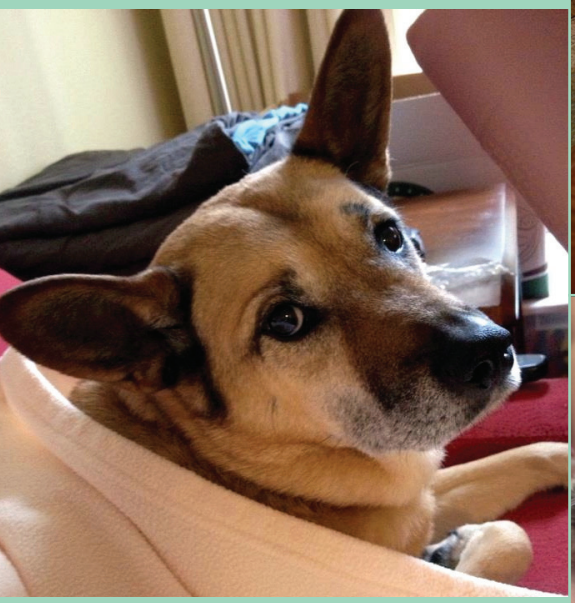
Small Animal Pathology

for Veterinary Technicians



Amy Johnson

with website

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Small Animal Pathology for Veterinary Technicians

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To my family and friends who put up with my absence throughout this process and support me in all my endeavors, no matter how crazy they may sound. Thank you Keith and Cooper.

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To my past animals who inspired my need for greater knowledge and became a part of this project as case studies or images.

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To my students who inspire me and believe in what I am doing for them.

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About the Companion Website

This book is accompanied by a companion website:

www.wiley.com/go/johnsonvettechpath

The website includes:

- Images from the book in PowerPoint for downloading
- Review questions and answers
- Case studies illustrating the process of handling the patient

Introduction

chapter 1

The Veterinary Technician's Role in Pathology

For a veterinary technician, there are certain tasks not allowable by law. These tasks include making a diagnosis, determining a prognosis, prescribing medication, initiating treatment, or performing surgery. Just because a technician cannot make a diagnosis does not mean he or she is not an integral part of the diagnostic team. Understanding pathology is an important part of the veterinary technician's job, meaning it cannot be overlooked.

TECH BOX 1.1: Veterinary technicians play a role as an integral part of the diagnostic team.

Why does the veterinary technician need pathology information? This question has many answers:

- The role of client education is often a task that is the job of the technician. Veterinary technicians will advise clients on the phone and in person on how to best care for their pets.
- It is important to understand disease to prevent the spread of pathogens from patient to patient. It is the role of technicians to make sure they are doing what they can to keep their patients in good health.
- As a technician, there is a need to understand how to appropriately care for the patient. This understanding of the disease process will facilitate patient care.
- An understanding of pathology will aid in protecting clients, co-workers, and the technician themselves from zoonotic diseases.
- A technician who knows the disease process is able to anticipate the veterinarian's needs, expediting patient care.

Technician Duties and Required Skills

Technician duties will include patient care, client education, laboratory diagnostics, assisting the veterinarian, and treatment. It is important to

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note that every veterinarian/clinic/hospital will have different thoughts as to what a technician's duties will be, thus making it important that the technician understands what his or her role is.

Some of the necessary skills involved in dealing with these ill patients include

- Client education and communication skills
  - The ability to speak with owners over the phone and in person.
  - The ability to speak clearly with owners during the intake process and answer questions in terms that are correct but on a level that the client will understand.
  - The ability to update clients on how their animals are doing and progressing.
  - The ability to convey information between the veterinarian and owner.
  - The ability to explain invoices/estimates to clients so there is an understanding of why the procedure and cost are necessary for the treatment of their pet.
  - The ability to discharge a patient and give owners any information needed to continue the care of their animal.
  - The ability to train owners how to medicate or perform treatments that may be necessary once the animal is home.
- Laboratory and other diagnostic skills
  - The ability to properly collect specimens including urine, feces, blood, and tissues.
  - The ability to properly submit and package samples to reference laboratories.
  - The ability to perform a complete blood count (CBC) and other basic hematological procedures.
  - The ability to run blood chemistry machines and enzyme linked immunosorbent assays (ELISA).
  - The ability to collect cytologic specimens, set up slides, and examine slides.
  - The ability to collect samples for bacterial evaluation and set up and read culture and sensitivity tests.
  - The ability to set up, perform, and develop radiographs, ensuring the safety of all persons and animals involved.
  - The ability to prepare and restrain patients for other diagnostic imaging techniques including ultrasound (US), magnetic resonance imaging (MRI), and computed tomography (CT) scans.
  - The ability to prepare the patient, set up and clean equipment, and restrain the patient for endoscopic procedures.
  - The ability to prepare the patient and equipment for other specialized diagnostic procedures.
- Treatment skills
  - The ability to place intravenous catheters (ICVs) in veins including cephalic, lateral saphenous, and jugular veins.
  - The ability to prepare fluid bags and medications.
  - The ability to calculate the patient's fluid rates.
  - The ability to administer medications through routes including injection, oral, and topical.
  - The ability to isolate infectious materials and prevent further spread of contagious diseases.
  - The ability to keep the patient comfortable and in clean quarters.
  - The ability to advocate for the patient and keep his or her best interests first and foremost.
- Other skills
  - The ability to perform dosage calculations and other important veterinary calculations.
  - The ability to induce the patient for surgery, maintain and monitor anesthesia, prepare the patient for surgery, and assist the veterinarian in surgery.
  - The ability to sterilize instruments, prepare surgical packs, and maintain sterility.
  - The ability to restrain patients for examinations and procedures, ensuring the safety of the animal and all persons involved.

- The ability to lift patients on to exam tables, into and out of cages, and help patients ambulate if they are unable to.
- The ability to perform euthanasia or aid in the process.
- The ability to maintain patient records and hospital logs.
- The ability to log and track controlled substances.
- The ability to triage patients and deal with multiple animals.

There are other additional skills and duties that will be discussed with specific pathologies and highlighted by "Technician Duty" boxes.

Diagnosis

The word "diagnosis" literally means "a state of complete knowledge" and is used to label the condition the patient is suffering from. Types of diagnosis include

- A presumptive diagnosis is the identification of the likely cause of disease.
- A definitive diagnosis is the identification of the definite cause of disease; this type of diagnosis involves diagnostic testing.
- A differential diagnosis is a list of possible diseases the patient could have. Testing will aid in ruling diseases out and narrowing the list.

