticosteroid therapy is not indicated in near-drowning victims, and use of this drug could be detrimental in animals with aspiration pneumonia.
- Use of enrofloxacin in young animals may result in cartilage erosion.

Alternative Drugs

- Alternative antibiotic choices for the treatment of secondary pneumonia are acceptable and might include ticarcillin with clavulanic acid (50 mg/kg IV q6h), clindamycin (10 mg/kg IV q12h) and enrofloxacin (see earlier) or cefotaxime (40–50 mg/kg IV q6h), or chloramphenicol (50 mg/kg IV q8h).
- Alternative bronchodilator therapy may include aminophylline 8 mg/kg IV q6–8h.

Nursing Care

- Patients may require intensive monitoring and care, especially if the need for mechanical ventilation arises. In such cases, it is necessary to keep the patient clean and dry, normothermic, perform frequent turning (~q4h), passive range of motion, oral care, eye lubrication, and close monitoring of comfort and level of sedation or anesthesia.

Diet

- Parenteral nutrition might be necessary for patients that require prolonged anesthesia for positive pressure ventilation.

Surgical Considerations

- Patients that develop pulmonary abscessation might require a thoracotomy for the removal of affected lung lobes.

COMMENTS

Client Education

- Animals that are deemed stable and ready for discharge should be monitored at home for changes in respiratory rate or effort, coughing, decreased appetite, vomiting or diarrhea, and neurologic abnormalities. Owners should consult with a veterinarian if any of these occur. Patients that suffered a near-drowning event due to an underlying disease or event should have further diagnostic evaluation to treat the predisposing condition (e.g., syncope).

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Patient Monitoring

- Frequent or continuous monitoring of heart rate and rhythm, respiratory rate, mucous membrane color and capillary refill time, urine output, arterial blood pressure, rectal temperature, neurologic status, +/- central venous pressure should be performed.
- Arterial blood gas, complete blood count, biochemical profile, coagulogram, and acid–base status should be rechecked as needed.

Prevention/Avoidance

- Close monitoring of animals (especially young and old/debilitated animals or those with seizures or diseases that might cause syncope) near bodies of water, bathtubs, buckets, etc. can help prevent near-drowning accidents.
- Place barriers around bodies of water, life jackets placed on animals, owners educated and trained to perform CPR.

Possible Complications

Patient

- Aspiration pneumonia, pulmonary abscess(es), pulmonary fibrosis, noncardiogenic pulmonary edema, acute respiratory distress syndrome, gastrointestinal bleeding, diarrhea, vomiting, acute kidney injury, permanent neurologic derangements, disseminated intravascular coagulation, central diabetes insipidus.

Owner

- Feelings of guilt and remorse may require counseling.

Expected Course and Prognosis

- The prognosis is directly related to the animal's status at time of admission: animals who present severely acidotic (pH <7.0), require cardiopulmonary resuscitation, or mechanical ventilation have a poor prognosis. Unlike human submersion victims, a retrospective study in dogs found that the level of consciousness at admission was not associated with outcome
- There was a 37.5% mortality rate in 15 dogs and one cat with fresh-water aspiration.
- A worse outcome may be associated with increased submersion times.
- Animals who present conscious have a good prognosis if no complications ensue within 2 hours of the event.
- Pulmonary and cerebral edema can occur up to 24 hours after the submersion injury.
- Cold-water submersion may be neuroprotective, but long-term neurologic sequelae are possible following all near-drowning injuries. Submersion time may be a more important predictor of survival than rectal temperature.

Synonyms

- Submersion injury, immersion syndrome (see Definition/Overview)

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Abbreviations

BAER: brainstem auditory evoked response
CBC: complete blood count
CPR: cardiopulmonary resuscitation
CT: computed tomography
IV: intravenous
MRI: magnetic resonance imaging
PEEP: positive end-expiratory pressure

Suggested Reading

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Author: Deborah C. Silverstein, DVM

chapter 25

Dystocia and Uterine Inertia

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DEFINITION/OVERVIEW

- Most owners lack medical knowledge regarding the birthing process, and as such, they frequently look to the veterinarian to answer questions and identify potential problems. The emergency clinician must therefore be familiar with normal reproductive behavior in addition to the common complications that may arise. Dystocia can occur due to either maternal or fetal causes. Uterine inertia is the most common maternal factor while malposition is the most common fetal factor.

ETIOLOGY/PATHOPHYSIOLOGY

- Normal gestation length in the dog may range from 57 to 72 days from the time of first breeding, with an average length of 65 days.
- If the day of ovulation is known, then the whelping date can be accurately predicted because 95% of bitches will whelp 63 ± 1 days from the date of ovulation.
- Because cats are induced ovulators, there is generally less variability in gestation length, which ranges from 63 to 65 days. Ovulation may not take place after the first breeding, however, so in the event of multiple breedings, uncertainties with regard to gestation length may still be present in the cat.
- As the whelping date approaches, a number of clues may point toward impending parturition. Mammary development, vulvar enlargement, mucoid vaginal discharge, and relaxation of the pelvic ligaments are early signs of approaching parturition. Onset of lactation may be noted in primiparous bitches within 24 hours of parturition, but in multiparous bitches, lactation may occur several days before parturition.
- A sudden drop in body temperature (<99 °F) is generally noted within 24 hours of parturition in dogs and cats as a result of decreases in progesterone levels, but this finding is not always reliable in cats.

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- Normal parturition proceeds in three stages.
  - Stage 1: the first stage is characterized by subclinical uterine contractions and progressive dilation of the cervix. During this stage, which typically lasts for 6–24 hours, bitches may show signs of restlessness, apprehension, panting, nesting behaviors, hiding, and anorexia. Queens may be tachypneic, restless, and vocal or may lie in their nesting boxes, purring. Active expulsion of the fetuses occurs during the second stage of labor.
  - Stage 2: the first fetus is usually delivered within 1 hour of onset of stage 2 labor in cats and dogs, with subsequent deliveries every 15 minutes to 4 hours. The entire process generally occurs over 2–12 hours, but may take as long as 24 hours with large litter sizes.
  - Stage 3: the third stage of labor results in expulsion of the placenta. One placenta should be identified for each fetus delivered. Placentas are usually still attached to the fetus by the umbilical cord and emerge with the fetus, but may emerge within 15 minutes to several hours later if they become detached. Lochia, a greenish vaginal discharge, indicates placental separation and may be seen during all stages of labor.
- Following parturition, the discharge gradually becomes red-brown, decreasing in volume over 4–6 weeks as uterine involution takes place.

Dystocia

- Dystocia may result from either maternal or fetal factors that prevent delivery from taking place. Uterine inertia is the most common maternal cause of dystocia, seen when the myometrium produces only weak and infrequent contractions that fail to expel a normal fetus through a normal birth canal.
- Primary uterine inertia is diagnosed when gestation has exceeded its expected length with no evidence of progression into active labor. Primary uterine inertia is termed partial if the bitch initiates parturition and expels one or more healthy fetuses, but then subsequently fails to deliver the remaining fetuses as a result of myometrial fatigue.
- Morphologic causes of dystocia are those in which an anatomic abnormality of the bitch or queen results in obstruction of the birth canal (e.g., small birth canal, pelvic fractures).
- Fetal factors that may result in dystocia include malpresentation, oversize, fetal malformations, and fetal death. It should be noted that posterior presentations are considered to be a normal variation in dogs and cats, occurring in approximately 40% of deliveries.
- Fetal oversize is a potential cause of dystocia, most commonly seen with single pup pregnancies (Fig. 25.1). Fetal death is an infrequent cause of dystocia, increasing the likelihood of malpresentation because of failure to rotate and extend the head and legs, which commonly occurs immediately prior to parturition.
- Fetal malformations are another potential cause of dystocia, with anasarca (generalized subcutaneous edema), hydrocephalus, cerebral and cerebrospinal hernias, abdominal hernias (Fig. 25.2), duplications, and ribcage malformations among the more commonly noted.

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■ **Fig. 25.1.** Ventrodorsal radiograph of a bitch with one singlet puppy much too large to be delivered naturally without a C-section.



■ **Fig. 25.2.** Eviscerated puppy presented on emergency after natural but difficult delivery.

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Systems Affected

- Reproductive
- Endocrine/metabolic



SIGNALMENT/HISTORY

- Primiparous bitches less than 2 years of age are predisposed to dystocia.
- Toy and small brachycephalic breeds, particularly bulldogs, are predisposed to dystocia.
- Signs of dystocia include active uterine contraction lasting more than 1 hour without passing a fetus, more than 3–4 hours without any active uterine contraction, and failure to initiate labor at the end of gestation.

Risk Factors/Causes

- Very large or very small litters predispose to dystocia.

Historical Findings

- Most often, the owner observes a change in behavior that may or may not be associated with the passing of a fetus. Most commonly, the owner will note active contractions without passing a fetus, or a period of more than 3–4 hours between puppies or kittens.



CLINICAL FEATURES

- On physical examination, the queen or bitch may be crying and biting at the vulvar area, have abnormal vaginal discharge (i.e., profuse hemorrhage; odorous mucopurulent discharge; green discharge without production of offspring, indicating placental separation), or signs of systemic illness in the dam – fever, weakness, tremors, etc.
- A vaginal examination should be performed using sterile technique.



DIAGNOSTICS

- Radiographs should be obtained in any animal experiencing dystocia. Radiographs are accurate for assessing the number, size, location, and position of fetuses, as well as maternal pelvic morphology and general status of the abdomen. Fetal viability is more difficult to assess from radiographs, unless evidence of fetal decomposition is present. Signs of decomposition include intrafetal or intrauterine gas patterns, awkward fetal postures, collapse of the spinal column due to loss of muscular support, and overlapping of the bones of the skull.
- Ultrasound may be a more useful tool for assessment of fetal viability, fetal malformations, and fetal distress. Normal fetal heart rates have been reported at 180–245 beats per minute

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in dogs and up to approximately 265 beats per minute in cats. Deceleration of fetal heart rates to less than 180 beats per minute and the presence of fetal bowel movements on ultrasound have been shown to correlate with severe fetal distress and may indicate a need for rapid intervention.

- A minimum database should include PCV/TS, serum calcium level, and blood glucose.



THERAPEUTICS

- If a fetus is in the birth canal, an attempt should be made to remove it with copious lubrication and gentle, steady traction in a posterior/ventral direction. Best results are obtained with the dam standing while using combined caudal abdominal palpation to direct the fetus into the birth canal. Obstetrical instruments should be avoided if at all possible.
- Medical management with oxytocin should be considered if there is no evidence of obstruction, and fetal and pelvic size appears normal. Calcium gluconate may be considered if weak, infrequent contractions are noted or when lab work reveals hypocalcemia. Retrospective studies have indicated that many patients who fail to respond to oxytocin alone may respond to a combination of calcium and oxytocin.

Drugs of Choice

- Oxytocin doses of 0.5–2 units are effective in increasing the frequency and quality of contraction. The oxytocin dose may be repeated in 30 minutes if expulsion of a fetus has not resulted. If labor proceeds and a fetus is delivered, oxytocin may be repeated every 30 minutes as needed to assist in expulsion of the remaining fetuses. If two doses of oxytocin fail to deliver a fetus, subsequent doses are not recommended. Note that prior to administration of oxytocin, clients should be cautioned that uterine rupture is a rare but possible complication.
- The dose for calcium gluconate (10% solution) as a uterotonic agent is 11 mg/kg diluted in saline and given subcutaneously, or added to intravenous fluids and given slowly while monitoring an ECG for dysrhythmias.
- If hypocalcemia is documented, a dose of 50–150 mg/kg IV should be used.
- Subcutaneous administration has been reported to result in irritation and potential granuloma formation, though this is an infrequent complication.

Surgical Considerations

- Surgical management should be considered for the following conditions.
  - Complete primary uterine inertia
  - Partial primary uterine inertia or secondary uterine inertia where large numbers of feti remain and response to drugs is unsatisfactory
  - Fetal oversize
  - Gross abnormalities of maternal pelvis (fractures, masses)
  - Fetal malformations

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■ **Fig. 25.3.** Intraoperative photograph of a uterine torsion. Note that puppies in the nontwisted uterine horn were still alive and viable at the time of C-section.

- Malpresentation that is not amenable to manipulation
- Past history of dystocia or C-section
- Fetal putrefaction
- Maternal evidence of systemic illness
- Suspicion of uterine torsion (Fig. 25.3), rupture, prolapse, or herniation
- Evidence of fetal distress with poor response to medical intervention

Anesthesia

- An anesthetic protocol for C-section should be selected with the goal of maximizing survival of neonates and dam.
- Attempts should be made to minimize exposure of the fetus to anesthetics by keeping the time from induction to delivery as short as possible.
- Ideally, the dam should be clipped and prepped prior to induction (Fig. 25.4), equipment should be out, and the surgeon should be scrubbed and ready.
- Induction agents (i.e., propofol 4–7 mg/kg IV slowly) should be given to effect.
  - Propofol (4–6 mg/kg IV) or mask inductions are most commonly used for C-section at this time and have been associated with reduced neonatal mortality in dogs.
- Regional techniques such as line blocks and epidurals may help to minimize the need for other drugs.
  - A line block can be performed using 2 mg/kg lidocaine infused along the ventral midline.
  - Alternately, epidural lidocaine may be administered in dogs at a dose of 2–3 mg/kg, not to exceed a total volume of 6 mL.

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■ **Fig. 25.4.** Preoperative photograph of shaving a bitch prior to anesthetic induction to minimize anesthetic time on the bitch and puppies.

- Anesthetic agents that have been associated with increased neonatal mortality include thiopental, ketamine, xylazine, medetomidine, and methoxyflurane.

COMMENTS

Client Education

- Owners of pregnant dogs or cats should be educated about the clinical signs of dystocia and given clear instructions concerning when veterinary intervention is required.

Expected Course and Prognosis

- The prognosis for medical management of dystocia is guarded, with success rates of 20–40% in the veterinary literature. Additionally, stillbirth rates have been shown to rise when dystocia is allowed to continue for greater than 4.5–6 hours from the onset of second stage labor in the dog. For these reasons, the decision to proceed to C-section should not

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be delayed if response to medical management is poor or unlikely to result in successful delivery. In recent studies, neonatal survival rates following surgical treatment of dystocia have been reported at 92% at birth, with 80% still alive at 7 days post C-section.

Abbreviations

ECG: electrocardiogram
IV: intravenous
PCV: packed cell volume
TS: total solids

Suggested Reading

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Author: Scott P. Shaw, DVM

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chapter 26

Electric Cord Injury



DEFINITION/OVERVIEW

- Electric cord injury is an uncommon event that occurs when an animal bites an electric cord.
- Other causes of electrocution are uncommon in dogs and cats but can occur.
- Household electrical currents are alternating (60 Hz) and dangerous.
- Injury can be due to thermal injury or due to disruption of normal electrophysiologic activity of excitable tissue.
- Pulmonary edema can be a sequela to electrocution and the pathophysiology is thought to be noncardiogenic and likely neurogenic and centrally mediated, leading to pulmonary hypertension.
- Cataract formation has been reported following electrocution.



SIGNALMENT/HISTORY

- Seen in dogs and cats.
- More commonly reported in dogs.
- More commonly seen in young or inquisitive animals. In published reports age ranged from 5 weeks to 1.5 years, Veterinary Medical Database suggests 2–12 months of age.
- No breed or sex predilections.
- No genetic basis.

Signs

- Burns of gingiva, tongue, palate (Fig. 26.1)
- Singed hair or whiskers
- Acute respiratory difficulty
- Coughing
- Tachypnea
- Orthopnea

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Fig. 26.1. Burn in commissure of mouth and tongue caused by biting an electric cord.

- Increased respiratory effort
- Cyanosis
- Crackles during pulmonary auscultation
- Cardiac arrhythmias
- Muscle tremors
- Tonic-clonic activity
- Collapse

Risk Factors/Causes

- Chewing electrical cord
- Young animals
- Primarily dogs but also reported in cats

DIFFERENTIAL DIAGNOSIS

- Primary upper airway disease.
- Primary lower airway disease.
- Primary pulmonary disease.

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- Left-sided congestive heart failure – may be due to congenital or acquired heart disease. The presence of cardiac murmur or dysrhythmia may help differentiate, but dysrhythmias may be seen with electric cord injury.
- Vitamin K antagonist, rodenticide intoxication – history, PT, PTT, and PIVKA.
- Thoracic trauma – history and thoracic radiographs.
- Pleural space disease – muffled lung sounds during auscultation and thoracic radiographs.
- Thermal or chemical injuries – history, physical examination, and thoracic radiographs.
- Exposure to fire and smoke inhalation – history and physical examination.
- Atypical pneumonia – history, physical examination, and thoracic radiographs.



DIAGNOSTICS

Complete Blood Count/Biochemistry/Urinalysis

- Typically no abnormalities
- Possible abnormalities related to tissue necrosis
- Hyperkalemia
- Myoglobinemia
- Myoglobinuria
- Hemoglobinemia
- Hemoglobinuria
- Hyperlactatemia
- Hyperglycemia

Other Laboratory Tests

- Arterial blood gas analysis may indicate hypoxemia.

Imaging

- Thoracic radiographs may help distinguish between cardiogenic and noncardiogenic causes of pulmonary edema.
- The radiographic pattern is usually a generalized, mixed alveolar bronchial pattern. The edema is often most notable in the diaphragmatic lung lobes (Fig. 26.2).
- Echocardiography may help identify underlying cardiac disease.

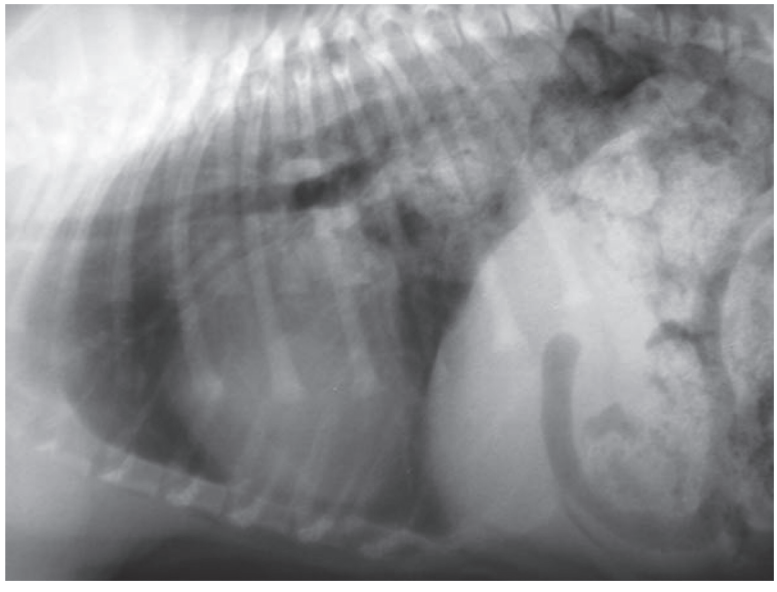
Diagnostic Procedures

- ECG may help distinguish cardiogenic disease from noncardiogenic disease, but dysrhythmias may be seen with electrocution

Pathologic Findings

- Pink, frothy fluid in airways
- Fluid-filled, congested lungs

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■ **Fig. 26.2.** Lateral thoracic radiograph of a dog with noncardiogenic pulmonary edema secondary to electrocution/electric cord injury.

- Subendocardial and subepicardial petechiae
- Circumscribed, pale gray or tan oral lesions



THERAPEUTICS

- If patient is close to live wire, turn off electricity or remove patient to safe area.
- Establish patent airway if patient is unconscious.
- Oxygen supplementation.
- Mechanical ventilation may be required.
- Establish venous access.
- Management of thermal injuries.

Drugs of Choice

- If in shock, treat with intravenous crystalloids (90 mL/kg per hour in dogs, 45–60 mL/kg per hour in cats) or colloids plus or minus hypertonic saline; administer in incremental boluses until blood pressure has normalized.
- If pulmonary edema is present, the use of furosemide is controversial (2–4 mg/kg IV or IM, 0.7–1 mg/kg/h IV CRI) if hemodynamically stable.
- Corticosteroids have been used but are of unknown value and are not recommended.

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- Inotropic support if required.
- Antiarrhythmic therapy if required.
- Oral and cutaneous burns – treat symptomatically.
- Analgesia if required, typically with opioid agents.

COMMENTS

Patient Monitoring

- Patient should be monitored until stable.
- Physical examination.
- Oral lesions should be monitored and may prevent the animal from eating.
- Electrocardiography.
- Central venous pressure.
- Blood pressure.
- Arterial blood gas analysis.
- Thoracic radiographs.

Prevention/Avoidance

- Damaged electric cords should be discarded.
- Avoid animal exposure to electric cords.
- Follow child safety rules for a safe home.

Possible Complications

- Infected burn wounds can occur but are uncommon.
- Oral–nasal fistula due to severe burns and tissue necrosis.

Expected Course and Prognosis

- The prognosis is based on the response to therapy.
- Pulmonary edema can develop as soon as 1 hour and as late as 36 hours after the incident.
- Pulmonary edema associated with electrocution is associated with high mortality (38.5%).
- If the patient survives the first 24 hours, the prognosis improves.
- Resolution of pulmonary edema may take 3–5 days.
- Most oral lesions resolve.
- Inappetence related to oral lesions resolves; severe cases may require placement of a feeding tube.

Associated Conditions

- Cataracts have been reported in one dog 18 months after electrocution.

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Abbreviations

CRI: constant rate infusion
ECG: electrocardiogram
IM: intramuscular
IV: intravenous
PT: prothrombin time
PTT: partial thromboplastin time
PIVKA: protein-induced by vitamin K absence/antagonism

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Author: Steven L. Marks, BVSc, MS, MRCVS

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chapter 27

Epistaxis

DEFINITION/OVERVIEW

- Hemorrhage from the nose (Fig. 27.1).

ETIOLOGY/PATHOPHYSIOLOGY

- Results from either local or systemic disease processes.
- With local disease, nasal blood vessels rupture after direct trauma or erosion by infection, inflammation, or neoplasia.
- Systemic diseases cause hemorrhage by hemostatic defects (such as thrombocytopenia, vitamin K antagonist rodenticide intoxication) or increased vascular fragility.

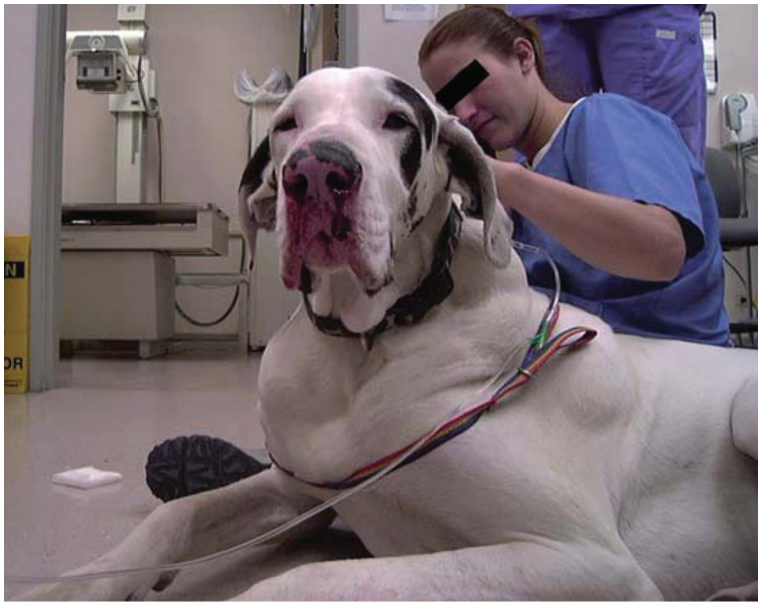


Fig. 27.1. Epistaxis secondary to bacterial rhinitis in a Great Dane.

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Systems Affected

- Respiratory: hemorrhage or sneezing
- Hemic/lymphatic/immune: anemia
- GI: melena or hematemesis

Incidence/Prevalence

- Local processes are the most common cause of epistaxis.

Geographic Distribution

- See other sources for areas endemic to fungal, rickettsial, and other infectious diseases.



SIGNALMENT/HISTORY

- Varies with underlying cause.
- Coagulopathies: young, purebred dogs
- Infectious/traumatic: young to middle-aged
- Immune mediated: middle-aged, female dogs
- Neoplasia: older animals

Historical Findings

- Nasal hemorrhage
- Sneezing
- Pawing at nose
- With coagulopathy: hematochezia, melena, hematuria, or hemorrhage in other areas of the body
- With hypertension: blindness, CNS signs

Physical Examination Findings

- Nasal hemorrhage
- Melena, commonly from swallowing blood
- Sneezing
- Pawing at nose
- Facial asymmetry, usually secondary to neoplasia
- Decreased nasal airflow
- Polypoid masses extending from the nares: rhinosporidiosis, pheohyphomycosis, cryptococcosis, or neoplasia
- Decreased ability to retropulse the globe
- Epiphora or exophthalmos

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- Regional lymphadenomegaly
- Oral exam: palatal defect, mass, oronasal fistula, or severe periodontal disease
- Fundic exam: chorioretinitis, retinal hemorrhage, tortuous retinal vessels
- With cryptococcosis: nasal convexity ("Roman nose")
- With aspergillosis or squamous cell carcinoma: ulceration or depigmentation of the nasal planum
- With coagulopathy: petechia, ecchymosis, hematomas, hematochezia, and hematuria

Risk Factors/Causes

Systemic Disease

Thrombocytopenia

- Spontaneous hemorrhage unlikely unless platelet count is less than 50 000/μL.
- Decreased production: infectious (i.e., ehrlichiosis, FeLV, FIV, RMSF, hepatozoonosis, septicemia, endotoxemia, anaplasmosis or leishmaniasis), toxic (i.e., cytotoxic drugs, estrogens, sulfas, methimazole, or phenobarbital), neoplasia (i.e., myelophthisis, plasma cell tumor/myeloma or hyperestrogenism), cyclic thrombocytopenia, myelofibrosis, or myelodysplasia.
- Destruction: immune mediated (i.e., idiopathic, secondary to drugs, neoplasia, infection, or vaccines) or microangiopathy.
- Sequestration in the spleen, liver, or tumors.
- Consumption: DIC, vasculitis, or hemorrhage.

Thrombocytopathia

- Congenital: vWD, platelet procoagulant activity deficiency in German shepherds or Glanzmann's thrombasthenia in Great Pyrenees, basset hound, and foxhound thrombopathia.
- Acquired: NSAID, hyperglobulinemia (i.e., ehrlichiosis, multiple myeloma, leishmaniasis), or uremia.

Coagulation Factor Defects

- More likely to cause intracavitary hemorrhage.
- Congenital: hemophilia A (factor VIII deficiency) or hemophilia B (factor IX deficiency).
- Acquired: anticoagulant rodenticide toxicosis, hepatic failure, or DIC.

Increased Vascular Fragility

- Hypertension: renal disease, hyperadrenocorticism, hyperthyroidism, pheochromocytoma, or idiopathic.
- Hyperviscosity: hyperglobulinemia, polycythemia, or leukemia.
- Hyperlipidemia.
- Vasculitis: immune mediated or rickettsial.

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Local Disease

- Trauma.
- Oral/dental disease: oronasal fistula or tooth root abscess.
- Foreign body inhalation: grass awns, splinters, or small objects.
- Infectious.
  - Viral: feline viral rhinotracheitis or calicivirus
  - Bacterial: *Bordetella*, *Pasteurella*, or *Mycoplasma*
  - Fungal: aspergillosis, penicilliosis, cryptococcosis, rhinosporidiosis, pheohyphomycosis, or pythiosis
  - Parasitic: mites or nematodes
- Immune mediated: lymphoplasmacytic rhinitis or allergic rhinitis.
- Neoplasia: adenocarcinoma (most common in dogs), lymphoma (most common in cats), undifferentiated carcinoma, chondrosarcoma, squamous cell carcinoma, fibrosarcoma, hemangiosarcoma, osteosarcoma, melanoma, TVT, plasmacytoma, myeloma or nasopharyngeal polyps.
- Environmental: humidity changes.
- Rupture of arteriovenous malformations.
- vWD: Dobermans, Shetland sheepdogs, Airedales, German shepherds, Scottish terriers, and Chesapeake Bay retrievers.
- Thrombasthenia: otterhounds and Great Pyrenees.
- Thrombopathia: basset hounds and foxhounds.
- Hemophilia A: German shepherds.
- Hemophilia B: cairn terriers, coonhounds, and Saint Bernards.
- Aspergillosis: German shepherds.
- Neoplasia: dolicocephalic breeds.



DIFFERENTIAL DIAGNOSIS

- In general, systemic causes should be ruled out prior to investigation of nasal disease.
- The signalment, history (including character and duration of nasal discharge/epistaxis), and physical examination can greatly help prioritize differentials.



DIAGNOSTICS

Complete Blood Count/Chemistry/Urinalysis

- Anemia: regenerative or nonregenerative depending on chronicity.
- Schistocytes: microangiopathy (neoplasia or DIC).
- Leukocytosis: chronic inflammation or infection.
- Leukopenia: chronic ehrlichiosis, cytotoxic drug administration, or sepsis.

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- Thrombocytopenia.
- Macroplatelets: platelet destruction or consumption.
- Pancytopenia: bone marrow disease.
- Panhypoproteinemia due to blood loss.
- Hyperglobulinemia: neoplasia or chronic infections.
- High BUN with normal creatinine due to ingested blood or GI hemorrhage.
- Azotemia: renal failure-induced hypertension or uremic thrombocytopathia.
- Elevated liver enzymes or hyperbilirubinemia: hepatic disease.
- Hematuria: coagulopathy.
- Isosthenuria: renal failure-induced hypertension or uremic thrombocytopathia.
- Proteinuria: hyperglobulinemia.

Other Laboratory Tests

- PT/aPTT: abnormal in patients with coagulation factor defects.
- BMBT: abnormal in patients with thrombocytopathia, inherently prolonged with thrombocytopenia (not indicated).
- vWF assay.
- Thyroid hormone levels in cats.
- Serology for heartworm, FeLV, FIV, *Cryptococcus*, *Aspergillus* (poor sensitivity and specificity), *Ehrlichia*, RMSF.
- Fecal flotation: ova of nasal parasites may be swallowed and shed in feces.
- Serum protein electrophoresis: to distinguish between monoclonal or polyclonal gammopathy.

Imaging

- Thoracic radiographs may show evidence of fungal disease or metastatic neoplasia.
- Skull radiographs are difficult to interpret but may reveal osteolysis with neoplasia or fungal rhinitis or opacity in the tympanic bulla in cats with nasopharyngeal polyps.
- Dental radiographs: tooth root abscesses.
- CT/MRI: superior to radiography for nasal disease.

Diagnostic Procedures

- Blood pressure.
- Sedated oral exam.
- Cytology of nasal discharge may identify *Cryptococcus* organisms.
- Rhinoscopy: visualization and biopsy of foreign bodies, masses, fungal plaques, and nasal parasites.
- Nasal cavity flushing to remove foreign bodies or retrieve tissue for histopathology.
- Exploratory rhinotomy/turbinectomy, if less invasive tests are nondiagnostic.
- Bone marrow aspirate or biopsy for diagnosis of thrombocytopenia or pancytopenia.

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THERAPEUTICS

- General: stop hemorrhage and provide supportive care.
  - Cage rest, ice packs, pressure to the nose or maxillary arteries
  - Sedation
  - Intranasal vasoconstrictors (i.e., epinephrine or phenylephrine)
  - Packing of the nasal cavity with gauze soaked in Vaseline or dilute epinephrine
  - Ligation of the external carotid artery in refractory cases
  - Intravenous fluids for hypovolemia and blood transfusion for severe anemia
- Specific: varies with cause.
  - vWD: fresh frozen plasma or cryoprecipitate
  - Thrombasthenia and thrombopathia: no treatment
  - Hemophilia: plasma or cryoprecipitate for acute bleeding; no long-term treatment
  - Anticoagulant rodenticide toxicosis: fresh or frozen plasma followed by vitamin K
  - Immune-mediated thrombocytopenia: immunosuppression
  - Hyperviscosity: plasmapheresis, phlebotomy, treatment of underlying cause
  - Vasculitis: doxycycline for rickettsial disease; corticosteroids for immune-mediated disease
  - Fungal rhinitis: intranasal or oral antifungal therapy or turbinectomy
  - Foreign body: removal by rhinoscopy or rhinotomy
  - Hypertension: ACE inhibitors, beta-blockers, calcium channel blockers, treat underlying disease
  - Neoplasia: varies by tumor type

Drugs of Choice

- Immune-mediated thrombocytopenia: prednisone 1–2 mg/kg every 12 hours or equivalent doses of other corticosteroids; vincristine 0.02 mg/kg IV once; additional immunosuppression may be needed in refractory cases.
- Anticoagulant rodenticide toxicosis: vitamin K 5 mg/kg PO divided daily, 15–20 mL/kg fresh frozen plasma.
- Fungal rhinitis: itraconazole 5 mg/kg PO every 12 hours for cryptococcosis.
- Vasculitis: doxycycline for rickettsial diseases (5 mg/kg every 12 hours for 3–6 weeks), corticosteroids at antiinflammatory doses.
- Hypertension: enalapril or benazepril 0.25–0.5 mg/kg every 12–24 hours, propranolol 0.5–1.0 mg/kg every 8 hours, atenolol 2 mg/kg every 24 hours, diltiazem 0.5–1.5 mg/kg every 8 hours (dog), 1.75–2.5 mg/kg every 8 hours (cat), or amlodipine 0.625 mg every 24 hours (cat).
- Yunnan Paiyao (Yunnan Baiyao):
  - Dog: open capsule and sprinkle on superficial wounds, bleeding tumors, etc.
    - <15 kg: 1 capsule PO twice a day
    - 15–30 kg: 2 capsules PO twice a day
    - >30 kg 2 capsules PO three times a day

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- Cat: open capsule and sprinkle on superficial wounds, bleeding tumors, etc.
  - 1/2 capsule PO twice a day

Contraindications

- Avoid drugs that predispose to hemorrhage (i.e., NSAIDs or heparin) or hypertension (i.e., phenylpropanolamine).

Precautions/Interactions

- Use sedation with caution to avoid hypotension.
- Animals with altered mentation or under sedation may aspirate blood.
- Monitor renal values when using ACE inhibitors.
- Substantial hemorrhage may occur after rhinoscopy or biopsy.



COMMENTS

- Platelet count in thrombocytopenic animals.
- Coagulation profile in animals with coagulation factor defects.
- Blood pressure in animals with hypertension.
- Rhinoscopy to confirm resolution of fungal rhinitis.
- Globulins or serum protein electrophoresis in animals with hyperglobulinemia.

Abbreviations

ACE: angiotensin converting enzyme
aPTT: activated partial thromboplastin time
BMBT: buccal mucosal bleeding time
BUN: blood urea nitrogen
CNS: central nervous system
CT: computed tomography
DIC: disseminated intravascular coagulation
FeLV: feline leukemia virus
FIV: feline immunodeficiency virus
GI: gastrointestinal
IV: intravenous
MRI: magnetic resonance imaging
NSAID: nonsteroidal antiinflammatory drug
PO: by mouth (per os)
PT: prothombin time
RMSF: Rocky Mountain spotted fever
TVT: transmissible venereal tumor
vWD: von Willebrand's disease
vWF: von Willebrand factor

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Author: Elisa M. Mazzaferro, MS, DVM, PhD

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chapter 28

Esophageal Foreign Body



DEFINITION/OVERVIEW

- Occurs when a solid object becomes lodged in the esophagus.



ETIOLOGY/PATHOPHYSIOLOGY

- Foreign objects that are too large or irregular to pass through the esophagus and become wedged at normal points of narrowing, including the thoracic inlet, at the heart base, and distal esophagus.
- Occasionally smaller items (e.g., kibble) become lodged proximal to an esophageal stricture.
- Common esophageal foreign bodies include bones, chew toys, fish hooks, dental chew treats, rawhides, and hair balls (cats).

Systems Affected

- Gastrointestinal: esophagitis, esophageal stricture
- Respiratory: aspiration pneumonia, pneumothorax



SIGNALMENT/HISTORY

- Small breed dogs are more frequently affected, with as many as 84% of esophageal foreign bodies occurring in dogs <8 kg when dental chew treats are responsible, and approximately 67% when the items are bones or toys.
- No sex, breed, or age predilection, although younger animals more often have dietary indiscretion.

Risk Factors/Causes

- Preexisting esophageal stricture.

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Historical Findings

- The most common clinical signs include gagging, regurgitation, vomiting, anorexia, ptyalism, lethargy, and cough.
- Ptyalism is observed more frequently in cats.



CLINICAL FEATURES

- Most commonly gagging or dysphagia, regurgitation, anorexia, ptyalism, lethargy, cough, pain, and frequent swallowing.
- Ptyalism is observed more frequently in cats.
- Tachypnea can be present due to concurrent pneumonia or pain.



DIFFERENTIAL DIAGNOSIS

- Esophagitis
- Megaesophagus, primary or secondary
- Esophageal stricture
- Vascular ring anomaly
- Esophageal neoplasia



DIAGNOSTICS

Thoracic and Cervical Radiographs

- Often reveal an esophageal foreign body; although it can be poorly defined and appear as a focal soft tissue density (Fig. 28.1).



Fig. 28.1. Dental chew treat lodged in the midesophagus in a dog.

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- The opposite lateral view can be helpful when the foreign body is poorly delineated.
- Pneumomediastinum, mediastinal edema, or pleural effusion are suggestive of esophageal perforation; repeat radiographs should be considered following endoscopic foreign body removal.

Radiographic Contrast Esophagram

- Some esophageal foreign bodies require radiographic contrast swallow to aid delineation, especially dental chew treats. Avoid barium if there is risk of aspiration.

Esophagoscopy

- Foreign body will be readily visualized (Fig. 28.2).

Pathologic Findings

- Esophageal foreign bodies can cause significant esophageal inflammation, which leads to a greater risk of stricture or cicatrix formation.
- Pressure necrosis and esophageal perforation can also develop.
- Esophageal perforation will lead to local inflammation and infection (mediastinitis/pleuritis); pneumomediastinum or pneumothorax may also be present.

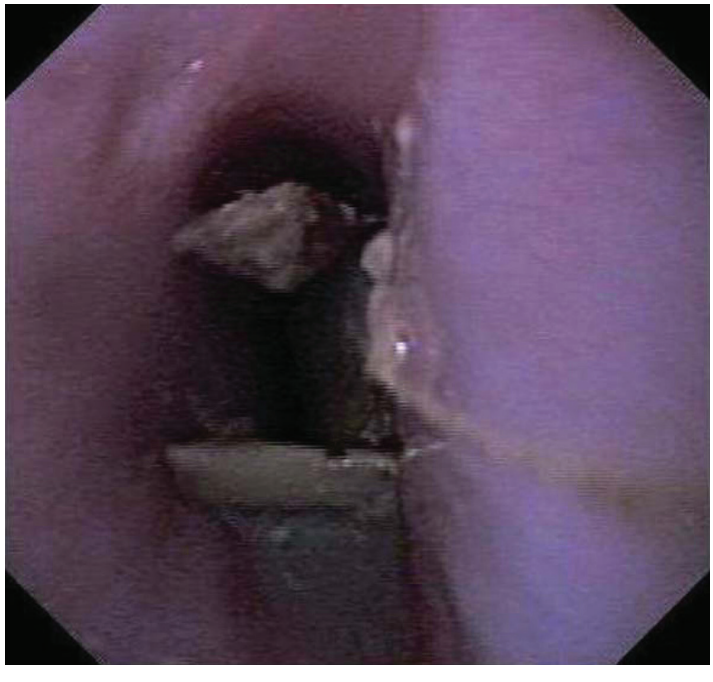


Fig. 28.2. Esophagoscopy demonstrating bone foreign body lodged in the esophagus.

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THERAPEUTICS

- The goal of therapy is to remove the foreign body and avoid complications.
- Esophageal foreign bodies are often removed via endoscopic techniques; orad removal is preferred and achieved in approximately 80% of cases. Various graspers and forceps are utilized.
- When objects are difficult to grasp, an inflatable object (e.g., Foley urinary catheter) can be passed distal to the object. The balloon is subsequently inflated, and the catheter withdrawn to pull the foreign object orad.
- If the foreign object cannot be removed in an orad direction, then attempts should be made to push it into the stomach. The endoscope itself or other rigid objects (e.g., orogastric tube, long-handled forceps, well-lubricated piece of PVC pipe) can be utilized.
- Judicious caution must be employed to avoid damaging a friable esophagus that might lead to esophageal perforation.

Drugs of Choice

- Antacids are often utilized following foreign body removal.
  - H_2 antagonists (famotidine, 0.5 mg/kg twice a day)
  - Proton pump inhibitors (omeprazole, 0.7–1.0 mg/kg PO twice a day, pantoprazole 1 mg/kg IV once a day), and sucralfate (0.5–1.0 g PO three times a day)
- Analgesics are indicated as esophageal injury is often painful.
- Opioids (e.g., tramadol 1–4 mg/kg 2–3 times a day)
- Avoid NSAIDs for management of pain following esophageal foreign body removal.
- Topical anesthesia can be fashioned with oral lidocaine mixtures.

Diet

- Gastrostomy tubes should be considered when there is severe esophageal inflammation and risk of developing an esophageal stricture.

Activity

- Restrict activity according to recovery from anesthesia or surgery.

Surgical Considerations

- Surgical exploration (thoracotomy) may be required when esophageal foreign bodies cannot be removed or esophageal perforation has occurred.
- Gastrotomy should be performed for esophageal foreign bodies that are advanced into the stomach and are likely to cause gastric or intestinal obstruction. Bone foreign bodies will often dissolve, but can possibly cause an obstruction if they move into the intestinal tract.

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COMMENTS

- Foreign body removal with rigid endoscopes has been reported to be more successful than flexible endoscopes.
- Repeat thoracic radiographs to assess for esophageal perforation (e.g., pneumomediastinum; Fig. 28.3) following foreign body retrieval should be considered when retrieval was difficult or the esophagus is particularly damaged.
- When there is significant esophageal inflammation, consider repeat endoscopy in 5–10 days to examine for esophageal stricture formation.

Client Education

- Following esophageal foreign body removal, clients should monitor closely for signs of regurgitation, which may indicate development of an esophageal stricture or cicatrix.

Prevention/Avoidance

- Bones, large chew toys, and dental chew treats should be avoided.

Possible Complications

- Esophageal stricture, approximately 10–20% of cases; more frequently with dental chew treats.

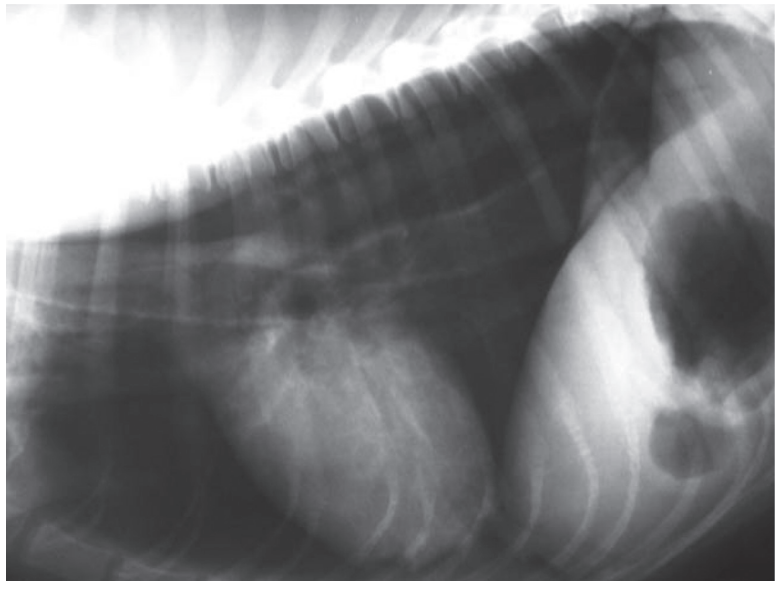


Fig. 28.3. Lateral thoracic radiograph of a dog with an esophageal foreign body caudal to the carina. Note the prominent aorta and soft tissues due to air in the mediastinal tissues.

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- Esophagitis occurs in the majority of cases (85%).
- Esophageal perforation and mediastinitis/pleuritis, approximately 12% of cases.

Expected Course and Prognosis

- Prognosis is fair to good. Overall mortality rates are 7–25%; esophageal foreign bodies due to dental chew treats tend to have more complications (e.g., perforation or stricture) and a higher mortality rate.

Abbreviations

IV: intravenous
NSAID: nonsteroidal antiinflammatory drug
PO: per os

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chapter 29

Ethylene Glycol Toxicity



DEFINITION/OVERVIEW

- Ethylene glycol is a dihydric alcohol compound that is water soluble, and is used in many commercially available antifreeze preparations used to prevent freezing of automotive radiators.
- Ethylene glycol often is sweet-tasting, making it desirable and palatable for consumption by small animals.
- Other potential sources of ethylene glycol include paint preparations, windshield wiper/deicing fluid, photography developing solutions, brake fluid, and rust and paint/wood solvents.



ETIOLOGY/PATHOPHYSIOLOGY

- Following ingestion, ethylene glycol is quickly absorbed from the gastrointestinal tract within 1 hour, and reaches peak concentrations in the blood and peripheral tissues within 3–4 hours of ingestion.
- Ethylene glycol is metabolized by hepatic alcohol dehydrogenase within 1–2 hours of ingestion to a number of toxic by-products that include glycoaldehyde, glycolic acid, glyoxylic acid, hippuric acid glycine, formic acid, oxalic acid, and calcium oxalate.
- The presence of by-products and acids causes a high anion gap metabolic acidosis.
- Serum calcium binds with oxalic acid and creates calcium oxalate crystals that precipitate in peripheral tissues, including the brain and kidneys.
- Precipitation of calcium oxalate crystals in the kidneys leads to AKI, ARF, and death.

Systems Affected

- Renal/urologic:
  - Precipitation of calcium oxalate crystals in renal tubules causes renal tubular damage.
  - Azotemia with elevated BUN and creatinine.
  - Hyperphosphatemia.
  - Calcium oxalate monohydrate crystalluria, renal tubular casts, glucosuria, and hematuria may be present.

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- Metabolic:
  - High anion gap metabolic acidosis
    - Anion gap (AG) = $(Na^+ + K^+) = (Cl^- + HCO3^-)$
    - Normal is 10–20; unmeasured anion metabolites of ethylene glycol cause AG to become characteristically > 40
  - Hyperosmolality
  - Hyperglycemia
  - Hypocalcemia
  - Acidemia with pH <7.2
- Gastrointestinal: nausea, inappetence, ptyalism, vomiting, hematemesis
- Cardiovascular: tachycardia
- Pulmonary: panting/tachypnea
- Neurologic: ethylene glycol crosses the blood–brain barrier, and can cause sedation; once uremia secondary to renal failure occurs, seizure, somnolence, coma and death can result.



SIGNALMENT/HISTORY

- Ethylene glycol exposure can occur at any time but appears to be seasonal when antifreeze is being changed from motor vehicles, and opening or closing up recreational vehicles such as campers, motorhomes, and seasonal cabins (spring, summer, and fall).
- Witnessed ingestion.
- Potential for purposeful poisoning.

Risk Factors/Causes

- More commonly seen in animals that are allowed outdoors unattended, but often exposure occurs at home in a driveway or garage.
- More commonly reported in areas of the country that require antifreeze for automotive radiators to prevent freezing.
- Ethylene glycol has a sweet taste, making it palatable for dog and cats to ingest.



CLINICAL FEATURES

- Phase 1 (30 minutes to 12 hours after ingestion): ataxia, polydipsia, polyuria, increased respiratory rate
- Phase 2 (12–14 hours after ingestion): lethargy, depression, anorexia, nausea/ptyalism, vomiting, dehydration
- Phase 3 (12–24 hours or more after ingestion): progressive lethargy, vomiting, uremic oral and gastrointestinal ulcers, ptyalism, halitosis, dehydration, oliguria, anuria, seizure, coma, death

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DIFFERENTIAL DIAGNOSIS

- Toxins that cause ataxia/sedation: alcohol (ethanol, methanol), marijuana, xylitol, propylene glycol, ivermectin, barbiturates, benzodiazepines
- Other causes of high anion gap metabolic acidosis: diabetic ketoacidosis (DKA), salicylate intoxication, other causes of uremia, methanol, ethanol
- Hypoglycemia secondary to sepsis, hypoadrenocorticism, insulin overdose, xylitol
- Nonspecific gastroenteritis
- Other causes of renal failure: toxin (lily, aminoglycoside antibiotics, NSAID, grape/raisin, cyclosporine, acetaminophen), infectious (leptospirosis, Lyme nephritis), hypotension

DIAGNOSTICS

- Anion gap: high anion gap metabolic acidosis.
- Blood pH: metabolic acidosis with low blood pH/acidemia, and low $HCO3^-$ and low total CO_2.
- Electrolytes: hyperkalemia, hypocalcemia.
- Cageside test for ethylene glycol (Kacey test).
  - Measures ethylene glycol in blood. False-negative tests can occur after 12 hours of ingestion, if ethylene glycol has been metabolized to toxic metabolites.
  - In some instances, the level of detection/sensitivity of the test (50 mg/dL) is below the level required for toxicity, so false negatives can occur in cats and small dogs. False positives occur with propylene glycol and drugs with propylene glycol carriers (etomidate, diazepam), as well as with some activated charcoal preparations.
- Biochemistry: azotemia (elevated BUN and creatinine) occurs 24 hours or more after ingestion, hyperphosphatemia can occur early due to rust inhibitors, and later as renal failure progresses and renal excretion of phosphorus decreases.
- Urinalysis: isosthenuria (within 3 hours of ingestion), glucosuria, calcium oxalate crystalluria (monohydrate > dehydrate), proteinuria, renal tubular casts, hematuria.
- Serum osmolality: (normal 280–310 mOsm/kg) elevated up to 450 mOsm/kg within 3 hours of ingestion.

$$1.86(Na^+ + K^+) + \text{glucose}/18 + \text{BUN}/2.8 + 9$$

Other Laboratory Tests

- Ultraviolet lamp (Wood's lamp): may fluoresce green around paws, face/muzzle, and prepuce/vulva, due to fluorescent dye added to antifreeze solutions; false positives can occur.
- Human hospital GCMS.

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Imaging

- Abdominal radiographs: subjectively, kidneys may appear more radioopaque.
- Abdominal ultrasound: kidneys may be normal to enlarged, and have hyperechoic renal cortices (hyperechoic "rim sign").



THERAPEUTICS

- Induction of emesis (apomorphine 0.02–0.4 mg/kg IV) within 1 hour of ingestion.
- Intravenous crystalloid fluid for rehydration and maintenance fluid requirements (2–3× maintenance unless concurrent significant cardiac disease is present).
- Sodium bicarbonate to treat severe metabolic acidosis pH <7.1.
- Antiemetic drugs:
  - Maropitant 1 mg/kg IV, SQ q24h
  - Dolasetron 0.6 mg/kg IV q24h
- Proton pump inhibitors:
  - Pantoprazole 1 mg/kg IV q24h
  - Omeprazole 1 mg/kg PO q12h (canine), q24h (feline)
- Histamine receptor blockade:
  - Famotidine (0.5–1 mg/kg IV q12–24h)
- Sucralfate (0.25–1 g PO q8h)
- Phosphorus/phosphate binders:
  - Aluminum hydroxide (2–10 mL PO q6–8h)

Drugs/Antidotes of Choice

- 4-Methylpyrazole (4-MP): a direct inhibitor of the alcohol dehydrogenase enzyme, causing ethylene glycol to be eliminated unchanged, before conversion to toxic metabolites.
  - Is effective in dogs if administered within 8 hours of ingestion, effective in cats if administered within 3 hours of ingestion.
  - Canine dose: 20 mg/kg IV once, then 15 mg/kg 12 and 24 hours after first dose, then 5 mg/kg at 36 hours after first dose.
  - Cats: effective dose for cats is 125 mg/kg IV once, then 31.25 mg/kg IV at 12, 24, and 36 hours after the first dose.
  - CNS depression can occur.
  - Trembling, ptyalism, tachypnea, anaphylaxis (rare).
- Ethanol: continue until ethylene glycol test is negative.
  - Canine.
    - 20% ethanol, canine: 5.5 mL/kg IV every 4 hours for five treatments, then 5.5 mL/kg IV every 6 hours for five treatments
    - 5% ethanol as a constant rate infusion of 5.5 mL/kg/h until ethylene glycol test is negative
    - 5% ethanol 12 mL/kg IV, followed by 2 mL/kg/h IV CRI

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- Feline.
  - 20% ethanol 5.5 mL/kg IV every 6 hours for five treatments, then every 8 hours for five treatments
  - 30% ethanol 1.3 mL/kg IV then 0.42 mL/kg/h for 48 hours
- Vomiting, depression, hypoventilation, and hypothermia can occur as a result of ethanol therapy.



COMMENTS

- Neither 4-MP nor ethanol is effective if started more than 8 hours after ingestion of ethylene glycol.

Miscellaneous

- Prevention is the best method of avoiding ethylene glycol toxicity (keeping animals leashed/indoors/supervised).
- Hemodialysis may be effective in removal of ethylene glycol from circulation, if initiated within 5–6 hours of ingestion.

Patient Monitoring

- Urine output: maintain at a minimum of 1 mL/kg/h
- Serial body weight to measure hydration
- Urine/fluid "ins and outs"
- Blood pressure
- Ionized calcium, venous blood gas, electrolytes
- BUN and creatinine

Possible Complications

- Vomiting
- Somnolence/coma
- Dehydration
- Metabolic acidosis
- Renal failure
- Hyperkalemia
- Atrial standstill
- Oligoanuria
- Seizures
- Death

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Expected Course and Prognosis

- Prognosis is good with early recognition and treatment (antidote within 8 hours for dogs, and within 3 hours for cats).
- Prognosis is poor to grave if azotemia, oliguria or anuria exists.
- Recovery of renal tubular function may take weeks to months.
- Overall mortality ranges from 59% to 70%.

Synonyms

- Antifreeze

Abbreviations

4-MP: 4-methylpyrazole
AG: anion gap
AKI : acute kidney injury
ARF: acute renal failure
BUN: blood urea nitrogen
CNS: central nervous system
CRI: constant rate infusion
GCMS: gas chromatography mass spectometry
IV: intravenous
NSAID: nonsteroidal antiinflammatory drug

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chapter 30

Feline Bronchitis

DEFINITION/OVERVIEW

- Chronic bronchitis: inflammation in the airways (bronchi and bronchioles); presents clinically as a chronic cough of greater than 2 months' duration in dogs, although the duration of cough in cats has not been defined.
- Asthma: acute or chronic airway inflammation associated with increased responsiveness of the airways to various stimuli, airway narrowing due to smooth muscle hypertrophy/constriction, reversibility of airway constriction, and presence of eosinophils, lymphocytes, and mast cells within the airways.
- These criteria are often difficult to determine or document, thus feline bronchopulmonary disease (FBD) is used more commonly to describe the clinical disease in cats of acute or chronic coughing or wheezing accompanied by lower airway inflammation.

ETIOLOGY/PATHOPHYSIOLOGY

- Noxious (e.g., smoke) or allergic stimuli trigger inflammation within the lower airways. The inflammatory mediators cause mucosal airway damage that releases more inflammatory mediators.
- Bronchiolar smooth muscle constriction – reversible either spontaneously or in response to antiinflammatory drugs or beta-2 agonists.
- Smooth muscle hypertrophy implies chronicity: usually not reversible.
- Increase in mucosal goblet cells, mucus production, and edema of bronchial wall.
- Excessive mucus can cause bronchiolar obstruction and lead to atelectasis or bronchiectasis (dilated airways often obstructed with mucus).
- Chronic inflammation within the airways may lead to fibrosis.

Systems Affected

- Respiratory.
- Cardiac: chronic airway disease can lead to pulmonary hypertension and secondary right-sided heart disease; this is rarely appreciated in cats.

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Geographic Distribution

- Worldwide. Parasitic causes of airway inflammation are more common in southern and midwest US. Heartworm disease is more prevalent in southern US. *Paragonimus kellicotti* is found in the Great Lakes region. *Aelurostrongylus abstrusus* is found worldwide, and is particularly common in free-roaming cats.



SIGNALMENT/HISTORY

Species

- Cats.

Breed Predilections

- Siamese overrepresented in some studies.

Mean Age and Range

- Any age; more common between 2 and 8 years. Older cats (>12 years) rarely have new-onset FBD; if suspected, careful clinical evaluation should be performed to exclude other causes.

Predominant Sex

- One study shows females overrepresented but this is not a consistent finding.

Historical Findings

- Coughing (80%), sneezing (60%), labored breathing or wheezing (40%).
- Signs are typically episodic and can be acute or chronic.
- Lethargy and inappetence are occasionally reported.

Physical Examination Findings

- Severely affected cats may present with open mouth breathing, tachypnea, and cyanosis.
- Increased tracheal sensitivity is common.
- Chest auscultation may reveal crackles or expiratory wheezes or may be normal.
- Labored breathing, typically a moderate-to-severe increase in expiratory effort with an abdominal push on expiration. Inspiratory effort is usually much less affected.
- Heart rate is typically normal to bradycardic, although stress may result in tachycardia.

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Risk Factors/Causes

- Exposure to cigarette smoke, dusty cat litter, hair sprays, and air fresheners could possibly exacerbate disease in some cats.
- Parasitic lung infections are more common in outdoor cats in certain geographic locations.
- Use of potassium bromide has been implicated as a cause for signs of bronchitis or asthma in some cats and for this reason, KBr is rarely used in cats.
- Triggers of airway inflammation are largely unknown.



DIFFERENTIAL DIAGNOSIS

- Diseases primarily affecting lung parenchyma. Rule out infectious pneumonia (toxoplasmosis, FIP, bacterial pneumonia, histoplasmosis). Idiopathic pulmonary fibrosis may also result in respiratory distress.
- *Dirofilaria immitis* (heartworm) and primary lung parasites (*Aelurostrongylus abstrusus*, *Capillaria aerophilia*, and *Paragonimus kellicotti*).
- Primary or metastatic neoplasia also similar in clinical and, occasionally, radiographic appearance. Severe bronchial disease may occasionally be radiographically mistaken for metastatic disease.
- Primary cardiac disease may appear clinically and radiographically similar in certain instances, but these cats typically do not have a history of cough or have tracheal sensitivity on physical examination.



DIAGNOSTICS

Complete Blood Count/Biochemistry/Urinalysis

- Frequently normal.
- Less than 40% of cats with allergic airway disease have a peripheral eosinophilia. Peripheral eosinophilia is more common with parasitic infection or eosinophilic pulmonary granulomatosis.

Other Laboratory Tests

- Fecal exams: flotation examination for *Capillaria*, sedimentation for *Paragonimus*, Baermann technique for *Aelurostrongylus*. False negatives occur.
- Heartworm testing: both antigen and antibody test recommended.
- Pulmonary lung function testing: available in select academic institutions or specialty practices. Increased airway resistance is the classic finding; affects expiratory parameters more than inspiratory.
- RAST or intradermal skin testing: a correlation between skin and respiratory allergies has not been documented at this time.
- Feline NT-proBNP: a negative result may aid in excluding cardiac disease as a cause of labored breathing and heighten suspicion of bronchitis.

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Imaging

Radiography

- Classically, diffuse bronchial wall thickening and interstitial pattern (Figs 30.1, 30.2).
- Patchy alveolar pattern can be seen as well.
- The severity of radiographic changes does not necessarily correlate with clinical severity or duration.

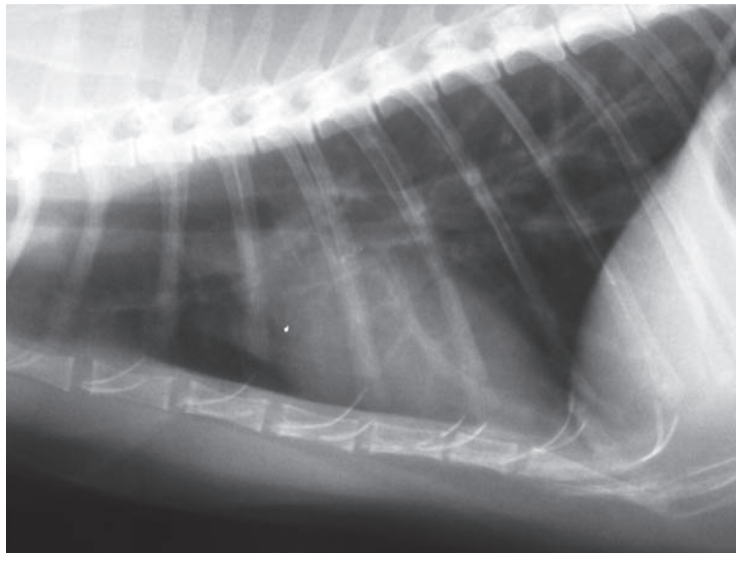


Fig. 30.1. Lateral thoracic radiograph of a cat with bronchitis. Note the bronchiolar thickening, also known colloquially as "donuts" and "tram lines," and hyperinflation of the diaphragm.

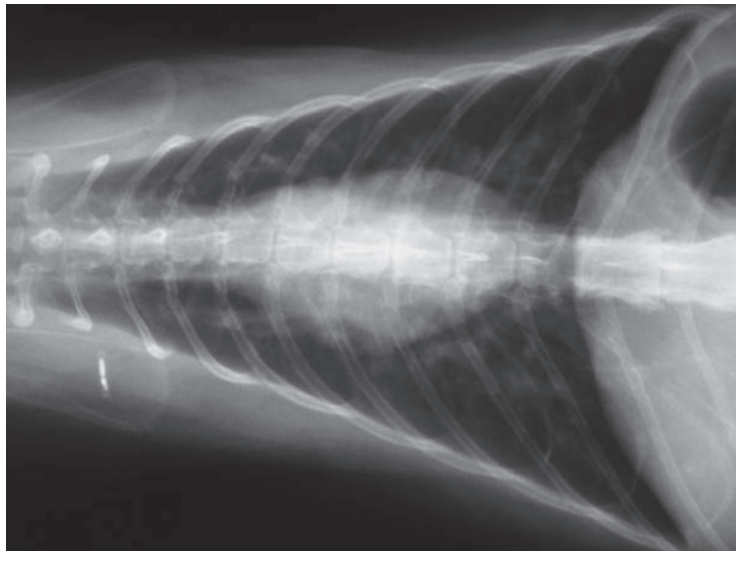


Fig. 30.2. Ventrodorsal thoracic radiograph of a cat with bronchitis. Note the bronchiolar thickening, also known colloquially as "donuts" and "tram lines," and hyperinflation of the diaphragm.

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- Hyperinflation of lung fields: characterized by a flattened and caudally displaced diaphragm, an increase in the distance between the heart and diaphragm or extension of the lungs to the first lumbar vertebra.
- Collapse of the right middle lung lobe has been reported with a frequency of 11% (Fig. 30.3).
- Pulmonary lobar arterial enlargement is suspicious for heartworm disease.

Echocardiography

- May be useful when evaluating for the possibility of heartworm disease or pulmonary hypertension secondary to chronic lung disease.

Diagnostic Procedures

Transoral Tracheal Wash

- Use a sterile endotracheal tube and polypropylene catheter to obtain a cytologic sample; this allows for sampling of airway fluids at the level of the carina.
- Does not allow for visualization of the lower airways or selective sampling of diseased lung.

Bronchoscopy/Bronchoalveolar Lavage

- Allows for visualization of trachea and bronchi. Excessive amounts of thick mucus are common with bronchitis. Mucosa of the airways is typically hyperemic and edematous.
- Biopsy or endoscopic brushing possible, although typically not needed with cases of bronchitis.

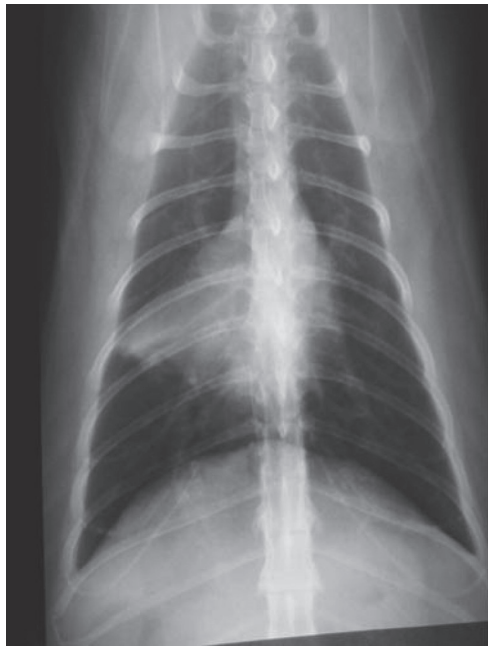


Fig. 30.3. Ventrodorsal thoracic radiograph of a cat with bronchitis and atelectasis of the right middle lung lobe. This may be mistaken in some cases for aspiration pneumonia. Note the thickened bronchiolar markings which are supportive of bronchitis, rather than aspiration pneumonia.

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- BAL should be performed during bronchoscopy; this allows for sampling of airway fluids from areas that appear most affected.

Cytology

- Eosinophils and neutrophils are the most prominent cell types. A mixed cell population occurs in about 21% of cats.
- In one study, 22% of cats had normal cytology (macrophages predominated).
- Up to 30% eosinophils on BAL cytology can be found in normal cats. Parasitic and heartworm infections will have a high percentages of eosinophils on cytology (Table 30.1).

Bacterial Cultures

- Quantitated cultures are recommended. Significant bacterial colony counts are uncommonly obtained in FBD. Significant bacterial growth on a quantitative culture is suggested to be >100–300 cfu/mL.
- *Mycoplasma* has been isolated from 21–44% of cats with FBD and was not isolated from healthy cats. Its role in bronchitis remains controversial.

Biopsy

- Keyhole biopsy is rarely indicated because of the invasiveness of the procedure. Rarely will the results alter the course of therapy, although they can help differentiate between idiopathic pulmonary fibrosis and bronchitis.

Pathologic Findings

- Histologic findings consistent with FBD include hyperplasia/hypertrophy of goblet cells, airway smooth muscle thickening, epithelial erosion, and inflammatory infiltrates.

TABLE 30.1. Mean (±SD) differential cell counts from bronchoalveolar lavage fluid obtained from normal cats in two independent studies.

Study	Padrid et al.	King et al.
Number	24	11
Total cell count/mL	303 (± 126)	241 (± 101)
% Macrophages	64 (± 22)	70.6 (± 9.8)
% Polymorphonuclear leukocytes	5 (± 3)	6.7 (± 4)
% Eosinophils	25 (± 21)	16.1 (± 6.8)
% Lymphocytes	4 (± 3)	4.6 (± 3.2)
% Mast cells	<1 (± <1)	Not reported
% Epithelial cells	2 (± 2)	Not reported
% Goblet cells	<1 (± <1)	Not reported

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THERAPEUTICS

Drugs of Choice

Emergency Treatment

- Combine the use of oxygen and a parenteral bronchodilator. A sedative often aids in decreasing anxiety associated with hypoxia. A short-acting parenteral corticosteroid may also be required. Patient manipulation should be minimized in order to avoid worsening respiratory distress.
- Injectable terbutaline (0.01 mg/kg IV or SQ). You can repeat this dose if no clinical improvement (decrease in respiratory rate or effort) in 20–30 minutes.
- Dexamethasone sodium phosphate (0.25–0.5 mg/kg, given IV or SQ). Can repeat if no improvement visible within 20–30 minutes. Prednisolone sodium succinate (Solu-Delta-Cortef) can also be used (50–100 mgIV).
- Butorphanol tartrate (0.4 mg.kg IV or IM) can be used for sedation. Also consider buprenorphine (0.01 mg/kg) or acepromazine (0.0.01–0.05 mg/kg).

Long-Term Management

Corticosteroids

- Decrease inflammation.
- Oral treatment is preferred over injectable because doses and duration can be more closely monitored.
- Prednisolone: 0.5–1 mg/kg PO every 12 hours. Begin to taper dose (50% each week) after 2 weeks if clinical signs have improved. If clinical signs resurface, dose should be increased again to 0.5–1 mg/kg PO every 12 hours. Maintenance therapy: 0.5–1 mg/kg PO every 24–48 hours. Some patients may be able to use steroids on a seasonal basis only.
- Dexamethasone sodium phosphate: used mainly for an acute crisis.
- Longer-acting parenteral steroids (Vetalog or Depomedrol) should be reserved for situations where owners are unable to administer oral medication on a routine basis.

Inhaled Corticosteroids

- Newer therapy: requires a form-fitting facemask, spacer, and MDI (Fig. 30.4). Veterinary brands include Aerokat (Trudell Medical).
- The corticosteroid most commonly used in cats as an MDI is fluticasone propionate (Flovent). The 220 µg or 110 µg Flovent MDI is recommended (2 actuations, 7–10 breaths every 12 hours) along with other bronchodilators and oral corticosteroids, depending on the severity of clinical signs.
- Flovent is used for long-term control of airway inflammation. Takes 10–14 days to reach peak effect; during this time oral steroids should be used concurrently.
- It has been shown that there is some suppression of the hypothalamic-pituitary axis in cats with the use of inhaled corticosteroids. Systemic side effects appear to be significantly less than with parenteral or oral medications.

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Fig. 30.4. Inhalation therapy with a metered-dose inhaler and facemask in a cat with lower airway disease.

- Methylxanthines (i.e., theophylline and aminophylline) inhibit smooth muscle constriction. The pharmacokinetics of aminophylline would suggest that it is unlikely to be an effective treatment. Sustained-release theophylline formulations are often recommended, but the pharmacokinetics can vary greatly depending on the brand. The most recent recommended brands (Inwood brand at 10–15 mg/kg PO once a day) are becoming increasingly difficult to obtain. In general, theophylline is rarely helpful.
- Beta-2 agonists (i.e., terbutaline, albuterol) should be used in a crisis, not routinely as resistance may develop.

Inhaled Bronchodilators

- Albuterol is the preferred inhalant bronchodilator therapy in cats, providing immediate relief of bronchoconstriction; its effect lasts less than 4 hours.

Anthelminthics

- These are routinely recommended for cats with clinical signs of FBD and airway cytology that is predominantly eosinophilic. Parasitic bronchitis can be difficult to diagnose based on airway cytology and fecal examination; empirical therapy is indicated with appropriate clinical signs and geographic location.
- Appropriate medication will depend on the specific parasite suspected in the geographic region. Consider fenbendazole as the most likely to be effective.

Antibiotics

- Use should be based on a positive quantitative culture. Choice of antibiotic therapy is based on susceptibility testing; the role of *Mycoplasma* is still being elucidated, but is likely important in diseased airways. Empiric antibiotic therapy should include coverage for *Mycoplasma*.

Contraindications

- Beta-2 antagonists (e.g., propranolol) are contraindicated because of their ability to block sympathetically mediated bronchodilation.

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Precautions/Interactions

- Bronchodilators may exacerbate underlying cardiac disease.
- Long-term use of steroids increases risk of development of diabetes mellitus and predisposes to immunosuppression.
- Use of corticosteroids in cats may unmask occult cardiomyopathy and precipitate congestive heart failure.
- Fluoroquinolones decrease the metabolism of methylxanthines in dogs, although this has not been investigated in cats. Consider decreasing the dose of methylxanthine by 50% when used concurrently with a fluoroquinolone. Watch for toxic side effects of the methylxanthine (e.g., vomiting, diarrhea, tachycardia).

Activity

- Usually self-limited by patient.

Diet

- Calorie restriction for obese cats.

Appropriate Healthcare

- Removal of patient from the inciting environment may help.
- Patient should be hospitalized for an acute crisis of respiratory distress.

Nursing Care

- Oxygen therapy and sedatives may help in an acute crisis. Minimize manipulation during a crisis in order to lessen stress and oxygen needs of the animal.



COMMENTS

Client Education

- Most causes of bronchitis are chronic, progressive diseases.
- Do not discontinue medical therapy when clinical signs have resolved; subclinical inflammation within the lungs is common and can lead to progression of disease.
- Lifelong medication and environmental changes may be necessary.
- Most clients can be taught to give terbutaline subcutaneously and corticosteroid injections at home for a crisis situation.

Patient Monitoring

- Owners should watch for and report any increase in coughing, sneezing, wheezing, or respiratory distress. Medications should be increased appropriately if clinical signs recur.

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- Follow-up radiographs are helpful in the first weeks after initial diagnosis to evaluate improvement with medical therapy.
- Long-term use of corticosteroids will require blood glucose monitoring every 3–6 months to screen for diabetes mellitus. Urinary tract infections can occur owing to immunosuppression. Owner should watch for signs of PU/PD that may indicate diabetes mellitus or renal disease.

Prevention/Avoidance

- Eliminate any environmental factors that may trigger a crisis situation (see Risk Factors/Causes).
- Change furnace and air conditioner filters on a regular basis.
- Consider dust-free litters.

Possible Complications

- Refractory cases or untreated acute episodes can be life threatening.
- Right-sided heart disease may develop as a result of long-term bronchitis.

Expected Course and Prognosis

- Long-term therapy should be expected.
- Most cats do well if recurrence of clinical signs is carefully monitored and medical therapy appropriately adjusted.
- A few cats will be refractory to treatment; these carry a much worse prognosis.

Associated Conditions

- Cor pulmonale can be a sequela to chronic lower airway disease.

Pregnancy/Fertility/Breeding

- Glucocorticoids are contraindicated in the pregnant animal. Bronchodilators should be used with caution.

Synonyms

- Allergic bronchitis, chronic obstructive pulmonary disease, asthmatic bronchitis, feline lower airway disease, extrinsic asthma, eosinophilic bronchitis, immune-mediated airway disease

Abbreviations

BAL: bronchoalveolar lavage
BNP: brain natriuretic peptide

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FBD: feline bronchopulmonary disease
FIP: feline interstitial pneumonia
IM: intramuscular
IV: intravenous
MDI: metered-dose inhaler
PO: per os
PU/PD: polyuria/polydipsia
RAST: radioallergosorbent testing
SQ: subcutaneously

Suggested Reading

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Internet Resources

www.Aerokat.com: internet source for ordering facemasks and spacers for inhalant therapy

www.fritzthebrave.com: good source for clients to learn about feline bronchitis and research the possibility of inhaled medications in their cat

Author: Elizabeth Rozanski, DVM

Acknowledgment to the author of the original chapter in *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Emergency and Critical Care, First Edition:* Carrie J. Miller.

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chapter 31

Feline Infectious Peritonitis

DEFINITION/OVERVIEW

- FIP, first discovered in the 1960s, is the term describing a complex systemic clinical syndrome of domestic, and certain nondomestic, cats resulting from infection with a virulent biotype of FCoV.

ETIOLOGY/PATHOPHYSIOLOGY

- The clinical syndrome called FIP is known to be caused by a virulent FCoV called FIPV.
- All FCoVs have the potential to cause FIP. However, not all cats with FIPV infection will develop clinical disease.
- Genetic susceptibility among certain breeds (e.g., Persian), and lines within breeds, may influence whether or not clinical illness develops.
- The pathophysiology of FCoV infection, and subsequent development of FIP, is complex and is not completely understood.
- FCoV infection occurs subsequent to fecal–oral transmission, commonly between a queen and her kittens, especially through contaminated litter.
- Infection via the respiratory tract has been suggested.
- FCoV replication within intestinal epithelium is rapid; viral shedding can occur in feces within 2 days of ingestion.
- FCoV infection may be subclinical (asymptomatic), may lead to diarrhea only, or may cause multisystemic disease.
- Most infections are subclinical.
- It is not possible to predict the outcome following infection.
- Multisystemic disease associated with FCoV infection is an immune-mediated disease leading to widespread vasculitis, a characteristic lesion of FIP.
- Once systemic dissemination of virus occurs, either of two clinical variations, effusive ("wet") or noneffusive ("dry") FIP, may result.

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- Effusive FIP usually develops within 4–8 weeks following infection and typically develops in the peritoneal or pleural cavities. Effusion is the result of vasculitis, vessel destruction, and the formation of pyogranulomata.
- Noneffusive FIP is a more chronic disease resulting from a partial cell-mediated immune response; clinical signs manifest months or years following initial infection.
- Why some cats never develop FIP following FCoV infection is not clear.
- Protection is generally associated with CMI, while a systemic humoral immune response is considered to be harmful or may predispose cats to severe disease following subsequent infection (antibody-dependent enhancement).



SIGNALMENT/HISTORY

- Transient vomiting or diarrhea may warrant veterinary attention during the earliest stages of clinical FIP (coronaviral enteritis).
- Although cats of any age may develop clinical FIP, about 50% are 2 years of age or younger.
- A history of having been group-housed (i.e., shelter, breeding cattery, rescue group) is common.
- Cats with effusive FIP may present with a distended abdomen (abdominal cavity effusion) or respiratory distress/tachypnea (thoracic cavity or pericardial effusion) (Fig. 31.1).



Fig. 31.1. Abdominal distension in a 1-year-old cat diagnosed with effusive feline infectious peritonitis.

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Risk Factors/Causes

- Risk of FIP is greatest among young cats (usually less than 2 years of age) housed in multicat households.
- Certain breeds (e.g., Persian), and lines within breeds, appear to be more likely to develop FIP subsequent to FCoV infection.
- Stress (e.g., recent surgery, rehousing, cluster housing) may be an important factor contributing to viral shedding and the signs of FIP in FCoV-infected cats.



CLINICAL FEATURES

- Abdominal effusion (see Fig. 31.1) or respiratory difficulty, associated with pleural cavity effusion or pericarditis, are among the most common presenting clinical manifestations of effusive FIP.
- Other nonspecific signs may include pallor, icterus, lethargy, palpable abdominal masses (adhesions or lymphadenomegaly), scrotal enlargement, and fever.
- Cats with noneffusive FIP typically present with vague clinical signs.
    - Abdominal palpation may reveal nodular irregularities on viscera or enlarged lymph nodes.
    - Several ocular abnormalities have been described in cats with FIP, including iritis (color changes), anterior uveitis, keratic precipitates, and retinal detachment or hemorrhage.
    - Neurologic signs are reported in 25–33% of cats with noneffusive FIP. Nystagmus, ataxia, and seizures are among the most common neurologic signs reported.
    - FIP is not a recognized cause of "fading kitten syndrome" (i.e., death occurring between birth and weaning), but it is recognized as a cause of death among kittens after weaning.



DIFFERENTIAL DIAGNOSIS

- The lack of specificity among the spectrum of clinical signs associated with FIP warrants testing any sick cat for FeLV antigen and FIV antibody.
- Pregnancy, abdominal neoplasia (lymphoma), hepatic failure, and hydronephrosis should be considered in cats presenting with abdominal distension.
- Cats presented with respiratory distress should be evaluated for cardiomyopathy, congestive heart failure, heartworm disease, pneumonia, lymphoma, and restrictive pleuritis.



DIAGNOSTICS

- Antemortem diagnosis of FIP, particularly noneffusive FIP, is difficult and requires a comprehensive assessment of the patient's history, clinical signs, and laboratory test results.
- Besides histopathology, there is no single diagnostic test that can confirm a diagnosis of FIP.

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- Hematology may reveal lymphopenia and neutrophilia with a left shift and mild, nonregenerative anemia (hematocrit <30%).
- Hypoalbuminemia with hyperglobulinemia (albumin-to-globulin ratio ≤0.45) is highly suggestive of FIP in cats with associated clinical signs and is negative for FeLV and FIV.
- Approximately 75% of cats with noneffusive FIP have abnormal serum proteins, making this simple test an important diagnostic parameter.
- In cats with effusive FIP, fluid analysis is extremely useful in establishing a clinical diagnosis.
    - Physical appearance of the fluid collected from the abdominal or thoracic cavities reveals a viscous, yellow to straw-colored fluid that may form clots on standing and froths when shaken.
    - Cytological assessment reveals a relatively hypocellular fluid (<5000 cells/μL) consisting of neutrophils and macrophages (pyogranulomatous fluid) that is absent of bacteria or neoplastic cells (Fig. 31.2).
    - Additionally, effusion with a high total protein (>3.5 g/dL) or an albumin-to-globulin ratio <0.45 is highly supportive of FIP.
- Advanced diagnostics.
    - Serologic testing for FCoV antibody is commonly used in practice, but this test is neither a consistent nor reliable indicator of FIP and should not be used as a single-source diagnostic test.
    - Virus isolation from effusions of cats with FIP rarely yields positive results and, as such, availability is limited.
    - rtPCR has been successful in confirming the presence of FCoV in seronegative cats and detecting FCoV shedding. While this test is reported to have good sensitivity

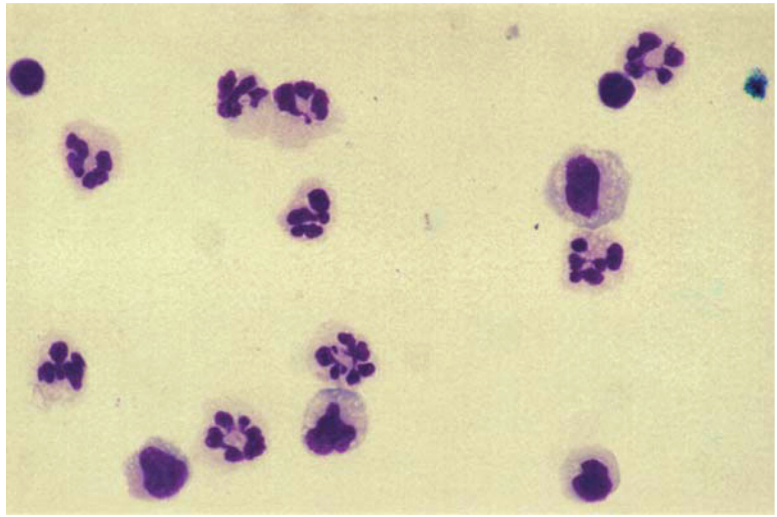


Fig. 31.2. Cytological appearance of abdominal effusion from a cat (see Fig. 31.1) with effusive feline infectious peritonitis; the pyogranulomatous effusion is characterized by the predominance of neutrophils and macrophages.

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and specificity, rtPCR for FCoV does not specifically identify FIPV or the cause of FIP, and a positive test does *not* predict impending FIP in a healthy cat.

- Recently, a commercially available rtPCR test for the detection of FCoV mRNA in circulating macrophages has become available. The test is based on the fact that although avirulent FCoV can be found in peripheral blood, replication within macrophages, and expression of mRNA, are unique to FIPV.

Screening Healthy Cats

- Veterinarians may be requested to screen healthy cats for evidence of FIP. In such cases, it is reasonable to submit serum for FCoV antibody. Cats with a negative titer (usually less than 1:10 or 1:20) are unlikely to have been exposed to FCoV and are therefore acceptable candidates for use in breeding programs. However, the clinician must use a reliable laboratory that will perform titers to this level.

Pathologic Findings

- Histopathology is the diagnostic test of choice in confirming FIP wherein pyogranuloma is the defining lesion. Kidney, spleen, and mesenteric lymph nodes are representative tissues likely to be affected in cats with FIP (Fig. 31.3). Vasculitis is characteristically seen in the microvasculature of affected tissues.
- Pyogranulomatous mass lesions in the ileocecal colic junction have been reported in cats.

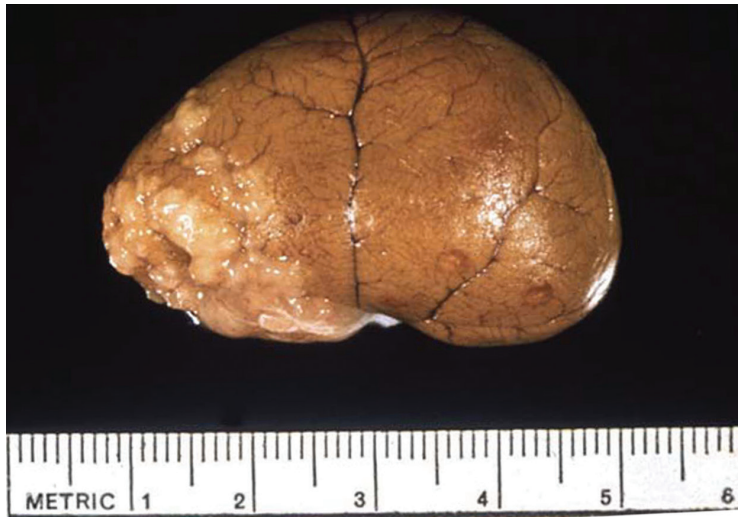


Fig. 31.3. Pyogranuloma on the cranial pole of a kidney from an adult cat that died of complications associated with noneffusive feline infectious peritonitis. Gross evidence of ischemic injury, subsequent to vasculitis, is present over the surface of the kidney.

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THERAPEUTICS

- In the absence of FCoV antiviral therapy, treatment centers on management of immune-mediated injury through judicious use of prednisolone (2–4 mg/kg per day PO once daily or divided into two doses; the dose can be reduced empirically every 10–14 days until an optimal response is achieved). Cats with FIP are expected to succumb from complications associated with the infection. Concurrent administration of antibiotics is indicated.
- Recombinant human IFN has been used in the United States as adjunctive therapy for cats with FIP. Because cats develop anti-IFN antibody, use of human IFN to treat FIP is no longer recommended. Recently, recombinant feline IFN-omega (currently not available in the United States) has been used concurrently with prednisolone to treat cats with FIP. However, benefits of supplemental IFN, without prednisolone, have not been established.
- Vitamins B_1 (thiamine), B-complex, C (ascorbic acid), and E, as well as anabolic steroids and thromboxane synthetase inhibitors, have been administered empirically as supportive treatment of cats with FIP. The clinical benefits associated with these products have not been defined.

Expected Course and Prognosis

- The prognosis of any kitten or cat with a confirmed diagnosis of FIP is poor.
- Cats with effusive FIP are not likely to live beyond a few weeks.
- Noneffusive FIP, however, is a more chronic clinical disease; with supportive treatment, affected cats may live for several months or years.
- Cats with neurologic disease caused by FIPV have a grave prognosis and are only expected to survive for days to weeks despite treatment.

COMMENTS

Client Education

- Beyond the prognosis, owners of cats with FIP who have additional, healthy cats in the household should be advised of the following.
  - Most cats that have been exposed to FCoV are likely to seroconvert within 18–21 days; however, a "positive" FCoV antibody titer does not define infection.
  - FCoV infection is common among group-housed cats but is not synonymous with FIP nor is it predictive of impending FIP.
  - Although FCoV is directly transmitted to cats, FIPV, the cause of FIP, is not generally considered to be a contagious virus.

Prevention/Avoidance

- Prevention of FIP centers on limiting exposure to FCoV, particularly among kittens.
- In breeding catteries, early weaning (by 5 weeks of age) and physical separation of kittens from adults (and their litter trays) have been shown to be effective measures in

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limiting FCoV infection, and subsequent development of FIP, in kittens housed in multicat households.

- Immunization of susceptible cats and kittens against FIP has proved to be difficult. To date, only one vaccine has been licensed as an aid in the prevention of FIP. The vaccine is a modified-live virus approved for intranasal administration only. Although the vaccine is generally regarded as safe, efficacy has not been definitively demonstrated in field studies.
    - Current feline vaccine guidelines stipulate that the FIP vaccine is "not generally recommended" on the basis that the vaccine has not been shown to consistently induce protective immunity.

Abbreviations

CMI: cell-mediated immunity
FCoV: feline coronavirus
FeLV: feline leukemia virus
FIP: feline infectious peritonitis
FIPV: feline infectious peritonitis virus
FIV: feline immunodeficiency virus
IFN: interferon
mRNA: messenger ribonucleic acid
PO: per os
rtPCR: polymerase chain reaction for viral RNA

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Acknowledgment to the author of the original chapter in *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Emergency and Critical Care, First Edition:* Richard B. Ford.

chapter 32

Feline Leukemia Virus Infection



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DEFINITION/OVERVIEW

- FeLV is a retrovirus (gamma-retrovirus, oncornavirus) that causes immunosuppression and lymphocytic neoplasia in domestic cats.

ETIOLOGY/PATHOPHYSIOLOGY

- Horizontal transmission (oronasal or bite wounds): viral replication is in local lymphoid tissues, infected cells carry virus to target lymphoid tissues (i.e., thymus, spleen, and lymph nodes), at which time it is common to see fever, diarrhea, leukopenia, lethargy, and marked lymphadenopathy. Virus then infects salivary glands and mucosal glandular epithelium, which secrete virus for horizontal transmission, and bone marrow, which produces infected WBCs and platelets.
- Acute infection takes one of three courses: (1) strong immune response that eliminates virus; (2) virus sequestered and causes latency; (3) persistent viremia. If latent for >1 year, reactivation is unlikely; viremia >16 weeks or bone marrow infected (detected by IFA) likely viremic, persistently infected, and infectious for life.
- Tumor induction occurs when DNA provirus integrates into cat chromosomal DNA in specific oncogene regions.

Systems Affected

- Hemic/lymphatic/immune: anemia (regenerative or nonregenerative); blood cell dyscrasias; bone marrow and lymphoid neoplasias; immunosuppression with secondary infections or neoplasia; immune-mediated diseases (IMHA, glomerulonephritis, uveitis with immune complex deposition, polyarthritis).
- Nervous: degenerative myelopathy, neuropathies (either direct neurotoxicity or secondary).
- Gastrointestinal: three clinical syndromes: FAE (i.e., vomiting/diarrhea, stomatitis, anorexia, weight loss); FAIDS (i.e., enterocolitis, crypt necrosis, villous atrophy, intractable diarrhea, and weight loss); FPLS (i.e., severe leukopenia, enteritis, destruction of crypt epithelium that mimics FPV with concurrent anemia, may actually be a FPV-FeLV coinfection).

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- Reproductive: fetal resorption, abortion, neonatal death; fading kitten syndrome (i.e., viremic kittens fail to nurse, severe dehydration, hypothermia, thymic atrophy, death within the first 2 weeks of life).
- All other body systems: immunosuppression with resulting secondary infections or neoplasia.

Incidence/Prevalence

- One to 8% worldwide; prevalence in the United States has been decreasing since the 1980s due to testing and removal programs.

Geographic Distribution

- Worldwide.



SIGNALMENT/HISTORY

Species

- Cats.

Breed Predilection

- Mixed breed-to-pure breed ratio: 1.3:1.

Mean Age and Range

- Prevalence highest between 1 and 6 years of age.

Predominant Sex

- Male-to-female ratio: 1.3:1 (outdoor-to-indoor ratio: 2.5:1).

General Comments

- Onset of FeLV-associated diseases may be delayed months to years after infection; clinical signs of virus are variable and nonspecific.
- Associated diseases: neoplastic or nonneoplastic; nonneoplastic or degenerative usually secondary to immunosuppression.
- FeLV-induced immunodeficiency is clinically indistinguishable from that induced by FIV.

Historical Findings

- Outdoor cat.
- Multicat household.

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Physical Examination Findings

- Depends greatly on presence of neoplastic or nonneoplastic disease and secondary infections.
- Lymphadenopathy: mild to severe.
- Upper respiratory tract: rhinitis, conjunctivitis, keratitis, or uveitis.
- Gastrointestinal: persistent diarrhea (either primary or due to secondary infections).
- Oral: gingivitis, stomatitis, or periodontitis.
- Integument: chronic recurrent skin or ear infections.
- Neurologic: peripheral or central neuropathies.
- Neoplasia: lymphoma, especially thymic and multicentric, occurs in up to 25% of cats which are positive for FeLV usually within 2 years of diagnosis; leukemias; fibrosarcomas due to coinfection with recombinant FeSV.

Risk Factors/Causes

- Feral or free-roaming cats.
- Male.
- Sexually intact.
- Horizontal transmission via saliva or blood, usually by biting, grooming, sharing water bowls; fecal shedding possible but lower risk of transmission.
- Vertical transmission via transplacental, transmammary, or grooming of neonates.



DIFFERENTIAL DIAGNOSIS

- FIV
- Primary infections: viral, bacterial, fungal, or parasitic
- Neoplasia of nonviral origin



DIAGNOSTICS

Complete Blood Count/Chemistry/Urinalysis

- Anemia: regenerative or nonregenerative, often severe; occasionally see IMHA.
- Lymphopenia.
- Neutropenia: possibly in response to secondary infections.
- Thrombocytopenia.
- Urinalysis and serum chemistry profile: findings depend on systems affected and type of disease.

Other Laboratory Tests

- General comments: no 100% accurate test.
- ELISA/ICGA: detect free soluble FeLV-p27 antigen in plasma, serum, or whole blood; more sensitive than IFA at detecting early or transient antigenemia; a single positive test

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cannot predict persistent infections, retest in 12 weeks or test with IFA; false-positives more common with whole blood, not recommended to test saliva or tears.

- IFA: identify FeLV-p27 antigen in neutrophils and platelets of fixed smears of whole blood or buffy coat preparations (for leukopenic cats); positive result indicates active infection in bone marrow that can usually be detected by 3 weeks post infection but may take up to 12 weeks to test positive; a positive result only indicates viral infection and not current illness, so it should be confirmed by another method and has no bearing on prognosis.
- Vaccinates are not detected by either ELISA/ICGA or IFA.
- Discordant results.
  - Two ELISA/ICGA tests differ: perform IFA.
  - Positive ELISA/ICGA and IFA: suggests persistent viremia and true positive.
  - Positive ELISA/ICGA and negative IFA: repeat assay in 6–8 weeks.
- PCR: detects viral RNA or DNA in blood, solid tissue, cell culture, or fixed samples; requires special equipment and expertise; can help determine status of cats with discordant ELISA/ICGA and IFA results or if suspicious of latent infections due to chronic conditions.

Imaging

- Thymic atrophy in fading kittens.

Diagnostic Procedures

- Bone marrow aspiration or biopsy: indicated if nonregenerative anemia is present; rule out aplastic anemia versus anemia secondary to myeloproliferative disease.

Pathologic Findings

- Lesions depend on concurrent disease state and presence of neoplasia.



THERAPEUTICS

- Outpatient unless severe secondary infections or other conditions require hospitalization and further treatment.
- Supportive care as needed.

Drugs of Choice

- Zidovudine (Retrovir, AZT) 5 mg/kg PO, SQ every 12 hours; antiviral, possible clinical improvement but poorer response *in vivo* than in cats infected with FIV; monitor for nonregenerative anemia, reduce dose compared to FIV treatment, do not treat cats with myelosuppression; other human antivirals are being studied but those with efficacy *in vivo* often have severe toxicity or are ineffective.

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- Interferon: immunomodulatory and direct antiviral effect; human IFN-alpha (Roferon) can be given for 6–7 weeks in high doses (10^5 to 10^6 IU/kg), after which neutralizing antibodies are generated. Oral administration (low dose: 15–30 U orally every 24 hours for 7 days on alternate weeks) may stimulate lymphoid tissue in the oral cavity with resulting systemic immunomodulation, but interferons are destroyed in the gastrointestinal tract so no measurable serum levels develop.
- Immunomodulatory therapy: nonspecific agents (i.e., *Acemannan*, *Staphylococcus* protein A, *Propionibacterium acnes*, and paraimmunity inducers) have showed equivocal results in clinical trials; nonspecific immune stimulation may increase viral replication and promote progression of disease, therefore it may not be recommended.
- Anemia: blood transfusions may be necessary in severe anemia; suspect *Mycoplasma hemofelis* (formerly *Haemobartonella*) in all cats with regenerative hemolytic anemia and treat appropriately; EPO may be helpful with nonregenerative anemias.
- Topical bovine lactoferrin (40 mg/kg every 24 hours as needed) may be helpful for treatment of stomatitis.
- Lymphoma: standard chemotherapy protocols.
- Myeloproliferative disease and leukemias: more refractory to treatment.

Contraindications

- MLV vaccines may theoretically cause illness.

Precautions/Interactions

- Systemic glucocorticoids or other immunosuppressive drugs should be avoided unless a very compelling indication exists.
- Inactivated boosters to only core vaccines (RV and FRVCP) should be considered for routine vaccination; cats who are positive for FeLV may not be able to mount adequate immune response to rabies vaccination so more frequent boosters than usually recommended (e.g., every 6 months) must be considered, especially in cats allowed to go outside in areas with high rabies prevalence.
- Perioperative antibiotics are recommended for all surgeries and dental procedures.
- Virus lives for only minutes outside the host and is susceptible to all disinfectants (including common soap) so routine cleaning procedures will prevent transmission in the hospital. Viremic cats should be kept away from sick cats to protect them from infectious diseases, and under no circumstances should they be placed in an "isolation ward" with cats suffering from infections such as viral respiratory disease.

Diet

- Normal unless secondary conditions necessitate alternative foods.

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Surgical Considerations

- No specific surgical treatments; surgical removal of tumors if present, treat oral lesions appropriately.
- Intact cats who are positive for FeLV should be spayed or neutered to reduce stress associated with estrus/mating and decrease roaming and aggressive behaviors.

COMMENTS

Client Education

- Emphasize the importance of keeping cats who are positive for FeLV indoors to prevent spread of the virus and protect the positive cat from exposure to secondary infections.
- Warn owners that adult cats who are negative for FeLV have a 10–15% risk of infection if they have lived with a positive cat for several months. The negative cats should receive FeLV vaccination if the owner refuses to separate the housemates.
- Warn owners that cats who have FeLV may have a slower response to treatment of secondary conditions.

Patient Monitoring

- Biannual exams with CBC, oral examination, lymph node measurements, and body weight are recommended to evaluate for secondary diseases and myelosuppression; minimum annual chemistry profile and urinalysis should be performed, more frequent monitoring may be warranted if concurrent diseases exist.

Prevention/Avoidance

- Prevent interaction with infected cats.
- Quarantine and test new cats before introducing into multicat households/catteries.
- Vaccine: test cats before initial vaccination and do not vaccinate cats who are positive for FeLV; warn owners that cat may be infected if testing is not performed; efficacy controversial and none is 100% effective; risk of vaccine-associated sarcomas with FeLV (and RV) vaccination is 0.1–0.01% overall, likely due to adjuvant; vaccinate low on left hind leg per AAFP recommendations.

Possible Complications

- Cats that are negative for FeLV that live with an infected cat have a 40-fold increased risk of lymphoma compared with cats who live in households that are negative for FeLV.

Expected Course and Prognosis

- Persistently viremic cats: >50% succumb to related diseases within 2–3 years after infection in multicat households; better survival rate in single-cat households.

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Associated Conditions

- Secondary infections
- Neoplasia: especially lymphoid, fibrosarcoma, other
- Immune-mediated disease: IMHA

Age-Related Factors

- Neonatal kittens are most susceptible to persistent infection (70–100%); risk decreases with age: <30% susceptible by 16 weeks of age.

Zoonotic Potential

- None known but infection of human bone marrow possible in cell culture.
- Potential transmission of secondary infections (e.g., *T. gondii*) to people who are immunocompromised.

Pregnancy

- Transplacental transmission causes abortions, stillbirth, and fetal resorption; up to 20% of kittens may survive to become persistently infected adults; infected queen can also infect kittens by transmammary transmission, biting umbilical cord, or grooming kittens.
- Kittens born to a queen who is FeLV positive may test negative but usually seroconvert within weeks to a few months and should be retested.

Abbreviations

AAFP: American Association of Feline Practitioners
AIDS: acquired immunodeficiency syndrome
CBC: complete blood count
ELISA: enzyme-linked immunosorbent assay
EPO: erythropoietin
FAE: FeLV-associated enteritis
FAIDS: feline acquired immunodeficiency syndrome
FeSV: feline sarcoma virus
FIV: feline immunodeficiency virus
FPLS: feline panleukopenia-like syndrome
FPV: feline panleukopenia virus
FVRCP: feline viral rhinotracheitis, calicivirus, panleukopenia
ICGA: immunochromatographic assay
IFA: immunofluorescent antibody
IFN: interferon
IMHA: immune-mediated hemolytic anemia
MLV: modified-live virus
PCR: polymerase chain reaction

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PO: per os
RV: rabies vaccine
SQ: subcutaneous
WBC: white blood cell

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chapter 33

Feline Lower Urinary Tract Disease



DEFINITION/OVERVIEW

- In approximately 65% of nonobstructed male and female cats with naturally occurring lower urinary tract disease, the exact cause(s) of hematuria, dysuria, pollakiuria, stranguria, and periuria are still unknown. After appropriate diagnostic evaluations, these cats are classified as having feline idiopathic LUTD (iLUTD) or feline idiopathic cystitis (FIC).
- Feline iLUTD is an exclusionary diagnosis established only after known causes have been eliminated.
- Since 1970 this condition has undergone a number of terminology and acronym changes as our understanding of the etiopathogenesis has changed. These naming modifications are a great example of the progress that has been made in understanding and defining this condition; however, more work needs to be done.
- Initially, the terms feline urological syndrome and FUS were used to describe disorders of cats characterized by hematuria, dysuria, pollakiuria, stranguria, and partial or complete obstruction. Because these signs may be associated with any cause of lower urinary tract disease, these terms have been recommended to be abandoned and replaced with more descriptive terminology pertaining to site of injury, cause(s), morphologic changes, or pathophysiologic mechanisms.
- Some cats with iLUTD exhibit findings similar to those observed in humans with interstitial cystitis, an idiopathic inflammatory disorder of the urinary bladder. These similarities prompted the hypothesis that some cases of feline iLUTD are analogous to human interstitial cystitis and lead to the proposed terminology of feline interstitial cystitis; however, further studies challenged this hypothesis. Studies demonstrated that these cats have significant alterations in the sympathetic nervous system. An increased acoustic startle response was also detected and these findings may explain why these cats are aggravated by environmental stressors and clinical signs wax and wane.
- Often cats with iLUTD have comorbid disorders such as gastrointestinal, endocrine, dermatologic, and/or behavioral problems. A similar pattern has been detected in humans with interstitial cystitis and some authors have questioned if these cats have a common disorder affecting all organs, which then respond in a stereotypical manner. The provisional term "Pandora syndrome" has been proposed to describe cats with recurrent iLUTD

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and comorbid disorders. The criteria for this syndrome also include an early adverse experience, waxing and waning severity of clinical signs associated with stress, and resolution with environmental modification.



ETIOLOGY/PATHOPHYSIOLOGY

- Exact etiology(ies) are unknown, likely multifactorial.
- Stress may play a role in precipitating or exacerbating signs; neuroendocrine abnormalities identified in cats with chronic forms of iLUTD are indicative of increased activity of the sympathetic nervous system and diminished adrenocortical responsiveness.
- Quantitative or qualitative defects in surface GAGs, as well as changes in the nonneuronal cholinergic system, and subsequent increased urothelial permeability have been hypothesized to be causative factors.
- Initial experimental and clinical studies implicated feline calicivirus as a potential etiological agent; however, a larger, follow-up study did not confirm these findings.

Systems Affected

Renal/Urologic

- Persistent urethral outflow obstruction results in postrenal azotemia.

Pandora Syndrome

- Gastrointestinal, dermatologic, pulmonary, cardiovascular, central nervous, endocrine, and immune systems.



SIGNALMENT/HISTORY

- Both males and females are affected.
- No breed predilection.
- May occur at any age, but is most commonly recognized in young to middle-aged adults (mean age 4.4 years).
- Uncommon in cats <1 year old and >10 years old.
- Indoor or outdoor cats have equal chance of developing iLUTD.
- No known genetic basis.
- Common clinical signs include pollakiuria, hematuria, stranguria, periuria (urination in inappropriate locations), dysuria, and urethral outflow obstruction.
- In cats with Pandora syndrome, common clinical signs include inappetence, avoidance behavior, and perichezia.

Risk Factors/Causes

- Stress: may play a role in precipitating or exacerbating signs; an unlikely primary cause.
- Age: younger age is associated with an increased risk of recurrence of signs.

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- Sex: males and female affected equally; however, neutered males and spayed females are reported in some studies to be at increased risk compared to intact animals.

Historical Findings

- Pollakiuria
- Hematuria
- Dysuria
- Stranguria
- Periuria (urinating in inappropriate locations)
- Urinary outflow obstruction
- With Pandora syndrome:
  - Perichezia (defecating in inappropriate locations)
  - Inappetence



CLINICAL FEATURES

- Periuria, pollakiuria, stranguria, and macroscopic hematuria are the most common clinical signs observed in cats with nonobstructive idiopathic cystitis.
- Clinical signs subside within 5–7 days without therapy in up to 92% of cats with acute nonobstructive idiopathic cystitis.
- Signs may recur after variable periods of time and again subside without treatment. Approximately 40–50% of cats with acute idiopathic cystitis will experience one or more recurrences of signs within 1–2 years.
- Recurrent episodes of acute idiopathic cystitis tend to decrease in frequency and severity as the cats become older.
- Recurrent clinical signs in patients with idiopathic cystitis are often assumed to be recurrence of the original disease; however, recurrent signs may also be the result of a delayed manifestation of the original disease (e.g., spontaneous or iatrogenic urethral stricture), or onset of a different lower urinary tract disease associated with similar clinical signs (e.g., urolithiasis).
- A small subset of cats with LUTD have persistent clinical signs that last for weeks to months or are frequently recurrent. These cats are classified as having idiopathic LUTD. Less than 15% of cats evaluated because of acute idiopathic cystitis will develop chronic forms of the disease.
- Physical examination may reveal a thickened, firm, contracted, painful, bladder wall; may detect urethral plugs or uroliths by examination of the distal penis and penile urethra.



DIFFERENTIAL DIAGNOSIS

- Metabolic disorders including various types of uroliths and urethral plugs.
- Infectious agents including bacteria, fungal agents, and parasites.

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- Trauma.
- Neurogenic disorders including reflex dyssynergia, urethral spasm, and hypotonic or atonic bladder (primary or secondary).
- Iatrogenic diseases including those due to urethral catheters, indwelling urethral catheters (especially open systems), postsurgical catheters, reverse-flushing solutions, and urethrostomy complications.
- Anatomic abnormalities including urachal anomalies and acquired urethral strictures.
- Neoplasia (benign and malignant).
- Behavioral disorders resulting in periuria.
- Clinical signs may be confused with constipation.



DIAGNOSTICS

- No single test is diagnostic; feline iLUTD is an exclusionary diagnosis established only after known causes have been eliminated.
- A complete medical history is a profoundly important initial diagnostic step and can help guide which diagnostic tests are needed.
- Hematuria and proteinuria are common urinalysis findings. Initial episodes of iLUTD usually occur in the absence of pyuria and significant numbers of detectable bacteria.
- Quantitative urine culture verifies absence of bacteriuria and excludes urinary tract infection; light microscopic examination of urine sediment is an unreliable method of detecting bacteriuria; collect urine specimens by cystocentesis to avoid contamination with organisms that normally inhabit the distal urethra.
- Survey radiography may exclude uroliths or urethral plugs.
- Positive-contrast retrograde urethrocystography or antegrade cystography may exclude urethral strictures, vesicourachal diverticula, and neoplasia.
- Double-contrast cystography may exclude small or radiolucent uroliths, blood clots, and thickening of the bladder wall due to inflammation or neoplasia.
- Ultrasonography may exclude uroliths in the urinary bladder and permits screening for urinary bladder masses and thickening of the bladder wall.
- Urethrocystoscopy may reveal petechial submucosal hemorrhages (so-called glomerulations) on the surface of the urinary bladder; may exclude urethral strictures, urocystoliths, blood clots, vesicourachal diverticula, and masses.
- Biopsies obtained with urinary catheters, cystoscopes, or via surgery may permit morphologic characterization of the inflammatory or neoplastic lesions; not routinely needed.

Pathologic Findings

- Mucosal ulceration and submucosal edema, hemorrhage, fibrosis, neovascularization, mastocytosis, and mononuclear inflammatory cell infiltration are common light microscopic lesions observed in the urinary bladders of cats with chronic iLUTD.
- Inflammatory cells may not be prominent in some forms of the disease, unless secondary bacterial infections have resulted from catheterization or perineal urethrostomy.

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THERAPEUTICS

- Patients with nonobstructive lower urinary tract disease are typically managed as outpatients; diagnostic evaluation may require brief hospitalization.
- Patients with obstructive lower urinary tract disease are usually hospitalized for diagnosis and management.
- Short-term use of pharmacologic agents is considered for any patient with severe signs.
- Long-term dietary and environment management strategies are used to minimize the risk of recurrences.
- Long-term pharmacologic inventions are usually reserved for cats with more severe chronic forms of the disease in which dietary and environmental management strategies have failed to adequately control clinical signs or frequency of recurrences.
- Over 70 agents or procedures have been recommended for management of nonobstructive iLUTD in cats; fewer than 10% of these proposed treatments have been evaluated in controlled clinical trials. Debate surrounding the efficacy of various treatments is complicated by the self-limiting nature of clinical signs associated with the majority of cases of idiopathic cystitis. In this setting, any form of therapy might appear to be beneficial as long as it is not harmful.

General

- Environmental enrichment and stress reduction: strategies aimed at decreasing central noradrenergic drive (e.g., stress) may be important in correcting the imbalance in the neuroendocrine system (excitatory sympathetic nervous system outflow inadequately controlled by cortisol). The goal is to increase behavioral choices, provide opportunity for appropriate behaviors, and ensure optimal litter box care (information available at http://indoorpet.osu.edu). A prospective, nonrandomized, observational study demonstrated significant reductions in lower urinary tract signs, fearfulness, and nervousness.
- Synthetic feline facial pheromone therapy (Feliway®) was associated with significant increases in grooming and food intake in a double-blind placebo-controlled clinical study, and a nonsignificant trend, in a second small study, for cats to show fewer days with clinical signs of cystitis and a reduced number of episodes.
- Antimicrobials (chloramphenicol, amoxicillin): no detectable effect on remission of clinical signs in cats demonstrated in controlled clinical trial.
- Fluid therapy (lactated Ringer's solution 100 mL, SQ, once): no detectable effect on remission of clinical signs in cats demonstrated in controlled clinical trial.

Diet

- Results of a nonrandomized, open, prospective study suggest that feeding moist (>60% moisture) rather than dry foods may minimize recurrence of signs. This approach results in dilute urine, which may decrease the concentration of irritating substances in the urine.

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- Other methods to increase water intake may be beneficial, including feeding 2–3 meals per day instead of a single meal, high-salt diets (do not use in cats with chronic kidney disease) or water fountains or special bowls. A goal urine specific gravity is 1.040 or lower. The efficacy of these treatments has not been evaluated by controlled clinical trials.
- Management recommended for persistent crystalluria in patients at risk for formation of crystalline-matrix urethral plugs.

Drugs of Choice

Antiinflammatory Agents and Analgesics

- Opioid analgesics: short-term opioid analgesic therapy for 24–72 hours may be of benefit in alleviating acute "flare-ups" of severe pollakiuria, dysuria, stranguria, and periuria in affected cats; butorphanol 0.2–0.4 mg/kg every 8 hours, PO for 1–3 days; buprenorphine 0.01–0.02 mg/kg every 6–12 hours, IM or SQ buccally for 1–3 days. There have been no reports of controlled studies evaluating the efficacy and safety of short-term opioid analgesics for management of cats with idiopathic cystitis.
- Corticosteroids (prednisolone 1 mg/kg every 12 hours, PO for 10 days): no detectable effect on remission of clinical signs in cats demonstrated in controlled clinical trial; predispose to bacterial urinary tract infections, especially in cats with indwelling transurethral catheters.
- NSAIDs: safety and efficacy of NSAIDs in the treatment of cats with iLUTD have not been evaluated by controlled clinical trials. Meloxicam (0.1 mg/kg every 24 hours, PO for 3–4 days) has been suggested to manage patient discomfort during acute episodes. Pending further safety and efficacy studies, exercise appropriate caution when considering use of NSAIDs to treat feline idiopathic cystitis.

Amitriptyline and Other Antidepressants

- Amitriptyline: a tricyclic antidepressant and anxiolytic drug reserved for treatment of cats with severe chronic iLUTD who have not responded to other forms of pharmacologic, dietary, and environmental management; suggested empirical dosage is 5–10 mg/cat every 24 hours PO given at night; may predispose to urine retention, bacterial urinary tract infection, and urolithiasis. Randomized controlled, short-term, clinical trial for 7 days revealed no significant difference in recovery and in amitriptyline-treated cats, clinical signs recurred faster and more frequently than in controlled cats. Amitriptyline does not seem to be beneficial for treatment of acute self-limiting episodes of iLUTD.
- Efficacy and safety of other tricyclic antidepressants, selective serotonin reuptake inhibitors, and anxiolytics have not been evaluated by controlled clinical trials.

Glycosaminoglycans (GAGs)

- Pentosan polysulfate sodium: a semi-synthetic low molecular weight heparin analogue that reinforces urothelial GAGs and reduces transitional cell injury. Oral pentosan polysulfate 50 mg every 12 hours PO may benefit some cats with chronic iLUTD. Results of

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controlled clinical studies evaluating the safety and efficacy of pentosan polysulfate have not been reported.

- Glucosamine/chondroitin sulfate: glucosamine, an amino sugar naturally produced in the body, is an important intermediate for the formation of GAGs. Chondroitin sulfate is one of the most abundant GAGs in the bladder surface GAG layer. No significant difference but a trend toward slight improvement, between the severity of clinical signs in cats treated with glucosamine (Cystease® 125 mg once daily PO for 6 months) and those treated with a placebo. The efficacy of oral glucosamine combined with chondroitin sulfate (Cosequin® 125 mg/100 mg per 4.5 kg cat once daily PO) has not been evaluated by controlled clinical trials.

Anticholinergics

- Propantheline (7.5 mg PO, once): a potent quaternary ammonium anticholinergic agent that minimizes the force and frequency of uncontrolled detrusor contractions; no difference in rate of recovery was observed between cats treated with propantheline and control groups.
- Oxybutynin chloride: a moderately potent anticholinergic agent with additional independent musculotropic relaxant and local anesthetic effects; efficacy of oxybutynin has not been evaluated by controlled clinical trials.
- Tolterodine: a moderately potent anticholinergic agent that may have a more pronounced *in vivo* effect on urinary bladder than oxybutynin; efficacy of tolterodine has not been evaluated by controlled clinical trials.

Precautions/Interactions

- Cats with urethral obstruction and postrenal azotemia are at increased risk for adverse drug events, especially with drugs and anesthetics that depend on renal elimination or metabolism.
- Indwelling urethral catheters, especially when associated with fluid-induced diuresis or coadministration of corticosteroids, predispose patients to bacterial urinary tract infections.
- Tricyclic antidepressants (e.g., amitriptyline): avoid short-term administration or abrupt discontinuation.
- Coadministration of oxybutynin or tolterodine with other anticholinergic agents (e.g., amitriptyline) may intensify adverse effects and should be avoided.
- Phenazopyridine: a urinary tract analgesic; may result in methemoglobinemia and Heinz body anemia.
- Methylene blue: a weak antiseptic agent; may result in Heinz body anemia.

Surgical Considerations

- Cystotomy to obtain biopsy specimens in patients with clinical finding consistent with iLUTD is not usually indicated.

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- Cystotomy to lavage and debride the bladder mucosa as a form of treatment is not recommended.
- When performing perineal urethrostomies to minimize recurrent urethral obstruction, it is important to localize the obstructive disease to the penile urethra by contrast radiography.



COMMENTS

Client Education

- Hematuria, dysuria, and pollakiuria: often self-limiting; subside within 5–7 days, but signs may recur unpredictably in approximately one-half of affected cats.
- Lack of controlled studies that demonstrate efficacy of most drugs used to treat symptomatically.
- Recurrent episodes of acute iLUTD tend to decrease in frequency and severity as the cats become older.

Patient Monitoring

- Males should be monitored for signs of urethral obstruction.

Prevention/Avoidance

- Feed moist diets.
- Reduce environmental stress by minimizing changes in the home, maintaining a constant diet, providing opportunities for play and safe places to hide, refining cat–owner interactions, managing conflict between cats and ensuring clean litter boxes.

Possible Complications

- Urethral obstruction due to formation of urethral plugs: most common in male cats with concurrent crystalluria.
- Vesicourachal diverticula: acquired vesicourachal diverticula often are self-limiting with resolution of clinical signs.
- Indwelling transurethral catheters often cause trauma; predispose to ascending bacterial urinary tract infections.
- Perineal urethrostomies may predispose to ascending bacterial urinary tract infections and urethral strictures.

Expected Course and Prognosis

- Hematuria, pollakiuria, and dysuria often are self-limiting in patients with most iLUTDs, subsiding within 5–7 days.
- Signs will recur unpredictably in approximately half of affected cats; the frequency of recurrence declines with advancing age.

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Synonyms

- Feline idiopathic cystitis
- Feline sterile idiopathic cystitis
- Feline interstitial cystitis
- FUS

Abbreviations

FUS: feline urological syndrome
GAG: glycosaminoglycan
iLUTD: idiopathic lower urinary tract disease
IM: intramuscular
NSAID: nonsteroidal antiinflammatory drugs
PO: per os
SQ: subcutaneous

Suggested Reading

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Acknowledgment to the author of the original chapter in *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Emergency and Critical Care, First Edition:* John M. Kruger, Carl A. Osborne, and Jody P. Lulich.

chapter 34

Feline Panleukopenia

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DEFINITION/OVERVIEW

- FPL is the common term used to describe a systemic, immunosuppressive disease of cats caused by highly contagious FPV.

ETIOLOGY/PATHOPHYSIOLOGY

- FPL virus (also called FPV) is a single-stranded DNA virus.
- Because FPVs are contagious and resilient to destruction, the virus is found worldwide and continues to pose a significant threat to all members of the family Felidae.
- The high degree of seroprevalence in unvaccinated, healthy cats (75%) suggests that exposure rates are high, but most clinical infections are subclinical.
- Transmission of virus can occur following direct cat-to-cat contact as well as following indirect, oral contact with contaminated fomites (e.g., litter trays, hands, feeding dishes, cages, bedding).
- Infected cats usually shed virus for 1–2 days, but recovering cats may shed virus in feces and urine for as long as 6 weeks.
- The ability of the virus to persist in the environment for sustained periods, rather than shedding, explains how the virus is maintained in the population.
- Virus undergoes initial replication in oropharyngeal lymphoid tissue within the first week post infection then disseminates via plasma to virtually all body tissues, particularly those with high mitotic activity (e.g., the gastrointestinal tract). Damage to intestinal crypt epithelium culminates in malabsorptive disorders and diarrhea.
- Virus replication in the CNS can occur subsequent to *in utero* infection of healthy queens.
- FPV can infect fetal CNS tissues subsequent to vaccination of pregnant queens with MLV vaccine.
- Neonates vaccinated with MLV FPV vaccine prior to 4 weeks of age can experience vaccine virus dissemination and replication in the CNS. Viral DNA can be found in optic nerves, retina, and cerebellum.

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- Cerebellar hypoplasia in kittens can be caused by FPV infection and MLV virus.
- Prenatal and neonatal infections have been associated with feline myocarditis and cardiomyopathy.



SIGNALMENT/HISTORY

- Most infections are subclinical, but severe clinical illness caused by FPV is most common among young kittens.
- Mortality is greatest between 3 and 5 months of age.
- FPV is also regarded as a cause of "fading kitten" syndrome (death occurring after birth but before weaning). The course of infection is acute, with most deaths occurring as early as 12 hours to 5 days post infection.
- Cats that survive acute illness for more than 5 days have a better prognosis for survival with supportive care.
- Clinical signs of inappetence and lethargy, followed by vomiting within 2–3 days.
- No gender or breed predisposition for FPV infection.
- Outbreaks of FPV have been reported in adult cats, particularly in high-density housing environments where seroprevalence can be particularly low (e.g., shelters, rescue groups).



CLINICAL FEATURES

- High fever (104–107 °F).
- Dehydration.
- Evidence of vomiting may be present on the hair coat around the face.
- Diarrhea is uncommon and, when seen, usually occurs in the later stages of infection.
- Affected cats may resist abdominal palpation; palpation of thickened (rope-like consistency) intestines and enlarged mesenteric lymph nodes is characteristic.
- If treatment is delayed, cats may become profoundly lethargic or comatose, dehydrated, and hypothermic.
- Death attributed to secondary bacterial infection, DIC, or severe dehydration is common among kittens with acute, untreated FPV infection.
- Kittens infected *in utero*, naturally infected before 9 days of age, or vaccinated early in life with MLV FPV vaccine (<4 weeks of age) may manifest neurologic signs: ataxia, incoordination, and intention tremors typically associated with cerebellar hypoplasia. Affected kittens walk with a wide stance and a characteristic hypermetric gait. Neurologic manifestations are typically absent when the cat is at rest. Blindness can occur.



DIFFERENTIAL DIAGNOSIS

- "Fading kitten" syndrome
- Acute gastrointestinal toxicity (e.g., salmonellosis)

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- Intussusception
- Septicemia
- Feline leukemia virus infection
- Feline immunodeficiency virus infection
- Linear foreign body



DIAGNOSTICS

Complete Blood Count

- Severely affected kittens may have total white blood cell counts between 50 and 3000 cells/μL.
- Less severely affected cats may have counts between 3000 and 7000 cells/μL.
- Cats that recover from acute FPV infection will manifest rebound leukocytosis with white blood cell counts exceeding 30 000 cells/μL.
- Anemia and thrombocytopenia are less common; variable laboratory findings.

Biochemistry

- No specific biochemical abnormalities occur in cats with FPL.

Serology

- Serological testing (virus neutralization is recommended) and virus isolation for the diagnosis of FPL are available from selected laboratories but these methods offer little value in the clinical setting due to the short clinical course.
- Serological testing is generally reserved for assessment of a protective immune response in vaccinated or exposed cats.

Enzyme-Linked Immunosorbent Assay

- ELISA-based canine parvovirus test for the presence of fecal antigen can be used in cats but important limitations apply.
- The canine fecal antigen tests have not been standardized for use in cats with confirmed FPV infection.
- Furthermore, fecal shedding of FPV in cats may cease prior to the onset of clinical signs.

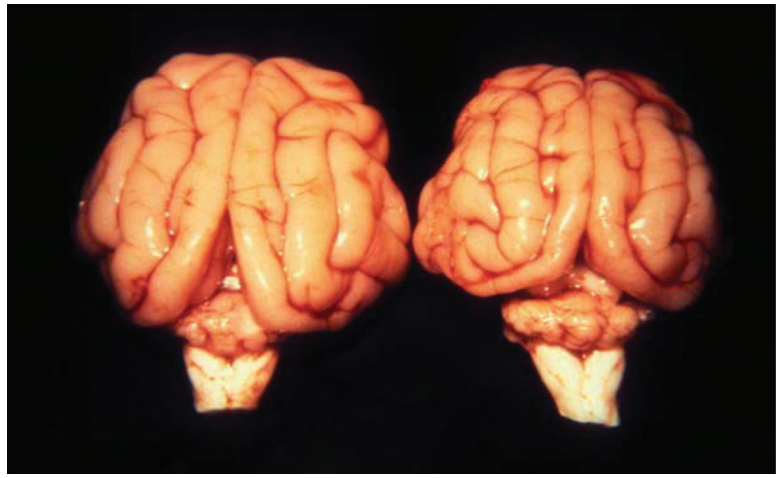
Histopathology

- Histopathology remains the confirmatory diagnostic test of choice for FPL.

Pathologic Findings

- Gross pathologic findings in cats that die of complications from FPV infection are minimal and usually limited to the small intestine (i.e., dilated bowel and serosal hemorrhages).

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■ **Fig. 34.1.** Cerebellar hypoplasia in the brain of a kitten that was euthanized (*left*) because of neurologic signs, compared to a normal cat brain (*right*).

- Significant histological findings center on changes in the crypt epithelium of the jejunum and ileum and lymphoid depletion.
- Cerebellar degeneration and hypoplasia (Fig. 34.1) may be seen in kittens that experienced neonatal or *in utero* infection from FPV or MLV FPV vaccine.



THERAPEUTICS

- Parenteral fluid and electrolyte replacement therapy.
- Insensible fluid loss should be replaced at the rate of 44 mL/kg per day using a balanced isotonic crystalloid fluid such as lactated Ringer's solution, Normosol-R, or Plasmalyte-A with supplemental potassium as indicated.

Nutritional Support

- Oral administration of food or liquid should be avoided during the acute clinical infection.

Antiemetic Therapy

- Maropitant (1 mg/kg IV q24h) or dolasetron (0.6 mg/kg IV q24h) can be administered to control vomiting.
- Gastroprotectant therapy with pantoprazole (1 mg/kg IV q24h) or famotidine (0.5–1 mg/kg IV q12h) may be considered if vomiting is severe.

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Transfusion

- Plasma or blood transfusions are limited to patients that become anemic or hypoalbuminemic (<2.0 g/dL) or hypoproteinemic (<5.0 g/dL).

General

- Antibiotic therapy (ampicillin 22 mg/kg IV q6h, Unasyn 30 mg/kg IV q8h, with enrofloxacin 5 mg/kg IV q24h). Aminoglycoside antibiotics should be avoided unless the patient is well hydrated and maintains normal blood pressure and renal perfusion.
- B-vitamin combination.
- Parenteral therapy should be continued at least until vomiting ceases and the cat resumes eating.
- Low-dose diazepam (2.5 mg total) may be administered to promote eating in cats that have stopped vomiting and appear to be making a clinical recovery (interest in food, leukocytosis).

Early Vaccination

- Initial vaccination of kittens beginning at 4 weeks of age, and every 2–4 weeks thereafter until 16 weeks of age, is only recommended in group-housed cats where a reasonable risk of FPV exposure exists (e.g., shelter-housed kittens).
- Current vaccination recommendations for kittens not in a shelter environment are to start at 6 weeks of age, revaccinate every 3–4 weeks through 16 weeks of age, then revaccinate at 1 year of age, then every 3 years thereafter. If a kitten is not vaccinated before 12 weeks of age, two vaccinations administered 3–4 weeks apart can be administered, then revaccination at 1 year.
- Early vaccination is most likely to be effective in colostrum-deprived kittens.

Modified-Live Vaccine versus Inactivated Vaccine

- The author recommends use of MLV (nonadjuvanted) FPV vaccines in all cats with the exception of those known to be positive for FeLV or FIV and kittens less than 6 weeks of age.
- If vaccination of a pregnant queen is indicated, use of an inactivated vaccine is recommended to reduce risk of vaccine virus replication in the CNS of unborn kittens (cerebellar hypoplasia).



COMMENTS

Client Education

- Before introduction of a new cat into a household where a cat/kitten has recently been diagnosed with, or died from, FPL, all cages, feeding bowls, floors, litter trays, and such should be thoroughly disinfected (fresh solution containing 1 ounce household bleach/gallon of water (1:30 dilution)).

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- Ideally, the facility should be isolated from cats for several days following disinfection before introducing a new cat.
- Prior to entry, any new cat/kitten should have received at least two doses of vaccine, with the last dose administered at least 2 weeks prior to introduction.

Controlling Outbreaks

- When administering inactivated (killed-adjuvanted) FPV vaccine to cats, a protective immune response is typically delayed until 2–3 weeks following the administration of the second dose. For this reason, inactivated (killed-adjuvanted) FPV vaccine has been implicated as a contributing factor in FPL outbreaks within multicat housing environments.
- In high-exposure risk environments, or in the face of an outbreak, kittens should be vaccinated with an MLV (nonadjuvanted) vaccine as early as 4 weeks of age and every 2 weeks thereafter as long as they reside in the facility.

Prevention/Avoidance

- In the United States both killed-virus (adjuvanted) and MLV vaccines (nonadjuvanted) are available for the prevention of disease caused by FPV.
- Vaccination provides excellent protective immunity and is recommended for administration to all kittens beginning as early as 6 weeks of age; a dose should be administered every 3–4 weeks until 15–16 weeks of age.
- Current vaccination recommendations include revaccination of all adult cats 1 year following the last dose in the initial series.
- Booster inoculations are recommended triennially thereafter.
- Vaccine approved for intranasal vaccination of cats against FPL (a trivalent vaccine for protection against herpesvirus and calicivirus as well as FPV) is available but is regarded as less effective than parenterally administered vaccines. All intranasal FPV vaccines are MLV products and should not be administered to kittens less than 4 weeks of age.

Abbreviations

CNS: central nervous system
DIC: disseminated intravascular coagulation
ELISA: enzyme-linked immunosorbent assay
FeLV: feline leukemia virus
FIV: feline immunodeficiency virus
FPL: feline panleukopenia
FPV: feline parvovirus
IV: intravenous
MLV: modified-live vaccine

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chapter 35

Gastric Dilation-Volvulus Syndrome



DEFINITION/OVERVIEW

Gastric dilation-volvulus syndrome (GDV) is a combination of intragastric accumulation of gas and fluid and a rotation of the stomach along its long axis. This results in a variety of life-threatening conditions including circulatory shock and systemic inflammatory response syndrome (SIRS). Untreated, it leads to death.



ETIOLOGY/PATHOPHYSIOLOGY

- The mechanism that initiates a GDV episode remains unknown. The gas produced is primarily carbon dioxide with some hydrogen, likely from bacterial fermentation. The source of bacteria would be ingested food or duodenal reflux. Severe gastric distension manifests as visible abdominal distension, restlessness, excessive salivation and swallowing, and nonproductive retching. Intraperitoneal pressure elevation, obstruction of the compliant caudal vena cava, decreased venous return and cardiac output, hypoxemia, microcirculatory injury, and maldistribution of blood flow result in signs of circulatory shock. Cytokine production, cellular dysfunction, and reperfusion injury following resuscitation can induce SIRS and disseminated intravascular coagulation. These consequences can culminate in multiorgan failure and death.

Systems Affected

- Cardiovascular: translocation of fluid into the stomach and hemorrhage from rupture of the short gastric and epiploic vessels can cause significant hypovolemia. Hypoxemia, electrolyte disturbances, acid–base imbalances, and release of myocardial depressant factors can result in myocardial dysrhythmias (e.g., ventricular premature contractions). Pain is considerable, and the sympathetic response can contribute to cardiovascular compromise.
- Gastrointestinal: gastric rotation and tearing of gastric blood supply causes reduced gastric blood flow and ischemia. Increased intraperitoneal pressures and decreased cardiac output reduce splanchnic blood flow.

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- Respiratory: upper airway obstruction from regurgitation and aspiration of swallowed saliva; decreased thoracic capacity leading to hypoventilation and hypoxia.
- Endocrine/metabolic: circulatory shock leads to lactic acidosis; fluid translocation can result in hypokalemia. Reperfusion injury expected with resuscitation and gastric derotation.
- Hemic/lymphatic/immune: bacterial and endotoxin translocation through disabled GI mucosal barrier into the lymphatics and bloodstream is expected. As the stomach rotates, it can pull at the short gastric vessels, causing a splenic torsion or splenic ischemia.
- Hepatobiliary: because the liver may be compromised from venous congestion and circulatory shock, bacteria may not be removed from the portal system, resulting in septicemia.



SIGNALMENT/HISTORY

Risk Factors

GDV can occur in any breed or species of animal, although it is most commonly seen in large breed dogs with increased thoracic depth to width ratio (e.g., Great Dane, Gordon and Irish Setter, Weimeraner, Standard Poodle, Basset Hound, Saint Bernard). Other characteristics associated with GDV include having a first-degree relative that suffered GDV and owner's perception of high-level stress in the dog. Additional factors thought to influence the formation of GDV include lean body condition, increasing age, large drops in environmental temperature over a short period of time, feeding single large meals, overeating, small kibble size, elevated feeding bowls, postprandial exercise, anesthesia, and aerophagia. The addition of table food or canned food to the diet of large and giant breed dogs has been associated with a decreased incidence of GDV.

Historical Findings

- Common presenting complaints include "vomiting" or dry heaves, describing nonproductive retching, restlessness, increased salivation, and abdominal distension.



CLINICAL FEATURES

- Poor perfusion based on brick red or pale mucous membrane color, rapid or prolonged capillary refill time, weak or bounding pulse quality.
- Tachycardia
- Hypersalivation
- Excessive efforts to swallow
- Abdominal distension with gastric tympany (this may be hidden under the rib cage in some cases) Tachypnea with or without labored breathing
- Nonproductive retching
- Occasionally splenomegaly can be palpated

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DIFFERENTIAL DIAGNOSIS

- Gastric dilation without volvulus
- Splenic torsion
- Intestinal volvulus
- Abdominal organomegaly
- Abdominal effusion
- Megaesophagus or esophageal foreign body
- Severe oropharyngeal or esophageal inflammation

DIAGNOSTICS

The physical exam findings that lead to a suspicion of GDV are listed in the clinical features. An immediate packed cell volume (PCV), total solids by refractometer, electrolyte panel, venous blood gas, glucose and lactate levels are evaluated and abnormalities determined. Because GDV is considered an emergency surgical procedure, preanesthetic laboratory analysis is indicated. This includes a complete blood count, serum chemistry, and prothrombin/activated partial thromboplastin times.

A right lateral abdominal radiograph is usually the most diagnostic view for confirming the diagnosis. Typical radiographic signs include gas- or fluid-dilated stomach and displacement of the pylorus in a dorsal position causing compartmentalization or "Popeye arm" (Fig. 35.1). The presence of gas within the gastric wall may occur with significant gastric wall necrosis. Occasionally, a left lateral or dorsoventral abdominal radiograph is also needed.

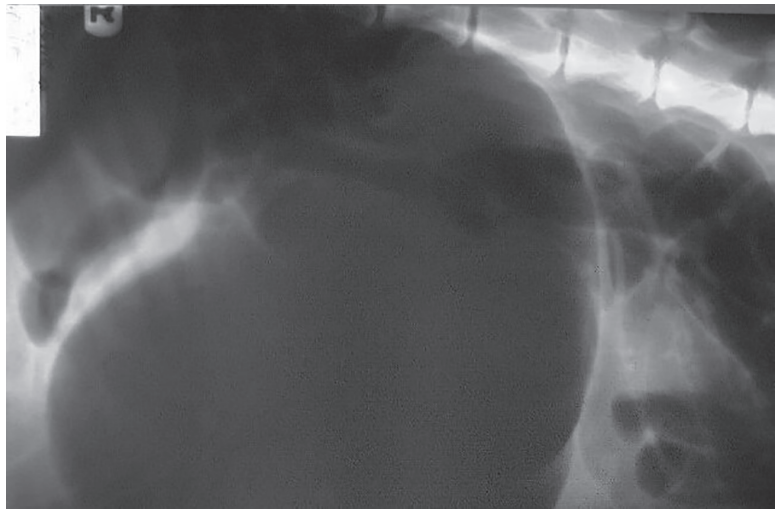


Fig. 35.1. Right lateral abdominal radiographs showing gas distension of the stomach with dorsocranial displacement of the pylorus, consistent with GDV.

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Right and left lateral and dorsoventral thoracic radiographs may be taken to rule out preexisting neoplastic disease or cardiomegaly suggestive of underlying cardiomyopathy. The presence of a megaesophagus is common and usually resolves with definitive treatment of GDV. Ventrodorsal radiographs are not recommended, since positioning can place extra pressure on the vena cava and additional compromise in venous return.

An electrocardiogram and blood pressure are also evaluated. It is not unusual to discover a ventricular dysrhythmia caused by myocardial hypoxia and circulating cytokines. Hypertension is also not uncommon, and usually corrects with fluid resuscitation and analgesia. When hypotension or bradycardia exist, severe to catastrophic shock may be occurring.

Pathologic Findings

Lactic acidosis is common, caused by severe circulatory shock. Anemia and hypoproteinemia can occur with hemorrhage and loss into the damaged GI tract. Increased lactate levels that do not improve with treatment are associated with increased complications. Decreased platelet count and increased clotting times are not unexpected, and can be caused by consumption from hemorrhage or a systemic inflammatory process. A significant coagulopathy with evidence of hemorrhage requires careful hemostasis during surgery and judicious use of synthetic colloids.

Ventricular dysrhythmias can occur pre-, intra-, and postoperatively. Esophageal dilation is also a reversible problem that usually corrects spontaneously following gastric repositioning.

The stomach can rotate in any direction. Most commonly, however, it rotates 180–270° in a clockwise direction (as viewed from caudal). This means the pylorus initially moves ventral and to the left and ultimately rests in the dorsal left quadrant of the abdomen. Various degrees of gastric injury will be encountered but most commonly, the cardia, up to the esophageal hiatus, and the greater curvature of the fundus of the stomach are affected. Changes can be minor, with only mild hyperemia seen, or major with black and ischemic gastric wall seen.

Peritoneal hemorrhage will most commonly occur from torn gastroepiploic and short gastric vessels. Additional intraoperative findings can include splenic enlargement or torsion, and pancreatic edema.



THERAPEUTICS

Intravascular volume resuscitation and analgesia therapy are the key to success, followed by decompression of the stomach.

Drugs of Choice

Fluid Resuscitation

Initially (multiple) peripheral IV catheters are inserted, and IV crystalloid and colloid fluids are administered in conjunction with analgesic drugs. A balanced, buffered, isotonic solution is rapidly bolused in incremental dosages (20–30 mL/kg) in conjunction with incremental dosages of colloids (such as hydroxyethyl starch or Oxyglobin™ if available; 5 mL/kg) until

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perfusion parameters are normal. These solutions preserve colloid osmotic pressure during fluid resuscitation. In addition, Oxyglobin™ carries oxygen to tissues with compromised blood flow and has a mild vasoconstricting effect which might be desired during SIRS states, such as occurs in the GDV patient. The rapidly deteriorating hypovolemic patient without significant hemorrhage may benefit from the infusion of hypertonic saline (4 mL/kg 7% solution) with hydroxyethyl starch in an effort to augment exogenous fluid infusion with interstitial fluid redistribution.

Balanced Analgesia

Pure agonist opioids (such as fentanyl 5 µg/kg IV followed by a continuous infusion of 3–5 µg/kg/h) are given with a benzodiazepine (such as midazolam, 0.2 mg/kg IV). Lidocaine (2–4 mg/kg bolus, followed by 50 µg/kg/h or 300 mg in 1 liter isotonic saline set at 2 mL/kg/h) is used as an adjunctive analgesic and when a significant ventricular dysrhythmia develops. Opioids and lidocaine can be mixed in the same fluid bag for continuous infusion.

Opioid infusion will not exacerbate attempts to vomit in the critically ill patient, and should not be withheld.

Antibiotics

Perioperative broad-spectrum antibiotics such as a first-generation cephalosporin or ampicillin (22 mg/kg IV) should be administered within 30 minutes of surgery. Although rarely required, additional antimicrobials may be selected for anaerobes or resistant gram-negative coverage based on findings at the time of surgery.

Gastric Decompression

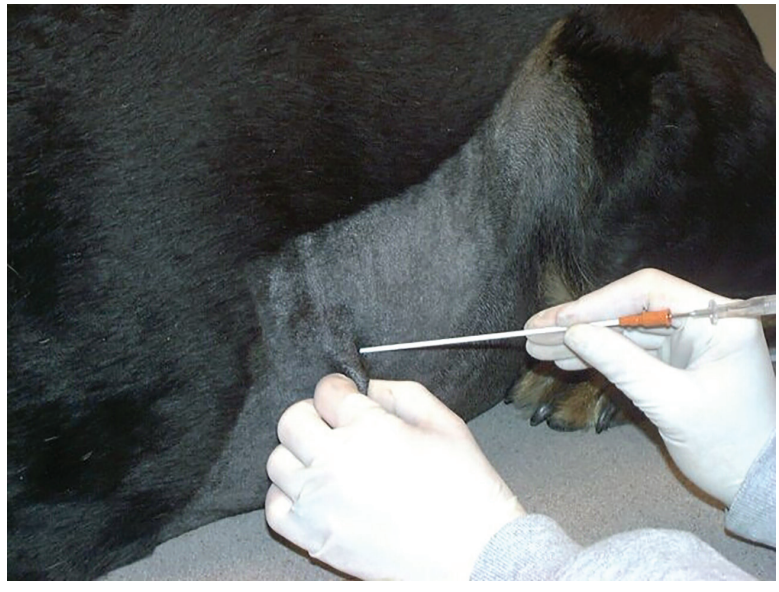
The stomach is decompressed if it can be percussed behind the ribs, or the distension is causing a significant degree of discomfort. Percutaneous decompression is less stressful, less painful, and requires minimal assistance compared to orogastric intubation and decompression. Percutaneous decompression is performed by sterilely preparing the skin over a tympanic region and percutaneously placing a large-bore (14–16 G) over-the-needle catheter. The stomach is gently pushed toward the catheter as it deflates. Once fluid and gas stop draining, the catheter is removed (Fig. 35.2).