What is involved in a diagnosis and what is the technician's role? Not many patients will present with signs so distinct that the veterinarian knows immediately what disease they have. Achieving a diagnosis takes work and there is a process involved. First a history will need to be taken and a physical examination performed. A problems list will be generated that will allow the veterinarian to form a differential diagnosis. Performing diagnostic testing or imaging will allow for conditions to be crossed off that list. Technicians play a crucial role in this process, and it does not stop there. Once the veterinarian initiates treatment, the technician will provide that treatment. Client communication is necessary throughout the animal's hospitalization, and more client education will be necessary upon the patient's release. What this means is the veterinary technician is a critical part of the whole process.

Immunity

Immunity is the ability of the body to fight off disease and can be categorized in several different ways.

Non-specific immunity/resistance is general protection that does not initiate a response against a specific pathogen. The first line of defense is provided by mucous membranes and skin providing a physical barrier. Innate immunity, including inflammation, fever, antimicrobial proteins, and phagocytes, is the body's second line of defense. Specific immunity/resistance is the body's third line of defense, giving the body the ability to target and destroy specific antigens. Specific immunity involves lymphocytes that produce antibodies and memory cells.

Active immunity is formed when the body is allowed to form its own antibodies against a pathogen. Examples of active immunity include antibodies formed when the body is exposed to a disease or a vaccine. Passive immunity is produced when the body receives preformed antibodies, such as in the instance of colostrum or plasma.

Cellular immunity (cell-mediated immunity) is immunity involving the activation of T cell lymphocytes. These T cells have different functions:

- Cytotoxic T cells have the ability to attach to the antigen and attack it.
- Helper T cells enhance the activities of other immune responses.
- Supressor T cells aid in control of the immune response.
- Memory T cells create a memory of the antigen for a quicker response with the second exposure.

Humoral immunity involves production of antibodies from B cell lymphocytes. B cells transform into plasma cells creating antibodies, which work by neutralizing the pathogen, preventing cell attachment, immobilizing bacteria, and enhancing phagocytosis. Antibodies formed are for specific antigens and initiate memory B cells that create a quicker response in future exposures.

Factors Involved in Infectious Disease

How can two animals come in contact with a disease in their environment and only one of them get sick? The answer involves factors or variables involved with each patient and circumstance. First are host factors, dealing with the patients themselves. Age, nutritional status, health status, medications, immunization status, and stress will all play a role in how well a patient's immune system will protect it. Next are environmental factors, which involve temperature, humidity, and sanitation. Lastly, agent factors involve the micro-organism. Virulence, mode of transmission, and the amount of exposure needed aid in determining how a patient's immune system will react to each pathogen.

Common Terminology Necessary for Understanding Pathology

- *Bacterial translocation*: The movement of bacteria or bacterial products across the intestinal lining to either the lymphatics or peripheral blood circulation.
- *Bacterin*: An immunization against a bacterial agent.
- *Biological vector*: An organism in whose body a micro-organism develops or multiplies prior to entering the definitive host.
- *Carrier*: A living organism that serves as host to an infection yet shows no clinical signs of the disease.
- *Clinical sign*: Objective changes an observer can see or measure in a patient.
- *Contagious infectious disease*: An infectious disease that can be passed from one animal to another.
- *Disease*: Any changes from the state of health disrupting homeostasis.
- *Endemic*: A disease that is present in the community at all times.
- *Fomite*: An inanimate object that transmits a contagious infectious disease.
- *Homeostasis*: The ability of an organism to maintain its internal environment within certain constant ranges.
- *Horizontal disease transmission*: Transmission of disease among unrelated animals; can occur through direct contact or vectors. Horizontal disease transmission occurs when an animal comes in contact with a disease in his or her environment.
- *Incubation period*: The period of time from when a pathogen enters the body until signs of disease occur.
- *Infection*: Invasion and multiplication of a micro-organism in body tissues.
- *Infectious disease*: A disease caused by a micro-organism.
- *Latent infection*: An infection where the individual does not show signs of disease, unless under stressful conditions.
- *Local disease*: A disease that affects a small area or part of the body.
- *Mechanical vector*: An organism that transmits a micro-organism by moving it from one location to another.
- *Morbidity*: A ratio of sick to well in a population; refers to how contagious a disease is.
- *Mortality*: The number of deaths among exposed or infected individuals.
- *Palliative*: Relieving clinical signs/symptoms without curing disease.
- *Pathogen*: An infectious agent or micro-organism.
- *Pathognomonic sign*: A hallmark sign or one that is unique to a particular disease.
- *Pathology*: The study of disease.

- *Prognosis*: The estimate of the likely outcome of disease.
- *Reservoir*: A carrier or alternative host that maintains an organism in the environment.
- *Resistance*: The ability to ward off disease (immune).
- *Subclinical or unapparent infection*: An infection where clinical signs cannot be observed.
- *Susceptibility*: The lack of immunity or vulnerability to disease.
- *Symptom*: Subjective changes not obvious to the observer, requiring the patient to report them.
- *Systemic disease*: A disease that affects a number of organs/tissues or body systems.
- *Vaccine*: An immunization against a viral agent.
- *Vector*: Anything that transmits a contagious infectious disease.
- *Vertical disease transmission*: Transmission of disease from parent to offspring in the period prior to birth or immediately after birth. Examples of vertical disease transmission include transplacental transmission of disease or transmission through colostrum or lactation.
- *Zoonotic disease*: An infectious disease that can be passed from animal to man.

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Canine Infectious Disease

chapter 2

There are numerous infectious agents ubiquitous in the environment with which dogs come into contact. Most of these agents can be fought off by the immune system, but multiple variables will allow that protection to fail (discussed in chapter 1). Vaccines will protect many dogs, and yet patients will still present to veterinary clinics with these infections.

Canine Distemper Virus (CDV) or Hard Pad Disease

Description

Distemper virus is a highly contagious systemic infection caused by an enveloped ribonucleic acid (RNA) virus from the family Paramyxoviridae. As a member of the *Morbillivirus* genus, it is very closely related to human measles virus. Distemper is seen in domestic dogs and ferrets but transmission can be linked to wildlife such as skunks, minks, raccoons, coyotes, wolves, and foxes. It is a fairly labile in the environment, being easily killed by common disinfection methods. Incubation for distemper virus is approximately 2 weeks.

Transmission

- The main transmission route for distemper is through aerosolization. Respiratory secretions contain virus, although all other secretions should be considered contagious. Distemper can be passed from mother to fetus across the placenta.

Clinical Signs

- Highest rate of infection is among young unvaccinated puppies.
- Dogs with distemper may have a fever accompanying the disease.
- Respiratory signs include severe ocular and nasal discharge and pneumonia (Figure 2.1).
- Integumentary signs include pustules on the abdomen and hyperkeratosis of the pads and nose. These tissues produce excess keratin,

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Figure 2.1 Nasal discharge from a dog with distemper virus. (Image courtesy Michael Curran)

causing a waxy hard surface commonly called "hard pad."

- Vomiting and diarrhea are clinical signs associated with the gastrointestinal (GI) tract.
- Dental disorders arise from enamel hypoplasia, as the enamel does not properly form on developing teeth in puppies with the infection (Figure 2.2).
- Seizures are common with distemper. If the dog is exposed to distemper after birth, the seizures may develop during the course of the disease or be delayed 1–3 weeks after recovery from the other clinical signs. These seizures will range from mild to severe. "Chewing gum" seizures and focal seizures in the facial muscles are common.

TECH BOX 2.1: Distemper is one of the most common causes of seizures in puppies less than 6 months old.

- Puppies exposed to distemper prior to birth will develop seizures within the first few weeks of life, while other clinical signs are absent.

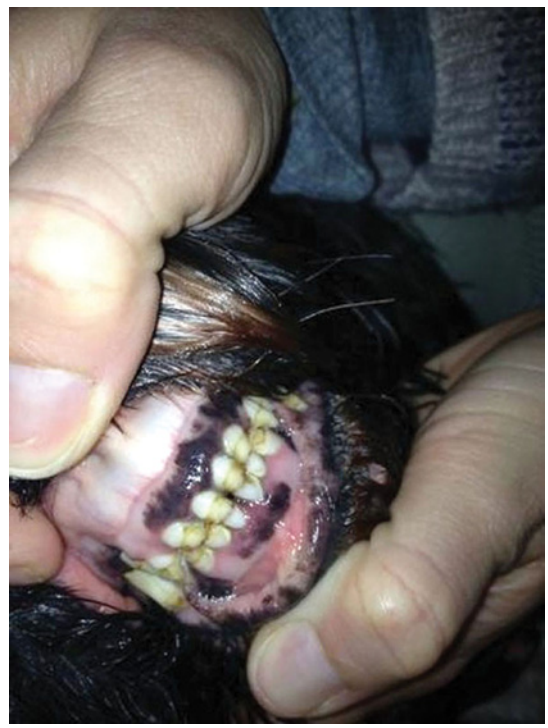


Figure 2.2 Enamel hypoplasia seen as a result of distemper virus. (Image courtesy Shawn Douglass)

Diagnosis

- Distemper is most commonly diagnosed based upon presenting clinical signs, physical exam, and history.
- Radiographs can be used to diagnose pneumonia (Figure 2.3).
- Reference lab testing includes polymerase chain reaction (PCR), antibody titers, and immunofluorescent antibody assay (IFA).
- In-house testing includes distemper antigen test kits and routine laboratory work (Table 2.1), although the lab values are not definitive. Distemper inclusions can be found in the red blood cells (RBCs) and white blood cells (WBCs) of infected patients (Figure 2.4) on a routine blood film.

Treatment

- Treatment is supportive care targeted at the patient's clinical signs.
- Treatment includes intravenous (IV) fluids, correction of electrolyte imbalances, antibiotic drug therapy to prevent secondary bacterial infections, anticonvulsants, and oxygen therapy.
- Even with treatment, the disease will most often be fatal.

Table 2.1 Distemper laboratory work

Morphology changes on blood film	Inclusions found in RBC and WBC: Dark purple Round to oval Inconsistent size
Blood cell count changes	Leukopenia first 3–6 days of infection
PCV/TP	Increase due to hemoconcentration
Blood chemistry	Hypoglycemia due to anorexia and vomiting
Electrolytes	Imbalances due to dehydration and anorexia
Urine changes	Increase in USG due to dehydration

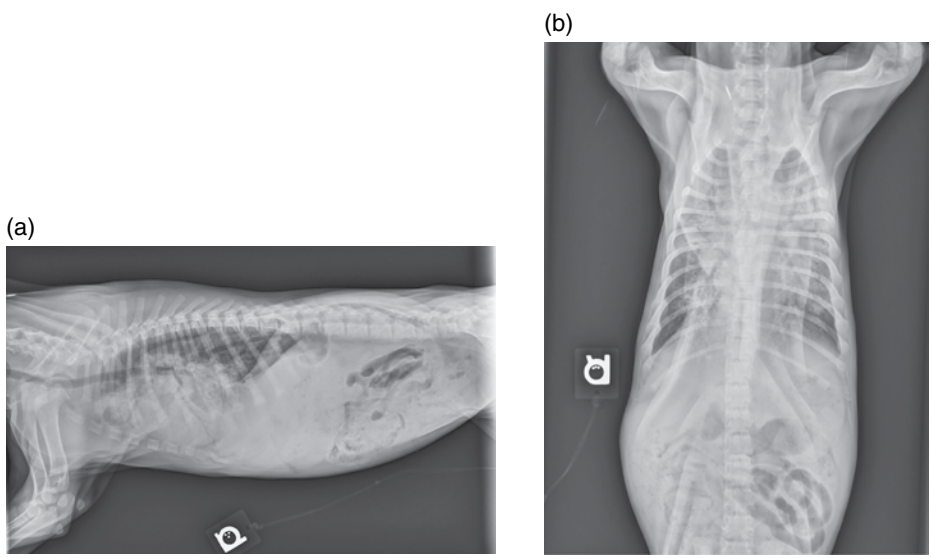
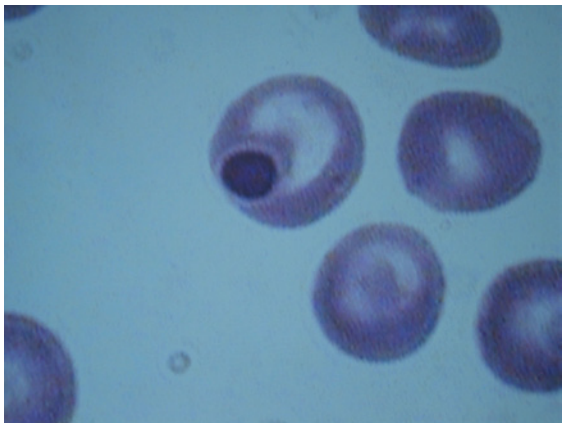


Figure 2.3 Radiograph of a puppy with pneumonia: (a) lateral, (b) ventral/dorsal. (Image courtesy Brandy Sprunger)

(a)