Orogastric intubation can also be performed, but the patient should be anesthetized and tracheally intubated to reduce stress and discomfort, and protect the airway. There is a risk of esophageal and/or gastric trauma and perforation. For catastrophic cases that are dying before your eyes, or when immediate surgery is not possible, nasogastric intubation can be attempted.

Precautions/Interactions

Nonsteroidal antiinflammatory agents are not used because of potential adverse effects to the GI tract. Corticosteroids have not been shown to reduce morbidity or mortality in the GDV patient, and may increase risk of GI ulceration and sepsis.

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■ **Fig. 35.2.** Percutaneous decompression of the stomach via trocarization, using a 14 gauge long over-the-needle catheter.

Appropriate Healthcare

Clients should be informed that a primary gastric motility disorder may not be resolved with treatment and gastropexy, and some cases will continue to suffer gastric distension. This may or may not respond to promotility agents (e.g., metoclopramide or cisapride). Chronic GI signs warrant GI biopsy collection at the time of surgery and gastropexy.

Nursing Care

Postoperative care should focus on ensuring analgesia and overall comfort, wound management of the incision site, and catheter and feeding tube maintenance.

Diet

Feeding multiple meals, and hydrating kibble prior to feeding may reduce the incidence of bloating.

Activity

Increased, in general.

Surgical Considerations

The stomach usually does not return to normal position without surgery, and the degree of gastric necrosis cannot be determined medically. In addition, the risk of recurrence of GDV

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without gastropexy is 54–80%. Therefore emergency surgery is recommended following resuscitation to return the stomach to normal position, remove necrotic or ischemic gastric or splenic tissue, and pexy the pyloric antrum to the peritoneal wall.

Surgical preparation of the abdomen, ventral thorax, and inguinal area is preferably performed prior to anesthesia to minimize the time under anesthesia. The lateral abdomen is prepared for possible gastrostomy or jejunostomy feeding tube placement. The inguinal area is prepared in case central femoral vascular access is required intraoperatively.

Rapid IV anesthetic induction is performed so that immediate control of the airway can be taken. Ketamine (5 mg/kg) and midazolam/diazepam (0.25 mg/kg) or fentanyl (10 µg/kg IV) with diazepam (0.5–1.0 mg/kg IV) have few negative cardiovascular effects. Propofol 2–4 mg/kg can also be used. The amount of pressure placed on the diaphragm by the dilated stomach will hinder effective chest expansion. Assisted ventilation is recommended regardless of spontaneous ventilatory efforts. Epidural analgesia can reduce the amount of inhalant anesthetic required.

Appropriate intraoperative monitoring includes constant ECG for dysrhythmia detection. Blood pressure monitoring is important, especially when the stomach is decompressed completely, and the pressure on the central vein is released. A sudden decrease in preload can produce acute hypotension. In addition, many anesthetic agents augment hypotension (iso-, sevoflurane) or potentiate dysrhythmias (halothane). End-tidal CO_2 helps monitor for adequate ventilation. Pulse oximetry monitors trends of change in hemoglobin oxygen saturation.

A routine approach to the abdomen is made from xyphoid to pubis. The peritoneal wall is carefully incised to avoid inadvertent incision of the distended GI tract. This approach allows adequate exposure for complete evaluation of the abdominal organs. If there is significant hemorrhage, decompression of the stomach must first be performed to permit adequate exposure of the abdominal organs. An orogastric tube is passed with the surgeon gently guiding the tube into the stomach by palpation of the esophageal hiatus. Decompression will greatly facilitate derotation of the stomach. Usually the stomach will be rotated with the pylorus traveling ventrally and to the left, so one hand is used to press the right side of the dilated stomach down and the pyloric region is grasped with the other hand and gently pulled up ventrally and to the right. If passage of the tube is not possible, and the stomach cannot be derotated, a large-bore catheter stylet or needle can be used for centesis or attached directly to a suction apparatus for gas and fluid removal. If there is a large amount of food in the stomach, a gastrotomy is performed and the ingesta shoveled out.

Hemorrhage is controlled with packing and/or ligation following derotation of the stomach. Any questionable stomach wall integrity is noted, and the rest of the abdomen is explored. Gastrectomy may not be required once blood flow is reestablished. The spleen is often congested and may be displaced. Evaluation for thromboses is made by palpation of the splenic arteries and inspection of the color of the parenchyma. Lack of blood supply or dark purple color means the tissues have lost their blood supply and a partial or complete splenectomy is warranted. The pancreas is inspected for loss of blood supply and edema. The remaining intestines are inspected for pathology. The hepatobiliary and genitourinary organs are routinely evaluated.

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Once the exploratory is completed, the stomach is reinspected. If there are any black or dark purple regions, these should be removed with TA staples or resection. Any area that does not bleed bright red blood is suspect and should be removed. Large invaginations of ischemic tissues are not recommended because of potential life-threatening hemorrhage that can occur when the tissues slough. In contrast to the serosa, when the mucosa is black resection is not always necessary. The mucosa will regenerate if there is a healthy submucosa. However, it is the author's experience that removal of compromised but viable tissue results in decreased hospital stay. Postoperative monitoring for intragastric hemorrhage is important. The spleen is reinspected and either partially or completely removed if infarcted areas exist.

The type of pexy performed is completely dependent on the surgeon's preference and the time involved. The incisional gastropexy is rapidly performed and provides reliable adhesion. It is not recommended to suture the gastric wall to the midline abdominal incision, even in the critical GDV patient. Any future abdominal surgeries will be severely compromised by the adhesions formed.

Maintaining gastric decompression postoperatively is recommended in the critical GDV patient. Nasogastric tubes are preferably placed intraoperatively with proper placement assured by palpation. Gastrostomy tube placement allows large-volume decompression and removal of large clots that can occur with large resections. Monitoring gastric residual volume and immediate enteral feeding are facilitated with feeding tube placement.

Jejunostomy or nasojejunostomy tube placement may be necessary if any significant gastric resection or pancreatic trauma occurs.

Copious saline lavage and suction of the abdomen is necessary. A routine three-layer closure is performed and dressings are placed over the incision site and ostomy tube sites. Any evidence of peritonitis warrants culture and sensitivity of the peritoneum and abdominal drainage.



COMMENTS

Client Education

Following the recovery period, few restrictions are necessary. More frequent, lower volume feedings may prevent a recurrence of gastric dilation.

Prevention/Avoidance

More frequent, lower volume meals may reduce the risk of GDV. Prophylactic gastropexy will reduce the incidence of GDV, and should be considered in dogs at risk, or when any intraabdominal surgery is being performed in dogs at risk. Finally, not breeding dogs that have an affected first-degree relative or are a breed at risk for GDV can reduce the overall incidence of GDV.

Possible Complications

Gastric necrosis, gastric perforation, septic peritonitis, circulatory shock, and death are expected to occur in the untreated GDV patient.

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Expected Course and Prognosis

Prognosis is good to excellent for dogs that receive immediate therapy and a gastropexy procedure.

Recently reported mortality rates are around 10% for dogs with GDV. Risk factors significantly associated with death prior to suture removal are a persistent hyperlactatemia, combined splenectomy and partial gastrectomy, hypotension at any time, peritonitis, sepsis, disseminated intravascular coagulation, and presenting in a comatose state. Incidence of acute kidney injury and dysrhythmias may be reduced by initiating a continuous infusion of lidocaine (2 mg/kg/h) during stabilization.

There is a reported recurrence rate of 4–6% of GDV in dogs following gastropexy. In addition, the gastropexy does not correct problems causing delayed gastric emptying.

Synonyms

Bloat

Abbreviations

ECG: electrocardiogram
GDV: gastric dilation-volvulus
GI: gastrointestinal
SIRS: systemic inflammatory response syndrome
IV: intravenous
PCV: packed cell volume
TA: thoracoabdominal

Suggested Reading

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chapter 36

Gastrointestinal Foreign Body/Obstruction

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DEFINITION/OVERVIEW

- Gastrointestinal obstruction is a common condition encountered in small animal medicine.
- Obstruction is classically categorized as either mechanical or functional. Mechanical obstruction in the gastrointestinal tract is most often due to entrapped foreign material within the gastrointestinal lumen, but it can also be caused by intussusception, intestinal entrapment, torsion, mucosal or muscular hypertrophy, or neoplasia. Functional obstruction of the gastrointestinal tract, also referred to as ileus, most often results from inflammation or infection and is specifically addressed elsewhere.

ETIOLOGY/PATHOPHYSIOLOGY

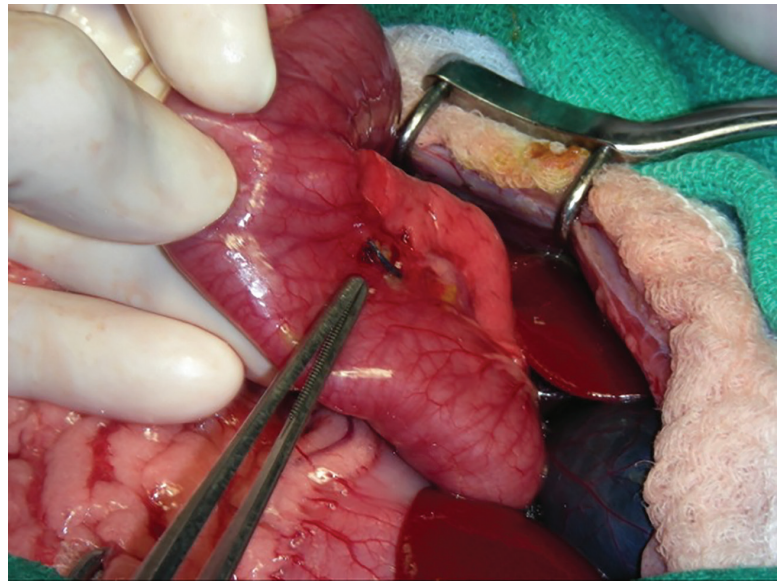
- Gastrointestinal mechanical obstruction may occur following indiscriminate ingestion of nonfood items. Frequently ingested items that may cause a focal obstruction include rocks, balls, toys, hair (trichobezoars), corncobs, peach pits, plant material, and clothing items.
- Focal obstruction most commonly occurs at areas where the stomach and small intestine narrow (i.e., pylorus, caudal duodenal flexure, and ileocolic junction), although items may become lodged at any point along the gastrointestinal tract.
- Gastrointestinal obstruction with linear material is a unique situation most commonly associated with cats, but can occur frequently in dogs as well. Foreign bodies such as string, thread, or cloth are ingested, become anchored at the base of the tongue or at the pylorus, and then cause a plicated obstruction as the intestinal tract attempts to move the piece of linear material caudally.

Systems Affected

- Gastrointestinal: complete obstruction results in gas and fluid distension orad to the foreign body. Blood supply to the mucosa and submucosa is damaged, and full-thickness wall necrosis with perforation may occur at the obstruction site. With linear foreign body

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■ **Fig. 36.1.** Intraoperative photograph of a kitten with a linear foreign body showing a perforation along the mesenteric border of the jejunum.

ingestion, peristalsis advances the material into the intestine, and as a result of the anchor, the intestine will gather around the foreign body. This plication of the intestines may cause a complete or, more often, partial obstruction. Intestinal peristalsis continues against the fixed material and may lacerate the mucosa and cause perforations along the mesenteric border (Fig. 36.1). The resulting peritonitis from gastrointestinal perforation secondary to foreign body ingestion is associated with poor wound healing and a high mortality rate.

- Cardiovascular: complete mechanical gastrointestinal obstructions can lead to breakdown of the mucosal barrier, wall necrosis, or perforation and have a high likelihood of causing hypovolemic and septic shock.



SIGNALMENT/HISTORY

- There is no specific signalment associated with gastrointestinal foreign body obstruction, although there is a tendency for dogs and cats to be relatively young (<4 years).
- Clinical signs associated with gastrointestinal obstruction are influenced by location, completeness, and duration of obstruction.
- Acute obstructions have a history most consistent with an acute abdomen: vomiting, bloody diarrhea, and abdominal pain (Fig. 36.2).
- Complete high obstructions are associated with protracted vomiting, severe dehydration, electrolyte imbalance, and hypovolemic shock.
- Animals with chronic, partial, or low obstructions may have more vague clinical signs: inappetence, weight loss, occasional vomiting, and diarrhea.

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Fig. 36.2. A 5-year-old female pug in the "praying" position demonstrating acute abdominal pain secondary to a small intestinal foreign body.

- Historical information may be valuable in aiding the diagnosis of gastrointestinal obstruction, as owners may report witnessing foreign material ingestion or know that objects from their home are missing.

CLINICAL FEATURES

- Physical examination may reveal a guarded painful abdomen. Animals will frequently display clinical signs consistent with dehydration: abnormal skin tent, dry mucous membranes, delayed CRT, and sunken globes.
- With linear foreign body obstruction, abdominal palpation may or may not painful, but may reveal a large mass of bundled intestines ventrally. Careful oral examination may reveal the foreign material anchored around the base of the tongue, particularly in cats.

DIFFERENTIAL DIAGNOSIS

- Gastroenteritis
- Acute pancreatitis
- Mesenteric torsion
- Intussusception
- Benign pyloric outflow obstruction
- Functional ileus
- Neoplasia
- Feline infectious peritonitis (cats)

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DIAGNOSTICS

- Survey abdominal radiographs: occasionally, radioopaque foreign material causing obstruction can be visualized on abdominal radiographs. However, when the foreign body cannot be seen, mechanical obstruction may cause varying degrees of gastric and small intestinal dilation, depending on the location, degree, and duration of obstruction.
- For linear foreign body obstruction, the classic radiographic pattern of gathered intestines to the right of the midline may be apparent, but intestinal loops are not usually abnormally dilated as the obstruction is incomplete. Small, eccentrically located luminal gas bubbles, tapered at one or both ends, are a more typical finding for linear foreign body obstruction.
- Additional imaging: if the diagnosis is still uncertain, survey radiographs may be repeated at a later time or other imaging techniques, such as upper gastrointestinal contrast studies or abdominal ultrasonography, may be performed.
- Abdominal ultrasound has been shown to be beneficial in the diagnosis of small intestinal mechanical obstruction. It may detect foreign bodies by the distal acoustic shadowing and may identify segmental small intestinal dilation.
- Abdominal ultrasound may also detect linear foreign body obstruction by identifying intestinal plication on either side of a central hyperechoic line (Fig. 36.3).
- Gastrointestinal contrast studies can be performed when radiographic or ultrasonographic imaging is uncertain. However, barium contrast agents are contraindicated if perforation

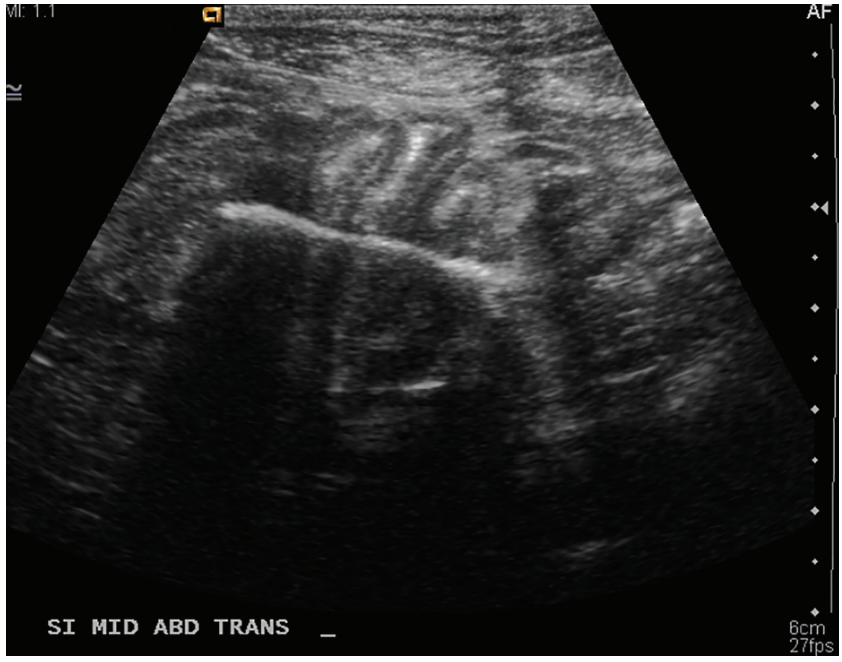


Fig. 36.3. A static ultrasonographic image demonstrating linear foreign body partial obstruction with small intestinal plication.

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is suspected and iodinated contrast agents may exacerbate dehydration. A pneumocolonogram is an easy negative contrast study that may be helpful in differentiating the large intestine from gas-distended small intestine.

- Laboratory findings may demonstrate a wide range of electrolyte abnormalities, acid–base disturbances, anemia, hypoalbuminemia, stress or inflammatory leukograms, and evidence of dehydration.
- Obstruction at the pylorus or in the proximal duodenum may result in a classic hypochloremic, hypokalemic metabolic alkalosis that occurs from loss of electrolytes in protracted vomiting. However, these derangements can occur with distal obstruction as well.



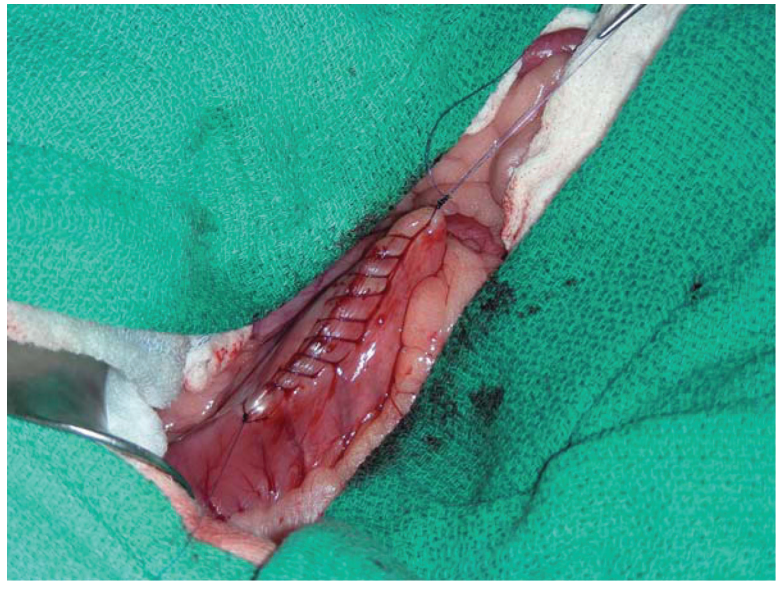
THERAPEUTICS

- Gastrointestinal obstruction is considered a surgical emergency. Prior to surgery, the animal's hemodynamic and electrolyte status should be stabilized, although surgery should not be delayed unnecessarily. A complete and thorough abdominal exploratory is necessary to locate suspected obstructions. Conservative management of linear foreign bodies has been reported in cats, but many animals managed medically ultimately require surgery.

Surgical Considerations

- Prophylactic antimicrobials are indicated as these surgeries are at a minimum classified as clean contaminated.
- Synthetic absorbable monofilament suture (3-0 to 4-0) with a swaged-on taper needle is the material of choice for most gastrointestinal surgery.
- There are numerous techniques described for closure of gastrointestinal incisions. Regardless of the suture pattern chosen, inclusion of the submucosal layer in the closure is vital. Full-thickness purchase of the tissue ensures that this holding layer is incorporated in the suture line.
- Gastrotomy is required for removal of gastric foreign bodies. The ventral body of the stomach is incised with a longitudinal incision between the lesser and greater curvature large enough to remove the foreign material. Specific options for gastrotomy closure include the following.
    - Single-layer full-thickness simple continuous pattern (Fig. 36.4)
    - Two-layer continuous inverting pattern
        - Full-thickness simple continuous pattern, followed by
        - Partial-thickness (seromuscular) Lembert or Cushing pattern
    - Two-layer continuous inverting pattern
        - Simple continuous to close the mucosa and submucosa, followed by
        - Partial-thickness (seromuscular) Lembert or Cushing pattern
- For intestinal foreign bodies, subjective criteria are most often used to determine the viability of the tissue at the level of the obstruction and to decide whether simple enterotomy

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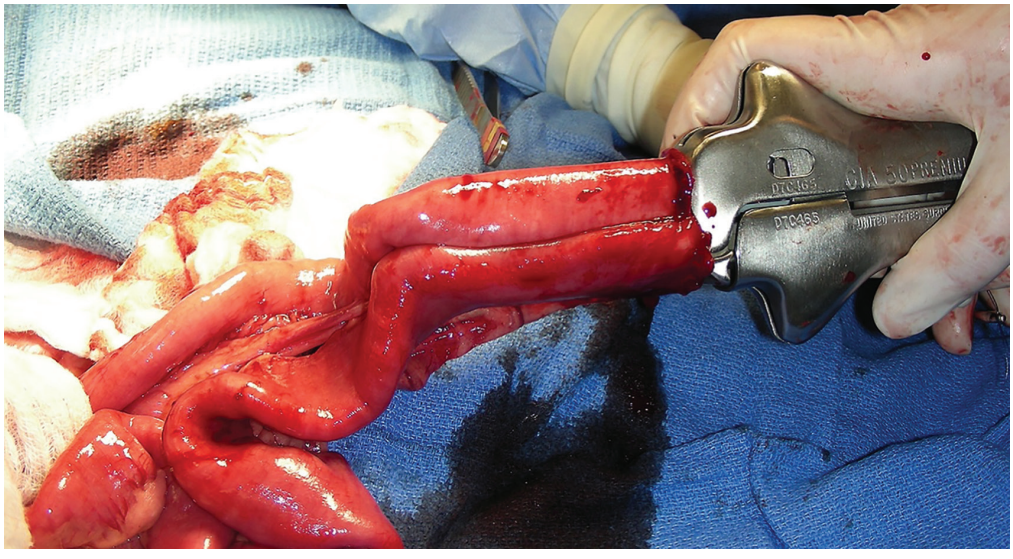


■ **Fig. 36.4.** Single-layer simple continuous closure of a gastrotomy incision following foreign body removal.

or intestinal resection and anastomosis is indicated to address the problem. These parameters include color, peristalsis, arterial pulsation, capillary (cut surface) bleeding, and tissue thickness. Multiple enterotomies are typically required for removal of linear foreign bodies. This allows for segmental removal of the material and minimizes the risk of iatrogenic perforation from too much traction.

- As with gastrotomy incisions, there are numerous ways to close simple enterotomies, but double-layer closures should be avoided due to the potential for luminal compromise. Some options for simple enterotomy closure include the following.
  - Single-layer simple continuous appositional pattern (Fig. 36.5)
  - Single-layer simple interrupted appositional pattern
- If intestinal resection and anastomosis are indicated, single-layer simple interrupted or modified simple continuous approximating patterns are also preferred. In a modified technique, two suture lines are used: one originating at the mesenteric border and the other originating at the antimesenteric border. A single-layer full-thickness continuous suture line is placed from the mesenteric knot to the stay suture at the antimesenteric knot with tissue purchases 2 mm from the wound edge and 2–3 mm apart. This is repeated on the other side from the antimesenteric knot to the mesenteric knot. There is no difference in reported rates of dehiscence between animals with simple continuous anastomotic closures and animals with simple interrupted closures.
- An alternative to performing a hand-sewn anastomosis following intestinal resection is to use surgical staples to create a functional end-to-end anastomosis.

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■ **Fig. 36.5.** Intraoperative photograph of creation of a stapled functional end-to-end anastomosis using a gastrointestinal anastomotic stapler (GIA).

COMMENTS

Possible Complications

- Early recognition may prevent most major complications, but intestinal necrosis, perforation, dehiscence, peritonitis, and sepsis are all possible complications for gastrointestinal obstruction.

Expected Course and Prognosis

- Following surgery, the prognosis for animals with mechanical gastrointestinal obstruction is generally good to excellent.
- A worse prognosis is associated with linear foreign body obstruction, long duration of clinical signs, and multiple intestinal procedures.
- If the gastrointestinal tract has been perforated and peritonitis is present, the prognosis becomes guarded to poor. Perforated intestine from foreign material can carry a mortality rate around 50%.
- If proper surgical technique is not used or if risk factors for poor intestinal healing are present (i.e., hypoalbuminemia, peritonitis, immunosuppression), dehiscence of gastrointestinal incisions may occur; mortality associated with this scenario approaches 75–85%.

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Abbreviations

CRT: capillary refill time

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chapter 37

Glaucoma

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DEFINITION/OVERVIEW

- The definition of glaucoma has changed considerably over time and glaucoma is now considered to be a group of related diseases in which an elevation of IOP is a consistent feature.
- The increased IOP is detrimental to vision and ocular health and results in discomfort.
- Primary glaucoma is a bilateral disease but in most cases, one eye becomes affected before the other.

ETIOLOGY/PATHOPHYSIOLOGY

- Arises from decreased drainage of AH from the eye.
- Intraocular fluid production cannot be high enough to increase IOP unless drainage through the iridocorneal (drainage) angle is severely reduced.
- Primary glaucoma is due to an anatomical defect in the iridocorneal angle; the angle may either be too narrow or may be covered with the remnant of an embryonic sheet of tissue.
    - It is a breed-related disease in dogs and cats; very common in dogs but rarely seen in cats.
- Secondary glaucoma is an increase in IOP due to reduced AH drainage secondary to another ocular condition, such as uveitis, neoplasia or lens luxation.
    - Secondary glaucoma is the most common type seen in cats and is also common in dogs.
    - The most common cause of secondary glaucoma in dogs is lens-induced uveitis secondary to cataract formation, although infectious causes and neoplastic causes are quite common as well.

Systems Affected

- Glaucoma only affects the ophthalmic system.

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SIGNALMENT/HISTORY

- Many breeds of dogs are affected with primary glaucoma (Box 37.1).
- In cats, breed predisposition for glaucoma has been reported in Siamese, Persian, Burmese, and European shorthair; glaucoma is rare in cats in general but may occur in older cats of any breed.
- Sex predilection for primary glaucoma in dogs varies by decade but in some breeds (e.g., American and English cocker spaniels, basset hound, Cairn terrier, Chow, Samoyed, Siberian husky, and Welsh springer spaniel), females are more often affected.
- No sex predilection has been noted in cats.
- There is no sex predilection for secondary glaucoma in dogs or cats except in cases caused by primary diseases with given sex predilections (e.g., female cats with aqueous humor misdirection syndrome).
- Age of onset of primary glaucoma in dogs varies considerably by breed but ranges from 4 to 10 years, with median age for many breeds falling around 6 years.
- No age of onset for glaucoma has been reported in cats, although primary noncongenital glaucoma is noted to be more common in senile cats.

Historical Findings

- Squinting and epiphora from the affected eye: varies from pronounced in some cases to absent in others.
- Red eye: due to episcleral congestion (Fig. 37.1) with or without conjunctival hyperemia.

BOX 37.1. Breeds of dogs reported to have primary glaucoma (adapted from Plummer et al. 2013).

Akita	Dalmatian	Norwich terrier
Alaskan malamute	Dandie Dinmont terrier	Poodle (toy/miniature)
Basset hound	English springer spaniel	Samoyed
Beagle	German shepherd	Scottish terrier
Border collie	Giant schnauzer	Sealyham terrier
Boston terrier	Greyhound	Shih tzu
Bouvier des Flandres	Irish setter	Siberian husky
Brittany spaniel	Italian greyhound	Skye terrier
Cairn terrier	Lakeland terrier	Smooth fox terrier
Cardigan Welsh corgi	Maltese	Tibetan terrier
Chihuahua	Miniature pinscher	Welsh springer spaniel
Cocker spaniel, American	Miniature schnauzer	Welsh terrier
Cocker spaniel, English	Norfolk terrier	West Highland white terrier
Dachshund	Norwegian elkhound	Wirehaired fox terrier

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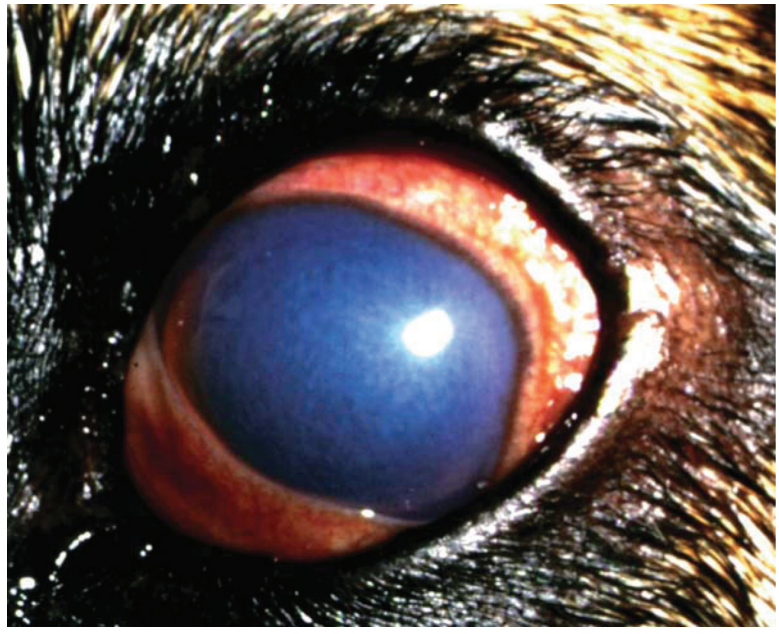


Fig. 37.1. Red sclera (episcleral injection) with corneal cloudiness (corneal edema) consistent with glaucoma.

- Cloudy eye: due to corneal edema; this may give the appearance of an enlarged eye even in the absence of true buphthalmos.
- Pets may be more quiet and depressed than usual.
- Cats often have no historical findings.
- Vision loss may or may not be noted, depending on the individual pet and the quality of vision in the contralateral eye.

CLINICAL FEATURES

Dogs

- Clinical signs vary according to stage and type of glaucoma.
- Early primary glaucoma is insidious; clinical signs may be very subtle and can be missed unless IOPs are obtained. The signs include slight or intermittent mydriasis, mild corneal edema, variable episcleral injection, and mild elevations in IOP (25–30 mmHg).
- Signs of moderate to advanced glaucoma include pronounced mydriasis, episcleral injection, distinct corneal edema, and variable vision loss. IOP usually ranges from 30 to 90 mmHg. Optic disk changes may be present (dark, cupped), depending on duration of pressure elevation.
- Chronic glaucoma is characterized by persistent mydriasis, blindness, corneal edema, episcleral injection, and optic disk changes (dark, cupped). Conjunctival hyperemia and regions of tapetal hyperreflectivity in the fundus may be noted as well. Some cases will

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■ **Fig. 37.2.** End-stage glaucoma in a Siberian husky. Note the cloudy cornea and buphthalmic appearance to the dog's left eye.

develop buphthalmos (ocular enlargement) over time, which may in turn lead to Haab's striae in the cornea and lens subluxation or luxation (usually posterior). Intraocular pressure may be very high (40–90 mmHg) (Fig. 37.2).

- Signs of secondary glaucoma are those of primary glaucoma, with the addition of the signs of the primary cause (e.g., uveitis, intraocular neoplasm, lens luxation). A miotic pupil in the presence of elevated intraocular pressure should increase suspicion for secondary glaucoma.

Cats

- Clinical signs are often more subtle than in dogs and therefore may be easily overlooked.
  - Frequently, clinical signs have been present for an extended period and blindness and buphthalmia are present.
  - Secondary glaucoma is more common than primary glaucoma in cats, however, and patients may present with signs of uveitis or intraocular neoplasms and subsequently be diagnosed with elevated intraocular pressures.
- Mydriasis is the most consistent finding in early glaucoma.
- Episcleral injection and conspicuous corneal edema (which occur in dogs) usually do not develop in cats.
- Elevated IOP is present.

DIFFERENTIAL DIAGNOSIS

- Conjunctivitis: redness limited to conjunctival hyperemia; less severe discomfort, which is usually alleviated by topical anesthetic.
- No corneal edema, episcleral injection, or buphthalmos; normal vision.

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- Uveitis: aqueous flare is present, IOP is low, pupil is usually miotic.
- Ulcerative keratitis: corneal fluorescence staining is positive, corneal edema is localized to one or several areas, and discomfort is alleviated by topical anesthetic.
- Corneal endothelial dystrophy: diffuse corneal edema but not painful, IOP is normal, and there is limited to no conjunctival hyperemia or episcleral injection.

DIAGNOSTICS

- Tonometry: elevated IOP. May range from 25 to 75+ mmHg depending on stage and severity. This is the definitive test, although false positives are possible for inexperienced operators or with fractious patients due to manipulation of eyelids or pressure on the jugular veins during restraint.
- Gonioscopy: allows for direct visualization of the iridocorneal angle (Fig. 37.3).
  - Facilitates the differentiation of primary from secondary glaucoma and prognosis of the unaffected eye.
- Ophthalmoscopy: to evaluate the optic disk for cupping.
  - Allows for prognosis. A cupped optic disk indicates a poor prognosis for vision.

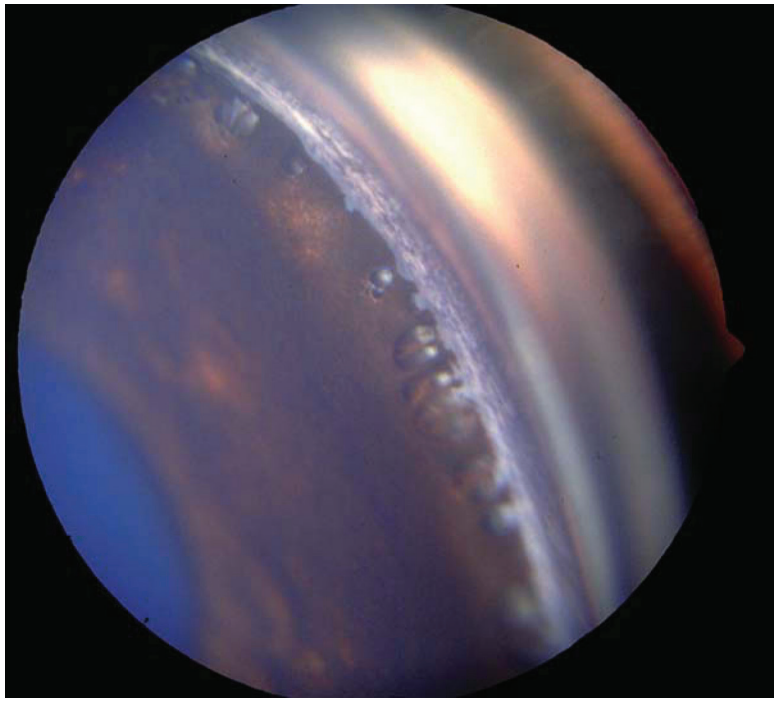


Fig. 37.3. Drainage angle in a dog with glaucoma.

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THERAPEUTICS

- Treatment modality is initially based on the absence or presence of vision on presentation, prognosis, willingness of the owner to treat the patient, cost of therapy, and patient temperament.
- Objective of therapy in sighted or recently sighted eyes is to lower the IOP as rapidly as possible with the goal of minimizing damage to the retina and optic nerve. This may preserve vision and will alleviate pain.
- Objective of prophylactic therapy in the contralateral, at-risk eye in primary glaucoma cases is to delay the onset of elevated intraocular pressure.

Medical Therapy for Primary Glaucoma

Initial Medical Intraocular Pressure Control (Emergency)

- For acute-onset glaucoma, emergency medications should be administered and intraocular pressure should be monitored to ensure response to medications. This may require patient hospitalization. After administration of initial medications, generally recheck pressure at 1 hour to assess for responsiveness and determine whether further intervention is needed.
- A sample protocol for acute primary glaucoma with significant pressure elevation might be:
  - 1 drop each of latanoprost and dorzolamide-timolol, recheck in 1 hour.
  - If significant drop, continue therapy at recommended dosing.
  - If moderate effect, recheck again in 1 hour .
  - If no effect or incomplete effect, consider addition of osmotic therapy.
  - See below for details and contraindications for specific medications.
- Prostaglandins: topical
  - Latanoprost, travoprost, or bimatoprost: topical; apply 1 drop every 12 hours.
  - Most effective class of antiglaucoma medication at eliciting a rapid drop in intraocular pressure in the emergency setting.
  - Cause profound miosis.
  - Ineffective in cats.
  - Contraindications:
    - Confirmed or suspected anterior lens luxation (for example, any Jack Russell Terrier with acute-onset corneal edema/discomfort where the lens cannot be visualized clearly to rule out anterior luxation).
    - Avoided if possible as first line of therapy in secondary glaucoma with uveitis, although may be indicated if other antiglaucoma medications fail to reduce IOP.
- Non-prostaglandin miotics, topical: less widely used now that prostaglandin analogs are less cost-prohibitive and easily available.
  - Parasympathomimetics.
    - Pilocarpine (1% or 2%): duration of action ~6 hours so requires 3–4 times daily dosing; irritating.
    - Demecarium bromide (0.125% or 0.250%): apply once daily; contraindicated in cats; compounded only.

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- Carbonic anhydrase inhibitors.
  - Topical: dorzolamide hydrochloride, brinzolamide.
    - Apply 1 drop every 8 hours.
    - Effective in dogs and cats.
    - No true contraindications, although dorzolamide is irritating in some patients; brinzolamide is more costly but frequently better tolerated in those patients.
- Beta-blockers.
  - Timolol (0.5%), betaxolol: apply 1 drop every 12 hours.
  - Limited effect as sole therapy in veterinary species; additive effect with carbonic anhydrase inhibitors.
  - Few side effects, use caution in patients with asthma or cardiac failure – systemic absorption is well documented.
- Fixed-combination dorzolamide hydrochloride 2%/timolol maleate 0.5%.
  - Apply 1 drop every 12 hours.
  - Synergistic effect.
  - See contraindications, etc., for individual medications, earlier in this chapter.
- Neuroprotective drugs.
  - Calcium channel blockers; oral.
    - Amlodipine *may* help protect retinal ganglion cells during initial treatment period; no specific dosing regimen is documented; generally dosing used is lower than that recommended for control of systemic hypertension.
- Osmotic diuretics: for **temporary** IOP reduction **only in an emergency setting**, in cases where medications alone are ineffective at reducing IOP; antiglaucoma medical therapy must be initiated concurrently to maintain IOP reduction.
  - Mannitol, intravenous: give 1–2 g/kg over 20-minute period; onset 30–60 minutes; duration 5–6 hours; can be repeated. Contraindicated if concurrent congestive heart failure, renal disease, systemic hypertension, or dehydration are present. Withhold water or intravenous fluids for 2–4 hours.
  - Glycerine (glycerine USP 75%, Osmoglyn 50%), oral: give 1–2 mL/kg; not as effective as mannitol; onset 10–30 minutes; duration 4–5 hours; can be repeated; contraindicated if concurrent diabetes mellitus, congestive heart failure, renal disease, systemic hypertension, or dehydration are present. Care should be taken to give slowly so dog does not inhale it. Gastrointestinal side effects may occur. Will not work well if concurrent uveitis is present.

Long-Term Intraocular Pressure Control

- Prostaglandin analogs, beta-blockers, and CAIs may be used long term at dosing frequencies indicated above.
- Often surgical therapy is required to maintain vision long term.
- For primary glaucoma, prophylactic treatment is recommended for the at-risk eye to delay onset of glaucoma.
  - BID timolol is an effective and inexpensive option.

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- Alternatively, for convenience, use the antiglaucoma medication employed in the affected eye for prophylaxis in the contralateral eye as well.
- Topical steroids may be beneficial in prophylaxis protocols.

Surgical Therapy for Primary Glaucoma

- Patient selection is important; must have sighted eye; increasing IOP despite medical therapy indicates possible candidates.
- Early surgical intervention is recommended; best potential to save vision.
- Two types are available; can use both approaches for some patients.
  - Surgeries that construct alternate drainage methods: anterior chamber shunts (gonioimplants); variable success rate long term, ~50% visual at 1 year; many complications; should be performed by ophthalmologist.
  - Surgeries that decrease AH formation: transscleral cyclophotocoagulation (LASER), endoscopic cyclophotocoagulation; these destroy part of the ciliary body which produces AH; should be performed by ophthalmologist.

Treatment of End-Stage Primary Glaucoma

- Used when the eye is blind.
- Objectives of therapy are to provide a pain-free, cosmetically acceptable situation.
- Available procedures.
  - Enucleation: resolves all need for topical antiglaucoma therapy, eliminates discomfort after short healing period, allows for histopathologic evaluation to confirm primary versus secondary glaucoma; high success rate.
  - Evisceration and intrascleral prosthesis: removal of the contents of the eye with intrascleral prosthesis placement; more cosmetically acceptable for some clients; the eye is still in place and is subject to trauma and ulcers. Should be performed by an ophthalmologist.
  - Pharmacologic cycloablation: injection of gentamicin (or cidofovir) into the eye to ablate the ciliary body epithelium and reduce aqueous humor production; not as reliable and second injection is required in some patients; eye is still in place, may become smaller and cloudy, and is subject to trauma and ulcers; performed with sedation rather than general anesthesia by many ophthalmologists, which may make this a preferable option for some cases; should be employed only for cases of primary glaucoma. Contraindicated in cats due to concern for posttraumatic sarcoma development. Performance by an ophthalmologist is recommended to avoid severe inflammation if the lens is damaged during the procedure.

Treatment of Secondary Glaucoma

- Objectives of therapy: treat the primary cause as well as lower IOP; treat uveitis (including cataract-associated inflammation) with topical antiinflammatories and systemic medications as indicated; surgical removal is indicated in lens luxation; enucleation indicated for most cases of intraocular neoplasia with elevated IOP.

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- Treatment to lower IOP is as listed previously.
- Atropine is contraindicated in eyes with elevated intraocular pressure.

Precautions/Interactions

- If uveitis is present, osmotic diuretics are ineffective and could worsen the glaucoma; miotics and prostaglandin analogs (that induce miosis) are contraindicated in cases of lens luxation; osmotics should not be used if there has been head trauma or if there is dehydration, congestive heart failure, hypertension or renal disease; glycerine should not be used in cases of diabetes mellitus; atropine is contraindicated with glaucoma; diphenhydramine may be contraindicated in glaucoma patients due to theoretical anticholinergic-mediated mydriasis.

Diet

- No changes in patient's diet are necessary.

COMMENTS

Client Education

- It is important to inform clients that primary glaucoma can only be controlled, not cured, and that in almost all cases vision will be lost eventually.
- Medications must be given as directed or IOP can elevate quickly and vision can be rapidly lost. Plan ahead for medication refills.
- Monitor for squinting, redness, and cloudy cornea.
- The contralateral eye is likely to develop elevated intraocular pressure within a year of diagnosis in the affected eye with primary glaucoma; use of prophylactic antiglaucoma medications significantly delays onset in the contralateral eye.

Patient Monitoring

- Successful reduction of intraocular pressure should be documented by monitoring response to treatments prior to discharge in acute-onset glaucoma, as persistently elevated intraocular pressure can cause significant, permanent damage in a matter of hours.
- After an acute glaucoma episode, the IOP should be checked in 24 hours and, if low, again in 5–7 days. Frequency of subsequent rechecks is based on perceived degree of intraocular pressure control.

Prevention/Avoidance

- There is no way to prevent primary glaucoma; because one eye often becomes affected before the other, the unaffected eye should be treated with an antiglaucoma drug as this has been shown to significantly delay the onset of elevated intraocular pressure.
- Discourage breeding of affected dogs (primary glaucoma).

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Possible Complications

- Few systemic complications; vision loss almost always occurs.

Expected Course and Prognosis

- Primary glaucoma will almost always progress to blindness; with therapy, vision can be retained for variable periods of time and pain can be eliminated.
- Many patients maintain a good quality of life even with vision loss, so long as pressure is controlled or salvage procedures are pursued to alleviate discomfort.

Abbreviations

AH: aqueous humor
BID: twice a day (bis in die)
CAI: carbonic anhydrase inhibitor
IOP: intraocular pressure

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chapter 38

Heat Stroke and Heat-Induced Illness

DEFINITION/OVERVIEW

- Hyperthermia can be categorized as pyrogenic hyperthermia (pyrexia or fever) or nonpyrogenic hyperthermia.
- Heat stroke, or heat-induced illness, occurs when heat-dissipating mechanisms are exceeded by heat gain, and the body cannot accommodate excessive heat. When left untreated and severe, heat stroke often leads to multiorgan dysfunction or failure, and could lead to death.
- Heat stroke, or nonpyrogenic hyperthermia, is most commonly observed during warm and humid months, although it can occur at any time of year, depending on the predisposing cause and patient risk factors.
- When body temperature exceeds 106 °F (41 °C) without signs of inflammation, nonpyrogenic hyperthermia exists (Fig. 38.1).

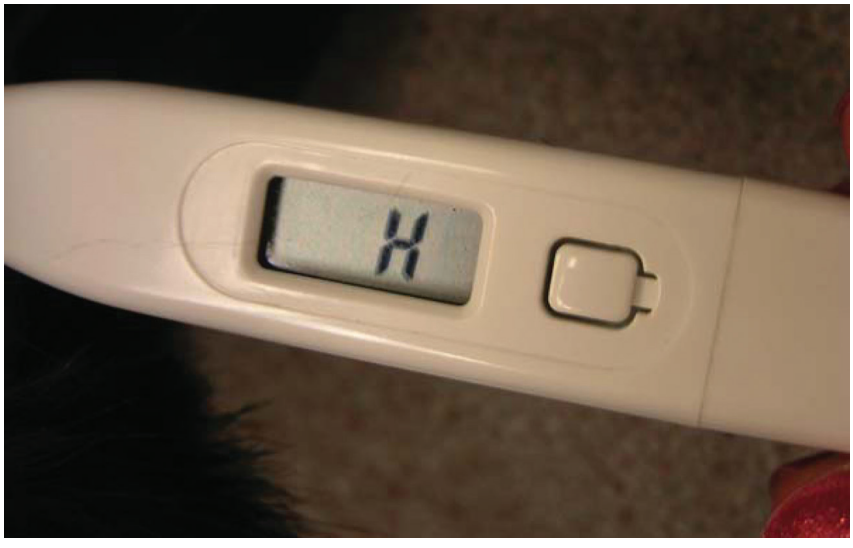


Fig. 38.1. Thermometer demonstrating rectal temperature too high to register (>109 °F) in a dog with heat stroke.

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- Malignant hyperthermia is an uncommon condition that causes nonpyrogenic hyperthermia that can occur due to inhalation of anesthetic agents and is familial in some breeds of dog, including the Labrador retriever.
- Other causes of hyperthermia and heat-induced illness include excessive exercise, thyrotoxicosis, hypothalamic lesions/dysfunction, and status epilepticus.



ETIOLOGY/PATHOPHYSIOLOGY

- Under normal circumstances, body temperature is regulated by a balance of heat gain and compensatory mechanisms for heat loss or dissipation. Nonpyrogenic hyperthermia, or heat stroke, occurs when heat-dissipating mechanisms become overwhelmed, and the core body temperature rises.
- Normal heat dissipation mechanisms in small animals include evaporative loss through the respiratory tract during panting and hypersalivation, convective and radiation losses from peripheral vasodilation, and seeking cooler locations. Warm blood at the periphery loses some heat as the body lies on a cool surface, for example.
- Nonexertional heat stroke occurs when an animal is enclosed in a warm environment and cannot escape.
- Exertional heat stroke occurs when an animal exercises in a warm environment and has not been acclimated or allowed to rest. Strenuous exercise in warm and humid environments can cause the normal heat-dissipating mechanisms to be overwhelmed, and core body temperature rises above the hypothalamic setpoint. Vascular endothelial injury, sludging of blood flow, vasodilation, and hypotension contribute to decreased vital organ perfusion and tissue ischemia. Without treatment, damage to the GI tract, liver, kidneys, heart, and central nervous tissue occurs, and can lead to SIRS, DIC, and MODS. The most extreme cases are often fatal.

Systems Affected

- Neurologic: direct thermal injury and oxidative injury to neurons cause cellular swelling, tissue hypoxia, and central nervous system dysfunction.
- Gastrointestinal: decreased mesenteric blood flow, decreased perfusion, bacterial translocation from GI tract, sloughing of basement membrane, and thermal damage to enterocytes.
- Hepatic: decreased splanchnic/portal blood flow, direct thermal injury to hepatocytes, hepatocellular ischemia and necrosis, cholestasis, decreased glycogenolysis, and gluconeogenesis.
- Renal: direct thermal injury to nephrons/renal tubular cells and glomerular basement membrane, hypoxic and oxidative damage, renal ischemia secondary to decreased perfusion/hypotension, myoglobinuria, and renal failure.
- Cardiovascular: direct thermal injury to cardiac myocytes, tissue ischemia, and dysrhythmias.

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- Respiratory: thermal injury to pulmonary endothelium, increased pulmonary vascular resistance, alveolar edema and hemorrhage, and ARDS in severe cases.
- Musculoskeletal: thermal damage to myocytes and rhabdomyolysis.
- Hemic/lymphatic/immune: hemoconcentration, increased nucleated red blood cells, thrombocytopenia, direct thermal injury to vascular endothelium, venous stasis, and exposure of tissue factor leads to Virchow's triad and consumption of platelets and clotting factors, DIC.
- Acid–base: respiratory alkalosis, metabolic acidosis, and lactic acidosis.

Geographic Distribution

- Can occur in any warm-weather climate, although also can occur when animals are locked in cars with the heater running on cold days.
- More common in warm and humid environments.



SIGNALMENT/HISTORY

Species

- Primarily dogs, but can occur in cats.

Breed Predilection

- Brachycephalic breeds (i.e., English bulldog, pug, Shar Pei, Pekingese, bull terrier), Belgian Malinois, breeds predisposed to laryngeal paralysis or tracheal collapse (golden and Labrador retrievers; Yorkshire terriers), boxer, Rottweiler, St Bernard, Weimaraner, Australian shepherds.

Mean Age and Range

- All ages, but often very young and older animals.
- Older dogs with preexisting disease.
- Younger dogs with overenthusiastic energy levels.

Historical Findings

- Identifiable underlying cause (i.e., hot day, locked in automobile or other confined area without adequate ventilation, grooming accident associated with heaters in drying cages, excessive exercise, limited access to water or shade).
- Predisposing underlying diseases (laryngeal paralysis, brachycephalic airway syndrome, cardiovascular disease, tracheal collapse, neuromuscular disease, upper airway mass lesion or obstruction, previous history of heat-induced illness).
- General anesthesia, faulty anesthesia equipment.

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Physical Examination Findings

- Panting
- Tacky or dry mucous membranes
- Hyperemic mucous membranes
- Markedly rapid or very delayed capillary refill time
- Neurologic abnormalities: altered level of consciousness, ataxia, cortical blindness, involuntary paddling, tremors, seizures, obtundation, coma
- Petechial hemorrhages
- Tachycardia
- Weak femoral pulses
- Pulse deficits
- Cardiac dysrhythmias
- Bloody diarrhea or melena, sometimes with GI mucosal sloughing
- Dark urine

Risk Factors/Causes

- Excessive environmental temperatures or humidity; note, it can occur in lower temperatures and in as little as 30 minutes in animals not acclimated to exercise.
- Unventilated areas (e.g., hot room, locked in an automobile, or a dryer cage at groomer's).
- Exercise.
- Toxicosis (strychnine, metaldehyde, mycotoxin, mushroom) that leads to seizures.
- Anesthesia (malignant hyperthermia).
- Previous history of heat-related illness.
- Age extremes.
- Obesity.
- Poor acclimatization to heat.
- Hyperthyroidism.
- Cardiopulmonary disease.
- Brachycephalic airway disease.
- Tracheal collapse.
- Upper airway obstruction.
- Thick haircoat.
- Dehydration.
- History of seizures.



DIAGNOSTICS

- Differentiating causes.

Complete Blood Count/Chemistry/Urinalysis

- May be helpful in identifying an underlying disease process.
- May be beneficial in identifying sequelae in hyperthermia.

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- CBC abnormalities may include a stress leukogram, leukopenia or transient leukocytosis, hemoconcentration or possibly anemia, nucleated red blood cells, thrombocytopenia.
- Biochemical profile may show azotemia, hyperbilirubinuria, elevated hepatocellular and cholestatic enzymes (i.e., ALT, AST, CK, alkaline phosphatase), hypernatremia, hyperchloremia, hyperglycemia or hypoglycemia, hyperphosphatemia, hyper- or hypokalemia, hyperbilirubinemia, or hypoproteinemia/hypoalbuminemia.
- Urinalysis may include hyposthenuria, proteinuria, cylindruria, renal tubular casts, hemoglobinuria, myoglobinuria, glucosuria; evaluate USG for renal concentrating ability prior to administration of intravenous fluids, when possible.

Other Laboratory Tests

- Blood gas analysis may show mixed acid–base disorder, respiratory alkalosis, or metabolic acidosis.
- Coagulation profiles may indicate prolonged PT, aPTT; FDPs or D-dimers may be positive. DIC may be present if PT and PTT are prolonged, and if FDPs or D-dimers are positive. Antithrombin levels may be decreased. Platelet count is usually decreased, or trends toward thrombocytopenia.
- Electrolytes: hyperkalemia (mild), hypophosphatemia, hypocalcemia.
- Lactate: elevated.

Diagnostic Procedures

- Minimum database: PCV/TS, peripheral blood smear evaluation, platelet estimate, blood glucose, BUN
- CBC
- Chemistry
- Urinalysis
- Lactate
- aPTT and PT
- ± D-dimers

Imaging

- Thoracic radiographs may help identify underlying cardiopulmonary disease or predisposing causes of heat stroke.
- Abdominal radiographs and/or ultrasound may help identify underlying disease process. May observe fluid-filled loops of bowel.
- CT or MRI may help identify hypothalamic or other mass lesion.



THERAPEUTICS

- The key to treatment is early recognition and immediate correction of hyperthermia.
- ABCs of emergency – airway, breathing, and circulation: establish an airway if airway obstruction, clear mucus or vomitus from oropharynx and trachea, if necessary; intubate

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if airway obstruction; need to perform emergency tracheostomy or tracheal oxygen in most severe cases of airway obstruction; if heat stroke has occurred secondary to seizures or airway obstruction, monitor patient carefully for development of noncardiogenic pulmonary edema.

- Institute cooling measures: place towels soaked in room-temperature water over patient and place a fan over the patient (Fig. 38.2). This will allow convective and radiant cooling measures. Do *not* immerse in cold or ice water or ice packs because this can promote peripheral vasoconstriction. Peripheral vasoconstriction is one of the primary means of heat dissipation. By removing the compensatory mechanism, core body temperature can continue to rise and cause worsening of clinical signs. As a general rule, patients should be cooled to a temperature of 39.4 °C (103 °F), then external cooling measures should be stopped, to avoid overcooling.
- Continuous temperature monitoring; hospitalization is required until temperature has normalized and is stable even in the least critical patients.
- Intensive care monitoring is common and necessary in most cases.
- Treat complications such as DIC, noncardiogenic pulmonary edema, ARDS, cerebral edema, seizures, renal failure, and MODS.
- Treat underlying disease and/or correct predisposing factors.
- Isotonic crystalloid fluid therapy based on degree of dehydration and fluid loss (GI tract).
- Synthetic colloids (i.e., hydroxyethyl starch) in incremental boluses (5 mL/kg) or 20–30 mL/kg per day may be required to aid in the treatment of hypotension or hypoproteinemia.
- Provide supplemental oxygen by nasal or nasopharyngeal oxygen catheter, tracheal oxygen, oxygen cage, or oxygen mask (50–150 mL/kg per minute).



Fig. 38.2. Dog with laryngeal paralysis that succumbed to heat stroke while playing ball on a very hot summer day. Note the cool towels placed over the dog to allow convective cooling to occur.

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- Mechanical ventilation may be required, depending on the degree of cerebral edema, hypoventilation, pulmonary disease, or airway obstruction.
- Antibiotics.
- Treat hypoglycemia if present.
- Gastroprotectants.
- Correction of acid–base and electrolyte abnormalities.
- Treat dysrhythmias.
- Treat hypotension.

Drugs of Choice

- Antibiotics: broad-spectrum antibiotics to cover both gram-negative and gram-positive infections; choices include ampicillin (22 mg/kg IV every 6–8 hours) with enrofloxacin (10 mg/kg IV every 24 hours in dogs, 5 mg/kg IV every 24 hours in cats), cefazolin (22 mg/kg IV every 8 hours) with enrofloxacin, Unasyn (22–30 mg/kg IV every 8 hours).
- Gastroprotectants.
- Antiemetics.
  - Dolasetron (0.6 mg/kg IV q24h)
  - Ondansetron (1 mg/kg IV q8–12h)
  - Maropitant (1 mg/kg IV q24h, 2 mg/kg PO q24h)
  - Metoclopramide (1–2 mg/kg per day IV CRI)
- H_2 blockers.
  - Famotidine 0.5–1 mg/kg IV q12h
  - Ranitidine 0.2–2 mg/kg IV or PO q8–12h
  - Cimetidine 5 mg/kg IV q6–8h
- Proton pump inhibitors.
  - Omeprazole 0.5–1 mg/kg PO q12h
  - Pantoprazole 1 mg/kg IV q24h
- Sucralfate (0.25–1 g PO three times a day)
- Antiarrhythmic drugs.
  - Lidocaine (2 mg/kg IV bolus, then 50–100 µg/kg per minute IV CRI)
  - Procainamide 6–10 mg/kg IV loading dose over 1–2 minutes, then 20–40 µg/kg min IV CRI in 5% destrose in water; or 10–22 mg/kg PO q8h
- Dextrose (0.5 g/kg 25% dextrose IV, followed by 2.5–5% dextrose CRI).
- Seizures.
  - Diazepam (0.5–1 mg/kg IV)
  - Phenobarbital loading (16–20 mg/kg IV); dose may be divided into four or five smaller doses and given every 30 minutes, provided that patient is rousable in between doses
  - Levetiracetam (mg/kg IV)
  - Propofol (4–7 mg/kg IV to effect, followed by 0.1–0.6 mg/kg per minute IV CRI)

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- Thrombocytopenia.
  - Platelet concentrate available from commercial blood banks

Contraindications

Glucocorticoids

- Although glucocorticoids reportedly can stabilize cellular membranes, there has been no documented benefit proven for their use in hyperthermic patients, and they are not recommended due to the risk of further decreasing mesenteric and renal perfusion.

Nonsteroidal Antiinflammatory Drugs

- NSAIDs of any form are contraindicated in heat stroke due to the risk of decreasing mesenteric and renal blood flow, and the risk of increasing GI ulceration. Dipyrone and flunixin meglumine are specifically contraindicated. Patients with exertional and nonexertional heat stroke are hyperthermic because of decreased ability to adequately dissipate heat. The mechanism of elevated body temperature is much different from that of pyrogenic hyperthermia, in which some NSAIDs may be indicated, on a case-by-case basis.

Overcooling

- Overcooling should be avoided due to the risk of overshooting and causing iatrogenic hypothermia. Hypothermia can induce shivering, which will increase the metabolic demands of the body, and can cause temperature to further increase.

Surgical Considerations

- Emergency tracheostomy may be necessary in cases of airway obstruction.



COMMENTS

Client Education

- Be aware of clinical signs.
- Educate client how to appropriately cool overheated animals prior to transport.
- An animal that has had prior episodes of heat stroke may be predisposed to additional episodes of heat stroke, even at lower environmental temperatures.

Patient Monitoring

- Monitor patients very closely when attempting to cool them and for a minimum of 24 hours afterwards because hypothermia can occur.
- Monitor the patient closely for signs of adverse sequelae, including DIC, renal failure, respiratory distress (ARDS, noncardiogenic pulmonary edema, aspiration pneumonia), and other organ failure.

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- Monitor closely for renal failure secondary to rhabdomyolysis and myoglobinuria.
- Monitor parameters such as heart rate, respiratory rate and effort, blood pressure, pulse oximetry and/or arterial blood gas, body temperature, ECG, coagulation status (platelet count, PT, aPTT), BUN, creatinine, electrolytes, lactate, glucose, urinalysis to check for renal tubular casts, urine output, PCV, and total protein.

Prevention/Avoidance

- Educate clients to not confine animals to areas where they cannot escape heat (e.g., cars, tied in yard without water or shelter/shade).
- Exercise animals during the coolest parts of the day.
- Acclimate the animal to short bursts of exercise or work before a longer training or working period.
- Rest animals that are working or exercising every 30 minutes and provide access to shade and water.
- Animals that are obese, have brachycephalic airway disease, laryngeal disorders, or any other predisposing factor should avoid exercise and remain in cool environments with fans or air conditioning.

Possible Complications

- Cardiac dysrhythmias
- MODS
- DIC
- Cerebral edema
- Coma
- Seizures
- Noncardiogenic pulmonary edema
- ARDS
- Acute renal failure
- SIRS
- Rhabdomyolysis
- Hepatocellular damage
- Bacterial translocation
- Sepsis
- GI ulceration
- Respiratory arrest
- Cardiac arrest
- Death

Expected Course and Prognosis

- Favorable if survive past 24 hours with no neurologic signs at time of presentation.
- Prognosis less favorable if neurologic signs (i.e., coma or progressive decline in neurologic status), hypothermic at time of initial presentation, persistent hypoglycemia, progressive

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worsening of azotemia, development of DIC or refractory hypotension, elevated total bilirubin, hypoproteinemia, respiratory difficulty or pulmonary edema, oliguria or anuria.
- Affected animals may have predisposition to other episodes of hyperthermia due to change in the sensitivity of the hypothalamic thermoregulatory set point.
- Prognosis more guarded in animals that present with hypothermia after cooling or neurologic signs (i.e., seizure, coma).

Synonyms

- Heat exhaustion, heat prostration, heat stroke, heat-induced illness

Abbreviations

ALT: alanine transaminase
aPTT: activated partial thromboplastin time
ARDS: acute respiratory distress syndrome
AST: aspartate aminotransferase
BUN: blood urea nitrogen
CBC: complete blood count
CK: creatine kinase
CRI: constant rate infusion
CT: computed tomography
DIC: disseminated intravascular coagulation
ECG: electrocardiogram
FDP: fibrin degradation products
GI: gastrointestinal
IV: intramuscular
MODS: multiple organ dysfunction syndrome
MRI: magnetic resonance imaging
NSAID: nonsteroidal antiinflammatory drug
PCV/TS: packed cell volume/total solids
PO: by mouth (per os)
PT: prothrombin time
SIRS: systemic inflammatory response syndrome
SQ: subcutaneous
USG: urine specific gravity

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chapter 39

Hemoabdomen

DEFINITION/OVERVIEW

- An accumulation of blood within the abdominal cavity.

ETIOLOGY/PATHOPHYSIOLOGY

- Blunt trauma such as automobile injury or high-rise fall.
- Penetrating trauma such as dog bite wounds, gunshot wounds, stab wounds, impalement by foreign object.
- Rupture of abdominal vessels secondary to gastric dilation and volvulus (GDV) or splenic torsion.
- Rupture of tumor associated with abdominal organs (i.e., liver, spleen, kidney, or adrenal).
- Coagulopathy, either congenital or acquired (i.e., anticoagulant rodenticide toxicity, hepatic disease, hemophilia).
- Postsurgical complication (i.e., slipped ovarian ligatures).

Incidence/Prevalence

- Unknown; more common in dogs than in cats.

Systems Affected

- Cardiovascular: hypotension due to blood loss, cardiac dysrhythmias may occur secondary to hypoxia.
- Hemic/lymphatic/immune: anemia due to blood loss, may not be apparent until fluid compartment shifts have occurred, often thrombocytopenia secondary to platelet consumption.

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SIGNALMENT/HISTORY

- Golden Retriever, German Shepherd, Labrador Retriever, and other large-breed dogs have a breed predilection for hemangiosarcoma.
- Middle-aged to older dogs in general have increased incidence of hemoabdomen associated with neoplasia.
- Intact free-roaming male dogs and outdoor cats have increased risk of traumatic hemoabdomen.
- Hemoabdomen due to neoplasia is rare in cats.
- Hemoabdomen in general can be seen in any age or breed.

Risk Factors/Causes

- Free-roaming dogs and cats have increased risk for blunt and penetrating trauma that result in hemoabdomen.
- Congenital or acquired coagulopathy.
- Exposure to anticoagulant rodenticides increases potential for acquired coagulopathy.
- Presence of abdominal mass, particularly spleen or liver associated, increases risk of hemoabdomen.

Historical Findings

- Weakness or ataxia
- Waxing/waning lethargy with recurrent bleeding episodes
- Acute collapse
- Abdominal distension
- Depression
- Anorexia
- Vomiting
- Possible history of trauma, history of coagulopathy, or history of exposure to anticoagulant rodenticides



CLINICAL FEATURES

- Lethargy
- Weakness
- Pale mucous membranes
- Abdominal distension with fluid wave
- Tachycardia with weak peripheral pulses
- Tachypnea
- Possibly abdominal mass

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- Possibly external wounds if hemoabdomen is traumatic
- Ecchymosis, tire marks, or abrasions from trauma
- Possibly bleeding from other sites, petechiae, or ecchymosis with coagulopathy or trauma



DIFFERENTIAL DIAGNOSIS

- Peritonitis: can be differentiated by the presence of large numbers of degenerate neutrophils and the presence of bacteria on cytologic examination of abdominal fluid.
- Hypovolemic shock: may coexist with hemoabdomen; hypovolemic shock due to other causes of fluid and blood loss may not have concurrent abdominal effusion.
- Uroabdomen: abdominal fluid is generally serosanguineous rather than hemorrhagic with uroabdomen. BUN equilibrates rapidly across the peritoneum, therefore creatinine and potassium ratios relative to serum should be used for comparison and not BUN ratios.
- Splenic torsion: hemoabdomen may occur secondary to splenic torsion but is not present in all cases. Best differentiated on basis of abdominal ultrasound.
- Immune-mediated hemolytic anemia: differentiated by the absence of abdominal fluid and by evidence of immune-mediated red blood cell destruction on complete blood count (red blood cell agglutination, spherocytosis).



DIAGNOSTICS

- Complete blood count: anemia but only after enough time has elapsed for fluid shifts to occur (several hours). With acute hemorrhage, patient may appear hypovolemic but not be anemic.
- Leukocytosis may be noted due to stress, neoplasia, or trauma. Thrombocytopenia may be noted due to platelet consumption. Primary thrombocytopenia may rarely cause hemoabdomen.
- Chemistry panel: hypoalbuminemia may occur secondary to blood loss.
- Coagulation panel: elevations in PT and/or PTT may indicate underlying congenital or acquired coagulopathy or DIC occurring secondary to abdominal hemorrhage. In one study 84% of dogs with nontraumatic hemoabdomen had a secondary coagulopathy.
- Abdominal radiographs: generally not very rewarding as the presence of abdominal fluid causes a generalized loss of detail although a mass effect may be seen with neoplasia.
- Abdominocentesis.
    - Can be "blind" four quadrant or ultrasound guided. May reveal the presence of nonclotting blood (or other fluid) within the abdominal cavity; positive abdominocentesis indicates that there is >5 mL/kg of blood within the abdominal cavity.
    - Clotting of the blood indicates either rapid, ongoing hemorrhage or perforation of a vessel or organ during the abdominocentesis procedure.
    - PCV/TS of abdominal fluid: PCV of abdominal fluid will typically be greater than that of peripheral blood if active bleeding is occurring.

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- Diagnostic peritoneal lavage (DPL).
  - May be helpful if abdominocentesis is negative.
  - Instill 10–22 mL/kg of warm fluid into the abdomen using a large-bore, multiple fenestrated over-the-needle catheter, distribute fluid by rotating or walking the patient, then aspirate fluid back by routine abdominocentesis.
  - PCV >5% is indicative of hemorrhage; serial hematocrits can be performed on DPL fluid to assess for ongoing hemorrhage.
- Abdominal ultrasound.
  - Very sensitive (96%) in detecting abdominal fluid.
  - Useful for guiding abdominocentesis and evaluating for primary and metastatic abdominal masses.
- Thoracic radiographs.
  - Should be performed with any bleeding abdominal mass to evaluate for metastasis and should be performed with blunt trauma to evaluate for concurrent thoracic injury or diaphragmatic hernia.

Pathologic Findings

- Vary depending on the cause for hemoabdomen.
- Other traumatic injuries may be present with blunt or penetrating trauma.
- With neoplasia, mass of liver, spleen, kidney or adrenal gland may be noted and possibly evidence of metastatic disease.
- With coagulopathy, hemorrhage in other areas may be noted.

Histopathologic Findings

- Vary depending on underlying cause.
- With neoplasia, hemangiosarcoma of the spleen is the most common tumor causing hemoabdomen but benign splenic hematoma, hepatocellular carcinoma, carcinomatosis, and adrenal adenocarcinoma can also result in hemoabdomen.



THERAPEUTICS

- Treat hemorrhagic shock by administration of supplemental oxygen and intravenous fluids.
- Isotonic crystalloids (lactated Ringer's solution, Normosol-R, Plasmalyte-A) can be administered at shock dosages (up to 90 mL/kg in the dog and 40–55 mL/kg in the cat).
- Fluid administration can be titrated to reach a systolic blood pressure of approximately 80–100 mmHg (small volume resuscitation).
- Synthetic colloids such as Hetastarch can be administered in 5 mL/kg incremental doses as necessary to improve perfusion and blood pressure.
- Transfusion with packed red blood cells (11 mL/kg) should be utilized when a patient is considered to be clinical for their anemia (e.g., tachycardic, hypoxemic, etc.) or bleeding acutely enough to warrant transfusion anticipating future anemia-related issues.

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- If available, hemoglobin-based oxygen carriers (such as Oxyglobin 5–10 mL/kg) can also be utilized to treat hemorrhagic shock.
- Treat underlying primary or secondary coagulopathies.
  - Fresh frozen plasma (10–15 mL/kg) is used to restore clotting factors (factors II, VII, IX, X), albumin, and immunoglobulins, but does not contain factor VII or von Willebrand's factor.
  - Vitamin K_1 should be administered with hepatopathy or vitamin K antagonist rodenticide toxicity.
- Control hemorrhage.
  - External counterpressure can be applied by using towels secured with tape to wrap the hindlimbs, pelvis, and abdomen, starting at the hind feet. Avoid excessive pressure (bandage should be loose enough to fit your hand underneath) to avoid intestinal ischemia and monitor for respiratory compromise. Leave bandage in place 2–4 hours then gradually remove over 8–12 hours. This counterpressure is often not successful, but can provide some tamponade effect.
- Surgery on an emergency basis is indicated with bleeding abdominal masses and penetrating abdominal wounds.
  - Surgery is indicated with blunt trauma if the patient is not responding to fluid resuscitation, the abdominal PCV is rising, and there is progressive abdominal distension despite appropriate resuscitative measures.
  - Abdominal exploratory is rarely required in cases of traumatically induced hemoabdomen though would be recommended for unstable patients that cannot be stabilized and patients who experience prolonged and persistent hemorrhaging.
  - Surgery is contraindicated in patients with primary coagulopathy.
- Control pain from trauma or surgery using:
  - fentanyl IV CRI (2–5–µg/kg/h)
  - hydromorphone (0.05–0.2 mg/kg IV, IM or SQ q4–6h)
  - buprenorphine (0.01–0.02 mg/kg IV q4–6h)
  - morphine (0.05–0.2 mg/kg/h IV CRI or 0.25–2 mg/kg IM or SQ q4–6h, 0.2–0.5 mg/kg IV slowly to prevent histamine release q4h).
- Prevent infection in the case of penetrating abdominal wounds.
  - Clean and bandage the wounds.
  - Administer antibiotics (cefazolin 22 mg/kg IV q6–8h for gram-positive coverage, enrofloxacin (dogs 10–15 mg/kg, cats 5 mg/kg) IV q24h for gram-negative coverage).
  - With gastrointestinal visceral or bite wounds, the antibiotic spectrum should be expanded to include anaerobic organisms using cefoxitin (30 mg/kg IV q6–8h), ampicillin/sulbactam (30–50 mg/kg IV q8h), or a combination of ampicillin (22 mg/kg IV q6–8h) with enrofloxacin (dogs 10–15 mg/kg IV, cats 5 mg/kg IV q24h).

Precautions/Interactions

- Avoid aspirin and other nonsteroidal antiinflammatory medications that decrease platelet function and can affect the kidneys during a period of decreased renal perfusion during hypovolemia.

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- Hypertension should be avoided, as this can lead to disruption of clot formation and increased hemorrhage. Generally, resuscitation to low normal blood pressure (between 80 and 100 mmHg systolic) is considered optimal in order to maintain organ and tissue perfusion but avoid exacerbation of bleeding.

Activity

- Usually limited in order to allow recovery from abdominal surgery or to prevent further bleeding in cases of coagulopathy.

Surgical Considerations

- Emergency surgery is recommended for patients with bleeding abdominal masses and penetrating abdominal wounds.
- Surgery is indicated in blunt trauma patients who are not responding to fluid resuscitation or with rising abdominal PCV and progressive abdominal distension.
- These patients are often hemodynamically unstable and fluctuations in blood pressure and heart rate may complicate anesthesia.



COMMENTS

Client Education

- Client should be informed of cost and prognosis associated with treatment based on underlying causes for hemoabdomen.
- For spontaneous hemoabdomen, clients should be made aware that about 70% of cases are from malignant neoplasia and about 70% of those are from hemangiosarcoma. A discussion about hemangiosarcoma and its prognosis is required.

Patient Monitoring

- Assess blood pressure frequently (every 5 minutes) until patient is stable then gradually less often after the patient stabilizes.
- Electrocardiogram is recommended continuously for the first 24 hours in patients with abdominal or thoracic trauma or who have undergone splenectomy.
- PCV/TS should be monitored every 4–6 hours initially based on concern for ongoing hemorrhage.
- Coagulation panel is needed, particularly in patients with existing coagulopathy (either primary or secondary).

Prevention/Avoidance

- Abdominal masses cannot be prevented but surgical removal as soon as they are identified can prevent onset of bleeding.

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- Avoid allowing pets to roam free, which increases the potential for trauma and access to anticoagulant rodenticides.

Possible Complications

- Hemorrhage
- Cardiac dysrhythmias
- DIC
- Abdominal dehiscence
- Surgical site infection

Expected Course and Prognosis

- Varies depending on underlying cause of hemoabdomen and on presence of coexisting injuries in cases of trauma.
- Prognosis in general is guarded to good. In one study, splenic source of hemorrhage and surgical intervention for spontaneous hemoperitoneum were positive prognostic factors.
- Patients with hemoabdomen that are anemic and have a splenic mass have about a 70% likelihood of malignant neoplasia, with about 70% of those being hemangiosarcoma (i.e., about 50% likelihood of hemangiosarcoma).
    - With hemangiosarcoma, microscopic metastasis has typically occurred prior to the time of presentation.
    - Median survival times of 3–4 months with surgery and 9 months with adjunctive chemotherapy have been reported.
    - Common sites for metastasis include the liver and right atrium.

Synonyms

- Hemoperitoneum

Abbreviations

BUN: blood urea nitrogen
CRI: constant rate infusion
DIC: disseminated intravascular coagulation
DPL: diagnostic peritoneal lavage
IM: intramuscular
IV: intravenous
PCV/TS: packed cell volume/total solids
PT: prothrombin time
PTT: partial thromboplastin time
RBC: red blood cell
SQ: subcutaneous

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chapter 40

Hepatic Encephalopathy



DEFINITION/OVERVIEW

- Multifactorial neurologic syndrome that occurs when hepatic dysfunction (>70% function loss) causes systemic biochemical alterations that induce disruptions of cerebral metabolism.



ETIOLOGY/PATHOPHYSIOLOGY

- Clinical and biochemical abnormalities associated with this syndrome cannot be sufficiently explained by any single hepatic or cerebral metabolic defect.
- Most frequently reported cause in dogs is portosystemic vascular anomalies.
- Three types have been categorized.
  - Type A: associated with acute hepatic failure.
  - Type B: associated with portosystemic vascular anomaly without intrinsic hepatic failure.
  - Type C: associated with marked hepatic parenchymal disease and portal hypertension.
- Several theories have been proposed.
  - Hyperammonemia.
    - Ammonia-rich blood bypasses hepatic parenchyma in the presence of an extrahepatic portosystemic vascular anomaly.
    - Glutamate in astrocytes is converted to glutamine in the presence of ammonia; glutamine formation is increased, but secretion is impaired in hyperammonemic states, thus inducing cellular swelling and neuronal edema.
    - Glutamine in mitochondria is converted by glutaminase into glutamate and ammonia, inducing reactive oxygen and nitrogen species formation.
  - Inflammation: proinflammatory cytokines alter astrocyte glutamate uptake, expression of gamma-aminobutyric acid (GABA) receptors, and blood–brain permeability.
  - Glutamate transmission defect: hyperammonemia reduces glutamate uptake by astrocytes secondary to downregulation of glutamate transporter GLT-1.
  - GABA agonist elevations: liver dysfunction is associated with reduced GABA clearance, and type A hepatic encephalopathy is associated with a hyperpermeable

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blood–brain barrier. Subsequently, cerebral GABA levels increase to inhibit neurotransmission through hyperpolarization of neuronal membranes.

- Benzodiazepine-like substances elevations.
  - Benzodiazepine-like substances may come from vegetables or certain medications and may be produced by the intestinal microbiota.
  - The number of astrocyte-associated benzodiazepine receptors is increased in hepatic encephalopathy; these receptors are involved with the formation of tetrahydroprogesterone and tetrahydrodeoxycorticosterone (both potent GABA receptor agonists).
- Serotonergic system defect: elevated levels of serotonin, its receptor, and monoamine oxidases have been documented in patients with hepatic encephalopathy.
- Amino acid metabolism alteration: plasma concentrations of aromatic amino acids (AAA) increase due to reduced hepatic extraction, and elevated AAA levels overwhelm normal AAA metabolic pathways; use of alternative metabolic pathways results in the formation of octopamine and phenylethanolamine, both false neurotransmitters that block normal catecholamine activation.
- Manganese toxicity: elevated cerebral manganese levels have been associated with altered cerebral metabolism, reduced glutamate uptake by astrocytes, and modified glutamatergic and dopaminergic neurotransmission.
- Neurotransmitter alterations.
  - Taurine levels have been directly correlated with the severity of hepatic encephalopathy.
  - Intestinal microbiota produce multiple neurotransmitters that have been implicated in hepatic encephalopathy.

Systems Affected

- Musculoskeletal: muscle weakness, tremors.
- Nervous: depression, ataxia, obtundation, head pressing, altered conscious proprioception, circling.
- Gastrointestinal: weight loss, anorexia, vomiting, diarrhea, ptyalism.
- Renal/urologic: polyuria/polydipsia.
- Cardiovascular: bradycardia, hypertension.
- Respiratory: irregular respiratory patterns.
- Ophthalmic: cortical blindness, pupillary abnormalities.



SIGNALMENT/HISTORY

- No breed, sex or age predilection for hepatic encephalopathy; however there are predilections for specific diseases associated with this syndrome.
- Patients with congenital portosystemic vascular anomalies typically show clinical signs within 12 months of life, but these anomalies should still be considered in older patients.

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- Extrahepatic portosystemic vascular anomalies commonly occur in small/toy breed dogs and cats.
- Intrahepatic portosystemic vascular anomalies are typically documented in large breed dogs.
- Patients with acquired hepatic diseases commonly manifest clinical signs when greater than 1 year of age.

Risk Factors

- Excessive protein ingestion.
- Medical conditions.
  - Gastrointestinal hemorrhage
  - Constipation
  - Electrolyte derangements
  - Hypoglycemia
  - Alkalemia
  - Dehydration
  - Kidney disease
  - Infection
- Liver-metabolized drugs.

Historical Findings

- Subtle signs include mild irritability, hyporexia, and marginal confusion.
- Patients with extrahepatic portosystemic vascular anomalies are often the runts of their litters, have retained deciduous teeth and/or open fontantelles; a higher prevalence of cryptorchidism is reported in affected males.
- Polyuria/polydipsia, inappropriate urination, and/or difficulty house training may be reported.



CLINICAL FEATURES

- Clinical signs are divided into two groups: signs associated with hepatic failure and signs referable to the CNS.
  - Hepatic failure signs are nonspecific: anorexia/hyporexia, nausea, diarrhea, depression, weight loss, lethargy, and/or polyuria/polydipsia.
  - CNS signs: behavioral changes, aggression, compulsive behaviors, head pressing, tremors, vocalization, blindness, deafness, stupor, seizures and coma. CNS signs are divided into four stages; patients commonly fluctuate episodically between stages.
    - ☐ Stage I: confusion, hyporexia, dull mentation or irritability, but may appear normal.
    - ☐ Stage II: dull mentation, cortical blindness, ptyalism, lethargy, ataxia, personality changes.
    - ☐ Stage III: stupor, confusion, marked ptyalism (cats), seizure activity, copper-colored irises (cats), occasional aggression.
    - ☐ Stage IV: recumbency, coma that may rapidly lead to death.

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DIFFERENTIAL DIAGNOSIS

- Intracranial.
  - Brain neoplasia
  - Seizure disorder
  - Encephalitis (bacterial, viral, fungal, protozoal)
  - Meningitis (bacterial, viral, fungal, protozoal)
  - Hemorrhage
  - Ischemic events
  - Congenital malformations
- Extracranial.
  - Intoxications (e.g., lead)
  - Uremia
  - Sepsis
  - Hypoglycemia
  - Ketoacidosis
  - Hypercapnia
  - Hypothiaminemia (cats)
  - Hypocobalaminemia (dogs)
  - Thyroid dysfunction

DIAGNOSTICS

- Complete blood count: macrocytosis, anemia, and changes in red blood cell morphology (acanthocytosis, poikilocytosis, target cells).
- Serum biochemical profile.
  - Hepatocellular and cholestatic liver enzyme elevations.
  - Indirect evidence of liver dysfunction includes hypoalbuminemia, hypoglycemia, decreased blood urea nitrogen, hypocholesterolemia, and hyperbilirubinemia.
- Urinalysis.
  - Minimal urine specific gravity (1.015–1.030) or isosthenuria (1.008–1.015).
  - Ammonium urate crystalluria .
  - Bilirubinuria; proteinuria, hematuria, and pyuria also occasionally reported.
- Serum bile acids: pre- and/or postprandial levels elevated.
- Blood ammonia level: increased level in some patients with hepatic encephalopathy.
- Coagulation testing: prolonged prothrombin (PT) and activated partial thromboplastin (aPTT) time, as well as changes consistent with a hypocoagulable state via thromboelastrography.
- Diagnostic imaging.
  - Microhepatica via radiography in patients with extrahepatic portosystemic vascular anomalies.

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- Sonography may identify vascular anomalies, hepatomegaly, hepatobiliary neoplasia, biliary mineralization, peritoneal effusions, hepatic parenchymal changes, splenomegaly, and intraperitoneal lymphadenomegaly.
- Advanced brain imaging can help confirm hepatic encephalopathy and rule out other diseases.

- Blood manganese level: increased in some patients with hepatic encephalopathy.

Pathologic Findings

- Liver sampling to confirm primary or secondary hepatic inflammatory, infectious and/or neoplastic processes.



THERAPEUTICS

- The three goals of the management are to:
  - reduce the incidence of predisposing factors
  - alleviate neurologic signs
  - determine the primary underlying hepatic disease.

Drugs of Choice

- Acute.
  - Reduce cerebral edema: mannitol (0.5–1.5 g/kg IV over 10–20 min). Induces osmotic fluid shift, reduces cerebrospinal fluid production, decreases blood viscosity, and serves as a free radical scavenger.
  - Anticonvulsants: levetiracetam (20 mg/kg IV q8h).
- Chronic.
  - Nonabsorbable disaccharides: lactulose (1–3 mL/kg PO q6–8h; dose adjusted so patient produces 2–3 soft bowel movements per day).
    - May be administered per rectum in those with incomplete swallowing reflex (5–10 mL/kg cleansing warm water enema followed by 5–10 mL lactulose diluted 1:3 q8h).
    - Bacteria produce various acids (acetic, butyric, propionic, and lactic) to form an acidic environment that promotes conversion of ammonia to ammonium, the latter being subsequently eliminated in feces.
    - Induces osmotic diarrhea that increases colonic motility and reduces ammonia-producing microbiota.
  - Antibiotics: neomycin (20 mg/kg PO q6–12h), metronidazole (7.5 mg/kg PO q12h), Clavamox (13.75 mg/kg PO q12h). Decrease bacterial deamination and reduce the production of aromatic amino acids, short chain fatty acids, mercaptans, and false neurotransmitters.

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Precautions/Interactions

- Mannitol contraindicated with hypernatremia, chronic hyponatremia, and hyperosmolality (>320 mOsm/L).
- Benzodiazepine antagonists not recommended for management of chronic hepatic encephalopathy.
- Repeat dosing of sedatives that undergo hepatic metabolism augments degree and/or duration of sedation.
- Avoid diazepam.
- Lactulose may cause diarrhea, abdominal cramping, hypernatremia, and abdominal distension.
- Neomycin should never be administered parenterally.
- Chronic high-dose metronidazole therapy has been associated with cerebellar and/or vestibular ataxia and nystagmus.

Alternative Drugs

- Acute.
  - Benzodiazepine antagonists: flumazenil (0.02 mg/kg IV) or sarmazenil (3 mg/kg IV). These drugs may be beneficial in a subset of patients with acute hepatic encephalopathy (i.e., comatose patients).
  - Anticonvulsants: sodium bromide (600–1200 mg/kg IV over 8 h, diluted in a 3% solution with 1 L sterile water) if levetiracetam is not available.
- Chronic.
  - Lactitol (0.5–0.75 g/kg PO q12h) if lactulose is not available.
  - Probiotics: Fortiflora (Purina Veterinary Diets), ProstoraMax (Iams Veterinary Formula), Proviable-DC (Nutramax Laboratories). Modify pathogenic bacterial substrates and decrease plasma ammonia levels.
  - Zinc (1–3 mg elemental zinc/kg PO q24h).
    - Deficiency impairs urea cycle enzymes and subsequently nitrogen detoxification.
    - Target level is 200–500 µg/dL.
    - Supplementation may be associated with vomiting and hemolytic anemia (with toxic doses).
  - L-Dopa (dogs: 6.8 mg/kg PO once; then 1.4 mg/kg PO q6h). Evidence suggests beneficial effect is due to increased renal ammonia excretion.

Appropriate Healthcare

- Avoid excessive protein intake. Large quantities stimulate microbiota to release ammonia and neuroinhibitor-like substances that are delivered to the CNS.
- Identify and treat any infection.
  - Increased protein metabolism to yield ammonia precursors and aromatic amino acids.
  - Proinflammatory cytokines exacerbate CNS derangements by promoting ammonia diffusion into the CNS and increasing the expression of benzodiazepine binding sites.

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- Reduce the use of drugs that undergo hepatic metabolism.
- Identify and correct electrolyte and acid–base derangements.
  - Hyponatremia causes neuronal myoinositol depletion that contributes to cerebral edema.
  - Metabolic alkalosis increases concentration of nonionized ammonia that passes freely into neurons.
  - Hypokalemia promotes intracellular acidosis and extracellular alkalosis that allows free movement of ammonia into cells
- Provide coagulation support as indicated: fresh frozen plasma (10–20 mL/kg IV over 4 h) and vitamin K1 (1.0 mg/kg SQ q24h).
- Consider gastroprotection: omeprazole (0.5–1.0 mg/kg PO q24h), pantoprazole (1.0 mg/kg IV q24h), famotidine (0.5 mg/kg PO q12h), raniditine (1–2 mg/kg PO q8h), sucralfate (0.25–1 g total dose q6–8h). Prevent bacterial translocation and decrease gastrointestinal hemorrhage.

Nursing Care

- Use strict aseptic technique when managing these patients.
- Ensure adequate unsoiled bedding.
- Change body position of recumbent patients frequently (q4h).
- Provide sterile ophthalmic lubricant for patients incapable of adequately blinking.
- Ensure in-hospital visibility due to potential for seizure activity.
- Serially monitor heart rate, pulse rate, respiratory rate, and respiratory effort.
- Monitor for and prevent constipation.
- Ensure perineum, tail, and medial thigh region remains clean and dry due to the potential for urine and/or fecal-induced moist dermatitis.
- Avoid jugular venipuncture and compression with suspected/confirmed increased intracranial pressure.
- Elevate cranial half of body 15–30° above the caudal half in patients with suspected/confirmed intracranial hypertension.

Diet

- Feed patients as much protein as they can tolerate; minimum intake dog 2.1 g/day; cat 4.0 g/day.
- Feeding small frequent meals may decrease catabolism.
- Methods to increase protein tolerance: oral antimicrobial therapy, lactulose, soluble fiber supplementation, ingestion of nonmeat protein sources.
- Protein restriction only recommended if efforts to increase tolerance fail.
- Once neurologic clinical signs resolve, dietary (dairy or vegetable) protein may be increased 0.3–0.5 g/kg q7 days.
- Manganese content should be minimized.

Activity

- Patients should generally be allowed to set their own pace.

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Surgical Considerations

- No safe sedatives for patients due to impaired hepatic clearance.
- Repeat dosing of sedatives may result in increased degree and/or duration of sedation.
- Surgical correction of single extrahepatic portosystemic vascular anomaly should be considered once clinical signs are adequately controlled and patient is no longer living in a catabolic state.



COMMENTS

Client Education

- Encourage families to maintain a detailed journal of their pet's appetite, medication schedule/administration, elimination habits, and potential adverse events (e.g., seizures).
- Review protocol for management of a seizure patient at home (safe transport to hospital, administration of anticonvulsant medication per rectum, etc.).
- Provide appropriate guidance on nutritional support.

Patient Monitoring

- Musculoskeletal: observe for resolution of muscle weakness, tremors.
- Neurologic: perform neurologic examination (q1–24h; variable per patient) to ensure appropriate resolution of depression, ataxia, obtundation, head pressing, altered conscious proprioception, circling.
- Gastrointestinal: serially weigh patients (q6–12h); maintain adequate nutrition records for hospitalized patients; maintain euglycemia to help reduce muscle catabolism induced by hypoglycemia, but avoid iatrogenic hyperglycemia.
- Cardiovascular: serially monitor (q6–12h) heart rhythm and blood pressure for resolution of bradycardia and hypertension, respectively.
- Renal: evaluate creatinine and urine specific gravity (q6–24h) given ~40% of human patients with acute hepatic failure develop acute kidney injury (hepatorenal syndrome).
- Respiratory: serially evaluate (q6–8h) respiratory pattern and effort to ensure patient is eupneic.
- Ophthalmic: periodically assess (q4–12h) vision and oculocephalic reflexes.

Prevention/Avoidance

- Avoid use of sedatives, anesthetics, and/or analgesics unless required to control neurologic signs.
- Avoid feeding red meat, fish, and/or eggs due to potential for enhancing heme metabolism (ammoniagenic potential).
- Avoid administration of medications with significant hepatic metabolism.

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Possible Complications

- Management of intracranial hypertension secondary to acute hepatic injury is challenging.

Expected Course and Prognosis

- Guarded with acute hepatic failure: common causes of death are cerebral edema, hemorrhage and/or sepsis.
- Guarded to fair when associated with chronic liver disease.
- If clinical signs do not resolve within 72 hours, investigation for a persistent precipitating factor is recommended.
- Reported negative prognostic factors are hyperbilirubinemia, prothrombin time >100 seconds, idiosyncratic drug reaction, viral hepatopathy, and/or extreme ages.

Synonyms

- Hepatic coma

Abbreviations

AAA: aromatic amino acids
aPTT: activated partial thromboplastin time
CNS: central nervous system
GABA: gamma-aminobutyric acid
IV: intravenous
PO: by mouth (per os)
PT: prothrombin time
SQ: subcutaneous

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chapter 41

Hyperglycemia

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DEFINITION/OVERVIEW

- Abnormally high blood glucose concentration.

ETIOLOGY/PATHOPHYSIOLOGY

- Insulin deficiency
- Increased hepatic gluconeogenesis
- Decreased consumption of glucose in peripheral tissues
- Increased glycogenolysis
- Excess of glucocounterregulatory hormones such as cortisol, ACTH, growth hormone, epinephrine, and glucagon

Systems Affected

- Metabolic: regulation of excess carbohydrate metabolism.
- Renal: polyuria secondary to hyperglycemic osmotic diuresis.
- Nervous: severe hyperglycemia leads to hyperosmolality that may cause CNS dysfunction.
- Ophthalmic: cataracts in dogs secondary to persistent hyperglycemia.

SIGNALMENT/HISTORY

- Variable depending on underlying cause.

Risk Factors/Causes

- Low glucose use, insulin deficiency, or insulin antagonism.
  - Diabetes mellitus
  - Pancreatitis
  - Acromegaly (cat)
  - Diestrus (bitch)

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- Increased glucose production.
  - Hyperadrenocorticism
  - Pheochromocytoma
  - Glucagonoma
  - Exocrine pancreatic neoplasia
- Insulin administration problems in confirmed diabetics.
  - Insulin overdosage with Somogyi phenomenon
  - Insulin resistance
  - Poor absorption of insulin
- Physiologic.
  - Stress hyperglycemia (especially in cats)
  - Postprandial (diets containing monosaccharides or disaccharides)
- Drugs.
  - Glucocorticoids
  - Progestagens
  - Megestrol acetate
  - Thiazide diuretics
- Iatrogenic.
  - Excessive dextrose-containing fluids
  - Parenteral nutrition
- Other.
  - Renal insufficiency: decreased renal clearance of glucose in diabetic animals can lead to severe hyperglycemia. Also, decreased tissue utilization can occur.
  - Head trauma.
  - Concurrent disease: hyperadrenocorticism, pancreatitis.
  - Obesity.
  - Diabetogenic drugs.
  - Dextrose-containing fluids.



CLINICAL FEATURES

- Clinical features are variable due to underlying cause. Many animals are asymptomatic.
  - Polyuria
  - Polydipsia
  - Polyphagia
  - Weight loss
  - CNS depression can be seen with severe hyperglycemia



DIFFERENTIAL DIAGNOSIS

- Mild, transient high blood glucose; rule out stress and postprandial fluctuation.
  - Recheck after 12-hour fast.
  - Recheck after minimizing stress; acclimate to hospital environment or consider home blood glucose monitoring.

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Laboratory Findings

- Drugs that may alter laboratory results.
  - High blood glucose concentrations: glucocorticoids, ACTH, dextrose-containing fluids, epinephrine, asparaginase, beta-adrenergic agonists, and diazoxide.
- Disorders that may alter laboratory results.
  - Lipemia, hemolysis, or icterus may interfere with spectrophotometric assays.
  - Delayed serum separation may falsely lower blood glucose results due to cellular glucose use; separate within 1 hour of collection.
  - Refrigerate or freeze serum samples not analyzed within 12 hours.
  - Blood glucose reagent strips used in home monitoring require whole-blood samples.
- Valid if run in human laboratory.



DIAGNOSTICS

Complete Blood Count/Biochemistry/Urinalysis

- CBC is usually normal in the uncomplicated diabetic patient.
- Biochemistry panel.
  - Hyperglycemia
  - Hypercholesterolemia
  - Lipemia may be secondary to low lipoprotein lipase, hyperadrenocorticism, pancreatitis, or postprandial blood sampling
  - Increased ALT (usually <500 IU/L) due to hepatic lipidosis
  - Increased ALP (usually <500 IU/L) due to hepatic lipidosis
  - Amylase and lipase have low specificity for concurrent pancreatitis
- Urinalysis.
- USG usually >1.025.
- Glucosuria in diabetes mellitus.
- Variable ketonuria.
- Bacteriuria and pyuria possible if concurrent urinary tract infection

Other Laboratory Tests

- Serum fructosamine concentration: marker of mean blood glucose concentration over the past 1–3 weeks. A normal fructosamine concentration in a hyperglycemic patient supports transient or stress-related hyperglycemia. Also used to monitor glycemic control in the diabetic patient. May be falsely lowered by hypoalbuminemia, hyperlipidemia, and azotemia.
- Blood glycosylated hemoglobin concentration: marker of mean blood glucose concentration over the past 3–4 months.
- Serum insulin concentration: rarely analyzed. Hyperglycemia should be accompanied by hyperinsulinemia in a normal patient. Hypoinsulinemia in the presence of hyperglycemia suggests diabetes mellitus.

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- ACTH stimulation test or low-dose dexamethasone suppression test to rule out concurrent hyperadrenocorticism.
- PLI: species-specific test. Positive result suggestive of concurrent pancreatitis.
- Intravenous glucose tolerance test, intravenous glucagon tolerance test: rarely indicated.

Imaging

- Abdominal ultrasound may be useful in identifying underlying causes (i.e., hyperadrenocorticism, pancreatitis).



THERAPEUTICS

- Insulin therapy for confirmed diabetic patients.
- Avoid rapid decrease in blood glucose levels.
- Discontinue diabetogenic drugs.
- Dextrose-free fluids.
- Dietary therapy.
    - Cats: feed low-carbohydrate, high-protein canned foods.
    - Dogs: feed high-fiber, low-fat diet.

Drugs of Choice

- Insulin.
    - Regular (crystalline) insulin has a rapid onset of effect but short duration of action. Most useful in the hospital setting in the anorexic or ketotic patient.
    - Intermediate and long-acting insulins (i.e., NPH, PZI, glargine, vetsulin) are used in the long-term management of diabetics.

Contraindications

- Diabetogenic drugs
- Dextrose-containing fluids

Precautions/Interactions

- Avoid rapid lowering of blood glucose levels.
- Avoid hypoglycemia.

Alternative Drugs

- Oral hypoglycemic agents (sulfonylureas such as glipizide) are more useful in type II (noninsulin-dependent) diabetic cats. However, response to these medications is poor and adverse side effects are common.

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COMMENTS

Patient Monitoring

- Recheck blood glucose after minimizing stress, discontinuing diabetogenic drugs to confirm diagnosis.
- Glucose curves measuring blood glucose concentration every 2 hours after insulin administration can be performed in the hospital or at home by the owner using a blood glucose monitoring system.
- Serum fructosamine concentration and glycosylated hemoglobin monitor long-term glucose control.
- Monitor for return of clinical signs: weight loss, polyuria, polyphagia, and polydipsia.

Possible Complications

- High incidence of infection (especially urinary tract infections and skin infections).
- Severe hyperglycemia may be associated with CNS depression and coma because of hyperosmolarity.
- Hypoglycemia may result as an effect of overaggressive therapy for hyperglycemia.

Associated Conditions

- Severe hyperglycemia is associated with hyperosmolarity.
- Diabetic ketoacidosis: hyperglycemia, acidosis and low total body concentrations of sodium, potassium, and phosphorus.

Pregnancy

- Pregnancy-related diabetes mellitus caused by high progesterone concentrations is reported in humans.

Synonyms

- High blood sugar

Abbreviations

ACTH: adrenocorticotropic hormone
ALP: alkaline phosphatase
ALT: alanine aminotransferase
CBC: complete blood count
CNS: central nervous system
NPH: neutral protamine hagedorn

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PLI: pancreatic lipase immunoreactivity
PZI: protamine zinc insulin
USG: urine specific gravity

Suggested Reading

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chapter 42

Hyperkalemia

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DEFINITION/OVERVIEW

- Hyperkalemia is a serum electrolyte disorder that occurs when serum potassium concentrations exceed 5.5 mEq/L. The increased serum potassium level leads to a depolarized state at the muscular cell membrane and causes skeletal and cardiac muscle dysfunction.

ETIOLOGY/PATHOPHYSIOLOGY

Systems Affected

Muscular

- Weakness.

Cardiovascular

- Excitation and conduction abnormalities, dysrhythmias.
- Translocation of potassium from intracellular to extracellular space caused by metabolic acidosis, rhabdomyolysis, massive tissue injury, drugs such as trimethoprim-sulfa and ACE inhibitors, insulin deficiency (i.e., diabetes mellitus/diabetic ketoacidosis), hypertonicity, and hyperkalemic periodic paralysis.
- Iatrogenic excess potassium administration in improperly mixed parenteral fluids after addition of potassium, failure to close the infusion port to the patient when potassium is added to the fluids, too rapid intravenous administration of potassium-containing fluids, too rapid injection of potassium penicillin.

Renal

- Urinary outflow obstruction or renal failure.

Drugs

- Potassium-sparing diuretics.

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Metabolic

- Aldosterone deficiency.
- Pseudohyperkalemia.
  - Escape of potassium in red blood cells to the serum or plasma. This latter condition has no effect on the patient.
  - Occurs in Akitas.



SIGNALMENT/HISTORY

- Can affect any age or breed.
- History of muscular weakness and signs due to underlying disease such as renal failure or hypoadrenocorticism.

Risk Factors/Causes

- Acute kidney injury
- Postrenal obstruction
- Metabolic acidosis
- Hypoadrenocorticism
- Errors in parenteral fluid supplementation
- Certain drugs

Historical Findings

- Progressive appetite depression
- Lethargy
- Often vomiting from an underlying problem such as uremia
- Skeletal muscle weakness



CLINICAL FEATURES

Dogs

- Muscles: weakness.
- Cardiac: conduction and rhythm disturbances including tall or deep T waves, shortened P waves, prolonged PR interval, bradycardia, atrial standstill, sine waves, asystole (Fig. 42.1).
- Other signs such as dehydration, weak pulses, prolonged capillary refill time, vomiting, sometimes diarrhea, mental depression; if due to acute kidney injury, urinary obstruction, acute hypoadrenocortical crisis.

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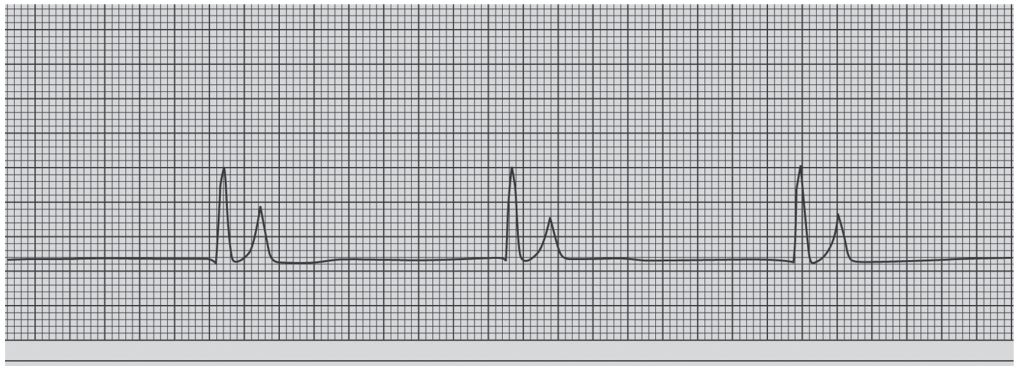


Fig. 42.1. Atrial standstill secondary to hyperkalemia in a blocked cat.

Cats

- Muscles: weakness.
- Cardiac: conduction and rhythm disturbances including tall or deep T waves, shortened P waves, prolonged PR interval, bradycardia, atrial standstill, sine waves, aberrant atrioventricular conduction, asystole.
- Other signs such as dehydration, weak pulses, prolonged capillary refill time, vomiting, sometimes diarrhea, mental depression; if due to acute kidney injury, urinary obstruction, acute hypoadrenocortical crisis.

DIFFERENTIAL DIAGNOSIS

Dogs

- Acute kidney injury
- Hypoadrenocorticism
- Intoxications
- Hypermagnesemia
- Hypoaldosteronism
- Drugs inhibiting potassium excretion
- Hyperkalemia periodic paralysis

Cats

- Acute kidney injury
- Hypoadrenocorticism
- Intoxications
- Hypermagnesemia
- Hypoaldosteronism
- Drugs inhibiting potassium excretion

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DIAGNOSTICS

- Serum magnesium concentration
- Serum potassium concentration
- Serum sodium concentration
- Electrocardiogram
- ACTH stimulation: to diagnose hypoadrenocorticism
- BUN, creatinine, urinalysis
- Abdominal ultrasound and other forms of imaging

Pathologic Findings

- None associated with hyperkalemia itself.
- Hypoadrenocorticism: small adrenal glands grossly. Histopathology will show adrenocortical atrophy with a varying degree of fibrous tissue, lymphocytes, and plasma cell infiltrates. Rarely other pathology from other diseases can occur.
- Renal disease: varying types of renal pathology and histopathology depending on the cause.



THERAPEUTICS

- Treat underlying disease, lower serum potassium level.

Drugs of Choice

- Hypoadrenocorticism: provide intravenous 0.9% saline for rehydration and fluid maintenance.
- Prednisone sodium succinate 1–2 mg/kg IV to provide glucocorticoid replacement in acute crisis, noting that this drug will interfere with serum cortisol measurement with the ACTH stimulation test. To avoid this interference, can use dexamethasone 0.5–2.0 mg/kg IV instead.
- Desoxycorticosterone pivalate 2 mg/kg IM every 21–28 days to replace mineralocorticoid can be given SQ subsequently.
- Abnormal cardiac function calls for additional treatment measures.
    - Calcium gluconate 10%, 1 mL/kg IV, will counter cardiomyotoxicity.
    - Insulin, glucose: give regular insulin at 0.25–0.5 U/kg IV with 2 g of dextrose for each unit of insulin given. This will move potassium ions from the extracellular into the intracellular space.
    - Sodium bicarbonate 1.0 mEq/kg IV.
- For renal disease, treatment consists of intravenous fluids and the same emergency measures (except for the steroids) mentioned previously for treating hyperkalemia-associated cardiac abnormalities.

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Precautions/Interactions

- Calcium gluconate and sodium bicarbonate should be administered in separate syringes to avoid precipitation which occurs when they are mixed.

Diet

- Diet usually unaffected with hypoadrenocorticism.
- With renal disease, a high biological value protein should be used once the animal resumes eating.

Activity

- Normal when the pet returns home.

Surgical Considerations

- Resolve the hyperkalemia before surgery in order to avoid cardiac complications.

Alternate Procedure

- Peritoneal dialysis.

COMMENTS

Client Education

- For renal patient, appropriate diet, water free choice, periodic evaluations by veterinarian.
- For patients with hypoadrenocorticism, be aware of early signs of decompensation: loss of appetite, weakness, and loss of vigor. Owners must provide maintenance medications lifelong and periodic veterinary reevaluations are required.

Patient Monitoring

- For hypoadrenocorticism, periodic serum sodium and potassium determinations with mineralocorticoid drug adjustments as necessary.
- For renal disease, BUN, creatinine, sodium, potassium, phosphorus, calcium, and urinalysis.

Prevention/Avoidance

- Diligent observation for signs of decompensation.
- Medication compliance.

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Possible Complications

- Hyperkalemia can kill if not detected and treated in a timely manner.

Expected Course and Prognosis

- Hypoadrenocorticism: excellent prognosis with treatment compliance.
- Renal disease: fair to grave prognosis depending on pathology.

Synonyms

- Hyper K^+
- $\uparrow K^+$

Abbreviations

ACE: angiotensin-converting enzyme
ACTH: adrenocorticotropic hormone
BUN: blood urea nitrogen
IM: intramuscular
IV: intravenous
SQ: subcutaneous

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Author: Michael Schaer, DVM

chapter 43

Hyperosmolarity

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DEFINITION/OVERVIEW

- Osmolarity: expressed in mOsm/L; represents the number of particles per liter of solution.
- Osmolality: expressed in mOsm/kg; represents the number of particles per kilogram of solution.
- Hyperosmolarity: a high concentration of particles per liter of solution.
- Serum concentrations >310 mOsm/L in dogs and 330 mOsm/L in cats are considered hyperosmolar.

ETIOLOGY/PATHOPHYSIOLOGY

- Serum sodium is responsible for most of the osmotically active particles that contribute to serum osmolarity; serum glucose and urea also contribute to serum osmolarity.
- Anything that causes water loss increases concentrations of solutes in plasma or serum, thereby increasing serum osmolarity.
- Blood volume, hydration status, and ADH secretion are intimately involved in controlling extracellular fluid volume.
- Low circulating blood volume stimulates carotid and aortic baroreceptors to respond to changes in blood pressure, causing the secretion of ADH.
- Hyperosmolarity affects the osmoreceptors in the hypothalamus and stimulates ADH secretion from the neurohypophysis; the hypothalamic thirst center is also stimulated and causes an increase in water consumption to counteract the serum hyperosmolarity by solute dilution.
- Rapid increases in serum osmolarity cause water movement across its concentration gradient from intracellular to extracellular spaces, resulting in neuronal dehydration, cell shrinkage, and cell death; cerebral vessels may weaken and hemorrhage.

Systems Affected

- Nervous: excessive thirst may be the first sign of hyperosmolarity. Central nervous depression may lead to coma.
- Cardiovascular: hypotension and decreased ventricular contractility.
- Renal/urologic: low urine output (<1–2 mL/kg per hour).

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SIGNALMENT/HISTORY

Dogs and Cats

- Hypodipsia and hyperosmolarity have been reported in young female miniature schnauzers.

General Comments

- Primarily neurologic or behavioral.
- Severity is related more to how quickly hyperosmolarity occurs than to the absolute magnitude of the change.
- More likely to occur if serum osmolarity is >350 mOsm/L and usually severe if >375 mOsm/L.

Historical Findings

- Anorexia, lethargy, vomiting, weakness, disorientation, ataxia, seizures, and coma
- Polydipsia followed by hypodipsia

Physical Examination Findings

- Normal or abnormalities may reflect underlying disease.
- In addition to historical findings, dehydration, tachycardia, hypotension, weak pulses, and fever may be detected.

Increased Solutes

- Hypernatremia, hyperglycemia, severe azotemia, ethylene glycol toxicity, salt poisoning, sodium phosphate enemas in cats and small dogs, radiographic contrast solution, administration of ethanol, aspirin toxicosis, shock, lactate in patients with lactic acidosis, acetoacetate and beta-hydroxybutyrate in patients with ketoacidosis, liquid enteral nutrition, and parenteral nutrition solutions.

Decreased Extracellular Fluid Volume

- Dehydration: gastrointestinal loss, cutaneous loss, third space loss, low water consumption, and polyuria without adequate compensatory polydipsia.

Risk Factors/Causes

- Predisposing medical conditions: renal failure, diabetes insipidus, diabetes mellitus, hyperadrenocorticism, hyperaldosteronism, and heat stroke.
- Therapeutic hyperosmolar solutions, hypertonic saline, sodium bicarbonate, sodium phosphate enemas in cats and small dogs, mannitol, and parenteral nutrition solutions.
- High ambient environmental temperatures.
- Fever.

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DIFFERENTIAL DIAGNOSIS

- Primary CNS disease and neoplasia may be characterized by altered mentation, but serum osmolarity is normal.
- Physical evidence or history of injury usually helps to rule out CNS depression caused by cranial trauma.
- Perform a thorough physical examination to assess hydration and obtain information regarding previous therapy that may have included sodium-containing fluids or hyperosmolar solutions.

Laboratory Findings

Drugs That May Alter Laboratory Results

- Excessive administration of sodium-containing fluids or hyperosmolar solutions increases serum osmolarity.
- Valid if run in human laboratory.

DIAGNOSTICS

Complete Blood Count/Biochemistry/Urinalysis

- High PCV, hemoglobin, and plasma proteins in dehydrated patients; serum electrolytes may also be increased.
- Hyperosmolarity is an indication to evaluate serum sodium and glucose concentrations.
- Without the presence of excessive unmeasured osmoles, estimated serum osmolarity may be calculated from serum chemistries. Many formulas exist, but in the presence of hyperglycemia and azotemia, the following formula has been determined to be most reliable:

$$\text{Cats: } [2(Na^+) + \text{glucose} + \text{BUN}]$$

$$\text{Dogs: } [2(Na^+) + [\text{glucose}/18] + [\text{BUN}/2.8]$$

- Normally, calculated osmolarity should not exceed measured osmolarity; if it is, it is considered a laboratory error.
- If measured osmolarity exceeds the calculated osmolarity, determine the osmolar gap.
- Osmolar gap is equal to measured osmolarity less calculated osmolarity.
- High measured osmolarity and normal calculated osmolarity with a high osmolar gap indicate the presence of unmeasured solutes (not Na, K, BUN, or glucose).
- High measured osmolarity and high calculated osmolarity with a normal osmolar gap usually indicate that the hyperosmolarity is caused by hyperglycemia or hypernatremia.
- Serum sodium concentration may be artifactually low in patients with severe hyperglycemia and hyperosmolarity.

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- Fasting hyperglycemia and glucosuria is supportive of a diagnosis of diabetes mellitus.
- Numerous calcium oxalate crystals in the urine suggest ethylene glycol toxicosis.
- High urine specific gravity rules out diabetes insipidus.
- Low urine specific gravity, especially hyposthenuria, suggests diabetes insipidus.
- Urine or serum ketones supportive of ketoacidemia/ketoaciduria – evaluate for diabetes mellitus and diabetic ketoacidosis.

Other Laboratory Tests

- Urine osmolarity lower than serum osmolarity suggests diabetes insipidus; concentrated urine rules out diabetes insipidus.

Imaging

- Renal ultrasonography may reveal bright, hyperechoic kidneys in patients with ethylene glycol toxicosis.



THERAPEUTICS

- Mild hyperosmolarity without clinical signs may not warrant specific treatment, but diagnose and treat underlying diseases.
- Hospitalize patients with moderate to high osmolarity (> 350 mOsm/L) and patients exhibiting clinical signs and gradually lower serum osmolarity with intravenous fluids while a definitive diagnosis is pursued.
- Administer D5W or 0.45% NaCl slowly IV.
- Free water deficit can be calculated by the following formula:

$$\text{Free water deficit less } 0.4 \times \text{lean body weight in kg} \times (\text{plasma Na}/140) - 1$$

  - The goal is to not drop sodium more than 15 mEq/L in an 8-hour period; that is, the ultimate goal is to not drop the sodium by more than 2 mEq/L per hour.
  - Initially, 0.9% NaCl may be used to restore normal hemodynamics and replace dehydration deficits; replace half of dehydration deficit over 12 hours, and the remainder over 24 hours, then switch to D5W or 0.45% NaCl.

Drugs of Choice

- Seizures can be controlled with diazepam, phenobarbital, pentobarbital, or propofol.

Contraindications

- Hypertonic saline and hyperosmolar solutions

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Precautions/Interactions

- May use 0.9% NaCl initially, but rapid administration may worsen neurologic signs.
- Rapid administration of hypotonic solutions (e.g., D5W, 0.45% NaCl) may also worsen neurologic signs.

Alternative Drugs

- Regular insulin (0.1 unit/kg) IM or IV can be administered if a hyperglycemic crisis occurs secondary to parenteral nutrition administration.

COMMENTS

Patient Monitoring

- Hydration status – avoid overhydration.
- Bladder size, urine output, and breathing patterns during intravenous fluids administration.
- Anuria, irregular breathing patterns, worsening depression, coma, or seizures may be signs of deterioration.

Possible Complications

- Altered levels of consciousness and abnormal behavior

Associated Conditions

- Hypernatremia and hyperglycemia

Abbreviations

ADH: antidiuretic hormone
BUN: blood urea nitrogen
CNS: central nervous system
IM: intramuscular
IV: intravenous
K: potassium
Na: sodium
NaCl: sodium chloride
PCV: packed cell volume

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chapter 44

Hypertension

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DEFINITION/OVERVIEW

- Systemic hypertension is an elevation of systemic arterial blood pressure.
- Systemic hypertension is of concern when the elevation of blood pressure is persistent and causing or likely to cause target organ damage.

ETIOLOGY/PATHOPHYSIOLOGY

- Systemic hypertension may be stress related (i.e., "situational" or "white coat" hypertension), idiopathic (no identifiable cause), or occur as a complication of another systemic disease ("secondary" hypertension).
- Secondary hypertension is the most common form of systemic hypertension in dogs and cats. The most common diseases associated with systemic hypertension are as follows.
  - Dogs:
    - Acute or chronic renal failure
    - Protein-losing (glomerular) renal diseases
    - Hyperadrenocorticism
    - Diabetes mellitus
    - Pheochromocytoma
  - Cats:
    - Renal disease of any variety
    - Hyperthyroidism
  - More rare causes in both species include:
    - Hyperaldosteronism (cats)
    - Acromegaly (cats)
    - Sex hormone abnormalities (dogs)
- Pathogenesis is unclear in most diseases. Both cardiac output and systemic vascular resistance affect blood pressure, and alterations in either of these parameters (or their contributing components) can lead to systemic hypertension.

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Systems Affected

- Any vascular bed may sustain damage from acute or chronic systemic hypertension. Most commonly affected organ systems include:
  - Eyes
  - Kidneys
  - Brain
  - Heart/vascular system

Ophthalmologic Effects ("Hypertensive Retinopathy," "Hypertensive Choroidopathy")

- Severe (often associated with acute blindness).
  - Retinal detachment (complete or partial)
  - Hyphema
  - Retinal hemorrhages
- Less severe (may only be obvious on ophthalmologic examination).
  - Papilledema
  - Vascular tortuosity and segmentalization
  - Periarterial intraretinal infiltrates

Renal Effects

- Elevated intraglomerular pressure due to abnormal renovascular autoregulation (ineffective vasoconstriction of renal afferent arterioles) leads to glomerular damage (glomerulosclerosis and atrophy, proliferative glomerulitis) and results in protein loss in urine (proteinuria).
- Proteinuria leads to tubular damage.
  - Tubular inflammation and atrophy
  - Interstitial fibrosis

Central Nervous System Effects

- Damage may result from focal hemorrhage or from cerebral edema.
- Intracranial abnormalities responsible for predominant signs ("hypertensive encephalopathy").
- Most common signs of hypertensive encephalopathy are:
  - Seizures (grand mal or focal facial)
  - Altered mentation: dementia, depression, stupor, coma
  - Isolated cranial nerve deficits
  - Nystagmus
  - Photophobia

Cardiovascular Effects

- Left ventricular hypertrophy
- Systolic heart murmur
- Gallop rhythm

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- Sensitivity to fluids (i.e., "normal" amounts of fluid administration result in congestive heart failure)
- Overt heart failure is rare unless fluids are administered
- Epistaxis



SIGNALMENT/HISTORY

- There are no known breed predilections for the development of systemic hypertension, but sighthounds (e.g., greyhounds, deerhounds) have normal blood pressure ranges that are higher than other breeds.
- There is no age predilection for development of systemic hypertension, but middle-aged to older animals are at increased risk for the systemic diseases likely to lead to systemic hypertension.

Risk Factors/Causes

- Some systemic illnesses have an increased probability of development of systemic hypertension as a complication.

Dogs

- Acute or chronic renal diseases, especially those associated with proteinuria.
- Adrenal diseases.
  - Hyperadrenocorticism
  - Hyperaldosteronism
  - Pheochromocytoma
  - Sex hormone abnormalities
- Diabetes mellitus.
- Use of medications associated with increased blood pressure (e.g., phenylpropanolamine, corticosteroids).

Cats

- Renal disease
- Hyperthyroidism

Historical Findings

- Overt abnormalities that may be noted by owner:
  - Acute blindness, hyphema, retinal detachment
  - Depression
  - Photophobia
  - Epistaxis
  - Seizures
  - Evidence of underlying disease (e.g., polyuria, polydipsia, etc.)

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- Subtle abnormalities that may be missed by owner:
  - Changes in behavior that may be attributed to aging
  - Hiding, depression
  - Subtle signs of underlying disease
  - Decreased vision

CLINICAL FEATURES

- Evidence of target organ damage.
  - Ocular changes (Fig. 44.1):
    - Acute blindness
    - Retinal and choroidal changes as outlined previously
  - Renal changes:
    - Palpably small, irregular kidneys
    - Polyuria/polydipsia
    - Proteinuria



Fig. 44.1. Ocular changes associated with hypertensive choroidopathy in a hypertensive cat. The hyperreflective appearance of the right eye is consistent with retinal detachment. This cat is also showing a mild degree of photophobia.

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  - Neurologic changes:
    - Seizures
    - Changes in mentation
    - Focal cranial nerve deficits
  - Cardiovascular changes:
    - Systolic heart murmur, typically left sided
    - Gallop rhythm
    - Epistaxis
- Evidence of underlying (causative) disease may be detected.



DIFFERENTIAL DIAGNOSIS

Ocular Changes

- Retinal detachment due to infection or inflammation
- Hyphema/retinal hemorrhage due to bleeding disorders

Renal Changes

- Acute or chronic renal disease of any cause
- Infectious causes of proteinuria

Neurologic Changes

- Seizures.
  - Epilepsy
  - Intracranial mass lesions (e.g., neoplasia, abscess)
  - Inflammatory brain lesions
  - Glucose or electrolyte abnormalities/toxins
- Changes in mentation.
  - Intracranial mass lesions
  - Cerebral edema due to infection/inflammation
  - Severe electrolyte abnormalities/toxins
  - Hypoglycemia
- Focal cranial nerve deficits.
  - Intracranial mass lesions
  - Inflammatory brain lesions
- Cardiovascular changes.
  - Heart murmur
    - Hypertrophic or dilated heart disease
    - Valvular disease
  - Gallop rhythm
    - Hypertrophic or dilated heart disease

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- Epistaxis
  - Nasal tumors, nasal infections
  - Bleeding disorders, including coagulopathies, thrombocytopenia, thrombocytopathia



DIAGNOSTICS

- Indications for blood pressure assessment for systemic hypertension include:
  - Evidence of target organ damage as described previously
  - Evidence of systemic disease associated with development of systemic hypertension
- Random blood pressure measurement on young, healthy animals is discouraged due to increased possibility of false-positive readings (i.e., falsely elevated due to activity or excitement).

Dogs

- Evidence of target organ damage.
  - Ocular findings consistent with hypertensive choroidopathy, hypertensive retinopathy, or intraocular hemorrhage as outlined previously
  - Proteinuria without urinary tract infection
  - Intracranial neurologic signs, including depression or changes in mentation
  - Unexplained left ventricular hypertrophy
  - Epistaxis
- Evidence of causative disease.
- Chronic or acute renal disease.
- Hyperadrenocorticism.
- Pheochromocytoma.
- Adrenal mass.
- Diabetes mellitus.

Cats

- The same for dogs as previously described but in addition:
  - Hyperthyroidism
  - Cats older than 10 years of age (due to high prevalence of renal disease and hyperthyroidism)

Blood Pressure Measurement Technique: Preparation

- Blood pressure should be measured prior to other diagnostic activities (e.g., blood sampling, radiographs).
- A period of acclimation (calming, preferably with only mild restraint) of at least 5–10 minutes is allowed prior to measurement.

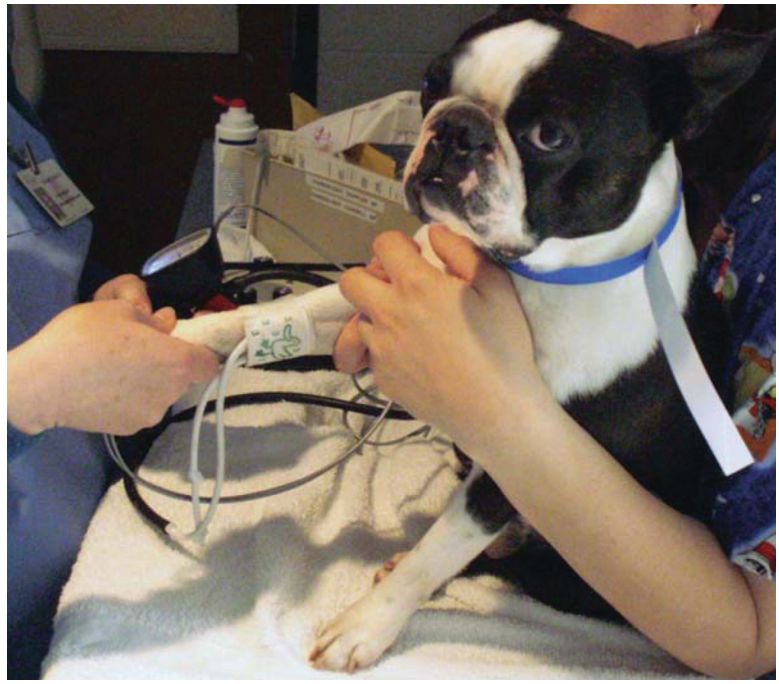
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- Choose an indirect (Doppler or oscillometric) blood pressure cuff of a width that is ~40% of limb circumference.
- Place cuff snugly at point of measurement (during measurement, limb or tail is positioned so that cuff is at the level of heart).
  - Tail head (cat or dog, Fig. 44.2a)
  - Forelimb at level of radius (preferred site in cats, Fig. 44.2b)
  - Hindlimb at metatarsal level (Fig. 44.2c)



Fig. 44.2. (a) Cuff at tailhead in a cat, (b) cuff at level of radius in a cat, and (c) cuff at metatarsal level in a dog.

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■ **Fig. 44.3.** Doppler sphygmomanometric blood pressure measurement in a dog. The cuff is placed around the forelimb at the level of the radius and the leg is elevated to the level of the heart during readings.

Doppler Sphygmomanometric Blood Pressure Measurement (Fig. 44.3)

- Coupling gel is applied to the Doppler crystal, which is then applied over the artery distal to the cuff. The sound generated by blood flow when the Doppler crystal is in the optimal position should be clear and crisp.
- The cuff is inflated until the Doppler signal is silenced. The cuff is then slowly deflated until the Doppler signal is again heard. The pressure (mmHg) at which the Doppler signal is again detected is recorded as the systolic pressure. After further deflation, the pressure at which the audible signal becomes muffled may be recorded as the diastolic pressure.
- Systolic pressure is most reliably estimated by this method. Diastolic pressure can be difficult to determine accurately in many animals.
- Five measurements should be taken and averaged for final value.

Oscillometric Blood Pressure Measurement (Fig. 44.4)

- Automated inflation and deflation reduce operator error.
- Oscillometric monitors deliver systolic, diastolic, and mean arterial pressures as well as heart rate.
- Five measurements should be taken and averaged for final value.

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■ **Fig. 44.4.** Oscillometric blood pressure measurement. The cuff is placed around the distal hindlimb at the level of the metatarsal arteries. The patient is resting comfortably with minimal restraint.

Assessment

- If clinical signs of target organ damage are present, therapy is warranted if systolic blood pressure >160 mmHg.
- If clinical signs of target organ damage are not present, the following guide may be used based on systolic blood pressure.
  - Systolic blood pressure <150 mmHg: no therapy required, monitor if possible causative disease is present.
  - Systolic blood pressure 150–160 mmHg: if presence of underlying disease is confirmed, repeat blood pressure measurement at another session to confirm abnormality. If confirmed systolic blood pressure >150 mmHg, consider therapy.
  - Systolic blood pressure 160–180 mmHg: confirm with second measurement session and treat if underlying disease is present.
  - Systolic blood pressure >180 mmHg: therapy for systemic hypertension is indicated if causative disease is present or strongly suspected.

THERAPEUTICS

- Objectives of therapy:
  - Immediate relief of clinical signs of target organ damage
  - Reduction of detected and undetected target organ damage

Drugs of Choice

Acute Systemic Hypertension (Clinical Signs of Target Organ Damage Present)

- Nitroprusside (intravenous).
  - Potent direct arterial dilator
  - Given as continuous rate infusion via an intravenous catheter with fluid pump

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- Continuous blood pressure monitoring (ideally, direct monitoring via indwelling arterial catheter) is recommended
- Dose (dogs only): 1–10 µg/kg per minute, start at lowest dose and titrate according to continuous blood pressure monitoring

- Amlodipine.
  - Dihydropyridine calcium channel antagonist normally used for long-term management of systemic hypertension (dogs/cats)
  - Dose: 0.2 mg/kg per hour PO until blood pressure normalizes, then decrease to q12–24h
- Hydralazine (oral).
  - Potent direct arterial dilator
  - Onset of action after oral dosage: approximately 1–2 hours
  - Dose (dogs or cats): 0.05–4 mg/kg every 12 hours PO; start at lowest dose and titrate to effect

Chronic Systemic Hypertension

- Underlying disease should be managed optimally (e.g., hyperadrenocorticism, hyperthyroidism).
- Efficacy of chronic antihypertensive medications should be confirmed by repeat blood pressure measurement approximately 7 days after beginning medication or changing dosage.
- Once blood pressure is controlled by chronic antihypertensive medication, blood pressure should be routine monitored approximately every 3 months.

Dogs

- ACEIs.
  - First-line drug for all systemic hypertension
  - Reduces proteinuria in affected dogs
  - ACEIs alone may control mild systemic hypertension adequately but are unlikely to control severe hypertension as monotherapy
  - Dose:
    - Enalapril: 0.5 mg/kg every 12 hours PO
    - Benazepril: 0.25–0.5 mg/kg every 12–24 hours PO
- Amlodipine besylate.
  - Long-acting dihydropyridine calcium channel antagonist
  - Effective, especially in combination with ACEI
  - Dose: 0.1–0.4 mg/kg every 24 hours PO
- Spironolactone.
  - Aldosterone antagonist
  - May be helpful as *adjunctive* medication for control of hypertension
  - Dose: 1–2 mg/kg every 12 hours

Cats

- Amlodipine besylate.
  - Drug of choice for long-term control of systemic hypertension in most cats
  - Well tolerated by most cats
  - Dose: 0.625 mg every 24 hours PO for cats up to 5 kg, 1.25 mg every 24 hours PO for cats >5 kg

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- ACEIs.
  - Less effective as a single agent in cats than in dogs
  - May be used as adjunctive therapy, especially if proteinuria is present
  - Dose: same as dogs
- Spironolactone.
  - Aldosterone antagonist
  - May be helpful as *adjunctive* medication for control of hypertension
  - Dose: same as dogs
- Beta-blocking medications (propranolol, atenolol).
  - Atenolol most frequently used to limit heart rate in cats with hyperthyroidism
  - May be used as *adjunctive* therapy to control blood pressure
  - Dose: 6.25–12.5 mg per cat every 12 hours PO

Precautions/Interactions

Hypotension

- May occur if any medications are overdosed or if dehydration occurs in a patient receiving antihypertensive medications.
- Clinical signs: collapse, weakness, tachycardia, prolonged capillary refill time, and prerenal azotemia.
- Discontinue medication temporarily. Fluid support may be needed until the effects of the medication subside and dehydration is remedied.
- Close monitoring is required when giving direct arterial dilators.
- ACEIs may cause worsening of azotemia if administered to dehydrated animals.

Alternative Drugs

- At this time, no alternative medications or treatments have been proven effective in control of systemic hypertension in dogs and cats.

Diet

- Although moderate sodium reduction and weight control are likely generally beneficial in aging animals, no particular dietary manipulation has been proven to be effective in controlling systemic hypertension in dogs and cats.



COMMENTS

- Many diseases associated with systemic hypertension can be controlled but not cured (e.g., renal disease, hyperadrenocorticism); antihypertensive medications in these patients are likely to be lifelong and may need upward adjustment over time.
- Some diseases (e.g., hyperthyroidism, pheochromocytoma) can be eliminated by appropriate therapy (medical or surgical). Systemic hypertension may resolve after treatment of

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underlying disease, but monitoring is required as blood pressure medications are slowly reduced. In the case of hyperthyroidism, systemic hypertension may become evident *after* therapy for the disease.

- Use of PPA may be associated with systemic hypertension. In dogs in which use of PPA is indicated, normal blood pressure should first be confirmed by a baseline measurement. If baseline blood pressure is within normal range, PPA can be initiated and blood pressure is rechecked approximately 1–2 weeks later. If blood pressure is normal while the patient is receiving PPA, PPA can be continued with blood pressure monitoring approximately every 3 months. If baseline or first recheck blood pressure is elevated, use of PPA should be avoided.

Client Education

- Control of systemic hypertension is most likely to be successful when blood pressure medications are given consistently.
- Optimal treatment of underlying disease is needed in addition to antihypertensive medications to control systemic hypertension.
- Successful antihypertensive therapy frequently involves use of multiple medications and is often lifelong.

Patient Monitoring

- Blood pressure should be monitored continuously in patients with acute hypertension associated with target organ damage until blood pressure is controlled.
- Blood pressure should be assessed within 1 week of initiation or change in chronic therapy.
- Once a patient's blood pressure is stable and underlying disease is controlled, blood pressure should be monitored approximately every 3 months.

Expected Course and Prognosis

- Cause of death or euthanasia is typically related to the underlying disease rather than systemic hypertension, but poorly controlled systemic hypertension contributes to worsening of renal disease and may result in additional complications.

Abbreviations

ACEI: angiotensin-converting enzyme inhibitor
PO: by mouth (per os)
PPA: phenylpropanolamine

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Acknowledgment to the author of the original chapter in *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Emergency and Critical Care, First Edition:* Rebecca L. Stepien.

Hypertrophic and Restrictive Cardiomyopathy

chapter 45



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DEFINITION/OVERVIEW

- Hypertrophic cardiomyopathy (HCM) is an idiopathic disease of the myocardium characterized by concentric hypertrophy and impaired diastolic relaxation. Concentric hypertrophy is a thickened ventricular wall with a normal to small chamber size, caused by a primary, inherent problem of the myocardium.
- Restrictive cardiomyopathy (RCM) comprises a diverse group of myocardial conditions including endocardial, subendocardial, or myocardial fibrosis or other infiltrative disease that results in decreased ventricular compliance.

ETIOLOGY/PATHOPHYSIOLOGY

- Unknown; recent investigations have revealed a genetic origin of HCM in two breeds of cat, the Maine coon and Ragdoll.
- Concentric hypertrophy causes a noncompliant ventricle and leads to reduced diastolic filling and increased diastolic pressures. Diastolic dysfunction may result in atrial dilation with congestive heart failure, left atrial thrombi formation with systemic embolization, impaired myocardial perfusion, myocardial ischemia with subsequent arrhythmias, dynamic ventricular outflow obstruction, and end-stage myocardial failure.
- RCM is also characterized by abnormal diastolic function or a restriction of diastolic filling that results in many of the same pathophysiologic and hemodynamic consequences as HCM.

Systems Affected

- Cardiovascular: impaired myocardial perfusion, myocardial ischemia with subsequent arrhythmias
- Respiratory: tachypnea or cyanosis due to pulmonary edema or pleural effusion
- Musculoskeletal: systemic embolization (pelvic limbs most common) resulting in paresis/paralysis and musculoskeletal pain

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SIGNALMENT/HISTORY

Species

- HCM is the most common cardiac disease in cats and is very rare in dogs.

Gender

- More prevalent in males (up to 87% in some studies).

Age

- Most common in middle-aged cats (4–7 years), but has been reported in cats from 3 months to 17 years of age.

Breed

- Domestic shorthair (most common), Maine coon, American shorthair, Persian, and Ragdoll.

Historical Findings

- Many cats are asymptomatic.
- Tachypnea.
- Inappetence.
- Lethargy.
- Syncope.
- Lameness, paresis (pelvic limbs most common), or vocalization.
- Sudden death.



CLINICAL FEATURES

- Tachypnea
- Orthopnea
- Muffled respiratory or heart sounds with pleural effusion
- Crackles with pulmonary edema
- Heart murmur; variable-intensity systolic murmur with point of maximal intensity in the parasternal region
- Gallop rhythm
- Premature beats
- Jugular pulsations or distention
- Poor body condition score
- Hindlimb paresis/paralysis with extreme pain, absent to weak femoral pulses, cyanosis of nail beds and footpads, and hypothermia with thromboembolic disease

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DIFFERENTIAL DIAGNOSIS

- HCM is considered a primary disease of the myocardium and all other causes for concentric hypertrophy must be ruled out. Other causes for concentric hypertrophy include:
  - Systemic hypertension
  - Hyperthyroidism
  - Aortic stenosis
  - Acromegaly (rare in cats)

DIAGNOSTICS

Laboratory Tests

- Pleural or abdominal effusion cytology: modified transudate is most common, but pure transudate or chylous effusion is also possible.
- Thromboembolic disease: elevations in creatine kinase, enzymes of hepatocellular damage (i.e., ALT, AST), renal values (i.e., BUN, creatinine), potassium, and cardiac troponin I; lactate obtained from vein in affected limb(s) is often higher than lactate concentration in nonaffected limb(s).

Electrocardiography

- Highly variable and nonspecific
- Left axis deviation (partial left bundle branch block)
- Ventricular or atrial premature complexes or tachyarrhythmias

Thoracic Radiography

- Mild-to-moderate generalized cardiomegaly (Fig. 45.1)
- Left atrial or biatrial enlargement (Fig. 45.2)
- Interstitial or alveolar infiltrates consistent with pulmonary edema
- Pleural effusion
- Pulmonary vascular distension

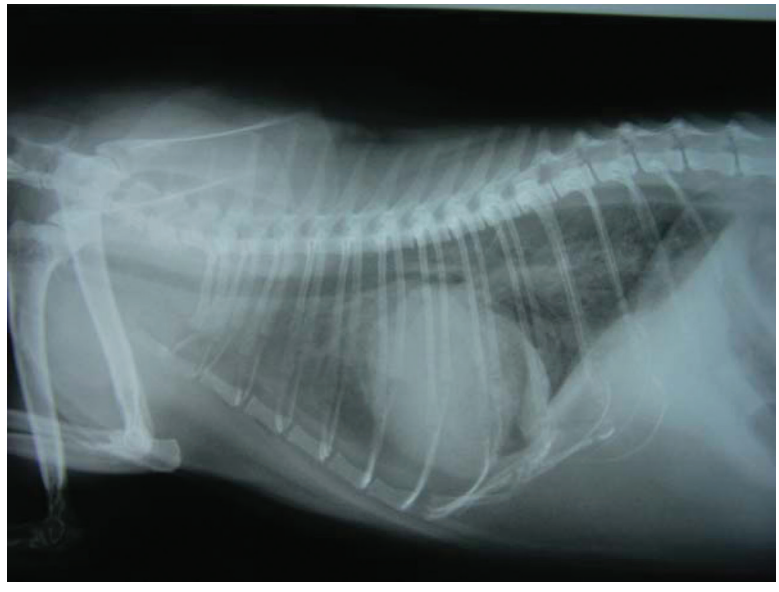
Echocardiography

- Gold standard for antemortem diagnosis
- M-mode and two-dimensional echocardiography

HCM

- Left ventricular wall thickness at end-diastole >5.5 mm
- Papillary muscle hypertrophy
- SAM of the mitral valve is present in approximately 50% of cases
- Left atrial enlargement with advanced disease (left atrial to aortic diameter >1.5)

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■ **Fig. 45.1.** Lateral thoracic radiograph of a cat with generalized cardiomegaly and pulmonary edema secondary to hypertrophic cardiomyopathy.



■ **Fig. 45.2.** Dorsoventral thoracic radiograph of biatrial enlargement of a cat with severe hypertrophic cardiomyopathy. This is the classic "valentine-shaped" heart sometimes seen with this disease.

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RCM

- Variable echocardiographic findings in RCM.
- Severe left atrial dilation is a common feature.
- Left ventricular internal dimensions are typically mildly to moderately reduced.
- May demonstrate loss of normal left ventricular symmetry, distorted or fused papillary muscles, and mild left ventricular hypertrophy with biatrial enlargement.