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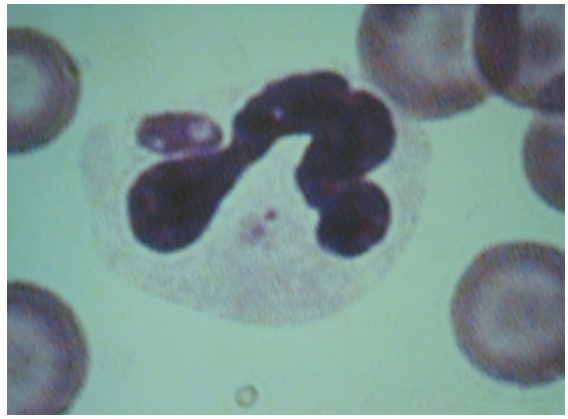

Figure 2.4 Blood film with distemper inclusions in (a) RBC, (b) WBC. Stained in routine hematology stain (Diff Quik). (Image courtesy Tammy Schneider)

Client Education and Technician Tips

- Distemper is one of the leading causes of death in unvaccinated dogs.
- Vaccination, isolation, and sanitation are key in preventing the spread.
- High-risk young puppies can be given human measles vaccine. This offers cross-protection as the antibodies formed will recognize distemper virus but will not interfere with maternal distemper antibodies.
- If a dog survives distemper, he or she may have lifelong problems, including dental and central nervous system (CNS) problems (seizures).

TECH BOX 2.2: With distemper, the long-term prognosis is questionable. Patients may not recover from neurological clinical signs.

- "Old dog encephalopathy" (ODE) is a condition seen in surviving dogs as they age. The virus remains long term in their brain tissue and can cause encephalitis. It is important to note that these dogs are not contagious and will not develop any other signs of distemper. Dogs with ODE will exhibit CNS signs such as seizures, ataxia, and head pressing.

Canine Parvovirus Type 2 (CPV-2)

Description

Canine parvovirus type 2, a highly contagious virus, will cause an acute severe gastroenteritis in dogs. CPV-2 is seen in wild canids as well as domestic dogs. This non-enveloped deoxyribonucleic acid (DNA) virus is from the Parvoviridae family, and although there are many species that are affected by viruses in this family, CPV-2 will not cross species lines. Dogs with parvovirus start to exhibit clinical signs within 4–9 days after exposure. Viruses in the Parvoviridae family are some of the most resistant viruses known. CPV-2 will live in the environment for approximately a year, possibly longer. The virus is resistant to some disinfectants, extreme temperatures, and changes in pH; however, dilute bleach will kill the virus on hard surfaces.

Transmission

- Parvovirus is spread through the feces. Dogs are infected via the fecal-oral route. The virus is spread through direct contact with the infected dog, feces, or though vectors, especially fomites.
- The virus is shed in the feces of infected dogs for up to 3 days prior to onset of clinical signs and up to 3 weeks post-recovery.
- Parvovirus initially replicates in the lymphoid tissue of the oral cavity and pharynx, then spreads to the bloodstream. The virus attacks rapidly dividing tissue or cells, including the bone marrow, lymphopoietic tissue, and intestinal crypt cells.

Clinical Signs

- Common signalment is puppies less than 1 year of age, although the virus cannot be ruled out in older dogs with clinical signs consistent with CPV-2.
- Acute onset of vomiting, diarrhea, anorexia, and lethargy are common presenting clinical signs. Diarrhea is most often hemorrhagic and has a distinct odor to it.
- Fever often accompanies the other clinical signs.
- Some dogs can be asymptomatic carriers of parvovirus.

Diagnosis

- The most common diagnosis is through the use of an in-house ELISA test. This test detects the parvovirus antigen in the feces of infected dogs and is considered definitive (Figure 2.5).
- Reference tests are available but are rarely used due to access to in-house testing.
- Laboratory blood testing may help add to the developing diagnosis but is not definitive if used alone (Table 2.2).

(a) (b)

Figure 2.5 (a) An IDEXX ELISA test and fecal sample for parvo testing. (Image courtesy Amy Johnson and Bel-Rea Institute of Animal Technology) (b) A positive IDEXX ELISA for CPV-2 antigen in the feces. (Image courtesy Hillary Price)

Table 2.2 Parvo laboratory work

Blood cell count changes	Leukopenia, especially lymphopenia and neutropenia
PCV/TP	Increase due to hemoconcentration
Blood chemistry	Hypoglycemia due to vomiting and anorexia
Electrolytes	Imbalances due to dehydration and anorexia
Urine changes	Increase in USG due to dehydration

TECH BOX 2.3: A definitive diagnosis of parvovirus is easily obtained in house. The testing is easily available, fairly inexpensive, and will give the owners and veterinarian a quick diagnosis.

Treatment

- Treatment is supportive and aimed at correcting electrolyte and fluid imbalances, stopping bacterial translocation and septicemia, and controlling clinical signs.
- Often dogs with CPV-2 will be taken off of any oral food, water, or medications until the vomiting has subsided. Many veterinarians advocate parenteral feeding early in treatment. Getting the enterocytes nutrients will speed the patient's recovery. In order to make the early enteral nutrition (EEN) successful, the patient's vomiting must be controlled.
- Parvo puppies present severely dehydrated as a result of vomiting and diarrhea, making rehydration and electrolyte balance a priority. Ideally the patients will receive IV crystalloid fluid therapy, as subcutaneous (SQ) fluids pose a higher risk for infection due to contamination and often cannot keep up with the dog's hydration needs. Once an IV catheter is placed it is important to replace it every 48–72 hours to avoid infection and inflammation.
- Most clinics will have their own "parvo cocktail" used for treatment of the patient. These vary but will often contain a mixture of crystalloid fluid with dextrose, broad-spectrum antibiotics, electrolytes, antiemetics, and analgesics. Some may also include immune-boosting vitamins.
- Dogs with parvo should be hospitalized in an isolation ward. Due to a weakened immune system these dogs are susceptible to secondary infections. Keeping them in the isolation ward protects them from the infections of other hospitalized patients. This isolation also serves to protect the other patients from infection with the highly contagious parvovirus.

Technician Duty Box 2.1

It is important to keep parvo patients and their cages clean and free of urine, feces, and vomit. This can be a difficult task based on the amount of diarrhea excreted, so the veterinary technician must stay on top of monitoring these patients.

Client Education and Technician Tips

- Vaccination, isolation, and sanitation are key in preventing the spread of this virus.

TECH BOX 2.4: Although parvoviruses are very difficult to kill in the environment, dilute bleach will kill the virus on hard surfaces.

- Some breeds have been found to be more susceptible than others. These breeds include Rottweilers, Doberman Pinschers, Pit Bulls, German Shepherds, and Labrador Retrievers. These breeds may require an extra vaccine for full protection from the virus.
- Most dogs presenting to clinics for parvo are puppies, but we must not overlook the fact

that CPV-2 can also be seen in adult dogs. Unvaccinated or inappropriately vaccinated old dogs or dogs with weakened immune systems and dogs with vaccine failures may be at risk for CPV-2.
- Most dogs that develop and survive parvo will be immune to the disease. Owners do not need to worry about the dogs re-infecting themselves when they go home.
- With intensive in-hospital treatment the prognosis for dogs with CPV-2 is good.

Canine Adenovirus Type 1 (CAV-1) or Infectious Canine Hepatitis (ICH)

Description

Infectious canine hepatitis is a multisystemic infection of domesticated dogs as well as wild canids and bears. The infection is caused by a non-enveloped DNA virus from the Adenoviridae family. As a result of the virus lacking an envelope it will survive in the environment for months, especially in cool climates. The virus is susceptible to dilute bleach and many other disinfectants. Infectious canine hepatitis is adenovirus type 1; although it is closely related to canine adenovirus type 2 (a common cause of canine infectious tracheobronchitis), they are two distinct viruses. The incubation period of CAV-1 is 4–9 days.

Transmission

- CAV-1 enters the body through contact with infected urine, feces, or saliva in the environment. Dogs with CAV-1 will shed virus in their urine for up to 6 months.
- Vectors are an important route of transmission, especially urine-contaminated fomites.
- Once in the body, the virus replicates in the tonsils and spreads to associated lymph nodes. The virus will spread via the bloodstream to tissues of the liver, kidney, spleen, lung, and eye.

Clinical Signs

- CAV-1 is most often seen in dogs less than 1 year of age.
- Clinical signs will vary and will range from subclinical infection to acute death.
- Because initial replication occurs in the tonsils, dogs with CAV-1 may have tonsillitis, although this most often goes unnoted by the owners or veterinarians.
- CAV-1 is often accompanied by a fever.
- Hepatitis and liver necrosis will cause hepatoencephalopathy. Hepatoencephalopathy is a condition in which hepatic dysfunction leads to increased ammonia levels in the blood. Ammonia has a toxic effect on the brain, causing clinical signs including seizures, stupor, blindness, ataxia, and head pressing.
- Hepatitis may also cause coagulation dysfunction, as the liver is responsible for production of many of the clotting factors. Patients will present with peticiation, bruising, bloody diarrhea, hematemesis, and other bleeding disorders. This hemorrhage can be severe and may lead to disseminated intravascular coagulopathy (DIC).
- Depending on the extent of the liver damage, patients may present with icterus tissues, serum, and urine (Figure 2.6).

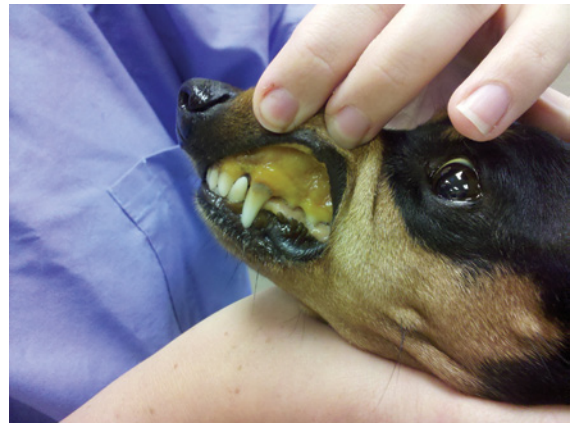

Figure 2.6 Icteric mucous membranes in a dog. (Image courtesy Brandy Sprunger)

- Viral colonization in the kidney can lead to pyelonephritis, resulting in chronic renal disease.
- CAV-1 will often cause ocular disorders, including anterior uveitis and corneal edema. Corneal edema is referred to as "blue eye" because of the bluish opacity seen in the eye. This will spontaneously resolve in most surviving dogs.

Diagnosis

- CAV-1 is most commonly diagnosed based on presenting clinical signs, physical exam, history, and laboratory work-up (Table 2.3).
- Reference lab tests include virus isolation, serum antibody titers, and IFA.
- Histopathology at the time of necropsy reveals intranuclear inclusions in hepatocytes.

Table 2.3 CAV-1 laboratory work

Blood cell count changes	Leukopenia, especially lymphopenia and neutropenia Thrombocytopenia Anemia if hemorrhaging
PCV/TP	Decrease if hemorrhage Decrease in TP due to liver damage
Blood chemistries	Increase in liver enzymes: ALT, AST, alk. phos., GGT Hyperbilirubinemia Increased ammonia Decreased BUN Hypoalbuminemia Decreased clotting factors Hypoglycemia due to anorexia and decreased glycogen production
Bleeding times	Increase due to lack of clotting factors
Urine changes	Hyperbilirubinuria Hematuria if bleeding in urinary tract or pyelonephritis

Treatment

- As with other viruses, supportive care is imperative to the dog's survival.
- Supportive care includes IV fluids with dextrose and electrolytes. SQ fluids are not advisable, especially in dogs with coagulation disorders.
- Due to leukopenia and a compromised immune system, CAV-1 infected dogs are often given broad-spectrum antibiotics to treat and prevent secondary infections.
- Blood transfusions are often performed to improve immune function by providing WBCs and aiding in correction of coagulation dysfunction. The blood transfusion provides clotting factors and platelets if the patient is deficient.

Client Education and Technician Tips

- Vaccination, isolation, and sanitation are key in preventing the spread. Infectious canine hepatitis is not as common now as it once was as a result of vaccination protocols.
- Vaccines for CAV-2 (infectious tracheobronchitis) should be used, as CAV-1 vaccines carry the risk for adverse side effects. Vaccines using CAV-1 were found to cause "blue eye" and renal dysfunction. The two viruses are so closely related that vaccination for CAV-2 will protect the dog from both adenoviruses.

TECH BOX 2.5: Vaccines labeled as DHLPP include CAV-2 and not CAV-1 as the name would suggest.

Canine Infectious Tracheobronchitis or Kennel Cough

Description

Any contagious respiratory disease of dogs that causes coughing can be considered kennel cough. This is a very broad diagnosis

that includes many viruses, bacteria, or fungi. Common viral causes of kennel cough include canine adenovirus type 2 (CAV-2), parainfluenza virus, and canine herpes virus. *Bordetella bronchiseptica* is the bacteria commonly implicated in the infection. It is common to see dogs with dual infections. Most agents responsible for causing kennel cough are fairly labile and will not survive in the environment for long. Incubation periods will vary by organism, yet most are approximately a week.