Pathologic Findings

HCM

- Thickening of the left ventricular myocardium (the interventricular septum and free wall), with the entire wall commonly being 8–11 mm thick
- Papillary muscle hypertrophy
- Smaller than normal left ventricular chamber

RCM

- Patchy or diffuse endocardial, subendocardial, or myocardial deposition of fibrous tissue.
- The endocardium may appear whitish-gray, opaque, and thickened.



THERAPEUTICS

Acute Congestive Heart Failure

- Oxygen supplementation.
- Furosemide: 1–4 mg/kg IV or IM every 1–2 hours initially and then adjusted as tachypnea resolves (usually 1–2 mg/kg every 6–12 hours); must be used with caution in patients with concurrent renal disease; hypokalemia can occur as a secondary complication of furosemide therapy.
- Serial renal values and electrolytes.
- Thoracocentesis as needed to control pleural effusion.

Acute Thromboembolic Crisis

- Treatment of congestive heart failure.
- Analgesia: use one of the following medications:
  - Hydromorphone: 0.05–0.1 mg/kg IV, IM, or SQ every 2–6 hours
  - Morphine: 0.05–0.2 mg/kg IM or SQ every 4–6 hours
  - Buprenorphine: 0.01–0.03 mg/kg IV, IM, or buccally every 6–8 hours
  - Fentanyl (2 µg/kg IV, then 3–5 µg/kg per hour IV CRI)

Anticoagulant Therapy

- Efficacy is unknown in cats.
- Additional studies are necessary to establish dosage and frequency.

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- Unfractionated heparin: 250 U/kg SQ every 8 hours, aPTT should be prolonged by 1.5–2 times normal
- Low molecular weight heparins:
  - Enoxaparin: 1 mg/kg SQ every 12 hours
  - Dalteparin: 100 U/kg SQ every 24 hours
- Aspirin: 5–81 mg PO every 72 hours
- Clopidogrel: 18.75 mg PO every 24 hours

Thrombolytic Therapy

- Tissue plasminogen activator or streptokinase: very expensive, severe or fatal complications including reperfusion injury or hemorrhage, no survival benefit compared to conservative therapy.

Chronic Congestive Heart Failure

Diuretics

- Furosemide: 0.5–2.0 mg/kg PO every 8–48 hours
- Hydrochlorothiazide: 1–2 mg/kg PO every 12–24 hours, typically used with furosemide in patients refractory to furosemide monotherapy
- Spironolactone: 1–2 mg/kg PO every 12–24 hours, typically used with furosemide

Angiotensin-Converting Enzyme Inhibitors

- Use only one.
  - Benazepril: 0.25–0.05 mg/kg PO every 24 hours
  - Enalapril: 0.25–0.5 mg/kg PO every 12–24 hours

Thromboprophylaxis

- Recommended in patients after thromboembolic events and for those patients at risk for systemic emboli.
- Aspirin 5–81 mg PO every 72 hours.
- Clopidogrel: 18.75 mg PO every 24 hours.
- Beta-blockers: never been shown to improve patient outcome, use extreme caution in patients with congestive heart failure, systolic dysfunction, hypotension, or bradyarrhythmias; may be preferred drug choice with systolic anterior motion of mitral valve.
  - Atenolol: 6.25–12.5 mg PO every 12–24 hours, slowly titrated to achieve a heart rate 140–160 bpm
- Calcium channel blockers: never been shown to improve patient outcome, use extreme caution in patients with congestive heart failure, systolic dysfunction, hypotension, or bradyarrhythmias.
  - Diltiazem: 7.5 mg PO every 8–12 hours

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COMMENTS

Patient Monitoring

- Asymptomatic patients should have echocardiography every 6–12 months.
- Patients receiving diuretics should be monitored for azotemia and electrolyte abnormalities (e.g., hyponatremia, hypokalemia).
- After a thromboembolic event, assess for return of limb function, reperfusion injury (e.g., hyperkalemia), limb necrosis, or self-mutilation.

Expected Course and Prognosis

- The prognosis for HCM and RCM is generally based on clinical presentation, echocardiographic and radiographic evidence of elevated diastolic pressures, and response to therapy. Asymptomatic cats with mild left atrial enlargement are believed to have a good long-term prognosis. Asymptomatic cats with marked left atrial enlargement are at risk for developing heart failure. Cats that present in heart failure have a poor prognosis with a short survival time (weeks to months). Cats that present in heart failure and respond favorably to therapy may do well for longer periods. Cats presenting with aortic thromboembolism have a poor prognosis.

Abbreviations

ALT: alanine aminotransferase
AST: aspartate aminotransferase
aPTT: activated partial thromboplastin time
BUN: blood urea nitrogen
CRI: constant rate infusion
HCM: hypertrophic cardiomyopathy
IM: intramuscular
IV: intravenous
PO: by mouth (per os)
RCM: restrictive cardiomyopathy
SAM: systolic anterior motion
SQ: subcutaneous

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chapter 46

Hyphema



DEFINITION/OVERVIEW

- Hyphema is the presence of blood in the anterior chamber of the eye.



ETIOLOGY/PATHOPHYSIOLOGY

- Trauma, either blunt or penetrating.
- Bleeding or clotting disorders (e.g. thrombocytopenia, inherited clotting deficiencies, consumption of clotting factors, liver failure).
- Blood dyscrasias (e.g., polycythemia, hyperviscosity).
- Systemic hypertension.
- Uveitis, especially with concurrent vasculitis (e.g., FIP in cats, rickettsial diseases in dogs).
- Other ocular disease (e.g., neoplasia, retinal detachment, chronic glaucoma, collie eye anomaly, persistent primary hyperplastic vitreous) generally due to stimulation of vascular growth on iris (preiridal fibrovascular membrane).

Systems Affected

- Ophthalmic



SIGNALMENT/HISTORY

- Hyphema can occur in any dog or cat regardless of age, breed or sex.
- Since there are many causes for hyphema, the signalment and history of the patient will vary and should be used as a tool to guide the clinician towards a diagnostic plan.

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Historical Findings

- Many dogs and cats with hyphema will present as an ocular emergency when the owners notice blood in the eye.
- If hyphema is caused by trauma, the patient may be presented for signs of acute ocular pain (e.g., epiphora and blepharospasm).
- Weight loss, lethargy, or decreased appetite may indicate a systemic cause.



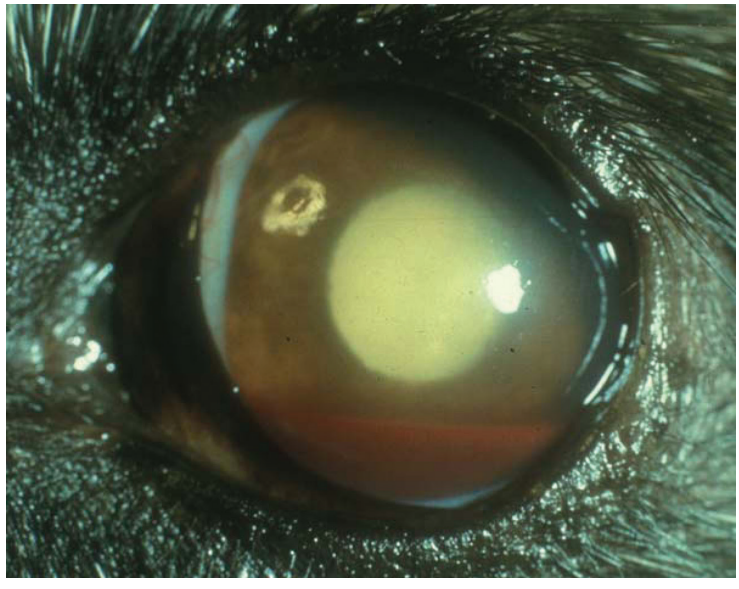
CLINICAL FEATURES

- The clinical features of hyphema will depend on the quantity of blood, whether or not the blood is clotted, the duration of the hyphema, and potentially the activity of the animal.
- Acute, traumatic hemorrhage will appear bright red (Fig. 46.1).
- If the blood partially fills the anterior chamber and the animal has been inactive for some time, the blood will settle out in a gravity-dependent manner (Fig. 46.2).
- Hemorrhage that completely fills the anterior chamber will generally clot within a few days and turn dark in color (referred to as an "eight ball" hemorrhage).
- Hyphema associated with infectious disease, such as FIP, will often be mixed with inflammatory cells in a fibrin clot. Clots will often localize over the pupil.
- Blood in the anterior chamber may or may not clot. The reasons for this are not well understood.
- Intraocular pressure may be elevated or may become elevated and should be monitored.



Fig. 46.1. Acute hyphema – blood is dispersed throughout the anterior chamber. The ability to see internal structures indicates that the hemorrhage is not extensive.

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■ **Fig. 46.2.** Blood has settled into the ventral anterior chamber. The amount of blood is less than one-third of the volume of the anterior chamber and carries a good prognosis for complete resolution.



■ **Fig. 46.3.** Complete hyphema in a proptosed globe. The anterior chamber is completely filled with blood as is the subconjunctival tissue. The prognosis for vision in this eye is grave. Hyphema this extensive generally leads to glaucoma or phthisis bulbi.

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DIFFERENTIAL DIAGNOSIS

- Iris injection/vascularization
- Corneal vascularization or intrastromal corneal hemorrhage

DIAGNOSTICS

- Hyphema is diagnosed by observing blood in the anterior chamber.
- Thorough and detailed history, physical and ophthalmic examination (including opposite eye) should be done to rule in/out trauma.
- Ophthalmic examination should always include fluorescein staining for corneal ulcers and intraocular pressure measurement for secondary glaucoma.
- Other diagnostic tests should be geared towards the suspected cause of hyphema once trauma is ruled out.
    - Minimum database (CBC, serum chemistries, UA) for all suspected systemic causes.
    - Coagulopathy: tests to assess hemostasis.
    - Metastatic neoplasia: thoracic radiographs and abdominal ultrasound.
    - Serum titers for suspected infectious disease.
    - Ocular ultrasound is indicated when blood prevents complete examination of the eye to identify neoplasia, retinal detachment and posterior segment (vitreal, retinal, subretinal) hemorrhage.

Pathologic Findings

- Blood (free or in proteinaceous matrix) is seen in the anterior chamber, iridocorneal angle, and trabecular meshwork.
- Blood or fibrinous adhesions (anterior synechia) may mechanically occlude the iridocorneal angle and cause secondary glaucoma.
- Minor to extensive posterior synechia may be present.
- Bloodstaining of the cornea may result with concurrent glaucoma.
- Fibrovascular membrane formation over the iris surface is common when hyphema is secondary to chronic ocular disease.

THERAPEUTICS

- The immediate objectives in the treatment of hyphema are to:
    - Keep the animal quiet to prevent rebleeding (especially when secondary to trauma)
    - Treat concurrent anterior uveitis
    - Eliminate or manage underlying cause when identified
    - Prevent or treat secondary glaucoma

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Drugs of Choice

- Topical 1% prednisolone drops or 0.1% dexamethasone ointment q6–8h for concurrent anterior uveitis.
- Topical 1% atropine sulfate drops or ointment q8h until the pupil dilates, then use to effect.
  - Atropine is used to prevent posterior synechia, pupil obstruction and iris bombé.
  - Pupil dilation can obstruct the iridocorneal angle and aqueous humor outflow so IOP should be carefully monitored
  - If IOP elevates after pupil dilation, atropine should be discontinued.
  - Because of the bitter taste, ointment is generally better tolerated than solution.
- Topical 2% dorzolamide drops q8h to decrease aqueous humor formation if intraocular pressure is elevated.
- Tissue plasminogen activator (tPA) 25 µg injected into the anterior chamber to dissolve formed clots.
  - tPA frequently causes rebleeding and should not be used in the presence of active hemorrhage or nonclotted blood.

Precautions/Interactions

- The use of topical or systemic nonsteroidal antiinflammatory agents is not recommended due to their effect on platelet function.
- Topical glucocorticoids should not be used in the presence of corneal ulceration.
- Topical ointment preparations should not be used if the eye has been perforated.
- Continuous dilation of the pupil with atropine may obstruct aqueous humor outflow through the iridocorneal angle.
- Close monitoring of the intraocular pressure is recommended when using atropine.
- Atropine is contraindicated if intraocular pressure is elevated unless pressure elevation is being caused by posterior synechia and iris bombé.

Activity

- The activity of the patient should be restricted for several days after hyphema has occurred. Cage rest or sedation may be necessary.

Surgical Considerations

- Surgery is rarely indicated or performed to treat hyphema.
- Uncontrolled glaucoma and complete hyphema that has been present for 5–10 days can be an indication for invasive surgery to remove the blood clot. Surgery often results in rebleeding.
- Uncontrolled glaucoma and complete hyphema carries a poor prognosis for glaucoma control or vision; enucleation may be required to relieve pain.

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COMMENTS

Client Education

- Hyphema often obstructs vision, increasing the risk of further injury to the eye.
- Glaucoma, if not already present, could develop, especially if hyphema is moderate to severe.
- Owner should contact veterinarian if signs of pain occur or worsen (squinting, tearing, rubbing, lethargy), since this could indicate glaucoma or corneal ulceration.
- If pupil is widely dilated, discontinue atropine until pupil begins to constrict.
- The necessity of treating the underlying cause of hyphema should be emphasized.

Patient Monitoring

- Monitor underlying disease as recommended.
- Monitor eyes with moderate-to-severe hyphema frequently.
    - If IOP is elevated, recheck daily; additional ocular hypotensive medications may be necessary.
    - If IOP is in normal range, monitor IOP every 2–3 days.

Possible Complications

- Chronic glaucoma and pain
- Blindness
- Phthisis bulbi
- Cataract

Expected Course and Prognosis

- Mild hyphema (<30% of anterior chamber filled when blood settles) carries a good prognosis for vision.
- Moderate hyphema (30–50% of anterior chamber filled when blood settles) carries a guarded prognosis for vision due to potential of secondary glaucoma.
- Severe hyphema (>50% of anterior chamber filled when blood settles) carries a poor prognosis for vision due to intraocular changes and uncontrollable secondary glaucoma.

Abbreviations

CBC: complete blood count
FIP: feline infectious peritonitis
IOP: intraocular pressure
tPA: tissue plasminogen activator
UA: urine analysis

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chapter 47

Hypoadrenocorticism

DEFINITION/OVERVIEW

- Primary hypoadrenocorticism is a disease in which the zona glomerulosa and the zona fasciculata of the adrenal cortex are destroyed, leading to a deficiency of the mineralocorticoid hormone aldosterone and the glucocorticoid hormone cortisol.
- In most cases, primary hypoadrenocorticism occurs as an immune-mediated destruction of the adrenal cortex, although more rarely, adrenal hemorrhage and adrenal neoplasia also can be inciting causes.
- Secondary hypoadrenocorticism occurs due to lack of adrenocorticotropic hormone release from the pituitary gland, and results in lack of glucocorticoid production by the adrenal cortex. Mineralocorticoid function is preserved.
- Iatrogenic hypoadrenocorticism can occur secondary to drug administration (trilostane, mitotane) or secondary to abrupt withdrawal of exogenous glucocorticoid drugs.
- Hypoadrenocorticism is rare in cats, and can be associated with adrenal lymphoma.

ETIOLOGY/PATHOPHYSIOLOGY

Pathophysiology

- Immune-mediated destruction of the zona fasciculata causes a lack of cortisol release. Lack of cortisol can result in a variety of clinical signs including inappetence, vomiting, diarrhea, weakness, tremors, muscle wasting, weight loss, and poor hair coat.
- Immune-mediated destruction of the zona glomerulosa causes deficiency of aldosterone. Deficiency of aldosterone results in lack of sodium resorption and lack of potassium excretion with resultant hyponatremia and hyperkalemia.
- The inability to regulate sodium and potassium eventually causes loss of the renal concentration gradient, leading to lack of water reabsorption. Isosthenuria and dehydration ensue. Prerenal azotemia can also be exacerbated by clinical signs of vomiting and diarrhea.
- Severe hyperkalemia can result in a loss of action potential generation of the cardiac myocytes, and can cause an idioatrial arrhythmia (a.k.a. atrial standstill), with loss of

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P waves, widened QRS complexes, and spiked T waves. Can eventually lead to ventricular asystole and death if left untreated.

Systems Affected

- Hematologic: lack of erythrocyte production, as well as gastrointestinal blood loss, can cause a microcytic anemia. Absence of glucocorticoid release during stress causes absence of a stress leukogram on CBC.
- Musculoskeletal: muscle wasting, weight loss, weakness, rarely, tremors.
- Gastrointestinal: inappetence, vomiting/hematemesis, diarrhea/hematochezia, gastrointestinal ulceration, megaesophagus.
- Metabolic: hypoglycemia, hyperkalemia, hyponatremia, hyperphosphatemia.
- Renal: isosthenuria, azotemia.
- Neurologic: mental dullness, tremors.
- Respiratory: aspiration pneumonia secondary to regurgitation and/or vomiting.



SIGNALMENT/HISTORY

- Primarily occurs in dogs, and rarely occurs in cats.
- Can occur in any age/breed/gender of dog; middle-aged female dogs more commonly affected.
- Median age of affected animals is 4–6 years.

Breed Predisposition

- Standard Poodle
- Bearded Collie
- Rottweiler
- Great Dane
- Portuguese Water Dog
- Bassett Hound
- West Highland White Terrier
- Labrador Retriever
- Wheaten Terrier
- Leonberger



CLINICAL FEATURES

- May be vague, and waxing and waning.
- Weight loss, muscle wasting.
- Inappetence, vomiting, regurgitation, diarrhea, sometimes with hematemesis/hematochezia.
- Lethargy.

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- Tremors.
- Cough, increased respiratory rate/effort secondary to aspiration pneumonia.
- Acute onset of severe hypovolemic shock may also be present.
- Relative bradycardia.

Bloodwork

- Not all bloodwork abnormalities may be present.
- Hypoglycemia.
- Hyponatremia, hyperkalemia, Na:K ratio <27 (<30 in cats).
- Serum sodium and potassium may be normal in animals with secondary or atypical hypoadrenocorticism (lack of cortisol alone, with normal aldosterone).
    - Dogs with *Trichuris* infestation, pregnant greyhounds, and dogs/cats with third spacing of fluids (pleural or abdominal effusion) can also have abnormally low sodium:potassium ratios.
    - Japanese Akita dogs can have pseudohyperkalemia.
- Hypochloremia.
- Hyperphosphatemia.
- Hypercalcemia.
- Hypocholesterolemia.
- Metabolic acidosis.
- Azotemia (elevated BUN and creatinine).
- Lack of a stress leukogram (lymphocytosis and normal neutrophil count on CBC).
- Eosinophilia.
- Nonregenerative anemia.

Urinalysis

- Isosthenuria to hyposthenuria in face of dehydration.



DIFFERENTIAL DIAGNOSIS

- Nonspecific primary or secondary gastroenteritis
- AKI, CKD
- Gastrointestinal parasitism (*Trichuris* spp.) or severe bacterial infection



DIAGNOSTICS

Definitive Diagnostic Testing

ACTH Stimulation Test

- Take 2–3 mL of whole blood to obtain a baseline serum cortisol sample.
- Administer 0.25 mg IV ACTH synthetic gel.

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- Obtain post-ACTH serum cortisol sample 1 hour (dogs) or 30 minutes and 1 hour (cats) after ACTH gel.
  - NOTE: Steroids such as prednisone, prednisolone, and methylprednisolone will all cross-react with cortisol assay. Dexamethasone and dexamethasone-SP will not cross-react with cortisol assay.
  - Resting cortisol levels in dogs/cats with hypoadrenocorticism are usually undetectable, and below the sensitivity of cortisol assay (<0.2 µg/dL).

Endogenous ACTH Levels

- ACTH is very labile and unstable.
  - Obtain 2–3 mL of whole blood directly into a cooled plastic tube (ACTH will bind to glass).
  - Immediately centrifuge, decant plasma, and immediately freeze at –20 °C in plastic tube.
- Used to differentiate between primary and secondary hypoadrenocorticism.
  - Must obtain sample prior to administration of any glucococorticosteroid drug.
  - In animals with primary hypoadrenocorticism, endogenous ACTH levels are extremely elevated >500 pg/mL.
  - Patients with secondary hypoadrenocorticism have extremely low or undetectable endogenous ACTH levels (<5 pg/mL).
  - Plasma aldosterone concentration may be decreased in patients with hypoadrenocorticism, even those with normal or abnormal Na:K ratios.
  - Plasma renin activity.

Diagnostic Imaging

- Thoracic radiographs may show signs of microcardia, small caudal vena cava (both supportive of hypovolemia), megaesophagus, and aspiration pneumonia.
- Abdominal ultrasound will show small adrenal glands.



THERAPEUTICS

- Intravenous fluids to treat hypovolemia (when present) and dehydration.
- Caution must be exercised to not increase serum sodium concentration by more than 15 mEq/L in a 24-hour period, to avoid complication of central pontine myelinolysis.
- Antiemetic drugs.
  - Maropitant.
  - Dolasetron.
- Gastroprotectant drugs.
  - Famotidine.
  - Omeprazole.
  - Pantoprazole.

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- Antibiotics.
  - Unasyn.
  - Enrofloxacin.
  - Metronidazole.
- Definitive therapy requires replenishment of glucocorticoids and administration of mineralocorticoid activity if patient also has electrolyte abnormalities.
- Dexamethasone or dexamethasone sodium phosphate 0.5–1 mg/kg IV once.

Long-Term Therapy

Glucocorticoid Replacement

- Predniso(lo)ne: 0.1–2 mg/kg/day (canine).
  - Glucocorticoid activity alone will also require mineralocorticoid supplementation if electrolyte abnormalities exist.
  - Increase frequency of dosing to q12h during periods of expected stress.

Mineralocorticoid Replacement

- Desoxycorticosterone pivalate (DOCP, Percortin).
  - Long-acting repository mineralocorticoid, no glucocorticoid activity.
  - 2.2 mg/kg IM or SQ q25 days.
  - Check electrolytes 1 week and then 3 weeks after administration.
  - After 2–3 months of chronic therapy, if electrolytes are stable at 25 days, some patients can be extended to less frequent dosing if monitored closely.

Glucocorticoid and Mineralocorticoid Replacement

- Fludrocortisone (Florinef).
  - Has both glucocorticoid and mineralocorticoid activity.
  - 15–30 µg/kg/day.
  - Start at lower end of dose; however, many animals require incremental adjustments based on serum electrolyte concentrations.



COMMENTS

Prevention/Avoidance

- Increase exogenous glucocorticoid administration in affected animals during periods of apparent stress.

Possible Complications

- Central pontine myelinolysis if serum sodium rises too quickly with therapy for acute severe hyponatremia/hypoadrenocorticism.

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- Aspiration pneumonia.
- Bacterial translocation and sepsis.
- Hypotension.
- Multiorgan failure.
- Death.

Synonyms

- Addison's disease

Abbreviations

ACTH: adrenocorticotropic hormone
AKI: acute kidney injury
CBC: complete blood count
CKD: chronic kidney disease
IM: intramuscular
IV: intravenous
SQ: subcutaneous

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chapter 48

Hypokalemia

DEFINITION/OVERVIEW

- Hypokalemia is a serum electrolyte disorder that occurs when the serum potassium (K^+) is less than 3.5 mEq/L (normal range 3.5–5.5 mEq/L).
- Occurs secondary to disorders of internal and/or external balance.

ETIOLOGY/PATHOPHYSIOLOGY

- Disorders of internal balance will translocate K^+ from the extracellular space (ECF) to the intracellular space (ICF).
- Includes metabolic alkalosis, insulin administration, increased levels of catecholamines, beta-adrenergic agonist treatment or intoxication, and refeeding syndrome.
- Includes renal potassium wasting (common in cats), inadequate intake, diuretic drugs (furosemide), osmotic and postobstructive diuresis, inadequate parenteral fluid supplementation (iatrogenic), aldosterone-secreting tumor, vomiting and diarrhea, diabetic ketoacidosis, and renal tubular acidosis.
- Most of the clinical signs are a result of impaired membrane sodium-potassium pump function and a sustained hyperpolarized cell membrane. This delays calcium ion influx into the cell which causes impaired organ function.

Systems Affected

- Cardiovascular: hyperpolarization with complex conduction abnormalities and impaired blood pressure control causing hypertension.
- Endocrine/metabolic: glucose intolerance.
- Gastrointestinal: ileus.
- Musculoskeletal: weakness.
- Renal: impaired tubular function (cats).
- Respiratory: impaired ventilation from muscle weakness.

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SIGNALMENT/HISTORY

- Renal tubular acidosis can be familial.
- History usually provides clues to the underlying problem such as predisposing medical problems and current medications.
- The most common clinical sign is muscle weakness.

Risk Factors

- Diabetes mellitus, body fluid losses, metabolic alkalosis, diuretic drugs, aldosterone-secreting tumor, renal disease (cats).

Historical Findings

- Weakness
- Lethargy
- Mental depression
- Polydipsia/polyuria
- Sometimes vomiting, depending on cause

CLINICAL FEATURES

Dogs

- Muscle weakness
- Hypoventilation in extreme cases (K^+ <2.0 mEq/L)
- Gastrointestinal ileus

Cats

- Muscle weakness (Fig. 48.1)
- Hypoventilation
- Gastrointestinal ileus
- Impaired renal function
- Ventral cervical flexion from skeletal muscle weakness possible in dogs and cats

DIFFERENTIAL DIAGNOSIS

Dogs and Cats

- Hyperkalemia
- Hypo- and hypercalcemia
- Hypomagnesemia

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Fig. 48.1. a, b. This diabetic cat has severe skeletal muscle weakness because of hypokalemic myopathy. Note the characteristic ventral cervical posture. Recovery was uneventful after it received parenteral fluids supplemented with potassium chloride.

- Peripheral neuropathy
- Combined neuromuscular disorders (myasthenia gravis)
- Organophosphate intoxication
- Myopathy

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DIAGNOSTICS

- History of underlying diseases, diet, and pattern of weakness all helpful.
- Serum potassium concentration is absolute and confirmative.
- Serum creatine kinase value might be elevated, and this may or may not represent necrosis.
- Abdominal ultrasound when searching for hyperaldosteronism.
- Serum aldosterone concentrations when searching for hyperaldosteronism.
- Antiacetylcholinesterase receptor antibodies.

Pathologic Findings

- Rarely muscle necrosis
- Ileus



THERAPEUTICS

- Correct the cause.
- Replace potassium.

Drugs of Choice

- Replace potassium deficits with potassium chloride solution intravenously or potassium gluconate powder orally mixed in food.
- Replacement deficits can be corrected with potassium chloride 10% solution given in amounts that are proportional to the severity of the hypokalemia, as shown in Table 48.1 which shows two different methods (a and b) for providing the potassium ions.
- The intravenous rate of administration for mild-to-moderate hypokalemia should usually not exceed 0.5 mEq/kg/h, but for life-threatening hypokalemia (serum level <1.5 mEq/L), the rate can be increased to 1.5 mEq/kg/h along with simultaneous ECG monitoring.
- Oral potassium gluconate is given as a powder added to food at a dose of ¼ teaspoonful (2 mEq) per 4.5 kg body weight twice daily.

TABLE 48.1. Methods for providing potassium ions.

Serum Potassium Concentration	(a) mEq KCl Added to Liter of Saline	(b) mEq KCl Per Kg Body Weight Given Over 24 Hrs*
3.0–3.5	30	1–3
2.5–3.0	40–50	3–5
2.0–2.5	60	5–7
<2.0	80	7–10

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- Potassium phosphate can be given if the patient is also markedly hypophosphatemic. One-half of the potassium replacement can be given intravenously at a dosage of 0.01–0.03 mmol/kg/h over 6 hours.

Precautions/Interactions

- Rapid administration of potassium salts intravenously can cause cardiac dysrhythmia, conduction defects, and death.
- Excessive potassium phosphate can cause hypocalcemia.

Alternative Drugs

- Potassium-sparing diuretics such as spironolactone might be indicated. The dose is 2–4 mg/kg once daily for dogs and 1.0 mg/kg twice daily for cats.
- Mild hypokalemia can sometimes be corrected with foods high in potassium concentration such as bananas.

Diet

- Diet should be balanced and contain adequate amounts of potassium.

Activity

- The muscle weakness will limit the patient's activity.
- Strength will return soon after a few days of eukalemia.
- Return to exercise should be gradual over several days.

Surgical Considerations

- Hypokalemic animals are an anesthetic risk because of the potential for cardiac dysrhythmias, especially at serum concentrations of <3.0 mEq/L.
- Any underlying metabolic alkalosis in addition to the hypokalemia should be corrected prior to surgery, if possible.

COMMENTS

Client Education

- Any necessary dietary adjustments should be followed.
- Any need for continued potassium supplementation should be delivered.

Patient Monitoring

- Frequency will depend on the underlying cause and correction of this.

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Prevention/Avoidance

- All intravenous fluids that do not contain potassium should be supplemented so long as the patient is tolerant to this cation (i.e., acute renal failure or acute hypoadrenocorticism).
- Patients on potassium-losing diuretics should have ample amounts of potassium in their diets.

Possible Complications

- Accidental overload of potassium salts during replacement treatment.
- Cardiac arrhythmias if hypokalemia persists.

Expected Course and Prognosis

- All signs are reversible.
- Underlying cause must be treated as well.
- Prognosis is good to excellent.

Synonyms

- Hypo K^+
- $\downarrow K^+$

Abbreviations

ECF: extracellular fluid
ECG: electrocardiogram
ICF: intracellular fluid

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Author: Michael Schaer, DVM

chapter 49

Hypotension

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DEFINITION/OVERVIEW

- Hypotension is an abnormally low arterial blood pressure, often defined as a mean arterial pressure below 60 mmHg or a systolic arterial pressure below 90 mmHg.

ETIOLOGY/PATHOPHYSIOLOGY

- Both cardiac output (volume of blood pumped by the heart per minute) and the vascular tone, or degree of systemic vascular resistance, contribute to arterial blood pressure. A dysfunction of either cardiac output or vascular tone or both can result in hypotension.
- Cardiac output is determined by venous return (amount of blood returning to the heart), contractility (strength of myocardial contraction), and afterload (the pressure against which the heart must pump). A decrease in venous return or contractility or an increase in afterload will all decrease cardiac output, and potentially lead to hypotension. Anything that causes vasodilation (endotoxemia, hypercarbia, anesthetic gases, etc.) can decrease vascular tone and lead to hypotension.
- Hypotension occurs in 22–32% of anesthetized dogs and 33% of anesthetized cats. The incidence of hypotension in traumatized or sick awake dogs is unknown, but would be expected to be high due to blood loss, sepsis, and related conditions.

Systems Affected

- Nervous: decreased cognitive function, and leads to an obtunded or comatose patient.
- Gastrointestinal: gastric and intestinal ischemia and subsequent mucosal sloughing.
- Musculoskeletal: generalized weakness.
- Hepatobiliary: hepatic ischemia which can decrease metabolism of drugs or clearance of toxins.
- Cardiovascular: ischemia, arrhythmias, decreased contractility.
- Renal/urologic: renal tubular necrosis and acute renal failure.

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SIGNALMENT/HISTORY

- There are no breed or sex predilections for hypotension.
- Any age patient can develop hypotension, although older animals may be more likely to develop it.
- There is no genetic basis for hypotension.
- Signs associated with hypotension include depression, lethargy, weakness, pale mucous membranes, cool peripheral extremities, tachy- or bradycardia, and a slow capillary refill time.

Risk Factors/Causes

- Anesthesia
- Trauma
- Inflammation
- Sepsis
- Myocardial disease/failure
- Hemorrhage
- Vomiting
- Diarrhea

Historical Findings

- Depression
- Lethargy
- Weakness
- Disappearance from home
- History of trauma/blood loss
- Exercise intolerance
- History of vomiting or diarrhea



CLINICAL FEATURES

- Depression
- Lethargy
- Weakness
- Slow capillary refill time
- Pulse quality may or may not be poor
- Blanched skin/mucous membranes
- Cool peripheral extremities

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DIFFERENTIAL DIAGNOSIS

- Sepsis: septic patients are often, but not always, hypotensive. Septic patients will tend to have an inflammatory leukogram and be hypoglycemic, hypoproteinemic, and have other clinical signs (i.e., vomiting, infectious nidus, etc.).
- Hypovolemia: patients who are severely dehydrated or have had blood loss may also be hypotensive. Hypovolemic patients rapidly respond to appropriate fluid therapy.
- Cardiac failure: patients with myocardial disease who have compromised contractility may be hypotensive. Any patient with no obvious cause for hypotension should have a thoracic examination, including auscultation, radiographs, and echocardiography, to rule out cardiac failure as a cause of hypotension.
- Arrhythmia: patients with third-degree atrioventricular block or other causes of bradycardia may suffer collapse and weakness due to inability of the heart to maintain cardiac output. The blood pressure in these patients is often normal. An ECG should be checked if the patient is bradycardic.
- Electrolyte disturbances: acidemia, hypocalcemia, hypokalemia, and hypomagnesemia may all complicate existing hypotension and should be ruled out by running an electrolyte and acid–base profile.
- Anemia: patients who are anemic without being hypovolemic (e.g., IMHA) may present with weakness similar to hypotensive patients. A complete physical examination, blood count, biochemistry profile, and evaluation for autoimmune disease should be conducted in cases of suspected anemia. Patients with anemia without blood loss will tend to have a normal-to-high protein level and will have no identifiable source of blood loss (i.e., internal hemorrhage into the chest or abdomen or external hemorrhage).
- Neurologic disorder: patients presenting obtunded or depressed should be given a thorough neurologic examination to rule out the presence of neurologic disease. Patients with neurologic disease but without hypotension should have a normal blood pressure on evaluation.
- Hypoxemia: patients with respiratory or cardiac illness who have impaired diffusion of oxygen in the lungs may resemble patients who are hypotensive in being depressed and weak, and may have pale or blue mucous membranes. Patients who are hypoxemic are usually normo- to hypertensive unless a concurrent disease is present. Patients who are hypoxemic can be diagnosed on the basis of respiratory distress, an abnormally low pulse oximeter reading, or an arterial blood gas analysis.

DIAGNOSTICS

- Indication: any patient presenting after trauma or with signs of depression, sepsis, lethargy, or any patient under anesthesia.
- Doppler: indirect method using a sphygmomanometer, blood pressure cuff, and Doppler sound device which gives systolic arterial pressure only.

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- Oscillometric: indirect method using a circumferential cuff which automatically inflates and deflates on a periodic basis, giving systolic, diastolic, and mean arterial pressures.
- Direct arterial pressure: direct method utilizing an arterial catheter and pressure transducer to give the gold standard real-time reading of systolic, diastolic, and mean arterial pressures.

Pathologic Findings

- Prolonged hypotension will cause ischemia, which can manifest in any organ. Ischemia will cause necrosis, and the brain, heart, and kidneys tend to be the most sensitive organs.



THERAPEUTICS

- The objective of treatment is to raise mean arterial blood pressure above 60 mmHg and diastolic blood pressure above 40 mmHg, or if no means to measure mean blood pressure are available, systolic arterial blood pressure above 90 mmHg.

Drugs of Choice

- Crystalloid fluids: balanced isotonic crystalloid fluid such as lactated Ringer's, Normosol-R, or Plasmalyte-A 60 mL/kg
- Dobutamine 5–10 µg/kg per minute IV CRI
- Dopamine 5–20 µg/kg per minute IV CRI
- Ephedrine 60 µg/kg IV or IM

Precautions/Interactions

- Any drug that has the potential to cause or worsen hypotension should be used cautiously or not at all in patients who are already experiencing hypotension. This includes most anesthetic agents, some antibiotics if given rapidly, and drugs that cause vasodilation (nitroglycerine, nitroprusside, ACE inhibitors).

Alternative Drugs

- Phenylephrine 1–3 µg/kg per minute CRI
- Norepinephrine 0.05–3 µg/kg per minute CRI
- Vasopressin 0.5–5 mU/kg per minute CRI
- Epinephrine 0.01–0.1 µg/kg per minute CRI

Activity

- Patients typically will be too weak to be active. Because many of the medications require continuous intravenous access, limiting patient movement while in a treatment cage may be helpful.

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Surgical Considerations

- Anesthesia should generally be avoided in patients who have existing hypotension, or the hypotension should be resolved before anesthesia. If surgery is necessary to correct the underlying cause of hypotension (i.e., septic abdomen), the patient should be stabilized and blood pressure-sparing anesthetic agents such as benzodiazepines (e.g., diazepam, midazolam), opioids (e.g., hydromorphone, fentanyl, morphine), and dissociatives (e.g., ketamine) should be used. Etomidate (0.5–1.5 mg/kg IV) has no cardiovascular side effects and may be ideal in hypotensive patients when combined with a benzodiazepine for rapid anesthetic induction. Use of etomidate alone can cause clonus.



COMMENTS

- Blood flow is what is actually desired. Blood flow is determined in part by blood pressure, but also by vascular tone. Drugs that increase vascular tone (e.g., phenylephrine, ephedrine, epinephrine) will increase blood pressure, but this may be at the cost of peripheral blood flow and perfusion. These drugs should be used cautiously to improve blood pressure in hypotension.

Client Education

- A patient with hypotension should not be discharged.

Patient Monitoring

- Blood pressure should be monitored at least every 5 minutes during anesthesia or in emergency situations. For long-term treatment, blood pressure should be checked every 15 minutes for the first hour, then hourly thereafter if the patient is critical or every 6 hours once the patient is stable.

Possible Complications

- Patients who are profoundly hypotensive may not regain consciousness, particularly after anesthesia.
- Possible clinical sequelae to hypotension include cerebral damage (especially blindness) and renal damage. These are typically noted before discharge. The owners should be informed that they should have regular bloodwork to ensure that the animal does not develop early-onset chronic renal failure due to the hypotensive episode(s).

Expected Course and Prognosis

- Hypotension can resolve rapidly with definitive treatment (such as fluids in a hypovolemic patient) and usually responds immediately to the drug treatments previously described.

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- Most patients that develop mild hypotension under anesthesia suffer no long-term consequences.
- Severe hypotension can cause cerebral and renal damage.
- In some cases, particularly severe sepsis, patients may not respond to initial drug treatments and more aggressive medications (given in alternative treatments) will be necessary.
- Patients can respond to medication initially and then gradually redevelop hypotension. In nonresponder cases, it is important to rule out and treat complicating conditions (e.g., acidosis, hypocalcemia, anemia). In patients who do *not* respond to initial medication measures or develop recurring hypotension despite treatment, the prognosis is poor to grave.

Synonyms

- Shock, hypoperfusion, low blood pressure

Abbreviations

ACE: angiotensin-converting enzyme
CRI: constant rate infusion
ECG: electrocardiogram
IMHA: immune-mediated hemolytic anemia
IV: intravenously

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Author: Erik Hofmeister, DVM, MA

chapter 50

Hypothermia

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DEFINITION/OVERVIEW

- Defined as a decrease in normal body temperature (37.8–39.2 °C or 100.0–102.5 °F) to below 37 °C (99 °F).
  - Mild hypothermia: 32–37 °C (90–99 °F)
  - Moderate hypothermia: 28–32 °C (82–90 °F)
  - Severe hypothermia: below 28 °C (82 °F)

ETIOLOGY/PATHOPHYSIOLOGY

- Under normal circumstances, the body produces heat as a byproduct of metabolic processes. Body heat is centered around the core, and by the process of thermoregulation, heat can be dissipated from the skin and respiratory tract. Activation of the autonomic nervous system and thermoregulatory system in the hypothalamus occurs in response to cold and body cooling, and causes shivering and increased cellular metabolism in an attempt to produce heat and rewarm the body.
- Even during some stages of mild hypothermia, when the body temperature drops to below 93 °F, adverse consequences such as peripheral vasodilation, decreased cellular metabolism, and decreased heat production occur. As hypothermia progresses to below 88 °F (31 °C), the animal can no longer thermoregulate.

Systems Affected

- Hepatic: decreased hepatic enzyme activity.
- Renal/urologic: increased glomerular filtration rate and diuresis, decreased sensitivity to antidiuretic hormone, fluid loss/dehydration; decreased renal blood flow, renal hypoxia, renal tubular necrosis.
- Coagulation: coagulopathies including decreased platelet aggregation, decreased thromboxane A2, and decreased coagulation factor activity. Predisposition to DIC due to sequestration of platelets in the reticuloendothelial system in the liver and spleen.

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- Cardiovascular: mild tachycardia with mild hypothermia, decreased tissue perfusion, and cellular hypoxia; vasoconstriction can increased systemic vascular resistance. Decreased response to catecholamines, bradycardia, decreased cardiac output, and hypotension. Atrial and ventricular fibrillation can occur with severe hypothermia.
- Respiratory: decreased tidal volume and respiratory rate, decreased minute ventilation, hypoxia.
- Neurologic: decreased cerebral perfusion and oxygen delivery. Neurologic abnormalities can range in severity from depression and weakness to stupor or coma.
- Immune system: decreased immune function secondary to decreased neutrophil activation, phagocytic capacity, chemotaxis; decreased cytokine and antibody production. Can lead to increased risk of infection and delayed wound healing.
- Metabolic: decreased off-loading of oxygen in peripheral tissues due to left shift of the oxyhemoglobin dissociation curve.

Incidence/Prevalence

- Unknown; most common cause is perianesthetic hypothermia.

Geographic Distribution

- None, although may be more common in cold, wet environments.



SIGNALMENT/HISTORY

Species

- Dogs and cats.

Breed Predilection

- No breed predilection, although short-haired, smaller breeds are at higher risk than large breeds.

Mean Age and Range

- Neonatal animals and geriatric animals are at higher risk.

Predominant Sex

- None.

Historical Findings

- Prolonged exposure to cold, wet environments or submersion in cold water.
- Unknown history if animal has wandered from home or may have experienced trauma.

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- History of anesthesia.
- Unresponsive, cold animal.

Physical Examination Findings

- Severity is dependent on degree and duration of hypothermia.
  - Mental depression
  - Lethargy
  - Weakness
  - Shivering
  - Muscle stiffness
  - Bradycardia
  - Hypotension
  - Decreased respiratory rate and depth
  - Stupor
  - Obtundation
  - Coma
  - Muffled to absent heart sounds
  - Bradycardia
  - Fixed dilated pupils
  - Death

Risk Factors/Causes

- Large body surface area to mass ratio in small animals.
- Decreased heat production in neonatal or cachectic animals.
- Prolonged preoperative preparation time.
- Intraoperative or perianesthetic hypothermia and inadequate warming.
- General anesthesia (worsens with duration of anesthesia).
- Inadequate shelter in cold or wet climates.
- Inadequate food and water in cold or wet climates.
- Hypothyroidism.
- Cold or moist ambient environments.
- Decreased thermoregulatory ability.
- Impaired ability to seek out heat/shelter or leave cold environment (nonambulatory, debilitated, or injured animals).
- Decreased ability to generate heat (hypothyroid, neonatal, geriatric animals).



DIFFERENTIAL DIAGNOSIS

Diagnosis

- In animals with severe hypothermia, it may be very difficult to differentiate between death and hypothermia until aggressive rewarming measures have been instituted.

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Differentiating Causes

- Must differentiate between other causes of neurologic depression, including trauma, hypovolemic or cardiogenic or septic shock, hepatic encephalopathy, toxin, neoplasia, hypoglycemia or other metabolic disorders.



DIAGNOSTICS

Complete Blood Count/Chemistry/Urinalysis

- Usually normal.
- Some patients may exhibit hyperglycemia and mild hemoconcentration.

Other Laboratory Tests

- Coagulation tests: thrombocytopenia, prolonged ACT, aPTT, and PT.
- Endocrine: decreased T_4 with concomitant elevation in eTSH may reveal true hypothyroidism versus euthyroid sick syndrome associated with other underlying illnesses.

Diagnostic Procedures

- Temperature probes (esophageal or rectal) are useful in assessing patient's temperature and rewarming.

Electrocardiography

- Sinus bradycardia with prolonged PR, QRS, and RT intervals.
- May see atrial fibrillation.
- Ventricular dysrhythmias are common.
    - Premature ventricular contractions, ventricular tachycardia, and ventricular fibrillation may be observed as hypothermia becomes progressively worse.



THERAPEUTICS

- Inpatient therapy is recommended until temperature, acid–base and electrolyte status, and correction of underlying cause of hypothermia have been resolved.
- Body temperature may initially decrease further during early stages of rewarming due to improved circulation of the peripheral tissues, vasodilation, and mixing of blood from the core to the cooler periphery.
- Goal is to provide ambient rewarming measures without promoting further loss of heat.
- Oxygen supplementation and endotracheal intubation are both necessary in obtunded patients. In most severe cases, mechanical ventilation may be necessary.
- Passive rewarming with insulated blankets.

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- Active rewarming with external heat sources such as forced warm air blankets or circulating warm water blankets or pads.
- Plastic bubble wrap or cellophane on limbs to prevent heat loss from feet, particularly during anesthetic procedures.
- Core rewarming techniques such as warm peritoneal, gastric, or urinary lavage, warm water enemas, warm intravenous fluids, and warmed airway air.

Drugs of Choice

- Oxygen supplementation: 50–150 mL/kg per minute
- Isotonic crystalloid fluids

Contraindications

- Avoid lactated Ringer's solution with severe hypothermia because lactate requires hepatic metabolism.

COMMENTS

Patient Monitoring

- Monitor core body temperature very closely during rewarming.
- Monitor ECG and blood pressure.
- Frostbite.

Prevention/Avoidance

- Avoid prolonged exposure to cold temperatures.
- Use warm water blankets or forced warm air blankets during anesthetic procedures.

Possible Complications

- Increased metabolic rate and shivering.
- Increased cellular oxygen demand.
- Thermal burns from inappropriate rewarming measures such as heating pads.
- Afterdrop phenomenon: core temperature of patient continues to fall during rewarming (passive and active).
- Rewarming shock: rapid vasodilation during rewarming that results in severe venous pooling; ideally, avoid warming body temperature more than 1 °F per hour.
- Frostbite.
- Cardiac arrest.
- Death.

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Expected Course and Prognosis

- Varies depending on degree and duration of hypothermia.
- Worse prognosis with debilitated animals.

Age-Related Factors

- Neonatal and geriatric patients are at higher risk.

Abbreviations

ACT: activated clotted time
aPTT: activated partial thromboplastin time
DIC: disseminated intravascular coagulation
ECG: electrocardiogram
eTSH: endogenous thyroid stimulating hormone
PT: prothrombin time
T_4: thyroxine

Suggested Reading

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chapter 51

Hypoxemia

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DEFINITION/OVERVIEW

- Hypoxemia is defined as an arterial partial pressure of oxygen (PaO_2) of less than 80 mmHg. Clinically significant desaturation of hemoglobin occurs at a PaO_2 <60 mmHg and warrants rapid intervention.

ETIOLOGY/PATHOPHYSIOLOGY

- The five causes of hypoxemia are:
  - Low fraction of inspired oxygen (FiO_2)
  - Hypoventilation
  - Mismatching of alveolar ventilation and pulmonary perfusion (V/Q mismatch)
  - Right-to-left cardiac or intrapulmonary shunting
  - Alveolar-capillary membrane diffusion defect
- Conditions that result in hypoxemia usually result from the combined effects of more than one of these causes.
  - Low FiO_2: the FiO_2 of environmental air is made of 21% oxygen. High altitudes have lower barometric pressures than at sea level and, subsequently, offer lower partial pressure of atmospheric oxygen. Patients attached to a breathing circuit (anesthetic circuit, ventilator) or in an enclosed environment (oxygen cage) could accidentally be exposed to air with a lower FiO_2. Both of these scenarios would result in a lowered driving force to oxygenate the blood due to decreased inspired oxygen partial pressures reaching the alveoli.
  - Hypoventilation: defined as a decrease in the alveolar ventilation for the amount of carbon dioxide produced, hypoventilation results in hypercapnia or a rise in the arterial partial pressure of carbon dioxide ($PaCO_2$). In such instances, hypoxemia may occur due to insufficient oxygen delivery to the alveoli. Conditions associated with central respiratory depression (CNS disease or an anesthetic overdose) or those affecting the respiratory apparatus (upper airway obstruction, restriction of the thoracic cage from pleural space disease, neuromuscular disease, thoracic trauma, diaphragmatic hernia, ascites or severe gastric dilation) can all cause decreased alveolar ventilation.

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- In addition to the afore-mentioned causes of hypoxemia, there are three additional mechanisms that include a degree of gas exchange impairment across the alveolar-pulmonary capillary membrane, resulting in an arterial PO_2 that is substantially lower than the alveolar PO_2.

Ventilation-Perfusion Mismatch

- Ventilation-perfusion mismatch occurs along a spectrum: at one end, alveoli are perfused but not ventilated (low V/Q mismatch). Diseases that create partial filling of alveoli with fluid (pneumonia, pulmonary edema, pulmonary contusions, neoplasia) or partial obstruction of the terminal airways (bronchitis) result in regions of impaired gas exchange. This leads to a reduced oxygen uptake from the capillaries perfusing this group of alveoli, with subsequent dilution of the systemic arterial blood with deoxygenated blood. V/Q mismatch is the most common cause of hypoxemia in small animal patients.
- At the other end of the spectrum, the alveoli are ventilated but not perfused, resulting in pure dead space ventilation (high V/Q mismatch). Patients with pulmonary thromboembolism represent this group.

Right-to-Left Cardiac or Intrapulmonary Shunting

- Physiological right-to-left shunts occur when areas of the lung are completely unventilated (V/Q = 0) due to lung lobe consolidation (from atelectasis, pneumonia, edema, neoplasia) and thus are unable to contribute to gas exchange. The deoxygenated blood coming from these unventilated areas of the lung dilutes the arterial partial oxygen pressure, leading to hypoxemia. Pathologic anatomic shunts are associated with abnormal communication between right and left sides of the circulation (R→L-sided shunting ventricular septal defects, R→L-sided shunting patent ductus arteriosus). These are much less common than the afore-mentioned physiologic shunts.

Alveolar-Capillary Membrane Diffusion Defect

- Diseases that decrease the area or increase the thickness of this gas exchange membrane (e.g., smoke inhalation, oxygen toxicity, and pulmonary fibrosis) may potentially cause hypoxemia if patients are exercised. Exercise exacerbates this diffusion block by increasing pulmonary capillary flow, allowing less time for oxygen transfer across the already impaired membrane.

Systems Affected

- Respiratory: decreased arterial partial oxygen pressure will eventually lead to hypoxia, or decreased oxygen delivery to the tissues.
- CNS: the most important organ to consider during hypoxemia and its resultant hypoxia is the CNS. Cerebral hypoxia can result in irreversible brain damage.

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- Gastrointestinal: the gastrointestinal tract is susceptible to bacterial translocation during periods of hypoxia.



SIGNALMENT/HISTORY

- Brachycephalic breeds may be more susceptible to hypoxemia secondary to upper airway obstruction.
- West Highland White Terriers and idiopathic pulmonary fibrosis (Westie lung disease).

Risk Factors

- Bronchopneumonia, pleural space disease, trauma, anesthesia, prolonged recumbency, cardiac disease, neuromuscular disorders, structural or dynamic upper airway obstruction (brachycephalic airway syndrome).

Historical Findings

- Owners may describe increased respiratory rate and effort, cyanosis, coughing, gagging, trauma, exercise intolerance, or collapse.



CLINICAL FEATURES

- Tachypnea, orthopnea, sitting with head and neck extended, restlessness, open mouth breathing, stridor, stertor, nasal flare, cyanotic or pale mucous membranes, coughing, bronchial sounds or crackles and wheezes on auscultation, tachycardia, heart murmurs, arrhythmias and pulse deficits.



DIFFERENTIAL DIAGNOSIS

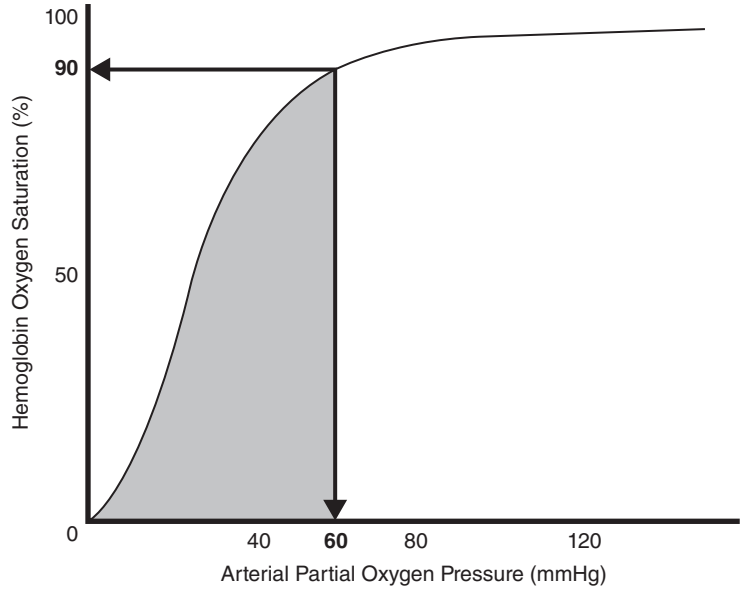
- Excitement/anxiety
- Hyperthermia/fever
- Pain
- Head trauma
- Anemia



DIAGNOSTICS

- The gold standard for assessing hypoxemia, as well as ventilatory and acid–base status, is through an arterial blood sample that is collected in an anaerobic manner.
- An alternative method of continuous analysis is with a pulse oximeter, which will indirectly determine the functional oxygen saturation of hemoglobin in the blood (SpO_2). The

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■ **Fig. 51.1.** The curve shows that the oxygen saturation of hemoglobin decreases sharply when the arterial partial oxygen pressure is less than 60. Animals with respiratory distress can compensate just above the flexure point of the curve when at rest, and their oxygen hemoglobin saturation is preserved (SpO_2 of 90%). Any stress may cause further decreases in the arterial partial pressure of oxygen which will bring the oxygen hemoglobin saturation into the steep downward part of the curve. Therefore, leaving the patient to relax with oxygen supplementation is the first priority upon arriving at the hospital.

PaO_2 and SpO_2 are related based on the oxyhemoglobin dissociation curve (Fig. 51.1). The SpO_2 remains greater than 90% when PaO_2 values are greater than 60 mmHg. SpO_2 values below 90–92% are considered abnormal.

- Disadvantages of pulse oximetry are that the probe is most accurate in nonmoving animals when clipped into the tongue or in nonpigmented mucous membranes. Also, pulse oximetry only indicates the percentage saturation of available hemoglobin molecules, irrespective of the amount of hemoglobin present or of their ability to function appropriately
- Therefore, it is important to correlate pulse oximetry findings with the patient's perfusion parameters (blood pressure and lactate levels), the patient's hemoglobin levels (estimated as one-third of the hematocrit), and the patient's ventilatory status (venous PCO_2 will suffice for this) to obtain a more complete assessment of the patient's overall ability to properly oxygenate the tissues.

Pathologic Findings

- Gross findings: pale or cyanotic mucous membranes.
- Histopathologic findings: tissue ischemic necrosis (GI tract, brain, heart).

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THERAPEUTICS

- The most common supportive treatment of hypoxemia is to provide supplemental oxygen therapy.
- Patients with severe hypoxemia despite oxygen therapy, severe hypoventilation, and those with excessive work of breathing may require mechanical ventilation.
- Oxygen can be delivered to the patient from an anesthetic machine via an oxygen facemask, from an oxygen tank and regulator through a cannula placed in the nares, through an intratracheal catheter or with an oxygen cage. At 1–3 L/min flow rates, up to 40% FiO_2 can be delivered via nasal cannula or up to 80% FiO_2 with intratracheal administration. A sealed oxygen cage can provide up to 40–50% FiO_2.

Drugs of Choice

- Oxygen

Precautions/Interactions

- Aggressive fluid therapy is contraindicated in patients with hypoxemia caused by cardiac failure and pulmonary edema.
- Diuretic therapy is indicated in patients with hypoxemia caused by cardiac failure and pulmonary edema.

Diet

- NPO if patient is in respiratory distress.

Activity

- Hypoxemic patients should be minimally stressed and handled. Those with pulmonary parenchymal disease or lung atelectasis will benefit from being maintained in sternal positions if recumbent. Coupage and short leashed walks help patients with bronchopneumonia dislodge and excrete pulmonary exudates.

Surgical Considerations

- For dogs showing early signs consistent with brachycephalic syndrome, widening the nostrils and possibly shortening the soft palate may confer a long-term favorable prognosis of avoiding collapsing episodes secondary to asphyxia.

COMMENTS

- Patients in respiratory distress should be minimally restrained and supplemented with oxygen.
- It is important to remember the oxygen dissociation curve when dealing with animals in respiratory distress. This curve relates the arterial partial pressure of oxygen with the percentage saturation of hemoglobin with oxygen (see Fig. 51.1).

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Client Education

- Owners should be instructed to monitor respiratory rate and effort when their animals are at rest.
- Check for cyanotic mucous membranes.

Patient Monitoring

- The effectiveness of supportive (oxygen therapy) and definitive treatment can be assessed by physical examination findings associated with a more comfortable and less anxious patient: reduced respiratory rate and effort, decreased tachycardia, and improvement of cyanotic mucous membranes (if present initially).
- Effectiveness of oxygen therapy can be objectively assessed by repeating arterial blood gas analysis and comparing trends based on the alveolar and arterial oxygen partial pressure gradients. If possible, the patient should be taken off oxygen supplementation for 5–10 minutes before performing these measurements of arterial oxygenation. This will remove the effects of supplementation on the patient's arterial blood gas parameters. The A-a gradient provides the most accurate comparison of the pulmonary function at different times. Pulse oximetry can also be used for continual assessment of the patient's oxygenation. Pulse oximetry readings on and off (for 5–10 minutes) oxygen support may be beneficial to identify the need for as well as the response to oxygen supplementation.

Prevention/Avoidance

- Weight loss for overweight patients.
- Activity restriction during hot weather.
- Early corrective surgery for brachycephalic breeds.

Possible Complications

- Exposure to 60% FiO_2 or greater for more than 24 hours can produce signs of oxygen toxicity due to oxygen-derived free radical formation and pulmonary damage.
- The full impact of these effects may not be evident until pulmonary damage becomes irreversible.
- Arterial blood gas analysis and pulse oximetry should be used to guide oxygen supplementation. If prolonged periods of high FiO_2 are anticipated, ventilator assistance should be considered.

Expected Course and Prognosis

- Depends on cause and patient response to therapy.

Abbreviations

CNS: central nervous system
FiO_2: fractional percentage of inspired oxygen

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GI: gastrointestinal
Hb: deoxyhemoglobin
HbO_2: oxyhemoglobin
NPO: nothing by mouth (nil per os)
PaO_2: arterial partial pressure of oxygen
$PaCO_2$: arterial partial pressure of carbon dioxide
SpO_2: indirect measurement of the hemoglobin oxygen saturation of arterial blood made via pulse oximetry

See Also

Smoke Inhalation

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chapter 52

Idioventricular Dysrhythmias

DEFINITION/OVERVIEW

- If conduction of sinus node pacemaker impulses to the ventricles is blocked or the impulses decrease in frequency, the lower regions of the heart automatically take over the role of pacemaker for the ventricles, which results in ventricular escape complexes (Fig. 52.1) or an idioventricular rhythm (Fig. 52.2).

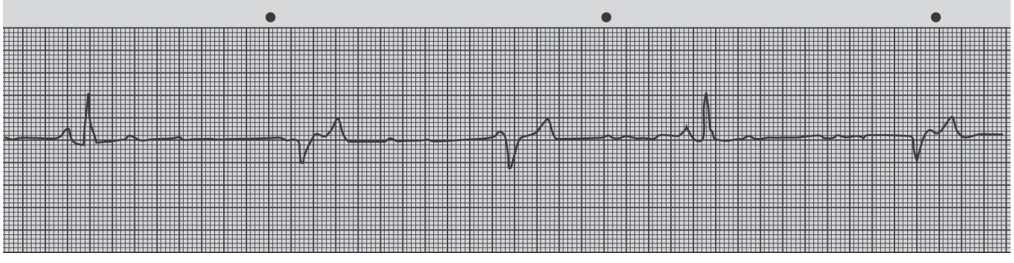


Fig. 52.1. Ventricular escape complexes during various phases in the dominant sinus rhythm in a dog during anesthesia. The sinus rate increased (*not shown*) after anesthesia was stopped; 1/2 cm 1 mv. Source: Tilley 1992. Reproduced with permission of John Wiley & Sons, Inc.



Fig. 52.2. Complete heart block. The P waves occur at a rate of 120, independent of the ventricular rate of 50. The QRS configuration is a right bundle branch block pattern. The regular rate and stable QRS indicate that the rescuing focus is probably near the AV junction. Source: Tilley 1992. Reproduced with permission of John Wiley & Sons, Inc.

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Features

- A series of ventricular escape beats with a heart rate <65 beats per minute in dogs and <100 beats per minute in cats; heart rates of 65–100 beats per minute in dogs and 100–160 beats per minute in cats are often termed *accelerated idioventricular rhythms*.
- P waves may be absent or may precede, be hidden within, or follow the ectopic QRS complex.
- P waves are unrelated to the QRS complexes.
- QRS configuration is wide and bizarre, similar to that of a ventricular premature complex.



ETIOLOGY/PATHOPHYSIOLOGY

- May be hemodynamically important with slow ventricular rates.
- Does not occur in healthy animals.
- Subsidiary pacemakers seem to discharge more rapidly in cats than in dogs.

Risk Factors/Causes

- Not a primary disease; a secondary result of a primary disease.
- The escape rhythm is a safety mechanism to maintain cardiac output.
- Causes of sinus bradycardia and sinus arrest.
- Increased vagal tone (i.e., high intracranial pressure, high ocular pressure).
- Drugs: digoxin, tranquilizers, propranolol, quinidine, and anesthetics.
- Addison's disease.
- Hypoglycemia.
- Renal failure.
- Hypothermia.
- Hyperkalemia.
- Hypothyroidism.
- Causes of AV block.
- Congenital.
- Neoplasia.
- Fibrosis.
- Lyme disease.
- Toxins (digitalis, lily of the valley).

Systems Affected

- Cardiovascular



SIGNALMENT/HISTORY

Species

- Dogs and cats.

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Breed Predilections

- Atrial standstill in English springer spaniels and Siamese cats.
- Pugs, miniature schnauzers, and Dalmatians prone to conduction abnormalities.

Historical Findings

- Some animals asymptomatic
- Weakness
- Lethargy
- Exercise intolerance
- Syncope
- Heart failure



CLINICAL FEATURES

Physical Examination Findings

- Irregular rhythm associated with pulse deficits
- Variation in heart sounds
- Possible intermittent "cannon" waves in the jugular venous pulses (with AV block)



DIFFERENTIAL DIAGNOSIS

- Ventricular tachycardia: dogs have a cardiac rate >100 beats per minute; cats >150 bpm.
- Slow heart rate in animals with right bundle branch block, left bundle branch block, or left anterior fascicular block; animals with these disturbances have the P waves associated with the QRS complexes.



DIAGNOSTICS

Complete Blood Count/Biochemistry/Urinalysis

- No specific findings.
- Complete blood testing may suggest a metabolic abnormality.

Other Laboratory Tests

- Drug toxicity
- Lyme's titer in animals with complete AV block

Imaging

- Echocardiogram may show structural heart disease.

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Diagnostic Procedures

- Electrocardiography

Pathologic Findings

- Depend on underlying cause.



THERAPEUTICS

Drugs of Choice

- Atropine or glycopyrrolate usually indicated to block vagal tone or increase the heart rate.
- If those drugs are ineffective, isoproterenol, dopamine, dobutamine, or artificial pacing may be needed.

Contraindications

- Lidocaine, procainamide, quinidine, propranolol, diltiazem, or any other drug that slows the cardiac rate or reduces contractility.

Precautions/Interactions

- Atropine is briefly vagotonic immediately post injection and can temporarily exacerbate the condition.

Diet

- No modifications or restrictions unless required for management of the underlying condition.

Activity

- Symptomatic animals may require cage rest.

Surgical Considerations

- Pacemaker implantation may be necessary.

Appropriate Healthcare

- Rhythm is an escape or safety mechanism for maintaining cardiac output; do *not* direct treatment toward suppressing this escape rhythm, but toward the primary disease process that allows the escape rhythm to assume pacemaker control of the heart.
- Symptomatic treatment is directed toward increasing the heart rate.

Nursing Care

- May be required for underlying disease.

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COMMENTS

Client Education

- Inform of the need to seek and specifically treat an underlying cause.

Patient Monitoring

- Serial ECG may show clearing of the lesion or progression to complete heart block.
- Serial blood profiles may be needed to monitor progress of the primary disease process.
- Serial echocardiograms may show improvement or progressive changes in cardiac structure.

Possible Complications

- Prolonged bradycardia may cause secondary congestive heart failure or inadequate renal perfusion.

Expected Course and Prognosis

- Arrhythmia may abate when the primary disorder is corrected.
- Guarded if condition is associated with cardiac or metabolic disorder; poor if the rate is not increased pharmacologically or if underlying cause cannot be identified and treated.

Abbreviations

AV: atrioventricular
ECG: electrocardiogram

See Also

Atrioventricular Block

Suggested Reading

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chapter 53

Immune-Mediated Hemolytic Anemia

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DEFINITION/OVERVIEW

- IMHA is a type II hypersensitivity reaction resulting from the production of antierythrocyte antibodies that cause the destruction or accelerate the removal of circulating erythrocytes.
- IMHA can be primary (idiopathic) or secondary to medication or disease.
- Immunosuppression is the mainstay of therapy. Additional therapies include blood product transfusion therapy, thromboprophylaxis, supportive care, and treatment of any underlying condition.
- Response to therapy is variable. Mortality is related to refractory anemia, thromboembolism (usually pulmonary), or adverse effects of therapy (immunosuppression or GI hemorrhage).

ETIOLOGY/PATHOPHYSIOLOGY

- Antierythrocyte antibodies form against normal erythrocyte surface antigens (primary IMHA) or against altered erythrocyte surface antigens (secondary IMHA).
- Cause of secondary IMHA include:
  - Neoplasia (especially lymphoma and hemangiosarcoma)
  - Drugs: sulphonamides, cephalosporins, methimazole, recombinant erythropoietin
  - Tick-borne disease: ehrlichiosis, babesiosis, mycoplasmosis, cytauxzoonosis
  - Bacterial infection: leptospirosis, bacterial endocarditis
  - Viral disease: FeLV, FIV, FIP
  - Heartworm disease
- Antibody or complement deposition on erythrocyte membranes results in either direct intravascular hemolysis or accelerated extravascular removal of the erythrocyte by the mononuclear phagocytic system in the spleen, liver, and/or bone marrow. Extravascular hemolysis is most common.
- Autoagglutination of erythrocytes is more likely when autoantibodies of two immunoglobulin isotypes are present.

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- The anemia of IMHA is usually regenerative. It may be nonregenerative early in the course of disease (2–5 days), if concurrent disease (e.g., renal failure) blunts regeneration, or if the targeted cell is an erythrocyte precursor, and results in the destruction of immature erythrocytes in the marrow.
- IMHA can occur alone or in combination with other immune-mediated disease (IMT, SLE).
- IMHA is relatively common in dogs; in many cases, an underlying or predisposing cause is not identified. It is uncommon in cats, and is usually secondary to underlying disease.

Systems Affected

- Hemolymphatic: destruction or removal of erythrocytes resulting in anemia, production and release of proinflammatory mediators, intravascular tissue factor expression, hypercoagulability, DIC, thrombocytopenia, and thrombosis.
- Hepatobiliary: prehepatic icterus, hyperbilirubinemia, and bilirubinuria are common. Elevated liver enzymes can occur secondary to anemia.
- Cardiovascular: tachycardia due to hypoxia; murmurs can occur as a result of decreased blood viscosity and turbulent blood flow; anemia may precipitate relative volume overload, particularly in cats.
- Respiratory: hypoxia, tachypnea secondary to anemia or pulmonary thromboembolism.
- Renal/urinary: pigmenturia with intravascular hemolysis; kidney injury may result from hypoxemia or possibly from severe hemoglobinemia.
- Skin: pallor, icterus.



SIGNALMENT/HISTORY

Breed Predilections

- Dogs: Cocker spaniel, English springer spaniel, poodle, Irish setter, collie, old English sheepdog, miniature Schnauzer; any breed
- Cats: None reported

Age

- Dogs: mean 5–6 years, range 1–13 years
- Cats: mean 2–3 years, range 0.5–9 years

Sex

- Neutered female dogs and male cats may have increased risk.

Risk Factors/Causes

- Recent drug administration (3–10 days).
- Evidence for vaccine association is limited.
- Associated underlying diseases.

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Historical Findings

- Lethargy, depression, inappetence
- Weakness, exercise intolerance
- Pallor with or without icterus
- Pigmenturia (hemoglobinuria)
- Vomiting, diarrhea

CLINICAL FEATURES

- Pallor with or without icterus (Figs 53.1, 53.2)
- Depression, weakness
- Tachycardia
- Bounding pulses
- Tachypnea
- Pigmenturia
- Splenomegaly or hepatosplenomegaly
- Heart murmur or gallop
- Fever
- Signs of underlying disease, e.g., lymphadenopathy, cavitary effusion
- Signs of other concurrent immune-mediated disease, e.g., petechiae/ecchymoses, mucocutaneous lesions, chorioretinitis, arthritis



Fig. 53.1. Icteric mucous membranes of a dog with immune-mediated hemolytic anemia.

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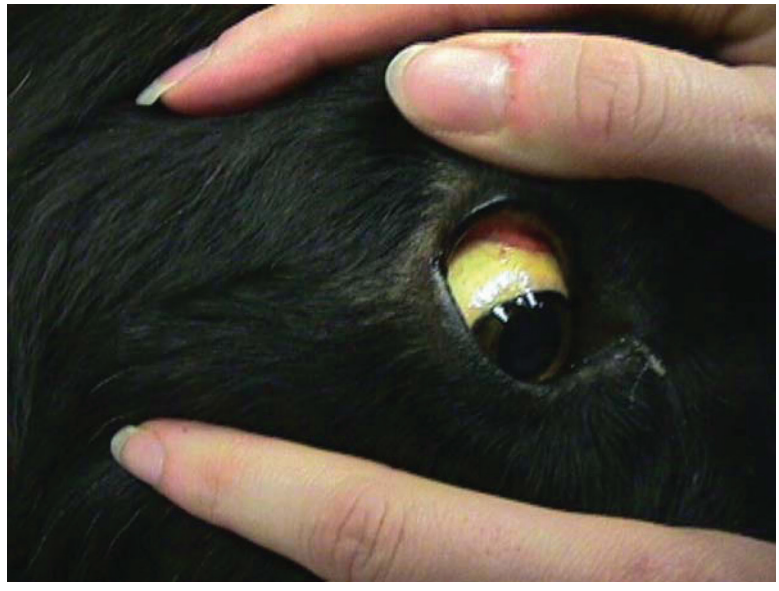


Fig. 53.2. Icteric sclera of a dog with immune-mediated hemolytic anemia.

DIFFERENTIAL DIAGNOSIS

- IMHA must be differentiated from other causes of hemolytic anemia.

Toxic Hemolysis

- Zinc toxicity
- Oxidative injury: acetaminophen, *Allium* spp., e.g., onions, garlic, propylene glycol, snake venom, methylene blue, cephalosporins, vitamin K, benzocaine, naphthalene (mothballs), skunk musk
- Copper toxicosis

Erythroparasites

- Babesiosis (*Babesia* spp.)
- Ehrlichiosis (*Ehrlichia* spp.)
- Hemobartonellosis (*Mycoplasma haemofelis*, *Haemobartonella canis*)
- Cytauxzoonosis (*Cytauxzoon felis*)

Microangiopathic Hemolysis

- DIC
- Splenic torsion
- Hemangiosarcoma
- Caval syndrome (dirofilariasis)
- Vasculitis

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Inherited

- Pyruvate kinase deficiency
- Phosphofructokinase deficiency
- B_{12} deficiency
- Osmotic fragility syndrome
- Nonspherocytic hemolytic anemia of beagles

Miscellaneous

- Hypophosphatemia
- Iatrogenic changes in osmolarity
- Histiocytic neoplasia
- Snake envenomation



DIAGNOSTICS

Complete Blood Count/Reticulocyte Count

- Anemia: usually moderately to markedly regenerative; may be nonregenerative.
- Spherocytosis in dogs.
- Anisocytosis, polychromasia, macrocytosis, nucleated red blood cells – can be seen if regenerative anemia.
- Neutrophilia, monocytosis.
- Macroscopic and/or microscopic agglutination may be present (Fig. 53.3).

Biochemical Profile

- Hyperbilirubinemia or hemoglobinemia
- Elevated hepatic enzymes
- Hypoalbuminemia
- Increased urea or creatinine

Urinalysis and Culture

- Hemoglobinuria or bilirubinuria
- Urine culture will be negative in primary IMHA

In-Saline Agglutination Test

- Agglutination persists after addition of 1–2 drops of normal saline to one drop of fresh or EDTA-anticoagulated blood on a glass slide.
- A negative test does not exclude a diagnosis of IMHA.

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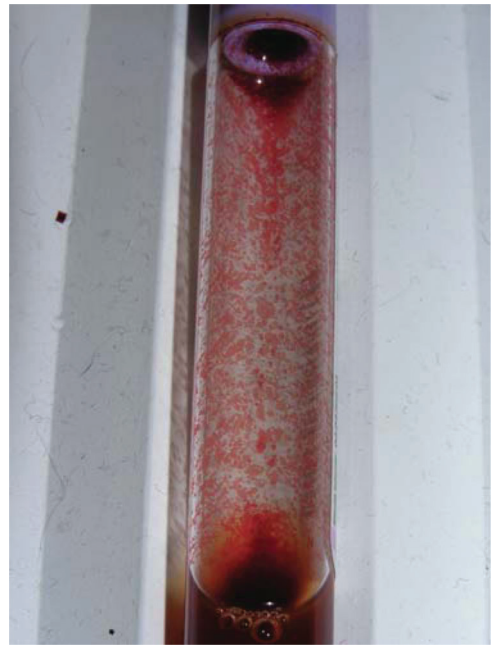


Fig. 53.3. Macroscopic agglutination observed from same dog with immune-mediated hemolytic anemia (IMHA). Macroscopic agglutination should increase an index of suspicion for IMHA, but it can also be present due to severe rouleaux formation. Therefore, the saline agglutination test must be performed and evaluated for microscopic agglutination.

Direct Antiglobulin Test (DAT) (Coombs test)

- Detects the presence of autoantibodies or complement on erythrocyte surface.
- Sensitivity of the DAT in IMHA is approximately 60% (range 37–89%).
- Specificity of the DAT in IMHA is good but a negative test does not exclude the disease.
- False-positive tests can occur with previous transfusions, as well as some infectious diseases, drugs, or neoplasia.

Coagulation Testing

- Platelet count, PT, PTT, D-dimers are often normal.
- Marked thrombocytopenia may indicate concurrent IMT.
- Mild-to-moderate thrombocytopenia, elevated D-dimers, or elevated PTT may indicate consumption (DIC or thrombosis).
- Thromboelastography typically suggests hypercoagulability but may provide prognostic information if relative hypocoagulability exists.

Imaging

- Thoracic and abdominal radiography, abdominal ultrasonography.
- Rule out presence of metallic foreign objects.
- Rule out underlying neoplastic process, e.g., lymphoma, hemangiosarcoma.

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- Splenomegaly or hepatosplenomegaly may be visible and nonspecific.
- Thorax usually normal; may be suggestive of PTE.

Bone Marrow Evaluation

- Indicated if nonregenerative anemia, or concurrent leukopenia or pancytopenia.
- Usually reveals erythroid hyperplasia; if anemia nonregenerative, may reveal maturation arrest or erythroid hypoplasia.
- Erythrophagocytosis sometimes evident.

Other Diagnostic Testing

- Aimed at ruling out differential diagnoses and identifying (or ruling out) an underlying cause for IMHA.
- FNA or other appropriate biopsy of mass lesions or enlarged lymph nodes.
- Serology and PCR: dogs – ehrlichiosis, babesiosis, leptospirosis; cats – FeLV, FIV, *Mycoplasma haemofelis*, with or without ehrlichiosis.
- Bacterial culture of any potential infections.



THERAPEUTICS

- The objectives of therapy are:
  - Support patient through the anemic crisis with transfusion therapy as required
  - Suppression of immune-mediated hemolysis, and reversal of anemia
  - Treatment of underlying disease, if present
  - Prevention of catastrophic consequences (e.g., thromboembolism)
  - Gradual weaning of therapy over subsequent months such that remission is sustained
- Inpatient care is indicated in the acute hemolytic state, where transfusion therapy is required, if the patient is unable to tolerate oral medication, is severely thrombocytopenic (<50 000 platelets/μL), or develops complications such as PTE, DIC, or heart failure.
- Inpatient care should focus on optimizing perfusion, oxygen delivery, acid–base status and hydration.
- Minimize stress.
- Minimize risk of thrombosis through use of thromboprophylactic drugs and minimizing additional risk factors, including use of minimally thrombogenic catheters.
- Restrict venipuncture and minimize sample volumes in small patients.
- Be cautious regarding the risk of volume overloading in patients with severe anemia.

Immunosuppressive Drugs of Choice

Glucocorticoids

- Prednisolone: dogs 1–2 mg/kg PO every 12 hours, cats 1–4 mg/kg PO every 12 hours. Once hematocrit is normal (typically after 3–4 weeks), begin to taper dose by 20–25%. If hematocrit remains stable, continue to taper by 20–25% every 3–4 weeks, to lowest effective dose.

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- Dexamethasone (0.3–0.5 mg/kg IV or SQ every 24 hours) is an alternative to prednisolone if oral medication is not tolerated.
- In cases that relapse with dose reduction of glucocorticoids, the clinician should consider changing to, or adding, another immunosuppressive agent.

Adjunct Immunosuppressives

- Adjunctive immunosuppressive therapy is generally recommended early in management in patients with intravascular hemolysis, autoagglutination, or as additional therapy in those refractory to glucocorticoid therapy (inadequate response by 5–7 days) or where steroid-related side effects necessitate dose reduction.

Dogs

- There is little evidence to support the choice of any one agent over another.
- Azathioprine (2 mg/kg PO every 24 hours). Decrease frequency to every 48 hours after glucocorticoid discontinuation, or if a decrease in white blood cell count. Note: may take 2–4 weeks to effect.
- Cyclosporine (5 mg/kg PO every 12–24 hours).
- Mycophenolate mofetil (10–20 mg/kg PO every 12–24 hours).

Cats

- Cyclosporine (5 mg/kg PO every 12 hours)

Blood Transfusion

- Indicated if anemic crisis or significant signs referable to anemia.
- Decision to transfuse should be based on patient's clinical signs, not merely PCV.
- In dogs: volume required (mL) = BW (kg) × 1.5 × desired change in PCV (%).
- Aim for PCV of 24–28% post transfusion.
- Packed red blood cells (PRBCs) are the preferred choice.
- If PRBCs are not available, fresh whole blood is an acceptable alternative.
- *All* cats *must* be blood typed or cross-matched prior to blood transfusion.
- It is preferable to blood type dogs prior to blood transfusion but not essential.
- Cross-matching should be performed with any subsequent transfusions in dogs and cats.
- Immunochromatographic blood typing cartridges (www.alvedia.com) enable blood typing to be performed where autoagglutination is occurring.
- Cross-matching cannot be performed where autoagglutination is occurring.

Thromboprophylaxis (Dogs)

- Aspirin (0.5 mg/kg PO every 24 hours)
- Clopidogrel (1–2 mg/kg PO every 24 hours)
- Individually dose-adjusted unfractionated heparin (see Helmond *et al.* 2010)
- Dalteparin (150 IU/kg SQ every 8 hours – dogs)

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- Enoxaparin (1 mg/kg SQ every 12 hours – cats)
- If thromboembolism present: low molecular weight heparin or heparin CRI protocol

Gastroprotectants

- Proton pump inhibitors (e.g., omeprazole, pantoprazole) orally or parenterally should be considered if gastrointestinal ulceration is suspected.
- Famotidine (0.5 mg/kg PO every 12 hours) can be considered for ongoing therapy while on high-dose glucocorticoids.

Intravenous Fluid Therapy

- If indicated for dehydration or hypovolemia.

Precautions/Interactions

- Avoid medications associated with secondary IMHA or bone marrow suppression.
- Do not use azathioprine in cats.
- If using azathioprine, monitor liver enzymes and CBC regularly.

Alternative Drugs

- Intravenous human immunoglobulin (0.5–1.5 g/kg IV infusion over 6–12 hours) has been suggested for patients with refractory IMHA, but a randomized controlled trial failed to identify any benefit of this intervention.
- Adjunctive therapeutic plasmapheresis has been successfully performed in rare cases.

Activity

- Enforced restricted activity until stable.

Surgical Considerations

- Splenectomy may be considered for cases refractory to medical management or where a splenic mass lesion is suspected to be an underlying cause. Splenectomy in the absence of a mass lesion is generally reserved for patients that fail to achieve sustained remission following multiple combination immunosuppressive regimens.



COMMENTS

Client Education

- IMHA and associated complications may be fatal.
- Successful treatment is possible in up to 75% cases.
- The patient will need to be on medication for months.

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- Lifelong treatment is necessary in some cases.
- Relapses and recurrences are possible.
- Regular recheck visits will be necessary, adding to the financial and time commitment.
- There are likely to be clinical adverse effects of corticosteroids; these will diminish as the dose is decreased, and will abate when drug is discontinued.
- Appropriate precautions for immunosuppressed patients.
- Monitor for signs of recurrent anemia, thromboembolism (e.g., tachypnea, respiratory distress), or adverse drug reactions (e.g., inappetence, lethargy, melena, pollakiuria).
- Drugs associated with secondary IMHA should be avoided.

Patient Monitoring

Inpatient Monitoring

- Signs of anemia: tachycardia, bounding pulses, tachypnea, decreasing PCV
- Signs of thromboembolism: tachypnea, respiratory distress, abdominal effusion
- Adverse effects of therapy (e.g., volume overload, transfusion reaction, secondary infection)
- PCV every 4–12 hours, depending on rate of change

Outpatient Monitoring

- CBC weekly until normal, then every other week for 2–3 months, then monthly until 3 months following discontinuation of drugs. If any evidence of relapse, increase frequency.
- Azathioprine: CBC as noted, chemistry profile (liver parameters) monthly or if concerns.
- Cyclosporine: trough drug concentration should be assessed if inadequate response or suspicion of toxicity.

Possible Complications

- Refractory anemia
- Thromboembolic disease (pulmonary or other organ)
- DIC
- Infection secondary to immunosuppression
- Gastrointestinal ulceration due to glucocorticoid therapy in dogs
- Diabetes mellitus secondary to glucocorticoid therapy in cats
- Azathioprine: potential for myelosuppression or hepatotoxicity
- Transfusion reaction
- Acute kidney injury
- Death

Expected Course and Prognosis

- Initial response (slowing of hemolysis, increase in PCV) is usually seen within 7 days; normalization of PCV may take weeks.
- Response is significantly slower in patients with nonregenerative anemia; it does not warrant a poorer prognosis.

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- A poorer prognosis is associated with intravascular hemolysis, serum bilirubin concentration >5 mg/dL, hypoalbuminemia, increased urea or creatinine, coagulation abnormalities.
- Mortality varies with severity of disease, and ranges from 25% to 60%.
- Most deaths occur within the first 2 weeks post diagnosis.
- Thromboembolism is a significant potential consequence, with reported prevalence of 50–80% in nonsurvivors.
- Patients that survive the acute crisis and leave the hospital have a relatively good prognosis.
- Patients need to be on immunosuppressive therapy for months.
- Relapse can occur with tapering of therapy, and is an indication for adjustment of the immunosuppressive therapy protocol.
- Recurrence is possible.

Synonyms

- Immune-mediated anemia
- Autoimmune hemolytic anemia (AIHA)

Abbreviations

BW: body weight
CBC: complete blood cell count
CRI: continuous rate infusion
DAT: direct antiglobulin test
DIC: disseminated intravascular coagulation
EDTA: ethylene diamine tetraacetic acid
FeLV: feline leukemia virus
FIP: feline infectious peritonitis virus
FIV: feline immunodeficiency virus
FNA: fine needle aspiration
GI: gastrointestinal
IgG: immunoglobulin G
IgM: immunoglobulin M
IMHA: immune-mediated hemolytic anemia
IMT: immune-mediated thrombocytopenia
IU: international unit
IV: intravenous
PCR: polymerase chain reaction
PCV: packed cell volume
PO: by mouth (per os)
PRBC: packed red blood cells
PT: prothrombin time

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PTE: pulmonary thromboembolism
PTT: partial thromboplastin time
SLE: systemic lupus erythematosus
SQ: subcutaneous

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chapter 54

Immune-Mediated Thrombocytopenia

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DEFINITION/OVERVIEW

- An immune-mediated disease that results from the development of antiplatelet antibodies that accelerate destruction and/or removal of platelets from circulation. IMT can be primary (idiopathic) or secondary to medication or disease.
- Diagnosis is aimed at the elimination of other potential causes for thrombocytopenia, and the identification of underlying disease.
- Morbidity and mortality are related to bleeding; this can be massive and occult, into the gastrointestinal tract or into a vital organ (e.g., brain, lungs).
- Immunosuppression is the mainstay of therapy. Additional therapies include blood product transfusion, supportive care, and treatment of any underlying condition.
- Response to treatment is generally rapid. If the patient survives the initial period until platelet count increases, long-term prognosis is relatively good.

ETIOLOGY/PATHOPHYSIOLOGY

- IgG antibodies bind to platelet surface glycoproteins (especially GPIIb/IIIa – the fibrinogen receptor) and result in accelerated platelet destruction and removal.
- Primary (idiopathic) IMT results from antibodies directed against normal platelet surface antigens. This can occur alone or in combination with other immune-mediated disease (i.e., IMHA, SLE).
- Secondary IMT occurs when antibodies target nonself antigens that have been adsorbed onto the platelet surface or when immune complexes bind to platelet surface. Causes include:
    - Tick-borne disease (ehrlichiosis), Rocky Mountain spotted fever, bartonellosis
    - Viral disease: FeLV, FIV
    - Neoplasia (especially lymphoma)
    - Drugs: potentiated sulfonamides
    - Live virus vaccination
    - Heartworm disease
    - Chronic bacterial infection

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Companion website: www.fiveminutevet.com/emergency

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- Significant thrombocytopenia (<30 000–50 000/μL) results in spontaneous bleeding, particularly in the skin and from mucosal surfaces.
- IMT is relatively common in dogs. It is uncommon in cats, and is usually secondary to underlying disease (often viral).
- Secondary IMT may have a higher incidence in areas where associated infectious diseases are endemic.

Systems Affected

- Hemic/lymphatic/immune: destruction or removal of platelets (thrombocytopenia), anemia (if bleeding or concomitant IMHA), and production and release of proinflammatory mediators may result in DIC.
- Cardiovascular: failure of primary hemostasis results in bleeding, usually manifested as petechiae or ecchymoses, especially of skin and mucosal surfaces.
- Skin: petechiae/ecchymoses (Figs 54.1, 54.2).
- Gastrointestinal: hemorrhage, hematemesis, melena.
- Urologic: urinary hemorrhage/hematuria.
- Respiratory: pulmonary hemorrhage, respiratory difficulty, cough, hemoptysis, epistaxis.
- Eyes: scleral or retinal hemorrhage, hyphema (Fig. 54.3).
- Nervous: intracranial hemorrhage, seizures.

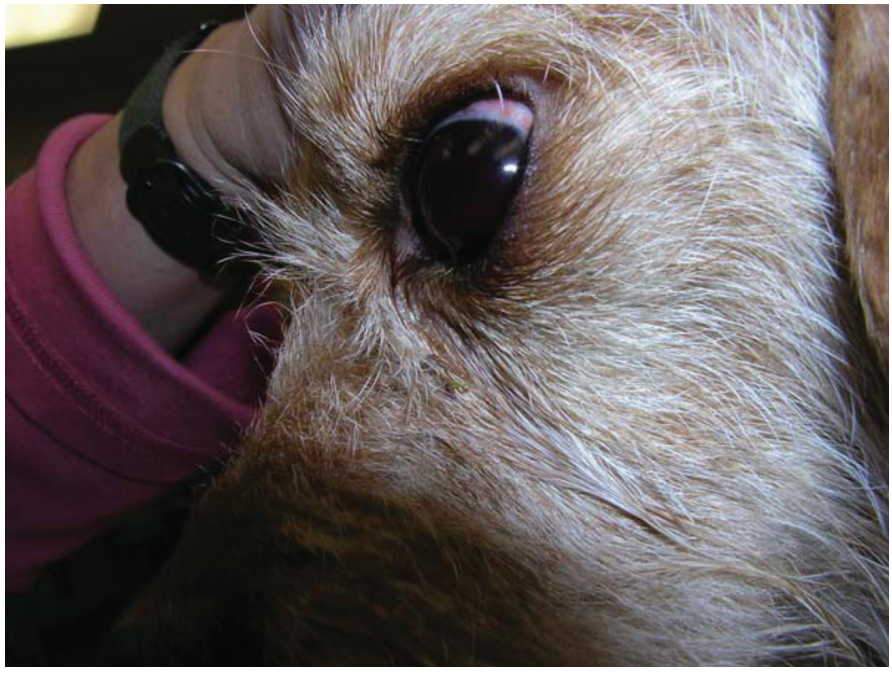


Fig. 54.1. Petechial hemorrhages on the gums of a dog with immune-mediated thrombocytopenia.

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■ **Fig. 54.2.** Severe petechiae on the ventral abdomen of a dog with immune-mediated thrombocytopenia.



■ **Fig. 54.3.** Scleral hemorrhage in a dog with thrombocytopenia.

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SIGNALMENT/HISTORY

Breed Predilections

- Dogs: Cocker spaniel, poodle, Old English sheepdog, Rottweiler, German shepherd dog have increased incidence.
- Cats: none reported.

Age

- Dogs: mean 4–8 years, range 0.5–17 years
- Cats: mean 2–3 years, range 0.5–9 years

Sex

- Female dogs have increased risk.

Risk Factors/Causes

- Recent drug administration (3–10 days)
- Limited evidence for vaccine association
- Associated underlying diseases

Historical Findings

- Bleeding that is apparent to the owner (e.g., petechiae, epistaxis, melena, pigmenturia)
- Lethargy, depression, inappetence, weakness
- Bleeding that is not apparent (e.g., blindness due to hyphema)



CLINICAL FEATURES

- Cutaneous or mucosal petechiae or ecchymoses
- Hyphema, scleral hemorrhage
- Epistaxis
- Melena, hematochezia
- Hematuria
- Hemorrhagic, hypovolemic shock
- Tachypnea, adventitious lung sounds
- CNS signs, including changes in consciousness and localized deficits



DIFFERENTIAL DIAGNOSIS

- A markedly decreased platelet count (<30 000/μL) in the absence of other cell line anomalies (nonregenerative anemia, leukopenia) is suggestive of IMT, but other potential causes must also be excluded. IMT must be differentiated from the following.

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Decreased Platelet Production

- Pure megakaryocytic hypoplasia (platelets only): vaccination (i.e., panleukopenia), viral (i.e., FeLV, FIV), estrogen toxicity, phenobarbital toxicity.
- Pancytopenia (including other blood cell lines): myelophthisis, myelofibrosis, hematopoietic neoplasia, infectious disease (i.e., FeLV, FIP, FIV, chronic ehrlichiosis, sepsis), or drug associated (i.e., chemotherapy, griseofulvin, methimazole, albendazole).
- Dysthrombopoiesis: myelodysplastic syndromes.

Increased Platelet Consumption or Sequestration

- DIC
- Profound, acute hemorrhage
- Splenic torsion
- Vasculitis
- Microangiopathy: hemangiosarcoma, splenic disease

Nonimmune Platelet Destruction

- Viral: FeLV, FIV, FIP, parvovirus
- Rickettsial, protozoal: ehrlichiosis, hemobartonellosis, cytauxzoonosis
- Bacterial: bacteremia, leptospirosis, salmonellosis
- Fungal: histoplasmosis, disseminated candidiasis
- Snake envenomation

Normal Breed Variation

- Low platelet counts can be normal in greyhounds, Cavalier King Charles spaniels, and Shiba Inus.

Spurious

- Platelet clumping due to errors in collection or handling will result in artificially low platelet counts.



DIAGNOSTICS

Platelet Count

- Should be performed in any patient with a possible bleeding disorder.
- Counts can be performed manually (hemocytometer) or via automated counters in dogs.
- Automated platelet counting may be inaccurate in cats; platelet counts must be verified manually.

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Blood Smear Evaluation

- Indicated where platelet count is not immediately available and to verify findings of platelet counts.
- The smear should be carefully examined for platelet clumps, which preclude an accurate count.
- A platelet count can be estimated by multiplying the average platelet count per high power field (100×) by 15 000.
- Large platelets are suggestive of regeneration in dogs.

Complete Blood Count

- Thrombocytopenia invariably present; usually marked (<30 000/μL).
- Anemia may be present if bleeding or concurrent IMHA. Usually regenerative; may be nonregenerative if early (<3–5 days) or concurrent disease precludes regeneration.
- Neutrophilia sometimes present.

Biochemical Profile

- No specific abnormalities; abnormalities may reflect underlying disease.

Coagulogram (PT, aPTT, D-Dimers)

- Should be performed to rule out consumptive coagulopathy.
- Normal with IMT, unless bleeding severe.

Thoracic Radiographs

- Indicated if respiratory compromise, or if potential for underlying disease.
- No specific abnormalities; may be suggestive of pulmonary hemorrhage or underlying disease, e.g., neoplasia.

Bone Marrow Evaluation

- Not necessary in patients where IMT is the most likely diagnosis since the marrow evaluation may not provide additional diagnostic or prognostic information.
- Megakaryocytic hyperplasia indicates platelet destruction and is typical (but not pathognomonic) for IMT. Megakaryocytic hypoplasia (less common) can occur in some cases of IMT if antibodies are directed against early precursors.
- Indicated with refractory thrombocytopenia, if multiple cell lines affected (i.e., leukopenia, nonregenerative anemia), or if atypical cells are evident on blood smear cytology.

Other Laboratory Tests

- Other diagnostic testing should be aimed at ruling out differential diagnoses and identifying (or ruling out) an underlying cause for IMT.
    - Abdominal ultrasonography
    - Serology for viral or tick-borne disease. Dogs: ehrlichiosis, bartonellosis, Rocky Mountain spotted fever. Cats: FeLV, FIV, with or without ehrlichiosis
    - Bacterial culture of any potential infections

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THERAPEUTICS

- Support the patient through the bleeding crisis with transfusion therapy as required.
- Prevention of catastrophic hemorrhage using platelet transfusions (platelet concentrates, platelet-rich plasma, fresh whole blood or DMSO-stabilized frozen platelets). Pharmacologic interventions (e.g., desmopressin, ε-ACA) might be tried as a last resort.
- Suppression of the immune system and thus the immune-mediated platelet destruction, such that platelet numbers normalize.
- Treatment of underlying disease, if present.
- Gradual weaning of therapy over subsequent months such that remission is sustained.
- Inpatient care is indicated with severe thrombocytopenia (<50 000/μL), active bleeding, inability to tolerate oral medication, or development of complications (pulmonary or CNS hemorrhage).
- Restrict venipuncture, and avoid phlebotomy from central veins.
- Minimize risk of bleeding through careful handling.
- Outpatient care when risk of bleeding is decreased (platelet count >50 000/μL), anemia is stabilized, and oral medications are tolerated.

Immunosuppressive Drug of Choice

Glucocorticoids

- Prednisolone. Dogs: 1–2 mg/kg PO every 12 hours. Cats: 1–4 mg/kg PO every 12 hours. Once platelet count is normal (>200 000/μL), typically after 3–4 weeks, begin to taper dose by 20–25%. If count remains stable, continue to taper by 20–25% every 3–4 weeks, to lowest effective dose.
- Dexamethasone (0.3–0.5 mg/kg IV or SQ every 24 hours) is an acceptable alternative to prednisolone if oral medication is not tolerated.
- Adjunctive immunosuppressive therapy should be considered in cases refractory to glucocorticoid therapy – if inadequate response by 5–7 days, or if platelet count decreases with attempts to wean glucocorticoids.

Intravenous Human Immunoglobulin

- Intravenous human immunoglobulin (0.5–1.5 g/kg IV infusion over 4–6 hours) has been demonstrated to significantly shorten the time to restoration of a normal platelet count in IMT in dogs.
- Infusion can be repeated, but only within 72 hours of the original administration since the product is antigenic.
- This product is not used for sustained immunosuppression and must be administered together with glucocorticoids with or without adjunctive immunosuppressive therapy.

Vincristine

- Vincristine (0.02 mg/kg strictly IV) has been demonstrated to significantly shorten the time to restoration of a normal platelet count in IMT in dogs.

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- Vincristine should only be administered once in the treatment of IMT.
- Patients must be monitored for myelosuppression following vincristine administration.
- This product is not used for sustained immunosuppression and must be administered together with glucocorticoids with or without adjunctive immunosuppressive therapy.

Adjunct Immunosuppressives (Dogs)

- There is little evidence to support the choice of any one agent over another.
- Azathioprine: 2 mg/kg PO every 24 hours. Decrease frequency to every 48 hours after glucocorticoid discontinuation, or if a decrease in white blood cell count. Note: May take 2–4 weeks to effect.
- Cyclosporine: 5 mg/kg PO every 12–24 hours.
- Mycophenolate mofetil: 10–20 mg/kg PO every 12–24 hours.

Adjunct Immunosuppressives (Cats)

- Cyclosporine: 5 mg/kg PO every 12 hours.

Gastroprotectants

- Proton pump inhibitors (e.g., omeprazole, pantoprazole) orally or parenterally should be considered if gastrointestinal ulceration is suspected.
- Famotidine (0.5 mg/kg PO every 12 hours) can be considered for ongoing therapy while on high-dose glucocorticoids.

Intravenous Fluid Therapy

- If indicated for dehydration or hypovolemia.

Blood Transfusion

- Indicated if anemic crisis or significant signs referable to anemia.
- Decision to transfuse should be based on patient's clinical signs, not merely PCV.
- In dogs: volume required (mL) = BW (kg) × 1.5 × desired change in PCV (%).
- Aim for PCV of 24–28% post transfusion.
- Packed red blood cells (PRBCs) are the preferred choice.
- If PRBCs are not available, fresh whole blood is an acceptable alternative.
- *All* cats *must* be blood typed or cross-matched prior to blood transfusion.
- It is preferable but not essential to blood type dogs prior to blood transfusion.
- Cross-matching should be performed with any subsequent transfusions in dogs and cats.

Precautions/Interactions

- Avoid medications associated with secondary IMT or bone marrow suppression.
- Avoid drugs that inhibit platelet function (e.g., NSAIDs).
- Do not use azathioprine in cats.

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- Vincristine should only be used following diagnostic work-up for underlying neoplasia.
- Complete blood counts should be monitored following vincristine administration.
- Vincristine must be given strictly IV; extravasation is extremely caustic.

Activity

- Enforce restricted activity until platelet count approaches normal range.

Surgical Considerations

- Splenectomy may be considered for cases refractory to medical management or where a splenic mass lesion is suspected to be an underlying cause. Splenectomy in the absence of a mass lesion is generally reserved for patients that fail to achieve sustained remission following multiple combination immunosuppressive regimens.



COMMENTS

Client Education

- IMT and associated complications can be fatal, particularly in the acute period until the platelet count approaches normal. If the patient survives this period, prognosis is good.
- The patient will be on medication for months. In some cases, lifelong treatment is necessary.
- Relapses and recurrences are possible.
- Regular recheck visits will be necessary, adding to the financial and time commitment.
- There are likely to be clinically significant adverse effects of corticosteroids; these will diminish as the dose is decreased, and will abate when drug is discontinued.
- Use appropriate precautions for immunosuppressed patients.
- Monitor for signs of recurrent bleeding or adverse drug reactions (e.g., inappetence, lethargy, melena, pollakiuria).
- Drugs associated with secondary IMT should be avoided.

Patient Monitoring

Inpatient Monitoring

- Signs of bleeding: respiratory rate and effort, mentation, stool, urine.
- Signs of anemia: tachycardia, bounding pulses, tachypnea, decreasing PCV.
- Adverse effects of therapy (e.g., volume overload, transfusion reaction).
- Platelet count every 48 hours until >50 000/μL.
- Complete blood counts following vincristine administration.

Outpatient Monitoring

- Platelet count: weekly until normal, then every other week for 2–3 months, then monthly until 3 months following discontinuation of drugs. If any evidence of relapse, increase frequency of counts.

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- CBC: performed with platelet counts if other cell lines affected, and if the patient is on azathioprine therapy.
- Azathioprine: CBCs as noted, chemistry profile (liver parameters) monthly or if concerns.
- Cyclosporine: trough drug concentration should be assessed if inadequate response or suspicion of toxicity.

Possible Complications

- Refractory thrombocytopenia
- Massive hemorrhage and hemorrhagic shock
- CNS hemorrhage
- Pulmonary hemorrhage
- Thromboembolism or DIC (rare, unless concomitant IMHA)
- Infection secondary to immunosuppression
- Gastrointestinal ulceration due to glucocorticoid therapy in dogs
- Diabetes mellitus secondary to glucocorticoid therapy in cats
- Vincristine: potential for myelosuppression
- Azathioprine: potential for myelosuppression and/or hepatotoxicity or pancreatitis
- Transfusion reaction

Expected Course and Prognosis

- Partial remission (platelet count >50 000/μL) generally occurs within 2–7 days.
- Complete remission (platelet count >200 000/μL) generally takes 1–3 weeks.
- Negative prognostic indicators include intracranial and intrapulmonary hemorrhage, and underlying neoplasia.
- Severity of thrombocytopenia is not a prognostic indicator.
- Initial response rates are 80–95 of cases.
- Mortality is reported to be 10–40%, with 10% more typical.
- Patients that survive the period from presentation to partial remission have a good prognosis.
- Transient thrombocytosis is not unusual following therapy, and likely poses minimal risk.
- Patients need to be on immunosuppressive therapy for months.
- Relapse can occur with tapering of glucocorticoid therapy, and is an indication for adjunctive immunosuppressive therapy.
- Relapse occurs in approximately 10% of cases.

Synonyms

- Idiopathic thrombocytopenic purpura
- Autoimmune thrombocytopenia

Abbreviations

aPTT: activated partial thromboplastin time
BW: body weight

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CBC: complete blood count
CNS: central nervous system
DIC: disseminated intravascular coagulation
FeLV: feline leukemia virus
FIP: feline infectious peritonitis
FIV: feline immunodeficiency virus
IgG: immunoglobulin G
IMHA: immune-mediated hemolytic anemia
IMT: immune-mediated thrombocytopenia
IV: intravenous
NSAID: nonsteroidal antiinflammatory drug
PCV: packed cell volume
PO: by mouth
PT: prothrombin time
PTT: partial thromboplastin time
SLE: systemic lupus erythematosus
SQ: subcutaneous

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chapter 55

Intussusception

DEFINITION/OVERVIEW

- Gastrointestinal intussusception is the telescoping or prolapsing of one portion of the gastrointestinal tract into the lumen of an adjoining part of intestine or stomach. This occurs most commonly in the jejunum (Fig. 55.1) or at the ileocolic junction, although gastroesophageal or pylorogastric intussusceptions are also possible. The parts of a typical intussusception are the proximal (orad) intussusceptum, the prolapsed portion, and the distal (aborad) intussuscipiens, the receiving portion. Reverse and multiple intussusceptions may also occur. Sliding intussusceptions (those that occur and then reduce spontaneously) have also been reported.

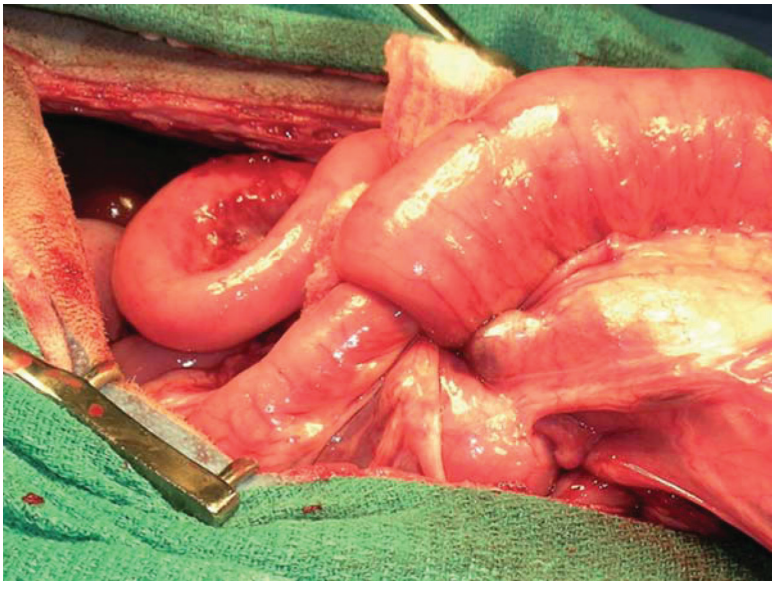


Fig. 55.1. A jejunojejunal intussusception in a 10-week-old Weimaraner.

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ETIOLOGY/PATHOPHYSIOLOGY

- Intussusceptions are often associated with gastroenteritis attributed to intestinal parasitism, focal and linear foreign bodies, intestinal mass, dietary indiscretion, or viral infection. Previous abdominal surgery has also been suspected as a causative factor, although in the majority of cases of intussusception the cause is unknown.

Systems Affected

- Gastrointestinal: intussusceptions may cause a complete or partial obstruction and result in vomiting, melena, or hematochezia. Inappetence and weight loss may occur with chronic or sliding intussusceptions. The tissue involved in the intussusception may become devitalized and result in gastrointestinal necrosis and leakage into the abdominal cavity.
- Cardiovascular: intussusceptions that result in complete gastrointestinal obstruction, breakdown of the mucosal barrier, or wall necrosis have a high likelihood of causing hypovolemic and septic shock.
- Respiratory: animals with acute gastroesophageal intussusception may present in severe respiratory distress due to the presence of the gas-distended stomach within the thoracic cavity or due to aspiration pneumonia from severe vomiting.



SIGNALMENT/HISTORY

- Intussusceptions occur in both dogs and cats and seem to predominantly affect younger animals less than 1 year of age. German shepherd dogs and Siamese cats may be overrepresented, although intussusceptions can occur in any breed. There does not appear to be a gender predisposition.
- Clinical signs associated with intussusception are influenced by location, completeness, and duration of obstruction. Acute intussusceptions have a history most consistent with an acute abdomen: vomiting, bloody diarrhea, and abdominal pain. Animals with chronic intussusceptions may have vaguer clinical signs: inappetence, weight loss, and diarrhea.
- Historical information may be valuable in aiding the diagnosis of intussusception because many animals will have a recent history of illness, abdominal surgery, or environmental change.



CLINICAL FEATURES

- Physical examination may reveal a guarded painful abdomen.
- Often an elongated large tubular structure can be palpated within the abdomen, representing a thickened loop of intestine.
- Occasionally, the intussusceptum may protrude through the anus, mimicking a rectal prolapse.

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DIFFERENTIAL DIAGNOSIS

- Focal gastrointestinal foreign bodies
- Linear gastrointestinal foreign bodies
- Intestinal volvulus
- Gastroenteritis

DIAGNOSTICS

- Survey abdominal radiographs: plain abdominal radiographs may have variable signs. A large soft tissue tubular structure may be visualized. Incomplete obstructions do not usually result in gas accumulation within the intestinal lumen. A barium enema or pneumocolonogram may help with diagnosis of ileocolic intussusceptions.
- Abdominal ultrasound: this modality is particularly useful in identifying intussusceptions. In a transverse plane, intussusceptions appear as a multilayered target-like image with alternating hyperechoic and hypoechoic concentric rings; in a longitudinal plane, there are alternating hyperechoic and hypoechoic parallel lines (Fig. 55.2). Although the "target sign" or "bulls-eye" is considered to be pathognomonic for intestinal intussusception, care must be taken to visualize the structure in multiple planes to avoid misdiagnosis. Healthy intestine, enteritis, and postpartum uterine involution can have an ultrasonographic target-like appearance in some imaging planes.
- Laboratory findings may include electrolyte abnormalities, acid–base disturbances, anemia, hypoalbuminemia, stress or inflammatory leukograms, and evidence of dehydration.

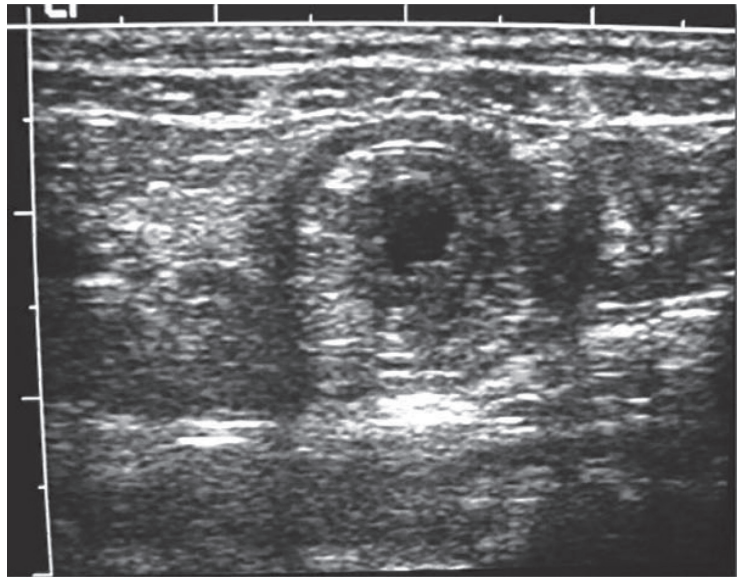


Fig. 55.2. A transverse ultrasound image of an ileocolic intussusception demonstrating the classic target sign appearance.

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THERAPEUTICS

- Although spontaneous reduction of intestinal intussusception is possible, surgical intervention is the treatment of choice. Prior to surgery, the animal's hemodynamic and electrolyte status should be stabilized, although surgery should not be delayed unnecessarily. Although intestinal resection may or may not be required, prophylactic antibiotics are indicated as the viability of the intestine is unknown and because there is increased infection risk with intestinal obstruction due to mucosal breakdown with potential bacterial overgrowth and translocation.

Surgical Considerations

- The objective of surgery is to reestablish a patent gastrointestinal tract either through reduction of the intussusception or by resection of the affected segment. Reduction of the intussusception should be attempted by placing gentle traction on the intussusceptum while gently pushing on the intussuscipiens. If reduced, the involved portion of the intestine is evaluated for perforations, necrosis, or other signs of poor viability. Subjective measures utilized to assess intestinal viability include color, thickness, peristalsis, and arterial pulsation. Resection and anastomosis have been reported to be necessary in over 80% of cases due to vascular compromise, devitalized tissue, intestinal perforation, and adhesions.
- Controversy exists over whether measures should be taken to prevent recurrence of intestinal intussusception. This is accomplished through enteroplication (or enteroenteropexy), which is the creation of permanent serosal adhesions between adjacent segments of small intestine (Fig. 55.3). To perform this procedure, gentle loops of small intestine from

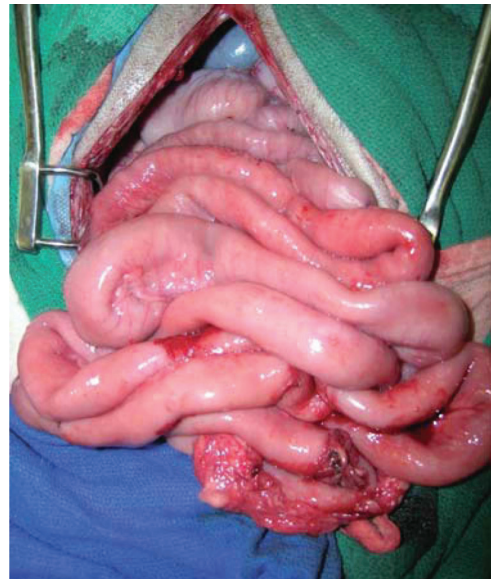


Fig. 55.3. Intraoperative photograph showing the small intestine gently looped in preparation for enteroplication.

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the duodenocolic ligament to the ileocolic junction are placed side by side and secured in this formation with variably spaced simple interrupted partial-thickness sutures using 4-0 absorbable monofilament material. Care must be taken to avoid abrupt angles of the intestinal loops to minimize the risk of postoperative intestinal obstruction. Both intussusception recurrence and severe postoperative complications associated with enteroplication have been reported in the dog and cat.



COMMENTS

Possible Complications

- The risk of possible complications from enteroplication must be weighed against the risk of possible recurrence. At this time, the only study investigating this problem found that there is similar likelihood (approximately 20%) between these two scenarios. Therefore, it is left to the discretion of the veterinarian whether enteroplication is to be performed.

Expected Course and Prognosis

- The prognosis for animals with intussusceptions is dependent on the location and completeness of the obstruction, as well as the duration of the condition. Rapid recognition and treatment contribute to improved survival. However, the prognosis worsens with intestinal perforation and peritonitis. Studies report successful outcomes in over 80% of cases.

Synonyms

- Intestinal invagination; intestinal telescoping

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Author: Catriona M. MacPhail, DVM, PhD

chapter 56

Ivermectin Toxicity

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DEFINITION/OVERVIEW

- Ivermectin and other macrolides, such as selamectin, dormectin, moxidectin, and milbemycin, are antiparasitic drugs that are effective against both external and internal parasites.
- In general, these drugs are commonly used, are very effective, and have wide margins of safety.
- Some collie-type breeds have a genetic mutation (ABCB1/MDR1) that causes them to develop neurotoxicity at lower doses.
- This discussion refers to ivermectin but applies to other similarly acting macrolides.

ETIOLOGY/PATHOPHYSIOLOGY

- Ivermectin and other similarly acting drugs bind to glutamate-gated chloride channels, causing hyperpolarization by enhancing the movement of chloride ions in the cell. This results in arthropod and nematode paralysis.
- Glutamate-gated chloride ion channels are not found in mammals.
- In mammals, ivermectin potentiates presynaptic release of GABA and postsynaptic binding of GABA. GABA is an inhibitor neurotransmitter.
- The multiple drug resistance gene, ABCB1 (MDR1), encodes for P-glycoprotein, a principal component of the blood–brain barrier. The mutation, mmdr1-1δ, results in loss of P-glycoprotein function, and thereby allows access to the brain by ivermectin and some other drugs that are also substrates of P-glycoprotein. Dogs with this mutant allele are susceptible to ivermectin toxicity at low doses.

Systems Affected

- The toxic effect is on the neurologic system; other organ systems are affected indirectly.

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SIGNALMENT/HISTORY

Genetic Basis

- Analysis of the history of the canine mutation shows that dogs carrying the mutant allele mmdr1-1δ are descendants of a dog that lived in Great Britain before 1873 and was one of the foundation dogs for a branch of working sheepdogs selected for show.
- Seven collie-type breeds and two sighthound breeds were identified as carrying this allele. These modern-day breeds are collie, Shetland sheepdog, old English sheepdog, McNab cattledog, Australian shepherd, miniature Australian shepherd, and English shepherd. Two other breeds of sighthound type but descended from the common ancestor are silken windhound and longhaired whippet.
- Within breeds, the frequency of the mutant allele varies from less than 4% in old English sheepdogs to more than 50% in collies.
- Ivermectin sensitivity has been reported in border collie, bearded collie, and Australian cattle dog. The mutant allele was not found in one study that included members of these breeds, indicating a low frequency of the allele or that another mutation is responsible.

Signs

- Toxicity is to the CNS with other systems affected secondarily.
- Ataxia.
- Tremors.
- Seizures.
- Coma.
- Death.

Historical Findings

- Ivermectin toxicosis should be considered in a dog belonging to a susceptible breed with a history of acute onset of neurologic signs combined with recent exposure to drugs in this group.
- In dogs with the mutant allele, doses of ivermectin of >100 µg/kg have caused toxicity (therapeutic heartworm prevention doses are well below this amount).
- In other breeds, toxicity occurs only with severe overdoses (>2000 µg/kg) usually caused by miscalculation using equine or bovine preparations.
- Exposure can also occur from dogs eating feces of treated large animals because the drug is eliminated in the feces.
- Young animals are more susceptible to toxicity because their blood–brain barrier is not fully developed.
- Toxicity has been reported in kittens at doses of 300–400 µg/kg.

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CLINICAL FEATURES

Physical Examination

- Ataxia or recumbency and altered mental state (e.g., depression, obtundation) are the primary neurologic signs with stupor, coma, and even death occurring in severely affected dogs.
- Those with depressed mentation may also have decreased gag reflex.
- Muscle tremors, seizures, and loss of menace and pupillary light response may occur.
- Spinal cord reflexes may be normal or increased.
- Other signs include bradycardia, mydriasis, miosis, blindness, hypersalivation, and vomiting.
- Clinical signs in dogs can range in severity, even with ingestion/exposure to similar doses.
- Onset of signs can be within a few hours after administration of a single dose.
- Animals being treated daily may develop signs of toxicity only after several days (accumulation in the body due to long half-life).



DIFFERENTIAL DIAGNOSIS

- Differentiation from other causes of acute-onset neurologic signs is based on a history of exposure to the drug in a susceptible canine breed or severe overdose in other animals.



DIAGNOSTICS

Laboratory Tests

- Ivermectin and other macrolides can be detected in serum, gastrointestinal contents, liver, and fat but levels in tissues do not correlate with clinical signs because it is the concentration of the drug in the brain that determines toxicity.

Other Tests

- Reversal of neurologic signs in response to the anticholinesterase physostigmine supports but does not confirm a diagnosis. Its use for this purpose is not recommended because of adverse side effects (see Therapeutics).
- A DNA-based test for the mutant genotype is available and could be used to confirm that a dog is at increased risk of toxicity.

Pathologic Findings

- No specific lesions are directly caused by these drugs.

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THERAPEUTICS

- The goals of therapy are decontamination and support.

Drugs of Choice

- There is no specific antidote.
- If the drug has been ingested in the past 30 minutes, emesis may be induced, provided that the patient is alert enough to protect its airway. If the patient is not able to protect its airway, orogastric lavage under general anesthesia with intubation could be performed.
- A dose of activated charcoal should be given if toxicosis is expected. Repeated doses of activated charcoal are indicated if symptomatic because of enterohepatic recirculation.
- Anticholinesterase drugs have been administered as a diagnostic test or for short-term improvement of clinical signs. Physostigmine may result in partial improvement of signs but the effect lasts less than 1 hour. The potential toxicities of physostigmine are tremors, seizures, ptyalism, lacrimation, urination, and defecation. It does not target ivermectin toxicity directly and is not recommended.
- Picrotoxin, a GABA antagonist, has also been used to treat ivermectin toxicity. Picrotoxin resulted in a rapid improvement in mental state followed by a seizure. Any improvement is likely to be transient, and it is not recommended because of the risk of adverse effects.

Supportive Care

- Patients should receive supportive care as appropriate for their clinical signs and must be closely monitored. Long-term intensive care nursing may be needed.
- Deterioration from the initial presenting signs can occur followed by gradual improvement over the next 6–18 days. Longer courses have been reported in sensitive animals.
- Special attention should be paid to the respiratory and cardiovascular systems because hypoventilation that requires mechanical ventilation and bradycardia that requires treatment with an anticholinergic agent such as glycopyrrolate have been reported.
- Aspiration pneumonia due to a combination of recumbency and a decreased gag reflex can occur.
- Lipid therapy (20% solution, 1.5 mL/kg bolus, followed by 0.25 mL/kg/min for 30–60 minutes, repeat in 4 hours if no lipemia) has had mixed results in ivermectin toxicosis. Some animals benefit from it, while others appear to receive little beneficial effect.
- Patients who are unable to eat and drink normally must receive fluid and electrolyte therapy and nutritional support. Oral intake of food or water should not be attempted in patients with altered mental states because they may have decreased gag reflexes and be unable to properly protect their airway. In these patients, nasogastric or gastric tube feeding is preferred but intravenous nutrition may be attempted.
- Severely affected patients are recumbent and meticulous care for the recumbent patient is needed.

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COMMENTS

Expected Course and Prognosis

- The prognosis for recovery without long-term sequelae is good even in severely affected patients, provided that aggressive intensive care support is administered.

Abbreviations

CNS: central nervous system
GABA: gamma-amino butyric acid

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Author: Tina Wismer, DVM

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chapter 57

Lily Toxicity

DEFINITION/OVERVIEW

- Lily toxicity causes acute nephrotoxicity in cats.

ETIOLOGY/PATHOPHYSIOLOGY

- A variety of Lilium and Hemerocallis lily species (Figs 57.1, 57.2) have been associated with nephrotoxicity, including Easter lily (*Lilium longiflorum*), tiger lily (*L. tigrinum*), asiatic hybrid lily (*Lilium* sp.), day lily (*Hemerocallis dumortieri, H. fulva*), early day lily (*H. sieboldii*), orange day lily (*H. graminea*), red lily (*L. umbellatum*), rubrum lily (*L. speciosum rubrum*), stargazer lily (*L. orientalis*), western lily (*L. umbellatum*), and wood lily (*L. umbellatum*). Both the leaves and flowers are toxic, although the flowers seem more toxic. The toxic principle is water soluble.



Fig. 57.1. Easter lily plant.

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Fig 57.2. Day lily plant.

Systems Affected

- Renal/urologic: the predominant component of lily toxicity is acute tubular nephrosis that causes acute kidney injury. Azotemia is apparent within 24 hours of ingestion.
- Gastrointestinal: vomiting is a common occurrence within hours of ingestion. Pancreatitis and pancreatic lesions can occur within 8 hours of ingestion.
- Nervous: seizures may occur as early as 8 hours after ingestion.
- Hepatobiliary: liver enzyme elevations have been reported in later stages of disease.
- Musculoskeletal: increased creatine kinase was noted in experimental cases, but no histologic lesions were seen in muscle tissue.



SIGNALMENT/HISTORY

- Lily toxicity affects cats. Dogs that ingest lily plants have mild, short-term gastroenteritis without renal failure.

Historical Findings

- Vomiting, anorexia, and lethargy are usually the initial signs, and appear within 1–5 days after ingestion. The initial gastrointestinal signs may resolve, followed by signs of uremia. Polyuria may occur as early as 12 hours after ingestion. Neurologic signs of ataxia, depression, tremors, and seizures have been reported.
- Owners may find parts of the plant in vomitus, or evidence that the plant has been chewed (Fig. 57.3).

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■ **Fig. 57.3.** Lily plant leaves that were vomited.

CLINICAL FEATURES

- If kidney failure has not yet developed, cats may have normal physical examination findings.
- Dehydration, renal pain, and renomegaly are common findings on initial examination if kidney failure is present. Mentation may vary from bright and alert to comatose. Urine output varies from anuric to polyuric; overhydration is common in anuric cats shortly after starting fluid therapy. Fever has been reported in some cases.

DIFFERENTIAL DIAGNOSIS

- Initial gastrointestinal signs (vomiting, anorexia).
  - Nonspecific gastroenteritis
  - Dietary indiscretion
  - Pancreatitis
- Acute uremia in cats.
  - Nephrotoxins (e.g., ethylene glycol, NSAIDs, aminoglycosides)
  - Ureteral obstruction
  - Pyelonephritis
  - Lymphosarcoma
  - Recent ischemic events (i.e., anesthesia, hypovolemia, hypotension)

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DIAGNOSTICS

Complete Blood Count/Biochemistry

- Azotemia: elevated BUN and creatinine with elevated phosphorus
- Hyperkalemia (common) or hypokalemia
- Metabolic acidosis, elevated anion gap
- Variable increased creatine kinase: decreases within a few days
- Alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase: increased in later stages
- Occasionally hypocalcemia

Urinalysis

- Consistent with acute tubular nephrosis
- Isosthenuria
- Glucosuria
- Proteinuria
- Granular casts

Pathologic Findings

- The kidneys are usually swollen and may have perirenal edema. Histologically, acute tubular nephrosis that affects predominantly the proximal tubule is characteristic, frequently with evidence of epithelial regeneration. Small numbers of birefringent crystals are occasionally seen, but lily toxicity is not caused by oxalate nephrosis.
- Pancreatic degeneration in early stages and fibrosing pancreatitis in late stages have been reported. Hepatic and lung congestion may occur.



THERAPEUTICS

- Gastric decontamination to limit absorption of toxic components.
- Early aggressive fluid treatment to counteract dehydration from polyuria and possibly to increase elimination of toxic component prior to onset of renal failure.
- Standard therapy to manage acute kidney injury.
- Treatment with fluid therapy is important even if clinical signs are not present. Peace lily (*Sathiphyllum* spp.) and lily-of-the-valley (*Convallaria majalis*) are not true lilies and do not cause nephrotoxicity.

Drugs of Choice

- Induce vomiting: apomorphine (administered via conjunctival sac or 0.03–0.04 mg/kg IV), 3% hydrogen peroxide (0.25–0.5 mL/kg PO), or xylazine (0.44 mg/kg IV, IM, or SQ).

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- Activated charcoal: 1–4 g/kg PO (protect airway with cuffed endotracheal tube if patient is unconscious).
- Fluid therapy: balanced polyionic crystalloid (i.e., lactated Ringer's solution or Plasmalyte-147) or saline IV at twice the maintenance rate for 48 hours prior to onset of renal failure.

Precautions/Interactions

- If urine production is decreased, aggressive diuresis will result in volume overload.

Alternative Therapies

- If medical management does not control uremia, hemodialysis or peritoneal dialysis has been used with favorable outcomes.

Diet

- Anorexia is common and may require placement of a feeding tube for management. A moderately protein-restricted diet is appropriate.

COMMENTS

Client Education

- If renal failure resolves completely (or does not develop), no special care is needed after discharge. If residual renal damage persists, standard care for chronic kidney disease will be required.

Patient Monitoring

- Early treatment: assessment of BUN, creatinine, electrolytes, and phosphorus at admission and after 48 hours of fluid therapy is recommended.
- After onset of renal failure: monitoring as for any cause of acute kidney injury. Particular attention to volume status because many cats develop oliguria or anuria.

Prevention/Avoidance

- Lily plants should not be available in the cat's environment. Lilies are common in floral arrangements, and day lilies are common in gardens.
- Cats will actively seek contact with lilies placed in the home.

Expected Course and Prognosis

- Early treatment (within 18 and up to 48 hours after ingestion): prompt decontamination and fluid therapy may prevent renal failure and has a good prognosis.

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- After development of renal failure: overall mortality rates for cats with acute kidney injury from nephrotoxic causes are about 50–60%. Cats that survive the acute phase may recover completely or have residual chronic kidney disease.

Abbreviations

BUN: blood urea nitrogen
IM: intramuscular
IV: intravenous
NSAID: nonsteroidal antiinflammatory drug
PO: by mouth (per os)
SQ: subcutaneous

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Lower Urinary Tract Infections – Canine and Feline

chapter 58



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DEFINITION/OVERVIEW

- Lower UTI encompasses a wide variety of clinical entities whose common denominator is microbial invasion of the bladder (cystitis), urethra (urethritis), prostate gland (prostatitis), or two or more of these sites.
- Bacteriuria refers to the presence of bacteria in urine. Identification of bacteria in urine is not synonymous with UTI because they may represent small particles which resemble bacteria but are false-positive bacteriuria (amorphous crystals, debris, lipid molecules or cytoplasmic organelles).
    - Significant bacteriuria is a term used to denote bacteriuria that represents UTI. A large number of bacteria in a properly collected and cultured urine specimen is indicative of UTI.
    - Subclinical bacteriuria (asymptomatic bacteriuria, covert bacteriuria) is defined as bacteria present in the urine as determined by positive bacterial culture that is unaccompanied by clinical signs or cytological evidence of UTI.
- Uncomplicated UTI: defined as a sporadic bacterial infection in which no underlying structural or functional abnormality of the urinary system can be identified. Most uncomplicated UTIs are likely to be caused by transient, self-limiting, and potentially reversible abnormalities of host defenses.
- Complicated UTI is an infection that occurs secondary to comorbidities (e.g., diabetes mellitus, urinary calculi, immunocompromising disorders, urinary or reproductive tract conformational abnormalities) that interfere with normal host defenses and predispose the patient to persistent or recurrent infections and/or treatment failure.
- Recurrent UTI is three or more episodes of UTI during a 12-month period. Up to 4.5% of all dogs with UTIs have recurrent or persistent UTIs. Recurrent UTIs can be further subclassified as follows.
    - Relapse UTI is a recurrent UTI by the *same* species and strain of microorganism within 6 months (typically within weeks rather than months) following apparently successful treatment of a UTI. This presumably indicates a failure to completely eliminate the pathogen.

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  - Reinfection UTI is a recurrent UTI by a *different* species and/or strain of microorganism within 6 months following apparently successful treatment of a UTI.
  - Persistent (or refractory) UTI is defined as a recurrent UTI caused by the *same* species and strain of microorganism that continues, despite appropriate antimicrobial therapy (*in vitro* susceptibility to the antimicrobial), without documenting a period of sterile urine.
- Appreciating evaluation of antimicrobial susceptibility patterns can be helpful; the persistence of the same microorganism should not be assumed based solely on the same bacterial species and antimicrobial susceptibility pattern. Ideally genotyping should be performed to determine if the same strain is present since unrelated organisms can have the same susceptibility pattern or the same organism can have a change in the pattern.
- Superinfection is defined as infection with an additional organism during the course of antimicrobial therapy.



ETIOLOGY/PATHOPHYSIOLOGY

- In the healthy state, the sterility of the lower urinary tract is maintained by multiple, concurrent factors including unidirectional urine flow following normal micturition, urethral sphincter function, mucosal integrity and defense barriers, local and systemic immunocompetence, and antimicrobial properties of the urine.
- Any disease process or therapeutic manipulation that interferes with normal micturition, alters the structural or functional characteristics of the urinary bladder or urethra, alters the volume or composition of urine, or impairs immune responsiveness predisposes the urinary tract to bacterial colonization.
- Usually caused by ascending migration of aerobic bacteria from the intestinal and lower genitourinary tract into the lower urinary tract under conditions that permit bacteria to persist in urine or adhere to urothelium and subsequently multiply. Certain strains of bacteria (i.e., uropathogenic *E. coli*) have specific virulence factors to permit adhesion and cytotoxicity.
- Aerobic bacteria are the most common uropathogens; *Escherichia coli* is the most common urine pathogen in cats and dogs, followed by *Staphylococcus*, *Proteus*, *Klebsiella*, *Enterococcus*, *Streptococcus*, *Pseudomonas*, and *Corynebacterium* spp. Cats can be infected by a unique strain of *Staphylococcus* – *Staphylococcus felis*.
- Approximately 72% of UTIs in dogs are caused by a single species of bacteria, 20% are caused by two species, and 8% are caused by three or more species.
- Similar to dogs, UTIs in cats are frequently (77%) caused by a single species of bacteria, 17% are caused by two species, and 5% are caused by three species.
- Anaerobic bacteria, fungal agents, parasites, viruses, mycoplasmas, and ureaplasmas are uncommon causes in dogs and cats.

Systems Affected

- Urologic: urinary bladder, urethra, prostate, vagina.

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Risk Factors/Causes

Cats

- UTIs are uncommon, between 1–3% of all cases of feline lower urinary tract disorders. Possibly more frequent in females.
- Risk factors include increasing age (>10 years), decreased body weight, chronic kidney disease (but not severity of azotemia), urinary incontinence, hyperthyroidism, diabetes mellitus (regardless of status of diabetic control), gastrointestinal disorders, and following indwelling urethral catheters and perineal urethrostomies.
- Equivocal increased risk in Siamese, Persian, and Abyssinian cats.
- Isosthenuria without other predisposing factors is unlikely as a risk factor for UTIs in cats.

Dogs

- UTIs are common in female (in particular spayed) dogs, less common in male dogs. Approximately 14% of dogs will develop a bacterial UTI.
- Risk factors include increasing age, prolonged urinary catheterization (>3 days), perineal urethrostomies, immunosuppressive conditions or therapies including long-term corticosteroid and/or cyclosporine administration, hyperadrenocorticism, diabetes mellitus, intervertebral disk herniation, and urogenital transitional cell carcinoma.
- Numerous breed predispositions for recurrent or persistent UTIs have been identified.

Historical Findings

- None in some patients (35% of cats in one study had no lower urinary tract signs)
- Pollakiuria
- Stranguria
- Dysuria
- Hematuria
- Malodorous urine
- Periuria – urination in inappropriate locations
- Urge or urinary incontinence – apparent loss of ability to voluntarily control urination
- Perivulvar dermatitis, licking or other signs of irritation



CLINICAL FEATURES

- May be clinically normal.
- Acute infection: urinary bladder may be small and painful; palpation may induce urination.
- Chronic infection: urinary bladder or urethral wall may be thickened; masses may be palpable.
- Other predisposing anatomic or functional abnormalities associated with complicated UTIs may be identified.

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DIFFERENTIAL DIAGNOSIS

Dogs

- Uroliths.
- Urogenital cancer (benign and malignant).
- Trauma.
- Neurogenic disorders including reflex dyssynergia, urethral spasm, and hypotonic or atonic bladder (primary or secondary).
- Iatrogenic disease including due to urethral catheterization or indwelling urethral catheters (especially open systems).
- Urethrostomy complications.
- Anatomic abnormalities including urachal anomalies and acquired urethral strictures
- Behavioral disorders resulting in periuria.
- Bleeding disorders resulting in hematuria.
- Prostatic disease (including prostatitis, benign prostatic hyperplasia).

Cats

- Idiopathic lower urinary tract disease.
- Uroliths and urethral plugs.
- Behavioral disorders resulting in periuria.
- Neoplasia (benign and malignant).
- Trauma.
- Neurogenic disorders including reflex dyssynergia, urethral spasm, and hypotonic or atonic bladder (primary or secondary).
- Iatrogenic disease including due to urethral catheterization, indwelling urethral catheters (especially open systems), postsurgical catheters, reverse-flushing solutions, and urethrostomy complications.
- Anatomic abnormalities including urachal anomalies and acquired urethral strictures.
- Constipation causing straining to defecate and confused for straining to urinate.

DIAGNOSTICS

- Results of CBC and serum biochemistries are usually normal.
- Urine sediment should be evaluated by microscopic techniques; dipstick leukocyte and bacteria test pads are unsatisfactory for canine and feline urine specimens; staining of urine sediment with Wright's, Gram's, or new methylene blue stain may significantly improve detection of bacteriuria.
- Pyuria: commonly associated with UTI but may occur with inflammation that is due to other noninfectious causes (e.g., uroliths); absence does not rule out UTI because it may occur without an inflammatory response. Pyuria is not a sensitive (53%) nor highly specific (86%) indicator of UTI in cats.

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- Hematuria and proteinuria: commonly associated with UTI but may be associated with other inflammatory and noninflammatory disease processes; absence does not rule out UTI because it may occur without an inflammatory response.
- Bacteriuria: presence of bacteria in urine; commonly associated with UTI but may represent specimen contamination during collection or storage or misinterpretation of nonbacterial look-alikes in urine sediment; absence does not rule out UTI because large numbers of bacteria are required for visualization by light microscopy. Bacteriuria is not a highly sensitive (73%) nor specific (68%) indicator of UTI in cats.
- Diagnostic quantitative urine culture and sensitivity testing.
    - Necessary for definitive diagnosis are isolation and identification of bacteria, quantification of the number of bacteria, and determination of the organism's susceptibility to antimicrobial agents.
    - Identifies the presence of antimicrobial resistant bacteria and can help differentiate reinfection from relapse should a UTI return.
    - Proper collection, storage, and handling of urine specimens are important for interpretation of culture results. Ideally, cystocentesis should be used for sample collection. Catheterized samples can be evaluated for culture but free-catch (midstream voiding or manual expression) samples should not be used.
    - Antimicrobics should be discontinued for 3–5 days before diagnostic urine culture.
    - Urine should be refrigerated in a sterile closed container immediately after collection and submitted to the laboratory as quickly as possible. Culture results from samples that take >24 hours to reach the laboratory can produce both false-positive and false-negative readings, particularly if a urine preservative was not used. Commercially manufactured collection tubes containing preservatives combined with refrigeration may be used to preserve specimens for up to 72 hours.
    - Quantification of bacteria in a urine sample provides the level of bacterial growth (colony forming unit (cfu) or organisms per mL (opm) of urine) which can be used in interpreting the relevance of results.
        - For urine collected by cystocentesis, any level of bacterial growth may be significant; however, detection of $\geq 10^3$ cfu/mL is considered significant bacteriuria in dogs and cats and indicates a probable UTI.
        - For catheterized urine samples, detection of $\geq 10^4$ cfu/mL in males and $\geq 10^5$ cfu/mL in females is typically considered significant.
        - In urine samples collected by voluntary micturition or manual compression of the urinary bladder, detection of $\geq 10^5$ cfu/mL of urine in dogs and $\geq 10^4$ cfu/mL of urine in cats has been suggested to be consistent with a UTI. However, studies in normal dogs and cats have shown that contamination of voided urine samples may result in colony counts of $\geq 10^5$ cfu/mL. Consequently, voided urine samples are generally unsatisfactory for routine diagnostic bacterial culture.
    - Other factors may influence the results of quantitative cultures of urine, including microbial species, collection method, time elapsed between collection and culture,

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method of preservation, *in vivo* growth characteristics of the bacteria, frequency of micturition, magnitude of diuresis, site(s) of infection, and administration of antimicrobics prior to bacterial culture.
    - *In vitro* susceptibility testing that determines the MIC of each drug against the bacterial isolate is the preferred method of identifying appropriate antimicrobics. Any antimicrobic whose urine concentration is at least four times the MIC of the bacterial isolate is likely to be effective.
- Aerobic culture of bladder wall biopsy specimens may detect UTI in patients with negative urine cultures, in particular in patients with uroliths.
- In-clinic screening tests have been evaluated as an adjunct test to microscopic examination of urine sediment or as an inexpensive means to screen dogs and cats for a bacterial UTI. A catalase-based urine test (Accutest Uriscreen™) was more sensitive but less specific than sediment examination. A veterinary urine dipstick paddle embedded with 2 standard culture media (cysteine lactose electrolyte-deficient medium and the other side with eosin methylene blue medium) was a sensitive and specific test (89.1% and 98.6%, respectively, compared to urine culture) for screening patients for bacterial UTI, but uropathogen identification was not always accurate.
- Survey and contrast radiographic and ultrasonic evaluations of the lower urinary tract may exclude other causes of lower urinary tract signs and identify predisposing factors (e.g., neoplasia) or sequelae (e.g., infection-induced struvite uroliths). Rarely, intramural gas formation may be observed (emphysematous cystitis).
- Endocrine testing: screening for hyperadrenocorticism (dogs) and hyperthyroidism (cats) may be appropriate based on the clinical history.
- Urethrocystoscopy may exclude other causes of lower urinary tract signs and identify predisposing factors or sequelae.
- Biopsies obtained with urinary catheters, cystoscopes, or via surgery may permit morphologic characterization of the inflammatory or neoplastic lesions; not routinely needed.

Pathologic Findings

- Varying degrees of mucosal ulceration and submucosal edema, vasodilation, hemorrhage, and inflammatory cell infiltration are common light microscopic lesions observed in urinary bladders of patients with acute and chronic bacterial cystitis.
- Follicular cystitis: a common form of chronic cystitis characterized by multifocal gray-white mucosal nodules often surrounded by a zone of hyperemia; nodules are composed of proliferating aggregations of lymphocytes.
- Polypoid cystitis: an uncommon form of chronic cystitis characterized by mononuclear cell infiltration, epithelial proliferation, and development of villus-like sessile polyps.
- Granulomatous (proliferative cystitis): an uncommon form of chronic urethritis resulting in partial or complete urethral obstruction; lesions are generally characterized by severe, extensive, lymphocytic or chronic suppurative inflammation with granulation tissue and edema.

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THERAPEUTICS

- Therapeutic goals are to eliminate bacteria from urine and tissues and allow the urinary tract and its defense mechanisms time to recover sufficient function to prevent recurrence. Both improper therapy and potentially overuse and misuse of antibiotic should be considered. At a minimum, these situations can lead to persistent clinical signs, need to repeat treatment, additional diagnostic testing and/or antimicrobial resistance, which have been documented in cat and dog UTIs.
- Antimicrobials should be selected on the basis of antimicrobial susceptibility testing, their ability to achieve high concentrations in urine, ease of administration, relative cost, and safety.

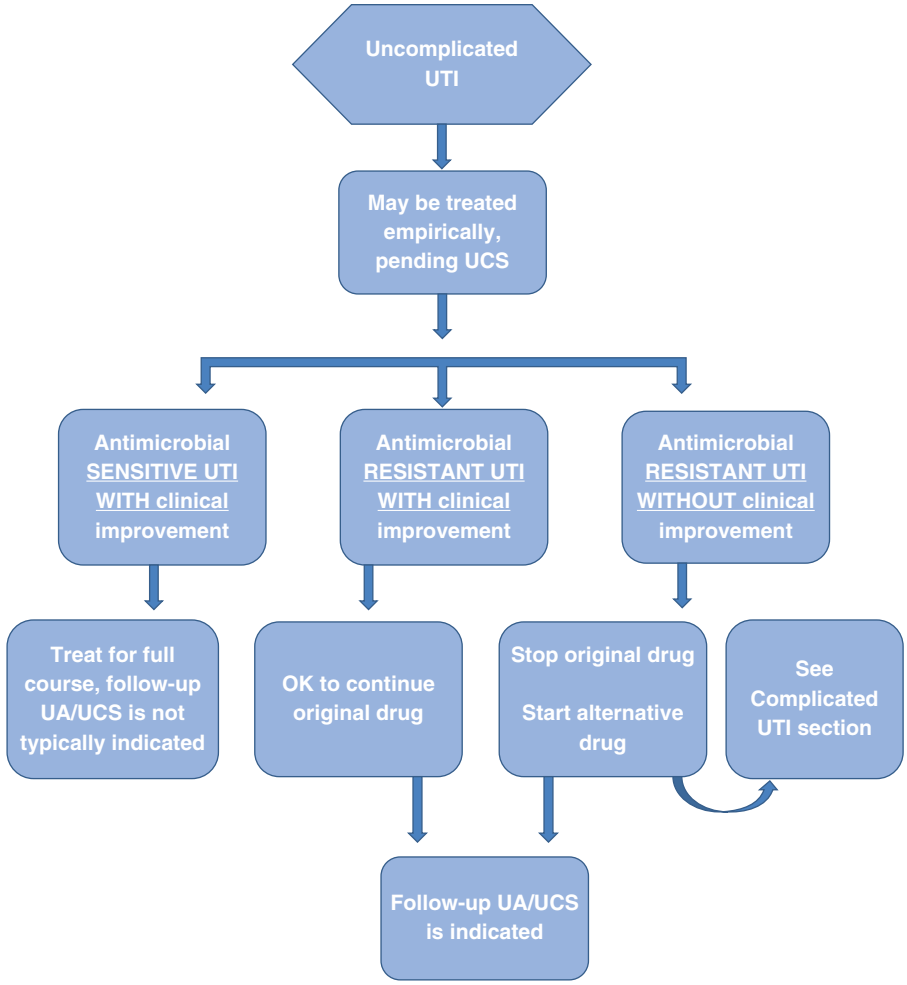
Uncomplicated Urinary Tract Infection (Fig. 58.1)

- May be treated empirically without results of susceptibility testing provided that patients have not had frequent or recurrent signs and have not been given antimicrobial drugs in the past 4–6 weeks.
- Initial therapy should consist of amoxicillin (11–15 mg/kg PO q8h) or trimethoprim-sulfamethoxazole (15 mg/kg PO q12h). Amoxicillin/clavulanic acid (12.5–25 mg/kg PO q8h) is acceptable but is not typically recommended. The optimal duration of treatment has not been determined; a 7–14-day course is typically recommended.
- In cats with uncomplicated UTIs, pradofloxacin 2.5% oral suspension (0.2 mL/kg PO q24h for 10 days) has been shown to be effective and has a palatable formulation.
- In dogs with uncomplicated UTIs, initial studies of short-duration antibiotic protocols have shown them to be not inferior to conventional antibiotic protocols. Trimethoprim-sulfamethoxazole (15 mg/kg PO q12h for 3 days) in female dogs. High-dose enrofloxacin (18–20 mg/kg PO q24h for 3 days) protocol in female (majority studied) and male dogs.
- If culture and susceptibility testing reveals an antimicrobial-resistant isolate to the antibiotic chosen, but there is clinical improvement, it is acceptable to maintain the current treatment; however, a follow-up urinalysis and culture should be performed after treatment has been completed. If an antimicrobial-resistant isolate is detected without clinical improvement, then the original drug should be discontinued and an alternative drug (see Complicated UTI) started.
- If the clinical signs resolve and the full course of antimicrobials is administered correctly for an antimicrobial-sensitive isolate, then no intra- or posttreatment urinalysis or urine culture is typically indicated.

Complicated Urinary Tract Infection

- Results of urine culture susceptibility testing are essential for patients with complicated UTIs. In patients having uroliths removed, culture of a urinary bladder wall biopsy should also be considered.

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■ **Fig. 58.1.** Algorithm for treatment of uncomplicated UTI.

- A detailed medical history should be obtained with a focus on recurrent or persistent clinical signs, prior antimicrobial therapy and client compliance with administration of treatments, concurrent medications (in particular immunosuppressive or dermatologic therapies), recent catheterization, and urinary tract surgeries.
- Ideally, testing should be performed to screen for an underlying anatomic or functional abnormality or comorbidity. Abnormalities should be corrected or controlled since, left unmanaged, it is possible therapy will ultimately be unsuccessful. In one study, 41% of pets with a corrected identifiable disorder had resolution of UTIs for >6 months, compared to only 5% treated with conventional antibiotic therapy.

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- If the patient's clinical condition permits, consideration should be given to waiting for susceptibility testing results prior to starting therapy.
  - If immediate treatment is deemed necessary for patient care reasons, therapy should be selected from the antimicrobials listed in the Uncomplicated UTI section; however, if possible, the drug class used should be different from that used for prior UTIs.
  - If therapy is started once susceptibility testing results are available, preference should be given to susceptible antimicrobials that are excreted in urine predominantly in an active form. See Selected and alternative antimicrobial treatment options for UTI in the dog and cat.
  - If treatment was initiated before culture and susceptibility results were available and an antimicrobial-resistant isolate was identified to the antibiotic chosen, a change to more active drug based on susceptibility testing should be made.
  - The optimal duration of treatment has not been determined; a 4-week course is typically recommended.
- For complicated UTIs, a repeat urine culture should be considered 5–7 days *after initiation* of therapy. In particular in patients with relapsing or refractory UTIs or at high risk for ascending or systemic infection, an additional urine culture should be considered 7 days *after cessation* of therapy in all cases to document clearance of the infection. If bacterial growth is detected and client compliance with administration of treatments is confirmed, additional screening for predisposing factors for relapse or reinfection should be performed.

Subclinical Bacteriuria

- In patients with no clinical signs of a UTI and no cytologic evidence of UTI based on examination of urine sediment, treatment may not be necessary.
- Treatment should be considered in immunocompromised patients, patients with underlying renal disease and/or a high risk of ascending or systemic infection.

Selected Antimicrobial Treatment Options for UTI in the Dog and Cat

- Amoxicillin 11–15 mg/kg PO every 8 hours
- Amoxicillin/clavulanate 12.5–25 mg/kg PO every 8 hours
- Cefovecin 8 mg/kg single SQ injection. Can be repeated once after 7–14 days
- Cefpodoxime proxetil 5–10 mg/kg PO every 24 hours
- Ceftiofur 2 mg/kg SQ every 12–24 hours
- Cephalexin, cefadroxil 12–25 mg/kg PO every 12 hours
- Ciprofloxacin 30 mg/kg PO every 24 hours
- Enrofloxacin (D) 10–20 mg/kg every 24 hours, (C) 5 mg/kg PO every 24 hours
- Marbofloxacin 2.7–5.5 mg/kg PO every 24 hours
- Orbifloxacin tablets: 2.5–7.5 mg/kg PO every 24 hours; oral suspension: (D) 2.5–7.5 mg/kg PO every 24 hours, (C) 7.5 mg/kg PO every 24 hours

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- Pradofloxacin 2.5% oral suspension (C): 0.2 mL/kg PO every 24 hours
- Trimethoprim-sulfamethoxazole: 15 mg/kg combined PO every 12 hours

Alternative Antimicrobial Treatment Options for UTI in the Dog and Cat

- Amikacin (D): 15–30 mg/kg IV/IM/SQ every 24 hours, (C)10–14 mg/kg IV/IM/SQ every 24 hours
- Chloramphenicol (D): 40–50 mg/kg PO every 8 hours, (C)12.5–20 mg/kg PO every 12 hours
- Imipenem/cilastatin: 5 mg/kg IV/IM every 6–8 hours
- Meropenem: 8.5 mg/kg every 12 hours (SQ) or every 8 hours (IV)
- Nitrofurantoin: 4.4–5 mg/kg PO every 8 hours

Precautions/Interactions

- Specific antimicrobial drugs are contraindicated in patients with known hypersensitivity to them.
- Trimethoprim-sulfa products have been associated with keratoconjunctivitis sicca, hepatitis, hepatic necrosis, anemia, granulocytopenia, and polyarthritis in dogs; use with caution in patients with preexisting hepatic disease.
- Enrofloxacin has been associated with retinal degeneration in cats.
- Ciprofloxacin is sometimes utilized due to lower cost than enrofloxacin; however, it has a lower and more variable oral bioavailability than enrofloxacin, marbofloxacin, and orbifloxacin.
- Chloramphenicol can cause myelosuppression, particularly with long-term therapy. Contact by humans should be limited because of rare idiosyncratic aplastic anemia.
- Because of potential nephrotoxicity associated with long-term administration of aminoglycosides, alternative safer antimicrobics should be selected whenever possible; use with extreme caution in patients with preexisting renal disease.
- Cefovecin should only be used if oral treatment is problematic.
- Enterococci are resistant to cephalexin, cefadroxil, cefovecin, cefpodoxime proxetil, and ceftiofur.

Diet

- Dietary modification may be indicated for management of other concurrent urinary tract disorders (e.g., urolithiasis).

Surgical Considerations

- Only indicated for detection or correction of abnormalities predisposing to UTI. In dogs treated with vulvoplasty (episioplasty) for management of recessed vulva and redundant vulvar skin folds, the incidence of UTI was reduced in 84% of cases.

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COMMENTS

Client Education

- Clinical improvement can be expected in 24–48 hours after initiating appropriate therapy; however, compliance with treatment recommendations is essential to allow adequate time for host urinary tract defenses to heal and regain sufficient function to prevent recurrence.
- Prognosis for simple uncomplicated UTI is generally good to excellent.
- Prognosis for complicated UTI depends on the nature of the infecting organism (virulence and antimicrobial susceptibility) and the ability to identify and correct or control predisposing abnormalities of host defenses.
- Compliance with recommendations for treatment and follow-up is crucial for optimal results.

Patient Monitoring

- Therapeutic urine culture: culture of urine at strategic times during antimicrobial therapy (so-called therapeutic cultures) is an effective method of assessing therapy, especially in patients with a high risk of morbidity or mortality associated with their UTI or in patients receiving potentially toxic antimicrobics. Therapeutic cultures allow timely testing of antimicrobial efficacy, verification of proper antimicrobial drug administration, early detection of emerging bacterial resistance, timely detection of persistent infections, and provision of justification for early discontinuation of potentially toxic antimicrobial. Initial response to therapy is considered effective only if culture of a properly collected urine sample does not result in growth of bacteria.
    - For complicated UTIs, a repeat urine culture should be considered 5–7 days after initiation of therapy, in particular in patients with relapsing or refractory UTIs or at high risk for ascending or systemic infection.
    - Therapeutic urine cultures are also performed if clinical signs or laboratory abnormalities recur during therapy.
- Surveillance urine culture: after antimicrobial therapy, timely follow-up evaluations (so-called surveillance cultures) are indicated to confirm eradication of the UTI and to detect recurrent infections at a subclinical state. Surveillance cultures are essential to differentiate relapses from reinfections.
    - For complicated UTIs, urine collected by cystocentesis should be cultured 7 days after completing antimicrobic therapy to confirm successful eradication and identify relapses.
    - Urine culture may be performed at 1–2 months and 3–6 months after completion of therapy to detect reinfections.
    - Urine culture and urinalysis should be performed any time clinical signs recur.

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Prevention/Avoidance

- Identify and eliminate or control predisposing abnormalities of host defenses.
- Avoid indiscriminate use of urinary catheters.
- Avoid surgical procedures that permanently alter host defenses (e.g., perineal urethrostomies).
- Cranberry juice or extract may be beneficial in the management of recurrent UTI following successful treatment of the UTI. The beneficial effect has been attributed to decreasing the ability of bacteria to adhere to the uroepithelium. Additional research on dosing cranberries is ongoing.
- Urinary antiseptics such as methenamine (methenamine hippurate) may also be beneficial in the management of recurrent UTI following successful treatment of the UTI.
- There is insufficient evidence to recommend chronic antibiotics (either pulse or low-dose therapy) to prevent UTIs. Although some patients likely benefit from these protocols, the risk of antimicrobial resistance needs to be considered.

Possible Complications

- Struvite urolithiasis: UTI with urease-producing microbes (*Staphylococcus*, *Proteus*, *Klebsiella*, *Pseudomonas*, *Corynebacterium*, and *Ureaplasma* spp.).
- Polypoid cystitis: uncommon; may be associated with chronic bacterial UTI.
- Emphysematous cystitis: uncommon; associated with infection with gas-producing microbes.
- Granulomatous (proliferative) urethritis: uncommon; may be associated with chronic bacterial UTI.
- Multidrug-resistant pathogens, including various Enterobacteriaceae, staphylococci, and enterococci, are becoming increasingly recognized and are problematic to treat.
- Pyelonephritis, prostatitis, diskospondylitis, endocarditis, or sepsis.

Expected Course and Prognosis

- Untreated UTIs persist indefinitely and may be associated with an increased risk for urolithiasis and bacterial invasion of other components of the urinary system (e.g., the kidneys) or tissues outside the urinary tract.
- Clinical signs typically improve within 24–48 hours after initiation of appropriate therapy; persistent signs may be due to treatment failure or the presence of other concurrent lower urinary tract disorders that cause similar signs (e.g., uroliths).
- Prognosis for simple uncomplicated UTI is generally good to excellent.
- Prognosis for complicated UTI depends on the nature of the infecting organism (virulence and antimicrobic susceptibility) and the ability to identify and correct or control predisposing abnormalities of host defenses.

Synonyms

- Bacterial cystitis
- Urethrocystitis
- Urethritis

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Abbreviations

C: cat
CBC: complete blood count
cfu: colony-forming unit
D: dog
IM: intramuscular
IV: intravenous
MIC: minimum inhibitory concentration
opm: organisms per mL
PO: by mouth (per os)
SQ: subcutaneous
UTI: urinary tract infection

Suggested Reading

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chapter 59

Macadamia Nut Toxicity

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DEFINITION/OVERVIEW

- Syndrome of tremors, weakness, agitation, vomiting, diarrhea, and paralysis in dogs associated with ingestion of macadamia nuts.
- Clinical signs can develop when 2.4–62.4 g/kg of macadamia nuts have been ingested.
- Clinical signs can develop within 12 hours of ingestion.
- No case fatalities have been reported.

ETIOLOGY/PATHOPHYSIOLOGY

- Mechanism of toxicity unknown.

Systems Affected

- Gastrointestinal: vomiting and diarrhea, often with visible macadamia nuts in the vomitus or stool.
- Nervous system: tremors, ataxia, weakness, ascending paralysis (primarily rear limbs).

SIGNALMENT/HISTORY

Species

- Dogs.

General Comments

- Witnessed ingestion of macadamia nuts.
- Chewed up packages that contained macadamia nuts.

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Historical Findings

- Vomiting or diarrhea, often with macadamia nuts visible in the vomitus
- Ataxia
- Tremors
- Muscle weakness
- Ascending paralysis
- Hyperthermia

Physical Examination Findings

- Ataxia, tremors, lower motor neuron signs to rear limbs, muscle weakness, paralysis, diarrhea, vomitus staining on muzzle, hyperthermia when severe tremors have occurred

Risk Factors/Causes

- Iatrogenic administration of macadamia nuts to dogs
- Access to macadamia nuts
- Free roaming
- Ingestion of macadamia nuts
- Toxic principle unknown



DIFFERENTIAL DIAGNOSIS

- Botulism
- Coonhound paralysis (polyradiculoneuritis)
- Tick paralysis
- Intervertebral disk disease
- Metaldehyde toxicity
- Mycotoxin ingestion
- Mushroom toxicity
- Seizures, idiopathic epilepsy
- Gastrointestinal
- Viral or bacterial gastroenteritis
- Pancreatitis
- Gastrointestinal parasitism
- Hypoadrenocorticism



DIAGNOSTICS

Complete Blood Count

- Serum biochemistry: elevated lipase
- Urinalysis: normal

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THERAPEUTICS

- Induce emesis within 3 hours of ingestion (apomorphine 0.03–0.04 mg/kg IV).
- Supportive care with intravenous crystalloid fluids to maintain hydration, turn frequently to prevent decubital ulcer formation, passive range of motion exercises, placement of a urinary catheter to maintain cleanliness, antiemetics to decrease vomiting and risk of aspiration pneumonia.

Drugs of Choice

- Antiemetics.
  - Metoclopramide: 1–2 mg/kg per day IV CRI, PO
  - Maropitant: 1 mg/kg SQ every 24 hours, 2 mg/kg PO every 24 hours
  - Dolasetron: 0.6 mg/kg IV every 24 hours
  - Ondansetron: 1 mg/kg IV every 8–12 hours
- Treat tremors.
  - Methocarbamol: 45–220 mg/kg IV slowly to effect; not to exceed 220 mg/kg IV
  - Guaifenasin: 44–110 mg/kg IV
  - Diazepam: 0.5–2 mg/kg IV to effect

Expected Course and Prognosis

- No reported deaths attributed to macadamia nut toxicity.
- Very favorable if vomiting is induced within 2–3 hours of ingestion, followed by activated charcoal.
- Will recover within 24–48 hours of onset of clinical signs.

Abbreviations

CRI: constant rate infusion
IV: intravenous
PO: by mouth (per os)
SQ: subcutaneous

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chapter 60

Magnesium Disorders

DEFINITION/OVERVIEW

- Hypomagnesemia.
  - Dogs: serum total magnesium <1.89 mg/dL and serum ionized magnesium <0.43 mmol/L
  - Cats: serum total magnesium <1.9 mg/dL and serum ionized magnesium <0.43 mmol/L
- Hypermagnesemia.
  - Dogs: serum total magnesium >2.51 mg/dL and serum ionized magnesium >0.60 mmol/L
  - Cats: serum total magnesium >2.6 mg/dL and serum ionized magnesium >0.70 mmol/L

ETIOLOGY/PATHOPHYSIOLOGY

- Magnesium is the second most abundant intracellular cation. The vast majority of magnesium is found in bone (60%) and muscle (20%). Less than 1% of total body magnesium is present in the serum.
- Because greater than 99% of total body magnesium is located in the intracellular compartment, serum magnesium concentrations do not always reflect total body magnesium status. Therefore, a normal serum total or ionized magnesium concentration can occur in the presence of a total body magnesium deficiency.
- In the serum, magnesium exists in three distinct fractions: a protein-bound fraction (30%), an anion-complexed fraction (5%), and an ionized fraction (65%). The ionized fraction is thought to be the physiologically active component.
- Serum ionized magnesium appears to equilibrate rapidly across the cell membrane; thus extracellular ionized magnesium concentrations are likely a more accurate indicator of intracellular stores.
- Magnesium is a coenzyme for the membrane-bound sodium-potassium ATPase pump and functions to maintain the sodium-potassium gradient across all membranes; as a result, it plays an important role in the activity of electrically excitable tissues.

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- Interference with the electrical gradient can alter resting membrane potential, signal transduction, and smooth muscle tone, resulting in cardiovascular abnormalities such as arrhythmias and inappropriate vascular tone.
- Magnesium decreases acetylcholine release from nerve terminals and depresses the excitability of nerve and muscle membranes. Therefore, hypermagnesemia results in hyporeflexia and flaccid paralysis and hypomagnesemia can result in muscle fasciculations and seizures.
- Magnesium regulates calcium movement into smooth muscle cells and can affect peripheral vascular tone and contractile strength.
- Because magnesium is necessary for the movement of sodium, potassium, and calcium into and out of cells, other manifestations of hypomagnesemia include metabolic abnormalities such as concurrent hypokalemia (often refractory to potassium supplementation), hyponatremia, and hypocalcemia.
- The renal system is the main regulator of serum total and ionized magnesium concentration and total body magnesium content; any condition or disease that lowers glomerular filtration rate can cause hypermagnesemia.
- Hypomagnesemia causes resistance to the effects of PTH and increases the uptake of calcium into bone.
- The incidence of hypomagnesemia in dogs ranges from 6% to 54%, with the higher incidence rates occurring in critically ill dogs.
- The incidence of hypomagnesemia in cats has been reported to be 28% in a population of cats admitted to an intensive care unit.
- The incidence of hypermagnesemia in dogs was reported to be 13% in a population of dogs admitted to a critical care unit. These dogs were 2.6 times more likely to not survive their illness when compared to dogs with normal serum magnesium concentrations.
- The incidence of hypermagnesemia in cats has been reported to be 17.5% in a population of cats admitted to an intensive care unit.

Systems Affected

- Cardiovascular.
  - Hypomagnesemia: tachyarrhythmias (i.e., atrial fibrillation, supraventricular tachycardia, premature ventricular contractions, ventricular tachycardia, torsades de pointes, and ventricular fibrillation), hypertension, coronary artery vasospasm, and platelet aggregation
  - Hypermagnesemia: arrhythmias (i.e., bradycardia, complete heart block, and asystole), hypotension, and hypothermia
- Endocrine/metabolic.
  - Hypomagnesemia: concurrent hypokalemia, hyponatremia, or hypocalcemia
- Gastrointestinal.
  - Hypomagnesemia and hypermagnesemia: dysphagia secondary to esophageal weakness
- Musculoskeletal.
  - Hypomagnesemia and hypermagnesemia: weakness

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- Neuromuscular.
  - Hypomagnesemia: hyperreflexia, muscle fasciculations, seizures, ataxia, and coma
  - Hypermagnesemia: hyporeflexia and flaccid paralysis
- Respiratory.
  - Hypomagnesemia and hypermagnesemia: hypoventilation and subsequent hypoxemia secondary to respiratory muscle weakness or paralysis



SIGNALMENT/HISTORY

- No breed, age, sex, or genetic predilection.
- Signs associated with hypomagnesemia include lethargy, depression, weakness, tachyarrhythmias, hyperreflexia, muscle fasciculations, ataxia, seizures, hypertension, dysphagia, and respiratory depression.
- Signs associated with hypermagnesemia include lethargy, depression, weakness, hypotension, bradycardia, hyporeflexia, flaccid paralysis, nausea, vomiting, dysphagia, and respiratory depression.

Risk Factors/Causes

- Hypomagnesemia: administration of magnesium-depleted intravenous fluids, total parenteral nutrition, or partial parenteral nutrition; administration of drugs that increase renal magnesium loss including digitalis, diuretics, aminoglycosides, cisplatin and cyclosporine; diseases that cause loss of magnesium via the gastrointestinal tract, including inflammatory bowel disease and protein-losing enteropathy; excessive loss of magnesium due to lactation; chelation of magnesium secondary to massive catecholamine release (sepsis, shock, and trauma) or to citrate administration (massive blood transfusions); and sequestration of magnesium secondary to pancreatitis.
- Hypermagnesemia: renal failure, hypoadrenocorticism, massive hemolysis, iatrogenic overdose of magnesium supplementation, thoracic neoplasia with pleural effusion, and excessive use of magnesium-containing cathartics, laxatives, or antacids, especially in patients with underlying renal disease.

Historical Findings

- Hypomagnesemia and hypermagnesemia: frequently nonspecific (i.e., lethargy, depression, weakness).



CLINICAL FEATURES

- Signs of hypomagnesemia and hypermagnesemia are often vague and multisystemic.
- Hypomagnesemia: depression, weakness, tachycardia, respiratory difficulty, hyperreflexia, and/or muscle fasciculations.

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- Hypermagnesemia: depression, weakness, hyporeflexia, flaccid paralysis, poor pulse quality, bradycardia, and/or respiratory distress.



DIFFERENTIAL DIAGNOSIS

- Hypomagnesemia: other electrolyte abnormalities such as hypocalcemia, hypokalemia, and hyperkalemia; cardiac disease, musculoskeletal disorders, seizure disorders, and intoxications.
- Hypermagnesemia: gastrointestinal disease, neuromuscular disease, renal disease, cardiac disease, intoxications, and other electrolyte disorders such as hyperkalemia.



DIAGNOSTICS

- Indications for determining magnesium status are the presence of clinical signs associated with hypomagnesemia or hypermagnesemia, and the presence of disease processes which may lead to alterations in total body magnesium concentrations.
- Determination of serum total and/or ionized magnesium concentrations is most commonly done to determine magnesium status. Serum ionized magnesium concentrations are preferred over serum total concentrations because ionized magnesium is thought to be the physiologically active component of the electrolyte and a more accurate indicator of intracellular magnesium stores.
- If serum total or ionized magnesium concentrations are not routinely measured, the presence of hypokalemia, hyponatremia, or hypocalcemia may indicate concurrent hypomagnesemia, especially if the patient's hypokalemia is refractory to potassium supplementation.
- Alternate methods of evaluating magnesium status may include mononuclear blood cell magnesium concentrations or quantifying retention of a loading dose of magnesium. Human studies suggest that retention of greater than 40–50% of an administered magnesium load indicates total body magnesium depletion, whereas retention of less than 20% indicates adequate magnesium stores. (These tests are rarely done in veterinary medicine.)
- Urinary magnesium determination may help differentiate abnormalities associated with urinary magnesium loss from diseases of decreased intake, gastrointestinal loss, or alterations in magnesium distribution within the body.
- Electrodiagnostics (e.g., electrocardiography and electromyelography) may reveal the effects of hypomagnesemia or hypermagnesemia but will not help differentiate the cause.



THERAPEUTICS

- Hypomagnesemia: correct underlying problem, supplement magnesium, and discontinue drugs that cause renal magnesium loss.

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- Hypermagnesemia: correct underlying problem, discontinue magnesium-containing medications and nutritional supplements, diurese and promote renal magnesium excretion, support patient's cardiopulmonary system, and antagonize the effects of magnesium.

Drugs of Choice

Hypomagnesemia

- For rapid correction of serum magnesium concentrations, magnesium chloride or magnesium sulfate can be administered IV in normal saline or 5% dextrose in water by continuous rate infusion at 0.75–1.0 mEq/kg per day for the first 24–48 hours.
- A lower dose of 0.3–0.5 mEq/kg per day can then be used for an additional 3–5 days, if needed.
- For the treatment of life-threatening ventricular arrhythmias, a dose of 0.15–0.3 mEq/kg of magnesium chloride or magnesium sulfate diluted in normal saline or 5% dextrose in water can be slowly administered IV over 5–15 minutes.
- In stable patients with mild hypomagnesemia, magnesium chloride, magnesium gluconate, magnesium oxide, or magnesium hydroxide can be administered orally at a dose of 1–2 mEq/kg every 24 hours.

Hypermagnesemia

- Diurese with 0.9% NaCl at 2–4 mL/kg per hour IV.
- Furosemide at 1–4 mg/kg every 8–12 hours PO, SQ, IM, or IV to increase renal magnesium excretion.
- Calcium gluconate at 50 mg/kg IV over 15–30 minutes to antagonize the effects of magnesium at the neuromuscular junction and to reverse the cardiotoxic effects of hypermagnesemia.
- Physostigmine at 0.02 mg/kg every 12 hours IV to antagonize the neurotoxic effects of hypermagnesemia.

Precautions/Interactions

Hypomagnesemia

- Parenteral administration of magnesium sulfate may result in hypocalcemia due to chelation of calcium with the sulfate. Therefore, magnesium chloride should be administered when hypocalcemia is concurrently present.
- Side effects of supplemental magnesium therapy include hypotension, atrioventricular, and bundle branch blocks. Adverse effects are usually associated with intravenous boluses rather than continuous rate infusions.
- Monitor ECG and blood pressure closely if administering magnesium rapidly IV.
- Stop or slow the infusion if heart rate or blood pressure decreases dramatically or if first-, second-, or third-degree atrioventricular block or bundle branch block develops.
- Decrease supplemental magnesium dose by 50% if renal disease or atrioventricular block is present and monitor ECG.

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- Avoid drugs such as aminoglycosides, cisplatin, and cyclosporine because they can cause renal tubular injury and lead to increased renal loss of magnesium.
- Avoid drugs such as furosemide, thiazides, and mannitol because they can increase renal loss of magnesium.

Hypermagnesemia

- Avoid magnesium-containing drugs such as cathartics, laxatives, and antacids.
- Monitor ECG while administering calcium gluconate.
- Do not administer furosemide to dehydrated, hypotensive, or shocky patients.

Appropriate Healthcare

- For patients with severe hypo- or hypermagnesemia, institute mechanical ventilation if the patient is hypoventilating and hypoxemic.
- For patients with severe hypo- or hypermagnesemia, consider placing a gastrostomy tube for nutritional support and to reduce the likelihood of regurgitation and aspiration pneumonia if the patient is dysphagic secondary to esophageal weakness.
- For patients with hypermagnesemia, institute hemodialysis or peritoneal dialysis if clinical signs are severe and the patient is in anuric or oliguric acute renal failure.

Diet

- Many veterinary critical care diets contain low concentrations of magnesium (range 0.1–0.22 mg/kcal). Given that many critically ill patients are fed at or below their resting energy requirement, the actual intake of magnesium may be well below the concentration needed to replete a magnesium-deficient animal.
- Magnesium supplementation in standard total parenteral nutrition formulations (0.13–0.22 mEq/kg per day) is also below the concentration recommended to treat hypomagnesemia (0.3–1.0 mEq/kg per day).
- Based on low concentrations of magnesium in critical care diets and total parenteral nutrition formulations, animals with moderate to marked hypomagnesemia will likely require intravenous or oral magnesium supplementation to normalize serum total or ionized magnesium concentrations, especially if they have disease processes resulting in continued loss of magnesium from the gastrointestinal tract and/or kidneys.

Activity

- Both moderate to marked hypo- and hypermagnesemia can cause weakness, and the patient may require assistance when standing or walking.

Surgical Considerations

- Due to magnesium's effect on the cardiovascular and neuromuscular systems, moderate to marked hypo- or hypermagnesemia should be corrected prior to anesthesia and surgery.

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COMMENTS

- 1 mEq of magnesium is equal to 12 mg of magnesium which is equal to 0.5 mmol of magnesium. To convert from mg/dL to mEq/L, divide by 1.2.

Client Education

- Monitor for signs of hypo- or hypermagnesemia. Unfortunately, many of the clinical signs for these electrolyte disorders are nonspecific (e.g., weakness, lethargy, and depression).

Patient Monitoring

- Recheck serum total or ionized magnesium concentrations until normalized. The recheck interval will depend on the severity of the electrolyte disorder, clinical stability of the patient, and underlying disease process.

Prevention/Avoidance

- Work with a veterinarian to resolve or control the underlying disease process that leads to the animal's hypo- or hypermagnesemia.

Possible Complications

- Severe, untreated hypo- and hypermagnesemia can be fatal.

Expected Course and Prognosis

- Depending on the underlying disease process, prognosis can range from guarded to good. The prognosis for correcting and resolving hypermagnesemia secondary to acute anuric renal failure is likely guarded, while the prognosis for correcting and resolving hypomagnesemia secondary to lactation is good.

Abbreviations

ECG: electrocardiogram
IM: intramuscular
IV: intravenous
NaCl: sodium chloride
PO: by mouth (per os)
PTH: parathyroid hormone
SQ: subcutaneous

See Also

Hyperkalemia
Hypokalemia

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chapter 61

Metabolic Acidosis



DEFINITION/OVERVIEW

- Decrease in plasma pH associated with a decrease in HCO_3^- concentration (dogs, <18 mEq/L; cats, <16 mEq/L) and a compensatory decrease in PCO_2.



ETIOLOGY/PATHOPHYSIOLOGY

- Metabolic acidosis may develop secondary to hyperphosphatemia (hyperphosphatemic acidosis), corrected hyperchloremia (hyperchloremic acidosis), and accumulation of metabolically produced strong anions (strong ion gap or high-AG acidosis). The most common causes of metabolic acidosis in critical care patients are presented in Box 61.1.
- Metabolic acidoses, especially lactic acidosis, uremic acidosis, and diabetic ketoacidosis, occur commonly in critically ill patients.
- Hyperphosphatemic acidosis.
  - Increase in plasma weak acids (e.g., inorganic phosphate) is associated with metabolic acidosis and increased AG. At pH of 7.4, a 1 mg/dL increase in phosphate concentration is associated with a 0.58 mEq/L decrease in HCO_3^- and a 0.58 mEq/L increase in AG.
- Hyperchloremic acidosis.
  - Hyperchloremic acidosis may be caused by chloride retention (e.g., renal failure, renal tubular acidosis), excessive loss of sodium relative to chloride (e.g., diarrhea), or by administration of substances containing more chloride than sodium as compared with normal extracellular fluid composition (e.g., administration of KCl, 0.9% NaCl).
  - In critically ill patients the source of hyperchloremic acidosis is often iatrogenic. A common cause is related to the volume of saline infused during resuscitation from shock.
  - Acidemia is usually not severe in patients with hyperchloremic acidosis.

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Companion website: www.fiveminutevet.com/emergency

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BOX 61.1. Common causes of metabolic acidosis in patients who are critically ill.

- Preexisting disease process
  - Hyperphosphatemic acidosis
    - Renal failure
    - Urethral obstruction
  - Hyperchloremic acidosis
    - Renal tubular acidosis
    - Renal failure
    - Diarrhea
  - High-AG acidosis
    - Diabetes mellitus
    - Renal failure
- Labile feature of an evolving process
  - Hyperphosphatemic acidosis
    - Phosphate-containing enemas
    - Acute renal failure
  - Hyperchloremic acidosis
    - Diarrhea
    - Post correction of chronic respiratory alkalosis
  - High-AG acidosis
    - Diabetic ketoacidosis
    - Uremic acidosis
    - Lactic acidosis
    - Toxicities (e.g., ethylene glycol, salicylates)
- Iatrogenic
  - Hyperphosphatemic acidosis
    - IV phosphate administration
  - Hyperchloremic acidosis
    - 0.9% NaCl administration
    - KCl administration
    - Total parenteral nutrition
  - High-AG acidosis
    - Propylene glycol intravenously as drug vehicle (e.g., nitroglycerin, diazepam)
    - Gelatin administration (increases anion gap without causing acidosis)

AG, anion gap; IV, intravenous; KCl, potassium chloride; NaCl, sodium chloride.
Source: Adapted from de Morais HA, Bach JF, DiBartola SP. Metabolic acid-base disorders in the critical care unit. Vet Clin North Am Small Anim Pract 2008; 38:563, with permission.

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- High-AG acidosis.
  - Increase in the concentration of other strong anions: addition (e.g., ethylene glycol toxicity), excessive production (e.g., lactate produced by prolonged anaerobic metabolism), or renal retention (e.g., renal failure) of strong anions other than chloride causes metabolic acidosis without increasing chloride concentration (so-called normochloremic or high-AG metabolic acidosis).
  - A mnemonic to remember causes of high-AG metabolic acidosis is A MUD PILE:

A: Aspirin (salicylates)
M: Metaldehyde
U: Uremia
D: Diabetic ketoacidosis
P: Paraldehyde
I: Iron
L: Lactic acidosis
E: Ethanol and ethylene glycol

Systems Affected

- Respiratory: increased H^+ stimulates peripheral and central chemoreceptors to increase alveolar ventilation; hyperventilation decreases PCO_2, which counters the effects of low plasma HCO_3^- on pH. In dogs, a decrease of approximately 0.7 mmHg in PCO_2 is expected for each 1 mEq/L decrease in plasma HCO_3^-. Little is known about compensation in cats, but it appears to be almost nonexistent.
- Renal/urologic: the kidneys increase net acid excretion, primarily by increasing excretion of NH_4^+ and chloride. This compensatory mechanism is not very effective in cats.
- Cardiovascular: a fall in pH results in an increase in sympathetic discharge but simultaneously causes a decrease in the responsiveness of the cardiac myocytes and vascular smooth muscle to the effects of catecholamines. In mildly acidemic conditions (pH greater than 7.2), the effects of increased sympathetic stimulation predominate and result in a mild increase in heart rate and cardiac output. More severe acidemia (pH below 7.1), especially if acute, may decrease cardiac contractility and predispose the heart to ventricular dysrhythmias and ventricular fibrillation.
- Endocrine/metabolic: acidemia produces insulin resistance that impairs peripheral uptake of glucose and inhibits anaerobic glycolysis by inhibiting phosphofructokinase.



SIGNALMENT/HISTORY

- Any breed, age, or sex of dogs and cats.

Risk Factors/Causes

- Preexisting diseases with a high risk for metabolic acidosis.
  - Chronic renal failure

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  - Diabetes mellitus
  - Hypoadrenocorticism
- Patients with poor tissue perfusion or hypoxia are at risk of developing lactic acidosis.

Historical Findings

- History associated with chronic disease processes that may lead to metabolic acidosis (e.g., renal failure, diabetes mellitus, hypoadrenocorticism, or diarrhea).
- Exposure to toxins (e.g., ethylene glycol, salicylate, and paraldehyde).



CLINICAL FEATURES

- Generally related to the underlying disease.
- Depression in severely acidotic patients.
- Tachypnea in some patients results from compensatory increase in ventilation.
- Kussmaul's respiration, typically seen in human beings with metabolic acidosis, is not commonly observed in dogs and cats.



DIFFERENTIAL DIAGNOSIS

- Low plasma HCO_3^- and hyperchloremia may also be compensatory in animals with chronic respiratory alkalosis, in which PCO_2 is low and pH is high or near normal, despite decreased HCO_3^- and increase in chloride concentration. Blood gas determination is required to differentiate.



DIAGNOSTICS

- Blood gas analysis reveals low HCO_3^-, low PCO_2, and low pH.
- Excessive heparin (>10% of the sample) decreases HCO_3^- and the degree of the acidosis may be overestimated.
- Anion gap.
  - Metabolic acidoses are traditionally divided into hyperchloremic and high AG by means of the AG. AG, the difference between the measured cations and the measured anions, is calculated as $AG = Na^+ - \left(HCO_3^- + Cl^-\right)$ or $AG = \left(Na^+ + K^+\right) - \left(HCO_3^- + Cl^-\right)$, depending on the preference of the clinician or laboratory. Normal values with potassium included in the calculation are usually 12–24 mEq/L in dogs and 13–27 mEq/L in cats. The negative charges of albumin are the major contributors to the normal AG; this should be taken into account when evaluating AG in patients with hypoalbuminemia. At pH 7.4 in dogs, a decrease of 1 g/dL in albumin is associated with a decrease of 4.1 mEq/L in the AG.

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  - Normal AG: hyperchloremic metabolic acidosis.
  - High AG: hyperphosphatemic acidosis, uremic acidosis, ketoacidosis, lactic acidosis, toxicities (e.g., ethylene glycol).
- Hyperglycemia: consider diabetes mellitus.
- Azotemia: consider renal failure.
- Hyperphosphatemia: consider renal failure, hypertonic sodium phosphate enema toxicity, and toxicity due to urinary acidifiers containing phosphate.
- High lactate concentration: consider lactic acidosis associated with poor tissue perfusion or poor metabolism of lactate (e.g., liver disease and lymphoma).
- Hyperkalemia: commonly associated with, but almost never caused by the acidosis. Hyperkalemia results from the disease process causing the metabolic acidosis (e.g., poor renal function in renal failure or lack of insulin in diabetes mellitus), not from the acidosis itself. Correction formulas to adjust potassium concentration based on pH changes should not be used.



THERAPEUTICS

- Acid–base disturbances are secondary phenomena; successful resolution depends on diagnosis and treatment of the underlying disease process.
- Treat patients with blood pH <7.1 aggressively while pursuing the definitive diagnosis.
- Discontinue drugs that may cause or contribute to metabolic acidosis.

Drugs of Choice

- $NaHCO_3$ may help patients with hyperchloremic, hyperphosphatemic or uremic acidosis but not patients with lactic acidosis or diabetic ketoacidosis.
  - Estimation of HCO_3^- dose: dogs, 0.3 × body weight (kg) × (21 – patient HCO_3^-); cats, 0.3 × body weight (kg) × (19 – patient HCO_3^-). Give half of this dose slowly IV and reevaluate blood gases before deciding on the need for additional administration. An empirical dose of 2 mEq/kg followed by reevaluation of blood gas status is safe in most patients.
  - Potential complications of $NaHCO_3$ administration: volume overload resulting from administered sodium, tetany from low ionized calcium concentration, increased affinity of hemoglobin for oxygen, paradoxical CNS acidosis, overshoot metabolic alkalosis, and hypokalemia.
- Hyperchloremic acidosis: $NaHCO_3$ is effective and should be considered whenever pH <7.2.
- Uremic acidosis: efficacy of $NaHCO_3$ in acute therapy of uremic acidosis is related to the shift of phosphate inside the cells and consequent amelioration of hyperphosphatemic acidosis.
- Lactic acidosis: $NaHCO_3$ increases lactate production and is of little to no value in lactic acidosis. Therapy should be directed at augmenting oxygen delivery to the tissues and

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reestablishing cardiac output. Small titrated doses of $NaHCO_3$ can be used as a temporizing measure to maintain HCO_3^- above 5 mEq/L, if needed.

- Diabetic ketoacidosis: $NaHCO_3$ adversely affects outcome in humans with diabetic ketoacidosis even when pH is below 7.0. Administration of $NaHCO_3$ to ketoacidotic patients cannot be recommended at any pH. Therapy should be directed at insulin and fluid administration. Reestablishing plasma volume and renal perfusion will allow the kidneys to excrete ketoanions, replacing them with chloride.

Precautions/Interactions

- Avoid $NaHCO_3$ in patients with respiratory acidosis because it generates CO_2. Patients with respiratory acidosis cannot adequately excrete CO_2, and increased PCO_2 will further decrease the pH.
- Avoid diuretics that act in the distal nephron (e.g., spironolactone).
- Avoid carbonic anhydrase inhibitors (e.g., acetazolamide).
- Use $NaHCO_3$ cautiously in patients with congestive heart failure because the sodium load may cause decompensation of the heart failure.

Activity

- Restriction dictated by the underlying disease leading to metabolic acidosis.

COMMENTS

Patient Monitoring

- Acid–base status: frequency dictated by the underlying disease and patient response to treatment.

Possible Complications

- Myocardial depression and ventricular arrhythmias.

Expected Course and Prognosis

- Prognosis is dependent on the severity of the underlying disease causing metabolic alkalosis and response to therapy.

Synonyms

- Nonrespiratory acidosis
- Hyperchloremic acidosis: normal-AG acidosis
- Normochloremic acidosis: high-AG acidosis
- Hyperphosphatemic acidosis: metabolic acidosis resulting from high phosphate concentration

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- Organic acidosis: metabolic acidosis resulting from accumulation of organic anions (e.g., ketoacidosis, uremic acidosis, and lactic acidosis)
- Dilutional acidosis: metabolic acidosis resulting from increased free water in plasma

Abbreviations

AG: anion gap
Cl: chloride
CNS: central nervous system
CO_2: carbon dioxide
H^+: hydrogen ion
HCO_3^-: bicarbonate
IV: intravenous
K: potassium
KCl: potassium chloride
Na: sodium
NaCl: sodium chloride
$NaHCO_3$: sodium bicarbonate
NH_4: ammonium
O_2: oxygen
PCO_2: carbon dioxide tension
pH: acid–base balance

Suggested Reading

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chapter 62

Metabolic Alkalosis

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DEFINITION/OVERVIEW

- Increase in pH associated with an increase in plasma HCO_3^- concentration (dogs >24 mEq/L; cats >22 mEq/L) and a compensatory increase in PCO_2.

ETIOLOGY/PATHOPHYSIOLOGY

- Metabolic alkalosis may develop secondary to corrected hypochloremia (hypochloremic alkalosis) or hypoalbuminemia (hypoalbuminemic alkalosis). The most common causes of metabolic alkalosis in critical care patients are presented in Box 62.1.
- Hypochloremic alkalosis.
  - Fluid rich in chloride and H^+ may be lost via the alimentary tract (e.g., vomiting, stomach drainage) or kidneys (e.g., diuretic therapy). Loss of chloride and H^+ is associated with an increase in plasma HCO_3^- concentration. With chloride loss and volume depletion, the kidneys reabsorb sodium with HCO_3^- instead of chloride, perpetuating the metabolic alkalosis.
  - Administration of fluids with high sodium and poor chloride concentration (e.g., sodium bicarbonate) also may result in transient metabolic alkalosis.
  - Hypochloremic alkalosis is not common in dogs and cats in a critical care setting and is usually secondary to vomiting or diuretic administration. Alkalemia is rare unless the patient also has respiratory alkalosis.
- Hypoalbuminemic alkalosis.
  - Decrease in plasma weak acids (e.g., albumin) is associated with metabolic alkalosis.
  - Hypoalbuminemic alkalosis is common in critically ill patients, but it rarely leads to alkalemia. However, presence of hypoalbuminemia complicates identification of increased unmeasured anions (e.g., lactate, ketoanions) because hypoalbuminemia decreases AG. At plasma pH of 7.4 in dogs, each decrease of 1 g/dL in albumin concentration is associated with a decrease of 4.1 mEq/L in the AG. Thus, the severity of the underlying disease leading to metabolic acidosis may be underestimated if the effects of hypoalbuminemia on pH, HCO_3^- concentration, and AG are not considered.

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BOX 62.1. Common causes of metabolic alkalosis in patients who are critically ill.

- Preexisting disease process
  - Hypoalbuminemic alkalosis
    - Liver failure
    - Protein-losing enteropathy
    - Protein-losing nephropathy
  - Hypochloremic alkalosis
    - Diuretic therapy
    - Vomiting of stomach contents
- Labile feature of an evolving process
  - Hypoalbuminemic alkalosis
    - Vasculitis
    - Third space losses
  - Hypochloremic alkalosis
    - Vomiting of stomach contents
    - Post correction of chronic respiratory acidosis
- Iatrogenic
  - Hypochloremic alkalosis
    - Bicarbonate administration
    - Diuretic therapy
    - Stomach draining

Source: Adapted from de Morais HA, Bach JF, DiBartola SP. Metabolic acid-base disorders in the critical care unit. Vet Clin North Am Small Anim Pract 2008; 38:561, with permission.

Systems Affected

- Respiratory: low H^+ reduces alveolar ventilation. Hypoventilation increases PCO_2 and helps offset the effects of high plasma HCO_3^- on pH. In dogs, an increase of approximately 0.7 mmHg in PCO_2 can be expected for each 1 mEq/L increase in plasma HCO_3^-. Limited data are available for cats, but the degree of respiratory compensation appears to be similar.
- Renal/urologic: the kidneys rapidly and effectively excrete excessive alkali. In patients with chloride deficiency (and, less importantly, volume depletion), the kidneys cannot excrete the excessive alkali and metabolic alkalosis is maintained. In these patients, chloride administration is required for renal compensation to occur, whereas volume expansion will hasten compensation.
- Nervous: muscle twitching and seizures occur rarely in dogs. Metabolic alkalosis and associated hypokalemia may precipitate hepatic encephalopathy in patients with liver failure.

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SIGNALMENT/HISTORY

- Any breed, age, or sex of dogs and cats.

Risk Factors/Causes

- Administration of loop or thiazide diuretics
- Vomiting
- Stomach drainage
- Vasculitis (hypoalbuminemia)

Historical Findings

- Administration of loop diuretics (e.g., furosemide) or thiazides
- Vomiting



CLINICAL FEATURES

- Signs related to the underlying disease or accompanying potassium depletion (e.g., weakness, cardiac arrhythmias, and ileus)
- Muscle twitching caused by low ionized calcium concentration
- Dehydration in volume-depleted patients
- Muscle twitching and seizures in patients with neurologic involvement (rare)



DIFFERENTIAL DIAGNOSIS

- High plasma HCO_3^- and hypochloremia in animals can also be compensating for chronic respiratory acidosis, in which PCO_2 is high and pH is low despite high HCO_3^- and low chloride concentration; blood gas determination is required to differentiate.



DIAGNOSTICS

- Metabolic alkalosis: blood gas analysis reveals high HCO_3^-, high PCO_2, and high pH.
  - Excessive heparin (>10% of the sample) decreases HCO_3^- and the degree of the alkalosis may be underestimated.
- Hypochloremic alkalosis: associated with a decrease in corrected chloride concentration.
- Hypoalbuminemic alkalosis: associated with a decrease in albumin concentration. *In vitro*, a 1 g/dL decrease in albumin concentration is associated with an increase in pH of 0.093 in cats and 0.047 in dogs.

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- Hypokalemia: common in patients with metabolic alkalosis. It likely results from the metabolic alkalosis or the underlying problem (e.g., vomiting of stomach contents or loop diuretic administration); hypokalemia-induced metabolic alkalosis does not occur in dogs and cats.



THERAPEUTICS

- Acid–base disturbances are secondary phenomena. Diagnosis and treatment of the underlying disease process are integral to the successful resolution of acid–base disorders.
- Severe alkalemia is uncommon but may be life threatening. Patients with chronic respiratory disease and respiratory alkalosis are at risk of developing severe alkalemia if they start vomiting or receive diuretics.
- Discontinue drugs that may cause metabolic alkalosis.

Drugs of Choice

- Hypochloremic alkalosis.
  - If there is evidence of volume depletion, the deficit should be repaired with a chloride-containing fluid (e.g., 0.9% NaCl).
  - If the metabolic alkalosis is associated with hypokalemia and total body potassium deficits, correcting the deficit with KCl is a particularly effective way to reverse the alkalosis.
- Hypoalbuminemic alkalosis.
  - Treatment for hypoalbuminemic alkalosis should be directed at the underlying cause and the decreased colloid oncotic pressure.

Precautions/Interactions

- In the absence of hypovolemia, the amount of saline required to correct hypochloremic alkalosis introduces a risk for volume overload.
- Avoid chloride-free fluids; they may correct volume depletion but will not correct metabolic alkalosis.
- Avoid using salts of potassium without chloride (e.g., potassium phosphate); potassium will be excreted in the urine and will correct neither the alkalosis nor the potassium deficit.

Alternative Drugs

- Acetazolamide (5–10 mg/kg every 8 hours PO) can be used as adjunct therapy for the correction of metabolic alkalosis in critical care patients.

Activity

- Restriction should be dictated by the underlying disease leading to metabolic alkalosis.

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COMMENTS

Patient Monitoring

- Acid–base status: frequency dictated by the underlying disease and patient response to treatment.

Expected Course and Prognosis

- Prognosis depends on the severity of the underlying disease causing metabolic alkalosis and response to therapy.

Synonyms

- Nonrespiratory alkalosis
- Hypochloremic alkalosis: metabolic alkalosis caused by low chloride concentration
- Hypoalbuminemic alkalosis: metabolic alkalosis caused by low albumin concentration
- Concentration alkalosis: metabolic alkalosis resulting from decreased free water in plasma
- Contraction alkalosis: metabolic alkalosis formerly attributed to volume contraction but now known to be caused by chloride depletion. Volume depletion is a common but not essential feature.

Abbreviations

AG: anion gap
H^+: hydrogen ion
HCO_3^-: bicarbonate
KCl: potassium chloride
NaCl: sodium chloride
PCO_2: carbon dioxide tension
pH: acid–base balance
PO: by mouth

Suggested Reading

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Author: Helio Autran de Morais, DVM, PhD

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chapter 63

Metaldehyde Toxicity

DEFINITION/OVERVIEW

- Syndrome of toxicity that occurs in dogs and cats after ingestion of metaldehyde, a common chemical in commercial slug baits.

ETIOLOGY/PATHOPHYSIOLOGY

- Metaldehyde is a tetramer of acetaldehyde and is a common component of commercial slug baits.
- Mechanism of toxicity unknown in small animals.
- LD_{50} is 100 mg/kg in dogs, and 207 mg/kg in cats. Clinical signs can occur after ingestion of 2 mg/kg. Clinical signs of toxicity can occur within minutes to several hours after ingestion.
- Proposed mechanism of toxicity involves the conversion of metaldehyde to acetaldehyde in the stomach when exposed to an acidic environment, with possible decreased concentrations of the inhibitory neurotransmitter GABA, decreased norepinephrine, and decreased 5-HT. The decreased concentration of inhibitory neurotransmitters has been linked with increased incidence of seizure activity and death.

Systems Affected

- Neurologic: nystagmus, muscle rigidity, tremors, opisthotonus, hyperesthesia, and seizures. Transient blindness has been reported.
- Musculoskeletal: muscle rigidity and tremors or fasciculations.
- Gastrointestinal: vomiting and diarrhea.
- Metabolic: respiratory alkalosis from panting, metabolic acidosis, or hyperthermia.
- Respiratory: panting, respiratory depression, and failure within hours of ingestion/exposure.
- Hepatobiliary: hepatic failure delayed up to 2–3 days after exposure.

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Incidence/Prevalence

- Unknown.

Geographic Distribution

- Can occur anywhere, but more common in geographic locations that have problems with slug infestations in gardens (i.e., southern United States, Hawaiian Islands, and Pacific coast).



SIGNALMENT/HISTORY

Species

- Dogs and cats.

Historical Findings

- History of exposure or ingestion.
- Acute onset of tremors, hyperexcitability, nystagmus, vomiting, diarrhea, or seizures.

Physical Examination Findings

- Tachycardia
- Blindness
- Tachypnea
- Nystagmus
- Tremors
- Hyperesthesia
- Ataxia
- Seizures

Risk Factors/Causes

- Exposure to metaldehyde.
- Free-roaming activity in a geographic location where slugs and gardeners coexist.

DIFFERENTIAL DIAGNOSIS

- Differential diagnoses include intoxication with bromethalin, 1080 compound, strychnine, chlorinated hydrocarbons, organophosphates, zinc phosphide, methylxanthines, tremorigenic mycotoxins, lead, and illicit recreational drugs such as cocaine, amphetamine, and methamphetamine.

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Complete Blood Count/Biochemistry/Urinalysis

- CBC: stress leukogram, thrombocytopenia in severe cases that are hyperthermic.
- Biochemistry: elevated ALT and CPK from muscle tremors, hyperglycemia, or hyperbilirubinemia.
- Urinalysis: myoglobinuria from severe muscle tremors.

Other Laboratory Tests

- Gas chromatography can be used to determine presence of metaldehyde or its metabolite acetaldehyde in urine, gastric contents, and plasma.

Diagnostic Procedures

- CBC, chemistry, urinalysis, tremorigen assay (Michigan State University Veterinary Diagnostic Laboratory).



THERAPEUTICS

- Decrease further absorption/induction of emesis.
- Treat tremors.
- Anticonvulsant drugs.
- Correct acid–base disturbances.
- Correct hyperthermia.
- Correct dehydration.
- Antiemetics.

Drugs of Choice

Apomorphine

- Induces emesis
- 0.03–0.04–mg/kg IV

Methocarbamol

- Muscle relaxant
- 55–220 mg/kg IV slowly to effect; no more than 330 mg/kg per 24 hours

Diazepam

- Muscle relaxant, control of seizures
- 0.5–1 mg/kg IV or per rectum

Phenobarbital

- Anticonvulsant
- 16–20 mg/kg IV may be administered as a bolus if animals are in status epilepticus, or divided into 4 mg/kg doses and administered every 30–60 minutes until total dose has

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been administered. Use caution because phenobarbital and acetaldehyde are metabolized by the cytochrome p450 enzyme system, and administration of phenobarbital can potentially decrease the metabolic degradation and excretion of acetaldehyde.

Activated Charcoal

- Prevents further absorption; repeat every 6–8 hours as long as patient is able to swallow and has an intact gag reflex.
- Administer dose according to manufacturer's recommendations.
- Sorbitol-containing activated charcoal solutions or concentrate may promote elimination from gastrointestinal tract but should be avoided in animals with diarrhea and vomiting.

Intravenous Crystalloid Fluids

- Dose is dependent on degree of hyperthermia, dehydration, and acid–base and electrolyte abnormalities. Make sure to correct fluid deficits, acid–base and electrolyte abnormalities, and provide maintenance needs.

Antiemetics

- Dolasetron (0.6 mg/kg IV every 24 hours)
- Ondansetron (1 mg/kg IV every 8–12 hours)
- Maropitant (1 mg/kg IV, SQ every 24 hours; 2 mg/kg PO every 24 hours)
- Metoclopramide (1–2 mg/kg per day IV CRI)

Contraindications

- Do not submerge in ice water baths.
- Caution with sorbitol in dehydrated animals with vomiting or diarrhea.

COMMENTS

Patient Monitoring

- Check liver enzyme activities several days after exposure because delayed hepatic failure can occur.

Prevention/Avoidance

- Prevent free roaming in geographic locations where slugs and gardeners coexist.

Possible Complications

- DIC
- Rhabdomyolysis
- Renal failure

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- Neurologic sequelae
- Hepatic failure
- Death

Expected Course and Prognosis

- Half-life of metaldehyde in dogs is unknown. Prognosis is generally good if exposure is witnessed and emesis is induced before onset of clinical signs. Prognosis is worse if severe hyperthermia, DIC, rhabdomyolysis, and organ failure occur. Survival of 83% was reported in one study.

Abbreviations

ALT: alanine transaminase
CBC: complete blood count
CPK: creatine phosphokinase
CRI: constant rate infusion
DIC: disseminated intravascular coagulation
GABA: gamma-aminobutyric acid
IV: intravenous
LD_{50}: lethal dose that kills 50% of animals tested
PO: by mouth (per os)
SQ: subcutaneous
5-HT: 5-hydroxytryptamine (serotonin)

Suggested Reading

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Author: Elisa M. Mazzaferro, MS, DVM, PhD

chapter 64

Murmurs

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DEFINITION/OVERVIEW

- Vibrations caused by disturbed blood flow.

Timing of Murmurs

- Systolic murmurs occur between S1 and S2 (systole).
- Diastolic murmurs occur between S2 and S1 (diastole).
- Continuous and to-and-fro murmurs occur throughout all or most of the cardiac cycle.
- Continuous murmurs are usually accentuated near S2 and to-and-fro murmurs are usually absent near S2.

Grading Scale for Murmurs

- Grade I: barely audible.
- Grade II: soft, but easily auscultated. Does not radiate far from PMI.
- Grade III: intermediate loudness; heard easily some distance from PMI, but not to opposite side of chest; most hemodynamically important murmurs are at least grade III.
- Grade IV: loud murmur radiating widely, often including opposite side of chest.
- Grade V: very loud, audible with stethoscope barely touching the chest; palpable thrill.
- Grade VI: very loud, audible without the stethoscope touching the chest; palpable thrill.

Configuration

- Plateau murmurs have uniform loudness and are typical of regurgitant murmurs such as mitral and tricuspid insufficiency and ventricular septal defect.
- Crescendo-decrescendo murmurs get louder and then softer and are typical of ejection murmurs such as pulmonic and aortic stenosis and atrial septal defect.
- Decrescendo murmurs start loud and then get softer and are typical of diastolic murmurs such as aortic or pulmonic insufficiency and mitral or tricuspid stenosis.

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Location

Dogs

- Mitral area: left fifth intercostal space at costochondral junction.
- Aortic area: left fourth intercostal space above costochondral junction.
- Pulmonic area: left second to fourth intercostal space at sternal border.
- Tricuspid area: right third to fifth intercostal space near costochondral junction.

Cats

- Mitral area: left fifth to sixth intercostal space one-fourth ventrodorsal distance from sternum.
- Aortic area: left second to third intercostal space just above the pulmonic area.
- Pulmonic area: left second to third intercostal space one-third to one-half ventrodorsal distance from sternum.
- Tricuspid area: right fourth to fifth intercostal space one-fourth ventrodorsal distance from sternum.



ETIOLOGY/PATHOPHYSIOLOGY

- Disturbed blood flow associated with high flow through normal or abnormal valves or with structures vibrating in the blood flow.
- Flow disturbances associated with outflow obstruction or forward flow through stenosed valves or into a dilated great vessel.
- Flow disturbances associated with regurgitant flow through an incompetent valve, septal defect, or patent ductus arteriosus.

Systems Affected

- Cardiovascular.



SIGNALMENT/HISTORY

Species

- Dogs and cats.

Historical Findings

- Relate to cause of the murmur.
- Owner may report having felt a cardiac thrill.

Risk Factors/Causes

- Cardiac disease.

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CLINICAL FEATURES

- Physical findings vary with the cause of the murmur.
- Murmurs may be systolic, diastolic, or continuous.
- Murmurs vary in loudness, radiation, and PMI depending on the cause and severity.
- Evaluation of jugular and femoral pulses may be helpful in narrowing the differential diagnosis (e.g., jugular pulse with tricuspid insufficiency, bb shot pulse with patent ductus arteriosus).

DIFFERENTIAL DIAGNOSIS

Differential Signs

- Must differentiate from other abnormal heart sounds – split sounds, ejection sounds, gallop rhythms, and clicks.
- Must differentiate from abnormal lung sounds and pleural rubs; listen to see if timing of abnormal sound is correlated with respiration or heartbeat.

Differential Causes

- Pale mucous membranes support diagnosis of anemic murmur.
- Location and radiation of murmur and timing during cardiac cycle can help determine cause.

Systolic Murmurs

- Mitral and tricuspid valve endocardiosis
- Cardiomyopathy and AV valve insufficiency
- Physiologic flow murmurs
- Anemia
- Mitral and tricuspid valve dysplasia
- Systolic anterior mitral motion
- Dynamic right ventricular outflow obstruction
- Dynamic subaortic stenosis
- Atrial septal defect
- Ventricular septal defect
- Pulmonic stenosis
- Aortic stenosis
- Tetralogy of Fallot
- Mitral and tricuspid valve endocarditis
- Hyperthyroidism
- Heartworm disease

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Continuous or To-and-Fro Murmurs

- Patent ductus arteriosus
- Ventricular septal defect with aortic regurgitation
- Aortic stenosis with aortic regurgitation

Diastolic Murmurs

- Mitral and tricuspid valve stenosis
- Aortic and pulmonic valve endocarditis

Age-Related Factors

- Murmurs present since birth are generally associated with a congenital defect or physiologic flow murmur.
- Acquired murmurs in geriatric, small-breed dogs are usually associated with degenerative valve disease.
- Acquired murmurs in large-breed dogs are usually associated with dilated cardiomyopathy.
- Acquired murmurs in geriatric cats are usually associated with cardiomyopathy or hyperthyroidism.



DIAGNOSTICS

Complete Blood Count/Biochemistry/Urinalysis

- Anemia in animals with murmurs secondary to anemia.
- Polycythemia in animals with right-to-left shunting congenital defects.
- Leukocytosis with left shift in animals with endocarditis.

Imaging

- Thoracic radiography is useful for evaluating heart size and pulmonary vasculature in the hope of determining cause and significance of murmur.
- Echocardiography is recommended when a cardiac cause is suspected and the nature of the defect is unknown.
- Doppler studies sometimes required to confirm cause of murmur.

Diagnostic Procedures

- Electrocardiography may be useful in assessing heart enlargement patterns in animals with murmurs.
- Blood cultures and serology/PCR for *Bartonella* if endocarditis is suspected.

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Pathologic Features

- Vary with underlying cause of the murmur.



THERAPEUTICS

Drugs of Choice

- None unless heart failure has developed secondary to the disease process causing the murmur or if the murmur is secondary to endocarditis.

Diet

- Sodium restriction if in congestive heart failure.

Surgical Considerations

- Surgery or interventional cardiology procedures may be options in cases of patent ductus arteriosus, pulmonic stenosis, aortic stenosis, or congenital mitral valve disease. Consultation with a veterinary cardiologist is recommended prior to surgical intervention.



COMMENTS

Client Education

- Murmurs are associated with many conditions, some benign and others serious. Echocardiography may be required if the cause of the murmur is not obvious from the history and physical examination.

Patient Monitoring

- Low-grade systolic ejection murmurs in puppies may be physiologic; most resolve by 6 months of age. If murmur is still present after 6 months, include diagnostic imaging.

Possible Complications

- If murmur is associated with structural heart disease, may see signs of congestive heart failure (e.g., coughing, dyspnea, ascites) or exercise intolerance.

Expected Course and Prognosis

- Varies with underlying cause.

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Abbreviations

AV: atrioventricular
PCR: polymerase chain reaction
PMI: point of maximal intensity
S1: first heart sound
S2: second heart sound

Suggested Reading

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Author: Francis W.K. Smith Jr, DVM

chapter 65

Mycotoxins – Aflatoxins

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DEFINITION/OVERVIEW

- Aflatoxins are metabolites produced by fungi (*Aspergillus* and *Penicillium* spp.) that may cause acute hepatic failure.
- Uncommonly diagnosed in dogs, but outbreaks involving more than 100 cases have been reported.

ETIOLOGY/PATHOPHYSIOLOGY

- Fungal invasion and toxin production in crops during the growing season, during harvest, or during transportation and storage result in accumulation of aflatoxin.
- Despite random screening for aflatoxins by most pet food manufacturers, small amounts often reach the final product undetected.
- Aflatoxins are absorbed via the GI tract and subsequently sequestered in the liver.
- Most dogs require several days to weeks of exposure prior to developing clinical signs of toxicity, although sudden acute intoxication is reported.
- Evidence suggests aflatoxins bind and interfere with DNA, RNA, and protein function leading to carcinogenic, teratogenic, mutagenic, and acute hepatotoxic effects.
- Aflatoxin B1 is most frequently reported to cause toxicity.

Systems Affected

- Hepatobiliary: aflatoxins accumulate in the liver.
- Gastrointestinal: GI ulcers and hemorrhage occur frequently via an unknown mechanism of action.
- Hemic: aflatoxins are reported to have a coumadin-like effect that may contribute to the frequent occurrence of hypocoagulable states.

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SIGNALMENT/HISTORY

- Young and pregnant animals appear to be more susceptible.
- Highly toxic to dogs (LD_{50} of 0.5–1.5 mg/kg of body weight) and cats (0.55 mg/kg of body weight).
- Naturally occurring feline aflatoxicosis has not been reported.

Risk Factors/Causes

- Outbreaks: accidental inclusion of contaminated corn during the manufacturing process or improper preparation of commercial dog foods.
- Individual cases may occur following ingestion of moldy food (particularly bread).
- Warm humid conditions promote mold growth and aflatoxin production.
- Low moisture content in pet foods decreases the risk of mold growth.

Historical Findings

- Feeding moldy commercial pet food or ingesting other moldy foods such as bread.
- Pet foods containing corn, peanuts, and cottonseed are most frequently contaminated with aflatoxins.
- Change in brands or recently opening a new bag of dog food.
- Anorexia.
- Depression.
- Vomiting.
- Polydipsia.
- Polyuria.
- Discoloration of the eyes and mucous membranes/icterus.
- Initial signs are often vague and insidious in onset.
- Animals may exhibit an aversion to the contaminated food for several days prior to the development of clinical signs.



CLINICAL FEATURES

- Acute hepatic failure in multiple dogs from the same region or sharing the same food source should prompt a strong suspicion of aflatoxicosis.
- Severity of signs varies depending on the length of exposure and concentration of the aflatoxins ingested.
- Most often related to acute liver failure.
    - Weakness
    - Depression
    - Anorexia
    - Vomiting

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- Diarrhea
- Melena
- Hematemesis
- Abdominal distension
- Icterus
- Epistaxis
- Bruising
- Petechiae
- Peripheral edema
- Seizures
- DIC



DIFFERENTIAL DIAGNOSIS

- Rule out other causes of acute hepatic failure.
- Leptospirosis.
- Hepatic abscess.
- Bacterial cholangitis/cholangiohepatitis.
- Liver lobe torsion.
- Hepatic abscess.
- Severe pancreatitis.
- Xylitol toxicity.
- Acetaminophen toxicity.
- Sago palm toxicity.
- *Amanita* spp. mushroom toxicity.
- Heavy metal toxicity.
- Antifungal medications.
- Phenobarbital.
- Potentiated sulfonamides.
- Tetracycline.
- Idiosyncratic drug reactions.
- Rodenticide intoxication.



DIAGNOSTICS

- Evaluate suspected feeds for the presence of aflatoxins, even if macroscopic evidence of mold is not detectable.
- Liver samples can be submitted for aflatoxin testing.
- Liver enzymes elevated.
- Bilirubin increased.
- Bile acids increased.

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- ACT, PT, and PTT often prolonged.
- Antithrombin and protein C levels decreased; not specific to aflatoxicosis.
- Dilute urine; granular casts may develop as toxicity progresses.

Pathologic Findings

- No pathognomonic findings.
- Hepatic cytology reveals microvesicular fatty vacuolation.
- Histologic findings include hepatic centrilobular necrosis, bile duct proliferation, fibrosis, hepatocellular fatty degeneration, and megalocytosis.
- Gross pathologic changes include generalized icterus, liver damage, ascites, widespread hemorrhage, and gallbladder edema.
- Renal proximal tubular necrosis has also been reported on histologic examination.



THERAPEUTICS

- There is no specific antidote.
- Treatment is directed toward limiting further hepatic injury, maximizing hepatic recovery, and supportive care.
- Intravenous fluids to correct dehydration, provide maintenance requirements and allow for ongoing losses.

Drugs of Choice

- Evidence-based medicine to support therapies in clinically occurring aflatoxicosis are lacking.
- SAMe 20 mg/kg PO once a day on an empty stomach.
- Oncotic support (synthetic colloids, canine albumin) may be required in hypoalbuminemic patients (efficacy of these therapies is lacking).
- Metoclopramide (1–2 mg/kg per day IV as a CRI) starting at low doses and tapering upward to effect until vomiting is controlled.
- Ondansetron (0.1–0.2 mg/kg IV as a slow bolus twice or four times a day) if metoclopramide is not effective.
- Famotidine (0.5 mg/kg IV once to twice a day) or pantoprazole (1 mg/kg IV once daily) if hematemesis or melena is present.
- Sucralfate (0.5–1 g PO three times a day) if hematemesis or melena is present.
- Packed red blood cells if significant hemorrhage is present or there are clinical signs associated with anemia.
- Vitamin K_1 may be helpful (0.5–1.5 mg/kg SQ or PO every 12–24 hours) if coagulation times prolonged.
- Plasma transfusions to correct coagulopathies when present (10–15 mL/kg IV given over 2–4 hours).

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Alternative Drugs

- Although unproven in dogs, milk thistle (silibinin-phosphatidylcholine complex 2–5 mg/kg PO once daily) may be beneficial.
- N-acetylcysteine may be hepatoprotective if SAMe cannot be given (140 mg/kg IV given slowly as a loading dose followed by 70 mg/kg every 6 hours until SAMe can be given orally); administer via a microfilter.

Diet

- Parenteral nutrition may be required with protracted vomiting.

COMMENTS

- Save samples (airtight containers or frozen) of suspect foods and GI contents for analysis.
- Save package information (product and date codes) to expedite proper identification of contaminated foods.
- Proper authorities should be contacted (product manufacturer and FDA consumer complaints coordinator or equivalent) if intoxication is linked to commercial pet foods.

Patient Monitoring

- Monitor for hypoalbuminemia and coagulopathies.
- Serially evaluate hepatic parameters to help guide therapy and determine prognosis in response to therapy.
- Serum electrolytes and hematocrit.

Prevention/Avoidance

- Discontinue all commercial dog foods and consider boiled rice and chicken until clinical signs of toxicity resolve.
- Avoid grossly moldy, discolored, or malodorous dog foods.

Possible Complications

- Aflatoxins are carcinogenic and immunosuppressive following chronic exposure.
- Long-term complications may include hepatic cancer and chronic liver failure.

Expected Course and Prognosis

- Prognosis varies with amount of toxin ingested and length of exposure.
- Prognosis is guarded to poor if signs of hepatic failure develop.
- Animals that recover will often do so within 3–5 days.
- Aflatoxins may bind albumin and have delayed toxic effects; administration of oral hepatoprotectants for up to 60 days has been suggested.

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Abbreviatons

ACT: activated clotting time
CRI: constant rate infusion
DIC: disseminated intravascular coagulation
DNA: deoxyribonucleic acid
FDA: Food and Drug Administration
GI: gastrointestinal
IV: intravenous
LD_{50}: lethal dose that kills 50% of animals tested
PO: by mouth (per os)
PT: prothrombin time
PTT: partial thromboplastin time
RNA: ribonucleic acid
SAMe: s-adenosylmethionine
SQ: subcutaneously

Suggested Reading

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Author: Søren Boysen, DVM

chapter 66

Mycotoxins – Tremorgens

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DEFINITION/OVERVIEW

- Fungal metabolites found in moldy foods, compost, and garbage that can induce signs of CNS excitation, including ataxia, tremors, and seizures, when ingested.

ETIOLOGY/PATHOPHYSIOLOGY

- Ingested and absorbed via the GI tract.
- Excretion in the bile, enterohepatic recirculation, and continued reabsorption may prolong the signs of toxicity.
- Two known tremorgens reported to cause toxicity in dogs: penitrem A and roquefortine C, both produced by *Penicillium* spp. of fungi.
- The two mycotoxins may act synergistically if ingested together.

Systems Affected

- Neuromuscular: exact mechanism of action unknown, although interference with neurotransmitter release at central and peripheral nerve synapses is suspected.
- Gastrointestinal: secondary to an unknown mechanism of action of the mycotoxins or the result of ingesting other vomitogenic materials in conjunction with the mycotoxins.

SIGNALMENT/HISTORY

- One of the more common intoxications occurring naturally in dogs.
- Probably underdiagnosed in the veterinary profession.
- No known cases reported in cats.

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Risk Factors/Causes

- Free-roaming dogs that have access to garbage are likely to be at higher risk.
- Environmental factors such as prolonged periods of rain/moisture may promote fungal and toxin production.

Historical Findings

- Ingestion of moldy foods or garbage, including dairy products, nuts, grains, bread, and pasta.
- Weakness.
- Panting.
- Vocalization.
- Ataxia.
- Tremors.
- Salivation.
- Vomiting.
- Tachycardia.
- Owners typically report signs within 1–3 hours of garbage ingestion.



CLINICAL FEATURES

- Ingestion of the toxin may not be witnessed.
- Diagnosis often based on possible exposure, history, clinical signs, and response to treatment.
- Should be suspected following acute onset of CNS stimulation and vomiting following ingestion of garbage or moldy foods.
- Symptoms vary depending on the concentration of the mycotoxin present in the food and the amount of toxin ingested.
- Ataxia.
- Fasciculation.
- Tremors.
- Hyperesthesia.
- Hyperextension of the limbs.
- Muscle contractions.
- Seizure-like activity.
- Hyperthermia.
- Salivation.
- Vomiting (often reported as black and tarry).
- Panting.

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DIFFERENTIAL DIAGNOSIS

- Other common neurologic toxins:
  - Strychnine
  - Metaldehyde
  - Pyrethroids
  - Organophosphates
  - Carbamates
  - Methylated xanthines (caffeine, theophylline, theobromine)
  - Lead
  - Illicit drugs
- Other less common neurologic toxins:
  - Bromethalin rodenticides
  - Hexachlorophene
  - Chlorinated hydrocarbons
  - Zinc phosphide
  - The insecticide diethyltoluamide
  - Mushrooms

Nontoxin-induced causes of tremors and seizures:

- Cerebellar disorders
- Tremor syndrome of white dogs (little white shakers)
- Hypomyelination
- Dysmyelination
- Hypoglycemia
- Hypocalcemia/eclampsia
- Hepatic encephalopathy
- Infectious diseases (rabies, distemper)

DIAGNOSTICS

- Laboratory analysis of the vomit, stomach/intestinal contents, or gastric lavage contents of suspected animals provides a definitive diagnosis.
- Moldy food samples can also be submitted for tremorgenic toxin analysis.
- Screen for other common neurologic toxins if tremorgenic toxins are not confirmed on laboratory analysis.
- The minimum database should include a complete blood count, biochemistry panel, and urinalysis to rule out other causes of tremors and seizures.
- Increased levels of serum aspartate aminotransferase and creatinine kinase have been reported with tremorgenic mycotoxicosis, which occurs secondary to increased muscle activity.

Pathologic Findings

- There are no typical findings on gross or histologic examinations.

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THERAPEUTICS

- The goals of therapy are to minimize absorption of the mycotoxin from the GI tract, control the tremors and seizures, and provide supportive care.
- Induction of vomiting, gastric lavage, and activated charcoal may limit GI absorption but should only be performed in dogs that are not at risk for aspiration pneumonia.
- Active cooling (application of soaked towels and the use of fans) should be initiated if the temperature is greater than 41 °C (105.8 °F) (stop active cooling efforts when the temperature falls to 39.5 °C (103.1 °F) to avoid overshoot hypothermia, and monitor the patient closely to identify the development of hypothermia or recurrent hyperthermia).
- Intravenous fluids should be provided to maintain normal hydration status.

Drugs of Choice

- Diazepam can be tried (0.5–1 mg/kg IV), although tremors and seizures refractory to diazepam have been reported.
- Pentobarbital (3–15 mg/kg IV administered to effect) and methocarbamol (55–220 mg/kg IV administered slowly to effect at a rate <200 mg/min) are currently recommended to control tremor-like activity.
- Pentobarbital can cause heavy sedation and appropriate monitoring is required.
- Methocarbamol provides muscle relaxation.
- Propofol (2–8 mg/kg IV to effect).

Precautions/Interactions

- Aspiration pneumonia may occur prior to or during therapy, especially following sedation to control tremors.
- Animals with active tremors and seizures should not have gastric lavage or activated charcoal administered until CNS excitement can be controlled.
- If medications to control the CNS excitement cause severe sedation and suppression of the gag reflex, administration of activated charcoal and gastric lavage should be approached with great care due to the risk of aspiration pneumonia.

Alternative Drugs

- Diazepam can be tried, but when given alone it may be insufficient to control severe mycotoxin-induced tremors.



COMMENTS

- Perform gastric lavage only under general anesthesia with cuffed endotracheal tube in place to prevent aspiration of gastric contents.
- Some affected animals can be intubated with minimal sedation.
- Patients under anesthesia should be endotracheally intubated and often require ventilation (monitor respiratory function).
- Activated charcoal can be administered by orogastric tube.

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Patient Monitoring

- Monitor for continued tremor activity for at least 24 hours and treat as needed.
- Monitor temperature: hyperthermia may occur with uncontrolled tremors or hypothermia may develop following pentobarbital administration and subsequent sedation.
- Monitor respiratory rate and effort, level of sedation, and the presence or absence of a gag reflex following administration of pentobarbital and other sedatives.
- Arterial or venous blood gas analysis should be performed to evaluate acid–base status and carbon dioxide levels if there are concerns about hypoventilation.
- Thoracic radiographs and arterial blood gas analysis should be performed if there is a suspicion of aspiration pneumonia.

Prevention/Avoidance

- Proper disposal of moldy food products and preventing animals from accessing garbage will decrease occurrence of tremorgenic mycotoxicosis.

Expected Course and Prognosis

- Varies with the severity of symptoms and promptness of therapy.
- Most dogs make a complete recovery within 2–3 days.
- Death has been reported in severe cases with uncontrollable seizure/status epilepticus.
- Concurrent aspiration pneumonia can contribute to significant morbidity and mortality and every precaution should be taken to avoid this complication.

Abbreviations

CNS: central nervous system
GI: gastrointestinal
IV: intravenously

Suggested Reading

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chapter 67

NSAID Toxicosis



DEFINITION/OVERVIEW

- Nonsteroidal antiinflammatory drugs (NSAIDs) are common over-the-counter (OTC) or prescription medications with antiinflammatory, antipyretic, and analgesic properties.
- NSAIDs, in both human and veterinary form, can result in accidental toxicosis in dogs and cats. Ingestion may be accidental or via well-intentioned pet owners who are unaware of the toxic dose or safety profile of these readily available medications.
- Examples of common human NSAIDs include aspirin, ibuprofen, naproxen, and diclofenac. Common veterinary prescription NSAIDs include carprofen, deracoxib, ketoprofen, firocoxib, meloxicam, and tepoxalin.
- Clinical signs of toxicosis often include gastrointestinal (GI) irritation or ulceration, acute kidney injury (AKI), and, rarely, central nervous system (CNS) impairment.
- Even with veterinary NSAIDs, potential adverse effects can be seen at therapeutic doses.

ETIOLOGY/PATHOPHYSIOLOGY

- NSAIDS work by inhibiting the conversion of arachidonic acid to prostaglandins via inhibition of COX enzymes. While prostaglandin inhibition is important in reducing inflammation, it has key roles in the body such as maintenance of blood flow to the kidneys and mucosa of the GIT and stimulation of bicarbonate from the stomach.
- There are several classes of NSAIDs, including azole derivatives (e.g., tepoxalin, phenylbutazone), indoles (e.g., indomethacin), propionic acids (e.g., ibuprofen, naproxen, carprofen, etc.), salicylates (e.g., aspirin), sulfonamides and sulfones (e.g., deracoxib, firocoxib), oxicams (e.g., piroxicam, tenoxicam, etc.), and fenamates (e.g., meclofenamic acid).
- Depending on the specific type of NSAID, the degree of COX-1 versus COX-2 inhibition may vary.
- NSAIDs are absorbed well from the GIT, and are highly protein bound with a low volume of distribution.
- Most NSAIDs are metabolized by the liver, with extensive enterohepatic recirculation occurring with some (e.g., carprofen, ibuprofen).

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Companion website: www.fiveminutevet.com/emergency

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TABLE 67.1. List of both prescription and over-the-counter NSAIDs with published data on half-life and toxic dose, if available.

NSAID	Plasma half-life (dogs)	Toxic dose (dogs)	Toxic dose (cats)
Ibuprofen	2.5–5.8 hours	GI: >50 mg/kg Renal: >150–175 mg/kg CNS: >400 mg/kg	GI: >10 mg/kg Renal: unknown
Naproxen	35–72 hours	GI: >5 mg/kg Renal: >10–25 mg/kg	GI: any dose Renal: unknown
Carprofen	6.7–9.3 hours	GI: >20 mg/kg Renal: >40 mg/kg	GI: >4 mg/kg Renal: >8 mg/kg
Deracoxib	3 hours	GI: >10–15 mg/kg Renal: >30 mg/kg	GI: >4 mg/kg Renal: >8 mg/kg
Etodolac	7–12 hours	GI: >40 mg/kg/day Renal: >80 mg/kg/day	
Firocoxib	7.8 hours	GI: >50 mg/kg	
Meloxicam	24 hours	GI: >1 mg/kg Renal: >2 mg/kg	GI: any dose above therapeutic dose Renal: >1.5 × therapeutic dose
Tepoxalin	12–13 hours	GI: 100–300 mg/kg	

CNS, central nervous system; GI, gastrointestinal.

- The half-life and toxic dose of certain NSAIDs vary between species. See Table 67.1 for more information. Unfortunately, limited information on toxic doses in cats is known.
- With veterinary NSAIDs, the GI toxic dose in dogs is generally considered to be five times a therapeutic dose, while the nephrotoxic dose is considered to be 10 times a therapeutic dose.

Systems Affected

- Gastrointestinal: GI irritation and/or ulceration secondary to lack of blood flow to the GIT and decreased bicarbonate secretion to the stomach. Rarely, septic peritonitis secondary to ruptured GI ulceration can occur.
- Renal/urologic: acute kidney injury.
- Nervous: rare, but with high dose, CNS signs, including, seizures may be seen.
- Hepatobiliary: with chronic dosing, idiosyncratic hepatopathy has been reported.
- Cardiovascular: with GI ulceration, severe GIT hemorrhage may occur, resulting in clinical signs of hemorrhagic shock. With septic peritonitis, hypovolemic or septic shock may be seen. Clinical signs include prolonged CRT, tachycardia, poor pulse quality, and collapse.
- Hemic/lymphatic/immune: common clinicopathologic findings seen with NSAID toxicosis include anemia and thrombocytopenia (secondary to GI bleeding) with concurrent panhypoproteinemia. While difficult to test for routinely, a thrombocytopathia may also be seen but is likely clinically insignificant with acute toxicosis.

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SIGNALMENT/HISTORY

Risk Factors

- Puppies and younger dogs appear to be overrepresented with poisoning due to their curious nature.
- Neonates, geriatric patients, or those with underlying renal or hepatic disease are more at risk for NSAID toxicosis due to abnormal or delayed metabolism.
- Cats are suspected of being at least twice as sensitive to NSAIDs as dogs.
- Coadministration of corticosteroids with NSAIDs.
- Chronic administration.
- Due to protein binding, patients with hypoalbuminemia may be at increased risk for toxicosis.

Historical Findings

- Evidence of a container or prescription bottle that has been tampered with or chewed
- Owner administration
- Anorexia
- Vomiting
- Diarrhea
- Black tarry stool
- Generalized malaise
- Abdominal pain
- PU/PD
- CNS signs

CLINICAL FEATURES

- Gastrointestinal.
  - Acute vomiting may occur within 2–6 hours of ingestion.
  - Common clinical signs include anorexia, hypersalivation, vomiting, hematemesis, diarrhea, melena, hematochezia, and abdominal pain.
  - Signs of GIT hemorrhagic may not be seen for 12 hours or up to several days post ingestion.
- Renal/urologic.
  - AKI is typically seen 24–36 hours post ingestion, but can develop as early as 12 hours or be delayed to 3–5 days post exposure.
  - Clinical signs or findings may include inappetence, anorexia, hypersalivation (secondary to uremic ulcers), polyuria, oliguria/anuria, uremic halitosis, abdominal pain (e.g., in the renal region), and renomegaly.
  - Clinicopathologic findings may include hemoconcentration secondary to dehydration, anemia (secondary to GI bleeding), panhypoproteinemia, isosthenuria, azotemia, electrolyte disturbances.

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- Nervous.
  - Rarely, with extremely high doses, CNS signs can be seen within 1–2 hours of ingestion.
  - Ataxia, decreased mentation, respiratory depression, seizures, and coma may be seen.
- Hepatobiliary: anorexia, lethargy, vomiting, diarrhea, melena, icterus, and seizures may be seen secondary to acute hepatic failure with chronic dosing.



DIFFERENTIAL DIAGNOSIS

- In patients presenting with AKI, other nephrotoxicants such as ethylene glycol, *Vitus* spp. (e.g., grapes, raisins), cholecalciferol, etc. should be ruled out.
- In patients presenting for acute gastrointestinal signs, underlying diseases such as infectious (e.g., parvovirus, *Salmonella*, etc.), inflammatory (e.g., gastroenteritis), parasitic (e.g., roundworms, etc.), dietary indiscretion, foreign body, endocrine (e.g., hypoadrenocorticism, etc.), metabolic (e.g., renal failure, liver disease, etc.), neoplasia, etc. should be ruled out.



DIAGNOSTICS

- If an ingestion approaching a nephrotoxic dose has occurred, baseline blood including a CBC, biochemistry panel, and urinalysis (pretreatment) should be performed at the time of admission.
- In hospitalized patients, a PCV/TS and renal panel should be performed every 24 hours (typically for 1–3 days). The fluid therapy plan and duration of hospitalization may need to be adjusted based on results of clinicopathologic testing.
- Less common: if there is clinical suspicion of septic peritonitis, additional diagnostics such as abdominal radiographs (+ contrast) or abdominal ultrasound is warranted. Rarely, a diagnostic peritoneal lavage (DPL) or abdominocentesis (with cytology and fluid analysis) may be necessary to rule out septic peritonitis secondary to a ruptured GI ulcer.



THERAPEUTICS

- The mainstay therapy for NSAID toxicosis is decontamination, intravenous (IV) fluid therapy, and gastrointestinal protectants. The reader is referred to a toxicology textbook for more information on decontamination.
  - With recent ingestion in an asymptomatic patient, emesis induction and administration of one dose of activated charcoal (1–5 g/kg, PO) with a cathartic (e.g., sorbitol) is warranted.
  - NSAIDs that undergo enterohepatic recirculation (e.g., ibuprofen, carprofen) or have a long half-life (e.g., naproxen) should be treated with multiple doses of activated charcoal (with the additional doses free of a cathartic).

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- Fluid therapy.
  - The use of a balanced, isotonic crystalloid is necessary. Aggressive fluid rates (6–10 mL/kg/h) are recommended to help counter the renal vasoconstrictive effects secondary to NSAIDs.
  - Goals of fluid therapy should be based on physical examination findings and evidence of hemodilution (e.g., PCV/TS 35%/5 g/dL) and isosthenuria.
  - In patients presenting with AKI secondary to NSAID toxicosis, severe polyuria may be seen. Fluid therapy should be adjusted based on calculated "ins and outs" and urine output (UOP).
- Gastrointestinal support.
  - Potent antiemetics should be used for patient comfort, to prevent aspiration pneumonia, and to help prevent secondary complications from activated charcoal administration (e.g., emesis, aspiration of charcoal, hypernatremia, etc.).
  - Gastroprotectants should be continued for 7–10 days.

Drugs of Choice

- Antiemetics.
  - Maropitant (1 mg/kg SQ q24h; extra-label use in cats and via IV route)
  - Ondansetron (0.1–0.5 mg/kg IV q12h)
  - Dolasetron (0.6 mg/kg IV q24h)
- Gastric protectants.
  - H_2 blockers (famotidine, 0.5–1 mg/kg SQ, IV, PO q12–24h)
  - Misoprostol (2–5 µg/kg PO q8h)
  - Proton pump inhibitors (omeprazole, 0.7–1 mg/kg PO q12–24h; pantoprazole, 0.7–1 mg/kg IV q12–24h)
  - Sucralfate (0.25–1 g PO q6–8h)

Alternative Drugs

- The use of intravenous lipid emulsion (ILE) as an antidote (for fat-soluble toxicants) is currently not recommended by this author for the treatment of NSAID toxicosis, with the exception of severe, life-threatening clinical signs (e.g., coma, seizures, etc.), until further evidence is available.

Precautions/Interactions

- Misoprostol can result in miscarriage in female pet owners handling the pills. As a result, this author prefers to use alternative gastric protectants or to carefully instruct pet owners to wear protective gloves.

Diet

- A bland diet should be fed for several days due to the risk for GI upset secondary to GI ulceration.

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Surgical Considerations

- Rarely, NSAID toxicosis can result in septic peritonitis secondary to perforated GI ulcers. Prompt diagnosis, recognition, and surgical correction are key to ensuring best outcome.



COMMENTS

Client Education/Prevention/Avoidance

- Owners should be educated to appropriately pet-proof the house. Education on crate training is imperative.
- Pet owners should be educated to never give an OTC or prescription medication to their pet without consulting their veterinarian first.
- Owners and veterinary professionals should be educated to call veterinary-specific poison control centers for consultation with a veterinary toxicologist for life-saving advice as needed.

Possible Complications

- Patients with AKI secondary to NSAID toxicosis are at increased risk for chronic renal insufficiency. Pet owners should be cautioned about long-term effects and chronic management and monitoring.

Expected Course and Prognosis

- Overall, the prognosis for NSAID toxicosis is fair to excellent with aggressive decontamination, fluid therapy, and symptomatic supportive care. However, financial limitations may preclude treatment, which often requires hospitalization for 24–72 hours.
- Rarely, severe complications such as hemorrhagic shock, septic shock, or septic peritonitis may be seen with NSAID toxicosis.

Synonyms

- Advil
- Midol
- Aleve
- Pamprin
- Rimadyl
- Deramaxx
- Etogesic
- Diclofenac
- Aspirin

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Abbreviations

CBC: complete blood count
GI: gastrointestinal
GIT: gastrointestinal tract
IM: intramuscular
IV: intravenous
NSAID: nonsteroidal antiinflammatory drug
OTC: over the counter
PCV: packed cell volume
PO: by mouth (per os)
TS: total solids

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chapter 68

Organophosphate Intoxication

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DEFINITION/OVERVIEW

- OPs and CMs are anticholinesterase chemicals used most commonly as household and industrial insecticides. They can harm people and veterinary species.
- OP exposure in dogs and cats is by ingestion, dermal contact, or inhalation.
- The dose required for OP intoxication and death varies significantly by compound.

ETIOLOGY/PATHOPHYSIOLOGY

- OP and CM compounds harm animals by inhibiting the action of acetylcholinesterase at neuronal synapses and the neuromuscular junction. Acetylcholinesterase is the enzyme that degrades acetylcholine, and thus prevents overstimulation of neurons and muscles for which acetylcholine is a neurotransmitter. Acetylcholine plays a stimulatory role at the skeletal neuromuscular junction, the parasympathetic neuromuscular junction, synapses of the autonomic nervous system, and some areas of the CNS. Inhibition of acetylcholinesterase leads to overstimulation of the skeletal neuromuscular junction, parasympathetic system, and occasionally the sympathetic nervous system, and can have variable effects on the CNS. Thus, clinical signs of acute OP or CM intoxication in dogs and cats involve skeletal muscle, the parasympathetic nervous system, and the CNS.
- OP and CM compounds are present in many home environments. These chemicals are used in some household insecticides, mixed fertilizer/insecticide preparations, and in some over-the-counter flea and tick products for topical use directly on dogs and cats. Both dogs and cats are susceptible to intoxication.
- OPs are most commonly associated with acute intoxication, as described here. However, OPs have also been associated with OP-induced delayed neuropathy and the intermediate syndrome, both of which are uncommon. More information on these syndromes in dogs and cats is available elsewhere (see Suggested Reading).

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Systems Affected

- Nervous: muscle tremors and twitching are common. Eventually, weakness to paralysis may occur. Obtundation is common; seizures to coma can be seen in severe cases.
- Cardiovascular: bradycardia is common. Rarely, tachyarrhythmias may occur.
- Gastrointestinal: hypersalivation, vomiting, diarrhea, and abdominal pain are common. Rarely, acute pancreatitis may occur in the dog.
- Ophthalmic: miosis is common, and can help distinguish OP or CM intoxication from that caused by other tremorgens. Lacrimation (excessive tearing) may also be present.
- Respiratory: occasionally, intoxicated patients have severe bronchoconstriction and bronchorrhea (excessive bronchial secretions), which can lead to respiratory distress and life-threatening hypoxemia. Diaphragmatic function may be impaired in severe cases.



SIGNALMENT/HISTORY

- Both dogs and cats of all ages and breeds are susceptible to OP and CM intoxication.
- Clinical signs generally occur within 30 minutes to 6 hours after chemical ingestion or inhalation.
- Time for dermal absorption can vary, but clinical signs may be present within a few hours.
- Clients may be aware of OP or CM exposure, such as in the case of purposeful topical insecticide application on the animal. Clients may still answer "No" when asked if toxins are available in the environment, even when exposure was through bathing or insecticide in the garden. Therefore, when the clinician has a high index of suspicion for OP or CM toxicity based on physical examination, it may be helpful to ask the client specific questions about flea and tick treatments, exposure to public gardens, and chemical use in the yard.

Risk Factors/Causes

- Household insecticides, garden insecticides, and topical chemicals used to treat flea or tick infestation on dogs and cats are the most common sources of exposure to OPs and CMs for pets.

Historical Findings

- Occasionally clients are aware of historical application of a topical insecticide or exposure to environmental insecticide products.
- More commonly, dogs and cats with OP or CM intoxication are presented with complaints of acute-onset muscle tremors, ataxia, hypersalivation, or gastrointestinal disturbance of unknown etiology.



CLINICAL FEATURES

- Physical examination most commonly reveals generalized muscle tremors, but clinical signs can progress to seizures or coma.

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- Clinical signs of parasympathetic overdrive are common, and can be remembered with the mnemonic DUMBELS:
  D: diarrhea
  U: urination
  M: miosis
  B: bradycardia/bronchospasm
  E: emesis
  L: lacrimation
  S: salivation
- Bronchospasm and excessive bronchial secretions can lead to life-threatening hypoxemia.
- OP or CM intoxication should be considered a top rule out in patients with acute-onset generalized tremors in combination with parasympathetic signs.
- Not all signs are present in all patients.



DIFFERENTIAL DIAGNOSIS

- Some other toxins that cause similar clinical signs include serotonergics; penitrem A, roquefortine, and other mycotoxins; metaldehyde; amitraz; strychnine; many toxic plants; blue-green algae; nicotine; some illicit drugs; physostigmine, neostigmine, and other similar medications used for treating myasthenia gravis; pyrethroids; and some other insecticide compounds.
- Other differentials for OP or CM toxicosis include nontoxic conditions such as inflammatory, infectious, idiopathic, and degenerative diseases of the CNS; neuromuscular diseases; and metabolic diseases that affect the neurologic system.



DIAGNOSTICS

- A tentative diagnosis can be made by considering signalment and historical data in combination with physical examination findings.
- Clinicopathologic data are nonspecific and therefore generally noncontributory; abnormalities can include azotemia, hematuria, leukocytosis, elevated serum creatine kinase concentration, hypokalemia, and hyperglycemia.
- Whole blood can be submitted to determine blood cholinesterase activity; activity <50% is suspicious for intoxication, whereas activity <25% is confirmatory.
- Definitive diagnosis requires toxicologic examination of blood, gastric contents, urine, or source material. Contact a veterinary toxicology laboratory to confirm sample handling procedures for whole blood cholinesterase activity and toxin identification.



THERAPEUTICS

- Supportive care is imperative for the intoxicated patient.
- Animals with tachypnea or respiratory distress should have oxygen supplementation. Patients with severe hypoxemia or poor diaphragmatic function (and subsequent

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hypoventilation) are at high risk of immediate death, and should have endotracheal intubation and positive pressure ventilation.

- Intravenous fluid therapy is required for hypovolemia or dehydration, which can occur due to vomiting, diarrhea, or sustained muscle activity.
- Seizures should be treated immediately.
- Antiemetics and gastric protectants may be considered for those with severe gastrointestinal disturbance.
- Decontamination strategies should be used to decrease systemic absorption of OP or CM toxins, when possible.
    - Induction of vomiting is important for animals that have ingested toxin within the past 1–2 hours but is less useful as time passes.
    - Animals that may have compromised gag function and those that have already vomited should not be made to vomit.
    - Activated charcoal is useful in OP and CM intoxication, though the risks of aspiration in animals with clinical signs must be weighed against the benefits.
    - Patients with the potential for topical exposure should be bathed with mild soap and water as soon as their clinical condition allows. They should be dried completely to prevent hypothermia. Patients with topical exposure may also benefit from gastrointestinal decontamination because they may have ingested some toxin during grooming, and because enterohepatic circulation of systemic toxin can occur even if exposure is topical.
- Antidotes are available for the muscarinic (parasympathetic) and nicotinic (skeletal muscle) signs of OP and CM toxicity. They can be used in addition to, but cannot replace, supportive care and good clinical judgment.

Drugs of Choice

- Supportive care should be provided, as clinically indicated, with supplemental oxygen, intravenous fluids, and gastrointestinal support.
- Seizures should be treated with diazepam 0.5–1 mg/kg (0.23–0.45 mg/lb) IV or another benzodiazepine at an appropriate dose.
- Activated charcoal can be administered at 1–4 g/kg (0.45–1.8 g/lb) and may be given once or repeated every 6 hours for the first day after exposure to bind enterohepatically circulated toxin. If repeated doses are administered, cathartics such as sorbitol should be avoided to help prevent electrolyte and acid–base abnormalities.
- Atropine is the most useful antidote and is used to treat potentially life-threatening muscarinic signs of bradycardia, bronchospasm, and excessive bronchial secretions. The initial atropine dose of 0.1–2 mg/kg (0.05–0.91 mg/lb) should be given one-fourth IV and the remainder SQ. The dose is based on severity of the muscarinic signs. Adequate treatment leads to clinical signs of atropinization: mydriasis, dry mouth, and mild sinus tachycardia. Depending on the severity of intoxication, atropine may need to be repeated every 20–30 minutes at smaller doses of 0.1–0.25 mg/kg (0.05–0.11 mg/lb) to achieve or maintain atropinization.

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- 2-PAM is an antidote for the nicotinic signs of acute, moderate-to-severe OP or CM toxicity. It has little impact on CNS or muscarinic signs. The efficacy of 2-PAM is debated, and the drug may be most beneficial with concurrent atropinization. 2-PAM can worsen clinical signs of mild OP or CM toxicity, so is not recommended in those cases. Patients should be monitored closely during 2-PAM administration and the drug discontinued if clinical signs worsen during treatment. The 2-PAM dose is 10–20 mg/kg (4.5–9.1 mg/lb) SQ or as a slow intravenous infusion every 12 hours. Refer to the package insert for information on drug compatibility and administration.

Precautions/Interactions

- Emesis should probably not be induced if ingestion occurred more than 2 hours before, and should not be induced if the animal has neurologic impairment or other cause for increased risk of aspiration, or has already vomited.
- Patients that present with tachycardia or tachyarrhythmia rather than the classic bradycardia should be monitored closely if they require atropinization for bronchospasm or bronchial secretions (this seems an unlikely scenario).
- The animal with bradycardia should probably not be treated with medications known to cause bradycardia (i.e., opiates, anesthetics) until it has been atropinized and has a normal heart rate.
- The major precaution when treating a patient with 2-PAM is that it may exacerbate clinical signs, in which case it should be discontinued.

Alternative Drugs

- Glycopyrrolate may be used if atropine is not available, though it has a slower onset of action. An appropriate starting dose would be 0.05 mg/kg (0.02 mg/lb) IV.

Nursing Care

- Recumbent patients should be turned every 4–6 hours to prevent pressure sores, and oral care procedures and intubation may be required to help decrease risk of aspiration.

COMMENTS

Client Education

- All OPs and CMs should be removed from the pet's environment.

Patient Monitoring

- Patients should be monitored closely in hospital for resolution or worsening of muscarinic, nicotinic, and CNS signs.

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- Vital signs should be checked frequently, and a continuous ECG is helpful to monitor for brady- or tachycardia.
- Blood pressure should be measured until the animal is stabilized.
- Pulse oximetry or PaO_2 may be used to assess patient oxygenation status. Arterial or venous PCO_2, or $ETCO_2$ in the intubated patient, can be used to check for hypoventilation that can occur due to diaphragmatic weakness.
- Time to resolution varies by severity of intoxication, but usually takes 1–3 days if the patient is adequately decontaminated.
- Once the animal is home, further monitoring is generally not required as long as the toxic source is removed from the environment.

Prevention/Avoidance

- All OPs and CMs should be removed from the pet's environment.

Possible Complications

- Acute OP toxicity has been associated with acute pancreatitis in humans. There is evidence that it could cause acute pancreatitis in dogs but probably not in cats.
- Other complications could include development of intermediate syndrome following the acute crisis or more typical complications such as aspiration pneumonia from hypersalivation or vomiting.

Expected Course and Prognosis

- Clinical course depends on the severity of toxicosis and varies from mild clinical signs with need for only brief observation to a severe clinical syndrome with need for longer hospitalization, mechanical ventilation, and a high risk of death. Regardless of initial clinical course, if the animal survives the acute crisis, prognosis for full recovery is good because development of intermediate syndrome appears to be rare in dogs and cats.

Synonyms

- Anticholinesterase intoxication or toxicity; carbamate intoxication or toxicity

Abbreviations

2-PAM: pralidoxime (2-pyridine aldoxime methiodide)
CM: carbamate
CNS: central nervous system
ECG: electrocardiogram
$ETCO_2$: end-tidal carbon dioxide tension
IV: intravenous
OP: organophosphate

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PaO_2: partial pressure of oxygen in arterial blood
PCO_2: partial pressure of carbon dioxide in blood
SQ: subcutaneous

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chapter 69

Otitis Media and Otitis Interna – Canine

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DEFINITION/OVERVIEW

- Otitis media: inflammation and effusion within the tympanic cavity.
- Otitis interna: inflammation of the inner ear; typically an extension of otitis media into the cochlea and/or vestibule.

ETIOLOGY/PATHOPHYSIOLOGY

Otitis Media

- Canine otitis media is uncommon. Most frequently, it is due to extension of otitis externa via damage of the tympanic membrane and subsequent infection in the tympanic cavity.
- The air-filled tympanic cavity is bordered by the tympanic membrane, tympanic bulla, and petrous temporal bone, and is lined with a mucociliary epithelium.
- Purulent, hemopurluent, mucoid, or keratinaceous effusion develops in the tympanic cavity.
- Bacterial and *Malassezia* infections are common.
- Bacterial species in the ear canal and tympanic cavity may differ.
- The facial and ocular sympathetic nerves pass through the middle ear: otitis media may lead to facial nerve paresis and Horner's syndrome.
- Bulla osteitis leads to temporomandibular joint pain in advanced cases.
- Tympanic membrane damage and tympanic cavity effusion reduce sound conduction.
- Rare causes of otitis media/interna include:
    - Ascending nasopharyngeal infection via the auditory tube
    - Nasopharyngeal neoplasia
    - Trigeminal nerve paresis and subsequent auditory tube dysfunction
    - Foreign body penetrating the tympanic membrane
    - Ototoxicity (Horner's syndrome) may arise after topical application of otic cleansers, gentamicin or ointments in the presence of a damaged tympanic membrane
    - Benign or invasive neoplasm of ear canal or middle ear (Fig. 69.1)
    - Cholesteatoma, a keratinizing, expansile, cystic mass in the middle ear (Fig. 69.2)

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- Primary secretory otitis media (PSOM) is accumulation of mucus in the tympanic cavity, in the Cavalier King Charles spaniel (Fig. 69.3).
- Pathophysiology is unknown and may involve auditory tube or mucociliary dysfunction.



Fig. 69.1. Otic neoplasm visible in the horizontal ear canal, 6-year-old Giant Schnauzer, cyst originating from the middle ear.

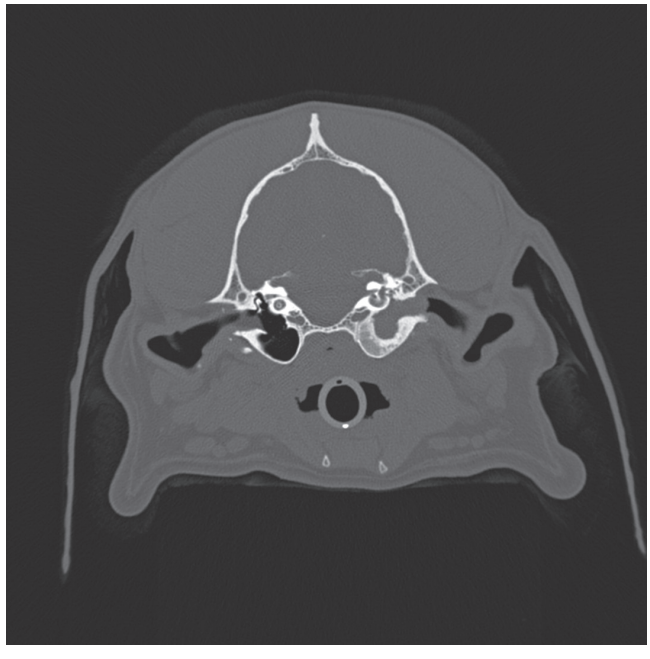


Fig. 69.2. CT scan of cholesteatoma, 8-year-old Labrador retriever. Note cystic mass arising from the tympanic cavity, extending into the ear canal and sclerosis of the bulla.

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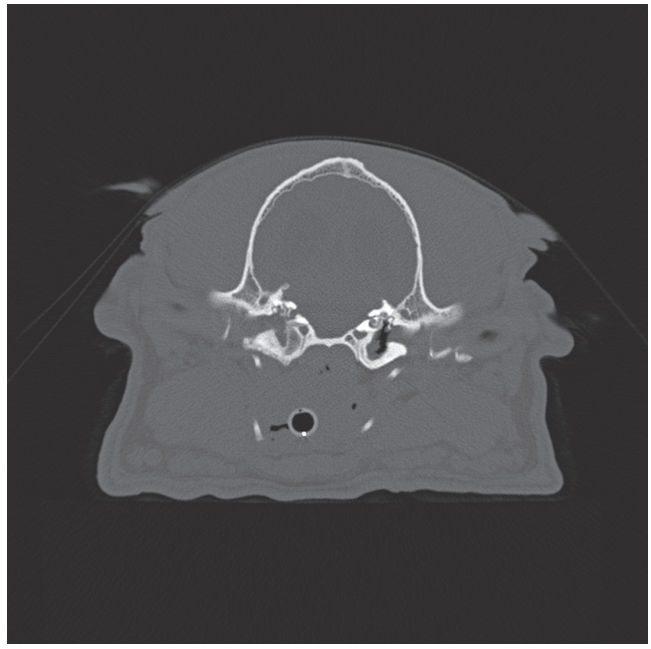


■ **Fig. 69.3.** Head tilt, 10-year-old Cocker spaniel, otitis interna resulting from chronic otitis externa.

Otitis Interna

- Canine otitis interna is rare.
- Otitis interna is usually progression of otitis media into the cochlea and/or vestibule within the petrous temporal bone.
- Aforementioned signs of otitis media may be present.
- Ipsilateral head tilt, ipsilateral slow-phase nystagmus, and neurologic hearing loss (see Fig. 69.3).
- Lysis of petrous temporal bone, due to infection or neoplasia, can result in meningoencephalitis and nonspecific signs of central neurologic dysfunction (see Fig. 69.4).
- Rare causes of otitis interna include:
  - Neoplasia of the middle or inner ear
  - Ototoxicity: topical or systemic gentamicin, otic cleansers, and ointments particularly if the tympanic membrane is damaged
  - Gentamicin can damage cochlear hair cells, resulting in hearing loss.

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■ **Fig. 69.4.** CT scan of otitis media/interna, 5-year-old English bulldog. Note unilateral lysis of the petrosal bone, with bilateral tympanic cavity effusion and bulla sclerosis.

Systems Affected

Otitis Media

- Nervous: Horner's syndrome and/or facial nerve paresis (Fig. 69.5).
- Ophthalamic: Horner's syndrome: pupil miosis, ptosis, globe retraction.
- Skin: draining tract and abscess as a result of bulla lysis.

Otitis Interna

- Nervous: peripheral vestibular syndrome: ipsilateral head tilt, circling, nystagmus.
- Nonspecific central neurologic signs if petrosal bone lysis and meningoencephalitis.
- Ophthalmic: nystagmus with ipsilateral slow phase.



SIGNALMENT/HISTORY

Otitis Media

- Adult dogs with a history of chronic otitis externa.
- Cholesteatoma is a potential complication of total ear canal ablation and bulla osteotomy (TECA-BO).
  - Cavalier King Charles spaniel: PSOM
  - Brachycephalic dogs: cholesteatoma

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■ **Fig. 69.5.** Facial nerve paresis, Cocker spaniel, otitis media due to chronic otitis externa.

- Clinical signs:
  - Head shaking, scratching
  - Otic discharge
  - Pain at base of ear
  - Reduced sound localization
  - Facial nerve paresis
  - Horner's syndrome
  - Reluctance to chew hard food

Otitis Interna

- Adult dogs with a history of chronic otitis externa.
- Adult dogs with otitis media
- Clinical signs:
  - Aforementioned signs of otitis media
  - Vestibular syndrome
  - Reduced hearing, reduced ability to locate sound

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Risk Factors (Otitis Media and Interna)

- Chronic, unresolved otitis externa
- Otic foreign body
- Otic neoplasia
- Total ear canal ablation and bulla osteotomy (cholesteatoma)
- Ototoxicity
- Stenosis of the ear canal due to chronic otitis externa or traumatic injury
- Trigeminal nerve dysfunction
- Congenital atresia of the ear canal

Historical Findings

Otitis Media

- Otic exudate, inflammation
- Head shaking, scratching
- Headshyness
- Reduced hearing, difficulty locating sound
- Reluctance to chew
- Malaise

PSOM

- Scratching at or near ear
- Head tilt
- Reduced hearing, reduced sound localization
- Pain with neck manipulation, reduced hearing

Otitis Interna

- Aforementioned signs of otitis media
- Difficulty navigating stairs, jumping up
- Head tilt, circling, or rolling
- Rapid eye movement
- Malaise

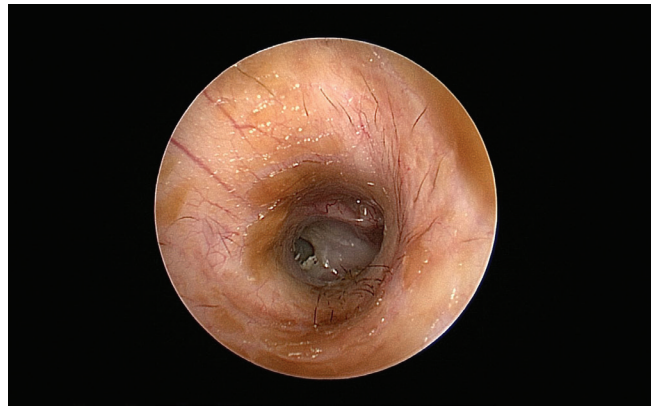


CLINICAL FEATURES

Otitis Media and Interna

- Otitis externa: erythema, exudate, hyperplasia, stenosis.
- Tympanic membrane pathology (Fig. 69.6).
  - Opaque
  - Absent or ruptured
  - Bulging pars tensa (rare) or pars flaccida (can be normal)

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■ **Fig. 69.6.** Opaque and ruptured pars tensa of tympanic membrane, 1-year-old German shepherd dog, otitis media due to penetrating grass awn foreign body.

- Pain.
  - Ear base palpation
  - Mouth opening
  - Chewing
- Horner's syndrome.
- Facial nerve paresis.
- Vestibular syndrome.
- Mass or foreign body in ear.

PSOM (Cavalier King Charles Spaniel)

- Bulging pars flaccida of the tympanic membrane (can be normal) (Fig. 69.7).
- Opaque material seen behind pars tensa of the tympanic membrane.
- Head tilt.
- Reduced sound localization and hearing.
- Neck pain.

DIFFERENTIAL DIAGNOSIS

- Otitis externa is common, and may be concurrently present with many other conditions.

Otitis Media/Interna Differentials

- Chronic otitis externa without otitis media/interna.
  - Otoscopic exam: normal tympanic membrane
  - Bulla CT or anesthetized, multiple-view bulla radiographs
    - Air-filled bulla with no effusion
    - Lack of bulla or petrosal lysis or sclerosis

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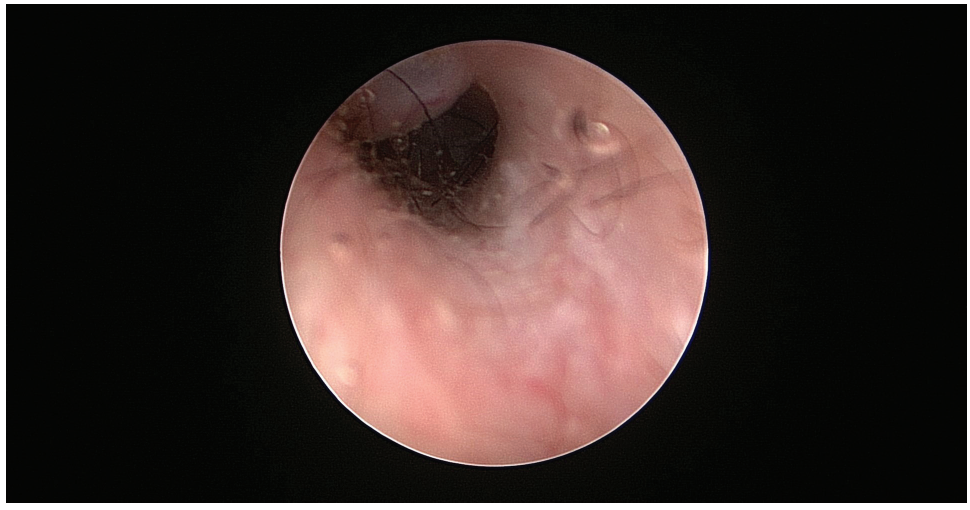


Fig. 69.7. Bulging pars flaccida, 1-year-old Cavalier King Charles spaniel, PSOM.

- Central vestibular syndrome.
  - Signs of central involvement: proprioceptive deficits, head pressing, seizures
  - MRI confirms central involvement, CT demonstrates lysis of petrous temporal bone

PSOM Differentials

- Chiari-like malformation.
  - MRI confirms Chiari-like malformation, syringohydromyelia
  - CT, bulla radiographs, and MRI confirm air-filled bulla with no effusion
  - Myringotomy and tympanic cavity aspirate negative for mucus
  - Note: PSOM and Chiari-like malformation may be concurrent

DIAGNOSTICS

- Otoscopic examination to assess tympanic membrane and ear canal.
- Elimination diet trial for patients with chronic allergic dermatitis.
- Assess for bulla pain: palpate ear base, open mouth.
- Cytology of the ear canal is recommended for patients with signs consistent with otitis externa.
- Neurologic examination.
  - All patients
  - Assess for Horner's syndrome and facial nerve, vestibular, and central nervous deficits

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- Imaging.
  - CT: ideal to confirm tympanic cavity effusion, bulla changes, neoplasia
  - Bulla radiographs: confirm effusion, bony changes
  - MRI: indicated if central involvement or Chiari-like malformation are differentials
- Cytology and culture of middle ear exudate.
  - Anesthetized patients with tympanic cavity effusion
  - Obtained via catheter aspiration and sterile saline flushing

Pathologic Findings

- Tympanic membrane: opacity, defect, absence.
- Canal stenosis, hyperplasia, exudate (Fig. 69.8).
- Tympanic membrane bulging.
  - Pars flaccida (can be present in normal dogs)
  - Pars tensa (rare)



Fig. 69.8. Otic stenosis and hyperplasia in a mixed breed dog.

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- Abscess, draining tracts at ear base.
- Mass in ear canal, middle ear.



THERAPEUTICS

- Identify and manage cause of chronic otitis externa.
- Remove otic exudate (Fig. 69.8).
- Treat otitis media topically and systemically based on culture/sensitivity.
- Manage pain, pruritus, stenosis and/or hyperplasia.

Anesthetized Myringotomy and Tympanic Cavity Flush (Fig. 69.9)

- Cytology and culture of tympanic cavity effusion.
- Foreign body removal.
- Biopsy, avulsion of ear canal neoplasms.

Surgery

- Indicated for neoplasia or end-stage pathology.
  - Polyp/cyst: avulsion
  - TECA-BO
  - Severe stenosis, hyperplasia of ear canal
  - Invasive/occlusive neoplasia
  - Bulla lysis
- Lateral resection.
  - Noninvasive neoplasm of vertical canal, normal horizontal canal

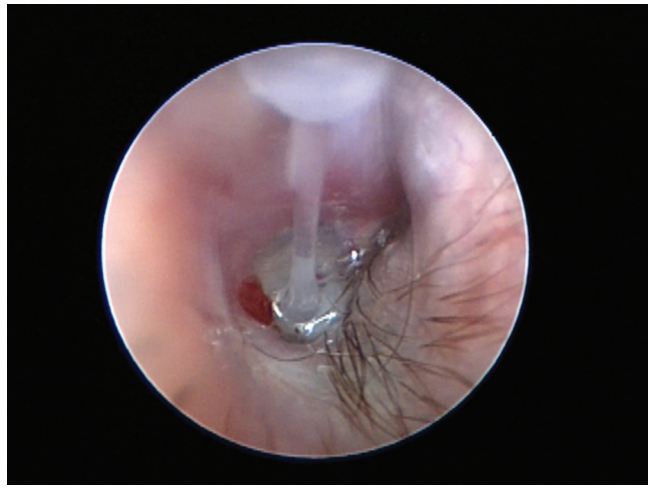


Fig 69.9. Aspiration of mucus from the tympanic cavity following anesthetized myringotomy in a Cavalier King Charles spaniel with PSOM.

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Drugs of Choice

Topical

- Avoid aminoglycosides, ointments to reduce ototoxicity risk.
- Use of culture and sensitivity data for topical antimicrobial selection is controversial.
- Triz-EDTA flush: every 24–48 hours.
- Enrofloxacin.
  - Commercial preparation with silver sulfadiazine
  - Compounded injectable enrofloxacin diluted in saline to 5 mg/mL
- Silver sulfadiazine.
  - Compounded silver sulfadiazine emulsion: 1 part cream to 9 parts sterile water
- Ticarcillin.
  - Compounded injectable diluted to 100 mg/mL
  - Aliquots stored up to a month in freezer
  - Recommended for enrofloxacin, silver sulfadiazine treatment failure
- Miconazole.
  - Compounded in sterile saline to a concentration of 5–20 mg/mL
- Dexamethasone.
  - Compounded injectable diluted in saline to 0.5–4 mg/mL
  - Indicated post middle ear flush, otitis externa

Systemic

- Predniso(lo)ne.
  - Pain and pruritus control: 0.25–0.5 mg/kg/day
  - Starting doses are tapered over a period of 4–8 weeks
  - Consider prior to topical therapy or anesthetized ear flushing
  - Severe stenosis, hyperplasia: 1 mg/kg/day for 7–21 days
- Antimicrobials.
  - Selection based upon middle ear culture/sensitivity
  - Treat 4–8 weeks, 12 weeks for bulla changes without surgery

Precautions Interactions

- Avoid topical aminoglycosides, ointments, and ear cleansers.
- Use TrizEDTA otic cleansers.

Alternative Therapies

- Acupuncture and therapeutic laser may reduce otitis externa pain and inflammation.
- Pursue systemic and topical treatments based on external ear canal cytology and culture if anesthetized flushing is declined.

Nursing Care

- Vestibular syndrome: supportive care for nonambulatory patients.
- Facial nerve paresis: artificial tears.

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Activity

- Supervise patients with vestibular syndrome when navigating stairs.

COMMENTS

Client Education

- Appropriate otic topical application techniques.
- Clients should recognize and report signs of ototoxicity.
- Follow-up is required to avoid relapse.
- Allergic otitis cases minimally require chronic maintenance topical corticosteroid therapy.
- Elimination diet trial is recommended for patients with nonseasonal otitis externa.

Patient Monitoring

- Recheck patient 7–14 days after initiation of therapy.
- Once managed and maintenance therapy instituted, reassess every 1–4 months or as needed.

Prevention/Avoidance

- Maintenance topical antiinflammatory 1–3 times weekly for chronic allergic otitis and for patients with permanent partial stenosis and otic hyperplasia.
- Identification and management of underlying cause.

Possible Complications

- Ototoxicity is possible with topical therapy if the tympanic membrane is compromised.
- Failure to manage allergic otitis may lead to relapse of otitis externa, media, and interna.

Expected Course and Prognosis

- Chronic allergic otitis: prognosis depends on degree of pathologic change. Chronic allergic patients will require ongoing therapy to prevent relapse and progression.
- Neurologic deficits may be permanent; vestibular and Horner's syndromes are typically temporary, but may take weeks of therapy to improve.
- PSOM: prognosis for reduction in signs is excellent with tympanic cavity flush.
- Multiple lifetime relapses may occur.
- TECA-BO for end-stage ear: temporary or permanent facial nerve paresis, temporary vestibular syndrome, cholesteatoma or abscess are rare complications.

Synonyms

- Labyrinthitis: otitis interna
- Auditory tube: eustachian tube

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- Facial nerve: cranial nerve VII
- Myringotomy: incision in the tympanic membrane
- Tympanic membrane: ear drum

See Also

Vestibular Disease

Suggested Reading

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Author: Jennifer S. Schissler, DVM, MS

chapter 70

Paraphimosis

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DEFINITION/OVERVIEW

- Paraphimosis is the inability to retract the erect or nonerect extruded penis into the preputial sheath. It must be differentiated from priapism, which is a state of continuous erection usually due to neurologic abnormality (Fig. 70.1).
- Paraphimosis of the erect penis is an emergency condition because constriction of venous blood flow will lead to greater engorgement, necrosis, damage to urethra, and gangrene. Chronic extrusion of the penis from the prepuce leads to drying, desiccation, licking, and trauma.



Fig. 70.1. Paraphimosis in a young dog. This patient was treated with cool sugar solution placed on the penis, lubrication of the penis with sterile lubricant, retraction of the edges of the prepuce to prevent the preputial orifice from inverting during replacement of the penis, and a small incision in the preputial orifice that was subsequently repaired with sutures and a purse-string suture.

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ETIOLOGY/PATHOPHYSIOLOGY

- Causes include chronic licking, sexual excitement, balanoposthitis, entrapment of penis outside prepuce during detumescense, neurologic disease (intervertebral disk herniation, encephalitis), constriction of the penis due to scar tissue or foreign material (e.g., malicious placement of string or rubber band), as well as anatomic issues (os penis fracture) or muscular issues such as inefficiency of preputial musculature.
- Paraphimosis may be idiopathic.
- Rarely, paraphimosis may be congenital, due to an abnormally large preputial opening, congenital preputial shortening, or paralysis of retractor penis muscle.

Systems Affected

- Renal/urologic
- Reproductive
- Skin/exocrine



CLINICAL FEATURES

- Dogs with paraphimosis of short duration may not have any signs other than the dog's licking of an exteriorized penis. After some hours of exposure, ischemic necrosis and urethral obstruction can develop.



DIFFERENTIAL DIAGNOSIS

- Priapism



THERAPEUTICS

- Paraphimosis requires immediate treatment. After 24 hours, the tissue damage and urethral obstruction may require penile amputation. If urethral patency is in question, place an indwelling urinary catheter. Replacement of the penis in normal position within the prepuce is the goal.
- Anesthesia may be necessary to perform the necessary manipulation to correct the problem.
- Cooled hyperosmotic agents such as dextrose 50%, concentrated sugar solution, concentrated magnesium sulfate solution (Epsom salts), or concentrated saline solution are used to reduce edema and shrink the penis.
- Cold packs may help reduce inflammation and swelling of the penis.
- Penis should be cleaned prior to replacement.
- Lubricants such as K-Y jelly or triple antibiotic ointment may aid in the return of the penis to the sheath.

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- Hair rings or foreign materials should be removed.
- Surgical release of the paraphimotic ring may be required.
- An abdominal compression bandage and indwelling urinary catheter to maintain the penis within the prepuce may also reduce localized edema.

Surgical Considerations

- Surgical release of the prepuce by making a small incision in the preputial orifice may be required to replace the engorged penis back into the prepuce. The preputial incision can be closed in two layers like any other laceration or incision.
- A purse-string suture around the preputial orifice may be considered to help prevent recurrence of paraphimosis until the penile swelling resolves. Use care to avoid overtightening of the purse-string suture, to allow normal urination and instillation of antibiotic ointment into the prepuce.
- Severe cases that result in penile and urethral necrosis may require penile amputation and prepubic urethrostomy.



COMMENTS

Client Education

- Owners should be instructed to prevent the dog from licking its penis and to avoid activities that may result in excitement and penile erection.
- Owners should be instructed to keep hair out of the preputial orifice to prevent hair rings from occurring.

Suggested Reading

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Author: Scott P. Shaw, DVM

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chapter 71

Parvoviral Enteritis – Canine



DEFINITION/OVERVIEW

- CPV is a highly contagious disease of immature and poorly vaccinated dogs.
- CPV affects the GI tract and lymphoid tissues, and if left untreated, is associated with high morbidity and mortality.



ETIOLOGY/PATHOPHYSIOLOGY

- Infection occurs by fecal–oral transmission.
- Incubation period ranges from 3 to 8 days.
- The virus initially replicates in the lymphoid tissues of the oropharynx, then subsequently spreads via the bloodstream to rapidly dividing cells of the GI tract, other lymphoid tissues (mesenteric lymph nodes, spleen, thymus), and bone marrow.
- CPV affects the germinal epithelium of the intestinal crypts, and results in destruction of epithelium and impaired normal cell turnover. This, in turn, results in villous atrophy and sloughing. Bacterial translocation, bacteremia, and endotoxemia are common sequelae of the impaired GI mucosal barrier.
- Bone marrow involvement results in leukopenia characterized by lymphopenia and neutropenia.

Systems Affected

- Gastrointestinal: vomiting and diarrhea are consequences of destruction of the intestinal crypt epithelium.
- Hemic/lymphatic/immune: leukopenia occurs secondary to bone marrow involvement.
- Cardiovascular: rapidly worsening dehydration may progress to signs of shock and cardiovascular collapse due to large fluid losses. *In utero* and early neonatal infection, although uncommon, can lead to early-onset heart failure.

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SIGNALMENT/HISTORY

- Signs and observations often reported by the owner include lethargy, anorexia, vomiting, and brown or hemorrhagic diarrhea.

Risk Factors/Causes

- Overcrowded and unsanitary conditions, intestinal parasitism and lack of protective immunity.
- Any breed may be affected, but the following breeds are at increased risk: American pit bull terrier, Doberman pinscher, Rottweiler, Labrador retriever, and German shepherd dog. It is not apparent whether a genetic predisposition exists, as opposed to breed popularity or increased proportion of unvaccinated dogs in these breeds.
- Puppies between 6 weeks and 6 months of age are most commonly affected. In dogs older than 6 months of age, intact males are twice as likely to acquire CPV infection as intact females.

CLINICAL FEATURES

- Depression/lethargy (Fig. 71.1).
- Fever.
- Dehydration characterized by tacky mucous membranes, prolonged skin tent, sunken eyes. Frequently, dehydration may progress to clinical signs of shock that include poor pulse quality, tachycardia, tachypnea, prolonged CRT, cool extremities.
- Painful abdomen; a mass effect may be palpable if intussusception is present.



Fig. 71.1. Profound lethargy is a frequently diagnosed clinical sign in canine parvoviral infections.

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DIFFERENTIAL DIAGNOSIS

- Other infectious diseases.
  - Bacterial: *Campylobacter, Salmonella, Clostridium*
  - Viral: coronavirus, CDV
- Severe parasitic or protozoal infestation: *Giardia*, *Coccidia*, *Cryptosporidium*, roundworms, hookworms, whipworms.
- Foreign body or intussusception.
- Dietary indiscretion.
- Toxin ingestion.
- Metabolic diseases: hepatic, renal, hypoadrenocorticism.
- Pancreatitis.
- Central nervous system disease.

DIAGNOSTICS

- Fecal ELISA antigen test is a practical cage-side test that has good sensitivity and specificity (Fig. 71.2).
  - Weak false positives may occur within 5–12 days of administration of an attenuated vaccine.
  - False-negative results may be seen due to a short period of fecal CPV shedding.



Fig. 71.2. Fecal antigen enzyme-linked immunosorbent assay (ELISA) is a quick and convenient bedside diagnostic test for canine parvovirus infections.

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- Fecal PCR: highly sensitive and specific; can aid in distinguishing between a vaccine and virulent strains of CPV; positive fecal PCR has been reported for up to 22 days after MLV vaccine. Although commercially available, PCR is not commonly used as a diagnostic tool.
- Serology: positive results are not diagnostic for an active CPV infection, as 25–90% of healthy animals may be seropositive secondary to a previous subclinical infection. Detection of IgM antibodies combined with suggestive clinical signs is necessary for diagnosis.
- Immunochemistry, latex agglutination, viral isolation, and electron microscopy may be used to demonstrate virus in tissue culture, feces, or tissues. These tests require specialized diagnostic laboratories.

Pathologic Findings

- Gross findings: thickening and segmental discoloration of distal duodenum, jejunum and ileum; denudation of intestinal mucosa; abdominal lymphadenopathy.
- Microscopic findings: shortening and obliteration of intestinal villi; crypt epithelial necrosis; necrosis of lymphoid tissues (spleen, thymus, Peyer's patches, mesenteric lymph nodes); viral intranuclear inclusion bodies may be visualized in epithelium of the GI tract.



THERAPEUTICS

- The objectives of treatment are correction of dehydration or shock, restoration of fluid and electrolyte balance, prevention or treatment of secondary bacterial infection, and aggressive supportive care while the GI tract is recovering.
- Intravenous fluid administration is the mainstay of therapy of CPV infections. High fluid rates are frequently required because of severe fluid depletion and significant ongoing losses in the form of vomiting and diarrhea. If an animal is in shock, an initial fluid bolus of 20–30 mL/kg of a balanced isotonic crystalloid solution over 15–30 minutes is indicated, followed by reassessment of perfusion parameters (i.e., heart rate, CRT, blood pressure) to determine if additional fluid resuscitation is required. The goal of resuscitation is return to normal hemodynamic parameters (i.e., blood pressure, mucous membrane color, CRT, heart rate). If an intravenous fluid bolus is not required or after it has been completed, the fluid rate may be determined by approximating the animal's fluid deficit (percent dehydration × body weight in kg) and replacing it over 12–24 hours, adding on maintenance needs (2–4 mL/kg/h) and ongoing fluid losses; typical fluid rates average ~10 mL/kg/h.
- If crystalloid solutions fail to restore perfusion, if total protein levels decrease to below 4 g/dL, or if a patient develops signs of peripheral edema, synthetic colloids may be beneficial. A 3–5 mL/kg bolus may be given to an animal in shock; alternatively, an infusion at the rate of 1 mL/kg/h may be administered if a bolus is not indicated. Traditionally, a total dose of colloids should not exceed 20–30 mL/kg per day; however, in patients that are critically ill that require very high fluid rates, this limit may be exceeded, if necessary. Despite recent concerns in human critical care, there is no current evidence to suggest that colloid use in dogs with parvovirus is independently associated with mortality.

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Drugs of Choice

Antibiotics

- Indicated due to destruction of the GI mucosal barrier and high potential for bacterial translocation, as well as leukopenia, which may predispose the animal to secondary bacterial infections.
- Combination therapy may be used to provide broad-spectrum coverage: beta-lactam (ampicillin 22 mg/kg IV, SQ every 6–8 hours) and fluoroquinolone (enrofloxacin IV, SQ 10–15 mg/kg every 24 hours) or aminoglycoside (gentamicin 6.6 mg/kg IV, SQ every 24 hours); see Precautions/Interactions.
- Single-agent broad-spectrum antibiotics may be used, such as cefoxitin (15–30 mg/kg IV, SQ every 6 hours) or ampicillin/sulbactam (22 mg/kg IV, SQ every 8 hours).

Antiemetics

- If vomiting or nausea persists, antiemetic therapy is indicated. The following medications may also be used alone or in combination with each other.
  - Serotonin antagonists (dolasetron 0.5–1 mg/kg every 12–24 hours)
  - Maropitant (1 mg/kg SQ every 24 hours for 5 days); not approved for use in dogs under 8 weeks
  - Phenothiazine derivatives (chlorpromazine 0.1–0.5 mg/kg IM, SQ every 8–12 hours); see Precautions/Interactions
  - Metoclopramide: intermittent injections (0.1–0.4 mg/kg SQ every 6 hours) or an intravenous CRI constant rate infusion (1–2 mg/kg per day); see Precautions/Interactions

Pain Medications

- As some animals with CPV infections may exhibit significant abdominal pain, pain management is a useful adjunct to therapy. Opioid medications may be used, such as buprenorphine (0.01–0.02 mg/kg IV, IM, SQ every 6–8 hours), hydromorphone (0.1 mg/kg SQ, IM, IV every 6–8 hours), fentanyl (3–10 µg/kg/h IV CRI); see Precautions/Interactions.

Supplements

- Patients with CPV infections are frequently hypokalemic because of the GI tract losses, as well as hypoglycemic because of decreased storage abilities and lack of oral food intake; potassium (maximum rate of 0.5 mEq/kg/h) and glucose (2.5–5% added to intravenous fluids) often require supplementation.

Precautions/Interactions

- One of the adverse effects of aminoglycosides is nephrotoxicity, which could be exacerbated by dehydration or hypovolemia. These agents should not be administered to dehydrated patients; hydration should be restored prior to initiation of therapy. If these drugs are used, daily urine sediment examinations should be performed to look for casts; therapy should be discontinued if casts are visualized.

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- Fluoroquinolones should be used with caution in immature dogs because cartilage abnormalities have been associated with their administration.
- Phenothiazine derivative medications cause vasodilation and sedation, which may potentiate hypotension in animals with suboptimal perfusion and increase the chance of aspiration of GI contents in vomiting animals.
- Sedation may also be a side effect of opioid medications.
- Anticholinergic agents should not be administered because they may increase the potential for intestinal ileus and intussusception.
- Prokinetics, such as metoclopramide, are contraindicated in animals with high suspicion of GI obstruction, as there could be an increased risk of GI rupture.

Alternative Drugs

- Oseltamivir has been advocated for use in CPV infection. Despite certain favorable trends, it has not been shown to make a significant difference in management of CPV.
- FFP may be beneficial, as it provides immunoglobulins, as well as serum protease inhibitors to aid in neutralizing the virus and combat associated inflammation. In dogs suffering from coagulopathy due to DIC, FFP will provide coagulation factors as well.

Diet

- Oral intake should be discontinued in vomiting patients with CPV infection until vomiting is significantly reduced to absent. To reinitiate oral intake, small amounts of water should be offered and if tolerated, followed by frequent small meals of highly digestible low-fiber, low-fat diet.
- Early enteral nutrition has been advocated in CPV infections and is associated with earlier clinical improvement and significant weight gain; this can be achieved by placing a feeding tube, if a patient is anorexic. Trickle feeding small amounts of enteral nutrition has been shown to decrease patient morbidity and mortality, even in the face of vomiting.
- In severely affected animals, parenteral nutrition may be life-saving.

Activity

- No activity restrictions are necessary during or after recovery from CPV infection.
- The virus can be shed in the animal's feces for 14–30 days, so the animal and its feces should be isolated from other animals during this potentially infective period.

Surgical Considerations

- Patients with CPV infection who develop an intussusception require emergency surgery; these patients may be at higher risk of developing perioperative complications due to their immunocompromised state. Fluid resuscitation is essential in dehydrated and hypovolemic dogs prior to surgery.

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COMMENTS

Client Education

- Other immature or poorly vaccinated dogs in the household are susceptible to CPV infection; they should be evaluated by a veterinarian and vaccinated as soon as possible.
- The household, premises, and any other objects that the affected animal has come into contact with should be disinfected with 1:30 dilution of bleach.
- CPV is ubiquitous and can survive in the environment for 5 months or longer, so owners should avoid obtaining a new puppy for at least 6 months.
- Convalescing animals typically shed large amounts of virus for up to 2 weeks after resolution of infection and therefore should be isolated for that period of time.
- Even though immunity to CPV infection is long lasting and possibly lifelong, it is imperative that the animal is vaccinated for other infectious diseases after recovery.

Patient Monitoring

- Frequent monitoring is essential to help guide therapeutic decisions. Physical parameters (i.e., temperature, mucous membrane color, CRT, heart rate, pulse quality, degree of abdominal pain) and blood pressure should be checked 4–6 times daily, as indicated.
- Packed red blood cell volume, total solids, and blood glucose should ideally be monitored at least twice daily. Electrolytes, acid–base status, and weight should be monitored at least once daily.