Transmission

- Transmission occurs through aerosolization of organisms in respiratory secretions.
- Patients are infected when in close proximity with other dogs. This includes not only boarding facilities but shelters, animal hospitals/clinics, or daycare facilities as well.

TECH BOX 2.6: Kennel cough is not just a disease contracted in boarding kennels. Any dog in situations with multiple dog contact is at risk.

Clinical Signs

- Kennel cough can be seen in any age or breed of dog with a history of confinement with other dogs.
- Dogs with kennel cough will have a harsh, dry cough often elicited by gentle tracheal palpation.
- The coughing may be followed by retching and gagging, which may lead the owners to believe the dog is vomiting.
- Most dogs with kennel cough are healthy with the exception of the cough. Generally no fever, anorexia, or other clinical signs of disease are present.
- Some dogs will develop a more severe disease. Stress and age may be factors in determining how severe an infection may be. Dogs in this category will develop a fever, anorexia, depression, purulent nasal discharge, and a change in the cough from a developing pneumonia.

Diagnosis

- Diagnosis is most commonly based on clinical signs, physical exam, and history.
- Definitive diagnosis can be obtained through reference lab testing but is often unnecessary, as testing generally does not alter the treatment or prognosis.

Treatment

- Kennel cough is most often self-limiting; the dog's immune system will clear the infection without medical intervention.

TECH BOX 2.7: Kennel cough is a self-limiting disease, meaning treatment is not always necessary.

- Hospitalization is usually avoided as a result of the contagious nature of the disease.
- Cough suppressants can be prescribed but are often more for the owner's comfort.
- Antibiotics may be used, especially if clinical signs worsen and pneumonia is suspected.

Client Education and Technician Tips

- Immunization, sanitation, and isolation are key to stop the spread of this infection. Yet even immunized dogs can get the disease. There are multiple organisms that will cause kennel cough; unfortunately not all of them can be protected against with immunizations.
- Most boarding facilities, daycares, grooming facilities, and veterinary hospitals will require dogs be immunized with a DA_2LPP and *Bordetella bronchiseptica* prior to being left in their care.

- If coughing is associated with walks, owners should be directed to use a harness that does not put pressure on the trachea the way a collar will. It is also best to recommend the dog not be walked while contagious.

Leptospirosis

Description

Leptospirosis is a bacterial disease of humans and other animals that has been found to be the most widespread zoonotic disease in the world. Although it is found in North America, it is seen more prominently in countries with poor water purification systems and poor water quality. This disease is caused by spirochete bacteria in the genus *Leptospira* (Figure 2.7). There are over 200 recognized serovars of the species *interrogans*. The most clinically significant in North America are *icterohemorrhagiae, canicola, pomona, grippotyphosa, bratislava*, and *autumnalis*. The *Leptospira* bacteria can survive for months in moist soil and water, although survival times are longest in temperate climates. The incubation period is between 2 and 20 days.

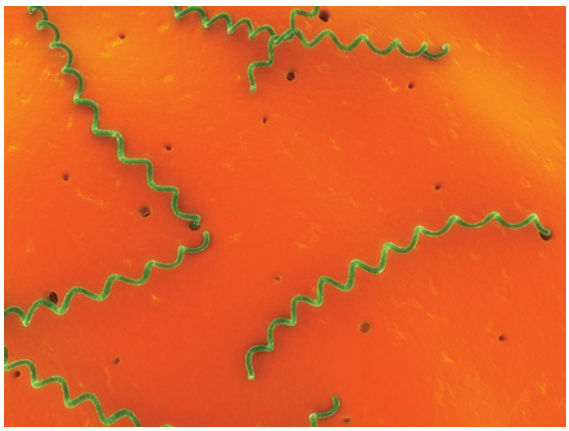

Figure 2.7 Electron microscope image of *Leptospira interrogans*. (Shutterstock image photo courtesy Sebastian Kaulitzki)

TECH BOX 2.8: Leptospirosis is the most widespread zoonotic disease in the world.

Transmission

- Leptospirosis can infect any mammal, although some are more resistant than others. Any breed and age of dog age can become infected.
- Most common transmission route is through infected urine. The bacterium is shed in the urine of infected animals for up to 1 year post-recovery. Urine enters the body through mucous membranes or abraded skin or through ingestion of contaminated food or water sources.
- Fomites play a large role in the spread of leptospirosis, as objects become contaminated with infected urine.
- *Leptospira* can also cross the placenta and can spread through venereal transmission.

Clinical Signs

- Clinical signs of leptospirosis are often nonspecific. Some dogs may be asymptomatic while others will die acutely.
- Acute phase dogs may present with fever, lethargy, anorexia, vomiting and diarrhea (V/D), polyuria and polydipsia (PU/PD), abdominal pain, muscle pain and weakness, and icterus. In this phase the bacteria are spreading through the blood and lymphatic system to all body tissues. During this phase, the body mounts an immune response to the infection and immunity starts to form.
- The convalescent phase, usually lasting 2 weeks, is the time frame when the immune system starts to clear the bacteria from many tissues, although the bacteria will remain in the kidney and potentially the liver. Clinical signs may wax and wane during this phase.
- The carrier or chronic phase occurs, as the bacterium is not eliminated from the kidneys,

liver, or eyes with proper antibiotic therapy. Patients will have clinical signs associated with chronic nephritis, active hepatitis, and uveitis.
- Death from leptospirosis is associated with acute kidney failure and/or liver necrosis.

Diagnosis

- Leptospirosis is a disease that cannot be diagnosed definitively in-house and requires reference lab testing. In-house laboratory diagnostics, however, can help support a developing diagnosis of leptospirosis (Table 2.4).

TECH BOX 2.9: Although no in-house testing for leptospirosis is available, patients should be labeled as "leptospirosis suspects" if the disease is on the rule out list.

Table 2.4 Leptospirosis laboratory work (may vary based on clinical signs)

Blood cell count changes	Leukocytosis Thrombocytopenia
PCV/TP	Increase due to hemoconcentration
Blood chemistries	Increase in liver enzymes: ALT, AST, alk. phos., GGT Hyperbilirubinemia Azotemia Increase in ammonia Hypoalbuminemia Decrease in clotting factors Electrolyte imbalances due to kidney dysfunction
Bleeding times	Increase
Urine changes	Bilirubinuria Proteinuria Glucosuria Increase in cellular casts Decrease in USG

- Reference lab tests include antibody titers, microscopic agglutination test (MAT), and PCR. These tests require blood and urine samples that are collected prior to antibiotic administration. Timing of sample collection often determines if a blood or urine test is best suited for each patient; it is often best to send both samples. The bacteria first appears in the bloodstream but is then cleared from the bloodstream and only found in the urine.

Treatment

- Antibiotics are used in conjunction with supportive therapy with this bacterial infection.
- The initial infection is most commonly treated with doxycycline or penicillin followed by long-term administration of doxycycline to eliminate the carrier state.
- Supportive care is targeted at the patient's presenting clinical signs. Treatments most often target the kidney and include IV fluids with correction of electrolyte and acid/base imbalances.

Client Education and Technician Tips

- Immunization with a bacterin is important in stopping the transmission, although immunized dogs can develop the disease. The bacterin only protects against serovars contained within the injection, with no cross-protection for other serovars seen. Despite this, immunization is still an important part of preventing the disease.
- It is important to isolate other animals from the urine of infected animals. This isolation is different with every clinic but may include collecting urine as the dog urinates, collection of urine through a catheter and closed collection system, or bleaching the areas where the dog urinated. It is important that the owners continue to treat urine from recovered dogs as a biohazard as well.

Technician Duty Box 2.2

Due to the zoonotic implications with leptospirosis, the urine must be disposed of in a manner that avoids possible contamination. This can be accomplished in many different ways, although most clinics will place a urinary catheter and collect the urine in a closed collection set. No matter how it is done, the key is to do so in a way that there is no exposure to the infectious urine.

- Rodents are possible vectors and reservoirs, making rodent control an important part of stopping the disease spread.
- Veterinary professionals are at great risk due to contact with infected dogs and their urine. Good personal hygiene and personal protective equipment is important. One should always wear gloves, a mask, and eye protection when dealing with infected urine and the patient.
- It must be stressed to owners to continue with antibiotic therapy, even if the dog's symptoms have resolved. It is important to follow through with the final phase of therapy to eliminate the carrier state.
- Good sanitation practices are necessary to clean fomites.

Canine Influenza Virus (CIV) or Dog Flu

Description

Canine influenza virus is a novel, highly contagious respiratory disease. It is the only influenza virus recognized to affect dogs. CIV was first reported in racing greyhounds in Florida in 2003 and has quickly spread throughout North America. It has been recognized as a newly emerging pathogen among the dog populations. Unlike the human counterpart, there is no seasonal pattern to CIV. CIV is in the Influenza A family and is labeled as subtype H3N8. It is thought to have originated as a mutation of an equine-associated influenza virus. Currently there is no evidence to support that it is zoonotic or will infect species other than dogs. The virus can survive in the environment for approximately 2 days and on hands and clothing for up to 24 hours. Dogs are most contagious during the incubation period, which lasts 2–4 days.

Transmission

- The main route of transmission is through aerosolization of respiratory secretions.
- Fomites and mechanical vectors play a role in transmission as well.

Clinical Signs

- Most dogs do not have immunity to this virus and will become sick when exposed. Any breed and age of dog is at risk.
- CIV is most commonly an upper respiratory disease causing cough, nasal discharge, mild fever, lethargy, and anorexia. It is often difficult to differentiate CIV from kennel cough.
- Severe cases will develop pneumonia and a high fever, but this is not common.

Diagnosis

- There are no in-house tests at the time of publication, but reference lab tests are available for diagnosis.
- Most cases are diagnosed based on clinical signs and exposure history.

Treatment

- The animal's immune system must clear the virus, making treatment supportive.
- Supportive care will include IV fluids and antibiotics to treat and prevent secondary infections.

Client Education and Technician Tips

- Morbidity of this disease is very high, making isolation and sanitation necessary to stop the spread. Luckily, the mortality is very low (1–5%).
- There is a new vaccine available for CIV. The vaccine doesn't prevent the dog from getting sick, but it will lessen the severity and duration of the illness. It is one of the most expensive canine vaccines on the market, making owners question the need for it in their dogs. It is not necessary for all dogs but recommended for dogs in high-risk categories. These include shelter, rescue, kennel, or pet store dogs as well as dogs staying at kennels and attending classes, daycare, groomers, dog parks, and events.

TECH BOX 2.10: Not all dogs need to be vaccinated for CIV; only those in the high-risk categories need to vaccinated.

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Feline Infectious Disease

chapter 3

Similar to the situation with dogs, the environment is also beset with feline infectious agents. Cats that remain strictly indoors with no access to other cats are the least at risk. The cats at higher risk for infection are indoor/outdoor cats, outdoor only cats, cats that have been in a shelter, or cats that have contact with multiple cats. Some of the infectious agents cats may encounter will offer a good prognosis, while infections with others pathogens may end in death or lifelong infections.

Feline Panleukopenia (FPV), Feline Distemper, Feline Parvo, Feline Infectious Enteritis

Description

Panleukopenia is a highly contagious, possibly fatal disease caused by a DNA non-enveloped virus in the Parvoviridae family. This virus is seen in all felids, and although closely related to canine parvovirus, it does not cause clinical disease in canids. Recent studies, however, have shown the reverse is not true. Canine parvoviruses have been isolated in cats with clinical signs of panleukopenia and healthy cats. Like other parvoviruses, FPV is very resistant in the environment and can survive a year or longer, although on surfaces where it is safe to use bleach the virus can be killed. The incubation period of the virus is 3–4 days post-exposure.

TECH BOX 3.1: Panleukopenia will survive in the environment up to twice as long as canine parvovirus.

Transmission

- Virus particles are shed through the cat's secretions, especially the feces. Cats will become infected oronasally through either

Small Animal Pathology for Veterinary Technicians, First Edition. Amy Johnson.

Companion website: www.wiley.com/go/johnsonvettechpath

direct exposure to infected cats, their secretions, or fomites.
- Vertical disease transmission in utero is also possible.
- FPV will destroy rapidly dividing cells in the bone marrow, lymphoid tissues, and intestinal epithelium. In developing fetuses and neonates the virus will attack cells of the cerebellum and eye.