Prevention/Avoidance

- Attenuated live vaccine is the method of choice for prevention of CPV infection. Vaccination should be initiated at 6–8 weeks of age and repeated every 2–3 weeks until 16–18 weeks of age. In high-risk breeds, evaluation of CPV titers may be useful in ensuring appropriate levels of protection. Maternal antibody interference is considered the biggest cause of vaccine failure; use of high-titer vaccines minimizes the chances of failure and is the current preferred vaccine.
- Early socialization of healthy puppies at training classes, prior to completion of the full vaccine series, does not increase risk of CPV; however, high traffic areas frequented by dogs with unknown health status, such as dog parks and pet stores, should be avoided prior to complete vaccination.

Possible Complications

- Aspiration pneumonia due to vomiting; septic shock secondary to GI bacterial translocation and leukopenia; thromboembolism due to a hypercoagulable state, which may be followed by hypocoagulability.
- Dogs may be coinfected with one or more infectious agents, including *Clostridium perfringens* alpha toxin (CPA), *Cryptosporidium* spp., and *Giardia* spp.

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Expected Course and Prognosis

- Average recovery time is 6 days.
- Prognosis for survival in animals that receive aggressive supportive care early in the course of disease is good to excellent; poor without treatment; and poor to guarded with outpatient care.

Synonyms

- Parvo

Abbreviations

CDV: canine distemper virus
CPV: canine parvovirus
CRI: constant rate infusion
CRT: capillary refill time
DIC: disseminated intravascular coagulation
ELISA: enzyme-linked immunosorbent assay
FFP: fresh frozen plasma
GI: gastrointestinal
IgM: immunoglobulin M
IM: intramuscular
IV: intravenous
PCR: polymerase chain reaction
SQ: subcutaneous

Suggested Reading

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Author: Cynthia M. Otto, DVM, PhD
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chapter 72

Pericardial Effusion



DEFINITION/OVERVIEW

- Pericardial effusion is defined as the accumulation of fluid within the pericardial sac.

ETIOLOGY/PATHOPHYSIOLOGY

- Pericardial effusion is a relatively common reason for dogs to present on an emergency basis.
- Pericardial effusion is much more common in dogs than cats.
- Similar to fluid accumulations in other spaces in the body, pericardial effusion occurs due to one or a combination of the following factors.
    - Increased vascular permeability (e.g., hemorrhage)
    - Decreased capillary oncotic pressure (e.g., severe hypoalbuminemia)
    - Increased capillary hydrostatic pressure (e.g., right-sided heart failure)
    - Decreased lymphatic drainage (e.g., neoplasia)
- Common etiology of pericardial effusion is as follows.

Dogs

- Neoplasia.
    - Hemangiosarcoma
    - Heart base tumor
        - Aortic body tumor (chemodectoma)
        - Ectopic malignant thyroid or parathyroid tissue
    - Mesothelioma
    - Lymphoma
    - Any metastatic neoplasia (e.g., carcinoma)
- Idiopathic pericardial effusion.
- Anticoagulant rodenticide intoxication.
- Trauma.

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Companion website: www.fiveminutevet.com/emergency

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- Infection (hematogenous versus direct seeding due to foreign object).
  - Fungal
  - Bacterial
  - Protozoal
- Cardiac conditions.
  - Left atrial rupture due to chronic mitral regurgitation and left atrial dilation
  - Congestive heart failure
  - PPDH
- Inflammatory conditions.
  - Pancreatitis
  - Renal failure
  - Immune-mediated diseases
- Hypoalbuminemia.

Cats

- Cardiac conditions.
  - CHF is the most common cause of pericardial effusion in the cat
  - PPDH
  - Pericardial cyst
- Neoplasia.
  - Lymphoma
  - Metastatic neoplasia
- Infectious disease.
  - FIP
- Hypoalbuminemia.
- Pericardial effusion should be differentiated from cardiac tamponade.
  - Low-volume pericardial effusion such as may occur with hypoalbuminemia or various systemic inflammatory disorders may not cause any clinical signs.
  - Cardiac tamponade refers to the condition in which pericardial effusion results in progressively increasing intrapericardial pressure that impairs diastolic filling and manifests with signs of cardiovascular compromise including both forward failure (shock) and right-sided CHF. Cardiac tamponade is more common in dogs than cats.

Systems Affected

Cardiovascular

- Increased intrapericardial pressures result in decreased diastolic filling.
- Decreased diastolic filling results in decreased stroke volume.
- Decreased stroke volume results in decreased cardiac output.
- Decreased cardiac output results in decreased oxygen delivery to the tissues (shock).
- Signs of right-sided CHF predominate.
- Cardiovascular manifestations of pericardial effusion are important to the diagnosis of the condition based on physical examination.

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Nervous

- Decreased mentation (i.e., obtunded, stupor, or coma) may occur secondary to hypotension and decreased oxygen delivery to the brain.

Gastrointestinal

- Gastrointestinal manifestations of pericardial effusion are likely a result of decreased gastrointestinal blood flow and may include vomiting or diarrhea.

Renal/Urologic

- Decreased renal blood flow may result in prerenal azotemia.



SIGNALMENT/HISTORY

- The underlying etiology of pericardial effusion may determine the patient population most commonly affected.
  - Adult to geriatric golden retrievers and German shepherds commonly develop pericardial effusion secondary to hemangiosarcoma involving the right atrium and surrounding tissues.
  - Brachycephalic breeds and retriever breeds may have a higher incidence of heart base tumors.
  - Young dogs may be more prone to developing pericardial effusion secondary to anticoagulant rodenticide ingestion.

Risk Factors/Causes

- Rodenticide ingestion
- History of neoplasia, especially hemangiosarcoma, lymphoma, carcinoma, and mesothelioma at local or distant sites
- Historical cardiac disease
- Known septic focus
- Conditions that cause systemic inflammation or hypoalbuminemia
- Recent cardiac surgery
- Recent trauma

Historical Findings

- Presenting complaints.
  - Collapse
  - Lethargy
  - Anorexia
  - Vomiting
  - Abdominal distension

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- Duration of signs.
  - Dogs and cats with pericardial effusion may have peracute history (minutes to hours) or a chronic history (days to weeks).



CLINICAL FEATURES

General

- Pericardial effusion resulting in cardiac tamponade should be highly suspected based on thorough physical examination. Echocardiography is critical to definitive diagnosis.

Physical Examination

Cardiovascular System

- Pale mucous membranes.
- Prolonged capillary refill time.
- Tachycardia.
- Poor pulse quality.
- Auscultation of the heart reveals decreased heart sounds. Lung auscultation is generally normal.
- Jugular venous distension (with or without pulsations) This finding helps differentiate pericardial effusion from hemoperitoneum.
  - Compression of the cranial abdomen may exacerbate jugular venous distension (with or without pulsation). This is termed the hepatojugular reflex.
- Pulsus paradoxus is a normal phenomenon in which pulse quality varies with the phase of the respiratory cycle. Pericardial effusion exacerbates pulsus paradoxus.
- Arrhythmia may be noted while concurrently palpating the pulse and ausculting the heart.
- Abdominal distension results from right-sided CHF. High intrapericardial pressures result in high caudal caval pressures, which are transmitted to the hepatic venous system, resulting in abdominal effusion.

Respiratory System

- Increased respiratory rate and effort may occur.
- Lung sounds are usually normal for the degree of respiratory effort. Lung sounds may be decreased if concurrent pleural effusion is present.

Central Nervous System

- Varying degrees of altered mentation may be noted due to a lack of oxygen delivery to the brain. These may include obtundation, stupor, or coma.

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DIFFERENTIAL DIAGNOSIS

Hypovolemia Due to Hemoperitoneum

- Hypovolemia due to blood loss (into any potential space) may result in many identical cardiovascular, respiratory, and CNS signs.
- Differentiating findings.
  - In hypovolemia, the jugular vein is generally small or flat.
  - Heart sounds are usually normal in hypovolemia although they may be quiet.
  - Pulsus paradoxus is not common in hypovolemia.
- Abdominocentesis will help rule out hemoperitoneum.

CHF Due to Other Cardiac Diseases

- Other cardiac disease causing CHF and cardiogenic shock may result in similar cardiovascular signs of pericardial effusion.
  - Right-sided disease
    - Dilated cardiomyopathy (dogs and cats)
    - Heartworm disease (caval syndrome)
    - Tricuspid regurgitation
    - Congenital cardiac anomalies
  - Left-sided disease
    - Dilated cardiomyopathy (dogs and cats)
    - Hypertrophic cardiomyopathy (cats)
    - Mitral regurgitation (dogs)
    - Congenital cardiac anomalies
- Differentiating findings.
  - Animals with CHF generally have very audible heart sounds.
  - Animals with CHF generally have heart murmurs or other auscultable abnormalities (gallop rhythm in cats).
  - Animals with CHF (especially dilated cardiomyopathy in dogs) often present with evidence of atrial fibrillation and other arrhythmias.
  - Animals in left-sided CHF often have pulmonary edema with increased lung sounds and more pronounced respiratory distress.
  - Echocardiography will definitively differentiate between CHF and pericardial effusion.



DIAGNOSTICS

Hemostasis Assessment

- Rodenticide intoxication is a cause of pericardial effusion.
- Hemostasis testing should be performed prior to pericardiocentesis
- Point-of-care coagulation analyzers are available to measure PT and aPTT.

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- ACT is an alternative to aPTT and may aid in the identification of severe intrinsic/common cascade abnormalities without the need for a coagulation analyzer.
- Blood smear can be rapidly performed to identify the presence of severe thrombocytopenia.

Diagnostic Tests

Echocardiography/Ultrasound

- Point-of-care ultrasound capabilities are critical to the management of dogs and cats in the emergency setting.
- Allows for rapid, definitive diagnosis of pericardial effusion.
- May aid in identification of an underlying cause (e.g., neoplasia).
- Minimally invasive.
- Dogs and cats are best imaged from the right cardiac notch.
- Findings.
    - Hypoechoic fluid between the epicardium and the echodense pericardium. (Fig. 72.1)
    - Inward deviation/compression of the right atrium
    - With or without presence of a mass (Fig. 72.2)
    - Due to stretch of the pericardium over time, it should be noted that small-volume pericardial effusion may be associated with severe clinical signs if accumulation occurs acutely. Conversely, if accumulation occurs slowly, larger volume accumulations may result in only mild clinical signs.

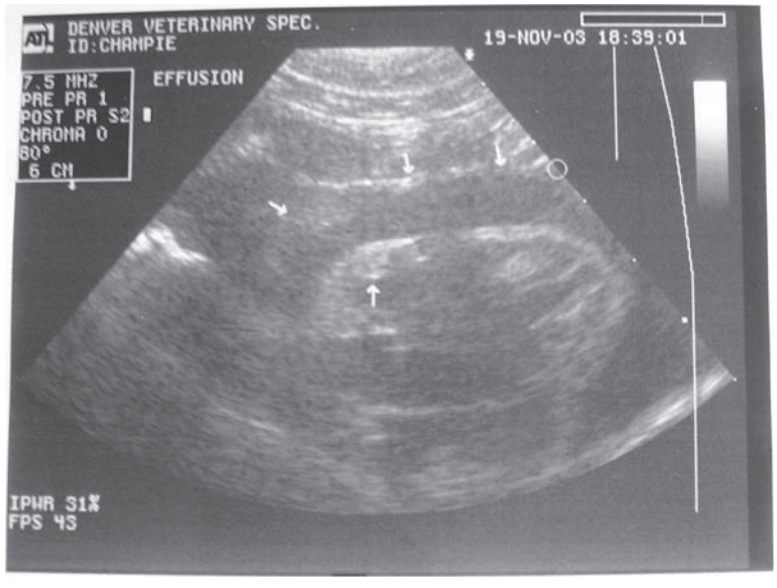
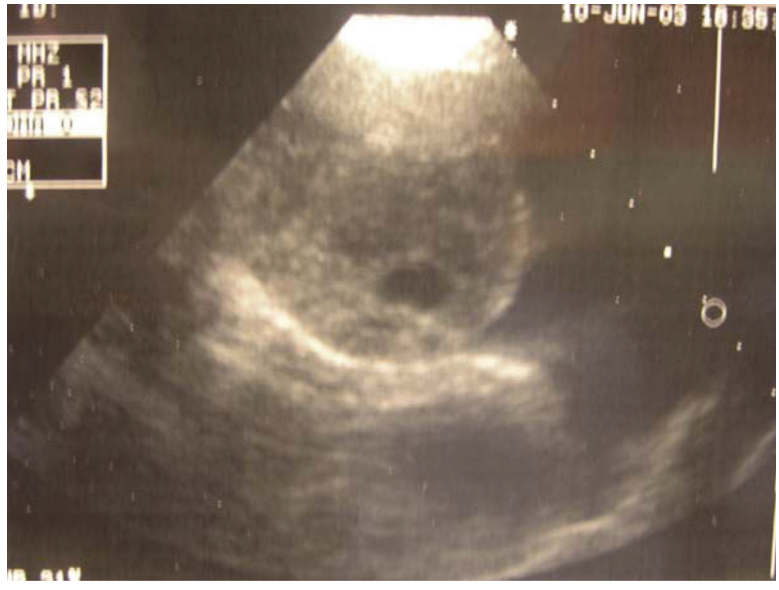


Fig. 72.1. Pericardial effusion in a cat. Note the hyperechoic pericardium and hypoechoic fluid in between the pericardium and the heart.

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■ **Fig. 72.2.** Ultrasound image of pericardial effusion and a heart base tumor in a dog.

Electrocardiography

- Electrocardiography provides a rapid diagnostic tool that will aid in the identification of pericardial effusion.
- Findings in lead II supportive of pericardial effusion (*indicates critical finding).
  - Decreased QRS complex amplitude (<1 mV)*
  - Electrical alternans resulting from movement of the heart within the pericardial sac to and from the positive pole of lead II (Fig. 72.3)
  - Tachycardia
  - Arrhythmias
  - ST segment changes (elevation or depression)

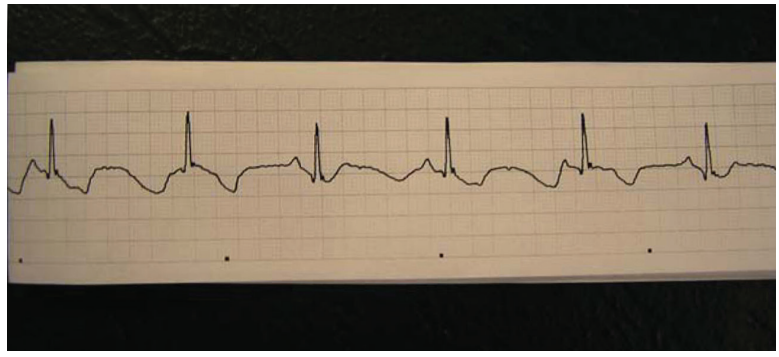
Abdominocentesis

- Although not diagnostic for pericardial effusion, abdominal effusion that accumulates in patients with pericardial effusion is frequently a modified transudate in character. Abdominocentesis can rule out hemoperitoneum, a condition that mimics some of the clinical findings of patients with pericardial effusion.

Blood Gas Analysis

- Although not diagnostic for pericardial effusion, blood gas analysis may reveal metabolic (lactic) acidosis associated with decreased oxygen delivery to the tissues due to cardiogenic shock.

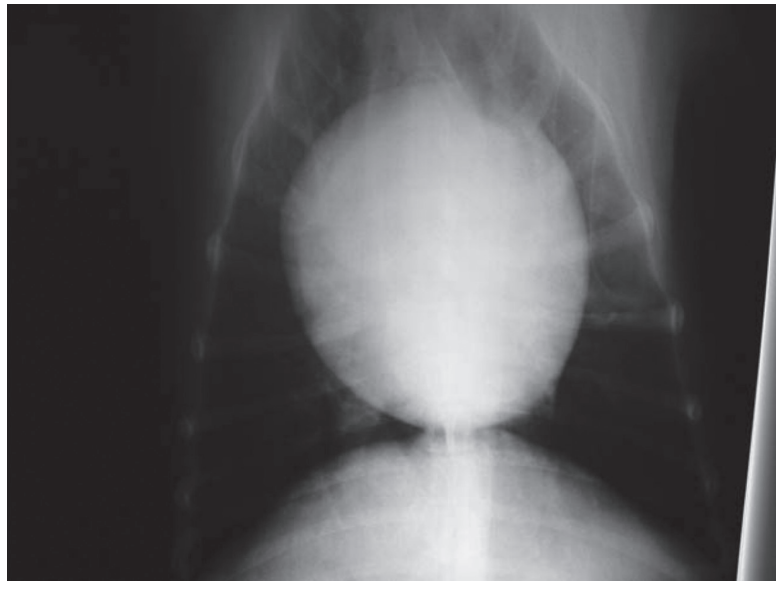
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■ **Fig. 72.3.** Electrocardiogram strip of electrical alternans in a dog with pericardial effusion. Note the higher amplitude complexes followed by lower amplitude complexes as the heart swings in the fluid within the pericardial sac. Electrical alternans is not always seen in all cases of pericardial effusion.

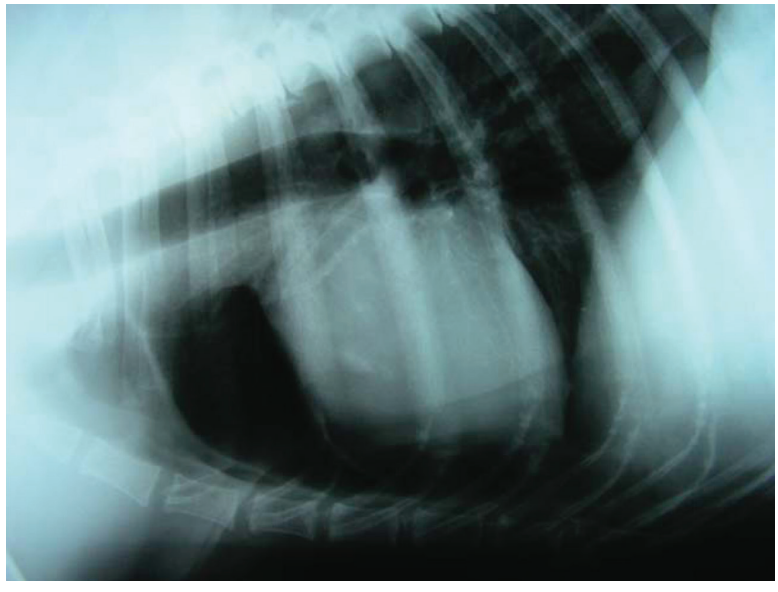
Thoracic Radiography

- Thoracic radiography should be avoided if possible in favor of echocardiography and electrocardiography because it may require significant patient manipulation and stress.
- If necessary, the patient should be stabilized prior to performing thoracic radiography.
- Views.
  - Dorsoventral
  - Lateral
- Findings.
  - Enlarged, globoid cardiac silhouette (Fig. 72.4)
    - Acute pericardial effusion may demonstrate a normal cardiac silhouette



■ **Fig. 72.4.** Globoid cardiac silhouette in a dog with pericardial effusion.

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■ **Fig. 72.5.** Pericardial effusion in a dog whose pericardial sac has not had time to stretch, resulting in a normal-appearing cardiac silhouette with an enlarged caudal vena cava.

  - □ Sharp border of the cardiac silhouette is due to its fixed location in contrast to a moving pericardium/epicardium in a dog that does not have pericardial effusion
  - □ Enlargement of the caudal vena cava
  - □ Low-volume pleural effusion may be present
  - □ Note that the cardiac silhouette may appear normal in cases of acute tamponade, when the pericardial sac has not had time to stretch. In such cases, the caudal vena cava still appears large (Fig. 72.5)
- Pulmonary changes
  - □ Pulmonary edema is uncommon in dogs with pericardial effusion
  - □ Pulmonary edema is common in dogs and cats with left-sided CHF
  - □ Pulmonary metastatic disease may be noted in dogs with hemangiosarcoma

Pericardial Fluid Analysis

- Pericardial fluid is retrieved with pericardiocentesis.
- Analysis should include assessment for clotting. Pericardial fluid that clots likely was retrieved from one of the cardiac chambers, or can be associated with ongoing hemorrhage (Fig. 72.6).
- Cytologic analysis may aid in the identification of the underlying cause, especially in bacterial and fungal pericarditis.
- Some types of neoplasia may also exfoliate cells into the pericardial fluid.
  - Mesothelioma; interpretive caution should be exercised as reactive mesothelial cells have many characteristics of malignancy
  - Lymphoma
  - Carcinoma

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■ **Fig. 72.6.** Evaluate the fluid carefully for clots because clots can form with inadvertent intracardiac punctures.

- Hemangiosarcoma and heart base tumors are rarely diagnosed cytologically.
- Aerobic, anaerobic, and fungal cultures should be performed if bacterial or fungal etiology is suspected cytologically.
- Acid–base analysis is not a useful predictor of the cause of pericardial effusion.

Cross-Sectional Imaging

- Only considered if traditional diagnostic testing is nondiagnostic and the patient has been stabilized with pericardiocentesis.
- CT or MRI gated to the cardiac cycle may provide additional information as to the underlying cause for the pericardial effusion, especially in cases of neoplasia.

Pathologic Findings

Gross

- Distension of the pericardium with fluid.
- Gross changes in dogs with pericardial effusion will be a reflection of the underlying disease process.

Histopathologic

- Histopathologic changes in dogs with pericardial effusion will be a reflection of the underlying disease process.

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THERAPEUTICS

- The objective of treatment for dogs and cats with pericardial effusion is the establishment or maintenance of hemodynamic stability.
- Dogs and cats with cardiac tamponade require emergency evacuation of their pericardial effusion in order to achieve hemodynamic stability.
- Although pericardiocentesis for diagnostic purposes may be indicated, patients with low-volume pericardial effusion and few clinical signs may not warrant therapeutic pericardiocentesis.

Pericardiocentesis

- Oxygen therapy maximizes oxygen saturation.
- Achieve peripheral intravenous access for the delivery of medications including sedation if needed.
- There is little place for fluid therapy, diuretics, or vasodilators in the acute management of pericardial effusion.
- If, based on assessment of coagulation status, severe coagulopathy is considered a likely cause of pericardial effusion, correcting coagulation abnormalities with FFP (15–20 mL/kg) should be attempted prior to pericardiocentesis. FFP can be delivered rapidly (<1 hour) if necessary. However, if the pericardial effusion is associated with lactic acidosis or thought to be immediately life threatening, pericardiocentesis should be performed as plasma is being delivered.
- ECG should be monitored throughout the procedure.

Sedation

- Light sedation should be considered to help alleviate stress and anxiety and to decrease the likelihood of patient motion during pericardiocentesis.
  - Midazolam 0.1–0.2 mg/kg IV
  - Opioid (choose one)
    - Butorphanol 0.1–0.2 mg/kg
    - Fentanyl 1–3 µg/kg IV
    - Hydromorphone 0.05 mg/kg IV

Equipment

- Fenestrated drape.
- Pericardiocentesis device.
  - 14–16 gauge 5.25-inch over-the-needle catheter (large dogs)
  - 18 gauge 1–2-inch over-the-needle catheter (small dogs and cats)
- Intravenous extension tubing: three-way stopcock, 20–60 mL syringe interconnected
- 3 mL syringe filled to 1.5 mL with sterile saline

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Positioning

- Sternal recumbency.
- Landmarks for pericardiocentesis.
  - Right hemithorax
  - Ultrasound can be utilized to identify the optimal rib space for pericardiocentesis
  - Alternatively, the apex beat (if palpable) is a reasonable target
  - Alternatively, the elbow can be pulled back to where it touches the chest (third to fifth rib space)

Preparation

- Clip and aseptically prepare the right side of the thorax, centering on the third to sixth rib spaces in the ventral half of the chest.
- Apply a fenestrated drape.
- Sterile technique should be utilized at all times.

Technique

- Apply 3 mL saline syringe to end of pericardiocentesis catheter and flush.
- Advance pericardiocentesis catheter through skin in the predetermined location just cranial to the edge of a rib (Fig. 72.7).
- The "closed" system (syringe attached to over-the-needle catheter) prevents pneumothorax.



Fig. 72.7. Insert the long catheter in between rib spaces as described in the text, and watch for a flash of blood in the hub of the needle. Note that this technique should be performed in a sterile manner. This photo demonstrates location and patient anatomy for instruction purposes.

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- Advance pericardiocentesis catheter in 2–4 mm increments until a "flash" of fluid is seen in the syringe. Many effusions are hemorrhagic.
- Advance pericardiocentesis catheter 2 mm.
- Advance catheter over the stylet and into the pericardium.
- Connect intravenous extension tubing: three-way stopcock, 20–60 mL syringe to catheter and evacuate pericardial space.
  - Collect initial sample to assess for clotting. If the sample clots, remove the catheter and start over.
  - Collect additional samples as indicated.
- On occasion, the catheter will become occluded on the epicardium or inner pericardium. Rotation of the catheter and repositioning may be effective.
  - Create a 2–3 mm sidehole in the catheter approximately 7–10 mm from its tip prior to pericardiocentesis to minimize the likelihood of this problem. Care should be taken not to destabilize the structural integrity of the catheter by creating too large a defect.

Additional Treatment Considerations

- If anticoagulant rodenticide is considered the cause of the pericardial effusion, initiation of vitamin K_1 therapy is indicated after FFP administration and pericardiocentesis.
- Yunnan Baiyao can be considered in cases where hemangiosarcoma or heart base tumor is suspected or confirmed based on hemorrhagic pericardial effusion.

Precautions/Interactions

- Pericardiocentesis is not without risk. Injury to the chest wall (i.e., intercostal vascular injury), lung (i.e., pneumothorax), and heart (i.e., arrhythmias, chamber puncture, or coronary artery laceration) are all possibilities although very rare.
- Ventricular arrhythmias may be noted when the catheter contacts the epicardium. If this occurs, the position of the catheter should be modified.

Diet

- The patient should feel much better soon after pericardiocentesis. Feeding should be considered after hemodynamic stability is achieved.

Activity

- Activity should only be restricted if ongoing bleeding is considered a significant possibility such as in animals with anticoagulant rodenticide intoxication.

Surgical Considerations

- There is a specific place for the surgical management of pericardial effusion in a variety of conditions.

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- Idiopathic pericardial effusion is generally initially managed through pericardiocentesis. If the effusion reaccumulates, then open surgical or thoracoscopic evaluation of the heart and thorax should be performed with subtotal pericardectomy or pericardial window to eliminate recurrence respectively. As is routine, biopsies should be acquired to rule out the possibility of neoplasia (especially mesothelioma).
- Cardiac or pericardial neoplasia is initially managed through pericardiocentesis. Tumors of the right atrial appendage can often be removed quite easily. There is significant debate as to the value of subtotal pericardectomy or pericardial window in dogs with masses of the heart. Pericardectomy in dogs with masses that have not previously demonstrated severe, acute, or large-volume hemorrhage is recommended. Heart base tumors (i.e., aortic body tumors) commonly behave this way. Biopsies should be acquired for definitive diagnosis if possible. Hemorrhage from biopsy sites may be severe.
- Bacterial or fungal pericarditis may occur due to hematogenous spread or direct implantation due to penetrating injury or migrating foreign body. Pericardiocentesis should be performed to achieve initial stability. Pericardectomy and establishment of ongoing pleural drainage with thoracostomy tubes coupled with aggressive appropriate antimicrobial therapy are then indicated for definitive treatment.
- Surgical repair of left atrial rupture secondary to chronic mitral valve insufficiency is possible.
- Pericardectomy plays a role in the management of restrictive pericarditis.
- PPDH is managed by replacing abdominal contents back into the abdominal space and repairing the defect in the diaphragm.



COMMENTS

Client Education

- Client education will be specific to the underlying cause of pericardial effusion. Clients should monitor for recurrence of clinical signs which could signify reaccumulation of pericardial fluid.

Patient Monitoring

- Pericardiocentesis is considered one of the most rewarding clinical procedures because it results in very rapid improvement in clinical signs. Postprocedural monitoring may include any (or all) of the following.
  - Intermittent physical examination will allow the practitioner to monitor for abnormalities associated with pericardial effusion.
  - ECG will demonstrate heart rate and the presence of arrhythmias. If pericardial effusion reaccumulates, the heart rate will rise.
  - Intermittent point-of-care assessment for the recurrence of pericardial effusion can be performed via ultrasound or echocardiogram.

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- Central venous pressure will rise above the baseline measurements when pericardial effusion accumulates.

Prevention/Avoidance

- If a predisposing condition such as anticoagulant rodenticide intoxication is diagnosed as the cause of pericardial effusion, eliminating the toxin from the environment is critical to preventing a second bout of intoxication.

Possible Complications

- The most common long-term complication of pericardial effusion is recurrence. Recurrence rates will be dependent on the underlying disease process and the treatment rendered. For example, idiopathic pericardial effusion and pericardial effusion secondary to neoplasia are likely to recur.

Expected Course and Prognosis

- As a general rule, pericardiocentesis results in immediate relief of symptoms of pericardial effusion.
- Prognosis over time will be dependent on the underlying cause of the pericardial effusion.
  - Neoplasia: prognosis in dogs and cats with neoplasia is dependent on the type of tumor and the treatment rendered. For example, dogs with hemangiosarcoma that undergo surgical intervention for tumor resection and treatment with chemotherapy have a median survival time of approximately 5 months. Dogs with aortic body tumors treated with pericardectomy had median survival time of approximately 2 years in one series.
  - Idiopathic: dogs with idiopathic pericardial effusion generally respond favorably to pericardiocentesis. If recurrence occurs, pericardectomy is recommended. A good prognosis is generally given.
  - Cats: the most common cause of pericardial effusion in the cat is CHF. Prognosis in cats with pericardial effusion is generally dependent on the prognosis for the underlying disease process.

Abbreviations

ACT: activated clotting time
aPTT: activated partial thromboplastin time
CHF: congestive heart failure
CNS: central nervous system
CT: computed tomography
ECG: electrocardiogram
FFP: fresh frozen plasma

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FIP: feline infectious peritonitis
IV: intravenous
MRI: magnetic resonance imaging
PPDH: peritoneo-pericardial diaphragmatic hernia
PT: prothrombin time

Suggested Reading

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Author: Matthew W. Beal, DVM

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chapter 73

Pleural Effusion





DEFINITION/OVERVIEW

- Abnormal accumulation of fluid within the pleural space.



ETIOLOGY/PATHOPHYSIOLOGY

- Increased production of fluid or decreased reabsorption of fluid.
- Increased vascular permeability.
- Increased hydrostatic pressure of the capillaries or lymphatics.
- Decreased intravascular oncotic pressure.

Systems Affected

- Respiratory: compression of the lung parenchyma results in a restrictive breathing pattern and, if severe, hypoxemia,
- Cardiovascular: severe cases may affect cardiac output by limiting cardiac filling.



SIGNALMENT/HISTORY

Species

- Dogs and cats.

Breed Predilection

- Varies with underlying cause; Afghan hounds have a higher reported incidence of chylothorax.

Mean Age and Range

- Varies with underlying cause; older animals are more likely to be affected with diseases that result in pleural effusion.

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Predominant Sex

- None.

Historical Findings

- Respiratory difficulty
- Tachypnea
- Orthopnea
- Open mouth breathing
- Cyanosis
- Exercise intolerance
- Lethargy
- Inappetence
- Cough

Physical Examination Findings

- Respiratory difficulty with increased effort.
- Restrictive, rapid, and shallow respiratory pattern.
- Muffled heart and lung sounds.
- Dullness on thoracic percussion of ventral lung fields.

Risk Factors/Causes

- Increased hydrostatic pressure.
  - CHF
  - Overhydration (including anuric renal failure)
  - Intrathoracic neoplasia
- Decreased oncotic pressure.
  - Hypoalbuminemia (due to protein-losing enteropathy, protein-losing nephropathy, or severe hepatic disease); this is a less common cause
- Vascular or lymphatic abnormality.
  - Infectious: bacterial, viral, or fungal
  - Neoplasia (e.g., mediastinal lymphosarcoma, thymoma, mesothelioma, primary lung tumor, metastatic disease)
  - Chylothorax (e.g., from lymphangiectasia, idiopathic, CHF, cranial vena cava obstruction, neoplasia, fungal granuloma, heartworm disease, diaphragmatic hernia, lung lobe torsion, trauma)
  - Pancreatitis
  - Diaphragmatic hernia
  - Lung lobe torsion (Fig. 73.1)
  - Pulmonary thromboembolism
  - FIP

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■ **Fig. 73.1.** Photograph of a 5-month-old male pug that presented for acute onset of anorexia, cough, respiratory difficulty, and pleural effusion.

- Trauma.
  - Hemothorax
  - Chylothorax from damage to thoracic duct (uncommon)
- Coagulopathies.
  - Vitamin K antagonist rodenticide intoxication



DIFFERENTIAL DIAGNOSIS

Differentiating Causes

- If there is historical or physical evidence of external trauma, hemothorax, chylothorax, or diaphragmatic hernia should be considered.
- Presence of fever is suggestive of inflammatory, infectious, or neoplastic cause.
- Suspect underlying cardiac cause if murmurs, gallop, ectopy or pericardial effusion.
- Concurrent ascites is suggestive of FIP, CHF, pericardial effusion, severe hypoalbuminemia, diaphragmatic hernia, metastatic neoplasia, coagulopathy, or pancreatitis.
- Cats with cranial mediastinal masses may have decreased compressibility of the cranial thorax.
- Concurrent ocular changes such as chorioretinitis or uveitis can be found with FIP, fungal disease, rickettsial disease, sepsis, or systemic neoplasia.

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DIAGNOSTICS

Complete Blood Count/Biochemistry/Urinalysis

- CBC may show inflammatory leukogram in patients with pyothorax, FIP, neoplasia, or lung lobe torsion.
- If pleural effusion is due to hypoalbuminemia, serum albumin is usually less than 1.5 g/dL; this is rare.
- Polyclonal hyperglobulinemia is usually found in cats with FIP.

Other Laboratory Tests

- If a coagulopathy is suspected (e.g., due to vitamin K antagonist rodenticide exposure), check clotting times (e.g., PT) before performing thoracocentesis.
- Pleural fluid analysis, including physical characteristics (i.e., color, clarity, odor, clots), pH, glucose, total protein, total nucleated cell count, and cytologic examination. See Table 73.1 for information on characterization of pleural fluid.
- Because mediastinal lymphosarcoma is often associated with FeLV infection, cats with mediastinal masses should be tested for FeLV.
- FIV/FeLV test should be done to look for underlying immunosuppression in cats with pyothorax.
- If cardiac disease is suspected, a heartworm test should be considered in dogs and thyroid levels should be evaluated in cats to rule out heartworm disease and hyperthyroidism.
- An NT pro-BNP test may be performed, and if elevated, is suggestive of cardiac disease (increased left atrial pressure).
- If pyothorax is suspected, aerobic and anaerobic bacterial culture and sensitivity should be done. Special stains (e.g., gram and acid-fast stains) of pleural fluid should also be considered.
- If chylothorax is suspected, cholesterol and triglyceride levels of the fluid and serum should be performed.

Imaging

Radiographic Findings

- Should be performed after thoracocentesis in patients with respiratory difficulty and evidence of pleural effusion on physical examination.
- Used to confirm pleural effusion.
    - Findings include separation of lung borders away from the thoracic wall and sternum by fluid density in the pleural space (Figs 73.2, 73.3), interlobar pleural fissure lines, silhouetting of fluid with the cardiac and diaphragmatic borders, blunting of the lung margins at the costophrenic angles (on the ventrodorsal view), and widening of the mediastinum (on the ventrodorsal view).

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TABLE 73.1. Characterization of pleural fluid.

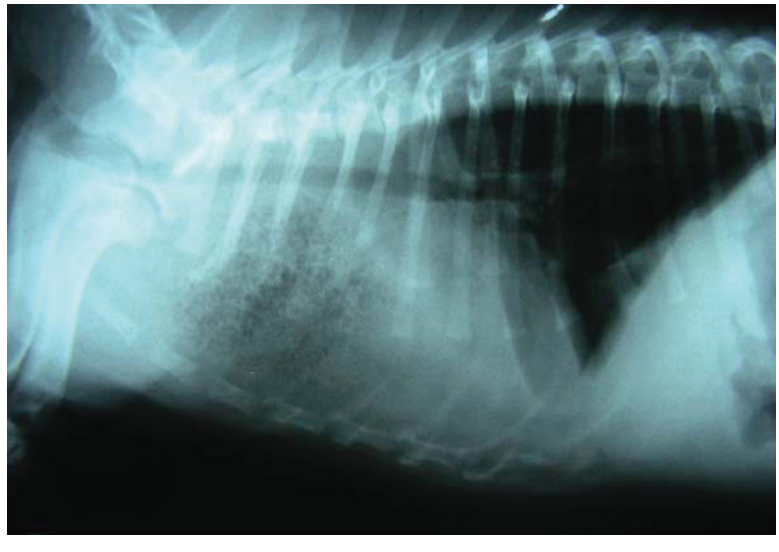
	Transudate	Modified transudate	Nonseptic exudate	Septic exudate	Chyle	Hemorrhage
Color	Colorless to pale yellow	Yellow or pink	Yellow or pink	Yellow to red-brown	Milky white	Red
Turbidity	Clear	Clear to cloudy	Clear to cloudy; fibrin	Cloudy to opaque; fibrin	Opaque	Opaque
Protein (g/dL)	<2.5	2.5–5.0	3.0–8.0	3.0–7.0	2.5–6.0	>3.0
Nucleated cells/μL	<1000	1000–7000 (LSA up to 100 000)	5000–20 000 (LSA up to 100 000)	5000–300 000	1000–20 000	Similar to peripheral blood
Cytology	Mostly mesothelial cells	Mostly macrophages and mesothelial cells; few nondegenerate PMNs; neoplastic cells in some cases	Mostly nondegenerate PMNs and macrophages; neoplastic cells in some cases	Mostly degenerate PMNs; also macrophages; bacteria	Small lymphocytes, PMNs, and macrophages	Mostly RBCs; macrophages and erythrophagocytosis
Disease associations	Hypoalbuminemia; early CHF	CHF; neoplasia; diaphragmatic hernia; pancreatitis	FIP; neoplasia; diaphragmatic hernia; lung lobe torsion	Pyothorax – penetrating chest wound, foreign body, ruptured esophagus, ruptured pulmonary abscess or tumor	Lymphangiectasia, CHF, cranial vena cava obstruction, neoplasia, fungal, heartworm disease, diaphragmatic hernia, lung lobe torsion, trauma	Trauma, coagulopathy, neoplasia, lung lobe torsion

CHF, congestive heart failure; FIP, feline infectious peritonitis; LSA, lymphoma; PMN, polymorphonuclear cell; RBC, red blood cell.
Source: Modified from Sherding RG. Diseases of the pleural cavity. In: Sherding RG, ed. The Cat: Diseases and Clinical Management, 2nd edn. New York: Churchill Livingstone, 1994; p. 1061.

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■ **Fig. 73.2.** Dorsoventral thoracic radiograph of pug with pleural effusion and abnormal soft tissue density in cranial thorax. Note the rounding and leafing of the lung lobes near the pleural effusion.



■ **Fig. 73.3.** Lateral thoracic radiograph of pug with pleural effusion and abnormal soft tissue density in cranial thorax. Note the rounding and leafing of the lung lobes near the pleural effusion.

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- In patients with fibrosing pleuritis due to chylothorax, pyothorax, or FIP, a rounding of the caudal lung lobe borders can be seen on the lateral view.
- Unilateral effusion is found most commonly with chylothorax, pyothorax, hemothorax, pulmonary neoplasia, diaphragmatic hernias, and lung lobe torsions.
- Postthoracocentesis radiographs should be taken to evaluate for cardiomegaly, intrapulmonary lesions, mediastinal masses, diaphragmatic hernias, lung lobe torsions, and evidence of trauma.

Echocardiographic/Ultrasonographic/CT Findings

- Ultrasonographic evaluation of thorax is recommended when cardiac disease, a diaphragmatic hernia, or a cranial mediastinal mass is suspected. An echocardiogram is the gold standard for diagnosis of cardiac disease.
- Computed tomography is also emerging as a diagnostic test for unexplained pleural effusion.
- T-FAST (screening ultrasound) is emerging as an extraordinarily useful point-of-care test for pleural effusion.

Diagnostic Procedures

- Thoracocentesis: allows the collection of a sample of pleural effusion fluid for analysis to help determine the underlying cause.
- Exploratory thoracotomy or thoracoscopy can be done to obtain biopsy samples of the lung, lymph nodes, or pleura.



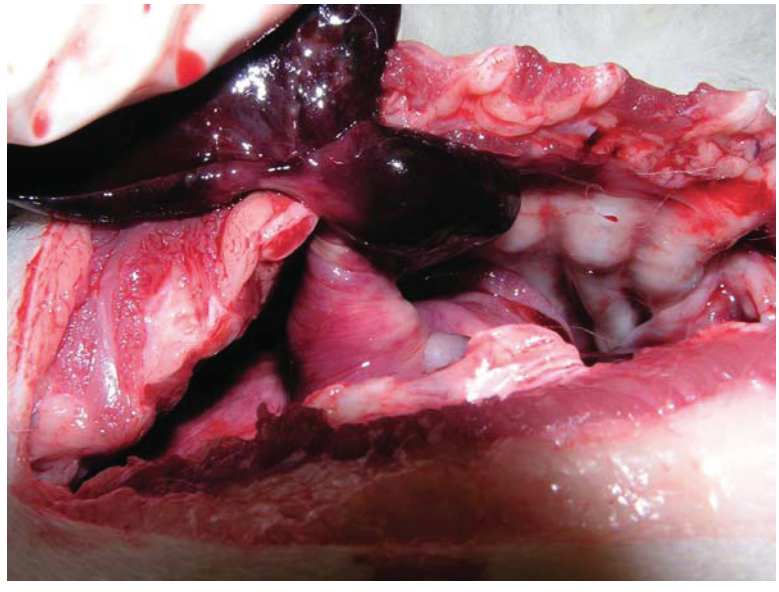
THERAPEUTICS

- Thoracocentesis should be done first in patients with respiratory distress.
- If patient is stable after thoracocentesis, outpatient management is possible in some cases (e.g., congestive heart failure).
- Most patients require hospitalization for diagnostics.
- For pets with pyothorax, surgical or intensive medical care is advised.
- Treatment for underlying disease is needed to prevent further fluid accumulation.
- Surgery is indicated for some neoplasias, diaphragmatic hernias, some cases of pyothorax, lung lobe torsion (Fig. 73.4), and cases of idiopathic chylothorax.
- For cases where fluid continues to accumulate despite treatment, repeated thoracocenteses or other palliative options such as placement of a subcutaneous vascular access port may be considered. Pleurodesis is not recommended in dogs and cats.

Drugs of Choice

- Treatment varies with underlying cause.
- Diuretics should only be used for patients with diseases that cause fluid retention and volume overload (e.g., CHF); otherwise, diuretics are largely ineffective for the treatment of pleural effusion not associated with CHF.

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■ **Fig. 73.4.** Necropsy photograph of lung lobe torsion that caused the pleural effusion. Lung lobe torsion has been reported in young pugs and is just one of the many causes of pleural effusion in dogs and cats.

COMMENTS

Patient Monitoring

- Ultrasonic evaluation is used to assess the efficacy of treatment in most cases; if ultrasound is not available, then radiographs may be used.

Possible Complications

- Death due to respiratory compromise.
- Reexpansion pulmonary edema can occur after removal of pleural effusion (rare).
- Iatrogenic pneumothorax during thoracocentesis; this is particularly likely in animals with recurrent effusion.

Expected Course and Prognosis

- Variable with underlying cause; guarded to poor in most cases.

Abbreviations

CBC: complete blood count
CHF: congestive heart failure
FeLV: feline leukemia virus

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FIP: feline infectious peritonitis
FIV: feline immunodeficiency virus
pH: acid–base status
PT: prothrombin time
T-FAST: thoracic focused assessment using sonography for trauma

Suggested Reading

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Author: Elizabeth Rozanski, DVM

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chapter 74

Pneumonia – Aspiration

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DEFINITION/OVERVIEW

- Aspiration pneumonia is caused by inhalation of liquid or particulate matter into the lower respiratory tract that results in inflammation/infection of the pulmonary parenchyma. Aspiration pneumonia is far more common in the dog than the cat.

ETIOLOGY/PATHOPHYSIOLOGY

- In the oronasal pharynx, the close proximity of the upper airway to the proximal aspect of the GI tract makes animals, especially dogs, susceptible to inhalation of regurgitated and vomited materials, and subsequent aspiration pneumonia.
- Aspiration pneumonia is commonly associated with vomiting, esophageal and laryngeal disorders, neurologic disease, and anesthesia.

Systems Affected

- Cardiovascular: tachycardia possible to compensate for hypoxia.
- Gastrointestinal: may have a history of GI symptoms such as vomiting and regurgitation.
- Hemic/lymphatic/immune: may or may not have leukocytosis, left shift.
- Musculoskeletal: may be weak secondary to hypoxia.
- Nervous: likely depressed or obtunded due to hypoxia.
- Respiratory: coughing, gagging, and tachypnea often present.
- Skin/exocrine: fever is possible; if hypoxia is severe enough, gray, pale, and cyanotic mucus membranes can be present.

SIGNALMENT/HISTORY

- A history of vomiting or regurgitation shortly followed by respiratory distress is typical of aspiration into the lower airways. Additional common history includes laryngeal or esophageal disorders and recent anesthesia. Older dogs and larger breeds are reported to

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most commonly develop aspiration pneumonia. Aspiration may also be a silent event, without a history of vomiting or regurgitation.

Risk Factors/Causes

- Animals with megaesophagus and laryngeal dysfunction are at significant risk for aspiration pneumonia. The aspiration event may not be observed in patients with laryngeal and esophageal abnormalities, but in these patients an increased index of suspicion is always present. Postoperative and heavily sedated patients often have an impaired gag reflex, making them susceptible to aspiration and subsequent pneumonia.
- In addition to anesthetized patients, those suffering from neuromuscular blockade or paralysis (e.g., botulism, tick paralysis, coonhound paralysis) may develop an impaired gag reflex and are also susceptible to the development of aspiration pneumonia.
- Animals that require mechanical ventilation also have an increased risk of aspiration because oropharyngeal flora may be transported to the lower airways during intubation and result in airway colonization.
- Nasogastric feeding tubes that have been misplaced or displaced can also predispose to aspiration pneumonia.

Historical Findings

- Commonly reported history includes recent vomiting or procedures requiring anesthesia. It is also important to question owners on subtle changes in their pet's attitude, voice, appetite and for presence of a cough, which may be indicators of laryngeal dysfunction as well as aspiration.



CLINICAL FEATURES

- Increased respiratory rate and fever are present in only approximately one-third of dogs that present with aspiration pneumonia, making them poor indicators of this disease.
- Abnormal lung sounds are commonly found during physical examination.
- Commonly described physical examination abnormalities include the following.

Auscultation

- Abnormal lungs sounds, most commonly crackles and loud lung sounds, have been reported to be the most consistent physical examination finding in dogs with any type of pneumonia. The lack of normal or abnormal sounds may be indicative of complete consolidation of that area of lung, which is common in aspiration.

Chest Percussion

- Percussion of the thorax may be helpful in identifying areas of lung consolidation. Percussion of consolidated areas of lung elicits a low-pitched or dull sound.

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Tracheal Palpation

- Tracheal palpation can be utilized to elicit a cough and confirm epithelial irritation. Pneumonia is most often associated with increased secretions and a moist or productive cough.

Oral/Pharyngeal Examination

- Information concerning laryngeal/pharyngeal anatomy and function is sought on examination. The following abnormalities may be noted on close inspection and can be associated with pneumonia: excess secretions, severe dental or tonsillar disease, masses, an abnormal gag reflex, or the presence of foreign material or gastric contents.



DIFFERENTIAL DIAGNOSIS

- Dogs: bacterial/fungal/viral pneumonia, near drowning, heartworm disease, pulmonary thromboembolism, electrocution, smoke inhalation, congestive heart failure, collapsing trachea, allergic airway disease, chronic bronchitis.
- Cats: congestive heart failure, allergic airway disease (asthma), pyothorax.



DIAGNOSTICS

Thoracic Radiographs

- Key in diagnosing aspiration pneumonia.
- Three views of the thorax (dorsoventral or ventrodorsal, right and left lateral) are recommended in order to examine all lung fields.
- Aspiration pneumonia is classically located in dependent lung lobes, right middle, left cranial, and right cranial.
- Common radiographic findings include air bronchograms (Figs 74.1, 74.2) and alveolar infiltrates, from patchy to lobar consolidation.

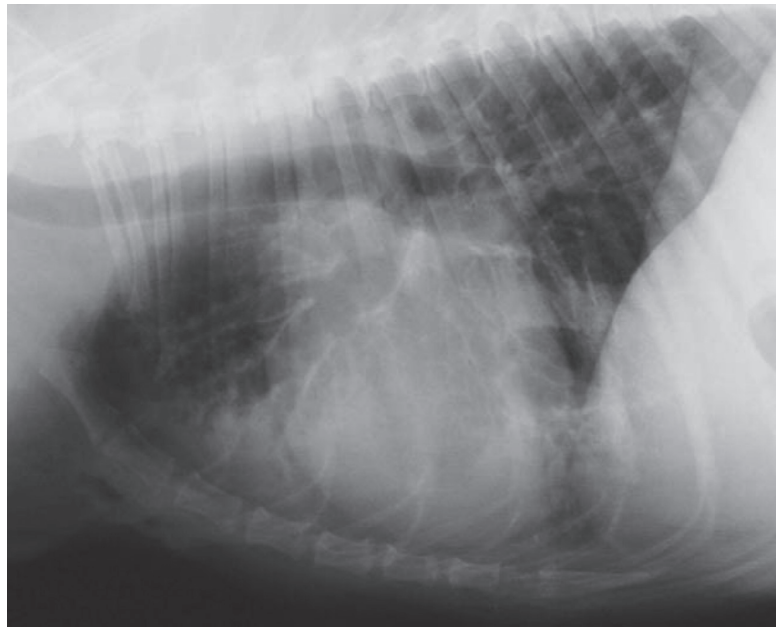
Additional Baseline Testing

- CBC, chemistry, and urinalysis, urine culture, fecal examination.
- Ancillary testing to rule out diseases that may increase susceptibility to aspiration such as ACH receptor antibody titers to diagnose myasthenia gravis.

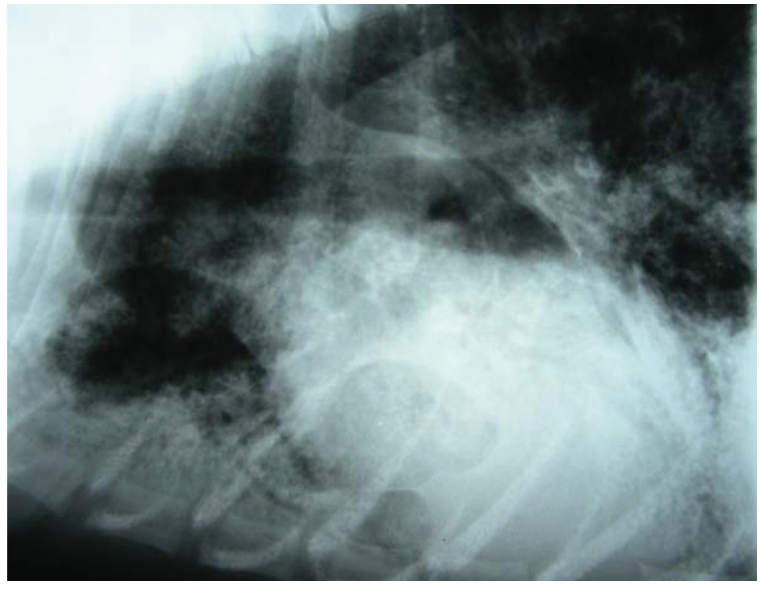
Advanced Diagnostics

- Arterial blood gas can be utilized to objectively (A-a gradient) monitor disease progression/resolution, and response to therapeutics.
- Pulse oximetry can also be used to monitor hypoxia and need for/response to oxygen supplementation in a noninvasive manner.

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■ **Fig. 74.1.** Lateral thoracic radiograph showing air bronchograms and bronchiectasis in right cranial lung lobe of a dog.



■ **Fig. 74.2.** Severe aspiration pneumonia in the right middle and cranial lung lobes of a Great Dane that ingested 1 gallon of used peanut oil from a turkey fryer after Thanksgiving dinner was served.

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- BAL is considered the best method for obtaining samples from the deep lung but should only be performed immediately after aspiration to alleviate airway obstruction by foreign material or to obtain cultures when disease does not respond to standard treatment.
- Transtracheal wash is not recommended unless obtaining cultures, BAL preferred; no visualization of lower airways.
- Culture and sensitivity are recommended on samples obtained from airways; quantitative cultures are best to allow differentiation between normal flora and infection. Cultures consistent with infection contain $>1.7 \times 10^3$ cfu.
- Gram-stained samples are helpful if culture is negative.



THERAPEUTICS

Antibiotics

- Pending culture and sensitivity results, bactericidal antibiotics with gram-negative spectrum should be utilized when aspiration is suspected. Oral antibiotics may need to be continued for extended periods of time (4–6 weeks after discharge). Antibiotic therapy should be continued 1–2 weeks past resolution of radiographic and clinical signs.

Fluid Therapy

- Tracheobronchial secretions are approximately 95% water; systemic dehydration results in increased secretion viscosity and retention in the lower airways. Decreasing the viscosity of secretions will assist in their clearance and the resolution of pneumonia.

Oxygen

- Animals with pneumonia can have lower PaO_2 <60 mmHg, lower SaO_2 <90–92%, and significantly greater A-a gradients and should be supplemented with oxygen when hypoxia is documented.

Nebulization

- Aerosol therapy has been utilized in pneumonic dogs in an attempt to decrease the viscosity of infectious secretions and improve mucociliary clearance.
- Sterile saline is currently the only fluid recommended for use in patients being nebulized.
- Ultrasonic nebulizers producing particles that range between 0.5 and 3.0 µm in size (mass median diameter) are necessary to ensure that fluid particles reach the lower airways.
- Nebulization is performed by placing the patient in a sealed cage for 30–45 minutes 3–4 times daily.
- Potential complications associated with cage nebulization include overhydration and overheating in very small patients as well as bacterial contamination.
- Nebulization of antibiotics, mucolytics, or other drugs is not routinely recommended, with the exception of *Bordetella* (gentamicin 6–7 mg/kg).

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Diet

- Malnutrition has been shown to alter pulmonary defense mechanisms, resulting in impaired clearance of organisms and foreign material. Once GI symptoms have been addressed, enteral nutrition should be attempted. In severe cases, parenteral nutrition may be necessary.

Activity

- Activity within the limits of the animal's pulmonary function should be encouraged, as it helps mobilize pulmonary secretions.

Surgical Considerations

- Lung lobectomy has been reported as a treatment for pneumonia, of various etiologies, that is nonresponsive to medical therapy. Lung lobectomy should be considered in chronic cases of aspiration pneumonia when a foreign body is suspected, or pneumonia is chronic or recurring.

COMMENTS

- Radiographic changes can lag behind clinical changes up to 48 hours; the most reliable, objective way to gauge clinical changes is with arterial blood gases. Hypoxic animals not responding to oxygen therapy alone could require ventilatory support.

Client Education

- Monitor for return of respiratory difficulty, anorexia, vomiting, and fever.

Patient Monitoring

- The A-a gradient is the most objective way to monitor disease progression. Unless there is an acute change in the animal's condition, it is calculated once daily.
- Radiographs are usually repeated at 2-week intervals once the animal is discharged.

Prevention/Avoidance

- If associated with anesthesia, make sure endotracheal tube cuffs are functioning properly.

Possible Complications

- Pneumonia may not resolve with medical management, especially if a foreign body is present, and surgical options may need to be considered.

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Expected Course and Prognosis

- Aspiration pneumonia without a permanent underlying cause such as megaesophagus is likely to resolve when treated appropriately; clinical improvement is often seen within 24–72 hours. Animals with diseases causing esophageal and laryngeal dysfunction are always susceptible to aspiration and many do not resolve. Greater than 75% of patients suffering from aspiration pneumonia are reported to survive.

Abbreviations

A-a: alveolar-arterial
ACH: acetylcholine
BAL: bronchoalveolar lavage
CBC: complete blood count
GI: gastrointestinal
PaO_2: partial pressure of oxygen in blood
SaO_2: oxygen saturation in blood

See Also

Pneumonia – Bacterial

Suggested Reading

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Author: Adam J. Reiss, DVM
Acknowledgment to the author of the original chapter in *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Emergency and Critical Care, First Edition:* Eleanor C. Hawkins.

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chapter 75

Pneumonia – Bacterial





DEFINITION/OVERVIEW

- Defined as any inflammation of the lung parenchyma that results in the filling of alveolar air spaces with exudate. Bacterial colonization of the lung is the most common cause of pneumonia in small animals. Bacterial pneumonia occurs more commonly in dogs than in cats.



ETIOLOGY/PATHOPHYSIOLOGY

- Animals that develop bacterial pneumonia often have impaired pulmonary defenses. Inhibition of pulmonary immunity is often caused by underlying metabolic diseases (such as diabetes mellitus and hyperadrenocorticism), lower airway disease (chronic bronchitis, ciliary dyskinesis), laryngeal and esophageal dysfunction, as well as those from exposure to environmental pathogens (*Bordetella*). Additionally, pathogenic bacteria may have specific physical and biochemical properties that allow them to thwart lower airway defenses and cause disease. The source of invading bacteria is often the oronasal and pharyngeal regions, upper GI tract, and normal lower airway flora. Hematogenous spread has also been proposed as a cause of bacterial pneumonia and is reported to occur more commonly in cats.

Systems Affected

- Cardiovascular: tachycardia possible to compensate for hypoxia.
- Hemic/lymphatic/immune: may or may not have leukocytosis with a left shift.
- Musculoskeletal: may be weak secondary to hypoxia.
- Nervous: likely depressed or obtunded due to hypoxia.
- Respiratory: coughing, gagging, and tachypnea with or without expectoration of debris or mucus often present.
- Skin/exocrine: fever is possible; if hypoxia is severe enough, gray, pale, and cyanotic mucous membranes can be present.

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SIGNALMENT/HISTORY

- No sex or breed predispositions.

Risk Factors/Causes

- Metabolic disorders: diabetes mellitus or hyperadrenocorticism.
- Acquired diseases: myasthenia gravis, polyradiculoneuritis, or tick paralysis.
- Immunosuppression: secondary to chemotherapy or other infections (i.e., viral, fungal).
- Laryngeal and esophageal disorders: laryngeal paralysis or megaesophagus.
- Other sites of infection: dermatologic, phlebitis, oral, urinary tract, or reproductive tract.
- Exposure to other pets: shelters, boarding grooming, shows, or dog parks.
- Vomiting.

Historical Findings

- Coughing, nasal discharge, tachypnea, orthopnea, anorexia, dehydration, or weight loss.



CLINICAL FEATURES

- No single clinical parameter is pathognomonic in the diagnosis of bacterial pneumonia.

Dogs

- Fever reported in <50% of dogs with bacterial pneumonia; cough, tachypnea, nasal discharge.
- Auscultation of all lung fields is an important part of the diagnosis of pneumonia. Common abnormalities detected during auscultation include crackles and loud lung sounds. Decreased sounds may be indicative of complete consolidation of that area of lung, which is common when aspiration is an underlying cause of bacterial pneumonia.
- Tracheal palpation can be utilized to elicit a cough and confirm tracheal irritation.
- Pneumonia is most often associated with increased secretions and a moist or productive cough.
- Oropharyngeal examination can also provide information relevant to bacterial pneumonia. Laryngeal and pharyngeal anatomy and function can be assessed, as well as the condition of the dentition and gums. Additional oropharyngeal abnormalities that may be noted and can be associated with pneumonia include excess secretions, tonsillar disease, masses, an abnormal gag reflex, or the presence of foreign material or gastric contents.

Cats

- May have clinical signs similar to dogs or none at all.
- Observe for oral erosions and ocular signs consistent with URI, which may be an underlying or associated disease.

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DIFFERENTIAL DIAGNOSIS

- Dogs: bacterial/fungal/viral pneumonia, near drowning, heartworm disease, pulmonary thromboembolism, electrocution, smoke inhalation, congestive heart failure, collapsing trachea, allergic airway disease, or chronic bronchitis.
- Cats: congestive heart failure, allergic airway disease (asthma), or pyothorax.

DIAGNOSTICS

Thoracic Radiographs

- Key in diagnosing pneumonia.
- Ventrodorsal or dorsoventral, and both left and right lateral views are recommended to examine all lung fields.
- Common radiographic findings include air bronchograms and alveolar infiltrates, from patchy to lobar consolidation (Fig. 75.1). Pleural effusion is uncommon but may be present in severe cases.

Additional Baseline Testing

- CBC, chemistry, urinalysis, urine culture, and fecal examination.
- Ancillary testing to rule out diseases that may increase susceptibility to aspiration such as ACH receptor antibody titers to diagnose myasthenia gravis.



Fig. 75.1. Alveolar lung pattern in right caudal lung field shown in this ventrodorsal thoracic radiograph from a dog with pneumonia.

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Advanced Diagnostics

- Arterial blood gas can be utilized to objectively (A-a gradient) monitor disease progression/resolution and response to therapeutics.
- Pulse oximetry can also be used to monitor hypoxia and need for/response to oxygen supplementation in a noninvasive manner.
- BAL is considered the best method for obtaining culture and cytology samples from small airways, alveoli, and pulmonary interstitium.
- Transtracheal wash does not provide visualization of the airways; samples collected are from larger airways.
- Culture and sensitivity are recommended on samples obtained from airways; quantitative cultures are best because they allow differentiation between normal flora and infection. Cultures consistent with infection contain >1.7×10^3 cfu.
- Gram-stained samples are helpful if culture is negative.



THERAPEUTICS

- Treatment objectives are to improve oxygenation and eliminate the infection. Concurrent treatment of underlying and secondary diseases may be necessary.

Antibiotics

- Pending culture and sensitivity results, broad-spectrum bactericidal antibiotics should be utilized. Severe cases should be started on intravenous antibiotics to provide good penetration of the pulmonary parenchyma. Maximum doses and dosing intervals should be utilized to ensure effectiveness. Mild cases can be started on oral antibiotics. Drug classes commonly effective in treating bacterial pneumonia include cephalosporins, potentiated penicillins, and fluoroquinolones. Oral antibiotics may need to be continued for extended periods of time (4–6 weeks after discharge). Antibiotic therapy should be continued 1–3 weeks past resolution of radiographic and clinical signs.

Fluid Therapy

- Tracheobronchial secretions are approximately 95% water; systemic dehydration results in increased secretion viscosity, retention in the lower airways, and often ventilation perfusion abnormalities. Fluids should be administered to correct dehydration and maintain hydration status until the animal can sustain itself.

Oxygen

- Animals with pneumonia can have lower PaO_2 <60 mmHg, lower SaO_2 <90–92%, and significantly greater A-a gradients and should be supplemented with oxygen when hypoxia is documented.

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Nebulization

- Aerosol therapy has been utilized in pneumonic dogs in an attempt to decrease the viscosity of infectious secretions and improve mucociliary clearance.
- Sterile saline is currently the only fluid recommended for use in patients being nebulized.
- Ultrasonic nebulizers that produce particles that range between 0.5 and 3.0 µm in size (mass median diameter) are necessary to ensure that fluid particles reach the lower airways.
- Nebulization is performed by placing the patient in a sealed cage for 30–45 minutes up to 3–4 times daily.
- Potential complications associated with cage nebulization include overhydration and overheating in very small patients as well as bacterial contamination.
- Nebulization of antibiotics, mucolytics, or other drugs is not routinely recommended with the exception of *Bordetella*. Cases of *Bordetella* that are resistant to systemic antibiotics may respond to the local effects of nebulized antibiotics (gentamicin 7 mg/kg diluted with sterile saline).

Physiotherapy

- Coupage is best described as physical therapy consisting of a firm clap (hands are cupped) on the lateral aspects of the thoracic cage. This percussion of the thoracic cage serves to loosen and help clear secretions by inducing coughing. Coupage should be performed for 5–10 minutes 3–4 times daily.

Diet

- Malnutrition has been shown to alter pulmonary defense mechanisms and result in impaired clearance of organisms and foreign material. Once GI symptoms have been addressed, enteral nutrition should be attempted.
- In severe cases, parenteral nutrition may be required.

Activity

- Activity within the limits of the animal's pulmonary function should be encouraged because it helps mobilize pulmonary secretions.

Surgical Considerations

- Lung lobectomy has been reported as a treatment for pneumonia, of various etiologies, that is nonresponsive to medical therapy. Lung lobectomy should be considered in chronic cases of lobar pneumonia when an aspirated foreign body is suspected as the underlying cause.

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COMMENTS

- Radiographic changes can lag behind clinical changes by up to 48 hours; the most reliable, objective way to gauge clinical changes is to obtain arterial blood gases. Hypoxic animals not responding to oxygen therapy alone could require ventilatory support.

Client Education

- Monitor for return of respiratory difficulty, anorexia, vomiting, and fever.

Patient Monitoring

A-a Gradient

- The A-a gradient is the most objective way to monitor disease progression. Unless there is an acute change in the animal's condition, it is recommended that the A-a gradient be calculated once daily.
  - To calculate the A-a gradient, obtain an arterial blood sample. The values of significance on the arterial blood sample include the PaO_2, barometric pressure, and the $PaCO_2$.
  - "Big A" (A) is the amount of oxygen available in the alveolus and is dependent on the FiO_2 (on room air, the FiO_2 is 21% or 0.21), the barometric pressure (think of this as the force that can help push the air into the lungs), and the diffusion capacity of air itself. Because CO_2 diffuses more readily than oxygen, there is a calculation that takes into consideration the CO_2 component.
  - "Little a" or (a) is the amount of oxygen actually in the bloodstream.
  - To calculate the A-a gradient, first calculate A.

$$A = FiO_2\,(\text{barometric pressure} - \text{water vapor pressure}) - PaCO_2 / 0.8$$

    Water vapor pressure is a constant and is equal to 47.
  - Next, subtract PaO_2 from A, and the value is equal to the A-a gradient.
- The A-a gradient allows you to determine the degree of diffusion impairment between the amount of oxygen available to the lung and that which actually enters the bloodstream.
- Normal values are <10, 10–15 suggesting moderate diffusion impairment, and >20 suggesting acute respiratory distress syndrome.

PaO_2/FiO_2 Ratio

- In an animal receiving supplemental oxygen, the A-a gradient will be negative. Instead, one must calculate the PaO_2/FiO_2 ratio.
- For facemasks, oxygen supplementation catheters, and hood or cage oxygen, the FiO_2 can be estimated at 40% or 0.40. Ideally, measure the FiO_2 with an oxygen meter.
- Normal calculated PaO_2/FiO_2 values are >400. Values between 200 and 300 are suggestive of acute lung injury. Values <200 are suggestive of acute respiratory distress syndrome.

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Radiographs

- Radiographs are usually as needed while the patient is in hospital and repeated at 2-week intervals once the animal is discharged.

Prevention/Avoidance

- Pets should be vaccinated for any infectious diseases that can predispose to bacterial pneumonia. Underlying diseases and conditions such as dental abscesses, diabetes, and hyperadrenocorticism should be addressed.

Possible Complications

- Severe pneumonia can result in pulmonary abscesses, pneumothorax and chronic airway issues; surgery and long-term/ongoing therapy may be required.

Expected Course and Prognosis

- A good prognosis can be given to those patients that show improvement of respiratory function within 24–72 hours of beginning appropriate antibiotic therapy.
- Poor outcomes, on the other hand, can be predicted in those animals that have antibiotic-resistant organisms cultured, underlying diseases that cannot be resolved (e.g., laryngeal paralysis, megaesophagus), the presence of concurrent disease (e.g., fungal and viral infections, neoplasia), and hypercarbia on presentation.

Abbreviations

A-a: alveolar-arterial
ACH: acetylcholine
BAL: bronchoalveolar lavage
CBC: complete blood count
FiO_2: fraction of inspired oxygen
GI: gastrointestinal
$PaCO_2$: partial pressure of carbon dioxide in blood
PaO_2: partial pressure of oxygen in blood
SaO_2: oxygen saturation in blood
URI: upper respiratory infection

See Also

Pneumonia – Aspiration

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Author: Adam J. Reiss, DVM

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chapter 76

Pneumothorax

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DEFINITION/OVERVIEW

- Free air that accumulates in the pleural cavity in between the lungs and the pleura.
- Can be spontaneous (rare) or more commonly caused by trauma.
- Spontaneous pneumothorax causes air to accumulate in the pleural space outside the lungs without trauma.
- Traumatic pneumothorax is air that accumulates in the pleural spaces as a result of trauma to the trachea, mainstem bronchi, lungs, or thoracic wall.
- Pneumothorax can also be categorized as being closed or open.
    - Closed pneumothorax occurs when there is no break in the integrity of the thoracic wall.
    - Open pneumothorax is associated with a break in the integrity of the thoracic wall, such that atmospheric pressure comes in contact with intrapleural pressure and causes the lungs to collapse.
- A tension pneumothorax results when damage to the bronchi or lungs causes a flap of tissue that acts as a valve. When the animal inhales, air leaks from the flap into the pleural space. Upon exhalation, the flap of tissue closes and prevents the air from leaving the pleural space. Pleural pressure continues to accumulate until there is severe pressure on the cranial vena cava that prevents blood from returning to the right-sided heart, thus impairing cardiac preload. As a result, cardiac output and blood pressure are adversely compromised. The animal with a tension pneumothorax can rapidly deteriorate and die without immediate aggressive intervention.

ETIOLOGY/PATHOPHYSIOLOGY

- The pleural cavity is lined with a thin layer of fluid and tissue called the parietal and visceral pleura. Normally, the visceral and parietal pleura and the lungs are contiguous, and freely move the lungs against the thoracic wall and diaphragm during normal respiration.
- Pressure in the pleural space is normally subatmospheric. An animal actively draws in a breath to expand the lungs, and then relaxation of the thoracic wall and diaphragm allows

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air within the lungs to leave the alveoli and return to the atmosphere via the trachea and upper airways. When air escapes from the lungs into the pleural space, it accumulates between the parietal and visceral pleura and causes the lungs to collapse.

- Collapse of any portion of the lungs causes that portion to be perfused but not participate in alveolar gas exchange or ventilation. This results in a ventilation-perfusion mismatch and hypoxemia.
- A pneumothorax can be bilateral, if the mediastinum is open, or unilateral, if the mediastinum on both sides of the thorax does not communicate.

Systems Affected

- Cardiovascular.
- Respiratory.

Incidence/Prevalence

- Unknown.
- It has been documented in more than 40% of cases of thoracic trauma, and 10–20% of cases of trauma caused by contact with automobiles.



SIGNALMENT/HISTORY

Species

- Dogs and cats.

Breed Predilection

- Spontaneous pneumothorax is documented more commonly in large, deep-chested breeds of dog.

Historical Findings

- In cases of traumatic pneumothorax, recent trauma may be documented, such as fall from height, motor vehicle accident, encounter with a large animal, or recent anesthesia with endotracheal intubation. Iatrogenic pneumothorax also can be caused by jugular venipuncture (although rare), thoracocentesis, acupuncture therapy or intrapleural misplacement of nasogastric feeding tubes.
- In spontaneous pneumothorax, historically there may be an acute onset of cough, cyanosis or pale mucous membranes, respiratory difficulty, exercise intolerance, lethargy, inappetence, or orthopnea, which may or may not be associated with previous history of respiratory disease.

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Physical Examination

- Rapid, shallow, restrictive respiratory pattern with muffled lung sounds, primarily dorsally but may be muffled in any area of the thorax. Tachypnea, orthopnea, extended neck, cyanosis, and billowing of the skin/cheeks in dogs, or obvious movement of the copulas, in front of the shoulder blades at the thoracic inlet in cats.
- When pneumothorax has been caused by a traumatic event, other signs referable to trauma may be present, including obvious hemorrhage or abrasions, rib fractures or other injuries to the thoracic wall, paradoxical chest wall motion in cases of flail chest, abrasions, frayed toenails, bite wounds, tachycardia, hypotension.
- In cases of open pneumothorax, bite wounds or penetrating injuries to the thoracic wall may be visible (Fig. 76.1). If recent anesthesia and endotracheal intubation caused the pneumothorax, subcutaneous emphysema may be present.

Risk Factors/Causes

- Blunt or penetrating trauma to the thorax or upper airways, penetrating cervical injuries, thoracocentesis, perforation of the esophagus from foreign bodies, migrating foreign bodies (grass awns, porcupine quills), neoplasia, pulmonary abscesses, parasitic infections (*Paragonimus*), cysts (congenital), pulmonary bullae, emphysema.
- Trauma, particularly in animals allowed to roam near open roads or near large animals; recent thoracocentesis, respiratory pathology, grass awns, encounters with porcupines; iatrogenic with overinflation of endotracheal tube cuff.



Fig. 76.1. Open pneumothorax caused by severe thoracic bite wounds. Notice the lung visible outside the thoracic cavity.

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DIFFERENTIAL DIAGNOSIS

- Differential diagnoses for pneumothorax include other forms of pleural space disease, such as pleuritis, pleural effusion, pulmonary contusions, diaphragmatic hernia, neoplasia, pneumonia, and pleural hemorrhage.

Differentiating Causes

- Pneumothorax can be differentiated from other causes of pleural space disease and restrictive respiratory pattern by lateral (Fig. 76.2) and dorsoventral (Fig. 76.3) thoracic radiographs.

DIAGNOSTICS

Complete Blood Count/Biochemistry/Urinalysis

- CBC, biochemistry, and urinalysis are usually unrewarding unless there is an infectious etiology that is associated with pulmonary pathology and sometimes a neutrophilic leukocytosis.

Other Laboratory Tests

- Arterial blood gases show hypoxemia (decreased PaO_2). Hypercapnia or hypocapnia may be present.
- Hypoxemia and SpO_2 <90% with pulse oximetry.

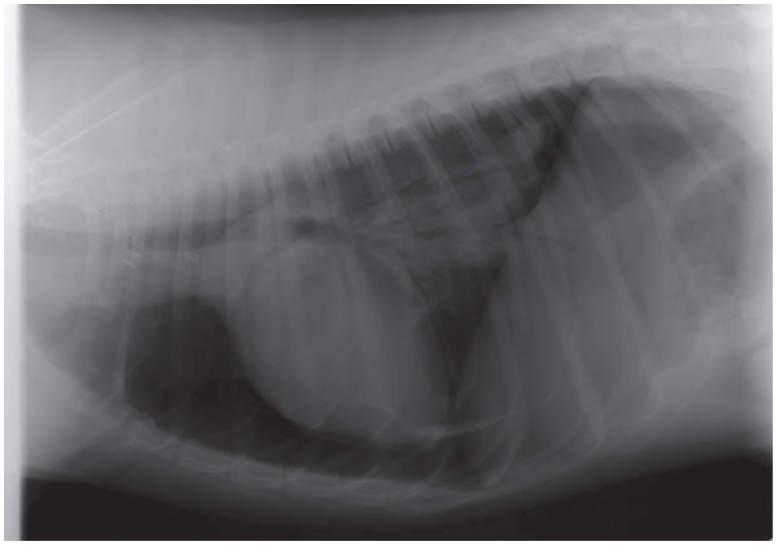
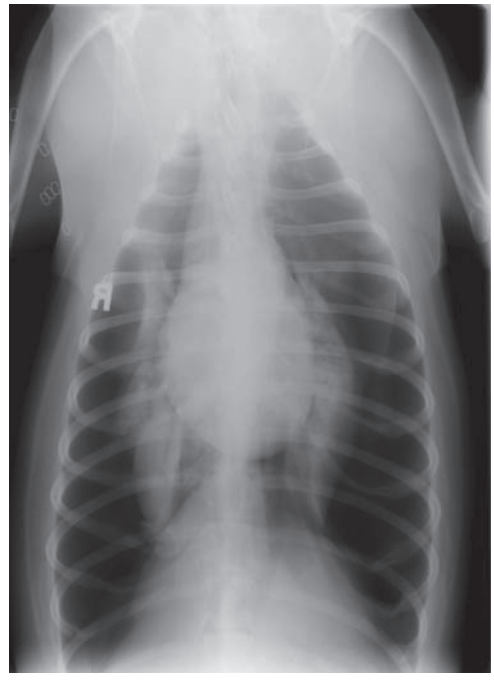


Fig. 76.2. Lateral thoracic radiograph of an animal with pneumothorax. Notice that the cardiac silhouette is elevated away from the sternum, and the lungs appear grayer in appearance, due to collapse and lack of air within the alveoli.

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■ **Fig. 76.3.** Dorsoventral thoracic radiograph of same dog with pneumothorax caused by migration of a porcupine quill.

Diagnostic Procedures

- Thoracic radiographs should be delayed until the patient is more stable, and attempts have been made to treat the pneumothorax prior to making a definitive diagnosis with radiography. Remember that thoracocentesis can be both diagnostic aand therapeutic in cases of pneumothorax.
- Thoracic radiographs may reveal free air within the pleural space, as evidenced by retraction of the lungs away from the thoracic wall and elevation of the cardiac silhouette from the sternum on a lateral view. The pulmonary vasculature will not be visible at the level of the thoracic wall. In cases of pneumomediastinum, the cranial vena cava, esophagus, and aorta will be visible. Rib fractures and foreign bodies, such as bullets, may be visible in cases of traumatic pneumothorax. If such findings are not present, often thoracic radiographs should be repeated after therapeutic thoracocentesis, to evaluate the lung for parenchymal disease such as bullae.
- CT scanning may be sensitive for identifying both pneumothorax and parenchymal pathology. A disadvantage of CT is the frequent need for heavy sedation or anesthesia in veterinary patients, as well as positive pressure ventilation to reinflate areas of collapse in order for bullae and blebs to be visible.

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THERAPEUTICS

- Treatment of pneumothorax largely depends on the degree of hypoxia and respiratory difficulty, severity of lung collapse, response to therapy, and whether the pneumothorax recurs.
- Initially, supplemental oxygen should be administered as rapidly as possible and with the least stressful method available.

Thoracocentesis

- Therapeutic thoracocentesis should be performed. To perform thoracocentesis, clip a square in the middle of the lateral thoracic wall, envisioning the ribcage as a box. Clip a box in the middle of the box, then aseptically scrub the area. Next, connect a hypodermic needle (usually 22 gauge, ¾ to 1½-inch needle, depending on the size and degree of obesity of the patient) to a length of intravenous extension tubing, to a three-way stopcock, to a 35–60 mL syringe. With the bevel of the needle facing down, insert the needle perpendicular to the thorax, in the middle of the box, in between rib spaces (Fig. 76.4). Once the needle has entered the pleural space, direct the needle so that it sits parallel with the thoracic wall. The bevel of the needle is now facing the inside of the pleural space and ready to aspirate air. The needle can be swept around like the hands of a clock, always parallel with the thoracic wall, to avoid iatrogenic puncture of the lungs. In smaller patients, such as neonates, a butterfly catheter and smaller needle can be used.
- As a rule of thumb, always perform bilateral thoracocentesis, in case the mediastinum does not communicate. If you cannot obtain negative pressure and fully evacuate the air from the pleural space, or if the air reaccumulates more than once, a thoracostomy tube should be placed.



Fig. 76.4. Inserting a 22 gauge hypodermic needle into the pleural space for thoracocentesis.

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Thoracostomy Tube Placement

- If a thoracostomy tube needs to be placed, clip the entire lateral thorax from the level of the vertebrae to the sternum, and from the 1st through 13th ribs. Aseptically scrub the clipped area and then drape the area with sterile field towels. Ideally, the animal should be anesthetized and intubated, so that you can maintain an airway and breathe for the patient, if necessary. Have an assistant pull the skin on the lateral thoracic wall cranially and ventrally, toward the elbow. This will facilitate making a tunnel through which the tube is placed. Trocarized thoracic drainage catheters are available from a variety of manufacturers. Any thoracic drainage tube should be, at minimum, the size of a mainstem bronchus, and not smaller, such that if a mainstem bronchus is the location of air leakage, suctioning the thorax through the tube can keep up with the air that is accumulating in the pleural space. Make a nick incision at the dorsal aspect of the 12th rib, then tunnel the tube under the skin, so that the tube can enter the 8th to 10th intercostal space. The trocarized thoracic catheter will need to be pulled so that it is perpendicular with the thoracic wall (Fig. 76.5). Grasp the base of the tube at the point of entry into the thorax and push it into the thorax, in between rib spaces. Take care to avoid pushing the tube and trocar in too far, to avoid iatrogenic puncture of lung, heart, or large vessels. An alternative to trocarized thoracic drain placement is to place a red rubber catheter. To place a red rubber catheter, make a small nick incision in the skin, then gently dissect through the subcutaneous tissues and intercostal muscles with the tips of a hemostat or Metzenbaum scissors. Once the pleural space is visible, grasp the tip of the red rubber catheter and push it into the thorax in between the 8th to 10th intercostal spaces. Push the catheter cranially



Fig. 76.5. Thoracostomy tube tunneled under the skin on the lateral thoracic wall, and being brought perpendicular to the thoracic wall before grasping it at its base and inserting it into the thorax.

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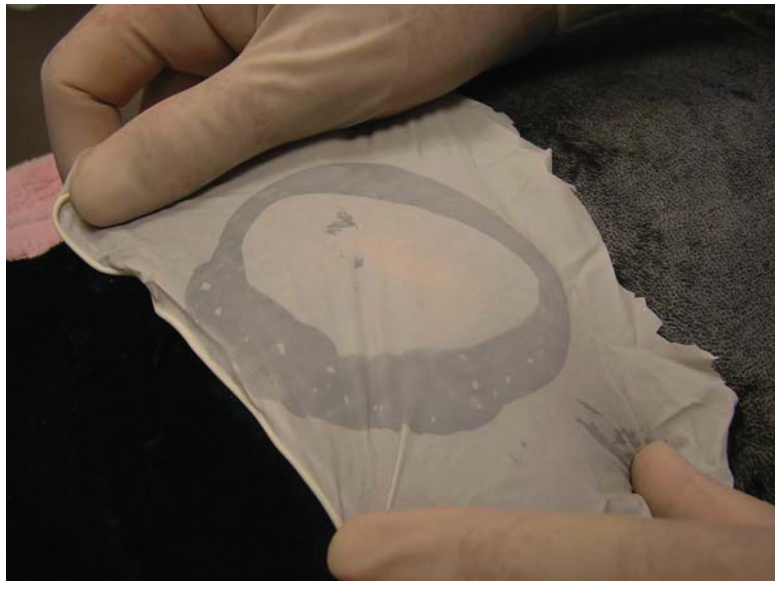
to the level of the 5th intercostal space. To make the tube more rigid during this process, insert a rigid polypropylene urinary catheter through the red rubber catheter. The polypropylene catheter can be removed, and then the tip of the tube connected to a Christmas tree adapter, length of intravenous extension tubing, three-way stopcock, and a syringe or continuous suction apparatus. The tube can then be secured with a combination of horizontal mattress and finger-trap sutures. The tube should be covered with a sterile wrap and checked daily for signs of erythema and infection.

- Open sucking chest wounds: if an animal has an open sucking chest wound, clip the wound and place sterile lubricant or antibacterial gel around it (Fig. 76.6). Next, place a sterile surgical glove over the wound (Fig. 76.7). The combination creates a seal from the atmosphere and the pleural space. The pleural space can now be evacuated using a combination of thoracocentesis and thoracostomy tube placement.
- Heimlich valves are one-way valves that allow air to escape from the thoracic cavity during inhalation. Heimlich valves can be useful in patients greater than 20 kg, who have enough strength to open the valve. Heimlich valves are not useful in smaller patients. Also, the apparatus can often become clogged with fibrin and not work properly. For this reason, their use is not recommended.
- Analgesia can be provided as systemic analgesia with opioids, as well as thoracic analgesia using lidocaine (0.25 mg/kg cat, 1 mg/kg dog), and bupivacaine (0.25 mg/kg cat, 1 mg/kg dog) through the chest tube every 8 hours.
- Tension pneumothorax may require creating a rapid hole in the thoracic wall and allowing gas to escape. The defect can be closed once a thoracic tube has been placed and the tension pneumothorax is under control.



Fig. 76.6. Sterile lubricant around an open sucking chest wound.

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■ **Fig. 76.7.** Placement of a sterile glove over the thoracic wound and sterile lubricant, creating a seal between the atmosphere and pleural space.

- Traumatic pneumothorax rarely requires surgical intervention, unless there is penetrating trauma to the thorax that has caused direct injury to the pulmonary parenchyma or bronchus/bronchi.
- Spontaneous pneumothorax often requires surgical removal of the diseased lung. In cases of bullae or blebs, however, rarely are the lesions isolated to one area and recurrence is common. For this reason, surgical intervention should not be performed without a preoperative CT scan to determine the extent of pulmonary parenchymal involvement.
- Autologous blood patch (a.k.a. autologous blood pleurodesis) can be used in cases of spontaneous pneumothorax in which surgical treatment is not an option due to disease severity or client finances. To perform this procedure, 5–10 mL/kg of whole blood is obtained via jugular venipuncture in 20–50 mL increments (no additives), and then infused into the pleural space using a thoracostomy tube (if in place) or an over-the-needle catheter.

Activity

- Restrict activity to short walks, only on a leash, for as long as the chest tube is required, and at minimum, at least 1 week after tube removal, to allow healing of pulmonary lesions.

Surgical Considerations

- When performing anesthesia on any patient with traumatic pneumothorax or pulmonary contusions, be careful to avoid overexpansion of the lungs to more than 20 cmH_2O, to avoid barotrauma and iatrogenic pneumothorax. Carefully inflate the cuff on endotracheal

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tubes, particularly in feline patients. When an animal needs to be rotated while under anesthesia, take care to disconnect the endotracheal tube from the anesthetic circuit when turning the patient, to avoid twisting and placing torque on the endotracheal tube and trachea, and causing an iatrogenic tear.
- Thoracoscopy may allow visualization of blebs and bullae in cases of spontaneous pneumothorax.
- Bronchoscopy rarely identifies the location of the tracheal tear because it is often dorsal under the trachealis membrane.
- Lung lobectomy may be required to remove lesions that are leaking air.

Nursing Care

- Oxygen therapy 50–150 mL/kg per minute of humidified oxygen.
- Analgesia.
- Maintenance of the thoracic drainage tube to ensure that all connections are tightly sealed and not allowing air to enter the thorax. Check the tube manually several times a day to ensure that it is maintaining negative pressure if it is attached to a continuous suction device. Check the entrance site at least once daily for signs of infection.
- Keep an Elizabethan collar on at all times to prevent the patient from licking or removing the tube.

COMMENTS

Patient Monitoring

- Careful monitoring of respiratory rate and effort, thoracic auscultation, and arterial blood gases or pulse oximetry to evaluate the patient's response to therapy.
- Rapid, shallow, restrictive respiratory pattern should signal the recurrence of pneumothorax.
- Thoracic radiographs can be performed to evaluate for the recurrence or resorption of free air.
- Quantitation of air can be performed by using a syringe and measuring the amount of air aspirated. If an animal is attached to a closed collection system, monitor the bubbles in the water trap that are visible as air is sucked into it.

Prevention/Avoidance

- Do not allow pets to free roam or have access to large animals that can cause bodily harm.

Possible Complications

- Possible complications range from death due to cardiovascular and respiratory collapse in the most severe cases, to iatrogenic lung laceration during thoracocentesis or thoracostomy

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tube placement. Infection, lung laceration, liver or diaphragm laceration, and reexpansion pulmonary edema can also potentially occur.

Expected Course and Prognosis

- The prognosis for traumatic pneumothorax is generally good and largely depends on the extent of other concurrent injuries. With simultaneous injuries such as internal hemorrhage, pulmonary contusions, and cardiovascular collapse, prognosis worsens. With spontaneous pneumothorax, the prognosis is more guarded, unless the location of the air leak is isolated to a small area of lung. With multifocal or generalized pulmonary parenchymal disease, prognosis becomes more and more guarded for long-term recovery.

Abbreviations

CBC: complete blood count
CT: computed tomography
H_2O: water
SpO_2: pulse oximeter oxygen saturation

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chapter 77

Proptosis

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DEFINITION/OVERVIEW

- Proptosis of the globe occurs when there is acute trauma that results in forward displacement of the globe such that the eyelid margins are located posterior to the equator of the globe and prevent return of the globe to its normal position (Fig. 77.1).
- Proptosis is usually a unilateral occurrence, but bilateral traumatic proptosis can occur.
- Proptosis often leads to blindness in the affected eye. Secondary bradycardia may occur via the oculocardiac reflex.
- Brachycephalic breeds are predisposed to proptosis; proptosis in nonbrachycephalic dogs and cats is generally secondary to significant trauma.



Fig. 77.1. Severe proptosis in a Boston terrier. This eye is not salvageable and will be enucleated.

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ETIOLOGY/PATHOPHYSIOLOGY

- Proptosis usually occurs due to a traumatic event, often secondary to fights between dogs in the home, such as big dog/little dog interactions. However, predisposed breeds may develop proptosis during less traumatic events such as an unusually abrupt jump from a high piece of furniture. In nonbrachycephalic dogs and cats, proptosis is more likely to result from incidents such as vehicular trauma.
- Proptosis can also occur when a collar around the neck is pulled quickly or suddenly.
- Grooming nooses have also been involved in cases of proptosis in certain dogs.
- Scruffing the fur at the back of the neck can also result in proptosis in predisposed breeds.

Systems Affected

Ophthalmic

- Blindness: due to traumatic optic neuropathy, severance of optic nerve, or severe hyphema.
- Lagophthalmos: prior to surgical correction, the eyelid margins are located posterior to the equator of the globe and the patient is unable to blink. Exposure keratitis is frequently present and severe corneal ulcers can develop rapidly if corneal lubrication is not initiated promptly and maintained until the proptosis is reduced.
- Lateral strabismus: traumatic rupture or disinsertion of extraocular muscles is commonly associated with proptosis, with the medial rectus muscle being the most frequently damaged.
- Keratoconjunctivitis sicca/neurotrophic keratitis: damage to the lacrimal system and innervation to the cornea can result in reduced tear production and keratitis after a proptosis.

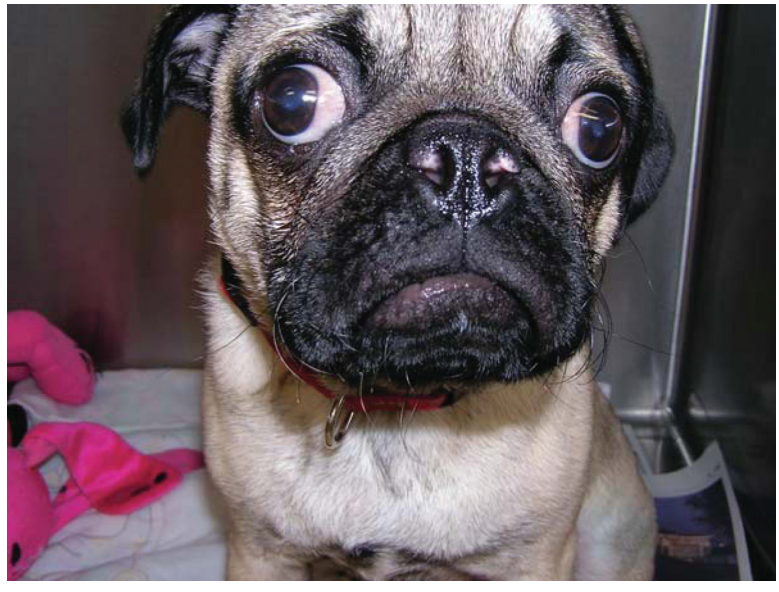
SIGNALMENT/HISTORY

- No age or sex predilection for this condition.
- Brachycephalic breeds are more prone to proptosis than mesocephalic and dolichocephalic breeds (Fig. 77.2).
- Brachycephalic breeds tend to have a much shallower orbit and a wider palpebral fissure.

Historical Findings

- Trauma, altercation with another animal.
- Dog or cat cannot close the eye.
- Severe redness and swelling all the way around the eye.
- The eye has “popped” out.

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■ **Fig. 77.2.** Pugs are just one brachycephalic breed that are predisposed to proptosis.

CLINICAL FEATURES

Dogs and Cats

- Forward displacement of the globe such that the eyelid margins are posterior to the equator of the globe and there is no ability to blink.
- Mild-to-severe chemosis.
- Mild-to-severe subconjunctival hemorrhage (see Fig. 77.2).
- Possible hyphema.
- Possible anisocoria.
- Possible pupillary light reflex deficits.

DIFFERENTIAL DIAGNOSIS

Dogs and Cats

- Moderate-to-severe exophthalmos due to trauma, cellulitis, abscess, or mass.
- Buphthalmos associated with end-stage glaucoma.
- The position of the eyelids differentiates proptosis from exophthalmos or buphthalmos. If the eyelid margins can be easily seen and identified, and they are not rolled inward or posterior to the equator of the globe, then the eye is exophthalmic or buphthalmic and not proptosed. Similarly, if the patient can blink, the eye is exophthalmic or buphthalmic and not proptosed.

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- If the eye is abnormally prominent but the eyelids are located anterior to the equator of the globe and a partial or complete blink is possible, exophthalmos and buphthalmos should be considered. An exophthalmic eye will have reduced retropulsion, whereas a buphthalmic eye will have normal retropulsion but appear larger in relation to the size of the palpebral fissure in comparison with the normal, contralateral eye.
- Sometimes severe exophthalmos and proptosis are treated similarly in order to take care of the globe and the ocular surface.



DIAGNOSTICS

- CBC, serum chemistry profile, and urinalysis are usually normal unless the patient has sustained additional trauma.
- Skull radiographs or CT may be useful if orbital fractures are suspected.



THERAPEUTICS

Initial Management

- Keep the cornea lubricated: frequent and prompt application of saline ocular irrigation solution, artificial tear products, or unmedicated petroleum is acceptable. If you receive a phone call from an owner describing a proptosis, it is a good idea to recommend institution of lubrication during transportation if appropriate products are available.
- Perform a physical examination to ensure patient stability and evaluate for additional trauma before proceeding to general anesthesia to reposition the globe.
- Treat the patient for shock or other trauma, if necessary, before performing surgery.
- Determine prognosis for vision and for comfortable retention of the globe, and discuss with owner whether repositioning of the globe or enucleation is indicated.
    - Repositioning should be considered if:
        - ☐ The owner will be able to provide follow-up care for the pet
        - ☐ Three or fewer extraocular muscles are estimated to have been ruptured based on globe position
        - ☐ Severe corneal pathology is not present
        - ☐ The optic nerve is not visible
        - ☐ The proptosis is relatively recent
        - ☐ The owner understands that vision may not return and that the eye is potentially at risk of keratitis, keratoconjunctivitis sicca, permanent blindness, and strabismus.
    - Note: Although a positive direct and/or consensual PLR are good prognostic signs, mydriasis and/or absent direct and/or consensual PLRs do not indicate definitive loss of vision.

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- Enucleation should be recommended if:
  - The owner is unable to make follow-up appointments and take care of the eye if further care is necessary after replacement of globe
  - The eye has ruptured or there is significant trauma to the globe itself
  - Three or more extraocular muscles have been torn – episcleral hemorrhage may be indicative of extraocular muscle rupture; the number of ruptured muscles can also be estimated based on globe orientation and position
  - If the optic nerve is visible, the eye is not a candidate for proptosis reduction but only for enucleation.
- Enucleation should be considered if:
  - The eye is completely filled with blood (reduced prognosis for vision; can be replaced if owner accepts risk of blindness, glaucoma, or other secondary complications, but wishes to attempt to save the globe)
  - There is severe corneal dessication.

Repositioning the Globe

- This should be done as soon as possible, as long as the patient is stable.
- This may be done using sedation and local anesthesia if absolutely necessary but is more efficiently done using general anesthesia, as long as the patient is stable.
- Lateral canthotomy may relieve some of the eyelid tension and aid in placement of temporary tarsorrhaphy sutures. Incise the lateral canthus using small Mayo or Metzenbaum scissors, temporarily elongating the palpebral fissure. If performed, a lateral canthotomy incision must also later be closed.
- Preplace 2–3 temporary tarsorrhaphy horizontal mattress sutures using a nonabsorbable 4-0 or 5-0 suture (Fig. 77.3). The suture should enter or exit the eyelid margin at the level of the meibomian gland openings and not at the conjunctival or epidermal surfaces of the eyelid. Splitting the thickness of the eyelid like this helps prevent the suture material from making contact with the cornea and causing an ulcer. If it is too difficult to pass the needle precisely through the eyelid margin with the globe proptosed, the sutures may be passed full thickness through the lid to reposition the globe. Once repositioned, the sutures are replaced with appropriately positioned tarsorrhaphy sutures.
- Apply an appropriate antibiotic ophthalmic ointment (i.e., neomycin, polymyxin B, bacitracin) to the cornea.
- Using a scalpel handle over the cornea but under the preplaced sutures, the globe is held in place while gentle traction is placed on all the sutures simultaneously, bringing the eyelids forward and into proper position over the globe. Note that the globe is *not* pushed back into the orbit, but rather the eyelids are pulled gently forward over the globe. Due to orbital tissue swelling, the globe will remain anteriorly displaced under the eyelids during initial healing; the goal of reduction is to replace the eyelid margins to the front of the globe for protection.

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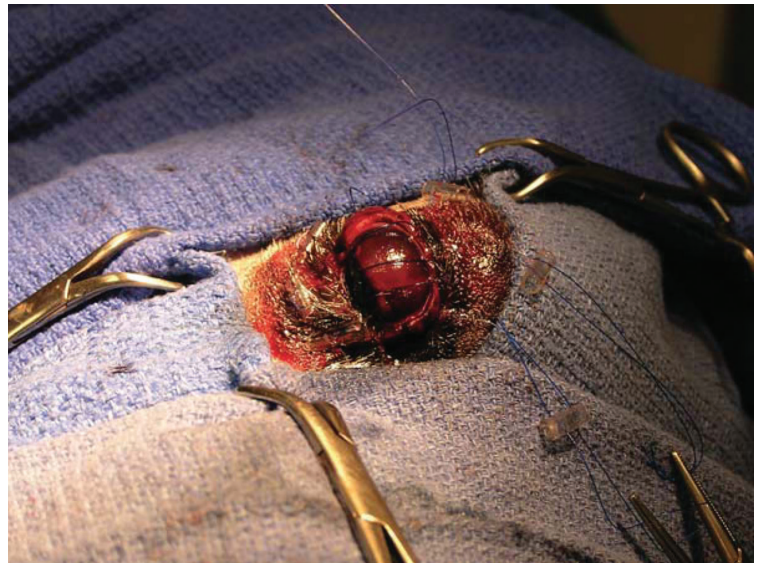


Fig. 77.3. Preplaced sutures during repositioning of the proptosed globe. This eye is filled with blood and the likelihood of restoring vision is poor. Another treatment choice in the patient would be enucleation.

- Tarsorrhaphy sutures are tied or replaced. Additional sutures are placed if necessary. The lateral canthotomy is closed. The palpebral fissure may be closed completely or a small gap can be left open at the medial canthus to allow application of ophthalmic medications.

Drugs of Choice

- Systemic antibiotics should be used for at least 7–14 days.
- If the medial canthus is left partially open, topical antibiotics should be used as long as sutures are in place. Frequency and type of topical antibiotic will depend on degree of corneal or conjunctival trauma or ulceration. Topical atropine may be used to relieve ciliary body muscle spasm and dilate the pupil to try to prevent posterior synechiae formation.
- Systemic antiinflammatory medication should be administered promptly, if the patient can tolerate it, and continued for 5–7 days.
- Systemic opioid analgesia may be indicated in addition to antiinflammatory medications.

Precautions/Interactions

- Do *not* use topical corticosteroids if the cornea is unhealthy.
- Do *not* use systemic corticosteroids if corneal ulceration is present or orbital infection is likely.
- Secondary bradycardia may occur via the oculocardiac reflex when manipulating the globe. Have atropine ready if necessary, although premedication with glycopyrrolate can help prevent bradycardia secondary to repositioning of the globe.

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Activity

- Walks using a leash and harness should be fine until the eye has healed. Keep the animal indoors or in a well-controlled environment until healing is complete.

COMMENTS

Client Education

- The patient should be kept quiet and the area around the eye clean while it is healing.
- Keep other animals from licking the affected eye.
- An Elizabethan collar may be necessary to protect the eye, and is recommended.
- Administer ophthalmic solutions or suspensions before applying ophthalmic ointments.

Patient Monitoring

- Monitor for excessive discharge from eye, and for excessive swelling, heat, or pain when opening mouth or eating, which may indicate orbital abscess formation.
- First recheck examination should be 5–7 days after repositioning the globe. Much of the retrobulbar hemorrhage and swelling may be reduced and significant suture loosening may occur at this time. This recheck is to make sure suture placement is still acceptable and not causing problems for the corneal surface.
- Sutures are usually removed sequentially and not all at the same time, starting at 10–14 days after repositioning. The last suture may not be removed until 3–4 weeks after placement or longer, if appropriate eyelid function has not returned, and there is still risk of drying out or injury to the ocular surface due to exposure. If sutures are removed prematurely, adequate fibrosis will not have taken place and repeat proptosis may occur.
- If lateral tarsorrhaphy sutures were placed, these can be removed at 10–14 days if adequate healing has taken place.

Prevention/Avoidance

- This injury is usually accidental and very sudden. Be careful introducing brachycephalic dogs to large dogs.

Possible Complications

- Lateral or dorsolateral strabismus due to extraocular muscle avulsion. May improve or worsen with time.
- Keratoconjunctivitis sicca.
- Neurotrophic keratitis.
- Permanent blindness.

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Expected Course and Prognosis

- If the globe can be successfully replaced and severe hyphema is not present, then there is a chance some vision may be preserved.
- Prognosis for vision is usually poor, regardless of how quickly the globe is repositioned.
- Prognosis is usually fair to good for being able to save the globe.
- Positive menace response, direct pupillary light reflex in the injured eye, and a consensual pupillary light reflex in the normal eye indicate a better prognosis for maintaining vision, but still guarded because damage to the eye may still be occurring.
- Pupil size or response is not an accurate indicator of visual outcome.

Abbreviations

CBC: complete blood count
CT: computed tomography
PLR: pupillary light response

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chapter 78

Pulmonary Arterial Hypertension

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DEFINITION/OVERVIEW

- PAH is present when the mean, sustained pulmonary arterial pressure is greater than 25 mmHg. MPAP is related to the PVR and PBF as evidenced in the equation:

$$\text{MPAP} = (\text{PVR} \times \text{PBF}) + \text{mean pulmonary capillary wedge pressure}$$

- Historically, PAH was classified as either primary (idiopathic) or secondary. However, following identification that some conditions within the category of secondary PAH resembled idiopathic disease pathologically and in their response to treatment, the World Health Organization introduced a new classification scheme devised on the basis of mechanisms. The groups, with examples of associated veterinary conditions, are as follows/
  - Group I: Pulmonary arterial hypertension
    - Idiopathic
    - PAH associated with systemic to pulmonary shunts
    - PAH associated with portal hypertension
    - PAH associated with drugs/toxins
  - Group II: Pulmonary venous hypertension
    - Mitral valve insufficiency/mitral stenosis
    - Myocardial disease with elevated left ventricular diastolic pressure
  - Group III: PAH associated with chronic respiratory disease/hypoxemia
    - Chronic obstructive pulmonary disease
    - Interstitial lung disease
    - Chronic exposure to high altitude
  - Group IV: PAH associated with chronic pulmonary thromboembolism
  - Group V: PAH due to miscellaneous disorders directly affecting the pulmonary vasculature
    - Heartworm (*Dirofilaria immitis*)
    - *Angiostrongylus vasorum*
    - *Aelurostrongylus abstrusus*

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Companion website: www.fiveminutevet.com/emergency

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ETIOLOGY/PATHOPHYSIOLOGY

- The principal vascular changes that produce PAH include vasoconstriction, smooth muscle cell and endothelial cell proliferation, and thrombosis. These processes reduce the cross-sectional area of the pulmonary arterial bed and the pulmonary compliance, thereby increasing PVR.
- Pulmonary endothelial cell dysfunction or injury likely contribute to these homeostatic imbalances via alteration of the complex interactions between various vasodilators (e.g., prostacyclin, nitric oxide) and vasoconstrictors (e.g., thromboxane A_2, endothelin-1, serotonin), growth inhibitors and mitogenic factors, and antithrombotic and prothrombotic determinants.
- Pulmonary venous hypertension secondary to myocardial disease in cats and myocardial and valvular disease in dogs may contribute to increased pulmonary arterial pressures via retrograde transmission of left atrial pressure to the pulmonary vascular bed, hypoxia-induced vasoconstriction of the pulmonary arteries, endothelial dysfunction, and vascular remodeling.
- Animals with PAH secondary to chronic respiratory disease presumably develop elevated pulmonary arterial pressure as a result of direct damage from the primary disorder, hypoxia-induced vasoconstriction, and vascular remodeling.

Systems Affected

- Respiratory.
  - Coughing, respiratory distress, and tachypnea may manifest as a result of any primary underlying pulmonary disease.
  - Vascular remodeling and hypoxemia of exertion associated with PAH may also independently reduce lung compliance and contribute to respiratory compromise.
- Cardiovascular.
  - Increased pulmonary arterial pressure contributes to right ventricular concentric hypertrophy in an effort to normalize ventricular wall stress.
  - Depending on the speed with which it develops and its severity, PAH may ultimately increase right ventricular end-diastolic and right atrial pressures, producing right-sided heart failure.
  - Exertional syncope may occur secondary to hypoxemia, exercise-induced vasodilation, and an inability to increase cardiac output/pulmonary blood flow; dysrhythmias or reflex-induced bradycardia and hypotension have also been hypothesized to contribute to syncope.
- Musculoskeletal.
  - Hypoxemia.
  - Ventilation-perfusion mismatch.
  - Lactic acidosis.
  - Fixed cardiac output contributes to exercise intolerance and possibly weakness.

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- Nervous.
  - Hypoxemia may contribute to depression and lethargy.
  - When combined with dysrhythmias or activity, PAH contributes to syncope.
- Hepatobiliary.
  - Concurrent right-sided congestion may contribute to cholestasis.



SIGNALMENT/HISTORY

- The signalment varies greatly based on the etiology that produces the PAH. Population characteristics from two large retrospective studies identified an age range from 2 months to 17 years although the largest percentages of dogs in both studies were younger than 10 years of age. Similarly, there is a wide weight distribution wherein dogs weighed between 2 and 67 kg. There is no apparent sex predilection and it is difficult to predict if there are breed predispositions, although:
  - Dogs predisposed to degenerative mitral valve disease will be at increased risk for developing pulmonary venous hypertension
  - Geographic distribution and heartworm preventive status may increase the likelihood of PAH associated with heartworm disease
  - West Highland white terriers with interstitial pulmonary fibrosis may also be at higher risk for development of PAH.
- Depending on the etiology, severity, and rate of onset of the PAH, owners may report a slow, insidious onset of clinical signs or an acute, rapid onset of life-threatening respiratory compromise.

Risk Factors/Causes

- Conditions that contribute to hypercoagulable states (e.g., protein-losing nephropathy, hyperadrenocorticism, neoplasia, and immune-mediated hemolytic anemia) may increase the risk for pulmonary thromboembolism and acute development of pulmonary hypertension.
- Acute development of PAH secondary to embolization of *Dirofilaria immitis* may occur following adulticide therapy.
- Large left-to-right congenital cardiac shunts may contribute to increased PBF and vascular remodeling, both of which contribute to PAH.
- Although it is generally accepted that dogs have low pulmonary vascular reactivity, there is some risk of developing mild-to-moderate PAH resulting from chronic hypoxemia associated with living at high altitude.

Historical Findings

- Exercise intolerance
- Coughing, tachypnea, or respiratory distress
- Syncope
- Abdominal distension/right-sided heart failure

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CLINICAL FEATURES

- Coughing, tachypnea, or respiratory difficulty/orthopnea.
- Crackles and wheezes during pulmonary auscultation.
- Murmurs of tricuspid or pulmonic insufficiency.
- A split S2 associated with delayed pulmonary valve closure.
- Dysrhythmias.
- Evidence of right-sided heart failure: jugular venous distension, positive hepatojugular reflux, hepatomegaly, and/or ascites.
- Cyanosis.
- Audible murmur or gallop correlating with the underlying etiology (e.g., left apical systolic murmur associated with mitral valve insufficiency or a left basilar continuous murmur associated with a left-to-right shunting PDA).



DIFFERENTIAL DIAGNOSIS

- Pneumonia
- Left-sided heart failure
- Heartworm disease
- Lungworm disease
- Tracheal collapse
- Feline asthma (bronchitis)
- Obstructive lung disease
- Bronchitis
- Pulmonary fibrosis
- Neoplasia
- Noncardiogenic pulmonary edema
- Pulmonary contusions
- Inflammatory respiratory disease
- Many of these disorders may produce varying degrees of PAH; therefore, they are not mutually exclusive.



DIAGNOSTICS

- Animals with severe PAH may be in an extremely critical condition and the diagnostic tests performed must be dictated by the clinical situation.

Thoracic Radiographs

- Although thoracic radiographs are insensitive for identification of PAH, they are useful for evaluation of underlying pulmonary disease.

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- In severe cases of PAH, dilation of the main pulmonary artery and right-sided heart enlargement may be identified.
- An enlarged caudal vena cava, ascites, hepatomegaly, and in some cases pleural effusion may accompany right-sided heart failure.
- Left atrial and left ventricular enlargement with pulmonary venous congestion may accompany left-sided heart disease that produces pulmonary hypertension.
- Dilated, tortuous, and truncated pulmonary arteries may be seen with heartworm disease.
- Peripheral pulmonary arterial markings may abruptly stop with pulmonary thromboembolism.

Electrocardiography

- Although electrocardiography cannot diagnose PAH, it may identify patterns of right ventricular enlargement, ST segment depression, or dysrhythmias that warrant institution of antiarrhythmic drugs.

Echocardiography

- Two-dimensional, spectral, and color flow Doppler echocardiography often allows for the noninvasive assessment of PAH.
- Right ventricular concentric and eccentric hypertrophy may be identified in cases of moderate-to-severe PAH in response to increased afterload, elevated end-diastolic pressures, and valvular insufficiencies.
- Diastolic (Fig. 78.1) or systolic (Fig. 78.2) interventricular septal flattening may be identified when right ventricular pressure exceeds left ventricular pressure.

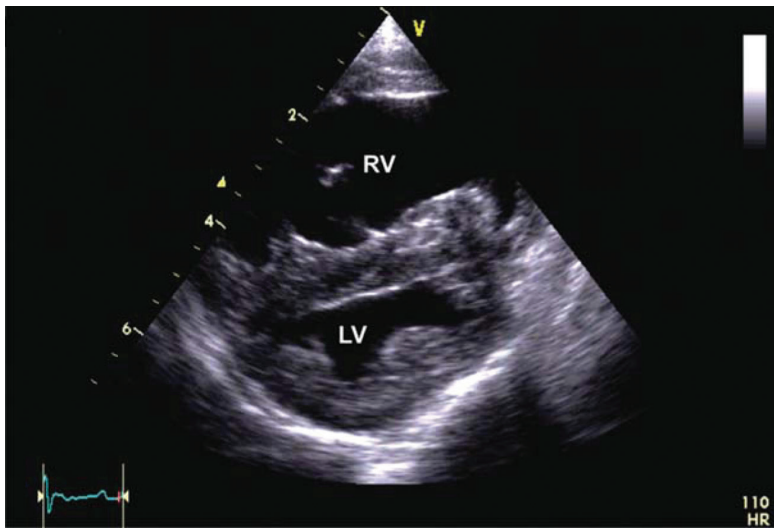
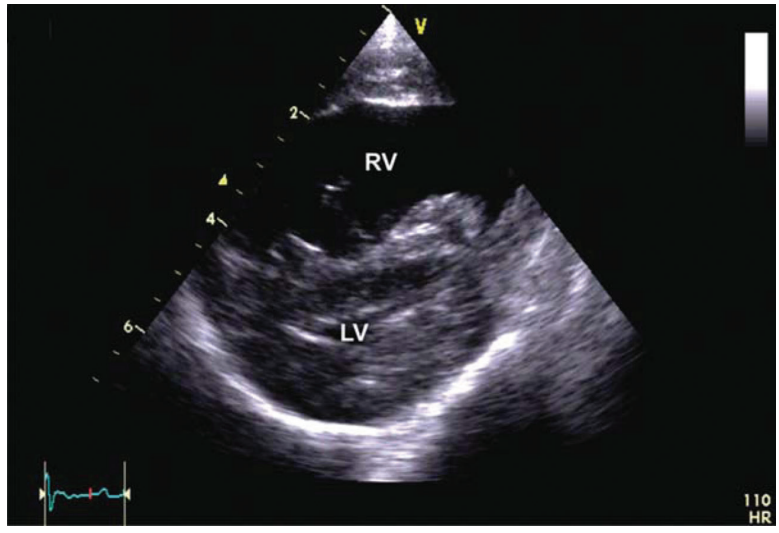


Fig. 78.1. A diastolic frame from a dog with severe pulmonary hypertension identified right ventricular (RV) dilation and thickening with interventricular septal flattening and reduced left ventricular (LV) diameters.

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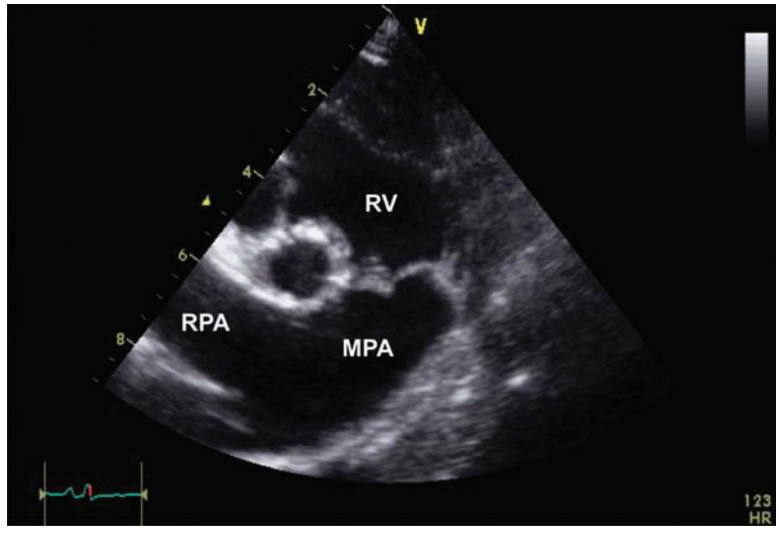
■ **Fig. 78.2.** A systolic frame from a dog with severe pulmonary hypertension identified marked septal flattening and end-systolic cavitary obliteration that may contribute to systolic anterior motion of the mitral valve. RV, right ventricle; LV, left ventricle.

- Dilation of the pulmonary artery and main branches (Fig. 78.3) may be present.
- Small end-diastolic left ventricular diameters may be identified resulting from reduced left ventricular preload in cases of severe PAH. This may also contribute to systolic anterior motion of the mitral valve.
- Pulmonic stenosis or other complex defects of the right ventricular outflow tract can be ruled out as etiologies for the right ventricular hypertrophy.
- Pulmonary arterial velocity profiles can be evaluated to increase the suspicion of PAH in the absence of quantifiable valvular insufficiencies.
- The presence of tricuspid insufficiency or pulmonic insufficiency enables estimation of pulmonary arterial pressure via use of the modified Bernoulli equation wherein the calculated pressure gradient = 4 × (velocity in meters/s)^2.
- Echocardiography enables identification of concurrent mitral valve insufficiency, myocardial disease or congenital heart disease that may contribute to PAH.
- Nuclear imaging and computed tomography may be useful for identification of large pulmonary thromboemboli.

Cardiac Catheterization

- Although infrequently performed, placement of a Swan–Ganz catheter to measure pulmonary arterial pressures, estimate left atrial pressure, perform thermodilution measures of cardiac output, and calculate PVR may be considered the gold standard for the diagnosis of PAH.

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■ **Fig. 78.3.** Echocardiography may reveal marked dilation of the main pulmonary artery (MPA) and right pulmonary artery (RPA) in comparison to the aorta. RV, right ventricle.

- Direct pulmonary arterial measures are further used in humans to assess the acute vasoreactivity of the pulmonary arteries to short-acting vasodilators and enable identification of candidates for chronic oral vasodilator therapy.
- Pulmonary angiography may help identify large pulmonary thromboemboli although life-threatening complications have been reported in humans with severe, acute PAH.

Other Tests

- A complete blood count, biochemical profile, urinalysis, and heartworm and tick-borne infection serology, and if possible TEG, should be performed to assess for underlying disease conditions that may contribute to PAH or hypercoagulability. These diagnostics also allow for investigation of consequences that may be attributable to the reduced cardiac output that frequently accompanies acute, severe PAH.

Pathologic Findings

- Dogs with PAH have been reported to have the same constrictive and complex lesions as those identified in people, including four main histologic subsets.
  - Isolated medial hypertrophy.
  - Medial hypertrophy-intimal thickening without plexiform lesion.
  - Variations of the plexiform lesion and medial hypertrophy-intimal thickening was the most common pattern in one study.
  - Isolated arteritis.
- Pulmonary and cardiac lesions vary markedly in animals with primary lung or heart disease, pulmonary thromboembolism, and heartworm disease.

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THERAPEUTICS

- The therapeutic objectives for the management of pulmonary hypertension would ideally include (1) resolution of any underlying pulmonary disease or complicating factor contributing to the elevated pulmonary arterial pressure, and (2) therapeutics aimed at reducing the pulmonary arterial pressure.
- Unfortunately, many of the primary pulmonary diseases are severe and irreversible by the time PAH is diagnosed.
- Similarly, although vasodilatory agents would seem beneficial, they may actually be contraindicated in the face of PAH related to pulmonary disease as they could exacerbate ventilation-perfusion mismatch by dilating areas that are not ventilated.
- Further complicating the use of vasodilatory agents (e.g., amlodipine) is the fact that most traditional arterial vasodilators more effectively reduce systemic arterial pressures and may produce symptomatic hypotension in the face of fixed right ventricular output.
- Many of the new and potentially promising vasodilatory agents used in humans, including endothelin receptor antagonists, constant rate infusion, and subcutaneous and oral synthetic prostacyclin analogs, are often cost-prohibitive or difficult to administer in veterinary species.

Drugs of Choice

- Oxygen.
  - Although dogs are generally accepted to have low pulmonary vascular reactivity, oxygen supplementation may be useful for reducing hypoxic-induced arterial vasoconstriction.
- Sildenafil.
  - A phosphodiesterase V inhibitor has been evaluated in a small number of dogs retrospectively.
  - There are varying reports regarding its ability to reduce echocardiographically determined pulmonary arterial pressure.
  - The retrospective studies suggest it may improve owner-perceived clinical signs and quality of life.
  - Whether the same results hold true in a prospective, large-scale, blinded study is uncertain.
  - There are no studies that clearly define the appropriate dose of sildenafil for the management of PAH in dogs but anecdotally the author has started with 1–2 mg/kg PO every 12 hours safely.
- L-Arginine (500 mg PO q12h).
- Diuretics, ACE inhibitors, and positive inotropes are used as indicated to treat valvular disease or myocardial disease that is contributing to PAH.
- Theophylline (10 mg/kg PO q12h).

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Therapeutic Procedures

- Abdominocentesis or thoracocentesis may need to be performed in animals compromised by ascites and pleural effusion, respectively.
- Atrial septostomy has been reported to improve hemodynamics and clinical signs in some humans with pulmonary arterial hypertension.
- Therapy in humans is usually palliative in an effort to prolong survival until a heart-lung transplantation can be performed.

Precautions/Interactions

- Excessive preload reduction may further limit cardiac output in animals with severe PHT producing weakness, lethargy, syncope, or sudden death.

Activity

- Many animals with moderate-to-severe PAH display exercise intolerance and should have restricted activity.
- Moderate-to-severe PAH may produce exertional syncope and increased myocardial oxygen demands may precipitate dysrhythmias.

COMMENTS

Client Education

- Clients should be informed that in most instances the medications will need to be administered throughout the remainder of the animal's life.
- Clients should be educated about the importance of periodic rechecks and should be instructed to monitor for clinical signs that may signify recurrence or progression of PAH.

Patient Monitoring

- Owners should be instructed to closely monitor general attitude, activity and exercise tolerance, respiration, and presence of right-sided heart failure.
- Follow-up diagnostic tests should be dictated by the underlying disease process that is producing the PAH.
- Echocardiographic monitoring of pulmonary arterial pressure can be performed after institution of sildenafil. Uptitration may be attempted based on clinical status, echocardiographic findings, and patient tolerance.

Prevention/Avoidance

- Owners should be cautioned about taking animals with moderate-to-severe PAH to excessive elevations.

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Possible Complications

- Reported complications that may be attributed to sildenafil therapy include lethargy, somnolence, clear nasal discharge, erect ears, and cutaneous flushing.
- One report suggests severe hypotension and death may develop if sildenafil is combined with organic nitrates (i.e., nitroglycerin paste).

Expected Course and Prognosis

- The expected course and prognosis for animals with PAH are difficult to define because there are many etiologies that contribute to the disease.
- Overall, with the exception of heartworm disease with resolution or reduction of PAH following adulticide therapy, the prognosis for animals with moderate-to-severe pulmonary hypertension is guarded to grave.
- In most cases wherein PAH is secondary to chronic respiratory disease, the underlying pulmonary pathology is severe and irreversible by the time PAH is diagnosed.
- Animals with mild-to-moderate PAH secondary to valvular disease or myocardial disease have a fair short-term prognosis and guarded long-term prognosis assuming the disease is progressive.

Abbreviations

ACE: angiotensin-converting enzyme
MPAP: mean pulmonary arterial pressure
PAH: pulmonary arterial hypertension
PBF: pulmonary blood flow
PDA: patent ductus arteriosus
PO: by mouth (per os)
PVR: pulmonary vascular resistance
TEG: thromboelastography

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chapter 79

Pulmonary Contusions





DEFINITION/OVERVIEW

- Syndrome that causes pulmonary parenchymal and alveolar damage secondary to compressive and decompressive forces associated with blunt trauma to the thorax.



ETIOLOGY/PATHOPHYSIOLOGY

- Compression then decompression of the thorax during blunt thoracic trauma transfers the energy or mechanical force to the pulmonary parenchyma and alveoli. The result is damage to the parenchyma and alveolar capillary membrane. Leakage of blood and plasma fluid into the alveoli and pulmonary interstitial space results in areas of lung with a ventilation-perfusion mismatch and hypoxia.
- Following initial injury, influx of fluid and inflammatory cells into the damaged area can perpetuate the pulmonary damage and injury by the generation of oxygen-derived free radical species and oxidative damage to the lung, and cause secondary lung injury. Depending on the extent of damage, ALI or ARDS can result.

Systems Affected

- Respiratory: areas of lung that are perfused but not well ventilated cause a ventilation-perfusion mismatch, and hypoxia, tachypnea, orthopnea, restrictive respiratory pattern, and hemoptysis may be present; can progress to ALI or ARDS.
- Other body systems are affected secondary to hypoxia.

Incidence/Prevalence

- Reported in 34–57% of dogs and 18% of cats with limb fractures following motor vehicle trauma.
- Occurs in approximately 50% of animals with thoracic trauma.

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Geographic Distribution

- Worldwide.



SIGNALMENT/HISTORY

Species

- Dogs and cats.

Historical Findings

- History of blunt trauma
- Free-roaming animal or on property with large animals
- Acute onset
- Respiratory difficulty

Physical Examination Findings

- Tachypnea
- Pale or cyanotic mucous membranes
- Orthopnea
- Cough
- Hemoptysis
- Increased bronchovesicular sounds and pulmonary crackles
- Often other physical examination indications of trauma
- Severity of respiratory signs associated with the degree of injury

Risk Factors/Causes

- Free-roaming off leash
- Fall from height
- Lives with larger animals or farm animals
- Potential for animal abuse injury
- Potential for coagulopathy



DIFFERENTIAL DIAGNOSIS

Coagulopathy

- Hemothorax or pulmonary hemorrhage from vitamin K antagonist rodenticide intoxication.

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- Can be differentiated from pulmonary contusions by coagulation tests, as ACT and PTs will be markedly prolonged with vitamin K antagonism. Severe thrombocytopenia <50 000 platelet/μL can be associated with pulmonary hemorrhage.

Neoplastic Cause of Pulmonary Hemorrhage

- Hemangiosarcoma is the most common.

Pneumothorax

- Muffled lung sounds; will see retraction of the lungs away from the thoracic wall on thoracic radiographs.

Diaphragmatic Hernia

- Loss of detail in pleural cavity, loss of demarcation of diaphragm, possible inclusion of abdominal contents in thorax.
- Cardiac failure with pulmonary edema.
- Inhaled or ingested toxins, such as paraquat.
- Bacterial or viral pneumonia.



DIAGNOSTICS

Complete Blood Count/Biochemistry/Urinalysis

- Not specific for pulmonary contusions, changes such as anemia, neutrophilic leukocytosis, or elevations in liver enzyme activities may be associated with trauma but not related to pulmonary contusion, *per se*.
- Urinalysis is usually normal unless damage to the kidneys or urinary bladder has occurred secondary to trauma.

Other Laboratory Tests

- PT or ACT to rule out vitamin K antagonist rodenticide toxicity.
- Platelet count to rule out severe thrombocytopenia and secondary pulmonary hemorrhage.

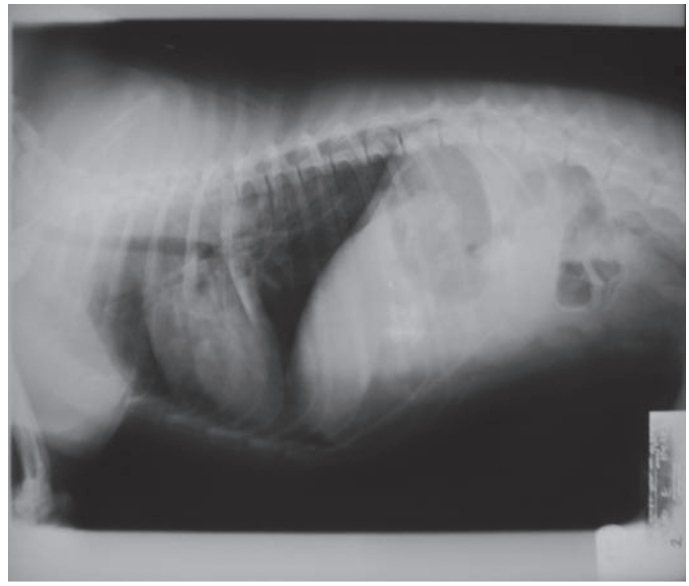
Thoracic Imaging

- Thoracic radiographs should be performed only *after* cardiovascular and pulmonary stabilization of the patient, whenever possible.
- Interstitial to alveolar lung pattern in any lung field (Figs 79.1, 79.2).
- Radiographic appearance of pulmonary contusions may lag behind clinical signs and may appear within 24–48 hours of trauma.

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■ **Fig. 79.1.** VD thoracic radiograph from a dog 12 hours after being hit by a car. Note the interstitial to alveolar pattern in the left cranial and left middle lung field.



■ **Fig. 79.2.** Lateral thoracic radiograph of the same dog. Note the alveolar pattern overlying the cardiac silhouette.

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- Lack of radiographic appearance of contusions does not rule out their presence or potential to form after trauma.
- Severity is related to degree of injury.



THERAPEUTICS

Drugs of Choice

- Oxygen supplementation (50–150 mL/kg per minute).
- The use of furosemide is controversial and not currently recommended because it could potentially worsen hypovolemic shock.

Contraindications

- Diuretic drugs such as furosemide have not been shown to be efficacious and can exacerbate hypovolemic shock.
- The use of NSAIDs in any traumatized patient is controversial and not recommended until the patient is normotensive and renal function has been proven to be normal.
- Antibiotics: less than 5% of patients with traumatic pulmonary contusions develop respiratory infections secondary to injury. The use of empiric antibiotics in a patient with pulmonary contusions can promote the development of bacterial resistance, and is therefore contraindicated.
- Overzealous crystalloid fluid therapy can be associated with worsening of pulmonary contusions; fluid therapy should be titrated according to patient blood pressure and cardiovascular perfusion parameters.



COMMENTS

Patient Monitoring

- Monitor respiratory rate and effort, mucous membrane color, presence of hemoptysis, respiratory difficulty/orthopnea, respiratory sound on thoracic auscultation; serial measurements of blood pressure, pulse quality, pulse oximetry or arterial blood gas analysis. ECG to evaluate for traumatic myocarditis and cardiac dysrhythmias.

Prevention/Avoidance

- Avoid interactions with motor vehicles and large animals or fall from height.
- Leash restriction or run in enclosed area, away from traffic.

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Possible Complications

- ARDS
- Bacterial pneumonia (extremely rare)

Expected Course and Prognosis

- Potential to worsen over first 24 hours post injury.
- Most patients can be discharged within 36 hours of initial injury.
- Severe contusions may require assisted ventilation with positive end-expiratory pressure until wounds heal.
- Potential for development of ARDS and death in severe cases; risk increases with the degree of multitrauma.

Abbreviations

ACT: activated clotting time
ALI: acute lung injury
ARDS: acute respiratory distress syndrome
ECG: electrocardiogram
NSAID: nonsteroidal antiinflammatory drug
PT: prothrombin time

Suggested Reading

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chapter 80

Pulmonary Edema – Cardiogenic



DEFINITION/OVERVIEW

- Cardiogenic pulmonary edema is the accumulation of fluid in the pulmonary interstitial space and alveoli subsequent to left-sided cardiac dysfunction and elevations in pulmonary venous and pulmonary capillary pressures.

ETIOLOGY/PATHOPHYSIOLOGY

- Cardiogenic pulmonary edema may occur secondary to any heart condition that impairs function of the left side of the heart, including diseases that promote volume overload, systolic dysfunction, or diastolic dysfunction.
- With progressive cardiac dysfunction, left atrial, pulmonary venous, and pulmonary capillary hydrostatic pressures increase.
- Pulmonary edema develops when the net movement of fluid, colloid, and solutes from the vasculature into the interstitial space exceeds its return to the circulation.
- Cardiogenic pulmonary edema impairs gas exchange and lung mechanics and may ultimately lead to death.
- Common congenital lesions that contribute to the development of pulmonary edema include:
  - Left-to-right shunting PDA
  - Left-to-right shunting ventricular septal defect
  - Mitral valve dysplasia
  - Subaortic stenosis (uncommonly).
- Common acquired conditions that contribute to the development of pulmonary edema include:
  - Chronic degenerative mitral valve disease (dogs)
  - Dilated cardiomyopathy (dogs)
  - Hypertrophic cardiomyopathy (cats).

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Systems Affected

- Respiratory: cardiogenic pulmonary edema impairs gas exchange and reduces lung compliance.
- Cardiovascular: while cardiac dysfunction often leads to the development of cardiogenic pulmonary edema, the associated hypoxemia may contribute to arrhythmogenesis and further impair cardiac function.
- Musculoskeletal: hypoxemia contributes to weakness.
- Nervous: hypoxemia may contribute to depression and lethargy or, when combined with arrhythmias or activity, contribute to syncope.
- Hepatobiliary: concurrent right-sided congestion may contribute to cholestasis and ascites.



SIGNALMENT/HISTORY

- The signalment varies based on the cardiac disease that leads to the development of pulmonary edema.
  - Left-to-right shunting PDAs can be seen in any young dog but females and many small breed dogs, including Chihuahuas, Maltese, toy and miniature poodles, Pomeranians, Bichon Frise, Cavalier King Charles spaniels, and Shetland sheepdogs, are reportedly overrepresented.
  - Chronic degenerative mitral valve disease is most common in middle-aged to older small breed dogs.
  - Dilated cardiomyopathy is more commonly identified in middle-aged to older large to giant breed dogs, with boxers, Doberman pinschers, Great Danes, and Irish wolfhounds reportedly overrepresented.
  - Purebred cats including Maine coons and Ragdolls have identified genetic alterations that may contribute to hypertrophic cardiomyopathy although the disease is also commonly diagnosed in domestic shorthair and domestic longhair cats.
- Depending on the etiology and severity of the cardiac disease and the presence of risk factors, owners may report a slow, insidious onset of clinical signs or an acute, rapid onset of life-threatening respiratory compromise.

Risk Factors/Causes

- Cardiac arrhythmias (e.g., development of atrial fibrillation or significant ventricular ectopy) may reduce cardiac performance and precipitate the development of pulmonary edema.
- General anesthesia and intravenous fluids in animals with otherwise compensated, yet severe cardiac disease may precipitate the development of pulmonary edema.
- Events that are difficult to predict, like rupture of chordae tendineae in dogs with mitral valve disease or systemic thromboembolism in cats, may be associated with the rapid development of cardiogenic pulmonary edema.

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Historical Findings

- Coughing (dogs).
- Tachypnea, respiratory distress, open mouth breathing.These signs may be exacerbated during recumbency.
- Exercise intolerance.
- Inappetence and weight loss.



CLINICAL FEATURES

Dogs

- Almost always have a cardiac murmur or gallop.
- Arrhythmias may be audible.
- Coughing, tachypnea, respiratory distress, or orthopnea.
- There may be audible crackles and wheezes during pulmonary auscultation.
- Many dogs with cardiogenic pulmonary edema will be tachycardic.
- Severe edema may be accompanied by expectoration of pink foam or cyanosis.

Cats

- Often have a cardiac murmur or gallop.
- Arrhythmias may be audible.
- Tachypnea, respiratory distress, open mouth breathing.
- Crackles may be audible although some cats with pulmonary edema have concurrent pleural effusion, which diminishes lung sounds and may produce an audible fluid line.
- Some cats will present bradycardic and hypothermic.
- Some cats present with concurrent systemic thromboembolization.



DIFFERENTIAL DIAGNOSIS

- Pneumonia
- Pulmonary thromboembolism and pulmonary hypertension
- Heartworm disease
- Tracheal collapse
- Feline asthma
- Obstructive lung disease
- Bronchitis
- Pulmonary fibrosis
- Neoplasia
- Noncardiogenic pulmonary edema
- Pulmonary contusions
- Inflammatory respiratory disease

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- Many dogs and cats with these disorders have concurrent cardiac disease that could lead to a false assumption of cardiogenic pulmonary edema if treatment decisions are based only on the physical examination.

DIAGNOSTICS

- Animals with cardiogenic pulmonary edema may be in an extremely critical condition and the diagnostic tests performed must be dictated by the clinical situation.
- Thoracic radiographs.
  - Common hallmarks of cardiogenic pulmonary edema in dogs include left atrial enlargement (Fig. 80.1), pulmonary venous congestion, and perihilar interstitial edema.
  - As the pulmonary edema progresses and alveoli are flooded, it obscures the pulmonary vessels and produces air bronchograms, often in the perihilar region and right caudal lung lobe.
  - Severe, fulminant congestive heart failure may produce diffuse alveolar disease (Fig. 80.2).
  - Instead of the classic pattern of cardiogenic pulmonary edema in dogs, cats tend to have left auricular enlargement on the ventrodorsal or dorsoventral view and their patterns of pulmonary edema tend to be patchy and unevenly distributed (Fig. 80.3).
  - Limitations of thoracic radiography include a time lag between the onset and clearance of pulmonary edema in comparison to clinical signs and the presence of preexisting lung pathology obscuring identification of pulmonary edema.

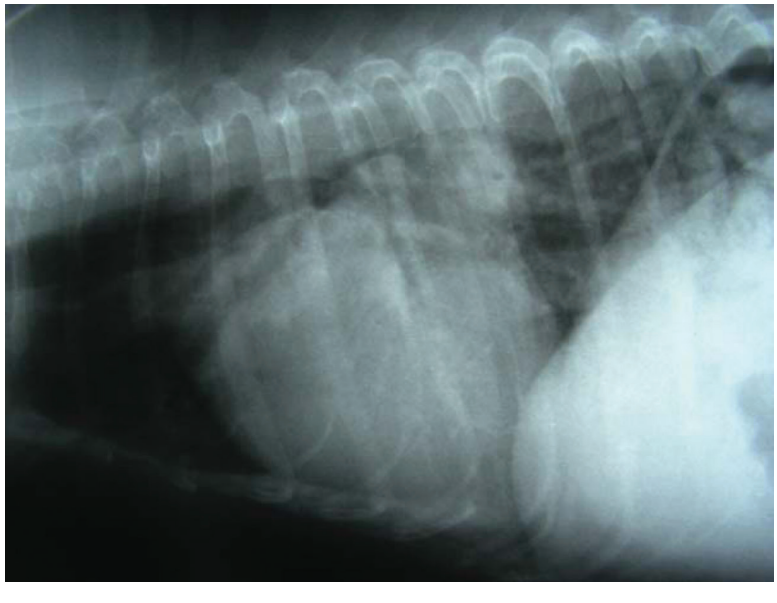
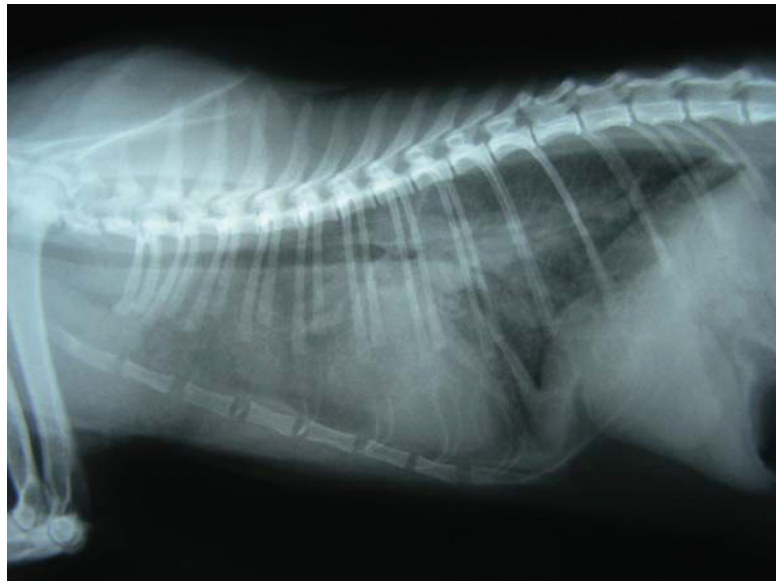


Fig. 80.1. Left atrial enlargement and perihilar interstitial to alveolar pulmonary infiltrates in a poodle with congestive heart failure secondary to mitral valve disease.

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■ **Fig. 80.2.** Generalized alveolar pulmonary pattern in a dog with dilative cardiomyopathy.



■ **Fig. 80.3.** Cardiomegaly and pulmonary edema in a cat with hypertrophic cardiomyopathy.

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- Electrocardiography.
  - Although electrocardiography is not able to diagnose pulmonary edema, it can identify arrhythmias that may warrant institution of antiarrhythmic therapy.
- Echocardiography.
  - Although echocardiography is not able to diagnose pulmonary edema, it is very useful for detecting the presence and severity of underlying cardiac disease and guiding therapy tailored for that disease.
- Cardiac catheterization.
  - Placement of a Swan–Ganz catheter to measure pulmonary arterial pressures and estimate left atrial pressure is rarely performed but may be useful for guiding therapy in animals with severe, life-threatening pulmonary edema or those with refractory congestive heart failure.
- Biochemical profile.
  - Severe cardiac compromise may reduce the glomerular filtration rate and make it more difficult to manage animals that require aggressive preload or afterload reduction.
  - Electrolyte imbalances may exacerbate arrhythmias, reduce the effectiveness of some therapeutics, or increase the likelihood of developing drug toxicities.
- Measurement of natriuretic peptides.
  - Recent studies suggest that measurement of natriuretic peptides, including atrial natriuretic peptide or B-type natriuretic peptide, may help identify animals with severe cardiac compromise and may be useful at distinguishing animals with respiratory distress of cardiac origin and noncardiac origin.



THERAPEUTICS

- The therapeutic objectives for the management of cardiogenic pulmonary edema include resolution of the pulmonary edema, maintenance of adequate tissue perfusion pressure, and adequate delivery of blood flow to vital tissues. To achieve these goals, it is important to use drugs with proven hemodynamic benefits and a rapid onset of action.

Drugs of Choice

- Oxygen.
  - Supplemental oxygen is readily administered to compromised animals by providing an oxygen-enriched environment (i.e., oxygen cage) or via nasal insufflation.
- Furosemide.
  - 2–4 mg/kg IV (dogs) and 1–2 mg/kg IV (cats).
  - These large doses may need to be repeated (initially every 1–2 hours) until the respiratory rate and level of distress start to decline.
  - Once stable, the dose should be reduced to 0.5–2 mg/kg every 6–12 hours, as dictated by clinical status.

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- Some animals will require more aggressive diuresis but care should be exercised because excessive administration may lead to profound dehydration, electrolyte depletion, renal failure, low cardiac output, and circulatory collapse.

- In dogs with severe mitral valve insufficiency (often those complicated with ruptured chordae tendineae) or severe dilated cardiomyopathy, additional therapies that may be required include the following.
  - Nitroprusside: a potent balanced vasodilator.
    - Constant rate infusion typically administered at 2–5 µg/kg per minute, diluted in 5% dextrose
    - It is light sensitive and should not be infused with other agents so a second intravenous catheter should be placed for administration of additional intravenous drugs
    - It promotes profound preload and afterload reduction so blood pressure should be continuously monitored
    - Rapid administration rates may produce cyanide toxicity
    - Discontinuation or a reduction in the infusion rate often promptly reverses side effects
  - Dobutamine: a rapid-acting positive inotropic agent.
    - Constant rate infusion typically administered at 5–15 µg/kg per minute, diluted in 5% dextrose
    - Although tachycardia/arrhythmias and vasodilation are uncommonly reported, ideally continuous ECG and blood pressure monitoring are performed
    - Discontinuation or a reduction in the infusion rate often promptly reverses side effects
  - Pimobendan, 0.25 mg/kg PO twice a day.
  - Hydralazine, 0.5–2.0 mg/kg PO twice a day.
  - Sedatives/tranquilizers: some animals with profound anxiety associated with their pulmonary edema may benefit from sedation.
- Long-term management of animals with a history of cardiogenic pulmonary edema often includes diuretics, angiotensin-converting enzyme inhibitors, positive inotropes, beta-blockers, calcium channel blockers, antiarrhythmics, or other drug classes as dictated by the underlying disease.
- Morphine (0.025 mg/kg IV) can decrease anxiety and work of breathing, and dilate splanchnic vasculature in dogs.

Precautions/Interactions

- In animals with known cardiogenic pulmonary edema, intravenous fluids, other than those used to prepare a constant rate infusion, should be avoided.
- Potent afterload reducers should be used cautiously in cats with hypertrophic cardiomyopathy complicated by systolic anterior motion of the mitral valve.

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Diet

- During the acute management of cardiogenic pulmonary edema, food and water should be withheld.
- Once stabilized, animals may have access to restricted water and food consumption.
- Low-sodium diets may be beneficial in the long-term management of animals with a previous history of cardiogenic pulmonary edema.

Activity

- During the acute management of cardiogenic pulmonary edema, strict rest should be implemented.
- After discharge, limited exercise can be resumed as the animal indicates but strenuous exercise should be avoided.



COMMENTS

Client Education

- Clients should be informed that in most instances, medications will need to be administered throughout the remainder of the animal's life.
- Clients should be educated about the importance of periodic rechecks and should be instructed to monitor for clinical signs that may signify recurrence of cardiogenic pulmonary edema.

Patient Monitoring

- Reductions in the respiratory rate and effort are the most reliable clinical signs that the cardiogenic pulmonary edema is resolving. These should be monitored and recorded at a minimum of hourly.
- Urine production should be monitored throughout hospitalization.
- A reduction in body weight may suggest effective diuresis.
- Radiographic findings often lag behind clinical signs so treatment should be based on clinical status in addition to results of diagnostic tests.
- Mild, presumably prerenal azotemia and electrolyte imbalances are common in animals treated for congestive heart failure. Biochemical profiles should be performed at initial presentation and 5–7 days after institution of therapy/discharge from the hospital to monitor the renal status.
- Radiographs should be rechecked approximately 5–7 days after institution of therapy/discharge from the hospital to assess the efficacy of therapy.
- Additional follow-up is dictated by the underlying cardiac disease, patient status, and complicating factors.

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Prevention/Avoidance

- Owners may not be able to specifically prevent the recurrence of pulmonary edema but they should make every effort to:
  - Closely monitor their animal's attitude, respiratory rate, appetite, and activity level daily
  - Develop a strict routine for administration and timing of cardiac medications
  - Follow up with instructions regarding periodic rechecks and diagnostic tests including radiographs, biochemical profiles, ECGs, and echocardiograms.

Possible Complications

- Complications, or side effects, of therapy are polyuria and polydipsia.
- Cardiogenic pulmonary edema often recurs as the cardiac disease progresses and clinical signs return or animals may die suddenly.

Expected Course and Prognosis

- Severe cardiogenic pulmonary edema is usually fatal if not treated rapidly and effectively.
- With appropriate treatment and monitoring, the short-term prognosis for animals with cardiogenic pulmonary edema is fair, assuming they respond appropriately to therapy and complicating factors (e.g., severe cardiac arrhythmias, ruptured chordae tendineae, systemic thromboembolization) are absent.
- Animals with concurrent severe renal insufficiency or acute renal failure have a poor short-term prognosis.
- Most animals with heart disease that is severe enough to produce pulmonary edema have a guarded long-term prognosis and many ultimately succumb to their heart disease. The overall median survival time is dependent on the particular disease, the presence of complicating factors, and the individual response to therapy.

Synonyms

- Left-sided congestive heart failure.

Abbreviations

ECG: electrocardiogram
IV: intravenously
PDA: patent ductus arteriosus
PO: by mouth (per os)

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chapter 81

Pulmonary Edema – Noncardiogenic

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DEFINITION/OVERVIEW

- The accumulation of abnormal fluid and solutes in the pulmonary interstitium and alveoli in the absence of heart disease.

ETIOLOGY/PATHOPHYSIOLOGY

- Common cause of all forms: increased pulmonary vascular permeability, associated with leakage of fluid, proteins, and other solutes into the interstitium and alveoli.
- The endothelial glycocalyx has recently been found to play a major role in vascular integrity and fluid homeostasis. The role of the endothelial glycocalyx in the development or prevention of pulmonary edema is still being researched and may provide for novel therapies for this patient population.
- Several proposed mechanisms, depending on underlying cause.
    - Neurogenic edema, electrocution, and upper airway obstruction.
        - Massive sympathetic discharge causes release of catecholamines, leading to systemic vasoconstriction, increased afterload, and temporary shunting of blood into the pulmonary circulation; this leads to transient pulmonary circulatory overload and endothelial damage
        - Severe decrease in intrathoracic pressure induced by inspiratory attempts against an airway obstruction; proposed additional mechanism for upper airway obstruction
    - Acute lung injury and acute respiratory distress syndrome, smoke inhalation, transfusion-related lung injury.
        - Direct chemical or inflammatory mediator damage (circulating histamine, complement, cytokines, or inhaled/ingested toxins) leads to epithelial and/or endothelial damage and increased pulmonary vasculature permeability
    - Hypoxia leads to pulmonary vasoconstriction and further increases in permeability of microvasculature.

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- For all forms, the inciting insult may trigger an inflammatory response cascade that often worsens in the first 24 hours after the initial episode; the most severe form is known as ARDS.
- Severity of clinical manifestation varies ranging from mild to severe; the most seriously affected patients may progress from normal to death in as little as a couple of hours after the incident.
- Overall incidence/prevalence is uncommon.

Systems Affected

- Respiratory: decreased compliance, pulmonary edema and atelectasis, decreased FRC.
- Hemic/lymphatic/immune: if severe and causing respiratory failure, may be associated with DIC and hypoproteinemia due to loss of protein into the pulmonary parenchyma.
- Cardiovascular: hypotension, tachycardia, other dysrhythmias, and shock.
- Renal/urologic: acute kidney injury.



SIGNALMENT/HISTORY

- Species: mainly dogs, occasionally cats.
- No confirmed breed or sex predilection; brachycephalic dogs are more prone to airway obstruction.
- Higher incidence in puppies younger than 1 year old.
- Young animals: associated with strangulation, head trauma, seizures, and electric cord bites.
- Older animals: associated with laryngeal obstruction, neoplasia, sepsis/SIRS, and seizures.
- No known genetic basis for the disease.
- Respiratory signs vary depending on underlying cause and severity.

Risk Factors/Causes

- Hypoproteinemia.
- Crystalloid fluid resuscitation.
- Upper airway obstruction: brachycephalic, laryngeal paralysis; leash/choke-chain/ strangulation injury; mass lesion; abscess; or other temporary airway obstruction.
- Electric shock.
- Acute neurologic disease: head trauma; prolonged seizures.
- Pulmonary and other neoplasia.
- Adverse drug reactions including certain anesthetic drugs (ketamine), thiazides, or certain antineoplastics such as vincristine, and cisplatin in cats.
- Chemicals (i.e., chlorine, minoxidil, waterproofing hydrocarbons).
- Blood transfusions (including human albumin in healthy dogs): not yet reported in veterinary medicine.
- Uremic toxins.
- SIRS: sepsis, pancreatitis, and endotoxemia.

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- Smoke inhalation.
- Anaphylaxis (cats).
- Hyperosmolar fluid/sea water inhalation/near drowning.

Historical Findings

- Predisposing cause.
- Acute onset of respiratory distress.



CLINICAL FEATURES

- Mild to severe respiratory distress.
- Increased respiratory rate and effort; open-mouthed breathing.
- Postural adaptations to respiratory distress in severe cases (e.g., orthopnea, neck extension).
- Restlessness, unwillingness to lie down.
- Pale or cyanotic mucous membranes (severe).
- Harsh lung sounds (early, mild) or generalized crackles (late, severe) on auscultation.
- Expectoration of pink froth or bubbles in severely affected animals; in intubated patients, large volumes of bloody fluid can flow out through the endotracheal tube.
- Normal cardiac auscultation; may note dysrhythmias; tachycardia common.
- Possible oral burns if due to electric shock.
- Patient may present with significant neurologic dysfunction consistent with forebrain disease or postictal state if secondary to seizure activity.
- Upper airway stridor in cases of brachycephalic syndrome, airway masses, foreign bodies, or abscesses.
- Smoky odor or burns indicative of smoke exposure.



DIFFERENTIAL DIAGNOSIS

- Cardiogenic pulmonary edema.
- Pulmonary infection: bacterial, viral, or fungal pneumonia.
- Pulmonary neoplasia.
- Pulmonary hemorrhage (e.g., anticoagulant rodenticide exposure).
- Pulmonary thromboembolism.
- Many of the above can occur concurrently with noncardiogenic pulmonary edema.



DIAGNOSTICS

- CBC: leukocytosis but possibly leukopenia and thrombocytopenia, owing to neutrophil sequestration in the lung and platelet consumption.
- Biochemistries: usually normal; may note hypoalbuminemia owing to pulmonary protein loss or as potentiating cause; mild hyperglycemia reported.

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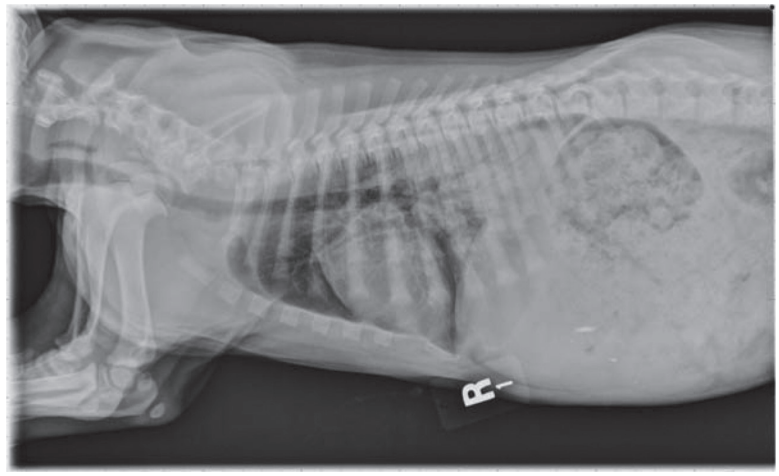


Fig. 81.1. Right lateral thoracic radiograph of 15-week-old puppy with noncardiogenic pulmonary edema secondary to strangulation.

- Urinalysis: usually normal, may show glycosuria in early cases of secondary acute kidney injury.
- Arterial blood gas analysis: usually demonstrates mild-to-severe hypoxemia and hypocapnia; results are not specific but indicate the severity of pulmonary dysfunction.
- Coagulation testing: may reveal mild-to-moderate prolongation of PT and PTT because of consumption of coagulation factors and DIC. Significant prolongations of PT (as in cases of anticoagulant rodenticide toxicity) and in some cases PTT may be more indicative that hemorrhage is the underlying etiology.
- Thoracic radiographs – vital: may simply reveal prominent interstitial pattern with mild or early disease; may note alveolar infiltrates with moderate or severe disease; alveolar infiltrates in the dorsocaudal lung fields common; alveolar infiltrates may be seen in other lung fields, are often asymmetrical and may demonstrate predominant right-sided involvement (Fig. 81.1).
- Echocardiography: rule out cardiogenic pulmonary edema.
- Troponin or NT Pro-BNP testing in cats and dogs may indicate underlying heart disease and thus support a diagnosis of cardiogenic edema. Findings are nonspecific, however, as both tests can be elevated due to severe myocardial hypoxia due to primary pulmonary disease.
- Computed tomography: optional to rule out differential diagnoses such as pulmonary neoplasia or thromboembolism, but may be contraindicated due to need for anesthesia.
- Pulse oximetry: noninvasive, continuous monitoring of arterial hemoglobin saturation; provides information about the severity and progression of pulmonary dysfunction.
- Pulmonary artery wedge pressure: confirms noncardiogenic origin; uncommonly used in small animal clinical patients.

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- ETL, TTW or BAL: if they can be safely performed, may demonstrate neutrophilic inflammation and cytologic evaluation may help rule out pneumonia of bacterial or fungal origin; culture can confirm presence or absence of infection.
- Pulmonary edema fluid to plasma ratio (EF:PL) compares protein in the edema fluid to plasma protein. An increased ratio (>0.65) indicates noncardiogenic pulmonary edema.

Pathologic Findings

- Lungs: may be heavy, red, or congested; may fail to collapse; may exhibit a wet cut surface; may note foam in the major airways.
- Histopathologic: depend on severity of the insult; early, mild: may note eosinophilic amorphous material filling the alveoli or may be near normal because fluid removed in processing; severe: alveolar hyaline membranes, alveolitis, and interstitial inflammatory infiltrates with neutrophils and macrophages evident and accompanied by atelectasis, vascular congestion, and hemorrhage; may be found only hours after a severe insult.



THERAPEUTICS

- Inpatient versus outpatient: depends on the severity of respiratory dysfunction and cause (e.g., dogs with upper airway obstruction, severe seizures, or pancreatitis may require hospitalization).
- Treat the underlying cause (e.g., relieve airway obstruction or treat seizures).
- Mild to moderate: patients generally improve on their own within 24–48 hours with complete resolution; offer support of pulmonary and cardiovascular function while the lung repairs.
- Severe: difficult to treat; may require PPV because of respiratory failure, plasma transfusions to treat DIC; many die despite extensive supportive care.
- Damaged endothelium in the pulmonary vasculature: no specific treatment available but complete recovery is possible if disease is mild and the inciting cause is resolved.
- Inflammatory response: generated by a variety of mediators and cascades; cannot be blocked by one specific antiinflammatory drug that leads to resolution of the edema.
- Oxygen therapy: vital in moderate-to-severe disease; administer via a mask or hood; oxygen concentration depends on the severity of disease; most patients do well on 40–50% oxygen, but severe disease may require 80–100% to sustain life
- Fluid therapy.
    - Used to maintain perfusion and as replacement solution in times of dehydration. Judicious use is important in these patients in order to prevent fluid overload and worsening of edema
    - Balanced electrolyte: give as replacement solution with dehydration or shock
    - Plasma, albumin (human or canine), or synthetic colloids: consider with hypoproteinemia, low measured colloid osmotic pressure, or coagulopathy.

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Drugs of Choice

- Diuretics: typically ineffective because edema is caused by changes in permeability, not high hydrostatic pressure; may use furosemide cautiously as a single bolus of 0.5–2.0 mg/kg IV, IM. Additional beneficial effects may include decreases in bronchospasm and bronchodilation.
- Corticosteroids: minimize airway swelling in animals with upper airway obstruction; generally ineffective for pulmonary inflammatory response; may predispose patients to infectious complications (e.g., bacterial pneumonia); if used, recommend an antiinflammatory dosage of a short-acting drug (e.g., dexamethasone sodium phosphate at 0.1–0.2 mg/kg IV).
- The use of beta-adrenergic agonists such as terbutaline may increase alveolar fluid clearance.
- Sedatives may be used cautiously if patient anxiety is contributing to respiratory distress or upper airway obstruction. The patient should be carefully monitored as sedation may decrease central respiratory drive leading to progression of respiratory failure.
- Additional therapy as indicated by underlying cause, such as anticonvulsants or analgesics for oral ulcerations.

Precautions/Interactions

- Minimize stress in animals with respiratory distress.
- Severe: may require PPV and PEEP.
- Aggressive fluid therapy can worsen pulmonary edema: increased circulating volume results in increased hydrostatic pressure in the pulmonary microvasculature, worsening the pulmonary vascular leakage.
- Excessive use of diuretics may cause dehydration and a marked decrease in intravascular volume with minimal resolution of edema, thereby exacerbating cardiovascular collapse or shock.
- Corticosteroids: may predispose patients to infectious complications (e.g., bacterial pneumonia).

Activity

- Rest and minimal stress vital for minimizing oxygen requirements in moderately to severely affected animals.

Surgical Considerations

- Relevant only for treating the underlying cause, particularly in cases of upper airway obstruction requiring possible tracheostomy and/or airway revision.



COMMENTS

Client Education

- Warn client that the condition may worsen before improving.
- Inform client that severe disease, which progresses rapidly to fulminant pulmonary edema and respiratory failure, is associated with a very poor prognosis.

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Patient Monitoring

- Observe respiratory rate and pattern and auscultate frequently (every 2–4 h) for the first 24–48 hours.
- Assess pulmonary function by pulse oximetry or arterial blood gas analysis (initially every 2–4 h).
- Perform PCV and total solids and evaluate mucous membranes, pulse quality, heart rate, ECG, blood pressure, and urine output every 2–4 hours to assess cardiovascular status and progression to shock.
- In severely affected animals, monitor renal function, coagulation status, and white blood cell count.
- Recheck thoracic radiographs to evaluate resolution and monitor for other contributing pulmonary disease.

Prevention/Avoidance

- Avoid contact with electric wire.
- Correct airway obstruction.
- Treat seizures or high intracranial pressure.

Possible Complications

- Usually none if patient recovers from acute crisis.

Expected Course and Prognosis

- Mild to moderate: uneventful resolution of signs in 24–72 hours; no specific treatment required except for oxygen and careful fluid supplementation.
- Severe: progression to ARDS usually results in mortality within 1–2 days.
- Overall survival rates: 80–100%. In electric shock, mortality up to 40% in dogs if severe, cats typically excellent prognosis.
- Long-term prognosis: excellent for recovered patients.

Synonyms

- Capillary leak syndrome
- Permeability edema

Abbreviations

ARDS: acute respiratory distress syndrome
BAL: bronchoalveolar lavage
CBC: complete blood count
DIC: disseminated intravascular coagulation
ECG: electrocardiogram

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ETL: endotracheal lavage
FRC: functional residual capacity
IM: intramuscular
IV: intravenous
PCV: packed cell volume
PEEP: positive end-expiratory pressure
PPV: positive pressure ventilation
PT: prothrombin time
PTT: partial thromboplastin time
SIRS: systemic inflammatory response syndrome
TTW: transtracheal wash

See Also

Acute Respiratory Distress Syndrome

Suggested Reading

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chapter 82

Pulmonary Thromboembolism

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DEFINITION/OVERVIEW

- Occlusion of the pulmonary arterial system with thrombi that may arise from distant sites.
- Usually refers to thrombi, but in some situations the term is also used for parasites, fat, or neoplastic cells.
- PTE is a secondary phenomenon; identification of a predisposing cause is beneficial in diagnosis and therapy.
- Prevalence in cats estimated at 0.06%; bimodal distribution with maximum at 0–2 and 10–12 years of age.
- Prevalence in dogs estimated at 0.9%.



ETIOLOGY/PATHOPHYSIOLOGY

- Stasis of blood, endothelial damage, and hypercoagulability (collectively referred to as Virchow's triad) predisposes the patient to the formation of a thrombus.
- Pulmonary vascular resistance increases due to mechanical obstruction of blood flow and vasoactive mediators released from the thrombus causing vasoconstriction of the vessels.
- PTE is considered a secondary condition.

Systems Affected

- Cardiovascular.
  - Jugular distension/pulsation.
  - Clinical signs of right-sided heart failure.
  - Pleural effusion.
  - Ascites.
  - Acute large PTE may result in low output cardiac failure and shock.

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Companion website: www.fiveminutevet.com/emergency

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- Respiratory.
  - Sudden-onset respiratory distress (58–96% of dogs, 55% of cats).
  - Tachypnea.
  - Coughing with or without hemoptysis.
  - Crackles or increased bronchial sounds.
- Possibly normal lung sounds.
- Abnormal gas exchange.
- Altered ventilator control.
- Hypoxia due to ventilation-perfusion mismatch, increased alveolar dead space, and intrapulmonary or intracardiac right-to-left shunting.
- Hypoxia can be made worse by edema due to overperfusion to nonthrombosed pulmonary tissue.



SIGNALMENT/HISTORY

- Middle-aged to older animals.
- No sex or breed predilection.

Risk Factors/Causes

- Reported primary causes in the dog include:
  - Immune-mediated diseases (such as immune-mediated hemolytic anemia)
  - Neoplasia
  - Hyperadrenocorticism
  - Glomerular disease and other protein-losing diseases
  - Amyloidosis
  - Recent surgery
  - Sepsis
  - Pancreatitis
  - Diabetes mellitus
  - Endogenous or exogenous corticosteroids
  - Vascular diseases like heartworm disease and vasculitis
  - Nephrotic syndrome
  - Iatrogenic causes such as indwelling catheters and transfusions
  - Hypothyroidism
  - Cardiac disease
  - Disseminated intravascular coagulation
  - Trauma
  - Endocarditis
  - Uncommonly, pulmonary fungal infections (blastomycosis).
- In cats, concurrent diseases that may serve as a primary cause include:
  - Neoplasia
  - Pancreatitis

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  - Nonhemolytic anemia
  - Hepatic lipidosis
  - Feline infectious peritonitis
  - Heart disease
  - Glomerulonephritis
  - Protein-losing enteropathy
  - Bacterial pneumonia
  - Encephalitis
  - Heartworm disease
  - Sepsis
  - Disseminated intravascular coagulation.
- Concurrent disease is not always present.

Historical Findings

- Reported historical complaints include:
  - Vomiting
  - Melena
  - Fever
  - Labored breathing
  - Lethargy
  - Altered mental state
  - Syncope.
- Some of these historical complaints are likely related to primary diseases that predispose to PTE.



CLINICAL FEATURES

- Labored breathing or tachypnea
- Tachycardia
- Restlessness
- Weakness
- Increased bronchial lung sounds and potential for crackles
- Cardiac murmurs (especially right-sided murmurs)
- Hepatosplenomegaly
- Cyanosis



DIFFERENTIAL DIAGNOSIS

- Airway obstruction
- Acute pneumonia
- Acute respiratory distress syndrome
- Other pulmonary parenchymal diseases
- Other causes of pulmonary hypertension

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- Acute congestive heart failure
- Noncardiogenic pulmonary edema
- Pleural space disease
- Asthma (bronchitis) in cats
- Pulmonary fibrosis



DIAGNOSTICS

- Diagnosis is difficult because symptoms are not specific for PTE and there is not a definitive diagnostic test.
- Lack of clinical suspicion contributes to the difficulty in diagnosis.
- Presence of predisposing factors may be the best indication to pursue diagnostics for PTE.
- Complete blood count.
  - Changes most likely due to primary diseases that put patient at risk for PTE.
  - Thrombocytopenia or thrombocytosis possible.
- Blood chemistry analysis.
  - Increased ALP
  - Increased ALT
  - Abnormal electrolyte values (i.e., hypokalemia, hyponatremia, and hypochloremia among the most common)
  - Hypoalbuminemia
  - Azotemia
- Urinalysis.
  - Changes most likely due to primary diseases that put patient at risk for PTE.
  - Dilute urine specific gravity.
  - Proteinuria.
  - Potential pyuria or bacteriuria.
- Coagulation profile FDPs: can be used to support diagnosis of disseminated intravascular coagulation.
  - Elevated FDPs may be seen but are considered insensitive indicators of thromboembolism.
  - Prolongation of the PT or aPTT.
- D-dimers are degradation products of cross-linked fibrin and specific for active coagulation and fibrinolysis.
  - False negatives are uncommon.
  - Positive results need to be considered with the clinical picture in mind.
  - Reported sensitivities and specificities at different levels include:
    - >500 ng/mL: 100% sensitive and 70% specific
    - >1000 ng/mL: 80% sensitive and 94% specific
    - >2000 ng/mL: 35% sensitive and 98.5% specific.
- Plasma antithrombin levels may be low.
- Thromboelastography to evaluate for hypercoagulable state.

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- Arterial blood gas.
  - Hypoxemia primarily due to ventilation-perfusion mismatch but physiologic shunting and increased dead space can contribute.
  - Hypocapnia.
  - Metabolic acidosis.
  - Increased alveolar-to-arterial gradient.
  - Response to supplemental oxygen variable.
- Thoracic radiography.
  - Normal (10–30% of dogs and 14% of cats with PTE on postmortem).
  - Possibly hypovascular lung regions.
  - Interstitial to alveolar lung infiltrates that do not correlate with the degree of respiratory distress.
  - Enlarged pulmonary arteries (especially centrally) with vascular tapering and oligemia.
  - Cardiomegaly and pleural effusion common in both dogs and cats.
- Echocardiography.
  - Potential to visualize thrombus if in proximal pulmonary arteries or right-sided heart.
  - Poorly contracting right ventricle.
  - Leftward shift of the septum during part of the cardiac cycle.
  - Pulmonary artery dilation.
  - High-velocity tricuspid regurgitation and pulmonic insufficiency jets. These can be used to estimate the pulmonary artery systolic and diastolic pulmonary arterial pressures respectively.
    - Pulmonary artery systolic pressure = 4 × (velocity of tricuspid regurgitation jet)^2 + estimated right atrial pressure.
    - Pulmonary artery diastolic pressure = 4 × (pulmonary insufficiency end-diastolic velocity)^2.
  - Diagnosis of pulmonary arterial hypertension is not analogous to PTE; it may increase index of suspicion when combined with other diagnostic tests.
- Pulmonary perfusion scintigraphy is accepted as safe and sensitive but only available at some referral institutions.
  - Involves intravenous injection of technetium-labeled macroaggregated albumin.
  - Perfusion deficits can also occur in nonventilated regions with reflex vasoconstriction.
- Ventilation-perfusion scan is a perfusion scan combined with a ventilation scan using technetium-labeled radioaerosol.
  - Increases sensitivity.
  - Uncommonly performed in veterinary medicine.
  - Because it cannot differentiate all primary pulmonary parenchymal diseases from PTE, it is frequently not definitive.
- Pulmonary angiography.
  - Most sensitive and specific test but uncommonly performed due to need for general anesthesia in veterinary patients.
  - Nonionic contrast should be used to minimize systemic hypotension.

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  - Patients with pulmonary hypertension are at increased risk of complications due to the procedure.
  - Diagnosis of PTE includes intraluminal filling defect in the pulmonary artery or abrupt vessel occlusion and visualization of the trailing edge of the thromboembolism.
- Magnetic resonance imaging with angiography is commonly used in human medicine but the need for general anesthesia limits its use in veterinary medicine.
- Recent report of a contrast-enhanced multidetector computed tomography study being used in a dog to definitively diagnose PTEs without anesthesia.

Pathologic Findings

- Pulmonary emboli visible.
  - Pleural effusion
  - Concurrent pulmonary pathology
  - Bronchopneumonia
  - Pulmonary edema
  - Pulmonary neoplasia
  - Interstitial fibrosis
  - Pulmonary calcification
  - Emphysema
  - Hyaline membrane disease
- Most common primary postmortem diagnoses include immune-mediated hemolytic anemia, sepsis, and amyloidosis.
- Most dogs have multiple disease processes.



THERAPEUTICS

- If primary cause can be identified, treatment should be instituted.
- Symptomatic respiratory support.
  - Supplemental oxygen.
  - Positive end-expiratory pressure ventilation may be required.
- Bronchodilator like theophylline.
  - May induce pulmonary vasodilation and improve diaphragmatic contractility.
- Anticoagulant therapy.
  - Unfractionated heparin: after baseline PT and aPTT and ACT, initial intravenous dose of 100–300 U/kg given followed by 100–300 U/kg SQ or IV every 6–8 hours. The dose is adjusted to maintain the aPTT at 1.5–2 times normal.
  - Low molecular weight heparin.
    - More expensive than unfractionated heparin but cost is theoretically balanced by not needing to evaluate coagulation times
    - Dalteparin dose (dog and cat): 100–150 IU/kg SQ every 12–24 hours.
    - Enoxaparin dose (dog and cat): 1 mg/kg SQ every 12–24 hours.

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- Warfarin.
  - Coagulation times must be closely monitored and are more difficult to regulate.
  - Uncommonly used.
  - If used, heparin must be administered during the initial hypercoagulable period (usually 5–7 days) due to warfarin effects on protein C.
  - Warfarin doses.
    - Dog: 0.1–0.2 mg/kg PO every 24 hours
    - Cat: 0.1–0.2 mg PO every 24 hours
- Antiplatelet drugs (i.e., aspirin, clopidogrel, ticlodipine).
  - Used primarily for prevention in patients considered at high risk.
  - Clopidogrel doses.
    - Cat: 18.75 mg once daily
    - Dog: 1–2 mg/kg/day PO
  - Aspirin doses.
    - Dog: 0.5 mg/kg PO every 12–24 hours
    - Cat: 5–81 mg PO every 72 hours
- Cautious use of parenteral fluids.
  - Hypotension may be present because of pulmonary arterial obstruction and impedance to left heart filling and may be aided by fluids.
  - Aggressive fluid therapy can further increase right-sided heart filling, displacing the intraventricular septum to the left, further impeding left-sided heart filling and compromising cardiac output.
- Thrombolytic therapy (rt-PA, streptokinase).
  - Not commonly used and reserved for early use.
  - Excessive bleeding and not uncommonly fatal bleeding is possible.
  - rt-PA activates bound plasminogen, making it more "clot specific."
    - Dog rt-PA dose: 1 mg/kg IV every 60 minutes for a total of 10 doses
    - Cat rt-PA dose: 0.25–1 mg/kg per hour for a total dose of 1–10 mg/kg IV
  - Streptokinase is not fibrin dependent and therefore leads to a systemic lytic state.
    - Dog streptokinase dose: 90 000 IU loading dose over 30 minutes followed by CRI of 45 000 IU/hour for 7–12 hours
    - Alternatively, a loading dose of 15 000–18 000 IU/kg can be used followed by same CRI protocol.
- Surgical thrombectomy could be considered but is rarely performed.



COMMENTS

Client Education

- Most client information provided will be related to primary disease.
- For patients on anticoagulant therapy, the owner should monitor for bruising or bleeding.

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Patient Monitoring

- Monitor patient's response to therapy (respiratory rate and effort and serial blood gas analysis).
- Monitor coagulation times (aPTT and PT) carefully.

Prevention/Avoidance

- Patients with a primary condition that increases their risk for PTE can be placed on prophylactic anticoagulant therapy.

Possible Complications

- Use of thrombolytic agents or anticoagulants increases the patient's chances of hemorrhage and should be monitored closely.

Expected Course and Prognosis

- Variable prognosis based on size of thrombus, underlying cause, and time course of clinical recognition and therapy.
- Poor prognosis with large thrombus in central pulmonary artery.

Abbreviations

ACT: activated clotting time
ALP: alkaline phosphatase
ALT: alanine transaminase
aPTT: activated partial thromboplastin time
CBC: complete blood count
CRI: constant rate infusion
FDP: fibrin degradation products
IV: intravenous
PO: by mouth (per os)
PT: prothrombin time
rt-PA: recombinant tissue plasminogen activator
SQ: subcutaneous

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chapter 83

Pyometra

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DEFINITION/OVERVIEW

- Cystic endometrial hyperplasia: hormonally mediated, progressive pathologic change in the uterine lining.
- Pyometra: secondary to cystic endometrial hyperplasia; develops when bacterial invasion of the abnormal endometrium leads to intraluminal accumulation of purulent exudate.

ETIOLOGY/PATHOPHYSIOLOGY

- Normal cycling bitches: 2-month diestrus, with ovarian secretion of progesterone after every estrus.
- Repeated exposure of the endometrium to high concentrations of estrogen followed by high concentrations of progesterone without pregnancy; leads to cystic endometrial hyperplasia.
- Bacteria: uterine secretions provide excellent media for growth; ascend from the vagina through the partially open cervix during proestrus and estrus; normal vaginal flora; *Escherichia coli* most common isolate.
- Cats: may be the result of estrogen at estrus followed by a progestational phase, caused by induction of ovulation by coitus, spontaneous ovulation, or other (as yet undefined) stimuli.
- Recent studies suggest that subclinical bacterial infections or foreign bodies may trigger pyometra in the absence of classic CEH.

Systems Affected

- Reproductive: accumulation of purulent material in uterine horns.
- Renal/urologic: bacterial toxins may impair renal function due to impaired ADH activity and lead to isosthenuric or hypothenuric urine.
- Hemic/lymphatic/immune: leukocytosis or leukopenia may be present.
- Hepatobiliary: extrahepatic cholestasis with severe sepsis.

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Companion website: www.fiveminutevet.com/emergency

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SIGNALMENT/HISTORY

- Older bitches and queens (younger than 6 years of age) are at greater risk.
- Bitches with short interestrous intervals are at greater risk.
- Young animals: if treated with exogenous estrogen or progestagen.
- Pyometra may be more prevalent, or prevalent at younger ages, in some family lines.

Risk Factors/Causes

- Administration of estrogenic or progestagenic medications to intact females.
- Dogs: usually diagnosed 1–12 weeks after estrus.
- Cats: onset relative to estrus more variable.
- Pyometra of the uterine stump (stump pyometra) in spayed animals may develop any time after ovariohysterectomy if exposed to estrogen from ovarian remnant or exogenous source.

Historical Findings

- Open cervix: purulent or bloody vulvar discharge
- Closed cervix: no vulvar discharge
- Increased drinking and urinating
- Depression, lethargy, and anorexia
- Vomiting and diarrhea in some cases

CLINICAL FEATURES

- Uterus: palpably large; careful palpation may allow determination of size; overly aggressive palpation may induce rupture; with open cervix may not be palpably large.
- Vulvar discharge: depends on cervical patency; sanguineous to mucopurulent (Fig. 83.1).
- Depression and lethargy.
- Anorexia.
- Polyuria and polydipsia.
- Vomiting.
- Abdominal distension.
- Fever or hypothermia.

DIFFERENTIAL DIAGNOSIS

- Pregnancy.
- Other causes of polyuria and polydipsia: diabetes mellitus; hyperadrenocorticism; primary renal disease.

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Fig. 83.1. Purulent vaginal discharge in a dog with an open pyometra.

- Severe vaginal disease: vaginitis, foreign body, bleeding mass.
- Hydrometra (serous intrauterine discharge); mucometra (mucoid intrauterine discharge); and hematometra (hemorrhagic intrauterine discharge).

DIAGNOSTICS

- CBC: immature neutrophilia seen in 74% of cases; leukopenia can occur with sepsis; normal CBC also can occur.
- Mild, normocytic, normochromic anemia chemistry panel: hyperglobulinemia and hyperproteinemia, azotemia with renal impairment.
- ALT and ALP both high with septicemia or severe dehydration.
- Electrolyte disturbances depend on clinical course.
- Urinalysis: sample collected by catheterization of the urinary bladder (least traumatic and most diagnostically accurate): evaluate for secondary UTI, impairment of renal function; cystocentesis is contraindicated if pyometra is suspected, due to risk of puncturing enlarged uterus and wicking bacteria into abdominal cavity.
- Cytologic examination of vulvar discharge: degenerative polymorphonuclear cells and bacteria; may be normal with closed cervix.
- Bacterial culture and sensitivity test of anterior vaginal sample, useful in determining appropriate antibiotic use.
- Radiography detects a large uterus (cannot differentiate from pregnancy prior to 45 days of gestation).

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- Ultrasonography best diagnostic tool for diagnosis of pyometra and selection of candidates for medical therapy.
  - Assess size of uterus and extent of cystic endometrial hyperplasia; nature of uterine contents.
- Rule out pregnancy 20–24 days after ovulation.
- Normal uterine wall: not visible as a distinct entity.
- Pyometra or cystic endometrial hyperplasia associated with a thickened uterine wall (with or without hypoechoic cystic areas) and intraluminal fluid.
- Pyometra may occur with pregnancy in dogs (rare).
- Vaginoscopy indicated only in dogs with purulent vulvar discharge and no apparent uterine enlargement; allows determination of site of origin of the vulvar discharge; not possible in cats.

Pathologic Findings

- Endometrium (dogs and cats): described as cobblestone (either condition).
- Cystic endometrial surface: covered by malodorous, mucopurulent exudate; thickened because of increased endometrial gland size and cystic gland distension.



THERAPEUTICS

- Determine if patient is desired for future breeding by owner and, if so, if she is a candidate for medical therapy (not septic or systemically affected, no free fluid in abdomen, uterine wall not thin, patient under the age of 6 years, owner willing to breed on next cycle).
- If patient is not planned for future breeding, correct dehydration, support renal function, start antibiotic therapy, and perform ovariohysterectomy.
- If patient is to be treated medically, start antibiotics pending culture results, and begin therapy with prostaglandin $F_{2\alpha}$ to lyse corpora lutea and cause myometrial contraction.

Drugs of Choice

Antibiotics

- Empirical, pending results of bacterial culture and sensitivity test.
- Use in all patients with pyometra.
  - Common choice is enrofloxacin (5–10 mg/kg PO every 24 hours).
  - Administration continued for 3–4 weeks.

Prostaglandin $PGF_{2\alpha}$

- Doses listed below for native compound only; analogues not recommended.
- Cats: 0.1–0.5 mg/kg SQ every 8–12 hours for 2–5 days until the size of the uterus nears normal.

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- Dogs: 0.05–0.25 mg/kg SQ every 8–12 hours for 3–7 days until no intrauterine fluid visible by ultrasound.
- Less conventional protocols also exist for lower doses given five times daily (10 µg/kg five times a day for 1 day, then 20 µg/kg five times daily for 1 day and then 50 µg/kg five times daily).

Cloprostenol

- Dogs: 1 µg/kg SQ daily for 7–14 days.

Miscellaneous

- Aglepristone: 10 mg/kg SQ days 1, 2, and 8; efficacy enhanced with concurrent treatment with prostaglandin; not available in United States.
- Cabergoline: 5 µg/kg PO daily for 7–14 days; used currently with prostaglandin to increase speed of luteolysis and cervical opening.
- Misoprostol: 1–3 µg/kg given intravaginally concurrently with prostaglandin $F_{2\alpha}$ to assist in opening cervix if needed.

Precautions/Interactions

- $PGF_{2\alpha}$ and cloprostenol with closed-cervix pyometra cause strong myometrial contractions.
- May cause uterine rupture or force purulent exudate through the oviducts, causing secondary peritonitis.
- $PGF_{2\alpha}$ and cloprostenol in a valuable breeding animal; always rule out pregnancy before administration.
- $PGF_{2\alpha}$ and cloprostenol not approved for use in dogs and cats.
- Adverse effects of prostaglandins: referable to contraction of smooth muscle; include hypersalivation; emesis; defecation; intense grooming of the flanks and vulva (cats); appear within minutes of injection; subside within 30–60 minutes; severity diminishes throughout the treatment regimen; may be diminished by diluting the drug with an equal volume of sterile saline before subcutaneous injection and by walking dogs for 20–30 minutes after injection; antiemetics can also be utilized.

Diet

- Timing of feeding should avoid the hour before and the hour after $PGF_{2\alpha}$ administration to prevent vomiting of meal.

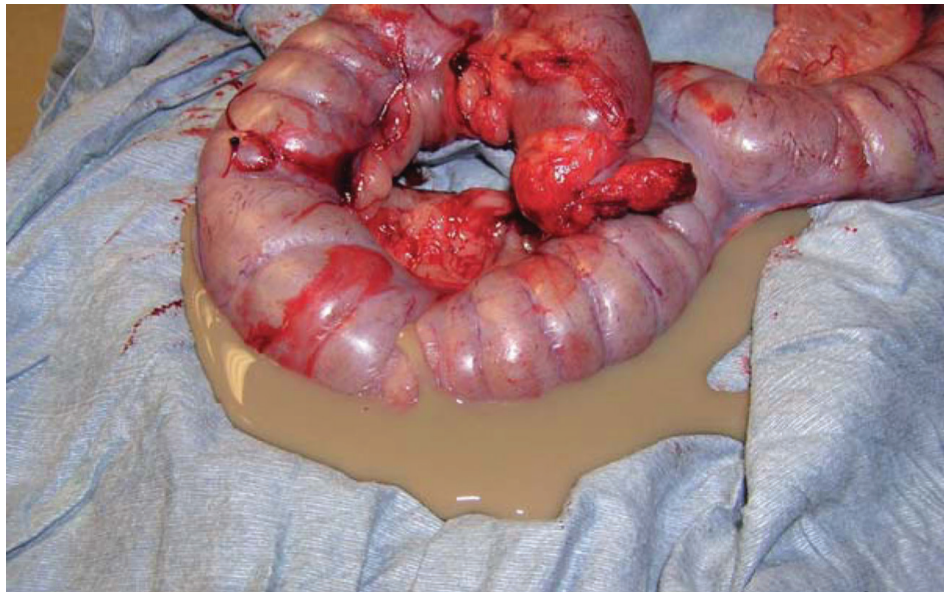
Activity

- Patients with pyometra typically are not interested in exercise and should be allowed to rest, apart from the walking post $PGF_{2\alpha}$ administration.

Surgical Considerations

- Pyometra (open and closed cervix): ovariohysterectomy preferred treatment; chronic progressive disease (Fig. 83.2).

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■ **Fig. 83.2.** Removal of pus-filled uterus at the time of ovariohysterectomy.

- Closed-cervix pyometra: use caution during ovariohysterectomy; enlarged uterus may be friable and prone to rupture.
- Uterine rupture or leakage of purulent material from the uterine stump: repeated lavage of the peritoneal cavity with sterile saline.
- Adequate fluid is necessary to maintain blood pressure and support renal function.
- Patients may have dramatic temperature drop after removal of progesterone source; exogenous heat support and monitoring of body temperature are important.

COMMENT

Client Education

- Inform client that ovariohysterectomy is the preferred treatment.
- Recommend medical treatment only for valuable breeding animals that are not azotemic and have an open cervix; warn client that except when breeding is planned, nonprogestational, estrus-suppressing drugs (mibolerone) should be given for life.
- Warn client that medical treatment of closed-cervix pyometra can be associated with uterine rupture and peritonitis.
- Inform client that medical treatment does not cure underlying cystic endometrial hyperplasia in patients with either open- or closed-cervix pyometra but may enable some affected bitches and queens to reproduce.
- Patients treated medically can cycle sooner than expected due to shortening of the luteal phase of the cycle.

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Patient Monitoring

- Medically treated patients should be monitored with ultrasound to gauge their progress, initially after 3–5 days of treatment, then every 2 days until resolution.
- After discharge, patients should be rechecked in 2 weeks for any vaginal discharge and overall health; ultrasound can be rechecked at this time.

Prevention/Avoidance

- Patient should be bred on next cycle and spayed after desired litter(s).
- If breeding is not planned, cycle should be suppressed in the bitch (no safe alternatives for this in the queen).

Possible Complications

- Infertility.
- Uterine rupture.
    - Bitch may enter estrus sooner after treatment than anticipated if medical treatment induced premature luteolysis.

Expected Course and Prognosis

- Dogs, medical treatment: underlying cystic endometrial hyperplasia still exists; predisposed to recurrence; breed patient to desired stud dogs in a timely manner; recommend ovariohysterectomy as soon as breeding life is over; use of subfertile stud dogs not recommended, overall success in future reproduction 50–75%.
- Cats, medical treatment, good success.
- Surgical treatment, dogs and cats, if not septic and stable, good success; patients who are septic and critically ill, with significant renal impairment, prognosis is fair to guarded.



COMMENTS

- Typically patients treated medically for pyometra still have underlying changes in the endometrium and should have estrus suppressed with anabolic steroid (mibolerone, from compounding pharmacy) until breeding is desired.
- Cycling will resume days to months after cessation of mibolerone (average 70 days).
- No safe estrus suppression drug is available for queens.

Abbreviations

ADH: antidiuretic hormone
ALP: alkaline phosphatase

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ALT: alanine transaminase
CBC: complete blood count
CEH: cystic endometrial hyperplasia
$PGF_{2\alpha}$: prostaglandin $F_{2\alpha}$
PO: by mouth (per os)
SQ: subcutaneous
UTI: urinary tract infection

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chapter 84

Pyothorax

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DEFINITION/OVERVIEW

- Pyothorax is characterized by the accumulation of a septic purulent effusion within the pleural space.
- Pyothorax occurs in both dogs and cats, but occurs more commonly in cats.

ETIOLOGY/PATHOPHYSIOLOGY

- The most common cause of pyothorax in cats is bite wounds to the chest from other cats, but environmental contamination from penetrating thoracic injuries can also occur. Additional causes include rupture or perforation of the esophagus, trachea, or bronchi, migrating foreign bodies (grass awns) or lung parasites, bacterial pneumonia leading to lung abscesses and rupture, or iatrogenic contamination from thoracocentesis or thoracotomy.
- In dogs, pyothorax may result from migrating foreign bodies such as grass awns, penetrating wounds to the chest, neck, or mediastinum, esophageal perforations, lung parasites, bacterial pneumonia with abscessation and rupture, hematogenous or lymphatic spread from other septic foci, spread from cervical, lumbar, or sternal diskospondylitis, neoplasia with secondary abscessation, and iatrogenic contamination from thoracocentesis or thoracotomy.
- The most common bacterial isolates from cats with pyothorax are the anaerobes *Peptostreptococcus* spp., *Bacteroides* spp., *Fusobacterium* spp., *Porphyromonas* spp., *Prevotella* spp., and the aerobes *Pasteurella* spp. and *Actinomyces* spp.
- The most common bacterial isolates from dogs are the anaerobes *Peptostreptococcus* spp., *Bacteroides* spp., *Fusobacterium* spp., and *Porphyromonas* spp., and the aerobes *Actinomyces* spp., *Pasteurella* spp., *Escherichia coli*, and *Streptococcus* spp. These bacterial isolates are similar to those identified from cats with the addition of the enteric *E. coli* and *Streptococcus* spp.
- Mixed populations of bacteria are often cultured from cats and dogs.

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- Once bacterial infection is present in the pleural space, inflammatory mediators are released, causing increased permeability of the endothelial lining of the pleural capillaries, impairment of lymphatic outflow and accumulation of fluid, protein, and inflammatory cells. Increased protein concentration in the pleural fluid causes an increased oncotic pressure, favoring additional fluid movement out of the capillaries and into the pleural space.

Systems Affected

- Respiratory
- Hemic/lymphatic/immune
- Cardiovascular
- Endocrine/metabolic



SIGNALMENT/HISTORY

Risk Factors/Causes

- Cats from multicat households.
- The hunting and working breeds of dogs, possibly caused by aspiration and subsequent migration of grass awns.

Historical Findings

- Presentation is often delayed for weeks to months after inciting incident.
- Penetrating thoracic injuries are often healed by the time respiratory compromise develops, making diagnosis of the underlying cause difficult.
- Dogs: exercise intolerance, respiratory difficulty or distress, reluctance to lie down, anorexia, lethargy, and cough are common.
- Cats: respiratory difficulty or distress, depression, lethargy, pallor, anorexia, and pain.



CLINICAL FEATURES

- Clinical presentation can vary widely, from mild respiratory signs to collapse from severe septic shock.
- Findings usually include varying degrees of respiratory distress: increased respiratory rate and effort, dull lung sounds ventrally, harsh sounds dorsally and orthopnea.
- Signs of septic shock, such as hyperthermia or hypothermia, tachycardia (or in cats, bradycardia), injected or pale mucous membranes, and bounding or weak pulses.
- Recent weight loss and a poor body condition may be noted.
- Additional findings may include depression and dehydration.

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DIFFERENTIAL DIAGNOSIS

- Other causes of pleural effusion: chylothorax, heart failure, hemothorax, neoplasia, hypoproteinemia, or FIP (cats).
- Other pleural space disease: diaphragmatic hernia or neoplasia.
- Pulmonary disease: pneumonia or pulmonary abscesses.

DIAGNOSTICS

Diagnostic Procedures

- Thoracocentesis: indicated if there is a suspicion of pleural effusion, is both diagnostic and therapeutic. It is often a thick, opaque and foul-smelling fluid and may be flocculent. Samples should be evaluated through cytology, cell counts, total protein, and aerobic/anaerobic culture.
- Fluid analysis: exudate, protein >3 g/dL, nucleated cell count >7 × 10^9/L.
- Cytology: neutrophils, often degenerative with signs of toxicity and bacteria, both intracellular and extracellular. Absence of bacteria does not rule out pyothorax, especially if antibiotics have previously been administered. "Sulfur granules," which are really macroaggregates of bacteria, may also be seen. If the pyothorax is chronic, there may be increasing numbers of macrophages and plasma cells.
- Aerobic and anaerobic culture and sensitivity testing.
- Fungal culture depending on geographic location.

Complete Blood Count/Biochemistry

- CBC: usually marked leukocytosis, with or without an increased number of band neutrophils and toxic change, but in severe cases a leukopenia and degenerative left shift may be found. Anemia may be present if the pyothorax has been chronic.
- Biochemical results may be normal or may show changes consistent with sepsis, including hypoalbuminemia, hypoglycemia, and increased ALT and total bilirubin. Changes consistent with dehydration and hemoconcentration are seen in some patients.

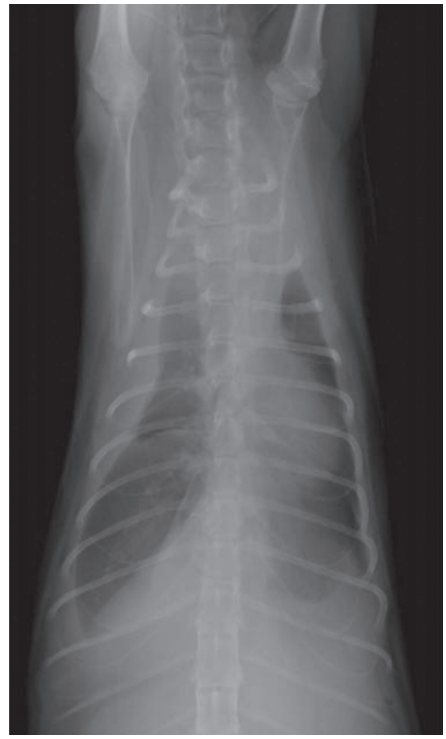
Other Laboratory Tests

- Retrovirus testing (cats): FeLV- or FIV-positive status may complicate treatment and increase risk of recurrence.

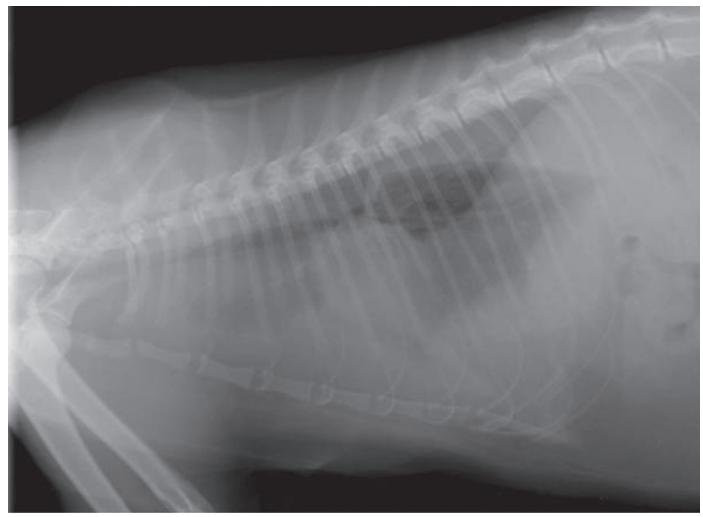
Imaging

- Thoracic radiography: useful in confirming the presence of pleural effusion, but patients in respiratory distress may not tolerate radiographs. More helpful in searching for underlying causes once pleural effusion has been removed. Useful to determine if disease is bilateral or unilateral (Figs 84.1, 84.2).

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■ **Fig. 84.1.** DV radiograph of a cat with pyothorax showing bilateral pleural effusion. Due to the presence of the effusion, the pulmonary parenchyma cannot be well evaluated for the presence of lung abscesses, foreign bodies, masses, or other underlying pathology.



■ **Fig. 84.2.** Lateral thoracic radiograph of a cat with pyothorax showing pleural effusion. Due to the presence of the effusion, the pulmonary parenchyma cannot be well evaluated for the presence of lung abscesses, foreign bodies, masses, or other underlying pathology.

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- Thoracic ultrasonography and CT: can identify abscesses or masses in pulmonary parenchyma or mediastinum.
- Thoracoscopy: can be used to explore thoracic cavity. May be helpful in some cases to flush thoracic cavity, determine cause, and remove affected lung lobes.

Pathologic Findings

- Pleuritis, pleuropneumonia, or pneumonia.
- Underlying etiology including lung lobe abscesses, puncture wounds into thoracic cavity, or foreign bodies may occasionally be seen.

THERAPEUTICS

- Initial stabilization: oxygen therapy, minimal handling and stress, thoracocentesis, intravenous catheter and intravenous fluids, including shock boluses if indicated and intravenous broad-spectrum antibiotics as soon as possible.
- Thoracic drainage: mainstay of therapy. Can be accomplished by intermittent thoracocentesis or by placement of thoracostomy tubes, with intermittent aspiration or continuous drainage. Thoracostomy tubes are preferred because they allow for more complete drainage of the thoracic cavity. Bilateral thoracostomy tubes are recommended unless effusion is unilateral (Fig. 84.3).



Fig. 84.3. Cat with a chest tube in place to allow for frequent intermittent drainage of pleural effusion from a pyothorax.

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- Lavage of the thoracic cavity with warm physiological saline or other balanced electrolyte solutions at 20 mL/kg, instilled over 10–15 minutes every 6–24 hours has been described. Not recommended unless effusion is too thick to aspirate through chest tube due to risk of nosocomial infection and potential inability to retrieve fluid. When tubes are first placed, a single lavage can be safely performed if fluid is too thick to drain.
- Fluid therapy: maintenance rates or higher if pleural effusion is still being produced. Essential to keep patient hydrated so effusion can be aspirated via thoracostomy tubes or thoracocentesis. May need to consider colloids if patient is very hypoproteinemic.

Drugs of Choice

Antimicrobials

- Broad-spectrum antimicrobials: gram-positive, gram-negative, and anaerobic coverage until culture and sensitivity results are available. Broad spectrum is essential due to frequency of infection with mixed bacterial population.
- Administer parenteral antibiotics while patient is critical and then switch to oral once stabilized.
- Adjust antibiotic therapy once culture and sensitivity results are available.
- Continue for several months following discharge from the hospital.
- Clindamycin 11 mg/kg IV every 8–12 hours and enrofloxacin 10–15 mg/kg IV every 24 hours are excellent initial choices for dogs. Can use ampicillin 22 mg/kg IV every 8 hours with enrofloxacin instead of clindamycin. Metronidazole 15 mg/kg IV every 12 hours can also be added.
- Clindamycin 11 mg/kg IV every 8–12 hours and cefotaxime 15 mg/kg IV every 6 hours initially for cats. Other options include ticarcillin/clavulanate 50 mg/kg IV every 6 hours or ampicillin 22 mg/kg IV every 8 hours with metronidazole 15 mg/kg IV every 12 hours.
- Long-term antibiotic therapy depends on culture and sensitivity testing but potentiated penicillins such as amoxicillin/clavulanate 22 mg/kg every 8–12 hours are often effective.

Analgesics

- Should be administered post thoracostomy tube placement and while tubes are in place or post thoracotomy.
- Intrapleural bupivicaine (1.5 mg/kg every 6–8 hours) via the thoracostomy tube may provide some analgesia; usually not sufficient alone.
- Opiates such as methadone 0.1–0.2 mg/kg IV every 4–6 hours, hydromorphone 0.1 mg/kg IV every 6–8 hours or fentanyl CRI 2–5 µg/kg per hour are often effective.

Precautions/Interactions

- Care with choice of antibiotics; aminoglycosides may cause acute renal failure, especially in patients that are not properly hydrated prior to initiating them. Potentiated sulfas may cause KCS, ITP, polyarthropathy, and other immune-mediated disease.

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Activity

- Light activity (short walks) while hospitalized should be encouraged to prevent atelectasis.
- Restriction of activity to moderate level when discharged from hospital until resolution of radiographic signs.

Surgical Considerations

Cats

- Medical treatment is usually successful.
- Surgery is indicated if not responding to medical therapy after a week or so, or if surgical lesion (abscessed lung lobe or foreign body) is identified.

Dogs

- In one study, dogs had improved outcome with surgical intervention. Surgery can be considered without first trying medical treatment.
- More recent studies have also shown excellent outcome in dogs treated with medical management.

Surgical Treatment

- Requires a complete exploratory thoracotomy to identify and remove any necrotic tissue or foreign material, as well as complete lavage of the thoracic cavity (Fig. 84.4).

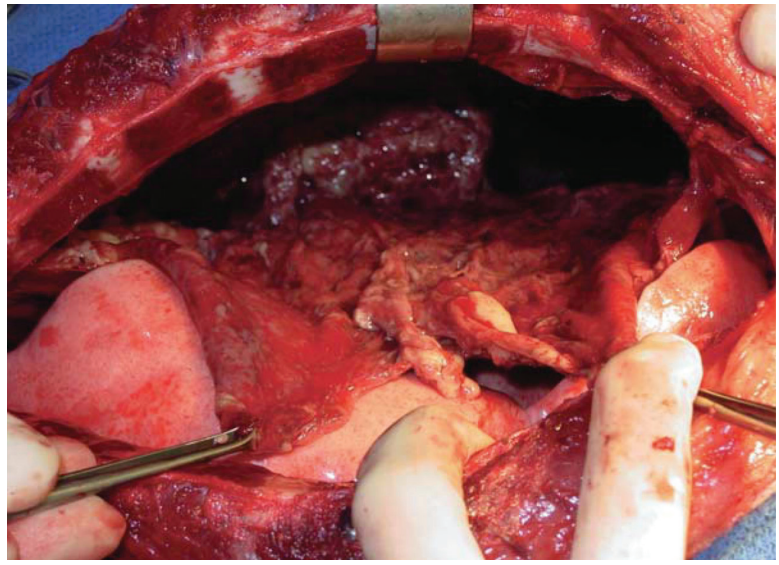


Fig. 84.4. Intraoperative picture of a 4-year-old intact male Weimaraner with pyothorax showing a very thickened, abnormal mediastinum, normal lung in the near field (*to the left of the forceps*), and very diseased lung in the far field (*just beneath the retractor*). A left pneumonectomy, partial excision and debridement of the mediastinum, and lavage of the thoracic cavity were performed. The underlying etiology of the pyothorax was not identified.

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- Samples for aerobic and anaerobic culture can be obtained at surgery.
- Options include median or sternotomy or lateral thoracotomy. Median sternotomy is preferred because it allows for full evaluation of the right and left hemithoraces.
- Video-assisted thoracic surgery is also increasingly utilized in dogs, and is often the first choice of therapy for humans.

Postoperative Care

- Patients require intensive care and monitoring, especially for the first 24–48 hours after surgery.
- Septic shock and SIRS may develop.
- Fluid therapy in the postoperative phase is very important and often consists of crystalloids, colloids, and blood products.
- Vasopressors may be needed to treat refractory hypotension once volume expansion has been accomplished.
- Respiratory, acid–base, and electrolyte abnormalities should be evaluated and treated, as needed.
- Patients are at risk for the development of DIC and need to have their coagulation parameters monitored closely.
- Frequent evaluation of the CBC and cytology of the pleural effusion should be done.



COMMENTS

Client Education

- Clients must be prepared for cost of treatment and long-term follow-up care.

Patient Monitoring

- In hospital: requires intensive care monitoring including respiratory rate/effort, heart rate, blood pressure, fluid ins/outs, temperature, oxygenation, pain management, nutrition monitoring, electrolytes, and acid–base balance.
- CBC and thoracic radiographs should be monitored every few days initially, then at increasing intervals.
- Once discharged, recheck examinations every few weeks for CBC and thoracic radiographs.
- Antibiotics should be continued until several weeks after resolution of radiographic changes.

Prevention/Avoidance

- Cats from single-cat households have reduced risk.
- Hunting may place dogs at increased risk.

Possible Complications

- Sepsis, septic shock, SIRS, death.
- Recurrence of pyothorax, especially in dogs treated medically.

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- Complications from thoracostomy tube including pneumothorax, hemothorax, or nosocomial infection.
- Fibrosing pleuritis (uncommon).

Expected Course and Prognosis

- With appropriate therapy (antibiotics and medical or surgical management depending on species), prognosis is fair to good.
- Outpatient therapy with antibiotics and intermittent thoracocentesis has a poor prognosis.
- Recurrence rate is low for cats treated medically and for dogs treated surgically.
- Many patients may be euthanized due to owners' financial constraints.

Synonyms

- Empyema
- Purulent pleuritis

Abbreviations

ALT: alanine transaminase
CBC: complete blood count
CRI: constant rate infusion
DIC: disseminated intravascular coagulation
FeLV: feline leukemia virus
FIP: feline infectious peritonitis
FIV: feline immunodeficiency virus
IM: intramuscular
ITP: idiopathic thrombocytopenic purpura
IV: intravenous
KCS: keratoconjunctivitis sicca
SIRS: systemic inflammatory response syndrome

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Author: Lori. S. Waddell, DVM

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chapter 85

Raisin and Grape Toxicity

DEFINITION/OVERVIEW

- Syndrome of vomiting, diarrhea, and ARF secondary to ingestion of raisins or grapes.

ETIOLOGY/PATHOPHYSIOLOGY

- Mechanism of ARF unknown.
- Acute tubular necrosis, possibly secondary to ischemic insult to kidneys.
- Degeneration of glomerular basement membrane.
- Tubular obstruction with proteinaceous debris and renal casts.
- Oliguric to anuric renal failure.

Systems Affected

- Gastrointestinal: vomiting and diarrhea, often with visible remnants of raisins and/or grapes (Fig. 85.1).
- Renal: ARF, isosthenuria to hyposthenuria, oliguria to anuria, azotemia, or uremia.
- Metabolic: metabolic acidosis, hyper- or hypokalemia, hyperphosphatemia, or hypercalcemia.
- Cardiovascular: atrial standstill possible with severe hyperkalemia.

SIGNALMENT/HISTORY

- Primarily dogs, but anecdotally reported in cats.
- Vomiting and diarrhea within 24 hours of ingestion of raisins or grapes.
- Lethargy.
- Oliguria that can progress to anuria.

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■ **Fig. 85.1.** Induction of emesis in a dog that ingested chocolate-covered raisins.

Historical Findings

- Witnessed ingestion of raisins or grapes.
- Findings of chewed boxes of raisins.
- Visible grapes or raisins in vomitus or diarrhea.
- Lethargy.
- Inappetence.
- Diarrhea.
- Vomiting.

Physical Examination

- Lethargy.
- Dehydration.
- Vomitus staining on muzzle.
- Uremic ulceration in oral cavity.
- Physical examination may be normal in early stages of ingestion.

Risk Factors/Causes

- Access to grapes or raisins.
- Free roaming.
- Preexisting renal disease.
- Preexisting vomiting, diarrhea, dehydration, or hypotension.
- Ingestion of even small quantities of raisins or grapes.

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- Toxic principle remains unknown.
- Ethylene glycol, herbicides, chlorinated hydrocarbon, carbamates, organophosphates, mycotoxins, and ochratoxin A all negative when tested.



CLINICAL FEATURES

- Vomiting
- Diarrhea
- Lethargy
- Anorexia
- Abdominal pain
- Ataxia
- Weakness
- Decreased urination



DIFFERENTIAL DIAGNOSIS

- Leptospirosis.
- Pyelonephritis.
- Urethral or ureteral obstruction.
- Toxins.
  - Ethylene glycol
  - Cholecalciferol rodenticide
  - Melamine
  - Aminoglycoside antibiotics
  - NSAID
  - Easter lily
- GI disease.
  - GI obstruction
  - Hemorrhagic gastroenteritis
  - Dietary indiscretion
  - Pancreatitis
  - Viral or bacterial enteritis
- Hypoadrenocorticism.
- Perform a thorough history and physical examination to determine risk and potential for exposure to grapes and/or raisins and to rule out other causes of acute renal failure.



DIAGNOSTICS

Complete Blood Count/Biochemistry/Urinalysis

- See Tables 85.1, 85.2.

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TABLE 85.1. Biochemical or complete blood count abnormalities in affected patients.

Abnormality	Percentage of affected patients
Azotemia (elevated blood urea nitrogen and creatinine)	100
Hypercalcemia	63
Hyperphosphatemia	90
Elevated calcium × phosphorus product	95
Hyperkalemia	45
Hyponatremia	33
Hypochloremia	33
Elevated anion gap	69
Elevated alkaline phosphatase	24
Elevated alanine aminotransferase	65
Thrombocytopenia	27
Anemia	41
Elevated lipase	41

TABLE 85.2. Urinalysis abnormalities and percentage of affected patients.

Abnormality	Percentage of Affected Patients (%)
Isosthenuria	41
Hyposthenuria	21
Proteinuria	56
Glucosuria	50
Microscopic hematuria	58
White blood cells	67
Crystalluria	33
Cylindruria	21

Other Tests

- Ethylene glycol negative.
- Leptospirosis urine PCR and serum titers negative.

Imaging

- Radiographs.
  - Microcardia and small caudal vena cava on thoracic radiographs.
  - Possibly pulmonary edema as anuria progresses despite intravenous fluid therapy.

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- Ultrasound.
  - Hyperechoic kidneys
  - Renomegaly
  - Perirenal fluid
  - Renal pelvis dilation
  - Hyperechoic mesentery near pancreas
  - Hypoechoic pancreas



THERAPEUTICS

- Induction of emesis within several hours of ingestion, followed by activated charcoal: 0.03–0.04 mg/kg IV apomorphine.
- Intravenous crystalloid fluids.
  - Calculate dehydration deficit, and replace over 24 hours.
  - Intravenous fluid diuresis (2–3 times maintenance) shortly after ingestion.
- Control nausea and vomiting.
- Maintain blood pressure and normotension.
- Stimulate urine output.
- Peritoneal or hemodialysis in anuric patients

Drugs of Choice

Diuretics

- Furosemide: 2–4 mg/kg IV at 30-minute intervals, or 1 mg/kg per hour IV CRI.
- Mannitol.
  - 0.5–1 g/kg IV slowly over 20 minutes.
  - Use caution in overhydrated patients, or those with pulmonary edema, tubular obstruction, or known cardiac disease.

Calcium Channel Blockade

- Diltiazem.
  - 0.3–0.5 mg/kg slow IV over 10 minutes, then 1–5 µg/kg per minute IV CRI.
  - Use caution as hypotension could occur.
  - Can be used in conjunction with furosemide.
  - Monitor body weight carefully, as diuresis can occur and lead to excessive fluid loss.
- Dopamine: 2–5 µg/kg per minute IV CRI.
- Fenoldopam: 0.1–1 µg/kg per minute IV CRI.
- Phosphate binders.

Antiemetics

- Dolasetron: 0.6 mg/kg IV every 24 hours.
- Ondansetron: 1 mg/kg IV every 8–12 hours.

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- Maropitant: 1 mg/kg SQ, 2 mg/kg PO.
- Metoclopramide: 1–2 mg/kg per day IV CRI.

H_2 Blockers

- Famotidine: 0.5–1 mg/kg IV every 12 hours.
- Ranitidine: 0.2–2 mg/kg IV, PO every 8–12 hours.
- Cimetidine: 5 mg/kg IV every 6–8 hours.

Proton Pump Inhibitors

- Omeprazole: 0.5–1 mg/kg PO every 12 hours.
- Pantoprazole: 1 mg/kg IV once daily.

Gastroprotectants

- Sucralfate: 0.25–1 g PO twice a day.
    - Treat hyperkalemia.
- Insulin and dextrose: 0.25–0.5 units/kg IV regular insulin, followed by 1 g dextrose IV per unit of insulin administered, followed by 2.5% dextrose CRI.
- Sodium bicarbonate: 0.25–1 mEq/kg IV.
- Calcium chloride or calcium gluconate: 0.5–1 mL/kg of 10% solution IV over 10–20 minutes, monitor ECG continuously.



COMMENTS

- Central venous pressure
- Urine output; ins and outs
- Blood pressure
- ECG
- Body weight
- Daily to twice-daily evaluation of BUN, creatinine, hematocrit, Hct, platelet count, electrolytes, and glucose

Possible Complications

- Aspiration pneumonia secondary to vomiting
- Pulmonary edema
- Hypertension
- Multiple organ failure/MODS
- Cerebral edema
- DIC
- Seizures
- Coma
- Death

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Histopathology

- Severe diffuse tubular degeneration
- Proteinaceous and cellular debris
- Mineralization of glomerular basement membrane
- Glomerular congestion

Expected Course and Prognosis

- Generally favorable if treatment is initiated immediately after witnessed ingestion.
- Overall, 53% survival in affected cases.
- Potential for long-term renal insufficiency.
- Negative prognostic indicators include oliguria or anuria, weakness, elevated calcium × phosphorus product, metabolic acidosis.

Abbreviations

ARF: acute renal failure
BUN: blood urea nitrogen
CRI: constant rate infusion
DIC: disseminated intravascular coagulation
ECG: electrocardiogram
GI: gastrointestinal
Hct: hematocrit
IV: intravenous
MODS: multiple organ dysfunction syndrome
NSAID: nonsteroidal antiinflammatory drug
PCR: polymerase chain reaction
PO: by mouth (per os)
SQ: subcutaneous

Suggested Reading

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chapter 86

Retinal Detachment

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DEFINITION/OVERVIEW

- Separation of the retina from the underlying choroid and sclera.
- Anatomically, the separation of the neurosensory layers of the retina from the retinal pigment epithelium (RPE).

ETIOLOGY/PATHOPHYSIOLOGY

- There are three main types of retinal detachments.
  - **Exudative detachments** (Fig. 86.1): accumulation of fluid under the neurosensory retina expands the potential space between the neurosensory retina and the RPE,

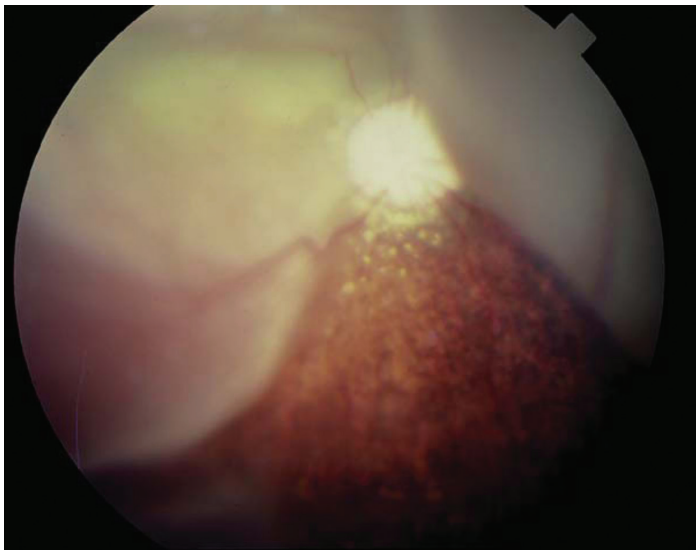


Fig. 86.1. Fundic photograph of a dorsal, serous, retinal detachment. Note the veil-like structure covering the tapetum. This is the detached retina.

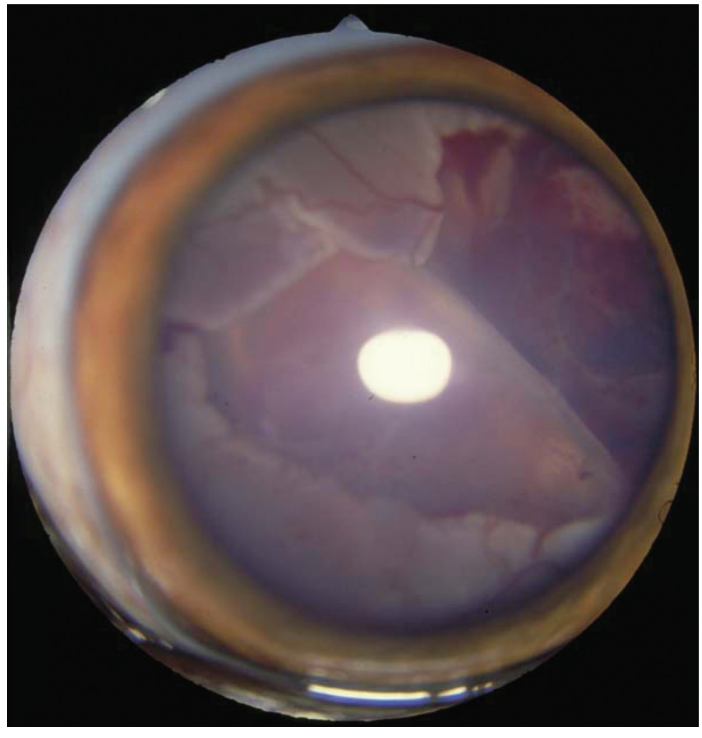
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causing the retina to billow anteriorly; may be partial or complete (Fig. 86.2). Fluid may be composed of:

- Exudate due to infectious causes/chorioretinitis (i.e., viral, fungal, rickettsial, protozoal, etc.)
- Transudate due to systemic hypertension
- Hemorrhage caused by systemic hypertension (a very common cause in cats), coagulopathies, anemia, hyperviscosity, trauma, vasculitis, infectious disease
- Idiopathic (steroid-responsive exudative retinal detachments) also occur infrequently when other etiologies have been ruled out.

- **Traction band detachments**: fibrous or fibrovascular tissue within the vitreous after hemorrhage or uveitis can contract and pull neurosensory retina away from the RPE; secondary to uveitis or trauma in most cases; rarely, secondary to congenital abnormalities.
- **Rhegmatogenous retinal detachments**: retinal detachment secondary to a tear or hole in the retina; vitreous or fluid dissects under the retina via the tear, causing progression of detachment; may progress to complete detachment with 360%



■ **Fig. 86.2.** Photograph of a detached retina taken through the pupil. Note the blood vessels in the retina and the hemorrhages.

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disinsertion, with the retina remaining attached only at the optic nerve head and hanging ventrally over the optic nerve head like a veil.
  - Iatrogenic: after cataract surgery
  - Traumatic
  - Breed-related/inherited predisposition
  - Secondary to congenital disease such as Collie eye anomaly, retinal dysplasia, Labrador Retrievers with oculoskeletal dysplasia
  - Secondary to uveitis, such as with cataracts and lens-induced uveitis or in association with chorioretinitis
- Congenital retinal "detachments" are not truly detachments, but rather failure of the embryologic layers to unite; seen in Collie eye anomaly, retinal dysplasia, multiple congenital anomaly eyes (Fig. 86.3); some may progress to develop tears and subsequent rhegmatogenous retinal detachments as the vitreous moves under the neurosensory retina.

Systems Affected

- Ophthalmic.

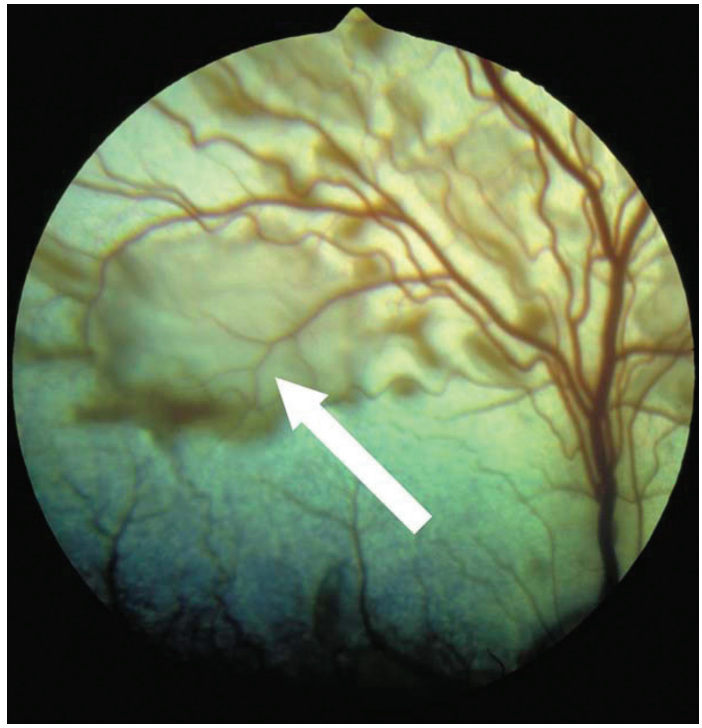


Fig. 86.3. Fundic photograph of a circumscribed area of retinal detachment (*white arrow*) due to retinal dysplasia. The worm-like areas are dysplastic retina (retinal folds).

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SIGNALMENT/HISTORY

- No breed or sex predilections in general, although numerous breeds have known predisposition to retinal dysplasia or multiple ocular anomalies that may lead to retinal detachment, and some terriers are thought to be at increased risk of rhegmatogenous retinal detachment.

Risk Factors

- Dogs developing cataracts and those undergoing cataract surgery are at higher risk and therefore those breeds predisposed to cataracts are at risk.

Historical Factors

- Vision loss: if unilateral, may be difficult for owner to discern; if incomplete and unilateral or bilateral, frequently no clinical signs are detected by the owner.
- Owner may notice cloudiness or redness of affected eye or the presence of a floating white tissue in pupil.
- Intraocular hemorrhage may be noted by the owner in cases of rhegmatogenous retinal detachments or those associated with systemic hypertension.

CLINICAL FEATURES

- Acute vision loss: if detachments are incomplete, visual field losses may be present but are very difficult to detect.
- A white/gray sheet of tissue may be visible through the pupil, behind the lens; it may contain blood vessels and possibly areas of hemorrhage. If close examination of the eye with a pen light or Finoff transilluminator allows visualization of retinal vessels, either the retina is detached or the lens is no longer in place as one should *not* be able to visualize retinal vessels without direct or indirect ophthalmoscopy. If the retina is detached, these vessels will not appear in their normal orientation, and the retina will appear as a white/gray membrane. Retinal detachments may thus be easier to recognize with the pen light/transilluminator than upon attempts at direct or indirect ophthalmoscopy in some cases.
- Neurophthalmologic findings.
    - Menace response will be absent in complete detachments, but present with some partial detachments.
    - The dazzle response will be significantly reduced with complete retinal detachment and become negative with chronicity.
    - Pupillary light responses may initially be only mildly slow or incomplete even with a complete retinal detachment, and become entirely absent only with chronicity in most cases.

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- Baseline pupil size is often moderately mydriatic; even in cases of unilateral, chronic, complete detachment where there is no persistent light perception, the pupil may not be widely dilated as there is a consensual from the contralateral eye in bright light.



DIFFERENTIAL DIAGNOSIS

- Any disease that causes acute blindness (e.g., SARDs, acute hyphema or vitreal hemorrhage, diabetic cataracts).



DIAGNOSTICS

- Close examination of the eye with a pen light or transilluminator may reveal a detached retina that is anteriorly displaced and located just behind the lens.
- Direct or indirect ophthalmoscopy: direct visualization of the detached retina.
- Ocular ultrasound: if the posterior part of the eye cannot be seen due to cataracts or intraocular hemorrhage.
- Complete blood count/serum chemistry/infectious disease testing as indicated: to rule out infection, bleeding disorder, renal insufficiency.
- Systemic blood pressure measurement: to detect hypertension (especially important in cats); followed by testing for hyperthyroidism if indicated in feline cases.
- Cardiac evaluation for cardiomyopathy.



THERAPEUTICS

- Goal: to optimize the environment for retinal reattachment; alternately, refer to retinal specialist for retinal reattachment surgery for certain types of rhegmatogenous retinal detachments. If unsure of cause or type of detachment, consider referral to a veterinary ophthalmologist for further evaluation.

Drugs of Choice

- Treat the primary cause: hypertensive therapy.
- Appropriate systemic antimicrobials if infectious disease is suspected.
  - Systemic corticosteroids can be administered if steroid-responsive retinal detachment is suspected; contraindicated in infectious diseases in most cases.

Precautions/Interactions

- Systemic corticosteroids are contraindicated if infectious disease is suspected.
- Corticosteroids should not be given in conjunction with NSAIDs.

Diet

- No special diet is needed unless systemic hypertension is causative.

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Surgical Considerations

- Retinal reattachment surgery, done by some veterinary ophthalmologists for certain cases of rhegmatogenous retinal detachments.
- Partial detachments may be prevented from progressing by laser retinopexy around the edges of the detachment.



COMMENTS

Expected Course and Prognosis

- Variable prognosis depending on cause.
- Surgically reattached retinas do not always regain visual function; prognosis is improved with earlier reattachment.
- Better prognosis if detachment is due to serous subretinal exudate secondary to hypertension if hypertension is promptly controlled.

Client Education

- The patient should be kept quiet with minimal activity or excitement.
- Because the animal will go blind with complete bilateral retinal detachments, he or she may become confused and require a period of adjustment.
- Distribution of references to caring for blind dogs (see Suggested Reading).

Patient Monitoring

- Frequent rechecks are recommended early in the disease (e.g., weekly); in cases of hypertension, blood pressure should be checked and medications adjusted accordingly.

Prevention/Avoidance

- Early detection of hypertension (e.g., monitoring blood pressure in older cats) can prevent retinal detachment.
- Discourage breeding of dogs affected with CEA or retinal dysplasia to prevent genetic transmission.

Possible Complications

- Permanent blindness.
- Secondary glaucoma may develop with chronic retinal detachment due to formation of a preiridal fibrovascular membrane that closes the iridocorneal drainage angle.
- Intermittent episodes of intraocular hemorrhage may occur with rhegmatogenous retinal detachments.

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Synonyms

- Retinal separation

Abbreviations

CEA: collie eye anomaly
NSAID: nonsteroidal antiinflammatory drug
RPE: retinal pigment epithelium
SARDS: sudden acquired retinal degeneration syndrome

Suggested Reading

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Author: Rachel Matusow Wynne, DVM
Acknowledgment to the author of the original chapter in *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Emergency and Critical Care, First Edition:* Juliet R. Gionfriddo.

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chapter 87

Schiff-Sherrington



DEFINITION/OVERVIEW

- Forelimb extensor hypertonia with hindlimb paralysis caused by an acute and severe thoracolumbar spinal cord lesion.



ETIOLOGY/PATHOPHYSIOLOGY

- Lumbar spinal cord L1–L7: dorsolateral section of the ventral gray matter and the location of inhibitory neurons "border cells."
- Border cells are responsible for extensor muscle inhibition of motor neurons in the cervical intumescence; highest concentration of cells are at L2–L4.

Systems Affected

- Nervous: forelimb hypertonia, most pronounced when in lateral recumbency, hindlimb paralysis, normal cranial nerves.
- Musculoskeletal: nonambulatory due to the hindlimb paralysis; however, the forelimbs can ambulate normally if the patient is supported. Spinal fractures should be ruled out prior to assessing.



SIGNALMENT/HISTORY

- Dogs: commonly associated with major trauma such as being hit by a car.
- Cats: rarely.

Historical Findings

- Trauma: hit by car most common.
- If unknown trauma, acute paralysis.

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Fig. 87.1. Extensor rigidity of forelimbs in a dog with a spinal fracture at T13–L1. Note that the patient is strapped down to a rigid board to prevent further movement.

CLINICAL FEATURES

- Nonambulatory.
- Forelimb extensor rigidity (Fig. 87.1).
- Hindlimb paralysis.
- Palpation of vertebral column trauma.
- Often other signs of trauma.
- Cranial nerves normal.
- Forelimbs: rigid extensor tone, normal gait, reflexes and postural reactions but "stiff."
- Hindlimbs: paralysis.
- Caudal to the lesion: reflexes normal.
- Level of the lesion: cutaneous trunci reflex cutoff; line is often caudal to the lesion by 1–2 spinal cord segments.

DIFFERENTIAL DIAGNOSIS

- Cervical spinal cord lesion: other forelimb neurologic deficits besides hypertonia.
- Decerebrate rigidity: hypertonia in forelimbs and hindlimbs.
- Decerebellate rigidity: forelimb hypertonia, flexed hindlimbs; the patient is unconscious and this is the distinguishing finding.

DIAGNOSTICS

- Radiographs, myelography, CT, MRI: detect spinal cord lesion.

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THERAPEUTICS

- The objective is immediate spinal cord stabilization to prevent additional trauma during diagnosis and stabilization of concurrent problems. Strap the patient to a stiff backboard until the spine can be cleared by imaging.
- See specific spinal cord diseases for further treatments.

COMMENTS

Client Education

- Posture may remain for several weeks, not an indication of prognosis. However, prognosis is guarded.
- Recovery can be seen if spinal cord hemorrhage and edema resolve, unless spinal cord is transected.

Patient Monitoring

- Neurologic exam q1–2h for change.

Expected Course and Prognosis

- Schiff-Sherrington does not change the prognosis; prognosis and clinical course should be based on the cause of the phenomenon.
- Grave prognosis if deep pain is absent after 48 hours.

Abbreviations

CT: computed tomography
MRI: magnetic resonance imaging

See Also

Spinal Shock
Spinal Fracture

Suggested Reading

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Author: Stacy D. Meola, MS, DVM

chapter 88

Scleral and Corneal Lacerations

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DEFINITION/OVERVIEW

- Corneal or scleral lacerations are the result of disruption of the corneal or scleral tissue, by blunt or sharp trauma, or by the presence of a foreign body.
- They may be partial or full thickness in nature, and they may or may not involve other intraocular tissues, such as the anterior or posterior uvea, lens, vitreous, or retina.

ETIOLOGY/PATHOPHYSIOLOGY

- Sharp trauma leads to partial- or full-thickness disruption of the sclera and/or cornea and is initiated at the surface of the globe. Perforating corneal lacerations due to cat claw injuries may be associated with significant intraocular damage, even when the entry wound is small; in such cases, phacoclastic uveitis or septic implantation syndrome can result in severe intraocular inflammation and loss of vision/globe if not treated aggressively at initial presentation.
- Blunt trauma usually leads to more severe damage to the intraocular structures and can lead to blow-out type wounds to the sclera or cornea, generally either at the posterior surface of the globe or near the limbus.

Systems Affected

- Ophthalmic: the extent of tissue involvement is highly variable. Sharp trauma may result in a superficial to deep corneal laceration, a perforating corneal laceration with or without lens involvement, or a corneoscleral laceration with involvement of the underlying uvea. Blunt trauma may result in lens luxation, retinal detachment, intraocular hemorrhage, and rupture of the globe near the limbus or posterior eye wall. Collateral damage to the conjunctiva, extraocular muscles, eyelids, and nictitating membrane may occur as well.

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SIGNALMENT/HISTORY

- Dogs or cats, no age, breed or sex predilection.
- Exception: young puppies are subjectively overrepresented for cat claw injuries to the eyes, especially prior to development of a strong menace response.

Risk Factors/Causes

- Fighting/interactions between dogs and cats or with wild animals.
- Spending time in tall grasses, brush, trees, dry vegetation.
- History of blunt trauma, such as tennis ball, vehicular accident.
- Poor protection of the ocular surface, such as with lagophthalmos, exophthalmos, blindness.

Historical Findings

- Acute onset.
- Possibly in altercation with another animal, most commonly a cat.
- Possibly in heavy or dry vegetation.
- Possibly hit by projectile (rock, pellet, ball, etc.).
- Trauma often not observed.



CLINICAL FEATURES

- Variable depending on location, depth, and blunt versus sharp trauma.
- Corneal laceration due to sharp trauma: variable depth and size, range from superficial to full-thickness perforations; examine periphery for extension into limbus.
- Corneal wounds may appear closed, gaping, or partially sealed with fibrin or uveal tissue.
- Scleral wound may not be visible beneath abnormal conjunctiva (i.e., chemosis, hyperemia, or subconjunctival hemorrhage).
- Uveal tissue may be prolapsed through a scleral or corneal wound and appears brown to black.
- Foreign body may or may not be present.
- Corneal edema, dyscoria, hyphema, cataract, lens laceration, vitreal hemorrhage, retinal detachment, or exophthalmia may be present.



DIFFERENTIAL DIAGNOSIS

- Identification of wound is diagnostic. If no wound is found, then one must consider traumatic incident without laceration.
- Bacterial keratitis or keratomalacia: deep stromal corneal ulcers frequently become apparent to the owner "overnight," and may progress to corneal perforation.

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- Hyphema or vitreal hemorrhage may also occur with retinal detachment, intraocular neoplasia, uveitis, or systemic disease causing coagulopathy or vasculitis.
- Lens luxation may also occur with primary lens instability or end-stage glaucoma with buphthalmos.



DIAGNOSTICS

- Note: Prompt referral to a veterinary ophthalmologist should be strongly considered and is recommended for any partial-thickness gaping corneal wound, perforating wound, or significant blunt trauma if treatment goals include maintaining vision and avoiding the need for enucleation.
- Identify tissues involved, possible cause, and nature of damage to the eye. Perform a complete ophthalmic examination to the extent that tissues can be visualized.
- Carefully examine the limbus for corneal lacerations crossing into the sclera (poorer prognosis/increased risk of injury to underlying uveal tissue) or blow-out wounds from blunt trauma.
- Dyscoria is evidence of perforating trauma.
- Direct and consensual pupillary light responses should be assessed.
- Seidel test may be used to assess for active leakage of aqueous humor at a wound; apply concentrated (dark orange) fluorescein directly from dry strip and watch for streaming of aqueous humor as the fluorescein turns yellow.
- Careful examination of the lens is recommended with any potentially perforating trauma. Anterior lens capsule lacerations often lead to blinding phacoclastic uveitis or septic implantation syndrome if not addressed at initial presentation, though clinical signs may not develop for several weeks. Such lacerations are easily missed if careful evaluation with mydriasis is not undertaken, ideally using slit lamp biomicroscopy.
- Be very careful not to put pressure on the globe until rupture or laceration has been ruled out.
- Consider scleral rupture when subconjunctival hemorrhage is present, shallow or deep anterior chamber is present, vitreal hemorrhage is present, or the globe is very soft.
- Blood work and urinalysis are usually not helpful if injury is limited to the eye. These should be considered if general anesthesia is required, or in preparation for treatment with systemic antiinflammatory medications.
- Consider cytology and/or culture and sensitivity of wound.
- Consider coagulation profile if intraocular bleeding cannot be explained due to trauma and no laceration is noted.
- Imaging, such as ocular ultrasound, orbital radiographs, CT or MRI, should be considered to ascertain extent of damage and possibly identify foreign body depending on the history and suspected degree and cause of damage.

Pathologic Findings

- Depend on extent of ocular damage.
- Posttraumatic sarcoma may occur in cats. May be months or years after initial trauma.

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THERAPEUTICS

Drugs of Choice

- Note: Medications that would otherwise have poor ocular penetration due to blood–eye barriers will likely enter the acutely inflamed eye. Inflammation reduces the effectiveness of these barriers and allows larger molecules to leave the bloodstream.

Antibiotics

- Penetrating but nonperforating and uncomplicated superficial wounds: topical antibiotics are indicated, systemic antibiotics should not be necessary. For example, 3–4 times daily neomycin-polymyxin B-bacitracin, neomycin-polymyxin-gramicidin, gentamicin, or tobramycin. Deeper stromal wounds may be an indication for topical antibiotics with better tissue penetration and broad spectrum of activity, such as topical fluoroquinolones, as well as increased frequency of treatment at 4–6 times daily.
- Perforating wounds: topical and systemic antibiotic therapy is indicated. Avoid ointments if there is any suspicion of corneal perforation. Broad-spectrum systemic antibiotics such as amoxicillin/clavulanic acid are indicated. Fluoroquinolones should be considered for topical antibiotic therapy for good tissue penetration (ofloxacin, gatifloxacin, or moxifloxacin) and should be administered every 1–6 hours depending on the severity of injury.

Antiinflammatories

- NSAIDs: systemic, if deemed safe for the individual patient.
- Topical antiinflammatories are generally not indicated for corneal or scleral lacerations, at least during the acute phase of healing. If phacoclastic uveitis arises or a lens capsule rupture is visualized, topical antiinflammatories may be prescribed at the discretion of a veterinary ophthalmologist.

Mydriatics

- Atropine 1% ophthalmic solution every 6–12 hours when there is significant miosis or aqueous flare. Frequency may be decreased to every 24–48 hours once mydriasis is achieved, as long as the pupil remains dilated. Potential contraindications: history of glaucoma, keratoconjunctivitis sicca.

Analgesics

- Topical atropine: mydriasis reduces the risk of axial posterior synechiae, but cycloplegia also alleviates discomfort by reducing ciliary body muscle spasms.
- Systemic antiinflammatories: see previous information.
- Systemic injectable or oral opioid pain medication as needed for comfort (buprenorphine, methadone, tramadol, etc.).

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Alternative Drugs

- Compounding fortified and noncommercial topical antibiotic solutions is possible with cefazolin, gentamicin, tobramycin, and others. More aggressive therapy is achieved by increasing the concentration in some of these solutions.

Precautions/Interactions

- Avoid ophthalmic ointments in cases where perforation has occurred. If ointment enters the eye, it will likely lead to granulomatous inflammation.
- Avoid systemic enrofloxacin and orbifloxacin in young dogs, risk of disruption of cartilage synthesis.
- Enrofloxacin in cats may lead to acute retinal degeneration and vision loss.
- Topical aminoglycosides: if used frequently or at high concentrations, they may be irritating and may impede reepithelialization; may need to carefully calculate dose in very small animals, especially if giving this kind of drug systemically as well.
- Topical steroidal and nonsteroidal antiinflammatories may delay wound healing.
- Atropine may exacerbate keratoconjunctivitis sicca and glaucoma.
- Systemic NSAIDs may potentiate nephrotoxicity of aminoglycosides; be sure animal is well hydrated and kidney function is adequate.

Activity

- Should be confined to indoors or small, well-controlled environment. Cats should remain indoors. Leash walking usually OK for dogs; consider using a harness to keep pressure off neck and avoid increasing intraocular pressure.
- E-collar should be used at all times in most cases, and should be placed prior to transport if referral is pursued.

Surgical Considerations

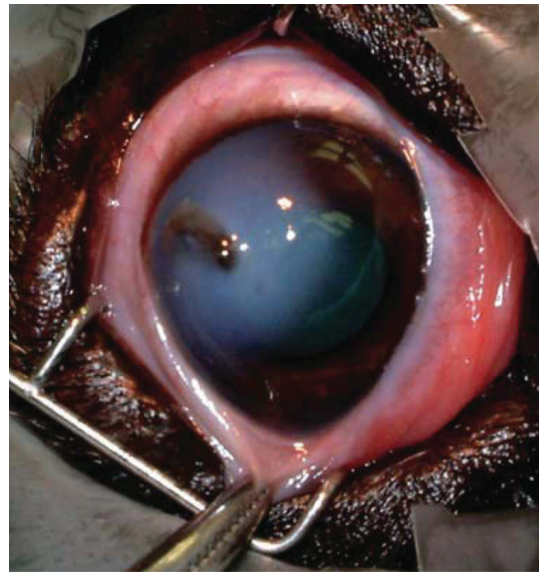
Injuries Possibly Requiring Surgery

- Small, full-thickness corneal laceration that has sealed and has no uveal prolapse (Figs 88.1, 88.2, 88.3).
- Nonperforating wound with moderate gaping: outcome ideal with surgical repair.
- Nonperforating wound that is deeper than two-thirds corneal thickness.
- Lens lacerations without obvious lens material in the anterior chamber: treatment may vary by ophthalmologist; surgery most commonly recommended.

Injuries Requiring Surgery

- Full-thickness sclera or corneoscleral lacerations, with or without positive Seidel test
- Full-thickness wounds with uveal prolapse
- Possible retained foreign body
- Possible posterior scleral rupture
- Lens lacerations with lens material in the anterior chamber

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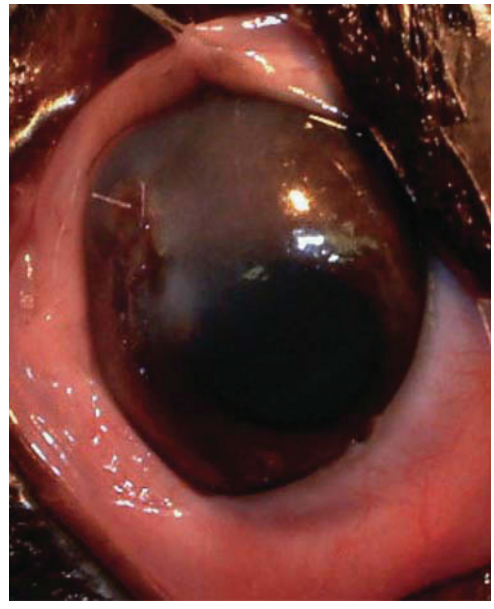


■ **Fig. 88.1.** Full-thickness corneal laceration in a boxer puppy after an altercation with a cat 20 hours prior to presentation. The iris has prolapsed through the corneal defect and has sealed the laceration such that aqueous humor is no longer leaking from the eye. There is also localized corneal edema.



■ **Fig. 88.2.** The iris tissue has been repositioned and is being held in place with one 8-0 simple interrupted suture.

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■ **Fig. 88.3.** Corneal laceration immediately following surgical repair with three interrupted 8-0 polyglactin 910 sutures.

Injuries Requiring Medical Management Alone

- Nonperforating wounds with little to no wound gaping.
  - Treat with topical antibiotics and possibly atropine, systemic NSAIDs, systemic opioid pain medications. Place an Elizabethan collar.
  - Reexamine in 3–5 days to make sure condition is not deteriorating.
- Uncomplicated, full-thickness, punctate corneal wounds that have sealed, there is no uveal prolapse, and there is a high degree of confidence that the lens has not been lacerated.
  - Treat with frequent topical antibiotic solutions (fluoroquinolones recommended) and possibly atropine, systemic NSAIDs, systemic opioid pain medications. Place an Elizabethan collar. If the globe has been penetrated, systemic antibiotics are indicated.
  - Recheck in 1–2 days to make sure condition is not deteriorating.

COMMENTS

Client Education

- Warn owners that full extent of ocular damage (i.e., cataracts, lens capsule rupture, retinal detachment, glaucoma, or infection) may not be apparent for days or weeks after initial injury; early assessment by a veterinary ophthalmologist will increase the chances of diagnosis of intraocular damage, to facilitate prompt intervention and improve prognosis.

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- With ocular disease, prognosis for vision and prognosis for keeping the globe and remaining comfortable must both be discussed, i.e. prognosis for a comfortable eye may be better than that for a visual *and* comfortable eye in some cases.

Patient Monitoring

- Deep or perforating wound (if referral for repair is not possible) should be checked every 24–48 hours to monitor for infection, adequate pain control, control of inflammation, and structural integrity of the globe.
- Superficial wounds should be rechecked every 3–5 days until adequate healing has occurred.

Prevention/Avoidance

- Carefully introduce new dogs, especially puppies, to new cats with front claws.
- Use caution when allowing dogs to run in thick vegetation, especially those dogs with significant ocular exposure (i.e., brachiocephalic breeds) or vision impairment.

Possible Complications

- Loss of vision
- Chronic ocular inflammation or pain necessitating removal of the globe
- Posttraumatic sarcoma in cats

Expected Course and Prognosis

- Most corneal lacerations and foreign bodies can be successfully treated.
- Injuries that have a poorer prognosis are those with posterior uveal involvement, lack of light perception in the injured eye, lens involvement, moderate-to-severe vitreal hemorrhage, or retinal detachment.
- An eye that sustains blunt trauma usually has a poorer prognosis than one that experiences sharp trauma.

Abbreviations

CT: computed tomography
KCS: keratoconjunctivitis sicca
MRI: magnetic resonance imaging
NSAID: nonsteroidal antiinflammatory drug

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chapter 89

Seizures



DEFINITION/OVERVIEW

- A seizure is a nonvolitional, rhythmic, repetitive excitatory discharge of neurons in the cerebral cortex.
- Seizures can be focal or generalized, convulsive or nonconvulsive (lacking gross movement).
- Focal seizures may manifest as repetitive, uncontrolled tonic-clonic movement of a single area of the body such as the jaw or a single limb.
- Generalized seizures usually are characterized by tonic-clonic movement of the entire body.
- The term "cluster seizures" refers to more than two seizures in a 24-hour period.
- In humans, epilepsy is defined as the occurrence of two seizures greater than 24 hours apart or the occurrence of one seizure with a high risk of subsequent seizures due to etiology.
- Status epilepticus is seizure activity lasting longer than 5 minutes or 30 minutes of seizure activity with incomplete recovery.

ETIOLOGY/PATHOPHYSIOLOGY

- Seizures may result from extracranial (provoked), intracranial (symptomatic), idiopathic, or cryptogenic causes.
- Extracranial causes of seizures are systemic disorders that result in the exposure of brain cells to epileptogenic substances or conditions.
- Intracranial causes of seizures result from structural derangement of the cerebral cortex.
- Idiopathic epilepsy is defined as epilepsy due to presumed genetic origin in which no neuropathologic alterations can be identified.
- Cryptogenic epilepsy is a seizure disorder that is likely due to an intracranial disease process, for which the cause has not been identified.

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Systems Affected

- Cardiovascular: possible arrhythmias.
- Endocrine/metabolic: lactic acidosis, dehydration, hyperthermia.
- Nervous.
- Respiratory: neurogenic pulmonary edema, possible dyspnea during
- seizure event.



SIGNALMENT/HISTORY

- Age, sex, and breed predisposition will vary according to the underlying etiology leading to seizures.
- Younger (1–5 year old), large breed, male dogs are commonly diagnosed with idiopathic epilepsy.
- Idiopathic epilepsy is increasingly recognized in cats, with some studies suggesting up to 25% of cats with seizures falling into this category.
- Small breed dogs, especially those with dome-shaped heads, are frequently affected by malformations like hydrocephalus.
- Older dogs and cats are more likely than younger animals to have intracranial neoplasia, and some tumor types, such as meningioma, may have breed and sex predilections.
- Animals of any age and breed can be affected by toxins, metabolic disorders, organ dysfunction, and encephalitis.
- Seizures (the ictus) are usually typified by a loss of consciousness, body/limb convulsions, and autonomic activity (tachycardia, salivation, urination, defecation).
- Animals that have seizures may have preictal behavioral changes.
- Postictal neurological signs are exceedingly common regardless of etiology, and may include blindness, mentation changes, and ataxia; typically, postictal deficits will resolve within hours or a few days and will very likely be symmetrical if the underlying etiology is extracranial.

Risk Factors

- Risk factors are dependent on the etiology leading to seizure activity.
- In general, animals with structural disease involving the forebrain are predisposed to developing seizures.
- Extracranial seizures may be precipitated by exposure to toxins, medications (e.g. metronidazole, antihistamines), systemic diseases of the liver and kidney, as well as diseases that alter electrolytes (e.g., Addison's disease) or result in hypoglycemia (e.g., insulinoma).

Historical Findings

- Owners may report an aura; the animal may hide, whine, seek attention, or appear restless prior to the ictus.
- During the postictal period, the animal may appear blind, walk into walls, circle, show ataxia, be nonambulatory, or have mentation changes.

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- The animal should lose consciousness during a generalized seizure and be unresponsive to the owner. Tonic-clonic movements are commonly noted.
- The owner may report finding feces and urine in the house that were produced during an unrecognized seizure event.



CLINICAL FEATURES

- Animals may have completely normal physical examinations or may show abnormalities that reflect underlying systemic disease, recent seizure activity, and/or neurologic examination findings suggesting structural disease.
- Abnormal neurologic exam findings include altered mentation, evidence of cortical blindness, circling, and postural reaction deficits.
- The neurologic examination abnormalities may disappear if they are postictal or remain if they are a result of structural intracranial disease.
- Posictal deficits are typically symmetrical if the underlying etiology is not structural.



DIFFERENTIAL DIAGNOSIS

- Syncope: defined as a loss of consciousness secondary to lack of adequate blood flow to the brain, which is usually sudden in onset and of short duration.
- Owner history is important in differentiating syncope from seizures.
- Syncopal episodes usually are preceded by excitement or exercise and can range in severity from rear limb weakness to total collapse.
- Recovery from syncopal episodes is usually rapid with no lingering deficits.
- Animals suspected of syncope should have a complete cardiac evaluation.
- Narcolepsy is characterized by sudden-onset REM sleep, cataplexy, sleep hallucinations, excessive daytime sleepiness, and sleep paralysis.
- Episodes are usually preceded by an excitatory stimulus such as feeding or seeing an intact member of the opposite sex.
- The Labrador Retriever and Doberman Pinscher are predisposed and have an autosomal recessive pattern of inheritance.
- Acute fulminant neuromuscular diseases such as myasthenia gravis and congenital myotonia may be mistaken for seizures.
- Owners may describe the collapse and weakness associated with myasthenia gravis as a seizure.
- Collapse is a feature of several uncommon neurological diseases, including exercise-induced collapse (Labrador), hereditary ataxia (Jack Russell), and myotonia.
- Acute vestibular attack: vestibular diseases (especially those that are idiopathic or vascular) can have a sudden onset of clinical signs.
- Affected animals may roll around and be unable to rise, which may be mistaken for a seizure.

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DIAGNOSTICS

- A complete blood count, chemistry panel with electrolytes, and urinalysis should be performed on all animals with seizures.
- Seizures are an uncommon manifestation of hepatic encephalopathy, but liver function testing (e.g., bile acids, ammonia) is nonetheless critical especially if long-term treatment with certain anticonvulsants is being considered.
- If the animal is currently on antiepileptic medications, serum should be drawn for blood drug levels prior to administration of AEDs other than diazepam/midazolam.
- Blood pressure monitoring and pulse oximetry are encouraged, especially in animals with recent severe seizure events or those exhibiting dyspnea.
- MRI and CSF examination should be recommended for seizuring animals without an etiologic diagnosis.
- CSF collection is contraindicated if high intracranial pressure or brain herniation is present.

Pathologic Findings

- Gross and histopathologic findings will vary based on etiology.



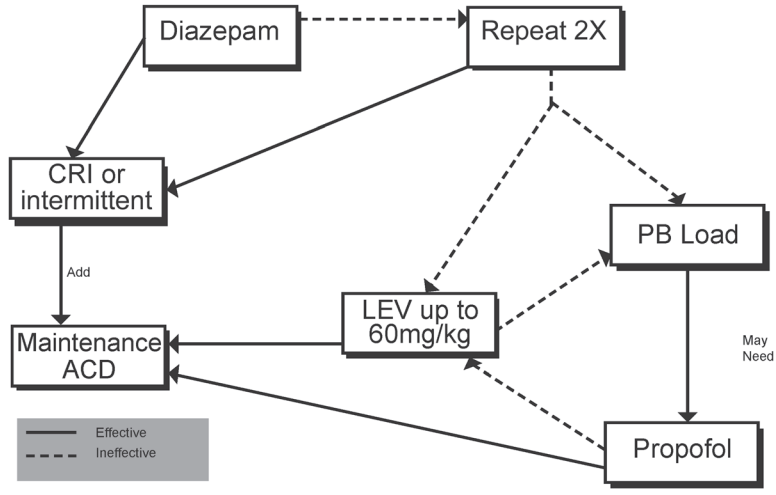
THERAPEUTICS

- The first objective of treatment is to stop ongoing seizure activity and to prevent future seizure activity.
- A rapid-acting AED should be administered to all patients actively seizuring at admission.
- If serum biochemistry suggests an underlying metabolic etiology, such as hypoglycemia or hypocalcemia, this should be corrected. Treatment of an animal with maintenance AEDs should be pursued if the patient presents in status epilepticus or has at least two seizures in a 24-hour period.
- If seizures resulted from intoxication or metabolic disease that is resolved, maintenance AEDs can be tapered over a few weeks.
- Animals with structural intracranial disease, idiopathic epilepsy, or unresolved metabolic disease will need to be continued on a maintenance AED.

Emergency Therapy

- Figure 89.1 outlines the typical approach to an animal with active seizures in the emergency setting.
- In general, benzodiazepine drugs are selected first and may be continued as a CRI if effective (midazolam preferred as it does not induce phlebitis).
- Phenobarbital and levetiracetam are maintenance ACDs that also can be used in the emergency setting. Acute ACD effects of phenobarbital may be delayed by up to 20 minutes.

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■ **Fig. 89.1.** An algorithm for emergency management of seizure activity in dogs and cats.

- Propofol is commonly used when animals are refractory to the above treatments or while waiting for phenobarbital to exert an ACD effect.
- General anesthesia is a treatment of last resort.
- Table 89.1 is a list of ACDs commonly used in the emergency setting with drug doses, routes, and common adverse events.

Precautions/Interactions

- AEDs are complex drugs, and any precautions or interactions listed here represent only a small proportion of those possible.
- In general, acepromazine has questionable proepileptogenic properties and some practitioners feel it should be used cautiously in dogs with seizures.
- Drugs that lower the seizure threshold should be avoided if possible.
- Animals on phenobarbital may have altered or increased metabolism of other drugs eliminated by the cytochrome p450 pathway in the liver.

Maintenance Therapy

- Maintenance AED therapy in veterinary medicine is rapidly evolving.
- Although some authors consider traditional drugs such as phenobarbital and potassium bromide as the "drugs of choice," others prefer newer drugs that have fewer reported side effects and less rigorous monitoring requirements.
- Potassium bromide is not an acceptable maintenance AED in cats because of adverse pulmonary effects.
- Diazepam has been used as a maintenance drug in cats, although most authors discourage this as severe, irreversible hepatopathy has been described.

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TABLE 89.1. Anticonvulsant drugs used in the emergency and maintenance setting.

Drug	Drug class	Dose range	Frequency	Route	Adverse events
Diazepam*	Benzodiazepine	0.5–1 mg/kg 0.2–2 mg/kg/h 1–2 mg/kg	Bolus CRI	IV Rectal	Sedation Phlebitis Liver (rare, cats receiving oral drug)
Midazolam	Benzodiazepine	0.5–1 mg/kg 0.2–2 mg/kg/h	Bolus CRI	IV, Nasal	Sedation
Phenobarbital	Barbiturate	12–20 mg/kg 2–3 mg/kg+	1–4 doses BID	IV, IM PO	Sedation Polyphagia Polydipsia Polyuria Liver Blood lines Movement disorder Osteomalacia Dermatopathy
Levetiracetam	Pyrrolidone derivative	20–60 mg/kg 20–60 mg/kg+	Bolus TID	IV IV, IM, PO	Sedation (mild) Gastrointestinal
Propofol	Novel hypnotic	2–8 mg/kg 0.1–0.6 mg/kg/h	Bolus CRI	IV	Profound sedation Respiratory depression/arrest Hypotension Cardiac arrest Heinz bodies (cats)
Potassium bromide	Ionic salt	500 mg/kg 30–40 mg/kg+	Divided 1–5d q24h	PO, rectal PO	Sedation Polyphagia Polyuria Polydipsia Ataxia (pelvic limb) Pancreatitis? Dermatopathy Gastrointestinal
Zonisamide	Sulfa	6–10 mg/kg+	BID	PO	Sedation Polyarthritis Hepatopathy Renal tubular acidosis Gastrointestinal Keratoconjunctivitis
Gabapentin	Synthetic GABA	10–20 mg/kg+	TID	PO	Sedation Weight gain
Pregabalin	Synthetic GABA	2–3 mg/kg+	TID	PO	Sedation Ataxia

* Dogs receiving p450-inducing drugs should be dosed at the upper end of the listed range.
+ Dose for maintenance therapy.
BID, twice a day (bis in die); CRI, constant rate infusion; GABA, gamma-aminobutyric acid; IM, intramuscular; IV, intravenous; PO, by mouth (per os); TID, three times a day (ter in die).

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- Some animals will not be controlled with single maintenance AEDs and may require bimodal or trimodal therapy.
- Phenobarbital and bromide require serum drug concentration monitoring.
- For phenobarbital, blood drug levels, CBC, serum biochemistry, and liver function testing are performed 10–21 days following a change in dosing and every 6 months.
- In the case of bromide, serum drug concentrations are obtained within 3 months and serial urinalysis and blood profiles are obtained every 6 months.
- Some newer drugs are dosed to effect.

Diet

- The animal's diet should not be changed without cause if the patient is receiving potassium bromide as different salt content of food can affect drug absorption.
- Animals on potassium bromide should also have a drug level checked 2–3 months after a change in the mineral composition of water.
- Diet may also affect blood phenobarbital level.
- Ketogenic diets, while effective in juvenile humans to reduce seizure frequency, do not work well in veterinary species.

Activity

- No alterations in activity are suggested.

Surgical Considerations

- Animals with central nervous system disease are at increased risk for anesthetic complications.
- Monitoring of end-tidal CO_2, blood pressure, heart rate, and other parameters is recommended during anesthesia, as brain herniation can be a sequela of anesthesia – signs of this could include Cushing's reflex (hypertension with bradycardia), anisocoria, miosis, or unresponsive pupils.
- Surgical therapy for certain structural central nervous system diseases can be attempted.
- Surgical resection of an epileptic focus is not typically performed in veterinary medicine.



COMMENTS

Client Education

- Clients need to understand that treatment will be life-long and will require monitoring of animals with idiopathic epilepsy or underlying structural intracranial disease.
- The client should be instructed to maintain a seizure diary for the patient.
- Regular dosing of the AED needs to be emphasized as well as the negative consequences of sudden cessation of medication.

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Patient Monitoring

- While hospitalized, animals should be under near constant observation for seizure activity.
- Some facilities will attach bells to animals in an effort to help alert staff to a seizure event.
- The frequency of seizures should be monitored by the owner; more than one seizure per month indicates the need for blood drug levels and/or a change in drug dosage.
- See other monitoring suggestions under specific AEDs.

Prevention/Avoidance

- Some owners report that certain activities can trigger seizures in their pets. These activities should obviously be avoided if such a link is suspected.

Possible Complications

- Patients who are seemingly well regulated for a period of time may develop breakthrough seizure activity and need medication adjustments.
- Prolonged seizures or cluster seizures can be life-threatening as they may place increased metabolic demands on the patient and interfere with energy delivery to cells.
- Patients that have breakthrough seizure activity that is untreated will likely be at risk for the expansion of their epileptic focus, which may make seizure control more challenging.

Expected Course and Prognosis

- The course and prognosis associated with seizures are variable and depend on the underlying etiology.
- Idiopathic epileptics have a good prognosis, although the expense of drugs and therapeutic monitoring can be cost-prohibitive for some owners.
- Some animals with idiopathic epilepsy will require therapy with multiple AEDs or may have to be switched to more expensive medications as a result of side effects.
- Animals with encephalitis have a variable prognosis ranging from guarded to excellent, depending on the etiology.
- Animals with brain tumors have a poor prognosis without treatment and a guarded-to-good prognosis and increased survival with radiation therapy or surgery.
- The prognoses of animals with organ dysfunction or toxicities will vary depending on the severity of the disease.

Synonyms

- Epilepsy

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Abbreviations

AED: antiepileptic drug
CBC: complete blood count
CRI: constant rate infusion
CSF: cerebrospinal fluid
CT: computed tomography
GABA: gamma-aminobutyric acid
MRI: magnetic resonance imaging
REM: rapid eye movement

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chapter 90

Septic Peritonitis

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DEFINITION/OVERVIEW

- Peritonitis is an inflammatory process that results from contamination of the abdominal cavity by either microorganisms or inflammatory chemicals.
- Peritonitis is most often generalized and involves the entire abdominal cavity, but it may be localized to a portion of the abdomen.

ETIOLOGY/PATHOPHYSIOLOGY

- Primary peritonitis is uncommon and refers to infection of the abdominal cavity without a direct intraabdominal source of infection. Primary bacterial peritonitis most likely occurs through hematogenous spread. Another example of primary peritonitis is feline infectious peritonitis, which is caused by feline coronoavirus.
- Secondary peritonitis is more common and occurs due to direct bacterial or chemical contamination of the abdomen. Examples are contamination of the abdominal cavity due to rupture of a hollow viscus or abdominal abscess or due to a penetrating abdominal wound. Perforation of the GI tract is the most common source, which results in a polymicrobial and mixed bacterial infection.
- Peritoneal injury leads to release of proinflammatory mediators and increased capillary permeability. Fluid, electrolytes, protein, and red blood cells are lost into the abdominal cavity and result in a decreased circulatory volume.
- White blood cells enter the abdominal cavity and begin to destroy bacteria. Proteases and endotoxins are released that contribute to the inflammatory response.
- Sympathetic stimulation causes GI stasis. Ileus and peritoneal stimulation lead to vomiting that contributes to fluid losses.
- The decreased circulatory volume leads to hypovolemic shock. Absorption of bacterial and tissue toxins causes septic shock (a form of distributive shock) that contributes further to hypotension and tissue hypoxia.
- Failure of one or more organ systems occurs due to hypoperfusion and through several other complex mechanisms.
- Eventually organ failure will result in death if left untreated.

Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Emergency and Critical Care, Second Edition. Edited by Elisa M. Mazzaferro.
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Companion website: www.fiveminutevet.com/emergency

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Systems Affected

Cardiovascular

- Hypovolemia and inappropriate vasodilation contribute to decreased cardiac output in sepsis. Sympathetic nervous system activation causes increased heart rate, increased cardiac contractility, and, if vasomotor tone is normal, vasoconstriction. Sepsis is characterized by a lack of vasomotor tone and reflex vasoconstriction; instead, several mechanisms result in inappropriate vasodilation. Decreased tissue perfusion results in myocardial ischemia. Myocardial ischemia and electrolyte imbalances may trigger cardiac dysrhythmias that in turn further decrease cardiac output.

Gastrointestinal

- Sympathetic stimulation inhibits peristalsis. Ileus and peritoneal inflammation lead to vomiting, which contributes to fluid losses. Sequestration of fluid within the bowel lumen secondary to ileus also contributes to hypovolemia. Gastrointestinal hypoperfusion can result in bacterial translocation.

Hemic/Lymphatic/Immune

- Irritation of the peritoneal lining stimulates the inflammatory cascade, including cytokines and arachidonic acid metabolites. White blood cells, red blood cells, proteins, electrolytes, and fluid enter the peritoneal space. White blood cells destroy bacteria and cause the release of proteases and endotoxins. The proinflammatory cascade is counterregulated by antiinflammatory mediators, but the pro- and antiinflammatory responses are imbalanced.

Coagulation

- Early in sepsis, activation of the coagulation cascade, along with impaired fibrinolysis and anticoagulant systems, results in a hypercoagulable state. As coagulation factors and platelets are depleted, a consumptive, hypocoagulable state follows.

Hepatobiliary

- Decreased perfusion and bacterial endotoxin cause hepatocellular necrosis and intrahepatic cholestasis. Hypoglycemia may occur due to bacterial consumption of glucose, depleted hepatic glycogen stores, and impaired gluconeogenesis.

Nervous

- Mental depression occurs as a late consequence when mechanisms to maintain cerebral blood flow are overwhelmed.

Renal/Urologic

- Renal ischemia may progress to renal failure, which manifests clinically as oliguria or anuria.

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Respiratory

- Respiratory rate increases in response to tissue hypoxia. Respiratory failure manifests as severe impairment of gas exchange.



SIGNALMENT/HISTORY

Risk Factors/Causes

- Recent GI surgery, abdominal trauma, source of infection within the abdomen: gallbladder necrosis, hepatic abscess, prostatic abscess, pyometra, pancreatic abscess, GI obstruction or perforation.

Historical Findings

- Lethargy
- Vomiting, anorexia
- Abdominal pain, abnormal posturing such as hunched posture
- Abdominal distension
- Possible history of recent GI surgery or trauma



CLINICAL FEATURES

- Lethargy
- Fever or hypothermia
- Dehydration
- Abdominal pain (more common in dogs than cats)
- Possible abdominal fluid wave
- Injected or pale mucous membranes
- Fast or prolonged capillary refill time
- Tachycardia
- Strong, short duration or weak peripheral pulses
- Tachypnea
- Incisional discharge following recent abdominal surgery



DIFFERENTIAL DIAGNOSIS

- Gastrointestinal obstruction without peritonitis
- Pancreatitis
- Hemoabdomen
- Sepsis without peritonitis
- Hypovolemic shock without peritonitis
- Splenic torsion
- Mesenteric torsion

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DIAGNOSTICS

- Abdominocentesis: diagnostic test most likely to provide rapid, definitive diagnosis of peritonitis. May be performed with ultrasound guidance or using four quadrant technique by aseptically inserting a 20 gauge needle or catheter into the abdomen (patient standing or in lateral recumbency) (Fig. 90.1). If fluid does not flow through the needle spontaneously, gently aspirate using a 3 mL syringe.
- With peritonitis, the fluid is typically turbid or purulent (Fig. 90.2).



Fig. 90.1. Abdominocentesis and fluid cytology can provide a rapid, definitive diagnosis of peritonitis. A 20 or 22 gauge 1 inch needle is inserted into the abdomen and fluid is allowed to drip out of the open end of the needle and is collected for analysis.

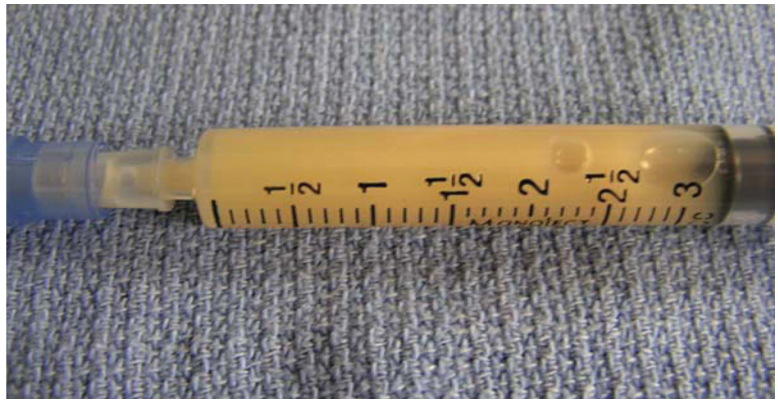


Fig. 90.2. Purulent abdominal fluid from a dog with septic peritonitis secondary to a perforated jejunum and gastrointestinal foreign body.

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- Diagnostic peritoneal lavage: if abdominocentesis is negative but peritonitis is suspected, infuse 22 mL/kg warm isotonic fluid into the abdominal cavity using aseptic technique. Roll patient from side to side and aspirate fluid via abdominocentesis for cytology.
- Abdominal fluid cytology: the presence of degenerative neutrophils with or without bacteria is indicative of peritonitis. Presence of intracellular bacteria confirms septic peritonitis. Bile pigment and plant material may be visualized on cytology with biliary and GI tract rupture, respectively.
- Abdominal fluid culture and sensitivity (aerobic and anaerobic): helpful to guide definitive antibiotic therapy.
- Abdominal fluid creatinine: at least two times higher than serum creatinine with uroabdomen.
- Abdominal fluid bilirubin: at least two times higher than serum bilirubin with biliary tract rupture.
- Ultrasound: free fluid and peritoneal inflammation can be visualized. Source of peritonitis may be identified (i.e., GI foreign body, pyometra, hepatic or prostatic abscess, etc.).
- Abdominal radiographs: loss of abdominal detail indicates the presence of abdominal fluid but not the type of fluid. Free gas may be noted within the abdominal cavity with rupture of hollow viscus or gas-forming bacteria. Generalized ileus may be noted.
- Contrast studies: contraindicated with suspected gastrointestinal tract rupture as barium and iodinated contrast materials can increase severity of peritonitis. Water-soluble iodinated contrast may be used to evaluate the integrity of the urinary tract.
- CBC: leukopenia or leukocytosis with left shift. Thrombocytopenia may be noted due to platelet consumption. Hemoconcentration may be noted due to dehydration.
- Chemistry panel: renal values may be increased due to dehydration or renal failure. Hepatic enzymes and bilirubin may be increased due to decreased hepatic perfusion and cholestasis. Blood glucose may initially be increased due to sympathetic stimulation but then becomes decreased due to hepatic dysfunction, increased metabolic rate, and bacterial consumption of glucose. Albumin is decreased due to increased capillary permeability, GI loss, malnutrition, and reduced hepatic synthesis. The severity of hypoalbuminemia often becomes apparent after fluid resuscitation. Electrolyte imbalances are common.
- Blood gases: metabolic acidosis.
- Lactate levels: increased due to tissue hypoxia, mitochondrial dysfunction, and altered carbohydrate metabolism.
- Abdominal fluid lactate at least 2.0 mmol/L greater than peripheral blood lactate strongly suspicious for septic peritonitis (dogs).
- Abdominal fluid glucose at least 20 mg/dL less than peripheral blood glucose strongly suspicious for septic peritonitis (dogs and cats).
- Coagulation panel: coagulation times may be prolonged due to consumption of coagulation factors.
- Note: free gas may be present within the abdomen for up to 1 month following abdominal surgery and increased TNCC (as high as 10 000 cells/μL) without bacteria or degenerate neutrophils can be noted on abdominocentesis for at least 3 days following abdominal surgery; these factors should be considered when evaluating postoperative patients.

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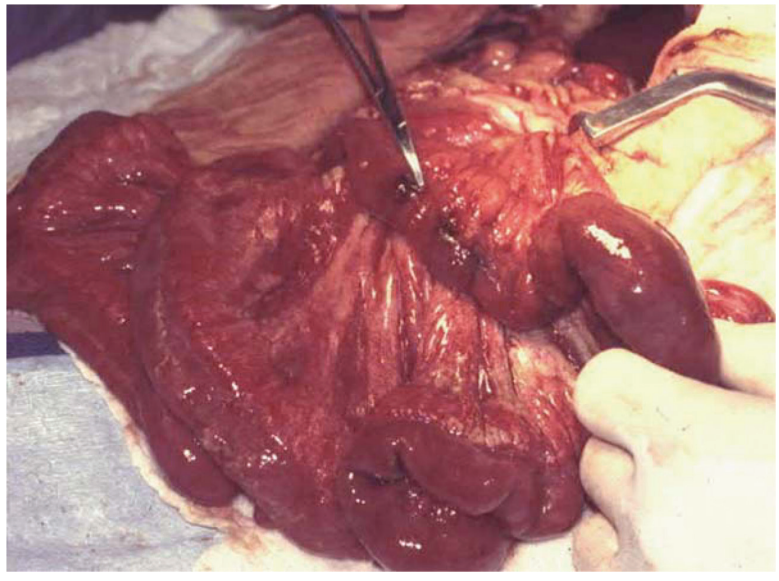


Fig. 90.3. This patient has a generalized peritonitis secondary to a linear foreign body that has perforated the intestine along the mesenteric border at multiple sites. Note the hemorrhagic appearance of the intestines and the petechiae throughout the mesentery.

- Note: in cases of gastroduodenal perforation, cytologic identification of bacteria and glucose/lactate comparisons of the abdominal fluid and peripheral blood may be less reliable due to low bacterial counts in the stomach and duodenum. History (drug use, liver disease) and imaging are important for diagnosis of peritonitis in these cases.

Pathologic Findings

- Free abdominal fluid which may contain plant material or bile.
- Inflammation of the peritoneal lining and serosal surface of the abdominal viscera.
- Omental and fibrin adhesions may be present (Fig. 90.3).
- Other pathology varies depending on the source of the contamination.

THERAPEUTICS

Medical Therapy Overview

- Intravenous fluid therapy with crystalloids and colloids. Colloids are an important part of fluid therapy due to increased capillary permeability and tendency for protein loss into the interstitial space.
- Correct electrolyte imbalances.
- Dextrose supplementation as indicated by blood glucose.
- Broad-spectrum antibiotics; start as soon as peritonitis is suspected.

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- Analgesics.
- Vasopressor and/or inotropic support as needed for hypotension once volume replacement is completed.
- Nutritional support via nasoesophageal, esophageal, gastrostomy, or jejunostomy feeding tube. Parenteral nutrition when enteral is contraindicated.
- Transfusion of blood and/or albumin products as needed to treat coagulation disorders, anemia, or hypoalbuminemia.
- Antiemetics and promotility agents to control vomiting.
- Gastroprotectants.

Surgical Therapy Overview

- Full exploratory laparotomy.
- Surgical correction of the underlying problem to prevent further contamination: resection and anastomosis of intestines with intestinal perforation, cholecystectomy for gallbladder rupture, urinary bladder repair, etc.
- Lavage of the abdomen with warm, sterile crystalloid irrigation solution.
- All foreign material, necrotic tissue, and blood clots should be removed from the abdomen at the time of exploratory surgery.
- Placement of closed-suction drain, open peritoneal drainage, or VAC should be considered to allow continued abdominal drainage (Fig. 90.4).
- Collection of appropriate samples for histopathology and culture.



Fig. 90.4. Closed-suction drainage can effectively be used in the treatment of generalized peritonitis. This is a Jackson–Pratt drain. The fenestrated end of the drain is placed within the abdominal cavity, and the tubing is tunneled through the abdominal wall and connected to the bulb portion of the drain. Squeezing the air from the bulb portion of the drain creates negative pressure and pulls fluid from the abdomen into the bulb ("grenade").

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Drugs of Choice

- Crystalloid fluids: isotonic, balanced electrolyte solution such as Normosol-R, lactated Ringer's solution, or Plasma-Lyte A; administer shock bolus of up to 90 mL/kg (dog) and 60 mL/kg (cat) initially then continue fluids as needed to maintain perfusion.
- Synthetic colloids (hydroxyethyl starches): bolus 5–20 mL/kg (dog) and 2.5–10 mL/kg (cat) for shock followed by CRI of 0.5–2 mL/kg per hour.
- Fresh frozen plasma: 5–15 mL/kg every 8–12 hours for hypocoaguability (prolonged PT/aPTT).
- Human serum albumin (25%): total dose based initially on the albumin deficit or empirical dose of 1.5 g/kg, but use recheck serum albumin levels and COP in addition to clinical assessment to determine actual dose administered. Can be given as initial bolus (1/3 to 1/2 albumin deficit or empirical dose of 0.5–1 g/kg IV over 4 hours) followed by CRI (0.05–0.1 g/kg/h). Monitor for reactions during and after use. Avoid repeat dosing. Exercise caution in cats due to risk of fluid overload.
- Antibiotics: combine drugs for broad-spectrum coverage.
  - Gram-positive: ampicillin (22 mg/kg IV every 8 hours), ampicillin/sublactam (22–30 mg/kg IV every 8 hours), ticarcillin/clavulanic acid (50 mg/kg every 6 hours), clindamycin (10 mg/kg every 12 hours).
  - Gram-negative: enrofloxacin (10–20 mg/kg dog, 5 mg/kg cat IV every 24 hours), amikacin (15 mg/kg IV every 24 hours, renal toxicity), cefotaxime (50 mg/kg IV every 8 hours).
  - Anaerobes: add metronidazole 15 mg/kg IV every 12 hours unless clindamycin chosen above for gram-positive coverage.
  - Base empirical therapy choices on individual hospital's susceptibility patterns for common isolates. Base continued antibiotic therapy on culture and sensitivity results.
- Analgesics: fentanyl CRI 2–5 µg/kg per hour or intermittent IV administration of other pure µ-agonists (methadone, hydromorphone, oxymorphone).
- Vasopressors (CRI in µg/kg/min): dopamine 5–20, norepinephrine 0.05–0.2, dobutamine 5–10 (risk of seizures in cats with doses >5 µg/kg/min).
- Antiemetics: metoclopramide (1–2 mg/kg/day CRI, also promotility), ondansetron (0.1–0.2 mg/kg IV every 6–12 hours), dolasetron (0.5–1 mg/kg IV every 24 hours), maropitant citrate (1 mg/kg SQ every 24 hours; IV use is extra-label but SQ absorption may be poor in this patient population).
- Gastroprotectants: pantoprazole (0.5–1 mg/kg IV every 24 hours), famotidine (0.5–1 mg/kg IV every 24 hours).

Precautions/Interactions

- Corticosteroids increase the risk of GI ulceration, cause immunosuppression, and impair wound healing, so their use is not recommended.
- Nonsteroidal antiinflammatory medications increase the risk of GI ulceration and renal toxicity, so their use is not recommended.

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Diet

- Due to ongoing protein losses and increased metabolic rate, these patients require aggressive nutritional support.
- Consider placement of a feeding tube to allow for enteral nutrition depending on the risks of added anesthesia time and potential tube site leakage.
- Parenteral nutrition can also be used to provide support but may not be readily available and does not help to maintain the intestinal mucosa.

Surgical Considerations

- Use of multifilament suture and surgical gut is contraindicated due to bacterial wicking and rapid absorption, respectively.
- Protect GI surgery sites with omentum or a serosal patch.
- Lavage: use enough volume to remove gross contamination. Excessive lavage can be deleterious. Lactated Ringer's solution is preferred because it is isotonic and balanced. Antiseptics and antibiotics should not be added to lavage fluid.
- Drainage: closed-suction drainage is effective, inexpensive, requires minimal nursing care, and allows abdominal fluid to be quantified and analyzed. Closed-suction drains are indicated in most cases of peritonitis. Open peritoneal drainage or VAC is considered for cases with severe peritonitis and/or when a second laparotomy is needed. VAC therapy decreases the frequency of bandage changes compared to open peritoneal drainage, among other benefits.



COMMENTS

Client Education

- The client should be informed of cost and prognosis prior to proceeding with treatment.

Patient Monitoring

- Mentation, body temperature, pulse, respiration every 3–6 hours.
- Continuous monitoring of vomiting/diarrhea, urine output, volume of abdominal fluid.
- Blood pressure at least every 3–6 hours.
- Continuous electrocardiogram, particularly if arrhythmias are present.
- PCV, total protein every 12 hours.
- Blood glucose every 6–12 hours.
- Electrolytes every 12 hours.
- Coagulation parameters every 12–24 hours.
- Albumin +/- select chemistry panel values every 24 hours.
- Abdominal fluid cytology: evaluate every 24 hours for white blood cell morphology and presence or absence of bacteria.

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Possible Complications

- Dehiscence of GI surgery sites
- Anemia
- Hypoproteinemia
- Hypoglycemia
- Hypotension
- Cardiac arrhythmias
- Altered coagulation: hyper- or hypocoagulability
- Multiple organ failure

Expected Course and Prognosis:

- Recent studies indicate survival rates of approximately 60% with surgery and aggressive therapy.
- Death is due to organ failure, often in the immediate postoperative period.

Abbreviations

CBC: complete blood count
COP: colloid osmotic pressure
CRI: constant rate infusion
GI: gastrointestinal
IV: intravenous
PCV: packed cell volume
TNCC: total nucleated cell count
VAC: vacuum-assisted closure

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chapter 91

Serotonin Syndrome

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DEFINITION/OVERVIEW

- Serotonin syndrome is a complex of clinical signs resulting from overstimulation of serotonin receptors in the central and peripheral nervous systems.
- In humans, serotonin syndrome is defined as a combination of symptoms that includes at least three of the following: myoclonus, mental aberration (dementia, disorientation, etc.), agitation, hyperreflexia, tremors, diarrhea, ataxia, or hyperthermia.

ETIOLOGY/PATHOPHYSIOLOGY

- Serotonin syndrome is caused by excessive stimulation of serotonin receptors. This most commonly occurs in pets following inappropriate ingestion of pharmaceuticals. Such pharmaceuticals can include drugs that enhance serotonin synthesis (i.e., L-tryptophan, L-5-hydroxytryptophan), drugs that increase presynaptic serotonin release (i.e., amphetamines and derivatives, MAOIs, cocaine), drugs that inhibit serotonin uptake into the presynaptic neuron (i.e., SSRIs, TCAs, amphetamines, cocaine, dextromethorphan, meperidine), drugs that inhibit serotonin metabolism (i.e., MAOIs), and drugs that act as serotonin agonists (i.e., buspirone, sumatriptin, LSD) (Table 91.1).

Systems Affected

- Gastrointestinal: increased smooth muscle contractility.
- Nervous: stimulation and altered mental status; increased autonomic function.
- Cardiovascular: decreased vascular tone, increased cardiac stroke rate and volume.
- Neuromuscular: autonomic dysfunction (hyperactivity).
- Respiratory: increased bronchial smooth muscle contraction.

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TABLE 91.1. Mechanism of action of serotoninergic drugs.

Action	Drug
Increase serotonin synthesis	L-tryptophan
	L-5-hydroxytryptophan (5-HTP)
Increase serotonin release	Amphetamines
	Dextroamphetamine and amphetamine (Adderall®)
	Lisdexamfetamine (Vyvanse®)
	Methamphetamine (Crystal)
	Methylphenidate (Ritalin®)
	N-Methyl-3,4-methylenedioxyamphetamine (MDMA, Ecstasy)*
	Cocaine
	Dexfenfluramine (Redux®)
	Fenfluramine (Pondimin®)*
	Reserpine
	Dextromethorphan (DXM)*
Decrease metabolism	Amphetamine metabolites
	Monoamine oxidase inhibitors
	Isocarboxazid (Marplan®)
	Linezolid (Zyvox®)
	Moclobemide (Manerix® Canada)
	Pargyline (Eutonyl®)
	Phenelzine (Nardil®)*
	Selegiline (Eldepryl®, Anipryl®)*
	Tranylcypromine (Parnate®)*
	St John's wort (*Hypericum punctatum*)
Inhibit serotonin uptake	Tricyclic antidepressants
	Amitriptyline (Elavil®)*
	Amoxapine (Asendin®)
	Clomipramine (Anafranil®, Clomicalm®)
	Desipramine (Norpramin®)
	Doxepin (Sinequan®, Adapin®)
	Imipramine (Tofranil®)*
	Maprotiline (Ludiomil®)
	Nortriptyline (Pamelor®)
	Protriptyline (Vivactil®)
	Trimipramine (Surmontil®)
	Selective serotonin reuptake inhibitors
	Citalopram (Celexa®)
	Escitalopram (Lexapro®)
	Fluoxetine (Prozac®)*

(*Continued*)

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TABLE 91.1. *Continued*

Action	Drug
	Fluvoxamine (Luvox®)
	Kanna (*Sceletium tortuosum*)
	Paroxetine (Paxil®)*
	Sertraline (Zoloft®)*
	Other serotonin uptake inhibitors
	Amphetamines
	Cocaine
	Desvenlafaxine (Pristiq®)
	Dextromethorphan
	Duloxetine (Cymbalta®)
	Meperidine (Demerol®)
	Milnacipran (Savella®)
	Mirtazapine (Remeron®)
	Nefazodone (Serzone®)
	Risperidone (Risperdal®)
	Sibutramine (Meridia®)
	Tramadol (Ultram®)
	Trazodone (Desyrel®)
	Venlafaxine (Effexor®)*
Direct serotonin receptor agonists	Almotriptan (Axert®)
	Buspirone (Buspar®)
	Eletriptan (Relpax®)
	Frovatriptan (Frova®)
	Lysergic acid diethylamide (LSD)
	Naratriptan (Amerge®)
	Rizatriptan (Maxalt®)
	Sumatriptan (Imitrex®)
	Zolmitriptan (Zomig®)
	Phenylalkylamines (mescaline)
Dopamine agonists	Amantadine (Symmetrel®)
	Bromocriptine (Parlodel®)
	Bupropion (Wellbutrin®)
	Levodopa (L-dopa)
	Pergolide (Permax®)
	Pramipexole (Mirapex®)
Nonspecific serotoninergic agents	Lithium (Lithobid®, Eskalith®)*

*High risk of serotonin syndrome.

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SIGNALMENT/HISTORY

- No breed, age, or sex predilection.
- Vomiting, diarrhea, seizures, hyperthermia, hyperesthesia, depression, mydriasis, vocalization, agitation, tremors, blindness, hypersalivation, respiratory distress, ataxia/paresis, disorientation, death, hyperreflexia, and coma.

Risk Factors/Causes

- Increased risk in animals with underlying hepatic or renal disease.
- Increased risk of signs if animal is already on a serotonergic drug.

Historical Findings

- Lethargy or agitation, mydriasis, ataxia, tremors, vomiting, diarrhea, hypersalivation, anorexia, seizures, nystagmus.



CLINICAL FEATURES

- Hyperthermia, pale mucous membranes, tachycardia, mydriasis, vomiting, diarrhea, agitation, tremors, hypersalivation, seizures, nystagmus.



DIFFERENTIAL DIAGNOSIS

- Toxicologic differentials include metaldehyde, lead, athylene glycol, hops, anticholinergic, or antihistamine toxicosis.
- Other disease processes such as malignant hyperthermia, heat stroke, meningitis (i.e., rabies, canine distemper, etc.), and neoplasia can cause similar CNS signs.



DIAGNOSTICS

- There are no diagnostic tests to confirm serotonin syndrome.
- Diagnosis is based on history of ingestion of serotonergic drugs and presence of appropriate clinical signs.
- Testing for various pharmaceuticals can be performed, but due to the turnaround time and lack of diagnostic levels in pets, this is rarely done.
- Electrolytes, BUN, creatinine, CK, urinalysis, and blood gases; assess metabolic acidosis and rhabdomyolysis.

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THERAPEUTICS

- Prevent development of signs or decrease the severity of signs once they occur by decreasing serotonin levels.

Drugs of Choice

- Emesis (if asymptomatic and recent ingestion) or gastric lavage.
- Activated charcoal with cathartic (may need to repeat).
- Phenothiazines (acepromazine 0.025–0.05 mg/kg IV, titrate up as needed) or benzodiazepines (diazepam 0.5–2 mg/kg IV) can be used to control agitation.
- Cyproheptadine is a nonselective serotonin antagonist (dog 1.1 mg/kg, cat 2–4 mg PO every 4–6 hours; may give rectally if vomiting). Do not give more than three doses.

Precautions/Interactions

- Contraindicated drugs (high risk of serotonin syndrome): amphetamines, SSRIs, MAOIs, TCAs, hydroxytryptophan, clarithromycin, dextromethorphan, lithium, St John's wort.
- Use with caution (low risk of serotonin syndrome): amantadine, bupropion, carbamazepine, codeine, and tramadol.
- Drug interactions: cimetidine (decreased metabolism of serotonergic drugs), class 1C antiarrhythmics (propafenone, flecainide – increased cardiac arrhythmias), metoprolol (bradycardia, hypotension), quinidine (decreased metabolism of serotonergic drugs), and theophylline (increased theophylline levels).

Diet

- NPO as needed with severe CNS signs.

Activity

- Confine until clinical signs resolve.

COMMENTS

Client Education

- No additional or long-term problems expected after recovery.

Patient Monitoring

- Heart rate, blood pressure, respirations, and temperature; take hourly at first and then as needed.

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Prevention/Avoidance

- Prevent access to serotonergic and all other medications.

Possible Complications

- Sequelae include rhabdomyolysis, DIC, and renal failure secondary to myoglobinuria.

Expected Course and Prognosis

- Prognosis is generally good with quick, aggressive therapy and most animals will recover over 12–24 hours. More severely affected animals may develop sequelae or die.

Synonyms

- Serotonin toxicosis, serotonin toxidrome, serotonin storm

Abbreviations

BUN: blood urea nitrogen
CK: creatinine kinase
CNS: central nervous system
DIC: disseminated intravascular coagulation
IV: intravenous
LSD: d-lysergic acid diethylamide
MAOI: monoamine oxidase inhibitor
NPO: nothing by mouth (nil per os)
PO: by mouth (per os)
SSRI: selective serotonin reuptake inhibitor
TCA: tricyclic antidepressant

Suggested Reading

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chapter 92

Shock – Cardiogenic



DEFINITION/OVERVIEW

- Shock is defined as any condition that results in inadequate effective circulating volume and oxygen delivery to the tissues. Shock has been broken down into several classification categories. In any classification scheme, the underlying problem is that the oxygen delivery is not sufficient to meet the oxygen needs of the tissues. This leads to a cellular oxygen and energy debt. It is important to note that although there may be one predominant category in which a specific patient falls, one must consider all aspects of each of the categories when formulating a treatment plan. For example, a patient with severe gastrointestinal disease and hemorrhagic diarrhea may initially present in hypovolemic shock, but as the clinical disease progresses, the gastrointestinal–blood barrier may become compromised, leading to bacterial translocation, systemic septicemia, and septic shock. This can then go on to affect the heart and lead to impaired myocardial contractility, thus exhibiting signs of cardiogenic shock. So although the different categories of shock will be presented, care must be taken to approach each patient with an open mind and the realization that the categories may overlap considerably.
- Cardiogenic shock refers to impaired tissue perfusion secondary to decreased cardiac output and diminished stroke volume.
- Distributive shock describes a condition of relative hypovolemia in which there is massive vasodilation resulting in pooling of the circulating blood volume and impaired tissue perfusion and oxygen delivery.



ETIOLOGY/PATHOPHYSIOLOGY

- Cardiogenic shock occurs secondary to anything that results in impaired cardiac output and compromised tissue perfusion.
- DCM is one of the most common causes of cardiac disease in large breed dogs.
    - DCM is characterized by decreased myocardial contractility, with subsequent ventricular dilation in an effort to maintain cardiac output.
    - Tachyarrhythmias are common with DCM.

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  - Impairs forward blood flow and results in decreased tissue perfusion.
  - DCM is currently one of the least common forms of heart disease in the cat but can still be associated with taurine deficiency.
  - DCM in cats can also present as the final stages of hypertrophic or unclassified cardiomyopathy.
- Hypertrophic cardiomyopathy is one of the most common causes of cardiac disease in the cat.
  - In severe cases, the inability of the ventricle to adequately relax during diastole leads to a decrease in cardiac output secondary to insufficient filling. The impaired cardiac output, combined with the congestive heart failure that occurs secondary to the poor relaxation, leads to significant decrease in oxygen delivery to the tissues.
- Pericardial tamponade is a condition in which pressure is placed on the heart from fluid accumulation in the pericardial sac.
  - This pressure prevents the heart from filling appropriately during diastole, causing a subsequent decrease in cardiac output. This may occur secondary to hemangiosarcoma (most commonly affecting the right atrial appendage or right atrium), heart base tumors, idiopathic or other neoplasia (lymphosarcoma, mesothelioma, other), right atrial rupture due to chronic valvular disease, or trauma.
- Patients with severe tachy- or bradyarrhythmias may not have adequate diastolic filling time or appropriate heart rate to provide cardiac output, resulting in secondary impaired perfusion of the tissues.
- Severe ventricular outflow or inflow obstruction secondary to neoplasia, thrombus, heartworm, anatomic abnormalities, stenosis, or granulomas.
- Infective endocarditis can result in significant valvular impairment, thrombus formation, or myocardial depression secondary to sepsis.
- Sepsis-induced myocardial depression is characterized by impaired contractility that leads to decreased cardiac output and potentially congestive heart failure.
- The compensatory responses of the body in cases of impaired cardiac output include tachycardia, peripheral vasoconstriction, and activation of the renin-angiotensin-aldosterone system. These compensatory responses increase the work of the heart and can further exacerbate the decrease in tissue perfusion.

Systems Affected

- Cardiovascular: the primary or secondary cardiac disease associated with cardiogenic shock is the cause of the impaired cardiac output. Decreased coronary blood flow further exacerbates the myocardial hypoperfusion and dysfunction and can lead to myocardial ischemia, dysrhythmias, and thrombi formation.
- Pulmonary: congestive heart failure and associated pulmonary edema are common in patients with cardiogenic shock. This decrease in the ability of the patient to oxygenate and subsequent hypoxia further amplify the decreased tissue perfusion.
- Renal: when the mean arterial blood pressure decreases below 60 mmHg, the kidneys are no longer able to autoregulate and hypotension directly impairs the blood flow and oxygen

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delivery. The kidneys normally receive 20–25% of the cardiac output, and when this is decreased the kidneys are extremely sensitive to impaired perfusion. This decrease in perfusion may result in renal ischemia and acute tubular necrosis, renal failure, and oliguria.

- Gastrointestinal: impaired splanchnic perfusion and decreased oxygen delivery can result in significant damage to the mucosa, ulceration, the loss of the intestinal–blood barrier and subsequent bacterial translocation, intestinal necrosis, and hemorrhage.
- Hepatic: decreased cardiac output results in decreased hepatic blood flow and hepatic congestion. Can lead to hepatic ischemia and impaired hepatic function, including decreased gluconeogenesis and subsequent hypoglycemia, decreased clotting factor production, decreased protein synthesis, and hyperbilirubinemia.
- Nervous: central nervous system dysfunction is a significant complication in patients with inadequate oxygen delivery. Coagulation abnormalities associated with cardiogenic shock can lead to vascular events (thrombosis or hemorrhage) that affect the central nervous system. This can result in cerebral ischemia and associated manifestations such as stupor, blindness, and decreased spinal reflexes. The permanence of these neurologic abnormalities is again related to the severity and duration of the shock event.
- Musculoskeletal: musculoskeletal weakness is a common complication in patients with decreased perfusion and oxygen delivery. Weakness may be profound, and patients should not be stressed until their perfusion is improved. In patients with a dynamic inflow or outflow obstruction, this weakness may be more pronounced when the patient has an increase in activity.



SIGNALMENT/HISTORY

- There is no age, breed, or sex predilection for cardiogenic shock.

Risk Factors/Causes

- Preexisting cardiac disease, particularly DCM.
- Severe systemic disease associated with a high risk of sepsis.

Historical Findings

- Variable pending the underlying cause of the cardiogenic shock
- A previous history of cardiac disease (murmur, dysrhythmia)
- Collapse, syncope, possibly associated with activity
- Respiratory distress



CLINICAL FEATURES

- Tachycardia or bradycardia.
- Dysrhythmia or pulse deficits.
- Pulsus paradoxus: a decrease in pulse pressure associated with inspiration, an increase in pulse pressure associated with expiration. This is generally associated with pericardial effusion.

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- Pale mucous membranes.
- Prolonged capillary refill time.
- Weak peripheral pulses.
- Distended jugular veins or jugular pulses.
- Tachypnea, crackles.
- Cough.
- Decreased arterial blood pressure.
- Hypothermia.
- Marked muscular weakness.
- Dull mentation.
- Cool extremities.



DIFFERENTIAL DIAGNOSIS

- Hypovolemic shock: often associated with a history of vomiting, diarrhea, blood loss, or fluid loss. Patients are often tachycardic or bradycardic (cats) but dysrhythmias or pulmonary edema are less common.
- Distributive shock: may present similarly to hypovolemic shock; may present with other clinical signs of infection such as abdominal pain.
- Hypoxic shock: patients in hypoxic shock have severe respiratory distress, with increased lung sounds, crackles, or cyanosis.



DIAGNOSTICS

- Thoracic radiographs may reveal cardiomegaly, distended pulmonary vasculature, pulmonary edema, or pleural effusion. In cases of DCM or pericardial effusion, a globoid heart may be appreciated.
- Echocardiography may show a decrease in cardiac contractility, inflow or outflow obstruction, valvular lesions, or pericardial effusion/tamponade.
- Electrocardiography may show dysrhythmias (atrial fibrillation common in cases of DCM), sinus tachycardia, or electrical alternans (alternation of QRS complex size between beats, associated with pericardial effusion).
- Blood lactate: in cases of impaired oxygen delivery to the tissues and disturbances in tissue perfusion, anaerobic metabolism results in the production of lactate. The normal lactate in adult dogs and cats is less than 2.5 mmol/L and levels over 7 mmol/L indicate severe tissue hypoxia. In resuscitation, serial measurements of blood lactate can help measure the effectiveness of therapy.
- Electrolytes: hyperkalemia may occur in patients with severe renal injury and oligoanuria. Pericardial effusion can lead to hyponatremia and hyperkalemia. Lasix therapy can result in hypochloremia and hypokalemia.
- Complete blood count may show a stress leukogram.

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- Liver enzyme activities are commonly elevated secondary to impaired hepatic perfusion.
- Renal values may be elevated either secondary to prerenal azotemia (associated with a concentrated urine specific gravity >1.025 in dogs, >1.035 in cats), or primary renal azotemia secondary to renal hypoxia and ischemic injury.
- Central venous pressure can help in assessing volume status objectively and monitoring for risk of congestive heart failure, with a goal of 5–10 cmH_2O in dogs and 2–5 cmH_2O in cats.
- A urinary catheter may be placed to monitor urine output if there is concern for oliguria or anuria. The goal is at least 0.5–1 mL/kg per hour.

Pathologic Findings

- Myocardial fibrosis and scarring may be present, as well as ischemia secondary to impaired myocardial blood flow and increased work of the myocardial cells to try and compensate for the decrease in cardiac output.
- Pulmonary edema and interstitial pneumonia may be seen in cases secondary to congestive heart failure, hypoxia, and ischemia in the pulmonary cells.
- Hyperemia and ulceration of the intestinal mucosa may occur secondary to impaired splanchnic perfusion and inflammatory mediators.
- Acute tubular necrosis occurs secondary to decreased renal blood flow and impaired oxygen delivery to the tubular cells.



THERAPEUTICS

- The objective of treatment is to improve cardiac output and oxygen delivery to the tissues.
- Pericardiocentesis is imperative for treatment of any patient with pericardial tamponade.
- Positive inotropic support should be provided if contractility is poor and associated with hypotension.
- Antiarrhythmia medications to control rate if arrhythmia is associated with severe pulse deficits, inadequate time for ventricular filling, or hypotension.
- Congestive heart failure should be treated to improve oxygenation and stabilize the patient.
- Oxygen supplementation, via mask, oxygen cage, or oxygen hood.

Drugs of Choice

- Positive inotropic support may be provided with pimobendan (dogs only), a calcium-sensitizing agent, dose 0.1–0.3 mg/kg orally every 12 hours, or dobutamine at 2–20 µg/kg per minute in dogs and 1–5 µg/kg per minute in cats.
- Treatment of the congestive heart failure with a diuretic, furosemide 2–4 mg/kg IV or IM every 30 minutes to correct failure, then 2 mg/kg every 6–12 hours.
- In severe cases, nitroprusside can be used in dogs and cats as a vasodilator to treat congestive heart failure and improve cardiac output by reducing afterload, at a dose of 2–10 µg/kg

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per minute. This is a potent vasodilator so blood pressure should be monitored closely to watch for hypotension. If hypotension occurs, the nitroprusside should be stopped immediately. In addition, sustained nitroprusside therapy can result in cyanide toxicity, particularly in patients with renal impairment.

- Supraventricular arrhythmias may be treated with diltiazem: dogs 0.25 mg/kg IV bolus over 2 minutes, repeat 0.25 mg/kg IV every 15 minutes until conversion occurs or up to a total dose of 0.75 mg/kg; cats 0.5–1 mg/kg, up to 1.5 mg/kg PO or per rectum every 8 hours.
- Oxygen supplementation should be provided; this can be done via oxygen mask, oxygen cage, or nasal cannula. In cases of severe respiratory distress unresponsive to diuretic therapy or in which respiratory arrest is imminent, sedation, intubation, and mechanical ventilation are warranted.

Precautions/Interactions

- In patients in cardiogenic shock, blood pressure must be monitored closely and continuously when the patient is on nitroprusside to avoid hypotension.
- Treatment of the tachycardia may result in hypotension if the tachycardia is a compensatory response to poor cardiac output. Treatment with a positive inotrope should be instituted to provide myocardial support.
- Nonsteroidal antiinflammatory medications should be avoided in patients with compromised perfusion to decrease risk of renal and gastrointestinal complications.

Diet

- Once the patient has stabilized, enteral or parenteral nutrition should be instituted to address the nutritional needs of the patient.

COMMENTS

Client Education

- Owners should be warned of the risk of sudden death in any patient that presents in cardiogenic shock.
- In patients that experience a severe hypotensive state and are discharged with residual neurologic impairments, the owner needs to be educated that full neurologic recovery may take weeks to months.

Patient Monitoring

- Serial lactate measurements every 8–12 hours once normalized.
- Central venous pressure: either continuous or every 8–12 hours to monitor.
- Blood pressure monitoring: every 8–12 hours to monitor for hypotension.
- ECG: every 8–12 hours or continuous to monitor for dysrhythmias.

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Possible Complications

- Renal dysfunction
- Cardiac dysrhythmias
- Gastroenteritis, hemorrhagic gastroenteritis
- Bacterial translocation, sepsis
- Residual neurologic impairment
- Cardiac arrest

Expected Course and Prognosis

- Variable depending on the underlying cause and severity of clinical condition.

Abbreviations

DCM: dilated cardiomyopathy
ECG: electrocardiogram
IM: intramuscular
IV: intravenous
PO: by mouth

See Also

Shock – Distributive
Hypertrophic and Restrictive Cardiomyopathy
Pericardial Effusion

Suggested Reading

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chapter 93

Shock – Distributive



DEFINITION/OVERVIEW

- Shock occurs when the patient is unable to provide effective circulating volume and oxygen delivery to the tissues. There are numerous classification categories for shock. In any case, the underlying problem is that the oxygen delivery is not sufficient to meet the oxygen needs of the tissues, resulting in a cellular oxygen and energy debt. It is important to note that although there may be one predominant category in which a specific patient falls, one must consider all aspects of each of the categories when formulating a treatment plan. For example, patients with septic shock often have significant protein and fluid losses resulting in hypovolemic shock, as well as myocardial dysfunction that may present as cardiogenic shock. So although the different categories of shock will be presented, care must be taken to approach each patient with an open mind and the realization that in any patient the categories may overlap considerably.
- Distributive shock describes a condition of relative hypovolemia in which there is massive vasodilation resulting in pooling of the circulating blood volume and impaired tissue perfusion and oxygen delivery.



ETIOLOGY/PATHOPHYSIOLOGY

- Distributive shock has numerous etiologies. The most common cause is septic shock, but it can also occur in anaphylactic and neurogenic shock.
- Sepsis is defined as a SIRS secondary to an infectious process. Septic shock is a progression of this inflammatory response resulting in cardiovascular dysfunction and compromise.
- Sepsis can be caused by gram-positive, gram-negative, aerobic or anaerobic infections, but gram-negative infections are a common cause of septic shock. In gram-negative infections, endotoxin, or LPS, is a component of the outer bacterial cell membrane and plays a significant role in stimulation of the inflammatory cascade.
- Endotoxin also stimulates the release of inflammatory cytokines such as TNF-alpha, IL-1, and IL-6. These cytokines have numerous systemic effects including vasodilation, myocardial depression, increased gut permeability, and pulmonary inflammation.

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- Common causes of septic shock include septic peritonitis resulting from rupture of gastrointestinal viscera and leakage of intestinal contents or rupture of the urinary or gallbladder with a concomitant urinary tract or biliary infection, pyometra, pneumonia, prostatic infection/abscess, pyelonephritis, meningitis, bite wounds, bacterial endocarditis, or bacterial translocation from severe gastrointestinal disease.
- Anaphylaxis is a severe allergic reaction to an allergen, antigen, drug or foreign protein that leads to release of histamine, extensive vasodilation, and increased capillary permeability.
- Neurogenic shock occurs when severe trauma to the spinal cord results in loss of autonomic and motor reflexes below the injury level. The lack of stimulation by the sympathetic nervous system leads to systemic vasodilation and hypotension. This form of distributive shock is very rare.

Systems Affected

- Cardiovascular: sepsis leads to the release of inflammatory cytokines that can cause direct myocardial dysfunction. This sepsis-induced myocardial depression is characterized by impaired contractility leading to decreased cardiac output and potential congestive heart failure. In humans, this myocardial depression is completely reversible if the underlying sepsis resolves. These cytokines also have direct effects on the vasculature, leading to vasodilation and capillary leak. In addition, the lactic acidosis that develops secondary to poor perfusion and impaired oxygen delivery to the tissues can impair the vascular response to catecholamines.
- Pulmonary: inflammatory cytokines, such as TNF-alpha have been shown to lead to capillary leak and acute lung injury/ARDS. Additionally, the myocardial dysfunction secondary to sepsis can result in pulmonary edema secondary to increased pressure in the pulmonary vascular bed.
- Renal: when the mean arterial blood pressure decreases below 60 mmHg, the kidneys are no longer able to autoregulate and hypotension directly impairs renal blood flow and oxygen delivery. The kidneys are extremely sensitive to impaired perfusion, and this may result in renal ischemia and acute tubular necrosis, renal failure, and oliguria. The significant systemic inflammatory response can also lead to differential vasodilation and vasoconstriction in the vascular beds, further diminishing oxygen delivery to the kidneys.
- Gastrointestinal: the splanchnic circulation is also compromised in cases of impaired perfusion, systemic inflammation, impaired vascular tone, and elevations in circulating cytokines. This can result in significant damage to the mucosa, ulceration, the loss of the intestinal–blood barrier, and subsequent bacterial translocation, intestinal necrosis, and hemorrhage. The combination of decreased splanchnic perfusion, pooling of blood in vascular beds, and significant systemic inflammation also affects the pancreas and can lead to pancreatitis and further release of inflammatory mediators into both the abdominal cavity and the peripheral circulation.

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- Hepatic: the liver is particularly sensitive to the impaired perfusion and circulating inflammatory mediators that occur in distributive shock. In addition, the inflammatory cytokines also have a direct effect on the hepatocytes and can lead to sepsis-induced cholestasis, hepatocellular dysfunction, and death. This may cause impaired hepatic function including a decrease in gluconeogenesis and subsequent hypoglycemia, decrease in the production of clotting factors, decreased protein synthesis, and hyperbilirubinemia.
- Nervous: central nervous system dysfunction is a significant complication in patients with impaired oxygen delivery and disturbances of the oxygen delivery to consumption ratio. In addition, the coagulation abnormalities associated with sepsis and severe systemic inflammation can lead to vascular events (i.e., thrombosis or hemorrhage) affecting the central nervous system. This can result in cerebral ischemia and associated manifestations such as stupor, blindness, and decreased spinal reflexes. The permanence of these neurologic abnormalities is again related to the severity and duration of the shock event.
- Musculoskeletal: musculoskeletal weakness is a common complication in patients with decreased perfusion and oxygen available to the myocytes. This weakness may be profound, and patients should not be stressed until their perfusion is improved.
- Endocrine/metabolic: relative adrenal insufficiency is a well-documented complication of sepsis and septic shock. Although the cause of adrenal insufficiency remains unknown, the most likely culprit is a cytokine-induced decrease in the synthesis or release of corticotropin-releasing hormone, ACTH, and cortisol. This has been associated with an increased vasopressor requirement, prolonged hospitalization, and increased mortality. Diagnosis is a decreased responsiveness to ACTH stimulation, although a definitive level at which relative adrenal insufficiency is defined has not been identified.



SIGNALMENT/HISTORY

- There is no age, breed, or sex predilection for distributive shock.

Risk Factors/Causes

- Preexisting immunosuppression, due to infectious disease (such as feline leukemia virus, feline immunodeficiency virus, or parvovirus), concurrent clinical conditions such as hyperadrenocorticism, diabetes mellitus, or medications such as long-term steroid use or chemotherapy.

Historical Findings

- Variable depending on the underlying cause of the distributive shock.
- Vomiting, painful, or distended abdomen.
- Previous surgery.
- Urinary tract signs in cases of urosepsis.

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- Concurrent conditions associated with immunosuppression such as feline leukemia, feline immunodeficiency virus, hyperadrenocorticism, or diabetes mellitus.
- Long-term steroid use or chemotherapy.
- Current or previous history of an allergic reaction (i.e., swelling, urticaria, erythema) associated with an antigenic stimulus (i.e., vaccination, insect bite, medication).



CLINICAL FEATURES

Early or Hyperdynamic Shock

- Tachycardia
- Strong peripheral pulses
- Rapid capillary refill time
- Bright red mucous membranes
- Normal to increased arterial blood pressure
- Normal mentation
- Normal muscular activity and ability
- Tachypnea
- Normal to increased rectal temperature
- This stage is rarely identified in cats.

Late or Decompensatory Shock

- Tachycardia or bradycardia (particularly in cats)
- Pale mucous membranes
- Prolonged capillary refill time
- Weak peripheral pulses
- Tachypnea
- Decreased arterial blood pressure
- Hypothermia (particularly in cats)
- Abdominal pain (cats)
- Marked muscular weakness
- Dull mentation
- Cool extremities
- Evidence of coagulopathy: petechiae, ecchymosis, hyphema, or epistaxis



DIFFERENTIAL DIAGNOSIS

- Cardiogenic shock may be associated with a preexisting cardiac condition, pulmonary edema, muffled heart sounds, or arrhythmias.
- Hypovolemic shock often presents very similarly to distributive shock and initial therapy is similar while investigating the underlying cause.

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- Hypoxic shock: patients in hypoxic shock are often severely orthopneic, with increased lung sounds, crackles, or cyanosis.



DIAGNOSTICS

- Blood lactate: in cases of impaired oxygen delivery to the tissues and disturbances in tissue perfusion, anaerobic metabolism results in the production of lactate. The normal lactate in adult dogs and cats is less than 2.5 mmol/L and levels over 7 mmol/L indicate severe tissue hypoxia. In resuscitation, serial measurements of blood lactate can help measure the effectiveness of therapy. Monitoring the lactate every 1–2 hours as resuscitation is performed can help guide the clinician in the resuscitation efforts and monitoring the lactate every 6–8 hours once it has normalized may alert the clinician to new problems if there is an acute increase in lactate levels. It is important to note that in patients with severely compromised perfusion, the lactate may initially rise after starting fluid therapy and the improved perfusion flushes lactate out of the poorly perfused periphery. This is a transient increase and as resuscitation continues and blood supply to the tissues increases, the levels should decrease to normal.
- Packed cell volume and total solids: in cases of severe systemic inflammation, vasodilation and increased vascular permeability, hypoproteinemia is common. In addition, underlying disease such as peritonitis, gastritis, and hepatic insufficiency exacerbate the decrease in serum protein levels. The hypoproteinemia may be present initially or may develop secondary to ongoing protein loss, decreased production by the liver, decreased intake through the gastrointestinal tract, and dilution from fluid administration. In addition, blood loss through the gastrointestinal tract or other ongoing losses can be exacerbated by blood sampling (particularly in cats) and decreased erythropoietin due to critical illness. Serial monitoring is important to monitor protein levels and identify when additional therapies such as colloid therapy or transfusions are indicated.
- Electrolytes: hyperkalemia may occur in patients with severe renal injury and oligoanuria, hypernatremia, and hyperchloremia may be seen in cases with severe dehydration and water loss.
- Complete blood count may show evidence of anemia, a neutrophilia with a left shift associated with infection, leukopenia in severe sepsis, and thrombocytopenia.
- Liver values are commonly elevated secondary to impaired hepatic perfusion.
- Renal values may be elevated secondary to prerenal azotemia (associated with a concentrated urine specific gravity >1.025 in dogs, >1.035 in cats) or primary renal azotemia secondary to renal hypoxia and ischemic injury.
- Central venous pressure can be an aid in assessing volume status objectively, with a goal of 5–10 cmH_2O in dogs and 2–5 cmH_2O in cats.
- Coagulation times (PT and PTT) to investigate for any coagulopathies.
- A urinary catheter may be placed to monitor urine output if there is concern for oliguria or anuria. The goal is at least 0.5–1 mL/kg per hour.

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- Thoracic radiographs may show pulmonary infiltrate associated with acute lung injury, pneumonia, or other parenchymal disease.
- Abdominal radiographs may show evidence of free air in the abdomen if there is a rupture of the gastrointestinal tract associated with septic peritonitis.
- ECG may document impaired myocardial contractility secondary to the sepsis-associated myocardial dysfunction.
- ECG to evaluate for any cardiac arrhythmias secondary to myocardial ischemia or electrolyte abnormalities.

Pathologic Findings

- Hyperemia and ulceration of the intestinal mucosa may occur secondary to impaired splanchnic perfusion and inflammatory mediators.
- Acute tubular necrosis occurs secondary to decreased renal blood flow and loss of autoregulation leading to impaired oxygen delivery to the tubular cells.
- Interstitial pneumonia may be seen in cases secondary to hypoxia and ischemia in the pulmonary cells with subsequent release of inflammatory mediators.
- Myocardial ischemia secondary to impaired myocardial blood flow and increased work of the myocardial cells to try to compensate for the decrease in cardiac output.



THERAPEUTICS

- The objective of treatment is to provide intravascular fluid volume to maintain cardiac preload and thereby blood flow and oxygen delivery to the tissues.
- Identification and treatment of the underlying source of infection are essential in the treatment of septic shock. This may include surgical drainage or resection of infected tissues or aggressive antibiotic therapy in cases of systemic infections such as pneumonia or pyelonephritis.
- Crystalloid therapy with a balanced isotonic solution at an initial rate of up to 90 mL/kg in dogs and 60 mL/kg in cats
- It should be noted that intravenous crystalloids should be used judiciously in cats because the lung is the shock organ in the cat and fluid overload and pulmonary edema can rapidly occur, particularly with concurrent hypotension and hypothermia. An initial bolus of 15–20 mL/kg every 15–20 minutes up to 60 mL/kg is often effective at providing fluid resuscitation while avoiding fluid overload and pulmonary edema.
- In patients that are hypoproteinemic or remain volume depleted despite crystalloid therapy, colloids such as hetastarch may be used as a bolus at a rate of 5–10 mL/kg in dogs and 3–5 mL/kg in cats. The total colloid administered should be less than 20 mL/kg per day to avoid secondary coagulopathies.
- Hypertonic saline is another hyperoncotic solution and may be administered at a rate of 5–10 mL/kg. The effects of this fluid are rapid but short-lived and care should be taken in hypernatremia patients.
- Oxygen supplementation, via mask, oxygen cage, or oxygen hood.

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Drugs of Choice

- In cases that remain hypotensive despite fluid resuscitation, vasopressor therapy with dopamine 5–20 µg/kg per minute, norepinephrine 0.05–2 µg/kg per minute, or epinephrine 0.005–1 µg/kg per minute may be utilized. Alternatively, positive inotropy may also be provided using dobutamine 2–20 µg/kg per minute in dogs and 1–5 µg/kg per minute in cats.

Precautions/Interactions

- Nonsteroidal antiinflammatory medications should be avoided in patients with compromised perfusion to decrease risk of renal and gastrointestinal complications.

Diet

- Once the patient has stabilized, enteral or parenteral nutrition should be instituted to address the hypermetabolic state the patient is in.

Surgical Considerations

- Aggressive drainage or debridement of any devitalized tissue is warranted to remove the source of infection.

COMMENTS

Client Education

- In patients that experience a severe hypotensive state and are discharged with residual neurologic impairments, the owner needs to be educated that full neurologic recovery may take weeks to months.

Patient Monitoring

- Serial lactate measurements every 8–12 hours once normalized.
- Central venous pressure: either continuous or every 8–12 hours.
- Blood pressure monitoring: every 8–12 hours to monitor for hypotension.
- ECG: every 8–12 hours or continuous to monitor for arrhythmias.
- Blood glucose: every 12–24 hours.

Possible Complications

- Renal dysfunction
- Cardiac arrhythmias
- Gastroenteritis, hemorrhagic gastroenteritis

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- Bacterial translocation, sepsis
- Residual neurologic impairment
- Cardiac arrest

Expected Course and Prognosis

- Variable depending on the underlying cause and severity of clinical condition.

Abbreviations

ACTH: adrenocorticotropic hormone
ARDS: adult respiratory distress syndrome
ECG: electrocardiogram
IL: interleukin
LPS: lipopolysaccharide
PT: prothrombin time
PTT: partial thromboplastin time
SIRS: systemic inflammatory response syndrome
TNF: tumor necrosis factor

See Also

Parvoviral Enteritis – Canine
Pneumonia – Aspiration
Pneumonia – Bacterial
Septic Peritonitis
Shock – Cardiogenic

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chapter 94

Smoke Inhalation



DEFINITION/OVERVIEW

- Inhalation injury from smoke and the noxious products of combustion in fires.



ETIOLOGY/PATHOPHYSIOLOGY

- The three primary mechanisms that lead to injury in smoke inhalation are: thermal damage, asphyxiation, and chemical irritation.

Thermal Damage

- Direct burns are usually limited to the nasal and oropharyngeal mucosa and the upper airways. Inflammation and edema of the oronasal cavity and larynx occur within the first several hours of injury. This may lead to upper airway obstruction.
- Steam and small particulate matter may reach the lower airways and the alveoli. Superheated particles cause thermal damage of the respiratory epithelium and vascular endothelium. This, in turn, leads to exudation of proteinaceous fluid into the terminal airways due to alveolar capillary damage.

Asphyxiation

- Tissue hypoxia can occur secondary to several mechanisms, including a decrease in the FiO_2 or fraction of inspired oxygen (hypoxic asphyxia), decreased ability to carry oxygen (functional anemia), decreased tissue perfusion (hypovolemic shock), and disruption of the cells' ability to utilize oxygen (altered cellular respiration) leading to anaerobic metabolism.
    - Combustion progressively consumes oxygen, significantly decreasing the ambient concentration of oxygen from average 21% to as low as 15%. The decrease in FiO_2 leads to hypoxemia.

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Companion website: www.fiveminutevet.com/emergency

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- Carbon monoxide (CO) is a common component of smoke, produced by the incomplete combustion of any organic material. It causes tissue hypoxia by decreasing the oxygen-carrying capacity of the blood. Hemoglobin binds CO with an affinity more than 200 times greater than the affinity for oxygen. CO also causes a left shift in the hemoglobin/oxygen saturation curve, preventing release of oxygen to the tissues. In addition, CO has been shown to poison the mitochondrial cytochrome oxidase chain and decrease myocardial contractility by binding to myocardial myoglobin.
- Combustion of common household substances such as plastics, polyurethane, wool, silk, nylon, rubber, and paper products can lead to the production of cyanide gas. Cyanide is a chemical asphyxiant that interferes with cellular metabolism by binding to the ferric ion on cytochrome c oxidase, subsequently halting cellular respiration. As a result, anaerobic metabolism ensues.
- Methemoglobinemia occurs in fire due to heat denaturation of hemoglobin, oxides produced in fire, and methemoglobin-forming materials such as nitrites. The pathophysiologic consequences of methemoglobin formation are a decrease in the oxygen-carrying capacity of the blood and a shift of the oxyhemoglobin dissociation curve to the left, similar to carboxyhemoglobin (HbCO) production.

Chemical Irritation

- Chemical irritants in smoke include sulfur dioxide, hydrogen chloride, ammonia and acroleins. Soot, a powder-like form of carbon, heightens the effect of these irritants by binding them and allowing them to adhere to and react with respiratory mucosal surfaces.
- Irritants cause direct injury by producing acid and alkali burns of the respiratory mucosa, by formation of free radicals, and by protein denaturation. This incites a reflex bronchoconstriction and pulmonary inflammation.
    - The injured mucosal cells produce copious exudates rich in protein, inflammatory cells, and necrotic debris. With progressive smoke exposure, mucosal sloughing ensues, forming casts of the airways and further blocking oxygen passage to the alveoli.
    - The mucociliary apparatus is also affected by chemical and thermal injury, thus promoting possible bacterial growth.
    - The irritants may also cause acute surfactant inactivation leading to atelectasis and decreased lung compliance.

Systems Affected

- Respiratory
- Nervous
- Skin
- Ophthalmic
- Cardiovascular

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SIGNALMENT/HISTORY

Risk Factors

- Fires in closed spaces; usually trapped in burning buildings.
- Residential fires are more common in the winter months.

Historical Findings

- History consistent with smoke exposure, altered mentation, coughing and gagging, respiratory difficulty, lethargy.



CLINICAL FEATURES

- Range of signs from nasal congestion, lacrimation, and mild CNS depression to life-threatening respiratory distress, hypothermia, and stuporous/comatose behavior, depending on the intensity of the heat produced, duration of exposure, and composition of the smoke.

Dogs and Cats

- Smoky smell, singed or burnt fur and whiskers
- Blepharospasm, hyperemic conjunctiva, corneal ulcers, and rubbing at the eyes
- Increased respiratory effort associated with upper airway sounds, expiratory wheezes, loud bronchovesicular sounds, and crackles
- Mental depression, ataxia (may be delayed)
- Seizures (may be delayed)
- Hyperemic mucous membranes
- Hypothermia



DIFFERENTIAL DIAGNOSIS

- Animals commonly have soot covering their fur and a smoky smell, suggestive of smoke exposure.

Dogs and Cats

- Anaphylaxis, ARDS, CHF, pneumonia, pleural space disease, pulmonary thromboembolism

Cats

- Feline bronchial disease
- Upper respiratory infection
- Pulmonary edema

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DIAGNOSTICS

- Emergency database: PCV, total solids, blood glucose, blood urea nitrogen, lactate, electrolytes and arterial blood gases should be performed in all critically ill patients with smoke exposure.
- Cooximetry may be helpful as a prognosticator. The test can be run at a human hospital from a venous sample. Persistent metabolic acidosis and lactic acidosis may indicate carbon monoxide, methemoglobinemia or cyanide intoxication and secondary tissue hypoxia.
- Fluorescein stain the cornea to assess for corneal burns and assess behind the nictitans for debris.
- Complete blood cell count: neutrophilia (inflammatory leukogram) or neutropenia (due to neutrophil sequestration in the lungs: poor prognostic sign).
- Serum chemistry: may reveal hypoxic damage to the liver or kidneys.
- Imaging: thoracic radiographs should be performed to establish a baseline of lung involvement and its progression. These can lag behind clinical progression, and vary from normal to bronchointerstitial to alveolar patterns.
- Bronchoalveolar lavage: transtracheal, endotracheal or bronchoscopically guided; should be performed if there is suspicion of superimposed bacterial pneumonia (moist cough, fever). This allows collection for cytologic and culture analysis of pulmonary fluid samples.

Pathologic Findings

- The clinical sequence of events can be divided into early, intermediate, and late phases. In the initial 24–36 hours after smoke exposure, the airway effects of heat, irritant gases, and soot predominate (i.e., upper and lower airway obstruction), as well as the systemic toxic effects of CO, cyanide, and methemoglobinemia (i.e., tissue hypoxia leading to immediate or delayed neurologic abnormalities). However, if there was minimal exposure to smoke, the damage may be limited to inflammation of the oronasal and ocular mucosal surfaces without any other major sequelae.
- If skin burns are present and the respiratory tract tissue displays burn edema, then the episode is more likely to be life threatening. Burn/edematous respiratory tissue enhances the toxic effects of smoke inhalation leading to progressive pulmonary dysfunction.
- Asphyxia, pulmonary edema, and bacterial pneumonia are potential complications that usually arise 48–72 hours post exposure; the latter is the most common complication with a mortality rate approaching 50%. Cerebral edema can also develop in the long term due to severe or prolonged hypoxia.



THERAPEUTICS

- Maintenance of airway patency, adequate oxygenation and ventilation, stabilization of hemodynamic parameters, and decontamination measures for skin and eyes.
- Animals with severe upper airway obstruction should be intubated. Supportive care and careful monitoring should be instituted early in the management of smoke inhalation patients as they have the potential to deteriorate rapidly.
- Tracheostomy should be considered for animals with complete airway obstruction.

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Drugs of Choice

- Oxygen therapy should be provided through a mask, oxygen cage or an endotracheal tube in order to improve hemoglobin oxygenation and decrease the half-life of carboxyhemoglobin. Administer humidified oxygen at a rate of 50–100 mL/kg/min for nasopharyngeal or endotracheal administration and 100–150 mL/kg/min for cage administration.
- Severe cases or those that remain hypoxic despite oxygen supplementation may require mechanical ventilation with positive end-expiratory pressure and repeated suctioning of airway secretions.
- Titratable IV fluid therapy to correct shock and maintain hydration. Consider the use of synthetic colloids (hydroxyethyl starch 20 mL/kg/day) to prevent pulmonary edema in the face of increased pulmonary vascular permeability.
- Nebulization of saline and coupage to help thin and clear airway secretions.
- Prophylactic antibiotic therapy is not warranted in animals with smoke inhalation. However, animals with fever and WBC changes on the CBC (persistent neutrophilia or neutropenia) should be treated with broad-spectrum antimicrobials (i.e., cefazolin or ampicillin each at 22 mg/kg IV q8h plus enrofloxacin 10 mg/kg q24h) while awaiting culture and susceptibility results of pulmonary fluid samples.
- Corticosteroids are not recommended as they may predispose to secondary bacterial pneumonia. However, nonsteroidal antiinflammatory drugs (carprofen 2.2 mg/kg SQ or PO dog q12h, meloxicam 0.1 mg/kg SQ or PO q24h) and analgesics (fentanyl 2–4 µg/kg/h IV CRI, buprenorphine 15 µg/kg IV, IM, or sublingual, cats only, q6–8h) can relieve inflammation and discomfort.
- Fast-acting bronchodilators such as beta-2-agonists (albuterol inhalers 90 µg per inhalation: 2 puffs q4h, albuterol prefilled vials of 2.5 mg albuterol in 3 cc sterile saline for nebulization: one vial nebulized q4h; or terbutaline 0.01 mg/kg cats IM, SQ or IV q4h up to four doses) may alleviate reflexive bronchoconstriction.

Precautions/Interactions

- Diuretics may cause hypovolemia without major beneficial effects on pulmonary or airway edema.
- Corticosteroids should only be used as a single antiinflammatory dose, if at all, as they may predispose the patient to bacterial pneumonia, gastric ulceration, and hyperglycemia.

Nursing Care

- Bathe to remove particulate matter on skin.
- Ocular rinse with sterile saline.
- Humidified oxygen to prevent dessication of nasal and respiratory mucosa.

Diet

- Nutritional support (enteral or parenteral) to preserve immune status and prevent a catabolic state.

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Activity

- Dependent on oxygen tolerance.

Surgical Considerations

- Debride any devitalized or necrotic tissue.
- Increased risk of anesthesia for patients with respiratory or neurologic compromise.



COMMENTS

- Dogs and cats presenting immediately after smoke inhalation may appear normal or exhibit only mild increases in respiratory effort. The full effect of smoke inhalation injury to the lungs may not be evident for 24–36 hours after exposure.
- The pulse oximeter can be misleading in the setting of CO exposure or methemoglobinemia because it measures oxygenated and deoxygenated hemoglobin only and not any other form of hemoglobin. It tends to overestimate the level of oxygenation of hemoglobin.
- Cooximeters transmit four wavelengths of light through a blood sample, instead of two wavelengths of light using a pulse oximeter, and are capable of detecting methemoglobin and carboxyhemoglobin (in addition to oxyhemoglobin and deoxyhemoglobin).
- Arterial blood gas analysis will also be unable to accurately determine the hemoglobin oxygen saturation as it cannot differentiate between oxyhemoglobin and carboxyhemoglobin saturation. However, serial analysis of the alveolar-arterial gradient (using the arterial partial pressure of oxygen and of carbon dioxide) will yield invaluable information about potential progressive pulmonary dysfunction due to the inflammatory responses within the lungs and secondary bacterial infection.

Patient Monitoring

- Respiratory rate and effort, auscultation of the neck and thorax, rectal temperatures (q4h), serial measurements of arterial blood gases, electrolytes and PCV/TS, BUN and lactate (q24h) to ensure adequate organ perfusion and resolving hypoxia.
- CBCs should be repeated (q24h) if there is any evidence of clinical deterioration (moist cough, fever).
- Thoracic radiographs should be repeated in 48 hours to rule out pneumonia, acute lung injury, and pulmonary edema.

Prevention/Avoidance

- Owners should be educated regarding residential sprinkler systems and smoke alarms, fire extinguishers, and stickers for doors informing rescuers of the quantity and type of animals that live in the building.

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Possible Complications

- Acute phases: development of bacterial pulmonary infections, acute lung injury, acute respiratory distress syndrome.
- Long term: from asymptomatic to possible chronic pulmonary changes (chronic bronchitis secondary to inflammation and scar tissue formation) and CNS dysfunction (ataxia and abnormal behavior or loss of hearing secondary to CO delayed effects on the CNS).

Expected Course and Prognosis

- Overall, there is a 90% survival rate in dogs and cats with smoke exposure (without concurrent evidence of severe skin burns).
- Deteriorating respiratory function, severe burns, and hypoxic organ damage occurring within 24 hours of hospitalization carry a poor prognosis.
- Dogs and cats with presenting signs that stabilize by 24 hours generally have a favorable prognosis.

Synonyms

- Smoke exposure
- Smoke toxicity

See Also

- Hypoxemia

Abbreviations

ALI: acute lung injury
ARDS: acute respiratory distress syndrome
BAL: bronchoalveolar lavage
CN: cyanide
CNS: central nervous system
CO: carbon monoxide
FiO_2: fraction of inspired oxygen
Hb: deoxyhemoglobin
HbCO: carboxyhemoglobin
HbO_2: oxyhemoglobin
met-Hb: methemoglobin

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chapter 95

Snake Bite – Coral Snakes



DEFINITION/OVERVIEW

- Coral snakes are members of the family Elapidae (fixed front-fang snakes).
- Two genera of coral snakes are found in the United States. The Sonoran coral snake *Micruroides euryxanthus* is found in central and south-eastern Arizona and south-west New Mexico. The other genus is represented by three subspecies of *Micrurus fulvius*, including the Texas coral snake (*M. f. tenere*), the Eastern coral snake (*M. f. fulvius*), and the South Florida coral snake (*M. f. barbouri*).
- The Texas coral snake is found in eastern and south central Texas, southern Arkansas, and Louisiana.
- The Eastern coral snake is found along the eastern seaboard from North Carolina south to Florida, west to Alabama and Mississippi, and throughout eastern Louisiana to the Mississippi River.
- The South Florida coral snake is found in southern Florida to the northern Florida Keys.

Natural History

- Coral snakes are distinctively colored with an alternating body pattern of black, yellow (occasionally white), and red. They can be distinguished from similar-appearing nonvenomous snakes by the color bands. In coral snakes, the color bands completely encircle the snake, and the yellow band touches the red.
- In North America only, coral snakes have a black snout. This is easier to differentiate than trying to remember the mnemonic "Red on yellow, kill a fellow. Red on black, venom lack." Simply look for the black snout. (Caution: This does not hold true for South America!)
- The popular rhyme "Red on yellow, kill a fellow" also fails because coral snakes can be albino, melanistic or other aberrant color morphs and although they lack the characteristic banding, they are nonetheless just as venomous.
- Coral snakes have round pupils, no facial pits for heat sensing prey, and their heads are not triangular.

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- Coral snakes have short, fixed front fangs and a relatively poor venom delivery system.
- Historically, a chewing action has been described as necessary for coral snakes to envenomate but several studies report the coral snake as capable of injecting its venom very rapidly. Human and animal victims often need the biting snake manually removed.
- The Sonoran coral snake genus *Micruroides* is shy, retiring, nocturnal, small, nonaggressive, and burrowing on contact with humans, dogs, cats, or livestock. Venom potency of this snake is relatively low. Bites to companion animals are unlikely. There are no reported fatalities in humans, dogs, or cats due to envenomation by this snake. The majority of bites in humans occur in handlers and collectors.
- The medically important coral snakes are the three subspecies of *Micrurus* (Texas coral, Eastern coral, and South Florida coral snake).
- *Micrurus* snakes are diurnal, docile, but aggressive if disturbed.
- *Micrurus* snakes can be very belligerent if disturbed. Biting snakes may stay attached to victims and need to be removed, causing what has been described as a "Velcro" sound.



ETIOLOGY/PATHOPHYSIOLOGY

- Coral snake venom is primarily neurotoxic, involving little local tissue reaction or pain at the bite site.
- The minor pain and clinically unimpressive local reactions that occur with coral snake bites contrast dramatically with the severe local reaction that crotalid envenomations display. However, lack of local symptoms does not signify that serious envenomation has not occurred.
- Coral snake envenomation can lead to serious systemic effects with little symptomatology at the actual envenomation site.
- Coral snake venom is a complex mixture of polypeptides including cholinesterase and acetylcholine.
- Venom of the Eastern coral snake is the most toxic of all the North American snakes in terms of milligrams of dried weight.
- Several neurotoxins may be involved and act as nondepolarizing postsynaptic neuromuscular blocking agents.
- Coral snake venom induces central nervous system depression, muscle paralysis, and vasomotor instability.
- These neurotoxins combine to produce a "curare-like" syndrome; unlike curare, this nondepolarizing blocking of acetylcholine receptor sites has a slow onset and a markedly prolonged duration.
- In dogs, the enzymatic fraction can cause hemolysis and a resultant hemolytic anemia with hemoglobinuria. It has been theorized that phospholipase A is responsible by directly damaging the red blood cell membrane.
- In cats, enzymatic effects may induce myoglobin release.

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- Hyaluronidase, phosphodiesterase, proteinase, ribonuclease, and desoxyribonuclease are other enzyme components of coral snake venom that can cause tissue destruction. Nevertheless, tissue necrosis and edema in coral snake bites are nowhere near what is seen in crotalid envenomations.
- Venom uptake can be delayed up to 12 hours or more and can take 7–14 days to clear the body.
- Although blood coagulation disorders have not been documented, Eastern coral snake venom has been shown to inhibit ADP-induced platelet aggregation and contains plasmin inhibitors.

Toxic Dose

- The severity of coral snake bites is related to both the volume of venom injected and the size of the victim.
- The human lethal dose is 4–5 mg of venom.
- A large coral snake can deliver 4–5 lethal doses in a single bite (20 mg of venom).
- Length of snake positively correlates with the snake's venom yield.
- Although bites by coral snakes are relatively rare (only 1% of all venomous snake bites in humans), it is estimated that the human fatality rate due to coral snake envenomation is as high as 10%.
- The volume of venom injected is proportional to the motivation of the snake (i.e., offensive, defensive, or agonal bite) along with the duration of the bite and the intensity of the chewing action.
- On account of their relatively primitive venom delivery system, it has been estimated that as much as 60% of coral snake bites are nonenvenomating. Nevertheless, initially it is impossible to predict if envenomation has occurred and some researchers claim the number of "dry bites" is greatly overestimated.



CLINICAL FEATURES

- In dogs, median time to onset of clinical signs following envenomation is 105 minutes. This parameter could not be determined for cats since they are much more unsupervised. However, the appearance of clinical signs in dogs, cats, and humans may be delayed as much as 10–18 hours.
- In dogs, hypersalivation, vomiting, acute central nervous system depression, hyporeflexia, decreased spinal reflexes in all limbs with quadriplegia, respiratory depression, and respiratory paralysis have been described. However, the appearance of clinical signs may be delayed as much as 10–18 hours.
- Regional signs around the bite can be minimal, involving only the puncture wounds, local pain, and possible regional paraesthesia.
- Clinical signs next progress to altered mental status, generalized weakness, and muscle fasciculations.

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- Next, the progression of signs involves paralysis of the limbs and respiratory musculature. This sequence of signs is consistent with bulbar dysfunction.
- Patients can display marked salivation, pharyngeal paralysis, spasms of the masticatory muscles, and respiratory failure.
- Aspiration pneumonia secondary to hypersalivation and dysphagia is not uncommon following coral snake bites.
- In addition to these general manifestations, clinical signs of coral snake envenomation vary depending on the species bitten.
- In addition, in canine victims, anemia, hemoglobinuria, intravascular hemolysis, and morphologic alterations of red blood cells may be seen.
- Hypotension and ventricular tachycardia can also be seen in dogs bitten by coral snakes.
- Generally, hemolysis occurs within 72 hours of envenomation.
- Some dogs can show diarrhea and red-tinged urine.
- In cats, clinical signs after coral snake envenomation include acute, ascending flaccid quadriplegia, reduced nociception, and central nervous system depression.
- Additional signs seen in cats can include hypothermia, anisocoria, loss of cutaneous trunci reflex, and loss of spinal reflexes in all four limbs.
- Cats display hypotension, respiratory depression, and decreased reflexes but maintain normal anal tone and micturition.
- Hemolysis and hemoglobinuria that are observed in dogs are not reported in cats.
- Clinical signs from bites of the Sonoran coral snake (*Micruroides euryxanthus*) are much less dramatic. Local pain, weakness of the bitten extremity, and mild local paraesthesia all resolve in about 24 hours in humans bitten by these snakes.
- The usual cause of death in coral snake bite victims is respiratory collapse.
- Clinical signs of coral snake envenomation in dogs and cats are listed in Boxes 95.1 and 95.2.

BOX 95.1. Clinical signs of coral snake envenomation (dogs).

Hypersalivation
Vomiting
Central nervous system depression
Decreased spinal reflexes (all limbs)
Decreased reflexes with quadriplegia
Muscle fasciculations
Respiratory depression
Anemia
Intravascular hemolysis
Tachypnea
Shallow breathing
Respiratory paralysis

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BOX 95.2. Clinical signs of coral snake envenomation (cats).

Central nervous system depression
Ascending flaccid quadriplegia
Reduced nociception
Hypothermia
Anisocoria
Decreased respiration
Loss of spinal reflexes (all limbs)
Loss of cutaneous trunci reflex
Loss of reflexes but normal tone and micturition
Hypotension
Vocalization
Erratic breathing



DIFFERENTIAL DIAGNOSIS

- Differential diagnosis of coral snake envenomation includes tick paralysis, botulism, macadamia nut ingestion (dogs only), bromethalin, ionophore poisoning, organophosphate toxicity, acute polyneuritis, iatrogenic drug administration, polyradiculoneuritis, myasthenia gravis, and neoplasia.



DIAGNOSTICS

- No specific diagnostic tests exist to establish a coral snake bite.
- Diagnosis of coral snake envenomation is likely in dogs showing concomitant lower motor neuron neuropathy, bulbar palsy, and hemolysis.
- Diagnosis depends upon eyewitness accounts, onset of clinical signs, time of year, regional probability and likelihood of companion animal–snake interaction, and clinician knowledge of local venomous species.
- Local lesions are nondiagnostic, small puncture wounds with minimal swelling.
- Laboratory work should include a complete blood count and biochemical profile.
- Hyperfibrinogenemia, moderate leukocytosis, and elevated creatinine kinase have all been reported in animals after coral snake bite envenomation.
- Elevation of creatinine kinase is an indication envenomation has occurred. This is not a foolproof envenomation indicator but increases in this enzyme are likely associated with myotoxic effects of phospholipases present in the venom. Thus, elevations in creatinine kinase levels lend tentative support for a diagnosis of coral snake bite.

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- In dogs, anemia, intravascular hemolysis, and hemoglobinuria can occur. Dogs bitten may exhibit burring and spherocytosis of red cells. Monitor canine patients for progressive anemia.
- In cats, increased myoglobin and alkaline phosphatase can occur as a result of rhabdomyolysis.
- Because aspiration pneumonia is a common complication of coral snake envenomation due to dysphagia and pharyngeal paralysis, radiographs of the thorax of envenomated animals may be indicated.
- Early diagnosis is critical as antivenom administration can reduce morbidity.



THERAPEUTICS

- The best predictor of successful treatment of coral snake envenomation is early, aggressive therapy.
- The best response in the field by owners of companion animals envenomated by coral snakes is swift transport to a fully equipped, 24-hour veterinary facility familiar with snake bite therapy and able to provide assisted ventilation.
- The wound should be cleaned and coral snake bite victims should be hospitalized for at least 48 hours of continuous monitoring.
- A pretreatment CBC, biochemical panel, and urinalysis should be collected on presentation to obtain a laboratory baseline.
- Supportive medical care for coral snake bite victims must anticipate what may develop.
- Administration of antivenom and provision of mechanical ventilation are cornerstones of successful therapy.
- The facility must be able to provide care for the paralyzed patient, prevent aspiration pneumonia, and possess capabilities for mechanical ventilation.
- Respiratory distress must be aggressively treated using endotracheal intubation to prevent aspiration.
- An intravenous catheter should be placed through which to administer fluids to counter hypotension.
- Suspected coral snake bite victims must be monitored vigilantly since the onset of clinical signs may be delayed for several hours.
- All suspected bite victims should be closely monitored for at least 48 hours.
- The only specific treatment for coral snake envenomation is administration of antivenom.
- Coral snake bite victims must be watched closely for aspiration pneumonia.
- Historically, two coral snake antivenoms have been available to emergency veterinary clinicians.
    - North American coral snake antivenom (NACSA) is an antivenom of whole IgG molecules from horses immunized with Eastern coral snake venom. This antivenom is no longer being manufactured in the United States. This antivenom is the only coral snake antivenom approved for use in humans by the FDA in the United States and is under very vigilant monitoring. At present, it is only available for human administration.

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  - Coralmyn® is a relatively new antivenom manufactured in Mexico, consisting of polyclonal antibodies from horses immunized with black banded coral snake venom. This antivenom has been shown to be effective at neutralizing Eastern coral snake venom and Texas coral snake venom in a mouse model. The USDA monitors and approves use of biologics for veterinary administration. This antivenom is available for use in veterinary medicine with a USDA import permit pending USDA or FDA (human) approval. Coralmyn is expensive.
- Experimental studies have shown protective cross-reactivity with Australian tiger snake (*Notechis scutatus*) and Mexican coral snake (*Micrurus*) antivenom in a mouse model.
- Antivenom treatment is recommended for animals where an eyewitness observed the bite, there is one or more fang marks from which blood can be expressed, with a history of the snake seen to be hanging from the victim, when a live, injured, or dead coral snake is found in the vicinity of the animal suspected of being envenomated, or the victim is from an area known for adverse snake–pet or snake–human interaction.
- Antivenom is most effective if given early. Published studies in rodents attest to its efficacy. The sole objective of timely antivenom treatment is to avoid the onset of respiratory paralysis that will increase the severity of the condition and the subsequent requirement for ventilator support.
- Paralysis can occur despite the administration of antivenom.
- Like equine-derived crotalid antivenom, final product coral snake antivenom is rich in equine proteins that can be responsible for allergic reactions.
- Studies defining dose–response correlation between administration of antivenom and coral snake envenomation have not been conducted in dogs, cats, or people. As a result, it is difficult to determine exactly how many vials a patient requires.
- One vial of NACSA neutralizes approximately 2 mg of dried venom while one vial of Coralmyn neutralizes approximately 5.5 mg of dried venom. A large coral snake may inject up to 20 mg.
- In people, recommendations for antivenom administration following a coral snake bite call for 4–6 vials of antivenom in the first 4–8 hours. More is given depending on the severity of clinical signs.
- In veterinary patients, expense limits the antivenom given to only 1–2 vials per patient.
- Treatment with antivenom should not wait until clinical signs appear.
- It is not known whether antivenom administration will shorten the period of paralysis. The objective of early antivenom treatment is binding the neurotoxins in the circulation before they enter the nerves.
- Antivenom is diluted (one vial in 100–200 mL) into crystalloid fluids and given slowly over 30–60 minutes while monitoring to prevent an allergic reaction. Reconstitution in the diluents may take 10–15 minutes. Warming the vial to body temperature may aid in dissolving.
- The most common reaction to antivenom is an anaphylactoid response. This reaction can be treated by administration of diphenhydramine and stopping the antivenom infusion.
- True anaphylaxis must be treated with epinephrine, corticosteroids, intravenous fluids, and stopping the antivenom infusion.
- Although the mouths of snakes contain a large array of organisms derived from the fecal material of their prey species, the incidence of infection following the bite of a coral snake

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is extremely low. Bacterial infection associated with coral snake bites has not been reported in either human or animal studies. For this reason, antibiotics are not recommended following coral snake bite.

- Likewise, at this time there is little or no evidence to justify the use of glucocorticoids or nonsteroidal antiinflammatory drugs (NSAIDs) following coral snake bite.
- Diphenhydramine and epinephrine should only be used if an anaphylactoid reaction to the antivenom is suspected.
- Medications that cause respiratory depression such as morphine or other narcotics are contraindicated, as are those that impair neuromuscular transmission such as aminoglycosides.
- Antivenom should not be injected into the bite site. Uptake of antivenom can be delayed up to 12 hours if given intramuscularly. In one study, when given intravenously, 85% of labeled antivenom accumulated at the envenomation site within 2 hours. In contrast, when given intramuscularly, there was a 1.4% local concentration, and a 5.6% local concentration if given subcutaneously.
- The entire initial antivenin dosage should be given within one half hour.
- Amount of antivenom needed is relative to the amount of venom injected and the body mass of the victim.
- Smaller patients require higher doses of antivenom since the dose of venom per kilogram of body weight is higher.
- Sonoran coral snake bite victims are much less seriously envenomated. At present, no antivenom is available for this venom. These victims are treated supportively and symptomatically.
- First aid treatment for coral snake bite in humans advocates compression bandages and pressure immobilization. Such treatments have not been validated for use in dogs and cats following snake bite. Tourniquet use is never indicated.
- Interventions to be avoided after coral snake envenomation include ice, cryotherapy, hot packs, incision and suction, extractor devices, electroshock, fasciotomy, compression bandages, and tourniquets (Box 95.3).

BOX 95.3. Interventions to avoid in coral snake bite.

Ice

Cryotherapy

Hot packs

Incision and suction

Extractor devices

Electroshock

Fasciotomy

Compression bandages

Tourniquets

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Prevention/Avoidance

- The most effective preventative effort in diminishing numbers of coral snake bite is companion animal avoidance of prime snake habitat in endemic regions.
- Cats should be kept indoors in areas of known coral snake populations.
- Dogs should never be left unsupervised or running loose in areas known to be home to coral snakes.
- In areas of high snake density, snake avoidance/aversion classes are often held to train dogs to avoid snakes. Many of these programs are based on negative reinforcement and are not that reliable. They may provide a false sense of security but can be effective in a well-trained dog.
- Rodents can be removed from houses, garages, sheds, and yards in an attempt to prevent snakes from being attracted to human habitations and biting companion animals.
- Client education to alert owners to the signs and appearance of snake bite envenomation is vital.

Expected Course and Prognosis

- Prognosis is closely dependent upon the length of time between time of bite and initiation of treatment. Also, the size of the snake, the size and species of the victim, and the site of the bite must be evaluated when considering outcome. The prognosis for all cats is guarded unless early, effective treatment is initiated.
- Even after envenomation, overall prognosis is good. One study showed 71% of dogs survived after being bitten by coral snakes. Symptomatic victims that do not receive antivenom have been successfully managed by providing supportive are which may include mechanical ventilation.

Abbreviations

ADP: adenosine diphosphate
CBC: complete blood count
FDA: Food and Drug Administration
NACSA: North American coral snake antivenom
NSAID: nonsteroidal antiinflammatory drug
USDA: United States Department of Agriculture

Suggested Reading

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chapter 96

Snake Bite – Pit Vipers



DEFINITION/OVERVIEW

- Of over 120 species of snakes found in North America, 20 species are venomous.
- Venomous snakes in North America can be divided into two families: Crotalidae (pit vipers, including rattlesnakes, copperheads, and cottonmouths (water moccasins)) and Elapidae (coral snakes).
- The group responsible for both the greatest number of bites and the most serious bites are members of the family Crotalidae. Each year 99% of snake bites in the United States are initiated by crotalids.
- It is estimated that 150 000 dogs and cats are bitten each year in the United States by rattlesnakes, copperheads, and water moccasins.
- In the US, 90% of venomous snake bites occur during the months of April and October, reflecting the snakes' hibernating habits.

Natural History

- Crotalidae are known as "pit vipers" because they possess a specialized, heat-sensing facial "pit" that aids them in locating and obtaining warm-blooded prey.
- These heat-sensing structures can differentiate a temperature gradient of 0.003 °C at a distance of 14 inches. They are located in facial pits between the eye and nostril.
- In addition to the bilateral heat-sensitive pits between the eyes and nostrils, pit vipers display folding retractable fangs, vertical elliptical pupils, triangular-shaped heads, and a single row of subcaudal scales caudal to the vent.
- There are three genera of Crotalidae in the United States: *Crotalus* (rattlesnakes), *Sistrurus* (pygmy rattlesnakes and massasauga), and *Agkistrodon* (which are the rattleless copperheads and water moccasins).
- Although many snakes rattle their tails when agitated, rattlesnakes have developed special keratinized rattles at the end of their tails to indicate their motivation, with the exception of one subspecies of rattleless rattlesnake (*Crotalus catalinensis*).
- Venomous snakes are found in every state in the United States except Maine, Alaska, and Hawaii.

Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Emergency and Critical Care, Second Edition. Edited by Elisa M. Mazzaferro.
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Companion website: www.fiveminutevet.com/emergency

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- Rattlesnakes do not always rattle before striking. Rattlesnakes can strike approximately one half their body length at a speed of 8 feet per second.
- Pit vipers can meter the amount of venom they release in a bite. "Offensive" bites on food objects release less venom than "defensive" bites against attackers. "Agonal" bites delivered by dying snakes can release all the venom they possess.



ETIOLOGY/PATHOPHYSIOLOGY

- The primary purpose of pit viper venom is the immobilization of prey and the initiation of predigestion of their tissues. The venom of snakes has been evolutionarily derived from modified salivary glands.
- Pit viper venom is a complex mixture of bioactive enzymes and peptides. The venom is composed of 90% water and over 50 enzymatic and nonenzymatic components.
- Hyaluronidase (spreading factor) and collagenase aid in spreading venom through interstitial spaces.
- Proteases lead to coagulopathies and necrosis.
- Phospholipases cause cytotoxic effects that lead to inflammation and endothelial cell death.
- Phospholipase A_2 is a major contributor to red blood cell abnormalities including echinocytosis, spherocytosis, erythrocyte ghosts, and erythroid loops causing alterations in erythocytic membrane and composition.
- In general, the clinical effects produced by pit viper envenomation can be classified into three groups.
    - Local tissue effects include soft tissue necrosis and chemically mediated inflammation. Over 90% of envenomated victims develop local tissue effects.
    - Hematologic venom effects include fibrinogen degradation and platelet aggregation and destruction. Decreased fibrinogen levels, elevated prothrombin time, and thrombocytopenia are revealed in laboratory findings.
    - Systemic venom effects include hypotension from direct cardiovascular toxicity, third spacing and vasodilation, nausea and vomiting, and neurotoxicity.
- Pit viper venom increases the permeability of capillary cell membranes. Damage to the microvasculature allows erythrocytes and plasma to escape, resulting in both edema and ecchymosis.
- Plasma loss to edema and third spacing can result in hypotension.
- Enzymes and proteins in snake venoms can result in coagulopathy. Fibrinolytics and thrombin-like enzymes in venom result in depletion of fibrin and fibrinogen, decreased ability to form intravascular clots, elevated fibrin degradation products, and prolonged clotting tests.
- Snakebite coagulopathy is unique and differs from other forms. Unlike true DIC, pit viper coagulopathies have normal antithrombin and factor XIII levels.
- Thrombocytopenia is common in pit viper envenomations and is the result of phospholipase damaging platelet membranes, triggering their destruction, venom-initiated platelet aggregation, and platelet consumption by inflammation at bite site.
- Renal failure can be seen secondary to crotalid envenomation. It is the result of hypotension, circulatory collapse, and toxic nephropathy of degratory by-products.

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- Neurotoxicity is primarily seen in envenomation by the Elapidae but neurological signs can be seen in bites by the Mojave rattlesnake (*Crotalus scutulatus*) of the south-western United States. Other snakes may also possess some neurotoxic activity.
- Mojave toxin-type ascending flaccid paralysis after an envenomation by a Southern Pacific rattlesnake (*Crotalus helleri*) has been described. Furthermore, Mojave-like toxins have been identified from the venoms of the Western diamondback rattlesnake (*Crotalus atrox*), the canebrake rattlesnake (*Crotalus horridus*), tiger rattlesnake (*Crotalus tigris*), and the prairie rattlesnake (*Crotalus viridus*).
- The bite of young snakes is toxic; young snakes are reported to have higher percentages of toxic fractions. However, adult snakes inject larger amounts of venom.
- Pit viper envenomation can induce myocardial injury and cardiac arrhythmias. Cardiac involvement has been confirmed through histopathologic changes with edema, hemorrhage, necrosis, interruption of the sarcolemma, and granular degeneration found in the myocardium.
- Rattlesnakes on average require 21 days to replenish utilized venom.

Toxic Dose

- The amount of venom injected varies with the age, size, species, and motivation of the snake. Eastern and Western diamondback venom yields can be as high as 800 mg whereas copperheads may yield only 40 mg.
- In general, rattlesnakes are more toxic than water moccasins (cottonmouths) which are more potent than copperheads.
- In terms of the LD_{50} for rodents, the Mojave rattlesnake has the most toxic venom (although larger snakes and larger species release more venom).
- Pit viper bite severity is related to the volume and toxicity of the venom injected, the location of the bite on the victim, and the rate of its uptake.
- Victim-associated factors determining bite severity include size of the victim, time elapsed between bite and initiation of treatment, and victim activity after bite occurs (activity increases venom absorption).
- An unknown percentage of crotalid bites are "dry" (nonenvenomating). However, many snake bite authorities feel the number of "dry" bites is overestimated.
- The severity of a pit viper envenomation is a factor of the species of snake involved, volume of venom injected, the location of the bite, size and species of the bite recipient, and the time since envenomation.



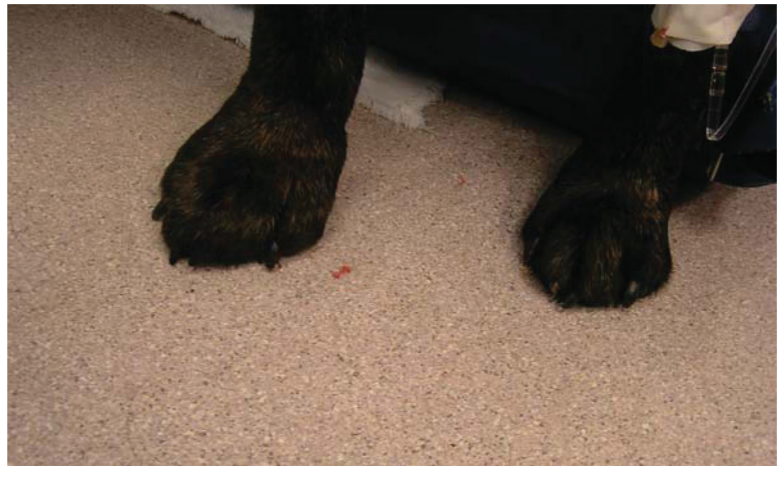
CLINICAL FEATURES

- In dogs, the majority of bites occur on the head and face. One study reported that in dogs over 80% are struck in the face. In cats, bites occur to the thorax, abdomen, and paws.
- The hallmark of crotalid envenomation is intense, rapid swelling (Figs 96.1, 96.2).
- Clinical signs include fang marks, swelling, edema, and considerable pain at the site. Other signs may include erythema, petechiae, ecchymosis, and tissue necrosis.

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■ **Fig. 96.1.** Facial swelling after envenomation by the prairie rattlesnake (*Crotalus viridis*).



■ **Fig. 96.2.** Swelling of the paw after rattlesnake envenomation.

- Although delayed effects of snake bites have been described both in humans and in dogs, the majority of victims display signs of envenomation within 30 minutes from the time of the bite.
- The signs of a localized bite can progress to systemic signs of vomiting, depression, respiratory distress, tachycardia, hypotension, fever, and coagulation abnormalities.
- Bites to the tongue are particularly insidious and can often be fatal.

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- Fang marks can be quite inconspicuous and covered with fur.
- Pit viper envenomations are painful and pain closely follows the progressive swelling and edema.
- Swelling can be intense and is not merely edema, but is caused by extravacularized blood seeping from damaged blood vessel walls.
- Intense swelling can interfere with breathing and swallowing. Animals may salivate profusely.
- Tissue around the bite may quickly become badly bruised; swelling worsens in the first 24 hours and may slough or ooze dark, watery blood.
- Cats have been reported to be more resistant (on a milligram of venom to a kilogram of body weight basis) to pit viper venom than dogs. However, cats are typically presented to hospitals in a more advanced clinical condition. Also, the severity of the bite is almost always inversely related to the victim's size.
- Main clinical signs of pit viper envenomation include intense swelling, pain, hypotension, coagulopathy with thrombocytopenia and prolonged clotting times, and potentially severe bruising and necrosis around the bite site. Myocardial injury and neurotoxic signs can be seen with some species.



DIFFERENTIAL DIAGNOSIS

- Differential diagnoses for pit vipers include trauma, angioedema from arthropod bites or stings, other animal bite wounds, draining abscesses, penetrating wounds, abscessed teeth, and neoplasia.



DIAGNOSTICS

- At present, there is no method available that can definitely establish that a pit viper envenomation has occurred and no way to measure the degree of that envenomation. Diagnosis must be based on an eyewitness of the event, clinician knowledge of venomous snakes in the region, and clinical signs.
- Traditional diagnostic tests used to assess hematologic abnormalities include a CBC, PCV, coagulation profile with either a PT and PTT or an ACT, and a serum fibrinogen measurement.
- After envenomation, laboratory abnormalities include echinocytosis, thrombocytopenia, anemia, hemoconcentration, elevated fibrin split products, hypofibrinogenemia, leukocytosis, and prolonged clotting times.
- For rattlesnake envenomation, hematologic abnormalities are reported in 81% of cases. Another retrospective study documented thrombocytopenia in 88% of dogs involved.
- Serial blood tests should be performed to monitor coagulopathies.
- Serum biochemical blood profile abnormalities include azotemia, hypoalbuminemia, hypoproteinemia, and elevated levels of creatine kinase, alkaline phosphatase, alanine transaminase, gamma-glutamyl transpeptidase, and aspartate aminotransferase.

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- Erythrocyte transformation into echinocytes (red blood cells with regular spiky membrane projections) has been described following snake bite envenomation in dogs, cats, and humans. The frequency of echinocyte formation has been reported to be 89–92% of dogs following snake bite envenomations.
- It has been suggested that the presence of echinocytes be used as a marker for envenomation in dogs; however, other disease processes can cause echinocytosis.
- Serial measurement of the swelling should be done at set intervals to monitor its progression secondary to the bite. This is a useful way to document stabilization and response to treatment.
- Serial ECG and blood pressure will identify cardiac arrhythmias and monitor hypotension.



THERAPEUTICS

- Any animal suspected of being bitten by a venomous snake should be evaluated by a veterinarian.
- Initial laboratory work should include complete blood count (with erythrocyte morphologic analysis), platelet count, biochemical profiles, coagulation profile (ACT), PT, aPTT, fibrinogen level, and a urinalysis. An ECG should be performed to monitor cardiac arrhythmia and blood pressure to monitor hypotension.
- Clip and prep the affected area gently with an antiseptic solution and measure the circumference of the swollen bitten area to monitor progress of swelling.
- Due to the rapid onset of hypotension, victims of pit viper envenomation should be immobilized, and intravenous fluids (crystalloids: lactated Ringer's solution, Plasmalyte-A, Normosol-R or 0.9% NaCl) initiated upon admission to the hospital.
- Colloids appear adequate and beneficial in maintenance of vascular volume and colloid oncotic pressure.
- Goals of treatment include management of pain, hypotension, swelling, neutralization of venom, and countering venom-induced coagulopathy.
- Traditional recommended therapy for pit viper bites for humans and animals has included intravenous fluids, analgesics, and antivenom.
- The only specific therapy against pit viper envenomation is the administration of antivenom.
- Currently, there are emerging controversies surrounding the use and efficacy of antivenom. Despite an effort in human medicine to develop a rational, unified approach to antivenom administration and dosage, no consensus has been reached.
- Currently, administration of antivenom is recommended when there is continued progression of local signs and swelling, hypotension is nonresponsive, there is significant coagulopathy, neuromuscular signs are present (paralysis, etc.), and evidence of cardiovascular collapse and/or myocardial injury.
- Antivenom is the cornerstone for humans bitten by pit vipers. The human mortality rate following envenomation has decreased from 5–25% to 0.5% since the advent of antivenom use in 1954. The average human patient receives 12 vials of antivenom following a bite.

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A coworker bitten at the author's rattlesnake study site received 32 vials of antivenom after he was bitten.

- Currently, several pit viper antivenoms are available to veterinary clinicians. Antivenin Crotalidae polyvalent (ACP) antivenom is made from whole IgG molecules from horses immunized with venom from Eastern diamondback (*C. adamanteus*), Western diamondback (*C. atrox*), tropical rattlesnake (*C. durissus*), and the fer-de-lance (*Bothrops asper*). This product has shown a failure to reverse neurologic effects of Mojave rattlesnake venom. In addition, time to reconstitute this antivenom is prolonged and averages 1 hour to dissolve. Currently, this is the only USDA-approved antivenom for veterinary patients.
- CroFab (F(ab) antivenom ovine) (Protherics, Nashville, TN) is a polyvalent antivenom derived from sheep immunized against Eastern diamondback (*C. adamanteus*), Western diamondback (*C. atrox*), Mojave (*C. scutulatus*), and the cottonmouth (*A. piscovorax*). The whole immunoglobulin molecules are enzymatically cleaved, leaving only monomers of antigen-binding fragments. This product is rapidly reconstituted (less than 10 minutes) and has been shown in animal models to be five times more potent at neutralizing pit viper venom than the ACP product. It is a small molecule which is rapidly cleared from circulation. Thus the clinical syndrome of reenvenomation has been seen in humans and may necessitate the use of several vials for a single envenomation. In one study in dogs treated with this antivenom, 1.25 vials per dog were administered on average.
- F(ab)2 antivenom is a polyvalent antivenom from horses immunized against the tropical rattlesnake (*C. durissus*) and the fer-de-lance (*Bothrops asper*). The whole immunoglobulin molecules are cleaved, removing the F_c portion of the molecule and leaving two antigen-neutralizing sites. Retrospective analysis of 180 dogs treated with this antivenom in Arizona found that one vial was sufficient to improve clinical signs in most dogs. This antivenom has shown efficacy in resolving both neurologic and myotoxic effects of rattlesnake envenomation. It is available for purchase from Mexico with a USDA import permit pending USDA approval.
- An unpurified equine plasma protein product called RTLR (MG Biologics, Ames, IA) consists of doses of equine plasma derived from horses vaccinated against Mojave (*C. scutulatus*), Eastern diamondback (*C. adamanteus*), Western diamondback (*C. atrox*), and prairie rattlesnake (*C. viridus*). Each bag contains 100 mL of unpurified equine plasma and the manufacturer recommends 4 mL/kg. Peer review literature evaluating either safety or efficacy of this product is not available at this time. The label reports that all dogs receiving this product experienced mild-to-moderate reactions around the injection site in addition to peripheral lymphadenopathy 48 hours after administration. It is recommended that this product be reserved for equine patients only until further studies have been completed.
- A recent study reported that both cost and duration of hospitalization were significantly greater in dogs receiving ACP (equine). However, this may reflect that more severely affected dogs were given antivenin and that more vials of antivenin may be required to neutralize the venom. The total number of vials administered to dogs is often limited by financial constraints of the owner.

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- A potentially more useful assessment of dosing may not be vials per dog but instead milligrams of antivenom per kilogram body weight, as is followed in human medicine.
- More studies are urgently needed to evaluate both the dosage and efficacy of various antivenoms available to envenomated companion animals.
- A recent investigation suggests that smaller patients may require higher dosages of antivenom. Also, early administration of antivenom is more effective than if given later.
- Antivenom must be given intravenously and never injected into the bite wound. If given intramuscularly, antivenom uptake is delayed by 12 hours.
- Antivenom does not reverse tissue necrosis once it has occurred but additional damage can be prevented. Antivenom is, however, effective in reversing venom-induced thrombocytopenia.
- Skin testing for allergic reaction to equine antivenom is hard to evaluate since false positives and false negatives occur. Animals may be pretreated with diphenhydramine and monitored for allergic reaction (hyperemia of the pinnae, pruritus, fluffing of the tail, etc.) as product is given. If anaphylactic reactions occur, they are treated with histamine blockers, systemic corticosteroids, epinephrine, and slowed or discontinued antivenin administration.
- Pain management of snake bite victims is critical. Buprenorphine (0.01 mg/kg IV every 6 hours) or fentanyl (2–3.0 µg/kg per hour CRI) may be employed. The animal must be monitored closely for respiratory depression.
- Morphine, while providing appropriate analgesia and having the ability of reversal with naloxone, should be used with caution due to the risk of histamine release, causing bronchoconstriction, urticaria, pruritus, lethargy, and hypotension.
- The oral cavity of pit vipers contains a large array of organisms derived from the fecal material of prey species. Even though studies reveal a host of potentially harmful bacteria in the mouths of snakes, retrospective studies demonstrating infection following snake bite reveal a very low incidence in both humans and dogs. Therefore, the use of prophylactic antibiotics in snake bite cannot be recommended. Use of antimicrobials should be reserved for bites with later abscess or excessive necrosis.
- Arterial tourniquet application is ineffective and can be associated with apparent harm and tissue damage when used to treat rattlesnake bite. At this time, since controlled studies are lacking, the use of pressure immobilization or lymphatic constricting bands cannot be recommended.
- The use of extractor devices is no longer recommended in humans, since they are ineffective, do not reduce swelling, can cause injury to surrounding tissue, and waste time that could be devoted to effective management.
- Use of NSAIDs cannot be recommended due to the theoretical harm associated with the platelet dysfunction caused by NSAIDs in a potentially thrombocytopenic patient.
- There is considerable controversy over use of glucocorticoids for human and animal snake bite victims. While corticosteroids are not recommended for the management of snake bites in humans, there is literature in animals to support their use. Further studies in rodents are needed that can eventually justify or discount their theoretical and documented usefulness.
- Following snake bite, later repair and surgical debridement of necrotic areas may be required.

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BOX 96.1. Treatments to avoid in pit viper snake bite.

- Ice
- Cryotherapy
- Nonsteroidal antiinflammatory medication
- Electric shock
- Dimethylsulfoxide
- Extractor devices
- Tourniquets
- Fasciotomy
- Wound excision and suction
- Lymphatic constriction

- Treatments that should be avoided in pit viper snakebite include extractor devices, tourniquets, ice and cryotherapy, wound excision and suction, electric shock therapy, fasciotomy, dimethylsulfoxide (DMSO) therapy, NSAIDs, pressure immobilization, and lymphatic constricting bands (Box 96.1).
- Emergency rooms treating snake bite victims must be prepared to perform urgent airway management (i.e., endotracheal tubes, tracheostomy kits, oxygen cages), particularly if the head, face, inside of the mouth, or tongue are involved. Swelling can be rapid, progressive, and dynamic.

Rattlesnake Vaccine

- A *C. atrox* toxoid rattlesnake vaccine (Red Rock Biologics, Woodland, CA) developed for prophylaxis against rattlesnake bite is now available. Two doses are initially given 1 month apart followed by a yearly booster. There are no peer-reviewed canine studies attesting to the efficacy of this product. Also, no venom challenge studies conducted on vaccinated dogs have been undertaken. The package insert states the vaccine does not completely prevent the effects of envenomation but decreases clinical signs. The effect of the vaccine is based on titer studies. At this time, the efficacy of this vaccine is difficult to evaluate and until controlled studies are done, its effectiveness is unknown.

COMMENTS

Prevention/Avoidance

- Keep dogs on leashes and closely supervised when in known or prime snake habitat with poor visibility.
- Similarly, keep cats indoors in areas of high snake density.
- In areas of known rattlesnake abundance, snake avoidance classes are often offered to teach dogs to avoid contact with snakes. Many are based upon negative reinforcement

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with variable results. These training measures are generally unreliable and can provide a false sense of security but may be efficacious in a well-trained dog.

- Dogs do not seem to learn about snakes. Several accounts have been reported of dogs being bitten in successive years.

Expected Course and Prognosis

- Prognosis for pit viper victims depends on the degree of envenomation, type of snake, motivation of the bite and amount of venom injected, location of the bite, size and species of the victim, and the time elapsed between envenomation and initiation of therapy. The sooner emergency intervention is begun and the more aggressive the management, the better the outcome. Prognosis is worse for animals with continued hypotension, hematologic complications, systemic venom effects, and extremely small size. Although smaller than most dog snake bite victims, and historically considered more resistant on a milligram per kilogram basis than dogs to pit viper venom, due to their aggressive behavior cats may receive a relatively larger venom dose than a dog. Despite this, the mortality of cats envenomated by a pit viper remains low. As a result of their increased popularity, clinicians may expect to see more cats presented for snake bite. Nevertheless, if managed vigilantly, the vast majority of animal snake bite victims survive with no permanent sequelae.
- At present, controversies exist concerning the administration and dosage of antivenin. The need for controlled studies in this area is clearly evident. From the evidence in humans, severely affected animals may benefit from antivenin administration. They may also benefit from more antivenom given per bite. Animals may likewise profit from a milligram of antivenom per kilogram of body weight system, as is employed in humans, rather than the vials per dog methodology used traditionally.

Abbreviations

ACT: activated clotting time
aPTT: activated partial thromboplastin time
CBC: complete blood count
CRI: constant rate infusion
DIC: disseminated intravascular coagulation
DMSO: dimethylsulfoxide
ECG: electrocardiogram
IV: intravenous
LD_{50}: lethal dose that kills 50% of animals tested
NaCl: sodium chloride
NSAID: nonsteroidal antiinflammatory drug
PCV: packed cell volume
PO: by mouth (per os)
PT: prothrombin time
USDA: United States Department of Agriculture

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chapter 97

Spider Bite – Black Widow

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DEFINITION/OVERVIEW

- Spiders are the planet's most successful terrestrial carnivore. Over 35 000 species have been described, and some researchers think there may be over 100 000 species. By comparison, there are only 4000 species of mammal.
- Over 20 000 spider species are found in North American and all but two are venomous. However, only 50 of these species have fangs capable of penetrating mammalian skin. Furthermore, the majority of these spiders possess small quantities of venom and weak delivery systems that limit severe toxicity.
- The majority of spider bites are of no medical consequence or concern. Spider bites have not been shown to transmit communicable diseases, spider envenomations do not generally become secondarily infected, and allergic reaction following a spider bite is extremely rare.
- A few species produce much more toxic venoms capable of causing skin lesions, systemic illness, neurotoxicity, and death.
- Spider venoms are composed of complex proteins and proteolytic enzymes designed to either initiate digestion of prey entrapped by web spinners or incapacitate prey ambushed by hunting spiders.
- In general, hunting spiders such as brown recluse spiders (*Loxosceles* spp.), funnel web spiders (*Atrax* and *Hadronyche* spp.), and South American armed spiders (*Phoeneutria* spp.) have more potent venoms than web spinners, with the notable exception of widow spiders (*Latrodectus* spp.).
- Some spiders, notably tarantulas, can use their hindlegs to launch urticating hairs from their dorsal abdomens to irritate the skin, eyes, and mucous membranes of pursuing attackers.
- Some reports state that you are never more than 1 meter away from a spider.

Natural History

- Widow spiders (*Latrodectus* spp.) are found in tropical and temperate latitudes worldwide (over 40 species identified), and five species reside in the US in every state except Alaska. In the continental US, the most common widow spider is *Latrodectus mactans* (black widow spider) (Box 97.1).

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BOX 97.1. Medically important Lactrodectus spiders in the US.

Latrodectus mactans (black widow spider, southern black widow), found throughout continental US, except Alaska
Latrodectus hesperus (western black widow), found in western US
Latrodectus variolus (northern widow spider), found in northern parts of US
Latrodectus bishopi (red widow spider, red-legged widow spider), found in central and southern Florida
Latrodectus geometricus (brown widow spider), found in Florida

- Black widow spiders prefer dark, dry, draftless areas. Preferred habitats include brick and rock piles, wood piles, wall crevices, crawl spaces, gas and water meters, barns, stables, attics, closets, cupboards, trash piles, garages, basements, and outhouses.
- The majority of bites in humans occur during the colder months when the spiders come into human habitations. The rest of the year they live outdoors. These spiders construct funnel-shaped webs, irregularly shaped with a tattered appearance. They spin web "tripwires" that may trail below their dark crevice retreats.
- Female black widow spiders are larger, darker, and more venomous than the smaller males. Females have a 30–40 mm leg span (male leg span 16–20 mm), but females can be as much as 20 times the size of males.
- Females can be 2–2.5 cm long and can cause life-threatening envenomations. Female black widows are dark gray to black and display the characteristic red or orange hourglass pattern on the ventral surface of their shiny, globous black abdomen. The hourglass becomes more prominent as the spider ages.
- Immature females are capable of delivering a severe envenomation, lack the typical swollen abdomen and hourglass of the adult female, and are red, brown, and beige. Immature females may take on the color of their last insect meal.
- Some females consume male mates after breeding, giving the spider its name. Males rarely bite and their bite is of no medical significance.
- Black widow females are shy, nonaggressive, and retreat from drafts and large currents of air. Nevertheless, females will protect their egg sac and the web. Like the brown recluse, black widow spiders are fairly long-lived and can live over 1 year.
- The five species of widow spiders found in the United States are *Latrodectus bishopi*, *L. geometricus*, *L. hesperus*, *L. variolus*, and *L. mactans*. The name *Latrodectus mactans* means"sneaky biter."
- It should be remembered that spider bites can occur well outside the normal geographic range, due to travel, commerce, and the innate ability of spiders to stow away.

Pathophysiology

- Black widow spiders produce one of the most potent venoms by volume.
- Through striated muscle control, these spiders can meter the amount of venom they inject with each bite. It has been estimated that 15% of bites are nonenvenomating (dry).

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- Clinical signs (latrodectism) from black widow bites are caused by a neurotoxic component of the venom, alpha-latrotoxin. This molecule causes massive presynaptic release of most neurotransmitters including acetylcholine, norepinephrine, dopamine, and glutamate.
- The alpha-latrotoxin targets the presynaptic neuronal membrane which leads to the massive release of both calcium-dependent (catecholamines) and calcium-independent (aceytylcholine, GABA, glutamate) neurotransmitters.
- It appears to do this through formation of stable transmembrane pores which allow calcium influx into the presynaptic nerve terminal.
- Initially stimulatory to endplate action potentials, the neurotoxin later blocks neurotransmission through depletion of synaptic vesicle contents and then subsequent inhibition of neurotransmitter reuptake.
- Black widow spider venom is a primary neurotoxin; it contains no locally active peptides that provoke an inflammatory action at the site of envenomation.

Toxic Dose

- In contrast to the brown recluse spider, people report that the bite of the black widow spider is moderately painful.
- Black widow spider venom on a volume-to-volume basis is 100 times more potent than the venom of a pit viper. Fortunately, since their prey objects are primarily small insects, only a small amount of venom is injected with each bite.
- It appears venom toxicity is increased in areas of higher environmental temperature and in bites occurring in the fall.
- For companion animals, a single bite can deliver a lethal envenomation.
- The brown widow (*L. geometricus*) has the most potent venom. The median lethal dose (LD_{50}) is 1.43 mg/kg body weight. The median lethal dose (LD_{50}) of the black widow is 1.39 mg/kg. The toxic fraction of the venom appears to be the same for the entire genus but varies in volume percentage between species.



CLINICAL FEATURES

- Clinical signs of latrodectism develop within 30 minutes to a few hours following the bite.
- Local tissue changes around the bite are absent or mild, swelling is uncommon, and a mild erythema may develop which is easily missed due to tissue pigmentation and dense coats.
- More severe systemic manifestations depend upon size of the spider, motivation of the spider (how much venom injected), and time of year (altered venom potency). Also the location of the bite, the size, age, species, and general health of the spider's victim all contribute to the possible severity of the bite.
- Dogs and cats show severe pain, muscle cramping, vomiting, diarrhea, tremors (Table 97.1) and abdominal rigidity without tenderness.

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TABLE 97.1. Comparison of widow spider and recluse spider bites.

Bite characteristics	Widow (*Latrodectus*) bites	Recluse (*Loxosceles*) bites
Initial bite	Moderately painful	Painless
Mechanism of venom	Massive neurotransmitter release	Local cytotoxicity
Predominant poison	Alpha-latrotoxin	Sphingomyelinase D
Incubation period	Rapid (1 hour)	Delayed (3 days)
Most common sign	Muscle rigidity – spasms	Rash and lesion
Signs of systemic illness	Fever, ataxia, paraesthesia	Hemoglobinuria and renal failure
Outcome of most bites	Resolution in 2–3 days	Ulcers may take weeks to heal

- Respiratory distress may become evident and if death occurs, it is usually due to respiratory collapse.
- Cats are extremely susceptible to the venom of black widow bites. Painful howling and vocalization can progress to ataxia and paralytic signs. Rhabdomyolysis and seizures are rare. Rarely are the small bite wound punctures identified.
- Envenomated animals can lose as much as 20% of their body weight in the first 24 hours following the bite.
- The muscle spasms, cramping, and abdominal rigidity are thought to be due to the massive release of neurotrasmitters and an influx of sodium. Deaths can occur in cats, in smaller animals, and in compromised or debilitated animals.
- The spider is rarely seen by the pet owner. Animals may vomit up the culprit spider. In our practice, a dead black widow was found between the lip and gingiva in a cat displaying signs similar to latrodectism.
- In one study, 20 of 22 cats envenomated by black widows died as a result of the bite.



DIFFERENTIAL DIAGNOSIS

- Differential diagnoses for black widow spider bites include acute abdomen, back pain from intervertebral disk disease, marijuana intoxication, bromethalin toxicity, macadamia nut poisoning, and various drug toxicities.



DIAGNOSTICS

- At present there is no diagnostic test for the presence of black widow venom.
- Unless the owners saw the spider, a history is generally not helpful in matching clinical signs to a cause.
- On physical exam, due to dense hair coat and pigmentation, the bite site is rarely discovered.

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- Black widow spider bites only swell minimally and do not possess the dermonecrotic behavior sometimes associated with brown recluse bites.
- On complete blood count, black widow envenomations typically display a leukocytosis.
- Blood chemistry and electrolyte analysis may reveal marked elevations in CK and hypocalcemia.



THERAPEUTICS

- Historically, a variety of treatments have been tried for black widow spider envenomations. Muscle relaxants, intravenous calcium gluconate, opioids and *Latrodectus*-specific antivenom have all been employed.
- Local wound care of *Latrodectus* bites should include thorough wound cleansing, pain management, and an initial complete blood count and serum biochemistry profile. Urinalysis is recommended.
- Although calcium gluconate has been recommended in the past, it has been shown to be ineffective for pain relief when compared to opioids, benzodiazepines, and *Latrodectus* antivenin.
- *Latrodectus* antivenin is indicated in patients manifesting severe regional or systemic toxicity and for animals displaying hypertension, seizures, or impending respiratory arrest.
- The older *Latrodectus* antivenin was produced from horses and could cause serum sickness, anaphylaxis, and death. The newer, purified Fab-fragment antivenins are now available in South America and Australia and have been shown to rarely cause immunologic complications.
- In the US, antivenin is available (Lyovac [*Lactrodectus*] antivenin, equine origin but it should be reserved for severe envenomations. One vial is generally sufficient. Risk of allergic reaction to antivenin can be diminished by a pretreatment intradermal skin test of the antivenin or pretreatment injection of diphenhydramine (2–4 mg/kg subcutaneously). Antivenin provides the fastest, most effective relief to the more severely affected individuals.
- If antivenin is not available, animals should be treated with aggressive wound management, intravenous fluids, and a combination of opioids and benzodiazepines for pain management.



COMMENTS

Expected Course and Prognosis

- Spider bites can be prevented in domestic animals by providing a clean, safe, hygienic environment. Clearing all litter and yard debris, restricting animals from woodpiles, and regular household cleaning all cut down on prime spider habitat. Not allowing animals unsupervised access to sheds, garages, attics, and basements can also reduce the number of bites. Finally, properly insulating houses, especially all windows, exterior doors, attics and basement crawl spaces, and sealing all cracks and leaks with insulation material will do much to diminish spider numbers.

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Abbreviations

GABA: gamma-aminobutyric acid

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chapter 98

Spider Bite – Brown Spiders

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DEFINITION/OVERVIEW

- There are more than 35 000 species of spider worldwide (some spider experts think this number is closer to 100 000 species), the majority of which are venomous.
- Fortunately, only a few species have fangs capable of penetrating mammalian skin.
- A definitive diagnosis of spider bite is difficult to obtain. Clinicians must base their diagnosis on multiple criteria including geography, clinical signs, their own knowledge and experience of area spiders, and finally authoritative identification of the spider by an arachnidologist.
- Unfortunately, numerous clinical cases are based on presumptive bites which has led to misconceptions about spiders and misdiagnosed etiologies.
- Approximately 200 species from 20 genera worldwide can inflict severe envenomations on humans and animals, causing discomfort, neurotoxicity, systemic illness, dermonecrosis, and death.
- The most medically important groups of biting spiders include widow spiders (*Latrodectus* spp.), recluse spiders (*Loxosceles* spp.), and two spiders confined to single countries: the Australian funnel web spider (*Atrax* and *Hadronyche* spp.) and the armed or banana spider (*Phoneutria* spp.) from Brazil (Table 98.1).
- Spider bites have not been shown to transmit communicable diseases, do not generally become secondarily infected, and rarely trigger an allergic reaction.
- The collective term arachnidism is used to refer to envenomating spider bites; this includes all arachnids (i.e., scorpions, etc.). Envenomation by spider bite is more correctly called *araneism*. Spider bites can further be classified by systemic manifestations, such as *loxoscelism* (from brown spider bites) and *latrodectism* (from widow spider bites).
- Other species commonly implicated in bites in the United States include *Cheiracanthium* species (or yellow sac spiders) and *Tegenaria* species (or hobo spiders).
- Some reports state that you are never more than 1 meter away from a spider.
- In terms of numbers of species, spiders are the world's number one terrestrial carnivore.

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TABLE 98.1. Common North American spiders of minor medical importance.

Genus	Common name
Araneus	Orb weaver
Argiope	Garden spider
Bothriocytrum	Trap door spider
Drussodes	Mouse spider
Herpyllus	Parson spider
Heteropoda	Huntsman spider
Liocranoides	Running spider
Lycosa	Wolf spider
Misumenoides	Crab spider
Neoscona	Orb weaver, barn spider
Peucetia	Lynx, green lynx spider
Phiddipus	Jumping spider
Steatoda	False black widow
Ummidia	Trap door spider

Natural History

- Spiders of the genus *Loxosceles* are indigenous to temperate regions of Africa, Europe, and the Americas. Over 80% of the 100 species in the genus are found in the Americas.
- Eleven indigenous and two nonindigenous species inhabit the US, but only six species are of medical importance: *Loxosceles reclusa* (brown recluse), *L. laeta* (Chilean recluse), *L. arizonica* (Arizona recluse), *L. deserta* (desert recluse), *L. devia* (Texas recluse), and *L. rufescens* (Mediterranean recluse). Other endemic *Loxosceles* have minimal human and animal contact.
- Spider venom from spiders of the genus *Loxosceles* can cause serious envenomations.
- The brown recluse spider (*L. reclusa*) is the most clinically important member of this genus in the US.
- Brown recluse spiders are also known as "victim" or "fiddle-back" spiders on account of the violin or fiddle-back marking on the dorsal cephalothorax with the neck of the violin pointing to the rear of the spider. This mark is not always distinct, may fade with age, and other species may have similar markings, so the violin pattern is not diagnostic and should not be used as the only identifying characteristic.
- A unique feature of recluse spiders, however, is that while most spiders have eight eyes, *Loxosceles* spiders have six eyes arranged in pairs in a semicircle on the top of the head (one medium pair and two laterals). The six eye pattern is more diagnostic than the violin mark.

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- *Loxosceles* species are shy, nocturnal spiders that favor dark secluded places such as basements, closets, sheds, wood piles, attics, under rocks, and trash receptacles. (They are not called the "brown extrovert"!)
- Avoiding light and drafts, recluse spiders build large irregular, untidy flattened cobweb-type webs.
- Recluse spiders vary in color from light brown to tan, to orange-red, to yellow brown to brown to gray-brown.
- Mature brown recluse spiders measure 8–13 mm in body length, legs measuring 20–30 mm. The long legs can be up to five times the body length.
- Female brown recluse spiders are larger than males. Females may produce many egg sacs from May to July with as many as 50 eggs per sac.
- A comparatively long-lived spider, the female brown recluse can live up to 2 years.
- Brown recluse spiders are endemic to the south-central US, ranging from Texas to Kentucky and Georgia, Louisiana to Iowa, through southern Illinois, Indiana, and Nebraska. The spiders are a south-central and south Mid-western occurrence. *Loxosceles* spiders are active at night and prefer warmer climates; the majority of reported bites in the US occur south of Interstate 80. Despite the well-established range of *Loxosceles* spiders, physicians and veterinarians continue to diagnose brown spider envenomations in areas not endemic to the spider.
- Without a reliable eyewitness history of a capture of the offending spider, any claim of a spider bite, particularly a brown recluse bite in a nonendemic area, should be viewed skeptically.
- Brown recluse spider bites are tremendously overreported. In 2004, 738 brown recluse bites were reported in South Carolina when only 44 brown recluse spiders have ever been verified at six sites in the state since 1953.
- Although brown recluse spiders in the US are only found in the southern Mid-west and southern states (not in either coastal or Canadian border states), one study found that in humans greater than 50% of alleged recluse bites came from regions where the spider is not endemic and has never been reported.
- In humans, brown recluse spider bites are reported to be initially painless or mildly stinging. This may be because bites most commonly occur at night and might not be felt by the sleeping victim.
- Most bites occur in humans when the spider is trapped between the victim and clothing or bedding or is laid upon.
- Companion animals are typically envenomated when they lie down and trap the spider between themselves and bedding or the floor.

Pathogenesis

- *Loxosceles* venom acts through a number of pathways and mediators. Sometimes it produces a dermonecrotic lesion and rarely severe systemic illness.
- The key component to *Loxosceles* venom is a family of phospholipase D which is involved in and leads to the development of dermonecrosis.

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- Brown recluse spider venom is cytotoxic. The primary cytotoxic agent has been identified as sphingomyelinase D, which activates complement, polymorphonuclear cells, and platelets.
- Sphingomyelinase D attracts white cells to the bite site, triggers a chain reaction releasing inflammatory mediators, and causes vessel thrombosis, tissue ischemia, platelet aggregation and necrosis, and cell death (necrotic arachnidism).
- Sphingomyelinase D is capable of inducing all the clinical signs of brown recluse venom. Sphingomyelinase D makes up about 0.02% of the venom.
- Hyaluronidase is also present and responsible for the spread of the lesion with gravity (a characteristic of *Loxosceles* envenomation).
- The remainder of the venom is composed of alkaline phosphatase, a variety of proteases, collagenase, esterase, ribonuclease, and deoxyribonuclease.
- Brown spider venom disrupts connective tissue basement membrane and stimulates release of large amounts of interleukin-8, monocyte chemoattractant protein-1, and granulocyte-macrophage-colony-stimulating factor.
- Dermal histopathology of experimentally envenomated rabbits demonstrated edema of dermal endothelial cells, endothelial thickening, deposition of intravascular fibrin, and infiltration of inflammatory cells, particularly polymorphonucleocytes. All these substances amplify the inflammatory response, accounting for the severe local reaction.
- Studies in humans and rabbits reveal that *Loxosceles* venom induces intravenous clotting, causing occlusion of dermal venules and arterioles, adding to the tissue hypoxia contributing to the tissue necrosis.
- Bites may progress to a central region of impending necrosis surrounded by concentric edema (the "bull's eye" or "target sign"), heralding "necrotic arachnidism." This cutaneous form occurs in less than 10% of cases.
- Brown recluse spider bites were confirmed to cause necrotic arachnidism in 1957.
- Systemic loxoscelism is much less common than the cutaneous form but the frequency seems to vary with different species (in less than 1% of cases, dependent on *Loxosceles* species involved).The most consistent features of systemic loxoscelism include intravascular hemolysis, disseminated intravascular coagulation, and renal failure.
- The hallmark lesion of systemic loxoscelism is acute intravascular hemolysis. The degree of hemolysis can be profound. A lack of hemolysis can rule out systemic involvement.
- Renal injury results from *Loxosceles* toxins binding to glomerular and tubule cells. Renal failure is infrequent but associated with less favorable outcome.

Toxic Dose

- *Loxosceles* species vary in venom strength. Likewise, venom amounts and strengths can also vary with the size and sex of spider involved. Males of equivalent size generate half the female volume.
- *Loxosceles* venom is remarkable when the amount of venom injected and the severity of the resultant tissue damage are considered. The average Eastern diamondback rattlesnake (*Crotalus adamanteus*) injects an average of 200–850 mg of venom per bite. *Loxosceles*

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envenomations typically produce an average of 30–65 mg of protein. Larger spiders can release up to 70 mg.

- A single bite can be lethal.
- Like venomous snakes, spiders are able to meter the amount of venom released. In offensive bites, hunting spiders release enough venom to immobilize and kill their insect prey. Defensive bites release more venom against attackers. In agonal bites, dying spiders release all of their venom. In contrast, bees and wasps release a consistent amount of venom with each sting.
- Not only can venom strength vary with species of *Loxosceles* and size and sex of the spider but also the venom produces very different toxicities in different mammals. Rats and mice develop dermonecrotic lesions while guinea pigs and rabbits do not. Brown recluse venom will lyse erythrocytes of humans and pigs but not those of dogs, rats or guinea pigs. On account of this variability, care must be taken with extrapolation between species involved.
- Despite its reputation and the fear surrounding this spider, in the US between 1983 and 2004 only eight deaths were attributed to brown recluse spider bites. Moreover, in all eight cases the envenomation was presumed – no definitive spider identification or positive confirmation of a bite was obtained.



CLINICAL FEATURES

- In humans, brown recluse spider bites are fairly painless, with victims often completely unaware of the event, so diagnosis is rarely based upon identification of the biting spider.
- For people, the hallmark lesion produced by the brown recluse spider is dermonecrotic skin, but a great range exists in the manifestation of this envenomation.
- The clinical spectrum of "loxoscelism" (brown recluse spider bites) can be divided into three classes.
    - First, the majority of bites are characterized by no clinical signs or local irritation. In this first class, very little venom if any is injected. A small, erythematous papule may be present and can be associated with a local urticarial response. The true percentage of minor bites is impossible to establish since victims who do not feel the bite will disregard minor symptoms or misdiagnose the actual cause.
    - In class two, more venom is introduced and is cytotoxic. The initially painless bite may now blister and bleed. The now stinging lesion may increase in diameter, ulcerate, and display central hemorrhagic vesiculation and necrosis. The central ulcer is surrounded by ischemic blanching and erythematous induration, worsening over the next 1–3 days. Necrosis of the central blister follows in 3–4 days, and eschar formation after 5–7 days. After 7–14 days, the bite wound eschar falls off, leaving an ulcer that must heal by secondary intention. Healing of this lesion may take weeks. This second class of clinical signs is call "necrotic arachnidism" or "cutaneous loxoscelism." This form is not as common and occurs less than 10% of the time, depending upon the species of spider involved and the species of victim involved.

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  - The third class is "systemic loxoscelism." This form is not predicted by the extent of the cutaneous reaction. It typically begins 24–72 hours after the bite. The hallmark of this stage is a Coombs negative hemolytic anemia. In addition to hemolysis, clinical manifestations of the systemic form include fever, weakness, vomiting, arthralgia, disseminated intravascular coagulation (DIC), thrombocytopenia, renal failure, and death. If hemolysis is not seen, it may be assumed that the systemic form is not present. Systemic loxoscelism is not common and rarely seen (less than 1% of the time) in both humans and animals bitten by brown recluse spiders in the US.
- The initial mild appearance of the lesion makes accurate clinical diagnosis difficult. Severity of brown recluse bites depends upon the amount of venom injected, site of the envenomation, and size and immune status of the victim.
- In humans, envenomations in pregnant women led to no fetal abnormalities.



DIFFERENTIAL DIAGNOSES

- Spider bite is greatly overdiagnosed and is poorly used as a "speculative" diagnosis.
- Prognosis is even harder to establish due to the mild appearance of the lesion and since the culprit spider is rarely seen or captured.
- The list of diagnoses that can cause dermonecrotic lesions is extensive and includes various bacterial, viral, and fungal infections, primary dermatides, vasculitides, thromboembolic phenomena, adverse drug reaction, diabetic and artificial ulcers, decubital ulcers, skin cancers and other neoplasia, bites of other arthropods or animals, and thermal and chemical burns.
- In humans, skin and underlying soft tissue infections with variously acquired methicillin-resistant *Staphylococcus aureus* (MRSA) are currently some of the most common causes of skin lesions attributed to spider bites.
- A list of disorders that mimic spider bites is included in Box 98.1.

BOX 98.1. Differential diagnoses of spider bites.

Allergic contact dermatitis
Chemical burns
Trauma
Bacterial skin infections
Dermatomycosis
Other arthropod bites and stings
Methicillin-resistant *Staphylococcus aureus* (MRSA) skin lesions

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DIAGNOSTICS

- Definitive diagnosis of a brown recluse spider bite is achieved only when the biting spider is positively identified.
- To be considered a confirmed envenomation, the spider must be recovered immediately in close proximity to the skin reaction.
- Ideally, the spider will also be identified by a qualified entomologist or arachnidologist.
- Possible envenomation must display a lesion typical of a brown recluse bite and have occurred in an area where the spider is endemic.
- Currently, there are no commercially available tests to confirm loxoscelism.
- Anatomic location of spider bites on the victim appears to be far more dependent upon the activity of the victim rather than on the spider's typical behavior, preferred habitats, or feeding habits. Thus, the location of a spider bite on a victim is nondiagnostic.
- The diagnosis of brown recluse bites is frequently overused for dermonecrotic lesions of uncertain etiology, especially for locations in the US where the spider is not even endemic.
- The most accurate diagnostic test for *Loxosceles* envenomation is detection of either the venom itself or circulating antibodies.
- Biopsy of the bite site is not recommended since histopathologic findings are nonspecific and not unique to loxoscelism.
- In general, positive diagnosis of brown recluse bite is not made since the victim rarely feels the bite, seldom is the spider seen, and presentation for medical treatment typically occurs several hours after the event.
- A passive hemagglutination test has been developed in guinea pigs for detection of the venom. The test measures the venom's ability to inhibit agglutination of venom-sensitive erythrocytes. This test displays 90% sensitivity and 100% specificity if performed within 3 days of envenomation. It is labor intensive, costly, and not available commercially.
- An enzyme-linked immunosorbent assay (ELISA) test specific for *Loxosceles* venom has been developed. A second ELISA test that uses hair, skin biopsies, or aspirated tissue near a suspected lesion has likewise been investigated. Currently, these tests are not commercially available in the US.
- Lymphocyte transformation following brown recluse envenomation has also been demonstrated but it too is costly, prohibitively labor intensive, and currently not commercially available.
- Although a variety of these tests are promising, until the ELISA test becomes commercially available, there is no gold standard diagnostic test for *Loxosceles* envenomation in the US, other than an eyewitness account of the event.



THERAPEUTICS

- A definitive therapeutic approach to management of loxoscelism has yet to reach consensus.
- A variety of interventions have been proposed, including steroids, dapsone, colchicine, surgical excision, vasodilators, antihistamines, antibiotics, topical nitroglycerine, anticoagulants,

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hyperbaric oxygen, electric shock, tourniquets, compressive bandages, suction devices, high doses of vitamin C, meat tenderizer, and antivenom.

- The one thing the host of recommended treatment protocols have in common is attempting to counter the dramatic influx of neutrophils, activation of complement, and subsequent tissue destruction of the classic lesion.
- One of the first interventions recommended for cutaneous brown recluse spider bites was surgical debridement and skin grafting. However, early surgical management has been found to be ineffective and often harmful as an initial management. As a result, surgical excision has fallen out of favor.
- In brown recluse spider bite wounds, it may take several days to weeks for the lesion to declare itself and demonstrate the final extent of necrosis and tissue destruction.
- In humans, less than 3% of cutaneous loxoscelism victims require surgery or skin grafting and this is usually because the lesion is greater than 2 cm or underlying problems such as peripheral vascular disease or diabetes mellitus impede primary healing.
- The theoretical mechanism of action of polymorphomuclear cell inhibitors (such as dapsone and colchicine) is that they halt expanding dermonecrosis through inhibition of leukocyte migration, degranulation, and cytokine release.
- Dapsone (diamino-diphenylsulfone) is an antimycobacterial used for leprosy management in humans and felines and as an alternative treatment for pemphigus in small animals. It inhibits influx of neutrophils but its adverse effects are numerous and can be severe. Adverse effects include anemia, leukopenia, thrombocytopenia, hepatotoxicity, gastrointestinal signs, neuropathies, and cutaneous drug eruptions that look similar to spider bite. If not given within hours of the bite, dapsone is not effective. Since its concrete effectiveness has not been demonstrated and for the sheer number of side effects, the administration of dapsone is not recommended.
- The majority of *Loxosceles* envenomations result in little more than a mild inflammatory reaction. Nevertheless, corticosteroids are often prescribed for short periods following the bite. There is insufficient evidence to merit the use of corticosteroids for brown recluse bites but the theoretical mechanism behind their use would be their immunosuppressive action blunting the immune system response to the bite. However, no investigative studies have been conducted to confirm the beneficial activity of steroids in clinical cases.
- Another possible intervention used in treatment of brown recluse spider bites is the application of hyperbaric oxygen. The proposed mechanism of this treatment is the promotion of neovascularization and increased oxygen availability to ischemic tissue. However, sphingomyelinase activity of the venom is not inhibited by hyperbaric oxygen and the majority of studies in animals showed no reduction in the amount of necrosis at the lesion site. One study claimed hyperbaric oxygen therapy to be effective if administered within 6 hours of envenomation. However, the vast amount of studies show that the majority of brown recluse spider bites on animals are not recognized or treated until more than 6 hours after the bite. At present, there is no conclusive evidence to support the use of hyperbaric oxygen treatment in treatment of loxoscelism.
- Topical application of nitroglycerine has been reported to decrease wound size, but controlled animal studies showed no impact on lesion size using topical nitroglycerine at the envenomation site.

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- Tetracycline protects against dermonecrosis and lesion formation in rabbits when applied topically but not if injected. Doxycycline was less effective than tetracycline but still proved capable of preventing an increase in size of the lesion while oral administration of both tetracycline and doxycycline was much less effective, possibly due to the level of concentration achieved. Sphingomyelinase D, the major component of *Loxosceles* venom, binds to molecules that stimulate the influx of neutrophils, lymphocytes, and eosinophils. Studies report that topical treatment with tetracycline significantly decreases this influx.
- The use of tourniquets, electric shock, heat, suction devices, meat tenderizer, and antihistamines has been shown to be ineffective in spider bite management. These treatments waste valuable time until effective therapy can be initiated and several can actually be harmful.
- Brown recluse spider bites in both humans and animals have a very low incidence of subsequent bacterial infection. Nevertheless, many suspected spider bite victims are treated with antibiotics. Many researchers report that antibiotic coverage following a brown recluse envenomation, even with developing necrotic ulceration, is unnecessary and inappropriate.
- Antivenoms for *Loxosceles* spiders have been produced in South America, none of which are commercially available in the US. All these are horse-derived F(ab)2 antivenoms, one whole IgG antivenom from Peru, and one rabbit-derived Fab (alpha-Loxd) anti-*Loxosceles* antivenom.
- Antivenom has shown some success in animal studies if given within 1 hour of the envenomating bite. The indications for antivenom administration are usually based upon severity of the envenomation. Unfortunately, delayed recognition of loxoscelism means antivenom is often given too late when patients have already developed substantial necrosis and/or systemic signs. Typically antivenom is not recommended more than 72 hours after the bite. The Brazilian Ministry of Health recommends antivenom use in cases of extensive necrosis or evidence of the effects of systemic loxoscelism. Institute Butantan in Brazil has produced antivenoms for *L. reclusa* and *L. rufescens*. Currently, no antivenoms for recluse spider bites are available in the US.
- Despite such a wide variety of recommended treatments, no clinical studies exist which support a definitively effective therapy. Clinical studies are urgently needed to establish a definitive method to diagnose brown recluse spider bites and to provide a safe and effective treatment for victims.
- Treatments not recommended for brown recluse spider bite are listed in Box 98.2.
- A concrete diagnosis of brown recluse spider bite in small animals is difficult to confirm. In addition, if small animals are bitten, only in a small percentage of cases are the effects serious (cutaneous loxoscelism about 4% of the time, systemic loxoscelism less than 1% of the time). As a result, treatment including observation and rest, thorough cleaning of the bite wound, ice, and elevation of the site must be the first line of defense.
- Systemic loxoscelism is very rare and may be due to decreased renal function. Furthermore, secondary infections are most common in immune-compromised or diabetic animals. Animals displaying systemic signs should be hospitalized and intravenous fluids initiated to maintain hydration and protect renal function.

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BOX 98.2. Treatments not recommended for brown recluse spider bites in animals.

Dapsone
Colchicine
Topical nitroglycerine
Electric shock
Meat tenderizers
Surgical excision
Suction devices
Tourniquets
Hyperbaric oxygen
Vitamin C

- In humans, wound care alone, including debridement of necrotic tissue, culture-directed antibiotic therapy of secondary infections, and delayed assessment and management of eschars, has proven to be successful. With proper wound management, brown recluse spider envenomations heal within 1–8 weeks with a 10% chance of significant permanent scarring.
- As in all facets of emergency medicine, in treating spider bites we must acknowledge the age-old notion, "First, do not harm"!



COMMENTS

Prevention/Avoidance

- The shy nature of *Loxosceles* species and a high tolerance to pesticides make extermination of these spiders extremely challenging.
- Distribution of pesticides in households rarely reaches under surfaces where brown recluse spiders live. *Loxosceles* species lack foot pads through which other arachnids absorb pesticides.
- Control by spraying is problematic. Spiders are killed by sprays only if the spray lands directly on them. Also, spray residual does not have a long-lasting effect. Spiders can walk over a sprayed surface in a few days (in some cases within a few hours) after treatment with no effect. The long legs of the brown recluse allow them to walk over most pesticides unharmed.
- Brown recluse spiders have shown resistance to many formulations, even exposure to dichlorodiphenyltrichloroethane.
- In certain indoor situations, sorptive dusts containing amorphous silica gel (silica aerogel) and pyrethrins can be applied. The dust contacts the outer covering of the spider and dries them out. A sorptive dust is applied as a dust-like film and left in place in crevices, cracks, and enclosed places in the home. This dust must only be applied by licensed professional pest control applicators.

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- Nonlethal, chemical repellants have been manufactured for a variety of spiders but in general do not seem to be effective.
- For the most part, the use of pesticides for spider control is impractical, ineffective, and unnecessarily toxic to nontarget species and the environment.
- Sticky traps can be effective and provide an idea of local spider population numbers.
- Regularly clean dark, unused areas of the house to decrease spider habitat.
- Vacuum up all visible spider webs and cobwebs.
- Decrease household insect numbers that are the prey of the spiders.
- Reduce and remove trash and rubbish from your property, such as woodpiles, boxes, old tires, plywood, and trash cans – especially if they are stored right next to the house. Declutter!
- Do not confine animals in areas of prime spider habitat. Peak period of envenomation is summertime.
- Bites often occur in beds when people and animals roll over on the spider during sleep.
- Pulling beds away from walls minimizes the chances that a spider may crawl onto the bed.
- Do not leave clothes and shoes on the floor or shake them before dressing if they are left out.
- Most spiders avoid well-lit, well-ventilated areas. The best preventive is to decrease clutter and areas spiders might inhabit.

Expected Course and Prognosis

- Dermonecrotic wounds of unknown or uncertain origin are attributed to the bite of a brown recluse spider.
- These bites are much overdiagnosed even in locations where the spiders are rare or not even found.
- Misdiagnosis of these wounds can lead to delays in appropriate care, adverse outcomes, and increased medical legal risk, particularly if there is specific treatment for the actual underlying cause.
- Early and aggressive wound management is the cornerstone of effective intervention.
- The majority of brown recluse spider bites heal uneventfully in 1–3 weeks.
- A very small percentage may require surgical intervention (which has fallen into disfavor as a treatment modality).
- An even smaller number may develop systemic illness and even more rarely death.
- The overall outcome of the vast majority of brown recluse spider bites is overwhelmingly favorable.

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chapter 99

Spinal Fracture

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DEFINITION/OVERVIEW

- Fracture, subluxation, or luxation of the spine causing neurologic deficits via laceration, compressive, or concussive injury to the spinal cord and/or nerve roots.

ETIOLOGY/PATHOPHYSIOLOGY

- Fractures/luxations: commonly at thoracolumbar region (50–60% of cases), followed by caudal lumbar to lumbosacral. Cervical fractures are uncommon, typically affecting the cranial cervical region, i.e., atlantoaxial and C2.
- Primary mechanical injury: initial compressive or concussive injury to the spinal cord.
- Secondary injury: includes spinal cord ischemia, hemorrhage, edema, and necrosis; mediated by endorphins, catecholamines, free radicals, and excitatory neurotransmitters.

Systems Affected

- Nervous: signs range from pain to paralysis without deep pain sensation.
- Musculoskeletal: concurrent appendicular injury is not uncommon.
- Soft tissue injury to the thoracic and/or abdominal structures.

SIGNALMENT/HISTORY

Species

- Dogs and cats.

Historical Findings

- Trauma: hit by car most common; bite and gunshot wounds.
- Unknown: acute paralysis or neurologic signs.

Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Emergency and Critical Care, Second Edition. Edited by Elisa M. Mazzaferro.
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Companion website: www.fiveminutevet.com/emergency

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CLINICAL FEATURES

- Initial physical and neurologic examination should be conducted prior to analgesia and sedation.
- Spinal pain; hyperesthesia.
- Ataxia to nonambulatory.
- Palpable spinal deformity.
- May have signs of spinal shock.
- Schiff-Sherrington posture: spinal cord injury may cause paraplegia with increased extensor tone in the thoracic limbs.
- Often other signs of trauma including intrathoracic and intraabdominal.

DIFFERENTIAL DIAGNOSIS

- Spinal cord compressive lesions; intervertebral disk disease, tumor, or hemorrhage
- Pathologic spinal fracture
- Spinal infarction, e.g., fibrocartilaginous embolism
- Myelitis
- Spinal epidural empyema

DIAGNOSTICS

- Radiographs: survey radiographs of the entire spine to determine severity and locations; accuracy is only 75% – multiple spinal fractures are seen in 20% of cases.
    - Sedation/general anesthesia may be necessary for a good radiographic study; caution as voluntary paraspinal muscle contraction will be lost and the spine may become more unstable.
    - Lateral and horizontal beam: ventrodorsal position may lead to neurologic worsening, i.e., consider advanced cross-sectional imaging via CT or MRI.
    - Radiographs show the current amount of displacement, not the amount of displacement that occurred at the time of the injury (Figs 99.1, 99.2).
    - Advanced imaging is mandatory when radiographs do not correlate with clinical neuroanatomic localization.
- Myelography, CT, and MRI: much improved accuracy in detecting the nature, number, location, and degree of spinal cord compression.

THERAPEUTICS

- The objective is immediate spinal cord stabilization (Fig. 99.3) to prevent additional trauma during diagnosis and treatment of life-threatening conditions.
- Treat all life-threatening conditions.

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■ **Fig. 99.1.** Lateral radiograph of a dog with a vertebral body fracture of L6.



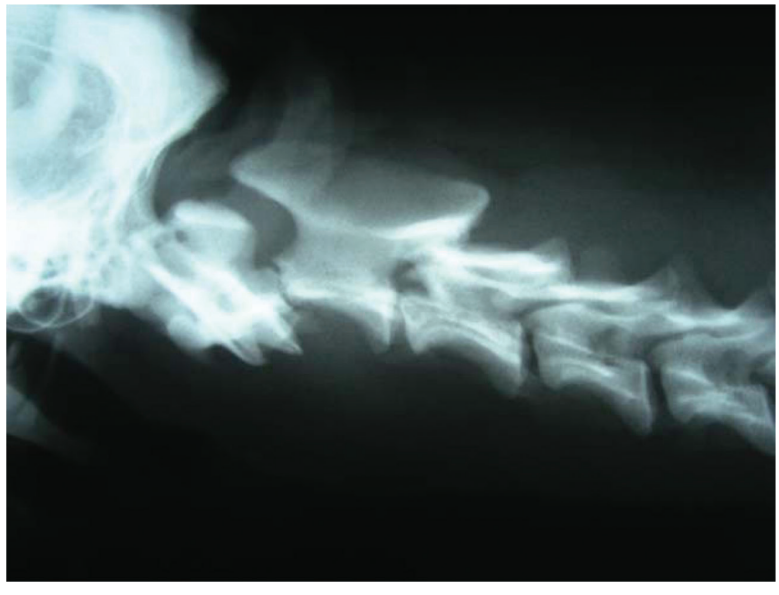
■ **Fig. 99.2.** Ventrodorsal radiograph of a dog with a vertebral body fracture of L6. This dog previously had a TPO for hip dysplasia.

- Maintain mean arterial blood pressure over 85 mmHg to increase spinal cord perfusion.
- Medical management: used in a stable spine with minimal spinal cord compression and mild or minimal neurologic deficits.
    - Splint/bandage stabilization: if cranial cervical, bandage should extend from the head to just caudal to the lateral canthi of the eyes (Figs 99.4, 99.5).

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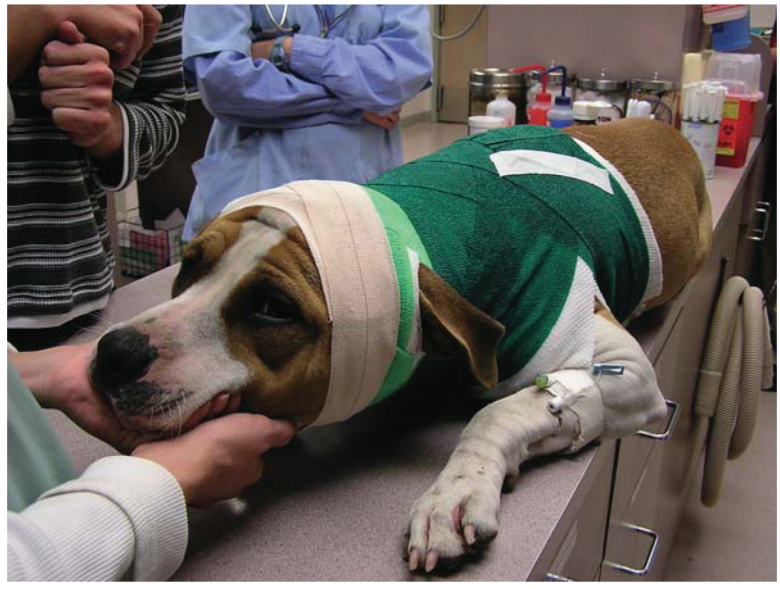
■ **Fig. 99.3.** Management of the patient with spinal trauma involves providing complete immobilization by securing the animal's head and shoulders and hips to a fixed firm surface, to prevent movement and further damage to the spinal cord if fracture or luxation/subluxation is present.



■ **Fig. 99.4.** Fracture of the dens of C2. The patient was ambulatory with no neurologic deficits and had presented on emergency for neck pain.

- Cage rest 4–6 weeks.
- Monitor temperature, ventilation, and irritation of the skin by friction of the bandage.

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■ **Fig. 99.5.** Conservative medical management of a patient with dens fracture of C2. Note that the splint is extended over the animal's head to prevent movement of the head and neck.

Drugs of Choice

- More recent clinical evidence contradicts the use of low- or high-dose corticosteroid protocols. These drugs have not shown significant improvement in clinical outcome. In addition, corticosteroids are associated with a significant increase in gastrointestinal complications and infection rates.
- Apart from their analgesic effect, NSAIDs have minimal benefit in acute spinal cord injury and increase the risk of complications (especially if used in conjunction with corticosteroids).
  - Dogs: carprofen 2.2 mg/kg PO every 12 hours, 4 mg/kg SQ once.
  - Cats: meloxicam 0.1 mg/kg PO every 24 hours for 4 days, 0.3 mg/kg IV or SQ once.
- Opioids: hydromorphone 0.05–0.2 mg/kg IV, IM, or SQ every 2–4 hours; fentanyl CRI 2–4 µg/kg/h.

Precautions/Interactions

- Do not combine corticosteroids and NSAIDs.
- Corticosteroids are absolutely contraindicated in head trauma.
- Corticosteroids increase the risk of gastric ulcers, sepsis, wound infection, and respiratory complications in these patients.
- Caution with NSAIDs in dehydrated patients.

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Activity

- Immobilization: stabilize on a board or gurney using straps or tape that will allow for treatments and diagnostics with limited movement of the spine.
- Cage rest for 4–6 weeks if medically managed.

Surgical Considerations

- Spinal cord compression causing severe neurologic dysfunction.
- May be indicated for inherently unstable spinal fractures regardless of neurologic status.
- Internal or external fixation: to be determined by the surgeon.

COMMENTS

Client Education

- The most important prognostic factor after spinal cord trauma is the presence or absence of nociceptive ability.
- Neurologic signs may progress, e.g., ascending/descending myelomalacia.
- Discuss intensive home care and physical therapy requirements after discharge.

Patient Monitoring

- Serial neurologic examination every 4–6 hours for deterioration
- Vital parameters
- Blood pressure
- Micturition ability

Possible Complications

- Decubital ulcers
- Urinary tract infection due to retention
- Aspiration pneumonia from recumbency
- Gastrointestinal ulceration with corticosteroid use
- Implant failure or infection

Expected Course and Prognosis

- Prognosis is based on the underlying trauma and the presence or absence of deep pain.
- In general, animals in which any degree of nociceptive ability is detected with certainty may be expected to be restored to locomotion.
    - Loss of deep pain <24 hours, <20% chance of recovery.
    - Loss of deep pain >24 hours, condition is grave.
    - Loss of deep pain with 100% vertebral displacement, virtually no chance of recovery.

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Abbreviations

CRI: constant rate infusion
CT: computed tomography
IM: intramuscular
IV: intravenous
MRI: magnetic resonance imaging
NSAID: nonsteroidal antiinflammatory drug
SQ: subcutaneous
TPO: triple pelvic osteotomy

See Also

Schiff-Sherrington
Spinal Shock

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chapter 100

Spinal Shock



DEFINITION/OVERVIEW

- The loss of muscle tone and segmental spinal reflexes caudal to a severe spinal cord injury.



ETIOLOGY/PATHOPHYSIOLOGY

- Poorly understood in companion animals; likely a sudden interruption in descending supraspinal input.
- Spinal reflexes are lost after acute spinal cord injury because descending facilitatory inputs are lost, causing a hyperpolarization of spinal motor neurons and a reduction in their excitability.
- Acute spinal cord trauma: usually a solitary lesion but spinal shock can give the appearance of a multifocal lesion and lead to confusion regarding neuroanatomic localization.
- Should be suspected with flaccid paralysis caudal to the level of injury with sudden return of reflexes in a quicker than expected recovery period.
- May only last 30–60 minutes in small animals.

Systems Affected

- Nervous: lesion localization is initially difficult to establish; loss of reflexes and muscle tone caudal to an acute spinal cord injury with rapid return of reflexes.
- Musculoskeletal: paralysis, decreased muscle tone.



SIGNALMENT/HISTORY

Species

- Dogs and cats: rare; likely underreported.

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Historical Findings

- Trauma: hit by car most common.
- Unknown: acute paralysis or neurologic signs.



CLINICAL FEATURES

- Initial physical and neurologic examination should be conducted prior to analgesia and sedation.
- Clinical features dictated by the cause of the acute spinal cord injury and spinal shock.
- Nonambulatory.
- Transient hypo- to areflexia in limbs caudal to the level of spinal cord injury.
- Although shock is relatively brief in animals compared with humans, it is likely to be encountered in animals presented promptly after injury, when it can complicate lesion localization.
- Often there are other signs of trauma.



DIFFERENTIAL DIAGNOSIS

- Myelomalacia: ascending/descending
- Spinal cord compressive lesions: intervertebral disk disease, tumor, hemorrhage
- Spinal infarction (e.g., fibrocartilaginous embolism) with secondary hemorrhage
- Spinal fractures, luxations
- Myelitis
- Spinal epidural empyema



DIAGNOSTICS

- Radiographs: lateral and horizontal beam.
- Myelography, CT, MRI: advanced imaging to evaluate multiple spinal cord segments and rule out additional lesions that may be responsible for the observed lower motor neuron signs.



THERAPEUTICS

- The objective is immediate spinal cord stabilization to prevent additional trauma during diagnosis and treatment of life-threatening conditions.
- Maintain mean arterial blood pressure over 85 mmHg to maintain spinal cord perfusion.
- Assisted micturition: manual voiding, intermittent or indwelling catheterization.
- Supportive care of the recumbent patient.

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Drugs of Choice

- More recent clinical evidence contradicts the use of low- or high-dose corticosteroid protocols. These drugs have been associated with a significant increase in gastrointestinal complications and infection rates.

Precautions/Interactions

- Do not combine corticosteroids and NSAIDs if used for analgesic purposes.
- Corticosteroids are contraindicated in head trauma.
- Corticosteroids increase the risk of gastric ulcers, sepsis, wound infection, and respiratory complications in these patients.
- Caution with NSAIDs in dehydrated patients.

Activity

- Immobilization: stabilize on a board or gurney using straps or tape that will allow for treatments and diagnostics with limited movement of the spine.

Surgical Considerations

- May be indicated for spinal stabilization or spinal cord decompression.

COMMENTS

Client Education

- Without advanced spinal imaging, complete neurologic assessment and lesion localization may not be possible for several days due to this transient phenomenon.

Patient Monitoring

- Serial neurologic examination every 4–6 hours for change
- Vital signs
- Blood pressure
- Micturition ability

Possible Complications

- Decubital ulcers
- Urinary tract infection due to retention
- Aspiration pneumonia from recumbency
- Gastrointestinal ulceration with corticosteroid use

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Expected Course and Prognosis

- Return of reflexes after experimental nonprimate spinal cord transection:
  - Anal sphincter reflex 15 min
  - Patellar reflex 30 minutes to 2 hours
  - Flexor withdrawal reflex up to 12 hours.
- Spasticity and hyperreflexia develop within 24–48 hours.
- Crossed extensor reflex may develop in 1–2 weeks post injury.
- Prognosis is based on the underlying trauma and the presence or absence of deep pain; the presence of spinal shock does *not* change the overall prognosis.

Abbreviations

CT: computed tomography
MRI: magnetic resonance imaging
NSAID: nonsteroidal antiinflammatory drug

Suggested Reading

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Author: Rainier Ko, DVM

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chapter 101

Splenic Torsion





DEFINITION/OVERVIEW

- Isolated splenic torsion uncommon.
- Can be seen in association with gastric dilation-volvulus syndrome.
- May also occur as a separate entity as acute or chronic form.

Pathophysiology

- Unknown.

Systems Affected

- Cardiovascular: torsion mechanically compresses the thin-walled splenic veins, resulting in vascular congestion.
- Hemic/lymphatic/immune: arteries become occluded with thromboemboli and resultant infarction.



SIGNALMENT/HISTORY

- More common in large breed, deep-chested dogs, such as German shepherds, standard poodles, Great Danes, and retrievers.
- No age predilection,
- Males more affected than females,



CLINICAL FEATURES

Historical Findings

- Acute: cardiovascular collapse and abdominal pain.
- Chronic: duration of clinical signs up to months, intermittent anorexia, vomiting, weight loss, and possibly hemoglobinuria.

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Physical Examination Findings

- Acute: pale mucous membranes, tachycardia, and other signs of hypoperfusion and shock.
- Discomfort, acute abdominal pain.
- Retching, drooling, weakness.
- Possible palpable abdominal mass (spleen) depending on duration of torsion and abdominal splinting.
- Chronic: vague signs, depression, lethargy.

Risk Factors

- Large breed and deep-chested dogs.
- Prior stretching of gastrosplenic, phrenicosplenic, and splenocolic ligaments (e.g., prior gastric dilation and volvulus).
- Historical gastric dilation.
- Excessive exercise, rolling, and retching may contribute.
- Nervousness and anxiety have been associated with an increased risk of GDV.



DIFFERENTIAL DIAGNOSIS

- Other splenic disease (e.g., neoplasia, especially hemangiosarcoma, trauma-hematoma, splenic abscess, immune-mediated disease)
- Acute gastrointestinal disease with abdominal pain
- GDV
- Other midabdominal masses (e.g., gastrointestinal, pancreatic, and renal)
- Other causes of intravascular hemolysis



DIAGNOSTICS

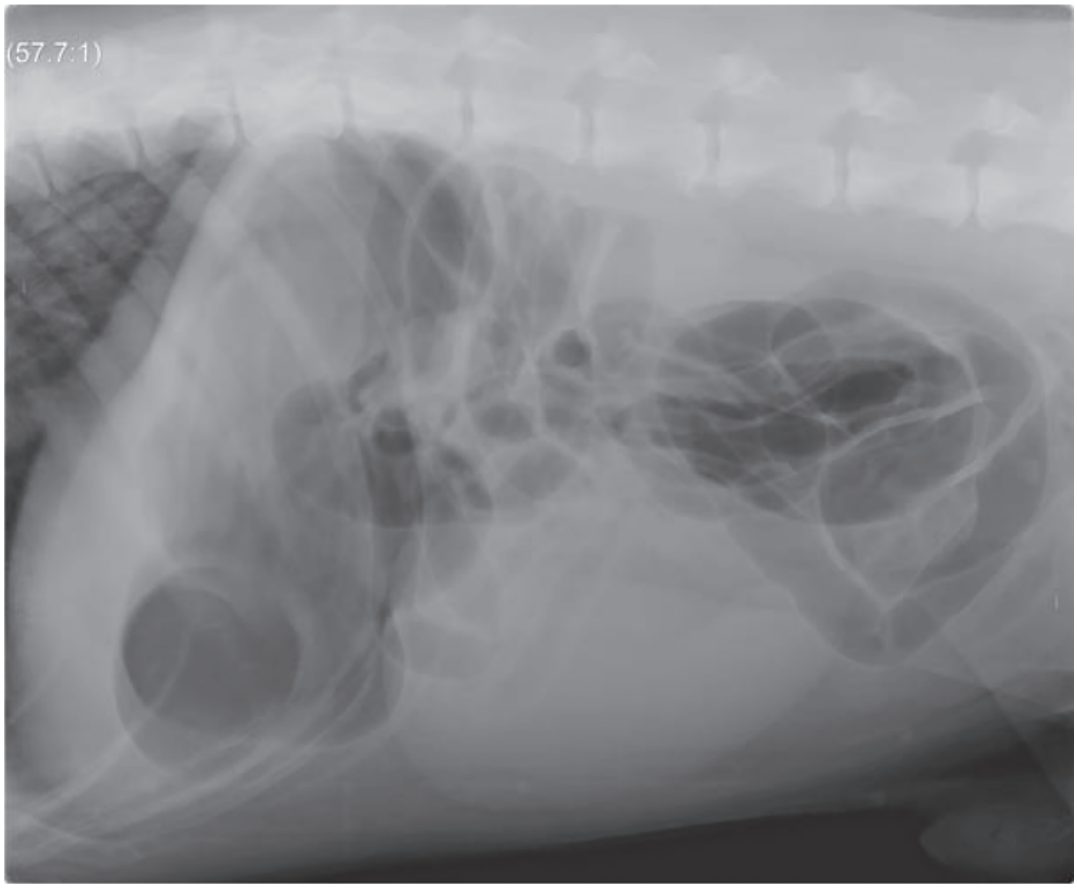
CBC/Biochemistry/Urinalysis

- Acute: blood work generally not helpful
- Chronic: variable changes
- Anemia
- Thrombocytopenia
- Leukocytosis
- High liver enzyme values
- High pancreatic enzyme values
- Hemoglobulinuria

Other Laboratory Tests

- Coagulation test: DIC (prolonged prothrombin time, partial thromboplastin time, and increased fibrin split products) because of accelerated consumption.

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■ **Fig. 101.1.** Lateral abdominal radiograph of a dog with splenic torsion.

Imaging

Abdominal Radiography

- Cranial or midabdominal mass may be seen (Figure 101.1).
- Spleen may be abnormally located, misshapen, multifocal gas opacities.
- Displacement of gastrointestinal tract.
- Loss of abdominal detail (possible).

Abdominal Ultrasonography

- Splenic congestion, splenomegaly.
- Dilated splenic veins (multiple parallel echogenic lines).
- Diffuse hypoechoic areas with linear echoes separating large, anechoic areas.
- Splenic infarction.
- No blood flow (absent velocity) in splenic veins with Doppler ultrasonography.
- Visible splenic vein intraluminal echogenicities compatible with thrombi.

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Computed Tomography

- In case of inconclusive radiology and ultrasound exam.
- Enlarged spleen with corkscrew-like soft tissue mass representing the rotated pedicle.
- Missing contrast enhancement as a sign of compromised splenic perfusion and indication for splenic removal.

Other Diagnostic Procedures

- ECG: may show ventricular arrhythmias as a risk factor for perioperative death.

Pathologic Findings

- Splenic congestion and infarction.



THERAPEUTICS

Surgical Emergency

- Anesthesia: avoid barbiturates, acepromazine or other drugs causing splenic congestion.
- Surgery: after adequate cardiovascular stabilization, a splenectomy should be performed without untwisting the splenic pedicle.
- Derotation of an acute torsion without splenectomy might be possible but is not recommended.
- Avoid ligation of pancreatic vascular supply by vessel ligation close to the spleen.
- The vascular pedicle cannot be untwisted in the chronic form due to fibrosis, thrombosis, and risk of splenic rupture.
- A splenic specimen should be submitted for histopathologic examination.
- Conflicting data regarding necessity of prophylactic permanent gastropexy.
- Fluid support and cardiovascular monitoring indicated after splenectomy.

Drugs of Choice

- No specific drugs required.
- Postoperative pain relief advised.
- Plasma transfusion may be considered if DIC and coagulopathy are documented.



COMMENTS

Follow-Up

- Surgical correction considered curative.
- Delayed treatment might lead to splenic necrosis, peritonitis, and sepsis.

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Abbreviations

CBC: complete blood count
DIC: disseminated intravascular coagulation
ECG: electrocardiogram
GDV: gastric dilation and volvulus

Suggested Reading

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Author: David E. Spreng, DVM, PhD

chapter 102

Supraventricular Tachycardia

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DEFINITION/OVERVIEW

- Repetitive supraventricular premature depolarizations that originate from a site other than the sinus node, such as the atrial myocardium or atrioventricular nodal tissue.

Electrocardiogram Features

- Heart rate: rapid, 150–350 beats per minute in dogs. The lower rate of SVT depends on the size of the patient. Smaller dogs typically have higher sinus nodal rates than larger dogs.
- Rhythm usually very regular (R-R interval is constant) and may be sustained, but there can be frequent or infrequent short runs of SVT, so-called paroxysmal SVT. Rarely, the rhythm during the tachycardia will be irregular, suggesting abnormal automaticity as the etiology (Fig. 102.1).
- Usually the QRS complexes are typical of normal sinus complexes, narrow with a normal mean electrical axis. In some cases, a coexisting bundle branch block or aberrant ventricular conduction makes it difficult, if not impossible, to differentiate an SVT from a ventricular tachycardia by examining the ECG.

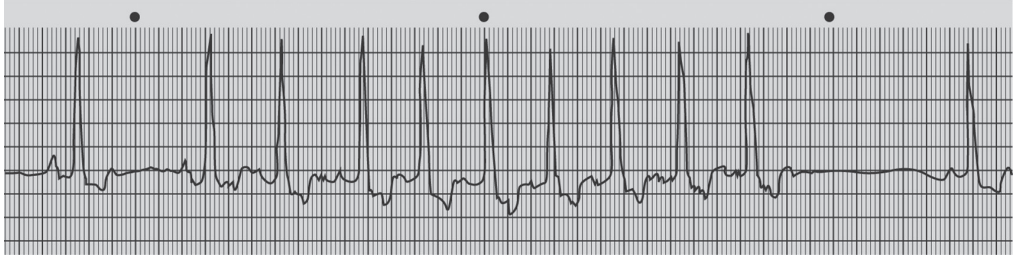


Fig. 102.1 Sinus with an atrial premature complex and paroxysmal supraventricular tachycardia. Abrupt initiation and termination of the tachycardia help distinguish it from sinus tachycardia (lead II, 50 mm/s, 1 cm 5 1 mV). Source: Tilley LP. Essentials of Canine and Feline Electrocardiography, 3rd edn. Baltimore, MD: Williams and Wilkins, 1992, with permission.

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- P waves can be normal or abnormal and typically differ in configuration from the sinus P waves. P waves may be buried in the previous T wave and therefore not visualized.
- Atrioventricular conduction is usually normal (1:1), but various levels of functional second-degree AV block may occur at higher atrial rates (2:1, 3:1, 4:1, etc.).



ETIOLOGY/PATHOPHYSIOLOGY

- SVT may be primary (idiopathic) or secondary to other cardiac disease, generally those creating atrial enlargement.
- May result from a reentrant mechanism or from abnormal automaticity in an ectopic focus.
- Reentrant SVT typically produces a very regular rhythm; SVT due to an automatic focus in atrial myocardium can produce an irregular rhythm.
- Most cases in dogs respond to drugs that specifically alter conduction and refractoriness in the AV nodal tissue, suggesting AV nodal reentry as the mechanism.
- Recent electrophysiologic studies revealed that some SVT in dogs is related to a congenital accessory pathway between the atria and ventricles that allows the electrical impulses to travel freely between the atria and ventricles without traversing the AV node and without conduction delay. In these patients, the SVT is caused by reentry through the accessory pathway and the AV node.

Systems Affected

- Cardiovascular: CHF may develop secondary to progressive myocardial failure associated with a chronically high heart rate (so-called tachycardia-induced myocardial failure).
- Neuromuscular: syncope or generalized episodic weakness due to reduced cardiac output and oxygen delivery.



SIGNALMENT/HISTORY

Species

- Dogs and rarely cats.

Risk Factors/Causes

- Heart disease
- Chronic valvular disease
- Cardiomyopathy
- Congenital heart disease
- Cardiac neoplasia
- Systemic disorders
- Ventricular preexcitation
- Electrolyte imbalances
- Digoxin toxicity
- Idiopathic

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Historical Findings

- Owners are generally unaware of the arrhythmia.
- Coughing or breathing abnormalities in dogs with CHF.
- Episodic weakness or syncope.



CLINICAL FEATURES

- Abnormalities noted on physical examination.

Physical Examination Findings

- Rapid, usually regular heart rhythm. However, in dogs with paroxysmal SVT, the rhythm may be normal and regular during the physical examination.
- May have evidence of poor peripheral perfusion: pale mucous membranes, a prolonged capillary refill time, and weak pulses.
- May have no signs other than the rapid heart rate.
- Findings may reflect an underlying cardiac condition (e.g., heart murmur).



DIFFERENTIAL DIAGNOSIS

- Sinus tachycardia.
- Atrial flutter.
- Atrial fibrillation.
- Ventricular tachycardia (SVT with right bundle branch block or aberrant conduction can look like ventricular tachycardia; resolution of arrhythmia after lidocaine administration usually confirms ventricular tachycardia).



DIAGNOSTICS

Imaging

- Echocardiography (including Doppler studies) may help characterize the type and severity of underlying cardiac disorders. Echocardiography is also important for assessing myocardial function in patients with idiopathic SVT.
- When viewed on an echocardiogram during bursts of SVT, the left ventricle has a normal end-systolic diameter and a small end-diastolic diameter, resulting in a decreased shortening fraction because of inadequate filling.
- Usually left or right atrial enlargement in dogs with SVT secondary to other cardiac disorders.

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Diagnostic Procedures

- Long-term ambulatory (Holter) recording of the ECG may detect paroxysmal SVT in cases of unexplained syncope. This is generally only helpful if the syncope is occurring regularly within a 24–48-hour period. Holter monitors may also help characterize the rate and frequency of sustained SVT and are useful in evaluating the efficacy of therapy.
- Event (loop) recorders may detect paroxysmal SVT in patients with infrequent episodes of syncope (<every 24–48 hours).
- Sustained SVT must be distinguished from sinus tachycardia because the two arrhythmias have different implications and treatment. A precordial thump may help differentiate sinus tachycardia from SVT when the heart rate is in the 150–250 beats per minute range; it will usually stop an SVT for at least one or two beats, while a sinus tachycardia will not slow. A vagal maneuver (e.g., ocular pressure or carotid sinus massage) may break an SVT abruptly but only gradually slows sinus tachycardia.



THERAPEUTICS

Drugs of Choice

Emergency Therapy

- Administer one of the following drugs.
  - Calcium channel blockers: verapamil (0.05 mg/kg boluses IV over 3–5 minutes up to three times) or diltiazem (0.05–0.25 mg/kg IV over 5–15 minutes).
  - Beta-adrenergic blockers: esmolol (0.25–0.5 mg/kg slow IV bolus administration followed by a constant rate infusion of 50–200 µg/kg per minute); moderate-to-severe myocardial failure is a relative contraindication to the administration of these drugs at these doses.
- Electrical cardioversion or intracardiac electrophysiologic pacing methods may be considered in extreme cases.

Long-Term Therapy

- Digoxin: administer at either a maintenance oral dose or double the maintenance dose for the first day to produce a therapeutic serum concentration more rapidly; contraindicated in patients with accessory pathways.
- Beta-adrenergic blocker: atenolol (0.2–1 mg/kg PO every 12–24 hours) can be administered as long as the patient does not have underlying moderate-to-severe myocardial failure.
- Diltiazem is the calcium channel blocker of choice for long-term control of SVT. The dosage required to control SVT has not been reported in the dog. Diltiazem is used more frequently to control the ventricular rate in patients with atrial fibrillation at a dosage of 0.5–1.5 mg/kg PO every 8 hours. Generally start in this dosage range but almost always increase the dose to 2.0–3.0 mg/kg PO every 8 hours to effect control of SVT.

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- Class I antiarrhythmic agents such as quinidine and procainamide can be tried when the afore-mentioned drugs are ineffective or when the SVT is thought to be due to an automatic rather than a reentrant rhythm. SVT caused by an automatic atrial focus may produce an irregular rhythm and may be refractory to conventional drug therapy. When the SVT is due to an accessory pathway, these drugs are more effective.

Alternative Drugs

- Emergency treatment.
  - Intravenous adenosine (1–12 mg IV rapidly). Adenosine is very expensive and short-lived.
  - Propranolol (0.02 mg/kg slow IV boluses up to a total dose of 0.1 mg/kg). Propranolol has a long half-life after IV administration and also has significant beta-2 blocking effects and is generally not recommended unless no other alternative is available.

Precautions/Interactions

- Calcium channel blockers and beta-adrenergic blockers have negative inotropic properties and should be used cautiously in dogs with documented myocardial failure.

Diet

- Mild-to-moderate sodium restriction if in CHF.

Activity

- Restrict until arrhythmia has been controlled.

Surgical Considerations

- Consider transvenous catheter ablation for patients with accessory pathways.

Appropriate Healthcare

- Asymptomatic patients can be managed on an outpatient basis; patients with a sustained SVT or signs of CHF should be hospitalized until stable.
- SVT is a medical emergency in dogs that exhibit weakness and collapse; nonpharmacologic interventions that may break an SVT include vagal maneuvers, precordial thump, and electrical cardioversion.
- Vagal maneuvers are often unsuccessful but may be used initially because of their ease of administration and noninvasive nature.
- Delivering a precordial thump can successfully (>90% of the time) terminate an SVT in dogs, but this maneuver may break the rhythm for only a brief period. At other times, the rhythm remains converted. To perform a precordial thump, the dog is placed on its

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right side and the left apex beat is located. This region is then "thumped" with a fist while recording the ECG.
- Emergency medical therapy is required in patients when a precordial thump is unsuccessful.

Nursing Care

- Treat CHF and correct any underlying electrolyte or acid–base disturbances.



COMMENTS

- Clinical signs may relate to the underlying cause.
- Dogs with slow SVT or infrequent paroxysmal SVT may exhibit no clinical signs.
- Dogs with fast SVT (heart rate usually >300 beats per minute) generally exhibit episodic weakness or syncope.

Client Education

- Owners should observe patients closely for signs of low cardiac output such as weakness and collapse.

Patient Monitoring

- Serial ECG or Holter monitoring.

Possible Complications

- Syncope and CHF.

Expected Course and Prognosis

- Most cases are controlled effectively with medication.

Synonyms

- Atrial tachycardia, junctional tachycardia

Abbreviations

AV: atrioventricular
CHF: congestive heart failure
ECG: electrocardiogram
IV: intravenous
PO: by mouth (per os)
SVT: supraventricular tachycardia

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See Also

Atrial Fibrillation and Atrial Flutter

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Author: Larry P. Tilley, DVM

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chapter 103

Syncope

DEFINITION/OVERVIEW

- Temporary loss of consciousness and vascular tone associated with loss of postural tone, with spontaneous recovery.

ETIOLOGY/PATHOPHYSIOLOGY

- Inadequate cerebral perfusion, delivery of oxygen, and metabolic substrates lead to loss of consciousness and motor tone.
- Impaired cerebral perfusion can result from changes in vasomotor tone, cerebral disease, and low cardiac output caused by structural heart disease or arrhythmias.

Risk Factors/Causes

- Heart disease
- Sick sinus syndrome
- Drug therapy: vasodilators (e.g., calcium channel blockers, ACE inhibitors, hydralazine, and nitrates), phenothiazines (e.g., acepromazine), antiarrhythmics, and diuretics

Cardiac Causes

- Bradyarrhythmias: sinus bradycardia, sinus arrest, second-degree AV block, complete AV block, atrial standstill
- Tachyarrhythmias: ventricular tachycardia, supraventricular tachycardia, and atrial fibrillation
- Low cardiac output (nonarrhythmic): cardiomyopathy, AV valve endocardiosis, subaortic stenosis, pulmonic stenosis, heartworm disease, pulmonary embolism, cardiac tumor, or cardiac tamponade

Neurologic and Vasomotor Instability

- Vasovagal syncope: emotional stress and excitement may cause heightened sympathetic stimulation, leading to transient tachycardia and hypertension, which is followed by a

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compensatory rise in vagal tone, leading to excessive vasodilation without a compensatory rise in heart rate and cardiac output; bradycardia often occurs.
- Situational syncope refers to syncope associated with coughing, defecation, urination, and swallowing.
- Carotid sinus hyperactivity may cause hypotension and bradycardia, often the cause of syncope when one pulls on a dog's collar.

Miscellaneous Causes

- Drugs that affect blood pressure and regulation of autonomic tone.
- Hypoglycemia, hypocalcemia, and hyponatremia (rare).
- Hyperviscosity syndromes (e.g., polycythemia and paraproteinemia) cause sludging of blood and impaired cerebral perfusion (rare).

Systems Affected

- Cardiovascular
- Nervous



SIGNALMENT/HISTORY

Species

- Dogs and cats.

Breed Predilection

- Sick sinus syndrome: cocker spaniel, miniature schnauzer, pug, and dachshund
- Ventricular dysrhythmias: boxer and German shepherd

Mean Age and Range

- More common in old animals.

Historical Findings

- Acute collapse or weakness
- Suspected seizure



DIFFERENTIAL DIAGNOSIS

Differential Signs

- Must differentiate from other altered states of consciousness, including seizures and narcolepsy (a sleep disorder).

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- Seizures are often associated with a prodromal and postictal period; syncope occurs without warning, and the animal usually has rapid, spontaneous recovery. Unlike syncope, seizure activity is usually associated with tonic-clonic muscle activity rather than flaccidity.
- Like syncope, narcolepsy occurs suddenly, results in muscle flaccidity, and resolves spontaneously. Unlike syncope, narcolepsy can last for minutes and can be terminated by loud noises or harsh external stimuli.
- Must differentiate from other causes of collapse such as musculoskeletal disease and neuromuscular disease (e.g., myasthenia gravis), which are not associated with loss of consciousness.

Differential Causes

- Syncope with excitement or stress suggests vasovagal syncope.
- Syncope with coughing, urination, or defecation suggests situational syncope.
- Syncope with exercise suggests low output states associated with dysrhythmias or structural heart disease.
- A murmur supports heart disease but does not confirm cardiac cause for syncope.
- Healthy dogs may exhibit sinus bradycardia (rate as low as 30 beats/minute) and sinus pauses (as long as 3.5 seconds) normally during sleep.
- Bradycardia and sinus arrest due to normal or enhanced vagal tone.
- Drug-induced (digitalis, alpha-adrenergic antagonists, alpha-2-adrenergic agonists, calcium channel antagonists, cimetidine, opioids).
- Atrial standstill secondary to hyperkalemia or atrial disease.



DIAGNOSTICS

Complete Blood Count/Biochemistry/Urinalysis

- Usually normal
- Hypoglycemia or electrolyte disturbance in some animals

Other Laboratory Tests

- If animal is hypoglycemic, measure insulin concentration on same blood sample.
- Calculate an amended insulin-to-glucose ratio to rule out insulinoma.
- If animal is hyponatremic or hyperkalemic, consider an ACTH stimulation test.
- If low cardiac output is suspected, rule out occult heartworm disease.

Echocardiography

- May detect structural heart disease that could lower cardiac output.

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Diagnostic Procedures

- Have owner monitor heart rate during any syncopal episode.
- Electroencephalogram, computed tomography of the head, CSF tap if CNS origin suspected.
- Atropine response testing: indicated in dogs with sinus bradycardia, sinus arrest, and sinoatrial exit block. Administer atropine (0.04 mg/kg IM), and evaluate the ECG 20–30 minutes later. A normal (positive) response is >50% increase in heart rate with abolition of pauses; dogs with SSS generally have no response or an incomplete response to atropine.
- Electrophysiologic testing of sinus node recovery time and sinoatrial conduction time.
- 24-hour ambulatory ECG (Holter) or event recording to correlate clinical signs with arrhythmia.

Electrocardiographic Findings

- Postexercise ECG may reveal intermittent dysrhythmia.
- Holter monitoring (24-hour ECG recording) or ECG event (loop) recorder is useful for evaluating dysrhythmic causes.
- Carotid sinus massage with ECG and blood pressure monitoring useful in evaluating carotid sensitivity.



THERAPEUTICS

Drugs of Choice

Bradyarrhythmias and Tachyarrhythmias

- Symptomatic dogs are grouped into those showing primarily bradycardia, sinus arrest, and/or sinoatrial exit block and those with supraventricular tachycardia followed by sinus arrest.
- Atropine-responsive symptomatic dogs with bradycardia or sinus arrest: anticholinergic agents (propantheline: small dogs, 3.75–7.5 mg PO q8–12h; medium dogs, 15 mg PO q8h; large dogs, 30 mg PO q8h; hyoscyamine: 0.003–0.006 mg/kg q8h).
- Dogs with bradycardia and sinus arrest: may try theophylline (Theo-Dur, 20 mg/kg PO q12h), terbutaline (0.2 mg/kg PO q8–12h), or hydralazine (1–2 mg/kg PO q8–12h) if anticholinergic drugs are ineffective (avoid hydralazine if patient is hypotensive).
- Dogs with bradycardia-tachycardia whose clinical signs are due to tachycardia or tachycardia-induced sinus arrest: can give digoxin (0.005 mg/kg PO q12h) or atenolol (0.5–1 mg/kg PO q12–24h) in attempt to suppress the SVT (monitor closely for exacerbation of bradycardia).

Low Cardiac Output

- Institute treatment to improve cardiac output, which varies according to specific cardiac disease.

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Precautions/Interactions

- Drugs that lower blood pressure.
- Attempts to manage bradycardia-tachycardia syndrome medically without prior pacemaker implantation carry significant risk because drugs used to control SVT may worsen the bradyarrhythmias, and vice versa.
- Adverse effects of anticholinergic medication (constipation, difficulty voiding, keratoconjunctivitis sicca, emesis, anxiety) occur commonly.
- Avoid drugs that may worsen sinus node dysfunction (e.g., beta-adrenergic antagonists, calcium channel blocking agents, phenothiazines, class I and III antiarrhythmic agents, opioids, cimetidine, alpha-2-adrenergic agonists).

Surgical Considerations

- Pacemaker implantation for sick sinus syndrome, advanced AV block, and persistent atrial standstill.
- Permanent artificial pacemaker necessary for dogs failing to respond to medical treatment and those exhibiting unacceptable medication side effects.
- Permanent artificial pacemaker usually required for dogs with bradycardia-tachycardia syndrome (sick sinus syndrome).
- Therapy for tachycardias can only be considered once pacing is established to avoid worsening of bradyarrhythmias.

Appropriate Healthcare

- Avoid or discontinue medications likely to precipitate syncope.
- Treat as outpatient unless important heart disease is evident.

COMMENTS

Client Education

- Minimize stimuli that precipitate episodes.
- Low cardiac output: minimize activity.
- Vasovagal: minimize excitement and stress.
- Cough: remove collar.

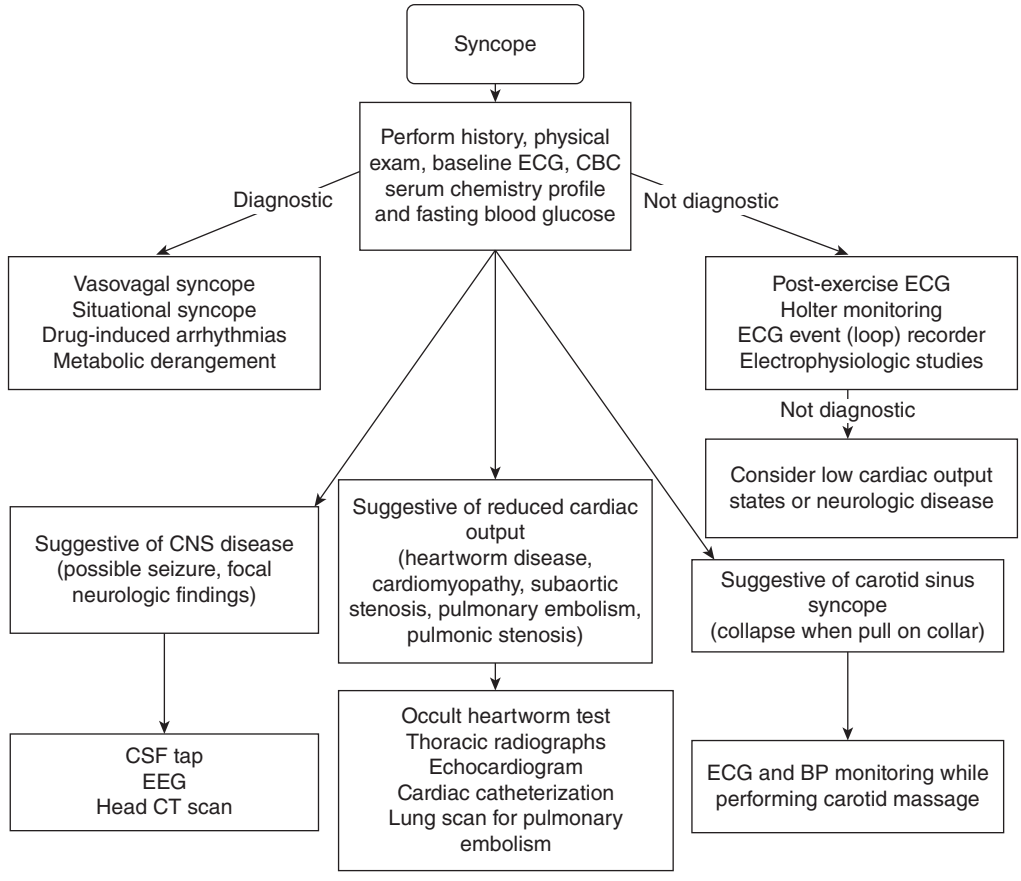
Patient Monitoring

- ECG or Holter monitoring to assess efficacy of antiarrhythmic therapy (Fig. 103.1).

Prevention/Avoidance

- See Client Education.
- Perform history, physical examination, baseline ECG, CBC serum chemistry, and profile.

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■ **Fig. 103.1.** Algorithm for syncope. BP, blood pressure; CBC, complete blood count; CNS, central nervous system; CSF, cerebrospinal fluid; CT, computed tomography; ECG, electrocardiogram; EEG, electroencephalogram.

Possible Complications

- Death
- Trauma when collapse occurs

Expected Course and Prognosis

- Most noncardiac causes are not life-threatening; cardiac causes may be treated, but syncope in patients with cardiac disease may suggest higher mortality risk.

Synonyms

- Fainting

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Abbreviations

ACE: angiotensin-converting enzyme
ACTH: adrenocorticotropic hormone
AV: atrioventricular
CBC: complete blood count
CNS: central nervous system
CSF: cerebrospinal fluid
ECG: electrocardiogram

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Author: Francis W. K. Smith, Jr, DVM

chapter 104

Tick Paralysis

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DEFINITION/OVERVIEW

- Flaccid, lower motor neuron paralysis caused by salivary neurotoxins from the female of certain species of ticks.

ETIOLOGY/PATHOPHYSIOLOGY

- The female tick of multiple species (*Dermacentor* and *Amblyomma* in the United States and *Ixodes* in Australia) injects salivary neurotoxins that interfere with the depolarization/acetylcholine release mechanism in the presynaptic nerve terminal.
- The Australian *Ixodes* tick neurotoxin depends strongly on temperature for activation and effect.
- Only one adult female *Dermacentor* or *Amblyomma* tick is necessary to cause neurologic signs, but a large larval or nymphal *Ixodes* tick infestation can also induce signs.
- Signs occur 6–9 days after initial tick attachment.
- Not all infested animals develop signs of tick paralysis and not all adult female ticks produce the neurotoxin.

Systems Affected

- Nervous/neuromuscular: peripheral nervous system and the neuromuscular junction most affected by the neurotoxin; cranial nerves can become involved, including the vagus, facial, and trigeminal nerves; sympathetic nervous system can also be affected.
- Respiratory: may see paralysis of the intercostal muscles and diaphragm; caudal brainstem respiratory centers may be affected especially with *Ixodes* tick infestation.

SIGNALMENT/HISTORY

- In Australia, both dogs and cats are equally affected by the *Ixodes* neurotoxin.
- In North America, however, cats appear to be resistant to the *Dermacentor* and *Amblyomma* neurotoxin.

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Risk Factors/Causes

- The incidence of tick paralysis is somewhat seasonal, with a greater incidence in the summer months.
- In warmer areas (southern United States and northern Australia), tick paralysis is a year-round problem.
- The overall incidence of tick paralysis is relatively low in North America, with a higher incidence of tick paralysis in Australia.
- In the United States, *D. variabilis* has a wide distribution over the eastern two-thirds of the country and in California and Oregon.
- *D. andersoni*, however, is found only from the Cascades to the Rocky Mountains.
- *americanum* has a southern US distribution from Texas and Missouri to the Atlantic Coast.
- *maculatum* has even a narrower range, preferring the high temperature and humidity of the Atlantic and Gulf of Mexico seaboards.
- In Australia, *I. holocyclus* is limited to the coastal areas of the east, especially in areas associated with bush and scrub.

Historical Findings

- The classic history would be that the patient walked in a wooded area approximately 1 week prior to the onset of signs.
- Onset of neurological dysfunction is gradual, starting with stumbling and weakness in the pelvic limbs, followed by a rapid progression to tetraparesis with possible cranial nerve and autonomic signs.

Neurologic Examination Findings

- In non-*Ixodes* ticks, once neurologic signs appear, there is rapid ascending lower motor neuron paresis to paralysis.
- Many dogs will become recumbent in 1–3 days, with hyporeflexia/areflexia and hypotonia/atonia.
- Occasionally, signs are much milder, with the dog still able to ambulate.
- Pain sensation is preserved.
- Cranial nerve dysfunction is usually not a prominent feature, with the most common signs being facial weakness and dysphonia.
- Respiratory paralysis is very rare in the United States.
- Urination and defecation are usually normal.
- With intoxication by the *Ixodes* tick, the neurologic signs are much more severe and rapidly progressive.
- The ascending motor paresis can progress to paralysis within a few hours, along with sialosis, megaesophagus, and vomiting or regurgitation.
- There is sympathetic nervous system dysfunction, manifesting as mydriatic, poorly responsive pupils, hypertension, tachyarrhythmias, high pulmonary capillary hydrostatic pressure, and pulmonary edema.

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- There is additional involvement of the caudal medullary respiratory centers, which add to the peripheral pulmonary changes, resulting in a progressive fall in respiratory rate without a change in tidal volume, leading to hypoxia, hypercapnia, and respiratory acidosis.
- Respiratory muscle paralysis is much more prevalent, with both dogs and cats progressing to respiratory distress, cyanosis, and respiratory paralysis within 1–2 days if not treated.



DIFFERENTIAL DIAGNOSIS

- Botulism
- Acute polyneuropathy
- Coonhound paralysis (acute canine polyradiculoneuritis)
- Distal denervating disease
- Generalized (diffuse) or multifocal myelopathy



DIAGNOSTICS

- Regular blood work will be normal in animals with tick paralysis.
- In severely affected patients, especially with the *Ixodes* tick, arterial blood gases will reveal a low PaO_2, a high $PaCO_2$, and a low pH.
- Thoracic radiographs will be normal in dogs affected by ticks in North America, but often will reveal a megaesophagus in patients affected by the *Ixodes* neurotoxin.
- Electrodiagnostic evaluation will reveal normal insertion activity and an absence of spontaneous myofiber activity (no fibrillations and positive sharp waves) on electromyography (normal), but motor nerve conduction studies will reveal a significant decrease or absence of compound muscle potential amplitudes.



THERAPEUTICS

- If tick paralysis is suspected, the patient should be hospitalized until either a tick is found and removed or appropriate treatment to kill a hidden tick is performed.
- Thoroughly search for a tick in all body areas including the trunk and limbs, ear canals, mouth, rectum, vagina, prepuce, and in between the digits and foot pads.
- If a tick is found, it should be immediately removed.

Drugs of Choice

- If the tick cannot be found, use a topical insecticidal product such as Frontline or dip the patient in an insecticidal bath.
- With tick paralysis in North America, this is often the only specific treatment needed.
- With *I. holocyclus*, circulating toxin must be neutralized via hyperimmune serum (0.5–1 mg/kg IV), depending on the severity of clinical signs.

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- If the clinical signs are severe and involve the sympathetic nervous system, phenoxybenzamine, an alpha-adrenergic antagonist (1 mg/kg IV diluted in saline and given slowly over 20 minutes) appears to be beneficial.
- Acepromazine (0.5–1 mg/kg IV) can be used as an alternative treatment because it has an alpha-adrenergic blocking effect.
- Antiemetics (dolasetron 0.6 mg/kg IV q24h or maropitant 1 mg/kg IV q24h) may be used in vomiting patients.

Precautions/Interactions

- Inpatient supportive care is essential until the patient begins to show signs of recovery.
- If the patient demonstrates hypoxia or hypoventilation, an oxygen cage should be used.
- Assisted ventilation may occasionally be necessary in severe cases, especially with *Ixodes* neurotoxicity.
- Intravenous fluid therapy is generally not required unless recovery is prolonged, the neurological dysfunction is severe, or there is evidence of megaesophagus.
- Administer intravenous fluids at a very slow rate to avoid further complications of pulmonary congestion in *Ixodes* neurotoxicity.
- Keep the patient in a quiet environment until recovery.
- The patient with *Ixodes* neurotoxicity should be housed in a cool, air-conditioned area because the neurotoxin is temperature sensitive. Avoid activity to prevent any increase in body temperature.
- Drugs that interfere with neuromuscular transmission are contraindicated. These include tetracycline, aminoglycosides, and procaine penicillin.
- Atropine is contraindicated in the advanced stages of *Ixodes* neurointoxication or with marked bradycardia.

Diet

- Withhold food and water if the patient has dysphagia or vomiting/regurgitation.
- Parenteral nutrition may be necessary if anorexia, dysphagia, vomiting or regurgitation is prolonged.
- Enteral nutrition using a nasojejunal feeding tube may be necessary.



COMMENTS

Client Education

- For non-*Ixodes* ticks, inform the client that good nursing care is essential, although the patient's recovery is rapid after removal of the tick.
- For the *Ixodes* tick, warn the client that signs often worsen despite tick removal. Therefore, more aggressive treatment to neutralize the toxin must be undertaken.

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Patient Monitoring

- For non-*Ixodes* ticks, reassess neurologic status after tick removal at least on a daily basis. Rapid improvement in muscle strength should occur within 24–48 hours.
- For the *Ixodes* tick, monitor neurologic status and respiratory/cardiovascular functions continuously and intensively even after tick removal because of the residual effect of the neurotoxin.

Prevention/Avoidance

- Vigilantly check for ticks after exposure (at least every 2–3 days). Remember that signs do not occur for 4–6 days after tick attachment.
- Advise the owner to regularly use a topical insecticide or give weekly insecticidal baths.
- Short-term acquired immunity develops after exposure to the *Ixodes* neurotoxin.

Expected Course and Prognosis

- For non-*Ixodes* ticks, prognosis is good to excellent if ticks are removed appropriately, with recovery occurring in 1–3 days.
- Prognosis is often guarded for the *Ixodes* tick, with a prolonged recovery. If left untreated, death will usually occur in 1–2 days.

Abbreviations

IV: intravenous
PaO_2: partial pressure of oxygen in blood
$PaCO_2$: partial pressure of carbon dioxide in blood
pH: acid–base

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chapter 105

Tracheal Collapse





DEFINITION/OVERVIEW

- Tracheal collapse generally refers to a condition of excessive collapsibility of the trachea.
- A collapsing trachea has a wide, flaccid dorsal tracheal membrane that is redundant and sags into the lumen; further, weakened cartilaginous rings may form a shallow arch impinging on the lumen of the trachea.



ETIOLOGY/PATHOPHYSIOLOGY

- Tracheal collapse has been associated with other conditions such as tracheal trauma, intra- and extraluminal masses, and tracheal hypoplasia.
- The typical syndrome of tracheal collapse in which the trachea is excessively collapsible has an unclear etiology. Several theories have been proposed including genetic, nutritional, neurological, and inflammatory causes.

Systems Affected

- Respiratory: severe tracheal collapse can lead to respiratory distress and hypoxemia. Concurrent bronchitis or pneumonia may be present.
- Cardiovascular: chronic cough and airway disease can be an underlying cause of pulmonary hypertension and right-sided heart failure.



SIGNALMENT/HISTORY

- Toy and miniature breeds are most commonly affected. Frequently affected breeds include toy poodle, Yorkshire terrier, Pomeranian, Maltese, pug, and Chihuahua.
- Tracheal collapse is occasionally reported in large breeds, and rarely reported in cats.
- Most dogs with tracheal collapse are middle-aged, and the reported average age of onset is 6.6–8 years. There is a biphasic age distribution with a subpopulation showing signs of tracheal collapse during puppyhood.

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Historical Findings

- Cough is the classic historical finding, and it may have been present for several weeks prior to the client seeking veterinary attention. The cough is often dry, hacking, and non-productive; a terminal retch or a moist cough may be present. The cough is most often described as a "goose-honking" cough. Initially the cough may have been mild but often progresses and there are bouts of severe, paroxysmal coughing episodes.
- Coughing bouts may be precipitated by excitement or tracheal irritation such as pulling on a neck leash. Severe cases can suffer respiratory distress at rest, cyanosis, and collapse. Exercise intolerance may be reported, although this may not be reported in dogs with a sedentary lifestyle.
- Up to two-thirds of patients are reported as obese.
- Gagging after eating or drinking is reported in 30% of dogs with tracheal collapse.



CLINICAL FEATURES

- Physical examination findings will vary from an apparently normal dog to findings of severe, paroxysmal cough with possible cyanosis or collapse.
- A harsh cough can be easily induced with gentle tracheal palpation. Thoracic auscultation will reveal harsh wheezing sounds in patients with concurrent chronic bronchitis.
- Cardiac auscultation is typically normal, although a split S2 from delayed closure of the tricuspid valve may be present in patients with pulmonary hypertension.
- Incidental chronic mitral valve disease and a loud systolic mitral murmur may be present, falsely raising the suspicion of congestive heart failure.



DIFFERENTIAL DIAGNOSIS

- Chronic bronchitis
- Infectious tracheobronchitis
- Tracheobronchial foreign body
- Brachycephalic airway syndrome
- Congestive heart failure
- Pneumonia



DIAGNOSTICS

Radiography

- Lateral radiographs of the cervical and thoracic regions are commonly obtained in diagnosing tracheal collapse; dorsoventral flattening of the trachea may be observed (Fig. 105.1).

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Fig. 105.1. Lateral thoracic radiograph of a dog with tracheal collapse at the thoracic inlet. Also present is a redundant dorsal trachealis membrane.

- Reported sensitivity for routine lateral radiographs is 60–84%; false-negative findings on routine films frequently occur.
- False positives may also occur with superimposition of overlying structures such as the esophagus or longus colli or longus capitis muscles.
- Because of the dynamic nature of tracheal collapse, both inspiratory and expiratory views should be obtained as intrathoracic tracheal collapse will be evident on expiration and cervical tracheal collapse will be evident on inspiration.
- Alternatively, fluoroscopy has the advantage of continuous documentation through all phases of respiration and is preferred by many clinicians.
- Routine thoracic and cervical radiographs are indicated in patients with cough or respiratory distress; patients should be medically stabilized prior to obtaining stressful diagnostic images.

Tracheobronchscopy

- Advances in fiberoptic and videobronchoscopy have increased the availability of this technique. Direct tracheal imaging allows visualization of dynamic tracheal lesions, intraluminal masses, and opportunity to grade the extent of tracheal collapse (i.e., cervical, thoracic, both), and exclude false-positive radiographic findings.
- Tracheal collapse grading system.
  - Grade I: slightly pendulous trachealis muscle with up to 25% luminal loss.
  - Grade II: the trachealis muscle is more pendulous and the lumen is reduced by up to 50%.
  - Grade III: severe flattening of the cartilaginous rings; the dorsal tracheal membrane is nearly in contact with the opposing wall and there is up to 75% luminal loss.

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  - Grade IV: total tracheal collapse; the dorsal tracheal membrane lies on the ventral floor, the lumen is obliterated.
- Tracheoscopy is typically performed with concurrent pharyngoscopy, laryngoscopy, and bronchoscopy as other forms of airway disease may exist concurrently with tracheal collapse.

Cytology and Bacteriology

- Obtaining airway wash samples for cytology and bacteriology is often advocated; however, 36% of clinically normal dogs have positive bacterial cultures from lower tracheal swabs. Common bacteria isolated include alpha-hemolytic streptococci, *Pasteurella multocida*, *Klebsiella pneumoniae*, and coagulase-positive staphylococci. Upper airway culture should be carefully interpreted with cytology to avoid inappropriate antimicrobial use.



THERAPEUTICS

- The objectives of therapy include resolving respiratory distress, resolution of severe cough, and return to adequate function.

Drugs of Choice

- Supplemental oxygen should be provided to patients in distress.
- Antitussives help break the cycle of cough-induced airway irritation that leads to more cough.
  - Butorphanol tartrate: 0.2–0.4 mg/kg SQ, IM, or IV every 4–6 hours.
  - Hydrocodone: 0.22 mg/kg PO every 6–12 hours.
  - Diphenoxylate: 0.2–0.5 mg/kg PO every 8–12 hours.
- Sedation of hyperactive animals can help break the cough-induced cycle.
  - Butorphanol has sedative properties.
  - Acepromazine: 0.05–0.1 mg/kg IM or IV every 8–12 hours.
  - Diazepam: 0.1–0.6 mg/kg IV.
  - Trazadone: 2–5 mg/kg PO every 8–12 hours.
- Bronchodilators may be helpful in cases where concurrent small airway disease is present.
  - Terbutaline: 0.01 mg/kg IM, or 0.625–1.25 mg/dog PO every 12 hours.
  - Theophylline: 10 mg/kg PO every 12 hours.
- Glucocorticoid treatment is controversial and may be advantageous in patients with concurrent small airway disease.
  - Antiinflammatory prednisone: 0.25–0.5 mg/kg every 12 hours.
- Antibiotic use should be restricted to cases with documented concurrent infection.

Diet

- Obese animals should be placed on a strict diet and a strict follow-up plan to assess weight loss.

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Activity

- Excessive activity and excitement can precipitate a paroxysmal coughing bout and routine tranquilization may be needed, especially during times of anticipated increased pet activity.

Surgical Considerations

- Surgical options commonly utilized include extraluminal ring prostheses and intraluminal stent placement.
- Procedures are intricate and should be performed by experienced specialists.
- Because the procedures carry significant morbidity and mortality, many authors reserve them for those patients which have failed medical management.

COMMENTS

Client Education

- The majority of patients can be managed medically.

Patient Monitoring

- Cough frequency will be the mainstay of patient monitoring.
- Patient return to activity.

Prevention/Avoidance

- Avoid neck leashes and other cervical irritants.
- Strive for ideal body condition score.

Possible Complications

- Complications of medical management are minimal. Prolonged prednisone usage may contribute to weight gain due to polyphagia.
- Complications of extraluminal tracheal prostheses include laryngeal paralysis, necrotizing tracheitis from blood flow disruption, and migration.
- Complications from intraluminal tracheal stent placement include stent migration, stent fracture, pneumonia, and tracheal granulation tissue.

Expected Course and Prognosis

- For severe cases the prognosis is fair to guarded.

Synonyms

- Collapsing trachea
- Collapsed trachea

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Abbreviations

IM: intramuscular
IV: intravenous
PO: by mouth (per os)
SQ: subcutaneous

Suggested Reading

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Author: Jonathan F. Bach, DVM

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chapter 106

Traumatic Myocarditis



DEFINITION/OVERVIEW

- Traumatic myocarditis is a term used by veterinarians to describe a presumed myocardial injury secondary to blunt nonpenetrating thoracic trauma that most commonly results in ventricular dysrhythmias in the dog.



ETIOLOGY/PATHOPHYSIOLOGY

- The most common mechanism of myocardial contusion in the dog is secondary to lateral chest compression that subjects the myocardium to compressive and concussive forces. Additionally, it has been proposed that distortion of the thoracic cage results in a rise in intrathoracic and intracardiac pressures, causing shearing stresses within the myocardium powerful enough to result in myocardial injury. Conditions other than direct injury to the heart, such as metabolic acidosis, hypoxia, electrolyte imbalance, intracranial injuries, and catecholamine release, may also cause dysrhythmias in traumatized dogs.

Systems Affected

- Cardiovascular: dysrhythmias, hypotension, weak pulses, or tachycardia.
- Musculoskeletal: weakness, rib fractures, or trauma severe enough to result in limb and pelvic fractures frequently causes myocardial contusion.
- Nervous: decreased responsiveness, disorientation, or weakness.
- Renal/urologic: decreased urine output.
- Respiratory: hypoxia or pulmonary contusion.
- Skin: bruising over the thoracic cage could be seen.



SIGNALMENT/HISTORY

Risk Factors/Causes

- There are no known medical conditions, medications, or environmental factors that predispose the dog or cat to developing this condition.

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Historical Findings

- Trauma is the only presenting complaint that is associated with traumatic myocarditis. Most commonly, automobile-induced injury is associated with this condition but other injuries such as animal attacks (i.e., bite, kick) and falls from height can also cause myocardial injuries.



CLINICAL FEATURES

Dogs

- The diagnostic gold standard in the identification of cardiac injury remains gross or histologic examination of the heart. Because visualization or myocardial biopsy is uncommon, a high index of suspicion for and understanding of myocardial injury are essential in making a diagnosis. Traumatic myocarditis should be suspected in traumatized dogs with the following injuries: (1) fractures of extremities, spine or pelvis, (2) external evidence of thoracic trauma, (3) radiographic evidence of chest trauma such as pulmonary contusions, pneumothorax, hemothorax, diaphragmatic hernia, and rib/scapular fractures, and (4) neurologic injury. Cardiac dysrhythmias are frequently delayed in onset up to 48 hours after the inciting event.

Cats

- This condition has not traditionally been reported in cats but remains possible, and the same features reported in dogs could easily be applied to cats once underlying cardiac disease is ruled out.



DIFFERENTIAL DIAGNOSIS

Dogs

- Dysrhythmias associated with trauma may also be caused by:
  - Underlying heart disease
  - Metabolic acidosis
  - Hypoxia
  - Electrolyte imbalance
  - Intracranial injuries
  - Pain (catecholamine release).



DIAGNOSTICS

- Blood work.
  - CBC, biochemistry panel, urinalysis, and arterial or venous blood gas.

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- Radiographs.
  - Thoracic – pulmonary contusions, pneumothorax, hemothorax, or rib fracture.
  - Abdominal– evidence of effusion (i.e., blood, urine), free abdominal gas.
  - Appendicular – fractures.
- Echocardiogram. Abnormal findings may include:
  - Increased end-diastolic wall thickness
  - Impaired contractility, indicated by wall motion abnormalities and decreased fractional shortening
  - Increased echogenicity
  - Localized areas of echolucency consistent with intramural hematomas.
- ECG: ventricular dysrhythmias most common, often delayed in onset up to 48 hours.
  - Intermittent
  - Continuous
  - Holter monitoring
- Serum myocardial isoenzyme/proteins.
- CPK-MB.
- Cardiac troponins (T and I).

Pathologic Findings

- Pathologic findings in the traumatized heart have been characterized by localized edema, ecchymosis, and intramyocardial hematoma formation. These injuries are often transmural, with the epicardial surface being more severely affected.
- Dysrhythmias and conduction defects are the most common physiologic consequences of myocardial injuries.
- The most common dysrhythmias encountered secondary to canine myocardial injuries include premature ventricular contractions, ventricular tachycardia, and nonspecific ST segment elevation or depression.
- Although less frequent than ventricular dysrhythmias, other dysrhythmias reported in dogs with chest trauma include atrial fibrillation, sinus arrest with ventricular or junctional escape complexes, and second- and third-degree AV block.



THERAPEUTICS

- Treatment is typically aimed at suppressing potentially life-threatening dysrhythmias such as multiform premature ventricular complexes, ventricular tachycardia, and the R-on-T phenomenon. Dysrhythmias that result in hypotension, weakness, pale mucous membranes, delayed capillary refill time, collapse, or syncope in patients that have received adequate fluids, electrolytes, oxygen, and pain control should also be addressed. The therapeutic goal of these recommendations is not necessarily total alleviation of the dysrhythmia; adequate therapy may be reduction of the heart rate (<140 beats per minute) or the return of hemodynamic stability.

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Drugs of Choice

- Lidocaine: 2 mg/kg intravenous bolus. Lidocaine boluses may be repeated as needed every 10–20 minutes until a cumulative dose of 8 mg/kg is given. If the therapeutic response is favorable to intravenous bolus therapy, an intravenous CRI of 40–80 µg/kg per minute should be initiated to maintain a normal rhythm. In many cases, additional boluses may be required to suppress the arrhythmia while steady-state blood levels of lidocaine are achieved by the CRI.
- When lidocaine fails to resolve ventricular ectopy, procainamide may be administered IV or IM (6–15 mg/kg every 4–6 hours). Procainamide may also be administered as a CRI (10–40 µg/kg per minute) or given orally (sustained-release formulation 20 mg/kg three times daily). Good success is noted in transitioning to mexiletine (4–8 mg/kg PO three times daily) as an oral antiarrhythmic for animals being discharged that require continued suppression of arrhythmias.
- The addition of a beta-blocker should be considered for the treatment of ventricular ectopy that remains unresponsive to class I antiarrhythmics in traumatized dogs. Use of beta-blockers should be reserved for patients that have been appropriately treated for shock and are not receiving positive inotropic medications.
- In some instances, ventricular ectopy will not be suppressed with antiarrhythmic drugs alone. Careful monitoring of the patient's electrolyte and acid–base status is necessary because acidosis, hypokalemia, and hypomagnesemia can be present and promote continued ventricular tachycardia. Judicious use of potassium supplementation (dose according to degree of hypokalemia) and magnesium chloride (0.75 mEq/kg/day IV CRI) should be considered when acid–base and electrolyte derangements are also present.

Precautions/Interactions

- High doses of lidocaine can result in seizures; cats are very sensitive to the adverse side effects of lidocaine.
- Hypotension and AV conduction block are serious potential side effects of procainamide administration.
- Beta-blockers can result in serious potential side effects such as AV block, hypotension, brochoconstriction, and decreased cardiac contractility.
- Oral antiarrhythmics can cause gastrointestinal upset such as anorexia and vomiting.
- Avoid administration of more than 0.5 mEq/kg per hour potassium supplementation.
- Oversupplementation with magnesium chloride can cause muscle fasciculation, tremors, and AV block.

Activity

- Activity restriction will depend on primary injuries, but the author recommends a minimum of 2 weeks of exercise restriction to allow cardiopulmonary injuries to resolve.

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Surgical Considerations

- If a patient with a myocardial injury must undergo anesthesia, drugs should be selected that are least likely to induce arrhythmias, such as acepromazine, butorphanol, isoflurane, and glycopyrrolate. Halothane, atropine sulfate, and the thiobarbiturates would be poor choices as they tend to exacerbate dysrhythmias and sensitize the heart to catecholamine-induced dysrhythmias.



COMMENTS

- Onset of dysrhythmias can be delayed for up to 24 hours. Treatment is only required in cases with potentially fatal arrhythmias (VT, R-on-T) and animals with clinical evidence of hypotension, weak, poor pulses or pulse deficits, or decreased cardiac output associated with a dysrhythmia. Dysrhythmias associated with trauma should only be addressed after the other physiologic consequences associated with sustained injuries are appropriately treated (i.e., fluids, oxygen, pain). Most dysrhythmias associated with myocardial trauma do not require treatment and resolve in 3–10 days.

Client Education

- Monitor for weakness, respiratory distress, pale mucous membranes, and irregular pulses.

Patient Monitoring

- It is recommended that intermittent ECG monitoring continue up to 1 week after discharge. Prior to reexamination, antiarrhythmic medications should be discontinued for a minimum of 24 hours. Holter monitoring, if available, would be the most sensitive way to detect complete resolution of arrhythmias after discontinuing antiarrhythmic medications.

Possible Complications

- Some dysrhythmias could be fatal if not suppressed. Myocardial dysfunction secondary to trauma could result in congestive failure. Persistent dysrhythmias and myocardial dysfunction could occur and require lifelong therapy.

Expected Course and Prognosis

- Prognosis is very good; most dysrhythmias resolve within 1–2 weeks, complications are rare.

Synonyms

- Blunt myocardial injury, myocardial cell injury, myocardial contusion, cardiac contusion

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Abbreviations

AV: atrioventricular
CPK-MB: creatine phosphokinase myocardial band
CRI: constant rate infusion
ECG: electrocardiogram
IM: intramuscular
IV: intravenous
PO: by mouth (per os)
VT: ventricular tachycardia

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chapter 107

Urethral Obstruction – Canine



DEFINITION/OVERVIEW

- Urethral obstruction refers to the restricted flow of urine from the urinary bladder to the external urethral orifice.



ETIOLOGY/PATHOPHYSIOLOGY

- Urethral obstruction is caused by any process which creates resistance to the flow of urine within the urethra. Dependent upon the duration and degree of the obstruction, metabolic abnormalities (hyperkalemia, azotemia, acidemia) may develop. Urethral obstruction can be mechanical or functional in origin.

Systems Affected

- Cardiovascular: bradyarrhythmias may develop in patients with hyperkalemia.
- Respiratory: patients with severe uremia may develop uremic pneumonitis.
- Gastrointestinal: uremia can result in gastrointestinal ulceration and vomiting.
- Renal/urologic: urethral obstruction can cause secondary renal injury when the pressure within the urinary bladder is high enough to prevent the flow of urine from the ureters into the bladder.



SIGNALMENT/HISTORY

- Urethral obstruction is more common in male than in female dogs. Breed predispositions exist for the individual causes of urethral obstruction, but not for urethral obstruction in general.

Risk Factors

- Urolithiasis
- Urinary tract neoplasia

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- Urethral stricture formation at a site of previous urethral injury or surgery
- Urethral blood clots
- Prostatitis
- Prostatic abscessation
- Paraprostatic pseudocyst formation
- Proliferative urethritis
- Penile fracture
- Perineal herniation
- Intervertebral disk disease
- Spinal neoplasia (intradural or extradural)
- Spinal trauma
- Reflex dyssynergia

Historical Findings

- The clinical signs associated with urethral obstruction can be divided into urinary signs and extraurinary signs.
- Urinary signs.
  - Stranguria
  - Pollakiuria
  - Hematuria
  - Attenuated urine stream
- Extraurinary signs.
  - Mental dullness/lethargy
  - Inappetence due to uremia
  - Vomiting due to uremia
  - Collapse/weakness
  - Abdominal distension
  - Abdominal pain



CLINICAL FEATURES

- Physical examination findings in patients with urethral obstruction can be divided into urinary and extraurinary findings.
- Urinary tract abnormalities.
  - Large, firm urinary bladder which is difficult or impossible to express.
  - Rectal examination may reveal a urethrolith, urethral mass, or prostatic mass.
  - Renomegaly (uncommon, typically associated with chronic, partial obstructions).
- Extraurinary abnormalities.
  - Dehydration
  - Mental dullness
  - Bradycardia

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- Cardiac arrhythmias
- Hypothermia
- Abdominal pain



DIFFERENTIAL DIAGNOSIS

- Constipation may cause a patient to have frequent, unproductive squatting.
- Unobserved urinary incontinence can cause a patient to have an attenuated urine stream.
- Other causes of abdominal pain include:
  - Peritonitis (e.g., pancreatitis, septic peritonitis)
  - Gastrointestinal distension due to obstruction
  - Acute nephritis
  - Referred pain (e.g., spinal pain).
  - Other causes of abdominal distension include peritoneal effusion, organ enlargement (e.g., hepatomegaly, splenomegaly), or gastrointestinal distension due to obstruction.
- Other causes of hyperkalemia include:
  - Acute kidney insult
  - Ureteral obstruction
  - Hypoadrenocorticism (Addison's disease)
  - Hypoaldosteronism
  - Iatrogenic.
- Other causes of bradyarrhythmia include:
  - Increased vagal tone
  - Atrioventricular block
  - Sick sinus syndrome.
- Other causes of azotemia include:
  - Acute or chronic kidney disease
  - Bilateral ureteral obstruction (or unilateral ureteral obstruction in a patient with concurrent disease affecting the nonobstructed kidney)
  - Severe dehydration.



DIAGNOSTICS

- Electrocardiogram: hyperkalemia can cause bradycardia, tall T waves, and a prolonged PR interval.
- Serum biochemical profile: may reveal azotemia, hyperphosphatemia, hyperkalemia, and acidemia.
- Complete blood count: may reveal a stress leukogram.
- Urinalysis: may reveal hematuria, proteinuria, pyuria, crystalluria, and dysplastic epithelial cells.
- Urine culture and sensitivity (via cystocentesis): bacterial infections may be associated with the etiology of obstruction or may be a sequela of obstruction.

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- Abdominal radiography: may reveal radiopaque cystic and/or urethral calculi, prostatomegaly, penile fracture, and loss of peritoneal detail in cases of secondary bladder rupture.
- Abdominal ultrasonography: may reveal cystic and/or proximal urethral calculi, masses of the proximal urethra, bladder trigone, or prostate, hydronephrosis/hydroureter consistent with concurrent obstruction of the upper urinary tract.
- Contrast radiography: may reveal radiolucent cystic and/or urethral calculi, masses affecting the bladder trigone or urethra, and urethral strictures.
- Cystourethroscopy: may reveal cystic and/or urethral calculi, masses affecting the bladder trigone or urethra, and urethral strictures. Cystoscopy-guided techniques are available for the treatment of some causes of urethral obstruction.



THERAPEUTICS

- The objectives of treatment in cases of urinary obstruction are to:
  - Resolve any metabolic abnormalities caused by the obstruction (e.g., hyperkalemia, azotemia, hyperphosphatemia)
  - Resolve dehydration
  - Provide the patient with a patent pathway for urine outflow
  - Prevent recurrence of obstruction in the future.
- Any life-threatening metabolic or cardiovascular abnormalities are addressed prior to anesthesia for urinary tract decompression/catheter placement.
- Procedures that may be attempted to temporarily decompress the urinary tract include the following.
  - Urinary catheterization: the goal of urinary catheterization is to establish urine flow and stabilize the patient prior to definitive treatment for the cause of the urinary obstruction.
    - Catheterization may require sedation in order to be successful.
    - If the catheter does not pass smoothly, saline can be infused into the urethra during passage of the catheter in attempts to retropulse the obstructing lesion into the urinary bladder.
    - In cases in which retrograde urinary catheterization is unsuccessful, antegrade urethral catheterization has been described.
    - Indwelling urinary catheters are attached to a closed-collection system.
- Cystocentesis: careful decompression of the bladder via cystocentesis can ease retrograde passage of a urinary catheter, and can also be performed as a short-term measure in preparation for definitive therapy if catheter placement is difficult or impossible.

Drugs of Choice

- 10% calcium gluconate: 0.5–1 mL/kg over 10 minutes.
  - Used to treat severely hyperkalemic patients with electrocardiographic abnormalities suggestive of cardiotoxicity/atrial standstill.

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  - The effects of calcium gluconate are typically short-lived, and if a patent conduit for urine flow cannot be obtained quickly, further measures will likely be necessary to treat hyperkalemia.
- Sodium bicarbonate: 1–2 mEq/kg slow bolus. Used to treat hyperkalemia as well as the metabolic acidosis.
- Regular insulin: 0.2–0.5 U/kg followed by 4 mL of 50% dextrose per unit of insulin administered (as a slow bolus, then CRI 2.5% dextrose). Used to treat life-threatening hyperkalemia.
- Intravenous fluids.
  - Buffered isotonic fluid (e.g. lactated Ringer's, Normosol-R, or Plasmalyte).
  - Used as needed to correct the patient's dehydration and/or hypovolemia.
- Antibiotic therapy.
  - Amoxicillin-clavulanic acid (Clavamox®):15 mg/kg PO BID.
  - Amoxicillin-sulbactam (Unasyn®): 22 mg/kg IV q8h.
  - Presumptive antibiotic therapy is used while pending urine culture results to guide definitive therapy.
- Diazepam: 0.2 mg/kg PO or IV q8h. Used for urethral skeletal muscle relaxation.
- Prazosin: 1 mg/15 kg PO q8h.
  - Alpha-antagonist used to reduce urethral tone.
  - Preferred over phenoxybenzamine because of its faster onset of action and reduced residual effects.
- Phenoxybenzamine (0.25 mg/kg PO q12–24h). Alpha-antagonist used to reduce urethral tone.

Precautions/Interactions

- Potassium-containing fluids should be used sparingly or not at all.
- Drugs that are known to cause hypotension should not be used until the patient's hydration and volume status have been assessed and addressed with intravenous fluid therapy as needed.
- Drugs that are excreted through the kidneys should be used sparingly or not at all until urethral obstruction has resolved.

Surgical/Interventional Considerations

- Many of the mechanical causes of urethral obstruction in dogs require interventions in order to be definitively treated.
- Cystotomy, laser lithotripsy, percutaneous cystolithotomy, and laparoscopy-assisted cystolithotomy have been described for the removal of uroliths from the lower urinary tract.
- Scrotal urethrostomy may be considered in male dogs with obstructive urethroliths that cannot be dislodged via urethral catheterization. However, many of these dogs may be treated with antegrade cystoscopic approaches, and scrotal urethrostomy should be avoided if possible.

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- Permanent cystostomy tube placement is a salvage procedure to be considered in cases in which the cause of urethral obstruction is not treatable, or in which previous treatment attempts have been unsuccessful.
- Urethral stent placement is appropriate in patients with urethral or prostatic tumors or urethral strictures.
- Urethral balloon dilation is used to open urethral strictures, but may require more than one attempt at dilation prior to successful resolution of the obstruction.

Nursing Care

- Any indwelling urinary catheters should be assessed frequently (every 1–2 hours) to ensure that they are still appropriately positioned, they have not sustained any damage, and they have not been soiled by feces.

Diet

- While in the hospital setting, patients with urethral obstruction that have developed postrenal azotemia may benefit from a bland diet, as they often have significant gastrointestinal upset.
- Patients with urethral obstruction due to urolithiasis may require long-term diet changes in order to prevent stone recurrence or, in the case of struvite uroliths, as part of a dissolution protocol.

Activity

- While a patient has an indwelling urinary catheter in place, their activity should be limited in order to prevent inadvertent removal of the catheter.
- The patient should wear an Elizabethan collar at all times while catheterized to prevent biting or licking the catheter.
- Following definitive treatment of the urethral obstruction, activity restriction is variable dependent upon the procedures that were performed.
    - Two weeks of restricted activity is recommended for patients following surgical procedures
    - Shorter periods of activity restriction (1–2 days) are recommended following laser lithotripsy or urethral stent placement.



COMMENTS

Client Education

- Urethral obstruction is a painful condition.
- There are many risks associated with urethral obstruction.
    - Renal damage
    - Life-threatening metabolic abnormalities
    - Bladder rupture

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- Treatment is mandatory for patients with complete urethral obstruction, and should be considered a three-part process.
  - Treatment of life-threatening metabolic abnormalities.
  - Decompressing the urinary tract.
  - Definitive treatment for the cause of urethral obstruction (which is determined from diagnostic testing).
- There are risks associated with decompression procedures, but these risks are much lower than those associated with ongoing urethral obstruction.

Patient Monitoring

- Regular monitoring of vital signs, hydration status, and cardiovascular stability is essential.
- Urine output measurements should be taken every 2–4 hours.
  - Adjustments should be made in fluid therapy based on these measurements.
  - Many patients with urethral obstruction will experience a postobstructive diuresis and have increased fluid needs following resolution of urethral obstruction.
- In patients with cardiotoxicity due to hyperkalemia, an ECG should be closely monitored until the hyperkalemia has been addressed and the ECG has normalized for a significant length of time.
- Patients that receive insulin as part of the treatment of hyperkalemia at the time of presentation should have frequent assessment of their blood glucose levels for at least 4–6 hours after insulin administration.
- Electrolyte levels should be reassessed frequently (every 6–8 hours) after the obstruction is relieved, as some patients will develop hypokalemia during a period of postobstructive diuresis.

Prevention/Avoidance

- The prevention of recurrent urinary obstruction is dependent upon its underlying cause.

Possible Complications

- Complications of urethral obstruction include death, cardiotoxicity due to hyperkalemia, and permanent damage to the kidneys, ureters, urinary bladder, or urethra.
- Possible complications associated with urinary catheterization include urethral injury and urethral rupture.
- Hypokalemia may result from postobstructive diuresis following relief of urethral obstruction.

Expected Course and Prognosis

- The prognosis for immediate relief of urethral obstruction following urethral catheterization is good to excellent.
- The prognosis for long-term relief of urethral obstruction is dependent upon the cause of obstruction.

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Synonyms

- Urinary tract obstruction

Abbreviations

BID: twice a day (bis in die)
CRI: constant rate infusion
ECG: electrocardiogram
IV: intravenous
PO: by mouth (per os)

Suggested Reading

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chapter 108

Urethral Obstruction – Feline

DEFINITION/OVERVIEW

- Urethral obstruction refers to the restricted flow of urine from the urinary bladder to the external urethral orifice.

ETIOLOGY/PATHOPHYSIOLOGY

- Urethral obstruction is caused by any process which creates resistance to the flow of urine within the urethra. Dependent upon the duration and degree of the obstruction, metabolic abnormalities (hyperkalemia, azotemia, acidemia) may develop. Urethral obstruction can be mechanical or functional in origin.

Systems Affected

- Cardiovascular: bradyarrhythmias may develop in patients with hyperkalemia.
- Respiratory: patients with severe uremia may develop uremic pneumonitis.
- Gastrointestinal: uremia can result in gastrointestinal ulceration, as well as vomiting.
- Renal/urologic: urethral obstruction can cause secondary renal injury when the pressure within the urinary bladder is high enough to prevent the flow of urine from the ureters into the urinary bladder.

SIGNALMENT/HISTORY

- Urethral obstruction is more common in male than in female cats.

Risk Factors

- Feline lower urinary tract disease (FLUTD) or feline idiopathic cystitis (FIC).
  - The most common cause of urethral obstruction in cats.
  - Obstruction is caused by urethrospasm and excess mucous and crystal production resulting in urethral plug formation.

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- Urolithiasis.
- Urinary tract neoplasia.
- Urethral stricture formation at a site of previous urethral injury or surgery.
- Urethral blood clots.
- Intervertebral disk disease.
- Spinal neoplasia (intradural or extradural).
- Spinal trauma.

Historical Findings

- Urinary signs.
  - Stranguria
  - Pollakiuria, recognized as frequent visits to the litterbox with small volumes of urine produced at each attempt
  - Hematuria
  - Attenuated urine stream
  - Vocalization during urination
- Extraurinary signs.
  - Mental dullness/lethargy
  - Inappetence due to uremia
  - Vomiting due to uremia
  - Collapse/weakness
  - Abdominal distension
  - Abdominal pain



CLINICAL FEATURES

- Physical examination findings in patients with urethral obstruction can be divided into urinary and extraurinary findings.
- Urinary tract abnormalities.
  - Large, firm urinary bladder which is difficult or impossible to express.
    - Occasionally, cats will have a small but very firm bladder when obstructed.
  - Renomegaly (uncommon, typically associated with chronic, partial obstructions).
- Extraurinary abnormalities.
  - Dehydration
  - Mental dullness
  - Bradycardia
  - Cardiac arrhythmias
  - Hypothermia
  - Abdominal pain

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DIFFERENTIAL DIAGNOSIS

- Constipation may cause a patient to have frequent, unproductive visits to the litterbox.
- Other causes of abdominal pain include:
  - Peritonitis (e.g., pancreatitis, septic peritonitis)
  - Gastrointestinal distension due to obstruction
  - Acute nephritis
  - Referred pain (e.g., spinal pain).
- Other causes of abdominal distension include:
  - Peritoneal effusion
  - Organ enlargement(e.g., hepatomegaly, splenomegaly)
  - Gastrointestinal distension due to obstruction.
- Other causes of hyperkalemia include
  - Acute kidney insult
  - Ureteral obstruction
  - Iatrogenic.
- Other causes of bradyarrhythmia include atrioventricular block.
- Other causes of azotemia include:
  - Acute or chronic kidney disease
  - Bilateral ureteral obstruction (or unilateral ureteral obstruction in a patient with concurrent disease affecting the nonobstructed kidney)
  - Severe dehydration.

DIAGNOSTICS

- Electrocardiogram: hyperkalemia can cause bradycardia, tall T waves, and a prolonged PR interval.
- Serum biochemical profile: may reveal azotemia, hyperphosphatemia, hyperkalemia, and acidemia.
- Complete blood count: may reveal a stress leukogram.
- Urinalysis: may reveal hematuria, proteinuria, pyuria, crystalluria, and dysplastic epithelial cells.
- Urine culture and sensitivity (via cystocentesis): bacterial infections may be associated with the etiology of obstruction or may be a sequela of obstruction.
- Abdominal radiography: may reveal radiopaque cystic and/or urethral calculi, and loss of peritoneal detail in cases of secondary bladder rupture.
- Abdominal ultrasonography: may reveal cystic and/or proximal urethral calculi, masses of the proximal urethra or bladder trigone, and hydronephrosis/hydroureter consistent with concurrent obstruction of the upper urinary tract.

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- Contrast radiography: may reveal radiolucent cystic and/or urethral calculi, masses affecting the bladder trigone or urethra, and urethral strictures.
- Cystourethroscopy (more readily performed in female than male cats): may reveal cystic and/or urethral calculi, masses affecting the bladder trigone or urethra, glomerulations (associated with FLUTD/FIC), and urethral strictures. Cystoscopy-guided techniques are available for the treatment of some causes of urethral obstruction.



THERAPEUTICS

- The objectives of treatment in cases of urinary obstruction are to:
  - Resolve any metabolic abnormalities caused by the obstruction (e.g., hyperkalemia, azotemia, hyperphosphatemia)
  - Resolve dehydration
  - Provide the patient with a patent pathway for urine outflow
  - Prevent recurrence of obstruction in the future.
- Any life-threatening metabolic or cardiovascular abnormalities are addressed prior to urinary tract decompression.
- Procedures that may be attempted to temporarily decompress the urinary tract include the following.
  - Urinary catheterization: the goal of urinary catheterization is to establish urine flow and stabilize the patient prior to definitive treatment for the cause of the urinary obstruction.
    - Catheterization usually requires sedation in order to be successful.
    - If the catheter does not pass smoothly, saline can be gently infused into the urethra during passage of the catheter in attempts to dilate the urethra and retropulse obstructing material into the urinary bladder.
    - In cases in which retrograde urinary catheterization is unsuccessful, antegrade urethral catheterization has been described.
    - Indwelling urinary catheters are attached to a closed-collection system.
    - In cats with FLUTD as the cause of urethral obstruction, longer periods of indwelling catheterization have been associated with a decreased risk of recurrent obstruction. The catheter should be kept in place until the urine is no longer hematuric.
- Cystocentesis: careful decompression of the bladder via cystocentesis can ease retrograde passage of a urinary catheter, and can also be performed as a short-term measure in preparation for definitive therapy if catheter placement is difficult or impossible.

Drugs of Choice

- 10% calcium gluconate: 5–15 mg/kg over 10 minutes.
  - Used to treat severely hyperkalemic patients with electrocardiographic abnormalities suggestive of cardiotoxicity.

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  - The effects of calcium gluconate are typically short-lived, and if a patent conduit for urine flow cannot be obtained quickly, further measures will likely be necessary to treat hyperkalemia.
- Sodium bicarbonate: 1–2 mEq/kg slow bolus. Used to treat hyperkalemia as well as the metabolic acidosis.
- Regular insulin: 0.5–1 U/kg IM or IV followed by 2 g dextrose IV per unit of insulin administered (slow bolus, followed by 2.5% dextrose as CRI). Used to treat life-threatening hyperkalemia.
- Intravenous fluids.
  - Buffered isotonic fluid (e.g. lactated Ringer's, Normosol-R, or Plasmalyte).
  - Used as needed to correct the patient's dehydration and/or hypovolemia.
- Antibiotic therapy.
  - Amoxicillin-clavulanic acid (Clavamox®):15 mg/kg PO BID.
  - Amoxicillin-sulbactam (Unasyn®): 22 mg/kg IV q8h.
  - Presumptive antibiotic therapy is used while pending urine culture results to guide definitive therapy.
- Diazepam: 0.2 mg/kg IV q8h.
  - Used for urethral skeletal muscle relaxation.
  - Should not be used orally in cats due to a risk of acute hepatotoxicity.
- Prazosin: 0.25–0.5 mg (total dose) PO q8–12h.
  - Alpha-antagonist used to reduce urethral tone.
  - Preferred over phenoxybenzamine because of its faster onset of action and reduced residual effects.
- Phenoxybenzamine: 2.5–7.5 mg (total dose) PO q24h.
  - Alpha-antagonist used to reduce urethral tone.

Precautions/Interactions

- Potassium-containing fluids should be used sparingly or not at all.
- Drugs that are known to cause hypotension should not be used until the patient's hydration and volume status has been assessed and addressed with intravenous fluid therapy as needed.
- Drugs excreted through the kidneys should be used sparingly or not at all until urethral obstruction has resolved.

Surgical/Interventional Considerations

- Perineal urethrostomy should be considered in male cats with repeated bouts of urethral obstruction due to FLUTD/FIC or with obstructive urethroliths that cannot be dislodged via urethral catheterization.
- Cystotomy, laser lithotripsy, percutaneous cystolithotomy, and laparoscopy-assisted cystolithotomy have been described for the removal of uroliths from the lower urinary tract.

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- Permanent cystostomy tube placement is a salvage procedure to be considered in cases in which the cause of urethral obstruction is not treatable, or in which previous attempts at treatment have been unsuccessful.
- Urethral stent placement is appropriate in cats with urethral strictures or urethral tumors.
- Urethral balloon dilation can be used to open urethral strictures, but may require more than one attempt at dilation prior to successful resolution of the obstruction.

Nursing Care

- Any indwelling urinary catheters should be assessed frequently (every 1–2 hours) to ensure that they are still appropriately positioned, that they have not sustained any damage, and that they have not been soiled by feces.

Diet

- While in the hospital setting, patients with urethral obstruction that have developed postrenal azotemia may benefit from a bland diet, as they often have significant gastrointestinal upset.
- Patients with urethral obstruction due to urolithiasis may require long-term diet changes in order to prevent stone recurrence, or, in the case of struvite uroliths, as part of a dissolution protocol.
- Increased water intake is an essential component of the long-term treatment plan for cats with FLUTD/FIC. One way in which water intake can be increased is a change from a dry diet to a canned diet.

Activity

- While a patient has an indwelling urinary catheter in place, their activity should be limited in order to prevent inadvertent removal of the catheter.
- The patient should wear an Elizabethan collar at all times while catheterized to prevent biting or licking the catheter.
- Following definitive treatment of the urethral obstruction, activity restriction is variable dependent upon the procedures that were performed.



COMMENTS

Client Education

- Urethral obstruction is a painful condition.
- There are many risks associated with urethral obstruction.
  - Renal damage
  - Life-threatening metabolic abnormalities
  - Bladder rupture

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- Treatment is mandatory for patients with complete urethral obstruction, and should be considered a three-part process.
  - Treatment of life-threatening metabolic abnormalities.
  - Decompressing the urinary tract.
  - Definitive treatment for the cause of urethral obstruction (which is determined from diagnostic testing).
- There are risks associated with decompression procedures, but these risks are much lower than those associated with ongoing urethral obstruction.
- FLUTD often requires life-long dietary manipulation to avoid recurrence of urethral obstruction.

Patient Monitoring

- Regular monitoring of vital signs, hydration status, and cardiovascular stability.
- Urine output measurements should be taken every 2–4 hours.
  - Adjustments should be made in fluid therapy based on these measurements.
  - Many patients with urethral obstruction will experience a postobstructive diuresis and have increased fluid needs following resolution of urethral obstruction.
- In patients with cardiotoxicity due to hyperkalemia, the ECG should be closely monitored until the hyperkalemia has been addressed and the ECG has normalized for a significant length of time.
- Patients that receive insulin as part of hyperkalemia treatment at the time of presentation should have frequent assessment of their blood glucose levels for at least 4–6 hours after insulin administration.
- Electrolyte levels should be reassessed frequently (every 6–8 hours) after the obstruction is relieved, as some patients will develop hypokalemia during a period of postobstructive diuresis.

Prevention/Avoidance

- The prevention of recurrent urinary obstruction is dependent upon its underlying cause.
- In patients with FLUTD/FIC, prevention of recurrence of obstruction is aimed at increasing water intake and reducing environmental stressors.
- In patients with FLUTD/FIC, longer periods of indwelling catheterization are associated with decreased risk of reobstruction in the short term.

Possible Complications

- Complications of urethral obstruction include death, cardiotoxicity due to hyperkalemia, and permanent damage to the kidneys, ureters, urinary bladder, or urethra.
- Possible complications associated with urinary catheterization include urethral injury and urethral rupture.
- Hypokalemia may result from postobstructive diuresis following relief of urethral obstruction.

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Expected Course and Prognosis

- The prognosis for immediate relief of urethral obstruction following urethral catheterization is good to excellent.
- The prognosis for long-term relief of urethral obstruction is dependent upon the cause of obstruction.

Synonyms

- Urinary tract obstruction

Abbreviations

BID: twice a day (bis in die)
CRI: constant rate infusion
ECG: electrocardiogram
FIC: feline idiopathic cystitis
FLUTD: feline lower urinary tract disease
IM: intramuscular
IV: intravenous
PO: by mouth (per os)

Suggested Reading

Adams LG, Syme HM. Canine ureteral and lower urinary tract diseases. In: Ettinger SJ, Feldman EC, eds. Textbook of Veterinary Internal Medicine, 7th edn. St Louis, MO: Saunders Elsevier, 2010; pp. 2086–2115.

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Reisner TM. Urinary tract emergencies. Vet Clin Small Anim 2005; 35:359–373.

Author: Megan J. Morgan, VMD

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chapter 109

Urethral Prolapse



DEFINITION/OVERVIEW

- Extrusion of the urethral mucosa through the urethral orifice, most commonly in males.



ETIOLOGY/PATHOPHYSIOLOGY

- Unknown: may occur after sexual excitement, concurrent with urethral infections or may be the result of increased intraabdominal pressure in brachycephalic breeds due to chronic increased airway resistance.
- Genetic or congenital abnormalities may be involved.

Systems Affected

- Urologic: hematuria, urinary incontinence, pollakiuria.
- Reproductive: intermittent erection.



SIGNALMENT/HISTORY

- Male dogs: English Bulldogs and other brachycephalic breeds, Boston Terriers, Yorkshire Terriers.
- Young dogs: 9–18 months old.

Historical Findings

- Penile hemorrhage
- Urinary signs
- Sexual excitement
- Licking of the penis
- Mass on the penis

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CLINICAL FEATURES

- Red pea-shaped structure at the end of the penis (Fig. 109.1)
- Urethral/penile bleeding

DIFFERENTIAL DIAGNOSIS

- Trauma
- Urinary tract infection
- Urethritis
- Cystic/urethral urolith(s)
- Penile neoplasia
- Prostate disease
- Testicular disease
- Os penis fractures
- Anatomic abnormalities – persistent penile frenulum, hypospadias

DIAGNOSTICS

- CBC and serum biochemistry: anemia (regenerative) if significant blood loss.
- Urinalysis: hematuria in a voided sample.



Fig. 109.1. Urethral prolapse in a Bulldog.

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- PT, PTT, BMBT, and platelet count to rule out coagulopathy.
- Urine culture and sensitivity.
- Abdominal radiographs: rule out radiodense uroliths, urethral obstruction, evaluation of the prostate; evaluate the os penis for fractures.
- Abdominal ultrasound: rule out radiolucent uroliths, bladder neoplasia and prostatic disease.
- Cystography: double contrast study to rule out uroliths, and other urethral, prostatic, and bladder diseases.



THERAPEUTICS

- The objective is reduction of the prolapsed urethral mucosa to restore urinary function, not necessarily reproductive function.
  - Medical management: consider if asymptomatic and minimal episodic bleeding, seldom curative.
  - An Elizabethan collar is necessary.

Drugs of Choice

- NSAIDs: carprofen 2.2 mg/kg PO q12h.
- Opioids: hydromorphone 0.05–0.2 mg/kg IV, IM, SQ q2–4h; tramadol 1–4 mg/kg PO q8–12h.
- Antibiotics appropriate to urinary and prostate diagnosis.

Precautions/Interactions

- Standard anesthetic precautions with brachycephalic breeds.

Activity

- Decreased activity, sedation may be necessary.
- Prevent contact with female dogs during recovery period.

Surgical Considerations

- Consider if extensive prolapse, ulceration, necrosis or severe bleeding; recommended therapy in most cases.
  - Surgical options: purse-string, surgical excision, and urethropexy.
  - A purse-string can be placed in the emergency setting. Pass a large urinary catheter into the urethra to reduce the prolapse. A purse-string is placed using nonabsorbable suture in the tunic of the penis. The purse-string should be placed as close to the urethral opening as possible. The urinary catheter is removed and the purse-string is left in place for a minimum of 5 days.
  - Castration: controversial for the treatment of sexual excitement.
- Treat any underlying urinary or reproductive disease.

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COMMENTS

Client Education

- Warn owners that recurrence is possible even with surgical correction.
- The patient should avoid sexual stimulation.

Patient Monitoring

- PCV.
- Ability to urinate.
- Urethral mucosa – progressing prolapse and trauma.

Prevention/Avoidance

- Avoid female dogs, if associated with penile erection.
- Correction of brachycephalic airway syndrome – may decrease intraabdominal pressure.

Possible Complications

- Anemia, self-trauma, surgical dehiscence, infection, and recurrence.

Expected Course and Prognosis

- Prolapse, bleeding, and trauma may progress until surgical correction is necessary.
- Prognosis: good, but recurrence is possible.

Abbreviations

BMBT: buccal mucosal bleeding time
CBC: complete blood count
PCV: packed cell volume
PT: prothrombin time
PTT: partial thromboplastin time

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Author: Stacy D. Meola, MS, DVM

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chapter 110

Vaginal Hyperplasia/ Prolapse

DEFINITION/OVERVIEW

- Protrusion of spherical or donut-shaped mass from vulva during proestrus or estrus.
- Type I: slight eversion of the vaginal floor but no protrusion through the vulva.
- Type II: vaginal tissue prolapses through the vulvar opening (tongue-shaped mass).
- Type III: donut-shaped eversion of the entire vaginal wall, including the urethral orifice, which can be seen ventrally on the prolapsed tissue (Fig. 110.1).
- Exaggerated response of vaginal mucosa to estrogen; some affected animals have follicular cysts.
- Despite the name, the change seen histopathologically is consistent with edema rather than hyperplasia or hypertrophy.
- Severe prolapse: may occlude the urethra and prevent normal urination.

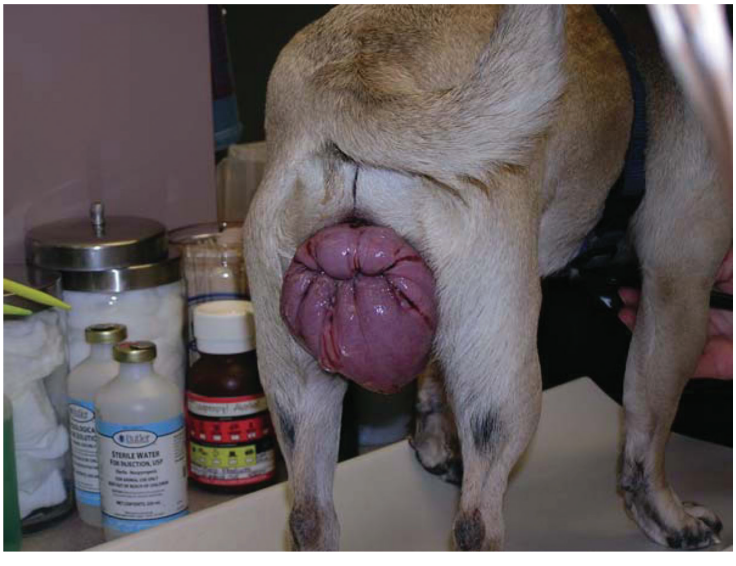


Fig. 110.1. Vaginal hyperplasia in a 3-year-old pug.

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ETIOLOGY/PATHOPHYSIOLOGY

- Exaggerated response of the vaginal lining to the presence of an estrogen level normally present during proestrus/estrus.
- Some bitches may have follicular cysts, but this is not common or necessary to produce vaginal hyperplasia.
- Exogenous exposure to estrogens, such as human skin to which hormone replacement therapy cream has been applied, may also result in vaginal hyperplasia.
- A few bitches have recurrence near the end of diestrus, in association with a rise in estrogen at that time.

Systems Affected

- Reproductive: protrusion of edematous vaginal tissue through vulva.
- Urologic: stage III can cause urethral occlusion and obstructive renal failure.

SIGNALMENT/HISTORY

- Usually seen in younger bitches, typically on their first heat cycle.
- A number of breeds have been reportedly affected; due to the increased incidence in some breeds, a genetic cause is postulated.
- Increased incidence has been reported in boxer, mastiff, bull mastiff, bulldog, American pit bull terrier and related breeds, and Labrador retriever.
- Also reported in Saint Bernards, German shepherd dogs, walker hounds, Chesapeake Bay retrievers, springer spaniels, Weimeraners, Airedale terriers, and mixed breeds.

Historical Findings

- Currently in proestrus or estrus
- Occasionally in late gestation or diestrus
- Protrusion of tissue through the vulva
- Failure to allow mating
- Difficulty urinating
- Excessive licking of vulva
- Prior occurrence

CLINICAL FEATURES

- Protrusion of round, tongue-shaped, or donut-shaped tissue mass from the vulva.
- Vaginal examination: locate lumen and urethral orifice; types I and II: lumen is dorsal to the prolapse; type III: lumen is central to the prolapse; urethral orifice is ventral to the prolapse.

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- Prolapse with all three types.
- Tissue may be dry or necrotic.



DIFFERENTIAL DIAGNOSIS

- Vaginal polyp: differentiated by vaginal examination.
- Vaginal neoplasia: transmissible venereal tumor and leiomyoma; differentiated by signalment, stage of cycle, and vaginal examination.
- Clitoral hypertrophy; differentiated by careful physical examination.



DIAGNOSTICS

- Vaginal cytology to determine stage of cycle and presence of estrogen stimulation.
- Biopsy in older bitch if needed to differentiate from neoplasia.
- Sedation and vaginoscopy if necessary to differentiate from other masses.

Pathologic Findings

- Careful vaginal examination differentiates from other causes of vaginal masses.
- Histopathology shows dramatic edema of vaginal lining.



THERAPEUTICS

- Outpatient, unless urethral obstruction.
- Breeding: possible by artificial insemination (discuss heritability).
- Prolapsed tissue: keep clean and lubricated with sterile water-soluble lubricant.
- Minimize tissue trauma.
- Instruct client to monitor patient's ability to urinate.
- If urethral obstruction present, place indwelling urinary catheter (Fig. 110.2).
- Regression: usually begins in late estrus; should be resolved during early diestrus.
- Recurrence rate: 66–100% at next estrous cycle.
- Ovariohysterectomy: prevents recurrence; may hasten resolution if performed early in proestrus.
- Severe condition: requires surgical reduction or resection; if possible, perform when the mass is beginning to regress; identify and catheterize urethra; 25% recurrence at next cycle after surgery.

Drugs of Choice

- GnRH (2.2 µg/kg IM) or hCG (1000 IU IM), if breeding not planned that cycle; may hasten ovulation and resolution but is not effective if given after ovulation.

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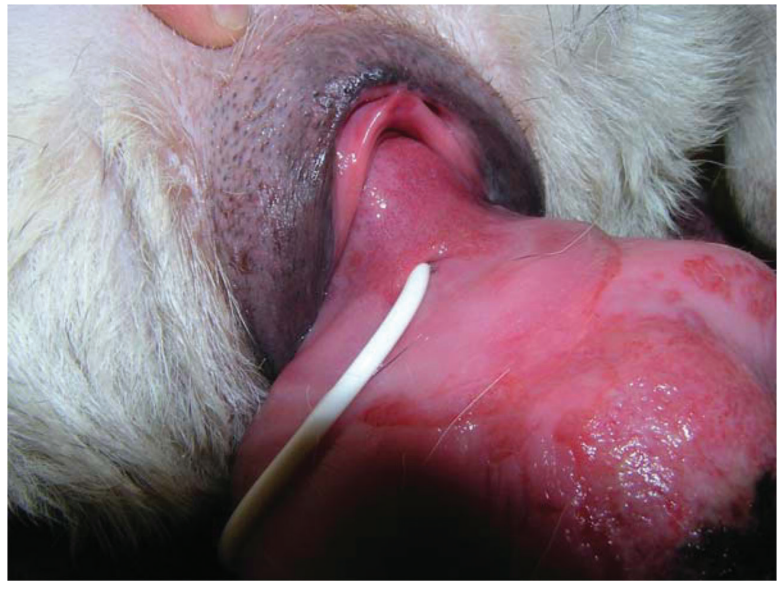


Fig. 110.2. Severe vaginal hyperplasia with urinary catheter in place.

Precautions/Interactions

- Avoid progestational drugs due to risk of inducing pyometra.

Activity

- Keep confined in clean area to prevent tissue damage.
- Apply Elizabethan collar to prevent self-trauma.
- Separate from other dogs to prevent trauma.

Surgical Considerations

- If ability to urinate is compromised or tissue is necrotic, surgical resection is necessary.
- If recurs near end of gestation, delivery of fetuses by caesarean section may be necessary.

COMMENTS

Client Education

- Keep prolapsed tissue clean and moist.
- Monitor ability to urinate.
- Consider removing from breeding program due to genetic basis.
- Breed via artificial insemination if breeding still desired.
- Monitor again near end of diestrus/gestation for recurrence.
- If recurs near end of gestation, caesarean section may be needed.

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Patient Monitoring

- Recheck if any concern about viability of tissue.
- Recheck if owner notes inability to urinate.

Prevention/Avoidance

- Ovariohysterectomy is recommended.

Possible Complications

- Urethral compromise and inability to urinate.
- Necrosis of prolapsed tissue.

Expected Course and Prognosis

- Tissue regresses with time and decrease in estrogen.
- If not spayed, may recur at end of gestation/diestrus.
- If not spayed, typically will recur at next proestrus.
- Ovariohysterectomy prevents recurrence later in cycle or in future.
- Prognosis for recovery good, except with urethral involvement.
- Surgical intervention for type III: prognosis good.

Abbreviations

GnRH: gonadotropin-releasing hormone
hCG: human chorionic gonadotropin
IM: intramuscular

Suggested Reading

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Author: Joni L. Freshman, DVM, MS

chapter 111

Ventricular Dysrhythmias

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DEFINITION/OVERVIEW

- If conduction of sinus node pacemaker impulses to the ventricles is blocked or the impulses decrease in frequency, the lower regions of the heart automatically take over the role of pacemaker for the ventricles, which results in ventricular escape complexes (Fig. 111.1) or an idioventricular rhythm (Fig. 111.2).

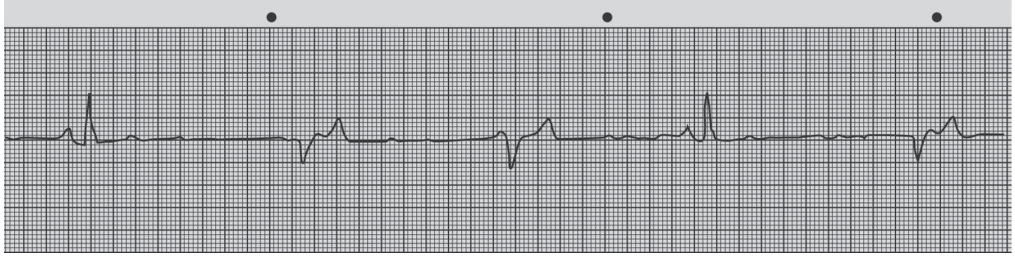


Fig. 111.1. Ventricular escape complexes during various phases in the dominant sinus rhythm in a dog during anesthesia. The sinus rate increased (*not shown*) after anesthesia was stopped; ½ cm 1 mv. Source: Tilley LP. Essentials of Canine and Feline Electrocardiography, 3rd edn. Baltimore, MD: Williams and Wilkins, 1992, with permission.

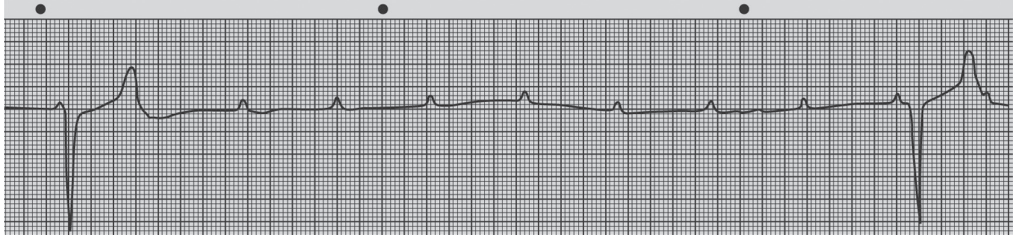


Fig. 111.2. Complete heart block. The P waves occur at a rate of 120, independent of the ventricular rate of 50. The QRS configuration is a right bundle branch block pattern. The regular rate and stable QRS indicate that the rescuing focus is probably near the AV junction. Source: Tilley LP. Essentials of Canine and Feline Electrocardiography, 3rd edn. Baltimore, MD: Williams and Wilkins, 1992, with permission.

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Features

- A series of ventricular escape beats with a heart rate <65 beats per minute in dogs and <100 beats per minute in cats; heart rates of 65–100 beats per minute in dogs and 100–160 beats per minute in cats are often termed accelerated idioventricular rhythms.
- P waves may be absent or may precede, be hidden within, or follow the ectopic QRS complex.
- P waves are unrelated to the QRS complexes.
- QRS configuration is wide and bizarre; similar to that of a ventricular premature complex.



ETIOLOGY/PATHOPHYSIOLOGY

- May be hemodynamically important with slow ventricular rates.
- Does not occur in healthy animals.
- Subsidiary pacemakers seem to discharge more rapidly in cats than in dogs.

Risk Factors/Causes

- Not a primary disease; a secondary result of a primary disease.
- The escape rhythm is a safety mechanism to maintain cardiac output.
- Causes of sinus bradycardia and sinus arrest.
    - Increased vagal tone (i.e., high intracranial pressure, high ocular pressure)
    - Drugs: digoxin, tranquilizers, propranolol, quinidine, anesthetics
    - Addison's disease
    - Hypoglycemia
    - Renal failure
    - Hypothermia
    - Hyperkalemia
    - Hypothyroidism
- Causes of AV block.
    - Congenital
    - Neoplasia
    - Fibrosis
    - Lyme disease

Systems Affected

- Cardiovascular.



SIGNALMENT/HISTORY

Species

- Dogs and cats.

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Breed Predilections

- Atrial standstill in English springer spaniels and Siamese cats.
- Pugs, miniature schnauzers, and Dalmatians prone to conduction abnormalities.

Historical Findings

- Some animals asymptomatic
- Weakness
- Lethargy
- Exercise intolerance
- Syncope
- Heart failure



CLINICAL FEATURES

- Irregular rhythm associated with pulse deficits.
- Variation in heart sounds.
- Possible intermittent "cannon" waves in the jugular venous pulses (with AV block).



DIFFERENTIAL DIAGNOSIS

- Ventricular tachycardia: dogs have a cardiac rate >100 beats per minute; cats >150 bpm.
- Slow heart rate in animals with right bundle branch block, left bundle branch block, or left anterior fascicular block; animals with these disturbances have the P waves associated with the QRS complexes.



DIAGNOSTICS

Complete Blood Count/Biochemistry/Urinalysis

- No specific findings.
- Complete blood testing may suggest a metabolic abnormality.

Other Laboratory Tests

- Drug toxicity.
- Lyme's titer in animals with complete AV block.

Imaging

- Echocardiogram may show structural heart disease.

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Diagnostic Procedures

- Electrocardiography.

Pathologic Findings

- Depend on underlying cause.



THERAPEUTICS

Drugs of Choice

- Atropine or glycopyrrolate usually indicated to block vagal tone or increase the heart rate. If those drugs are ineffective, isoproterenol, dopamine, dobutamine, or artificial pacing may be needed.

Contraindications

- Lidocaine, procainamide, quinidine, propranolol, diltiazem, or any other drug that slows the cardiac rate or reduces contractility.

Precautions/Interactions

- Atropine is briefly vagotonic immediately post injection and can temporarily exacerbate the condition.

Diet

- No modifications or restrictions unless required for management of the underlying condition.

Activity

- Symptomatic animals may require cage rest.

Surgical Considerations

- Pacemaker implantation may be necessary.

Appropriate Healthcare

- Rhythm is an escape or safety mechanism for maintaining cardiac output; do *not* direct treatment toward suppressing this escape rhythm but toward the primary disease process that allows the escape rhythm to assume pacemaker control of the heart.
- Symptomatic treatment is directed at increasing the heart rate.

Nursing Care

- May be required for underlying disease.

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COMMENTS

Client Education

- Inform owner of the need to seek and specifically treat an underlying cause.

Patient Monitoring

- Serial ECG may show clearing of the lesion or progression to complete heart block.
- Serial blood profiles may be needed to monitor progress of the primary disease process.
- Serial echocardiograms may show improvement or progressive changes in cardiac structure.

Possible Complications

- Prolonged bradycardia may cause secondary congestive heart failure or inadequate renal perfusion.

Expected Course and Prognosis

- Arrhythmia may abate when the primary disorder is corrected.
- Prognosis guarded if condition is associated with cardiac or metabolic disorder; poor if the rate is not increased pharmacologically or if underlying cause cannot be identified and treated.

Abbreviations

AV: atrioventricular
ECG: electrocardiogram

See Also

Atrioventricular Block

Suggested Reading

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Author: Larry P. Tilley, DVM

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chapter 112

Vestibular Disease



DEFINITION/OVERVIEW

- Vestibular disease refers to dysfunction of structures responsible for maintenance of balance.
- Peripheral vestibular structures include sensory receptors and first-order afferent neurons that form the vestibular part of the vestibulocochlear nerve (cranial nerve VIII).
- Central vestibular structures include the vestibular nuclei in the rostral medulla, vestibulospinal tracts, and vestibulocerebellum (the flocculonodular lobe and fastigial nucleus).
- Dysfunction of any of these structures results in vestibular signs, including disturbances of gait, posture, and eye position and movement.
- The distinction between central and peripheral vestibular disease is useful for establishing a prioritized list of possible etiologies and also for forming a diagnostic plan.



ETIOLOGY/PATHOPHYSIOLOGY

- Vestibular dysfunction often presents acutely or peracutely, even for disease processes that are more slowly progressive.
- Peripheral causes include the following.
  - Idiopathic (geriatric in dogs, not age-specific in cats): the condition is usually self-resolving within days to weeks, though recurrence is possible and mild deficits (e.g., head tilt) may remain after recovery.
  - Otitis media/interna: most commonly due to commensal organisms that colonize skin.
    - Can spread to central structures.
    - Can be related to foreign body migration through the ear canal.
  - Secondary to mass.
    - Nonneoplastic: nasopharyngeal polyp (cats), cholesteatoma (dogs).
    - Neoplastic: primary aural neoplasia is relatively rare.
    - Primary secretory otitis media (PSOM): Cavalier King Charles Spaniel.

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  - Toxic: any local application of substances to the external ear canal can result in vestibular signs, especially if the tympanic membrane is ruptured.
    - Topical aminoglycosides and chlorhexidine have been especially implicated in permanent toxicity to hair cells.
    - Systemic administration of aminoglycosides (streptomycin and gentamicin) and loop diuretics (e.g., furosemide).
  - Metabolic: hypothyroidism has been associated with vestibular dysfunction in dogs.
  - Trauma: may result in fracture, hemorrhage, contusion or edema causing dysfunction in local vestibular structures.
- Central causes include the following.
  - Meningoencephalitis (noninfectious versus infectious) (viral, bacterial, protozoal, rickettsial, fungal)
  - Vascular event (ischemic or hemorrhagic)
  - Trauma
  - Toxic: metronidazole
  - Otitis media/interna with extension (usually bacterial)
  - Neoplastic
  - Metabolic: thiamine deficiency (cats), hypothyroidism (dogs)

Systems Affected

- Nervous.
- Gastrointestinal: affected patients may have ptyalism, nausea, vomiting and/or hyporexia, especially early in their clinical presentation.



SIGNALMENT/HISTORY

- Any age or breed of dog or cat can develop vestibular disease; however, some specific signalments and historical data may suggest a particular etiology as more or less likely.
- Older (>5 years, usually >8 years), large-breed dogs are predisposed to idiopathic peripheral vestibular disease.
- Younger (<5 years, usually 1–3 years) cats are predisposed to inflammatory polyps arising from auditory tube epithelium.
- Cavalier King Charles Spaniels are prone to developing otitis media/interna secondary to PSOM.
- Several feline breeds (Siamese, Himalayan, Birman) are associated with congenital pendular nystagmoid eye movements that may not be noticed by owners. These movements are constant, conjugate, and lack a distinct fast and slow phase.
- A history of topical otic treatments and/or chronic otitis externa may suggest ototoxicity or otitis media/interna, respectively, as causes.

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CLINICAL FEATURES

- There are several cardinal signs of vestibular disease that are rarely seen with selective dysfunction of other nervous structures.
  - Head tilt: ventral deviation of one ear relative to the other; contrast a head turn, which is deviation of the rostrocaudal axis of the head due to lateral flexion of the cervical vertebral column.
  - Nystagmus: typically "jerk" nystagmus, which involves unequal alternating movements of the globe in opposing directions. Nystagmus is defined by its axis of movement (horizontal, vertical, or rotational), the direction of the fast phase of the movement, and whether the movement of the two eyes is conjugate or dysconjugate.
    - Physiologic nystagmus should be inducible in normal animals with head movement in horizontal rotation. Absence of this reflex indicates dysfunction in either the vestibular system or the extraocular muscles or their innervation.
    - Pathologic nystagmus may be spontaneous, or may be induced by placing the patient in certain head or body positions. Spontaneous nystagmus may change character with changes in head or body position (positional nystagmus).
- (Induced) strabismus: vestibular dysfunction often causes ventral to ventrolateral deviation of the globe ipsilateral to the lesion with head and neck extension.
- Ataxia: vestibular ataxia is characterized by asymmetry and usually presents as leaning, falling, rolling, and/or (tight) circling to one side.
- Bilateral peripheral vestibular disease may be evidenced by a more symmetrical ataxia with no or minimal nystagmus or head tilt and loss of the vestibuloocular reflex with no other signs of brainstem dysfunction. This may be more common in cats.

DIFFERENTIAL DIAGNOSIS

- Occasionally, acute vestibular episodes may be mistaken for seizures.
- Circling patients may have vestibular function or a forebrain lesion.
  - Circling is usually toward the side of the lesion with either localization.
  - Tight circles may be more commonly associated with vestibular dysfunction.
  - Wide, aimless circles are more suggestive of forebrain disease.
- Differentiation of central versus peripheral vestibular disease.
  - Positional nystagmus (spontaneous nystagmus that changes direction with head position, *not* nystagmus that is induced by changes in head position), vertical nystagmus, and dysconjugate nystagmus are more likely to indicate central vestibular disease.
  - Proprioceptive deficits that are lateralized (e.g., asymmetric right-left rather than or in addition to thoracic-pelvic differences) indicate central vestibular disease but can be difficult to assess in severely affected patients.
  - Lateralized paresis (decreased voluntary movement) is likewise indicative of central vestibular disease but can be difficult to assess in severely affected patients.

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  - Alterations in consciousness indicate either brainstem involvement or additional diffuse forebrain disease that demonstrates central localization.
  - Horner's syndrome is commonly seen ipsilateral to a structural peripheral vestibular lesion. It can be seen rarely with central vestibular and/or multifocal CNS disease.
  - Other cranial nerve abnormalities, especially lateralized deficits of cranial nerves V or XII, or deficits associated with cranial nerve IX–XI function, strongly suggest central disease. The *exception* is lateralized deficits of cranial nerve VII, which are equally common with both central and peripheral disease.
- In general, lateralized deficits and head tilt/falling/rolling/circling occur ipsilateral to the side of the lesion in both central and peripheral vestibular disease; the fast phase of nystagmus occurs away from the side of the lesion in both. The *exception* is paradoxical (central) vestibular disease, in which head tilt (most commonly) occurs opposite to the lesion if, and only if, the vestibulocerebellum, caudal cerebellar peduncle, and/or rostral and medial vestibular nuclei are selectively affected.



DIAGNOSTICS

- While you should allow your prioritized list of etiologies to guide your diagnostic and therapeutic plan, the following is an example of how localization of the lesion to a peripheral versus central source may influence the choice of appropriate diagnostics.

Peripheral Vestibular Disease

- CBC/biochemical panel/urinalysis for metabolic disease (e.g., hypothyroidism may result in microcytosis or hypercholesterolemia).
- Additional metabolic testing as indicated (e.g., free T4, TSH).
- The following tests will likely require sedation for optimal performance.
  - Otoscopic examination.
  - Bulla radiography and/or bulla ultrasound.
- The following tests will require at least sedation and possibly general anesthesia.
  - CT +/- contrast.
  - Video otoscopy.
  - Myringotomy with sampling for cytology and culture.
- The following tests will require general anesthesia.
  - Surgical biopsy with sampling for histopathology and culture.
  - MRI and/or CSF may be indicated if there is concern for central spread of peripheral disease or if no cause for vestibular disease is identified and signs are progressive.

Central Vestibular Disease

- CBC/Chem for metabolic disease.
- NIBP, UA, fundic exam, +/- TEG for predilection to vascular events/thromboembolic disease.

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- Alpha-transketolase for thiamine if history suggests.
- MRI (preferably *not* CT) and CSF.

Additional Testing

- Brainstem auditory evoked response (BAER).
  - May be performed in awake or anesthetized animals.
  - Noninvasive and inexpensive.
  - Can be useful for distinguishing peripheral from central vestibular disease in cases where neurological examination is not clear.
  - Can also be useful for identifying involvement of cochlear hair cells/auditory portion of CNVIII, which is less likely to recover function in cases of ototoxicity.



THERAPEUTICS

- There are no specific medications for control of vestibular signs independent of addressing the underlying cause, if known.

Drugs of Choice

- Antiemetics and antinausea medications may be used as needed for patients experiencing these signs.
  - Dolasetron: 0.6 mg/kg IV once daily.
  - Maropitant: 1 mg/kg IV, SQ once daily.
  - Meclizine: 25 mg/dog PO once daily, 12.5 mg/cat PO once daily.
- Diazepam at approximately 0.5 mg/kg IV once, followed by 0.5 mg/kg PO q8h for 3 days has been recommended to promote recovery from metronidazole intoxication.

Precautions/Interactions

- Metronidazole use should be discontinued in a patient with signs of central vestibular disease.
- Topical otic applications should be used with caution and only where necessary to treat definitively diagnosed otitis in patients with concurrent vestibular disease.

Appropriate Healthcare

- Prevent secondary injury due to falls by keeping the patient in a small, well-padded area.
- Use a chest harness or other body support for both thoracic and pelvic limbs when assisting the patient to walk to reduce the chance of falls.

Nursing Care

- Perform recumbent care as needed in patients who cannot right themselves or stand without assistance.

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Diet

- Patients may need fluid and/or nutritional support if unable to take water and food orally for a prolonged period of time.
- Recumbent or rolling patients should be placed in a stable, sternal position for feeding and water intake.

Activity

- Activity may be allowed as tolerated as long as the patient is protected from falling.

Surgical Considerations

- Certain causes of vestibular disease (e.g., otitis media/interna, with or without extension, aural or intracranial mass lesions) may have surgical treatment options. Surgery in these regions may temporarily or permanently worsen dysfunction in vestibular and associated (CNVII, sympathetic innervation to the eye) structures.



COMMENTS

Client Education

- The vestibular system is relatively robust in its ability to compensate for insult, even in adulthood. Dogs with relatively severe vestibular insults are likely to improve over time, as long as the insult is not progressive. However, certain signs, such as head tilt, may never resolve.
- Severe vestibular episodes may appear to owners to be seizures, because of the violent movements associated with rolling and attempted righting, as well as apparent disorientation that may be confused with altered mental status. Video recording of an episode may assist with distinguishing between the two.

Patient Monitoring

- Appropriate monitoring will depend on underlying etiology.
- Patients suspected of having idiopathic vestibular disease have been reported to have recurrent events.

Prevention/Avoidance

- Patients suspected of having or proven to have had a vascular insult should be evaluated for risk factors for thromboembolic disease (protein-losing nephropathy or enteropathy, hyperadrenocorticism, DIC) and appropriately treated if they are found.

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- Patients suspected of having or proven to have had otitis media/interna should be closely monitored for recurrent signs of otitis externa, including head shaking, ear scratching, aural discharge or visible changes to the pinna, although the two conditions may exist independently. Topical otic preparations should be used with caution.

Possible Complications

- It is important to prevent secondary injuries in dogs suffering from vestibular disease that may occur secondary to falls. Confinement to a small, well-padded area when not directly supervised is recommended during recovery, as well as assistance with a harness when walking.

Expected Course and Prognosis

- Variable depending on cause and response to therapy.

Synonyms

- Vertigo: this is a term from human medicine referring to the sensation of loss of balance, and should be avoided in animal patients whose perception of their condition cannot be known with certainty.

Abbreviations

BAER: brainstem auditory evoked response
CBC: complete blood count
CNS: central nervous system
CSF: cerebrospinal fluid
CT: computed tomography
DIC: disseminated intravascular coagulation
IV: intravenous
MRI: magnetic resonance imaging
NIBP: noninvasive blood pressure
PO: by mouth (per os)
PSOM: primary secretory otitis media
SQ: subcutaneous
TEG: thromboelastography
TSH: thyroid-stimulating hormone

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Author: C. Elizabeth Boudreau, DVM, PhD

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chapter 113

Vomiting/Hematemesis



DEFINITION/OVERVIEW

- Vomiting blood.



ETIOLOGY/PATHOPHYSIOLOGY

- Blood may be vomited because of mucosal disruption (ulcer/erosion) in esophageal, gastric, intestinal or gall bladder mucosa, coagulopathy or swallowed blood originating from inside (e.g., mouth, lungs) or outside the body.

Systems Affected

- Gastrointestinal: gastric ulceration/erosion (GUE) most commonly affects the stomach, then small intestines, then large intestines, then esophagus.

Incidence/Prevalence

- Unknown.



SIGNALMENT/HISTORY

Species

- More common in dogs than cats.

Mean Age and Range

- Unknown.

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CLINICAL FEATURES

General Comments

- Many dogs with GUE have no clinical signs.

Historical Findings

- Vomiting blood: may be fresh (red) or digested ("coffee grounds") blood (Figs 113.1, 113.2). Minimal bleeding due to mucosal trauma from vigorous vomiting may cause small "flecks" of blood.
- Melena: black, tarry stools; only seen when large amounts of blood enter the GI tract in a relatively short time. Much less common than hematemesis.
- Anorexia: very common sign of gastric disease, even in absence of vomiting.
- Abdominal pain: may assume "praying position" (infrequent).
- Weakness/dyspnea: either/both can be seen with severe anemia.

Physical Examination Findings

- Anemia: pale oral mucus membranes, tachypnea, systolic heart murmur, weakness, sometimes collapse.
- Abdominal pain: inconsistent; can be subtle when present.
- Melena: very infrequent.



Fig. 113.1. Frank blood in vomitus from a patient with hemorrhagic gastroenteritis.

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■ **Fig. 113.2.** Digested blood in a large amount of vomitus from a Rottweiler with immune-mediated thrombocytopenia and subsequent gastrointestinal bleeding. Note the "coffee-ground" appearance of the vomitus.

- Coagulopathy: petechiae, ecchymosis, hematochezia, epistaxis, retinal hemorrhage, hyphema, and/or hematuria.
- Edema: infrequent; only when serum albumin <1.6 g/dL.

Causes

- Causes of GUE.
  - NSAIDs: Cox-2 selective NSAIDs are safer than nonselective NSAIDs but still cause GUE. Flunixin meglumine is extremely ulcerogenic and should be avoided. Naproxen, ibuprofen, and piroxicam are particularly ulcerogenic in dogs.
  - Glucocorticosteroids: dexamethasone is probably the most ulcerogenic of commonly used steroids. Prednisolone is much less ulcerogenic unless other factors favor GUE.
  - Gastritis: often idiopathic; may be caused by ingestion of toxic/irritative substances (e.g., heavy metals, fertilizer, etc.). *Helicobacter* spp. do not cause canine/feline GUE. Pythiosis causes mucosal disruption and GUE. Gastric foreign bodies occasionally cause GUE, but they inhibit healing of GUE from any other cause. IBD has been associated with GUE, but cause and effect is questionable.
  - "Stress"/poor perfusion/shock: probably poor visceral perfusion coupled with increased endogenous steroids (*not* limited to veterinary superathletes).
  - Gastric hyperacidity: paraneoplastic effect (mast cell tumors, gastrinomas).
  - Neoplasia: disrupts gastric mucosa. Leiomyomas cause very severe gastric hemorrhage, but carcinomas and lymphomas are more common.
  - Iatrogenic: improper surgical closure of gastrotomy incisions. Iatrogenic from invagination of necrotic gastric serosa, submucosa, and mucosa during surgical

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repair of GDV. For this reason, gastric resection is preferable and invagination is not recommended.
  - Hepatic disease: severe hepatic failure (uncertain mechanisms).
  - Hypoadrenocorticism: rare but important; mimics primary GI disease – mucosal disruption due to lack of steroids.
- Nongastric causes of alimentary hemorrhage.
  - Hookworms: severe infestations cause upper duodenal bleeding with duodenal-gastric reflux.
  - Hemorrhagic gastroenteritis: assumed to be immune or bacteria induced.
  - Pancreatitis: duodenal hemorrhage in severe cases due to inflammation by direct extension.
  - Esophagitis: ulcers/erosions due to foreign bodies, caustics (e.g., tetracycline), gastroesophageal reflux of acid.
- Causes of coagulopathies: thrombocytopenia (numerous causes), von Willebrand's disease, vitamin K antagonism, DIC, congenital defects.
- Causes of ingestion of blood.
  - Endogenous blood: swallowing blood coughed up from airway due to airway disease, bronchopulmonary tumor, pneumonia, heartworms, or fungal infections; or that escaping the nose via the choana.
  - Exogenous blood: blood from any outside source (e.g., food, anal sacs) may be ingested and later vomited.

Risk Factors

- NSAID therapy increases risk for GUE.



DIFFERENTIAL DIAGNOSIS

- Hematemesis: hemoptysis, oral bleeding, food material resembling blood.
- Melena: ingestion of bismuth-containing compounds, activated charcoal, other dark substances.



DIAGNOSTICS

CBC/Biochemistry/Urinalysis

- Acute hemorrhage (<3 days) causes nonregenerative anemia plus hypoalbuminemia. Anemias >5 days old cause regenerative anemia plus hypoalbuminemia if not iron deficient. Chronic iron deficiency anemia causes poorly regenerative, microcytic anemia with hypoalbuminemia (i.e., low MCV). Panhypoproteinemia may or may not be found, depending on serum globulin concentration before blood loss. Thrombocytopenia may cause bleeding diathesis.

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- BUN:creatinine ratio: hemorrhage may increase BUN disproportionately compared to serum creatinine concentration; BUN raised by high-protein meals (e.g., blood) – requires major hemorrhage in relatively short period of time. Need BUN and creatinine values from before bleeding event to recognize this disparity.
- Hepatic disease may or may not have increased ALT, hyperbilirubinemia, increased serum bile acids, hypoalbuminemia.
- Hypoadrenocorticism may or may not cause hyponatremia/hyperkalemia, hypercalcemia, hypoglycemia, hypocholesterolemia, inappropriate lymphocytosis or eosinophilia in stressed dogs.

Other Laboratory Tests

- Pancreatic lipase immunoreactivity is the most sensitive test for pancreatitis, but specificity for clinically important disease is uncertain. Resting serum cortisol concentration is an acceptable screen for hypoadrenal patients that need ACTH stimulation testing. Serum gastrin needed to diagnose gastrinomas. Pre- and postprandial serum bile acids or blood ammonia for hepatic insufficiency. Coagulation profiles for coagulopathies. Serology/PCR for pythiosis. Fecal flotation for *Ancylostoma* ova. Buccal mucosal bleeding time is a reasonable screening test for coagulopathies severe enough to cause GI bleeding.

Imaging

- Abdominal radiographs may reveal foreign objects or spontaneous pneumoperitoneum (due to GI perforation).
- Abdominal ultrasound is specific but insensitive. Might demonstrate thickened infiltrated areas, ulcers, scant amounts of free fluid, foreign objects, hepatic disease, and/or pancreatitis. Ultrasound can guide fine needle aspiration of infiltrations or fluid pockets.
- Upper GI contrast radiographs not recommended in patients with upper GI hemorrhage.
- Thoracic radiographs may reveal esophageal foreign bodies or pulmonary lesions.

Diagnostic Procedures

- Gastroduodenoscopy is the most sensitive test for ulcers/erosions between the mouth and distal duodenal flexure. Once coagulopathy is eliminated, biopsies should be obtained of any lesion. Infiltrates are inflammatory or neoplastic; they may be sporadic, necessitating endoscopic guidance of the biopsy forceps to maximize the chance of diagnosis.

Pathologic Findings

- The main question is whether GUE is benign (idiopathic or fungal) or malignant.

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THERAPEUTICS

Drugs of Choice

- Therapy is often ineffective if the cause of GUE (e.g., NSAID drugs) is not removed.
- H2 receptor antagonists are much less effective than proton pump inhibitors. Cimetidine is the least potent and has most side effects. Famotidine is the most potent H2 receptor antagonist (1.0 mg/kg PO or IV, q12–24h). Ranitidine (1–4 mg/kg PO or IV q8–12h) and nizatidine (2.5–5 mg/kg PO q24h) are supposed to have gastric prokinetic activity.
- Proton pump inhibitors are noncompetitive inhibitors of gastric acid secretion and more effective than H2 receptor antagonists. Require 2–5 days to achieve maximal efficacy when administered orally, but immediate effects are superior to H2 receptor antagonists. Omeprazole (1–2 mg/kg PO q12–24h) is most commonly used. Pantoprazole is often used if need to give IV (1 mg/kg once daily).
- Orally administered antacids (e.g., aluminum hydroxide) not recommended: need 6+ daily administrations to be effective.
- Sucralfate (0.5–1 g PO q12–24h) binds to ulcer sites, stimulates healing, and protects them.
- Misoprostol: prostaglandin analog developed as prophylaxis against NSAID-induced GUE. Can also treat existing GUE (2–5 µg/kg PO q8–12h). It can cause diarrhea and/or abdominal cramping. It is an abortifacient.
- Antiemetics may be used symptomatically; maropitant (1 mg/kg SQ q24h or 2 mg/kg PO q24h).

Appropriate Healthcare

- The first goal is to stop GI bleeding; next is to stop clinical signs (e.g., anorexia, vomiting) and resolve the lesion to minimize the chance of perforation.

Supportive Care

- Maintain hydration to ensure good perfusion of GI tract.

Activity

- Allow patient to set their own pace.

Diet

- Avoid high-fat and high-fiber diets, which slow gastric emptying.

Client Education

- All NSAIDs increase GUE risk; if NSAIDs have previously caused GI bleeding, they must be used cautiously in the future.

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Surgical Considerations

- If bleeding due to GUE does not show improvement within 5–7 days of starting medical therapy or if GI blood loss is so severe that it puts the patient at inappropriate risk, scope the patient to see if the responsible lesion(s) appear(s) resectable. Leiomyomas are potentially resectable and should be removed as soon as possible. Patients apparently in danger of perforating should have lesions resected, if possible.

Possible Interactions

- Omeprazole should not be used with clopidogrel. Cimetidine inhibits hepatic p450 enzymes. Sucralfate may slow absorption of other orally administered drugs.

COMMENTS

Patient Monitoring

- Clinical monitoring (i.e., attitude, mucus membrane color, abdominal pain, vomiting, appetite) is usually sufficient to determine the efficacy of medical therapy. Hematocrits <20% (dogs), <18% (cats) must be monitored closely; serial hematocrits may be appropriate. Repeat endoscopy is seldom necessary in patients that are clearly improving clinically.

Prevention/Avoidance

- Avoid concurrent administration of NSAIDs and steroids; concurrent use of two NSAIDs; large or repeated doses of dexamethasone; large doses of any steroid in poorly perfused/hypoxic patients.

Possible Complications

- GUE perforation causes septic peritonitis. Vomiting is sometimes associated with aspiration pneumonia. Severe blood loss necessitates administration of red blood cells. Misoprostol (a synthetic prostaglandin) causes abortion in pregnant animals.

Expected Course and Prognosis

- Prognosis depends on cause. GUE due to NSAIDs and dexamethasone can usually be resolved. Gastric carcinomas have an extremely poor prognosis; leiomyomas are often cured with resection. Paraneoplastic hyperacidity (gastrinomas and mast cell tumors) are often palliated for months to a year with proton pump inhibitors. Severe hepatic failure and gastric pythiosis have a guarded to very poor prognosis, respectively. Hypoadrenocorticism has an excellent prognosis if diagnosed in a timely fashion.

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Abbreviations

ACTH: adrenocorticotrophic hormone
ALT: alanine transaminase
BUN: blood urea nitrogen
CBC: complete blood count
DIC: disseminated intravascular coagulation
GDV: gastric dilation-volvulus
GI: gastrointestinal
GUE: gastric ulceration/erosion
IBD: inflammatory bowel disease
IV: intravenous
MCV: mean corpuscular volume
NSAID: nonsteroidal antiinflammatory drug
PCR: polymerase chain reaction
PO: by mouth (per os)

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chapter 114

Von Willebrand Disease



DEFINITION/OVERVIEW

- Von Willebrand disease is a qualitative or quantitative deficiency in vWF, causing a dysfunctional interaction between platelets and vascular collagen (primary hemostasis), thereby reducing the adherence of platelets to sites of vascular injury.
- Circulating vWF complexes with factor VIII, preventing the enzymatic destruction of factor VIII.
- Stored sources of vWF multimers (sizes vary 100-fold), that are present in endothelial cells and platelets, can be instantaneously mobilized when the respective cells are activated.
- The most common inherited bleeding disorder in dogs, with inheritance being influenced by vWD type.



ETIOLOGY/PATHOPHYSIOLOGY

- Clinical manifestations are brought about by a reduction in two of the established functions of vWF: first, its role in supporting adhesion of platelets to damaged blood vessels (specifically subendothelial collagen), and second, postsynthetic association of vWF with factor VIII that protects factor VIII from proteolysis.
- The disease has been divided into three different major types.
    - Type 1 vWD is characterized by a partial quantitative deficiency of all vWF sizes that have a normal multimeric structure; most frequently documented type with clinical signs indirectly correlated with reduction in plasma vWF.
    - Type 2 vWD is characterized by both a quantitative and qualitative deficiency of vWF, with the quantitative deficiency primarily affecting the larger (more hemostatically active) multimers; least commonly diagnosed variant in animals.
    - Type 3 vWD is the most fatal type, due to a severe quantitative deficiency (vWF:Ag <1%) of vWF.
- Type 3 vWD is associated with the most severe hemorrhage, while type 1 vWD generally manifests with the least severe hemorrhage.

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Systems Affected

- Gastrointestinal: bleeding during teething or mucosal hemorrhage.
- Hemic/lymphatic/immune: anemia if hemorrhage is severe.
- Renal/urologic: hematuria.
- Reproductive: prolonged estral hemorrhage.
- Respiratory: epistaxis.
- Skin/exocrine: hemorrhage from wounds or surgical sites, cutaneous bruising, rarely petechiae.



SIGNALMENT/HISTORY

- No sex predilection.
- Most patients affected from birth.
- Type 1: many canine breeds (Doberman pinschers, Airedales, shelties); Himalayans.
- Type 2: German wirehaired and shorthaired pointers.
- Type 3: Scottish Terrier, Shetland Sheepdog; mixed-breed felines.

Risk Factors

- Known familial inheritance.

Historical Findings

- Bleeding may or may not be a historical feature.
- Excessive hemorrhage noted at the time of teething, during estrus or secondary to minor injury.
- Sometimes familial inheritance is known.



CLINICAL FEATURES

- Symptoms related to a deficiency in primary hemostasis, such as spontaneous mucosal hemorrhage (epistaxis, hematuria, GI hemorrhage); more commonly overt hemorrhage after trauma or during teething or surgery; mild-to-moderate bleeding is typical.
- Petechiation is uncommon in dogs and cats.



DIFFERENTIAL DIAGNOSIS

- Thrombocytopenia
- Inherited (Glanzsman's thrombasthenia, etc.) or acquired (aspirin therapy, etc.) thrombocytopathies

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DIAGNOSTICS

- Different subtypes of vWD require different diagnostic strategies.
- Prolonged BMBT (PFA-100 or other platelet function tests) with normal platelet count is typical, but BMBT is not sensitive enough to consistently detect mild vWD and a normal result does not rule out the disease; in addition, clinical utility is limited by problems with standardization of the procedure.
- The PT is usually normal, while aPTT may be prolonged due to reduced levels of plasma factor VIII; because the plasma level of factor VIII depends on vWF level, patients with milder forms of vWD usually have normal levels of factor VIII.
- Definitive diagnosis can be made with confirmation of low plasma vWF:Ag levels (immunologic test) with types 1 and 3, but results are typically normal in type 2 (Table 114.1).
- Being an acute phase reactant, transiently increased levels of vWF during hemorrhage may obscure the diagnosis; therefore, if possible, tests should not be performed in proximity to hemorrhagic events, acute infection, pregnancy or strenuous exercise.
- Genetic testing for a number of vWD genotypes is commercially available and may be used to confirm a diagnosis in an individual patient and/or eliminate positive individuals from a breeding program.
- If a laboratory diagnosis of vWD is made in an older canine patient with only a recent history of clinical signs, consider evaluating for hypothyroidism.

Pathologic Findings

- Hemorrhage possible at any site, but most commonly associated with areas of trauma or surgery.



THERAPEUTICS

- Different types of vWD require slightly different therapeutic strategies.

TABLE 114.1. Selected laboratory features of vWD types.

vWD type	Factor VIII	vWF:Ag	Multimer structure
Type 1	Decreased	Decreased	Normal
Type 2	Decreased or normal	Decreased or normal	Larger multimers decreased
Type 3	Moderate decrease	Absent or trace	None or trace

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Drugs of Choice

- Traditionally, DDAVP administration has been the treatment of choice with type 1 vWD, used to transiently increase the endogenous release of factor vWF from endothelial storage sites, as well as increasing factor VIII (source controversial).
- Use of DDAVP may avoid transfusion in patients requiring only temporary normalization of primary hemostasis (i.e., patients undergoing brief invasive procedures).
- Effectiveness of DDAVP in veterinary patients has recently come under scrutiny.
- Frozen or freeze-dried cryoprecipitate (concentrated form of vWF and factor VIII) may be used to replace vWF in all three types of vWD.

Precautions/Interactions

- Tachyphylaxis rapidly occurs to DDAVP, limiting its usefulness to just a few doses.
- Free water retention after DDAVP administration rarely leads to volume overload and/or hyponatremia.
- Increasing risk of immunologic transfusion reactions with chronic repeated administration of allogenic blood products.
- Acute administration of FFP may predispose to circulatory overload.

Alternative Drugs

- FFP or fresh whole blood may be used to provide vWF, if cryoprecipitate is not available.
- Administration of DDAVP to the blood donor, prior to unit collection, increases the level of vWF in the donated unit.
- Thyroid supplementation if concomitant hypothyroidism exists.
- Site-specific therapy may be necessary (direct pressure, use of hemostatically active agents, surgical hemostasis).

Activity

- Limit activity in patients manifesting spontaneous hemorrhage.

Surgical Considerations

- Prophylactic vWF replacement prior to invasive procedures, or at least have appropriate blood products readily available; topical application of cryoprecipitate may suffice.
- Avoid elective procedures, but if necessary, limit tissue trauma and maximize surgical hemostasis techniques.
- Avoid medications that may alter hemostasis.

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COMMENTS

Client Education

- Review mode of inheritance and discuss appropriate breeding practices; animals with clinical hemorrhage should not be used for breeding, asymptomatic carriers would preferably not be utilized or at a minimum be bred only with cleared partners.
- Recommend diagnostic testing for related animals.
- Over-the-counter therapies (aspirin, etc.) and herbal supplements, notably garlic, gingko, and ginseng, may exacerbate hemorrhagic tendencies.

Patient Monitoring

- Screening is important for breeds with a high prevalence.
- BMBT is an insensitive tool for monitoring therapeutic response.
- Evaluate fluid and electrolyte status if DDAVP was administered.
- Closely observe for hemorrhagic events, especially following surgical or accidental trauma.

Prevention/Avoidance

- Avoid breeding affected animals.
- Conscious measures should be taken to limit risk of hemorrhage (i.e., conservative nail trims, etc.).

Possible Complications

- Hemorrhage, especially with concurrent medical conditions known to influence primary hemostasis (thrombocytopenia, uremia, hepatic disease, etc.), or administration of medications known to inhibit platelet function (aspirin or other nonsteroidal antiinflammatories, phenothiazine tranquilizers, synthetic colloids, etc.).

Expected Course and Prognosis

- Disease is medically managed, but not cured.
- Hemorrhage may be fatal or is controlled with appropriate therapy.
- Long-term prognosis varies with vWD type.

Abbreviations

aPTT: activated partial thromboplastin time
BMBT: buccal mucosal bleeding time
DDAVP: desmopressin or 1-deamino-8-D-arginine vasopressin

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FFP: fresh frozen plasma
GI: gastrointestinal
PT: prothrombin time
vWD: von Willebrand disease
vWF: von Willebrand factor
vWF:Ag: von Willebrand factor antigen – quantified plasma concentration of vWF

Suggested Reading

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chapter 115

Zinc Toxicity



DEFINITION/OVERVIEW

- Toxicity from the ingestion of zinc-containing objects, most commonly US pennies minted after 1982.
- Causes severe intravascular hemolysis, gastrointestinal irritation, renal injury; may lead to multiple organ failure, DIC, and cardiopulmonary arrest; pancreatitis can occur as a possible sequela.



ETIOLOGY/PATHOPHYSIOLOGY

- The stomach's acidic environment mediates the rapid release of zinc from ingested foreign materials; rate of zinc release depends on gastric pH, presence or absence of food/ingesta, and the length of time foreign objects are in the stomach.
- After absorption, zinc is transported to the liver and excreted through feces and pancreatic secretions.
- Exact mechanisms of zinc-mediated hemolysis and zinc-mediated renal injury are unknown.

Systems Affected

- Hemic: zinc toxicity induces severe intravascular hemolysis.
- Renal/urologic: zinc-mediated renal injury possibly secondary to anemia, hypoxia, or direct tubular injury.
- Gastrointestinal: direct irritation and corrosive effects from presence of metallic object/zinc in stomach or intestinal lining; pancreatitis is also possible.
- Cardiovascular/respiratory: zinc toxicosis sequela includes DIC and cardiopulmonary arrest from severe hypoxia, anemia, and multiple organ failure.
- Hepatobiliary: zinc is transported to the liver and can cause hepatocellular damage.
- Nervous: there is one report of zinc neurotoxicity in a puppy possibly caused by secondary hypoxia and neuronal ischemia.

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SIGNALMENT/HISTORY

- Due to the higher incidence of dietary indiscretion in dogs, they are the most commonly reported species for zinc toxicity, with no sex or age predilections.

Risk Factors/Causes

- Ingestion of zinc-containing objects such as US pennies minted after 1982 (Figs 115.1, 115.2), galvanized metal, plumbing parts, and zinc oxide ointments.



Fig. 115.1. Penny ingested by a beagle puppy. Note that the penny, which was minted after 1982, was degraded significantly by gastric acid.



Fig. 115.2. Coins ingested by a beagle puppy.

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Historical Findings

- Most penny ingestions are not witnessed and the most common clinical signs reported by owners include depression, vomiting, weakness, abdominal pain, icterus, and signs of renal failure.



CLINICAL FEATURES

- Signs of intravascular hemolysis include icterus, weakness, depression, pale mucous membranes, and hematuria/hemoglobinuria.
- Signs of gastrointestinal irritation include vomiting/hematemesis, diarrhea/hematochezia/melena, anorexia, abdominal pain; signs of acute pancreatitis.
- Signs of acute toxic renal failure include PU/PD, oliguria or anuria, vomiting, depression, weakness, and anorexia.



DIFFERENTIAL DIAGNOSIS

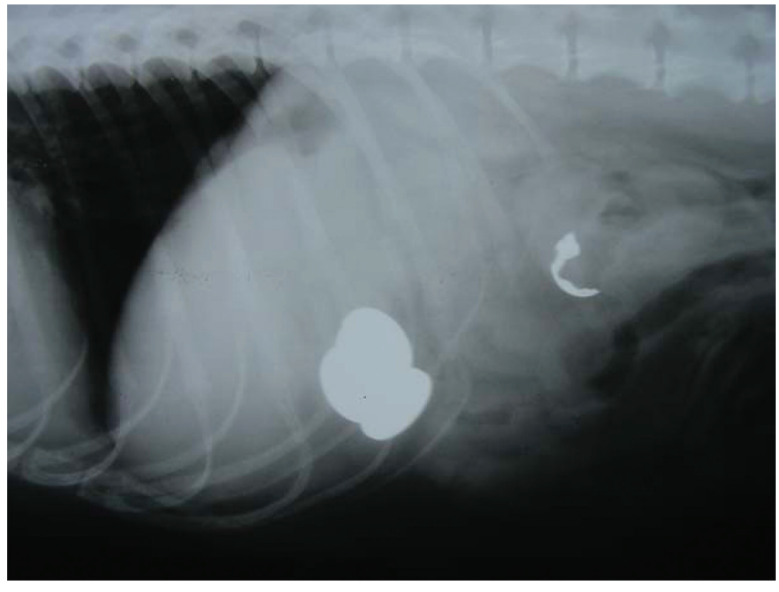
- Differential diagnosis includes any other causes of hemolytic anemia such as immune-mediated hemolytic anemia, tick-borne diseases, allium (chive, garlic, leek, onion, shallot) toxicity, toxicity from other metals (lead, copper), infectious disease (i.e., hemobartonellosis, heartworm disease, leptospirosis, feline leukemia virus infection), neoplasia, and splenic torsion.
- Note that bismuth-containing tablets (Pepto-Bismol) can appear similar to a metallic foreign body on abdominal radiographs.



DIAGNOSTICS

- The diagnosis of zinc toxicity is usually based on clinical signs and radiographic identification of metallic objects in the gastrointestinal tract (Fig. 115.3).
- Biochemical abnormalities.
  - Hemolysis: moderate-to-severe regenerative anemia, bilirubinemia, bilirubinuria, hemoglobinemia, and hemoglobinuria.
  - Renal failure: azotemia, hyposthenuria.
  - RBC abnormalities: nucleated RBC, basophilic stippling, target cells, polychromasia.
  - Evidence of multiple organ failure: elevated ALT, ALP, coagulation abnormalities (prolonged PT, PTT, high FDP, thrombocytopenia, elevated D-dimers).
- Zinc concentrations of serum or urine: dog normal is 0.7–2.0 ppm, a concentration above 10 ppm is diagnostic for zinc toxicity.
- Zinc toxicosis may cause a positive outcome for a Coombs test in the absence of a primary autoimmune disorder.

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■ **Fig. 115.3.** Lateral abdominal radiograph from the beagle puppy, demonstrating coins in the stomach and small intestine.

Pathologic Findings

- Postmortem changes include tubular nephrosis; centrolobar to midzonal hepatic necrosis, vacuolar degeneration; pancreatic inflammation, necrosis, and fibrosis; gastrointestinal hyperemia and serositis, and scattered neuronal shrinkage associated with hypoxia.



THERAPEUTICS

- Goals of therapy include patient stabilization, minimizing zinc absorption via removal of the metallic object, supportive care for subsequent hemolysis and renal failure.
- Initial patient stabilization includes treatment of shock with intravenous crystalloid fluids, supplemental oxygen therapy, blood transfusions, antiemetic and gastroprotectant drugs.
- Removal of the zinc-containing object is critical and may be achieved with emesis in asymptomatic patients if ingestion was recent. Surgical or endoscopic removal may be required, depending on location and duration of the foreign body, as coins often adhere to the gastric mucosal lining.

Drugs of Choice

- H2 receptor blockers help reduce zinc absorption by decreasing gastric acidity (famotidine 0.5 mg/kg PO, SQ, or IV).
- Proton pump inhibitors (omeprazole 1 mg/kg PO q24h, pantoprazole 1 mg/kg IV q24h).

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- Diuresis with a balanced crystalloid solution is indicated to address dehydration, prevent hemoglobinuric nephrosis, and promote renal excretion of zinc at twice maintenance rate.
- Activated charcoal is *not* recommended because it is ineffective in adsorbing elemental zinc.
- Sucralfate (0.5–1.0 g orally 2–3 times daily) after removal of zinc-containing object as a gastroprotectant.
- Antiemetics for gastrointestinal upset (maropitant 1 mg/kg IV or SQ, dolasetron 0.6–1.0 g/kg IV q24h, ondansetron 0.1–0.2 mg/kg IV q6–12h, 0.1–1 mg/kg PO q12h; metoclopramide hydrochloride 0.2–0.4 mg/kg PO, SQ, or IV constant rate infusion at 1–2 mg/kg per day).
- Transfusions: packed RBC transfusions (10 mL/kg) after blood typing, fresh frozen plasma (10 mL/kg) in cases in which pancreatitis or coagulopathy are evident.
- Chelation therapy is controversial as it may increase gastrointestinal absorption and potential for nephrotoxicity.
- Calcium EDTA: 25 mg/kg SQ q6h for 2–5 days.
- Oral penicillamine 35 mg/kg four times daily for 7–14 days.

Surgical Considerations

- Surgical or endoscopic removal of the zinc-containing objects may need to be attempted; stabilize the patient before anesthesia.

COMMENTS

Patient Monitoring

- Monitor blood urea nitrogen, creatine, phosphorus, packed cell volume, urine output, urine specific gravity, and coagulation profiles throughout the course of treatment.
- Monitoring clinical signs of anemia such as respiration rate, heart rate, and energy level may help in deciding if a blood transfusion is necessary.

Prevention/Avoidance

- Inform client about the hazards of ingesting zinc-containing objects and to avoid using zinc oxide ointments.

Possible Complications

- Sequelae include DIC and multiple organ failure with cardiopulmonary arrest.

Expected Course and Prognosis

- Supportive care may be needed for several days to weeks.
- Prognosis depends on the severity of clinical signs and patient response to treatment.

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Abbreviations

ALP: alkaline phosphatase
ALT: alanine transaminase
DIC: disseminated intravascular coagulation
EDTA: ethylenediamine tetraacetic acid
FDP: fibrin degradation products
IM: intramuscular
IV: intravenous
pH: acid–base
PO: by mouth (per os)
PT: prothrombin time
PTT: partial thromboplastin time
PU/PD: polyuria/polydipsia
RBC: red blood cell
SQ: subcutaneous

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