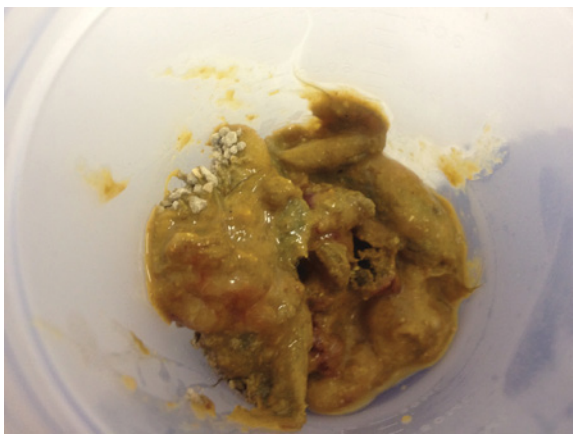
Clinical Signs

- Queens infected early in gestation are likely to experience mummification or resorption of fetuses, abortions, or stillbirths.
- Exposure in later gestation will result in cerebellar hypoplasia (CHP) and optic nerve damage. Cats born from infected queens will be ataxic and have tremors, a wide-based stance, and possible blindness. These are lifelong consequences, although CHP signs can lessen with time as the cat learns to cope.
- Horizontal exposure signs include acute fever, anorexia, lethargy, vomiting possibly followed by diarrhea, weight loss, and abdominal pain. The resulting dehydration and electrolyte imbalance can cause acid/base abnormalities, which can lead to death (Figure 3.1).
- Intestinal crypt cell damage will result in diarrhea, malabsorption, and the risk of bacterial translocation.

Diagnosis

- Diagnosis is commonly based on patient history, physical exam, and presenting clinical signs. High-risk categories include unvaccinated young kittens, feral cats, or cats with outdoor access.
- Although there is no in-house test available for FPV, there is a possibility of cross-reaction with a CPV ELISA antigen test. A positive test is considered a definitive diagnosis for FPV. A negative test result does not necessarily mean the cat is negative. Cats shed smaller amounts of the virus than dogs, and they shed virus infrequently.

(a)



(b)

Figure 3.1 (a) Bloody diarrhea associated with feline panleukopenia virus. (b) Third eyelid visible as seen with severe dehydration. (Images courtesy Amy Johnson and Bel-Rea Institute of Animal Technology)

Table 3.1 Panleukopenia laboratory work

Blood cell count changes	Leukopenia, especially neutropenia and lymphopenia Will rebound in 24–48 hours Left-shift is seen Toxic neutrophils
PCV/TP	Increase due to hemoconcentration
Blood chemistry	Hypoglycemia due to anorexia and vomiting
Electrolytes	Imbalances due to dehydration and anorexia
Urine changes	Increase in USG due to dehydration

- Blood work, including a CBC, blood chemistry profile, and electrolyte panel, may support a developing diagnosis (Table 3.1).
- Reference labs offer other testing methods including PCR, virus isolation, IFA, and antibody titers.

Treatment

- Primary goals of treatment are aimed at restoring fluid and electrolyte balances, preventing secondary infections, controlling nausea, and preventing bacterial translocation.
- Aggressive fluid therapy with electrolytes is essential, along with dextrose, if the cat is hypoglycemic.
- Nutrient support is imperative for the sick and recovering cat, which may require force-feeding, nasogastric tube, or gastroesophageal feeding. The sooner feeding can begin, the better the cat's chances of recovery. EEN within the first 12 hours of hospitalization will improve survival rates as compared to traditional NPO (nothing per os/nothing by mouth) treatment.

Technician Duty Box 3.1

It is important to get panleukopenia patients eating as soon as vomiting has subsided. The veterinary technician will be the one experimenting with good-tasting smelly foods, heating food, force-feeding, or feeding through external devices.

- Antiemetics can be used to control vomiting; this is especially important to control if EEN is to be successful.
- Antibiotics are important in preventing secondary infections and bacterial translocation. Additionally, as important is to keep the cat isolated from other infections.
- In severe cases blood or plasma transfusions may be necessary to restore deficient blood cells and plasma proteins.

Client Education and Technician Tips

- The American Association of Feline Practitioners (AAFP) considers the vaccination a core vaccine and vaccination protocols have proven very successful in prevention of the disease. Other factors in prevention include not feeding kittens outside, especially in warm months, controlling flies and other vectors, and avoiding exposure to unvaccinated cats. FPV is not diagnosed as much as it once was but is still seen in feral populations, and outbreaks in shelters are common.
- Cats can shed the virus for up to 6 weeks post-recovery and can infect other cats during this period.

- Cats who have recovered from FPV have mounted an intense immune response and will not re-infect themselves. However, their contaminated environment can infect other cats, especially kittens. If FPV is in the environment owners should wait at least a year before bringing a new, unvaccinated kitten into the household.
- CHP kittens can have a good quality of life with owners that understand their needs and can provide a safe environment.

Feline Leukemia Virus (FeLV)

Description

FeLV is caused by an enveloped RNA virus from the family Retroviridae, subfamily Oncoviridae. It is often labeled as an oncorna virus; onco for tumor, plus RNA. The virus is one of the major causes of mortality in cats due to severe immunosuppression, anemia, and neoplasms. FeLV is a virus seen in domestic as well as wild felids throughout the world. The virus is labile in the environment, only surviving for several hours. The incubation period of the virus is between 2 and 6 weeks, but acute signs of the disease are rarely detected.

Transmission

- Horizontal transmissions occur through contact with secretions. Saliva is where most of the virus is shed, but other secretions include urine, feces, and tears. Fighting, grooming, and exposure to fomites are all possible transmission methods. Cats that have become persistently infected serve as the reservoir for the infection.
- Vertical transmission occurs in utero or shortly after birth during nursing or grooming.
- Most cats acquire the infection as a kitten. Kittens are at highest risk due to an immature immune system.
- The oronasal route allows the virus into the body, where the virus will spread through lymphoid tissue and blood.
- Once infected, there are a number of possible outcomes. Cats who can mount a sufficient immune response will have a transient viremia, otherwise known as a primary infection. These cats will usually clear the infection from their body within 16 weeks. If the virus is allowed to spread to the bone marrow, it is said to be a secondary or persistent infection. The bone marrow is often referred to as "the point of no return," as the immune system is not able to suppress the virus and most cats will be infected for life.

Clinical Signs

- Acute signs of the disease include fever, lethargy, lymphadenopathy, and blood cell deficiencies.
- Clinical signs associated with persistent infection include anemia (Figure 3.2), immune system suppression, and enteritis.
- Lymphoma and leukemia are common neoplastic disorders seen with FeLV.
- Possible neuropathies may be apparent, including anisocoria (Figure 3.3), blindness, hind limb paralysis, ataxia, seizures, and urinary incontinence.
- Immunosuppression predisposes the cat to concurrent or secondary infections.

Diagnosis

- Laboratory blood work up may reveal hematologic changes but is not definitive for the disease (Table 3.2).
- In-house testing for FeLV is done with ELISA test kits to detect viral antigens. The advantage of this testing method is a quick, easy, relatively inexpensive definitive diagnosis. The FeLV ELISA tests commonly use

(a) (b)

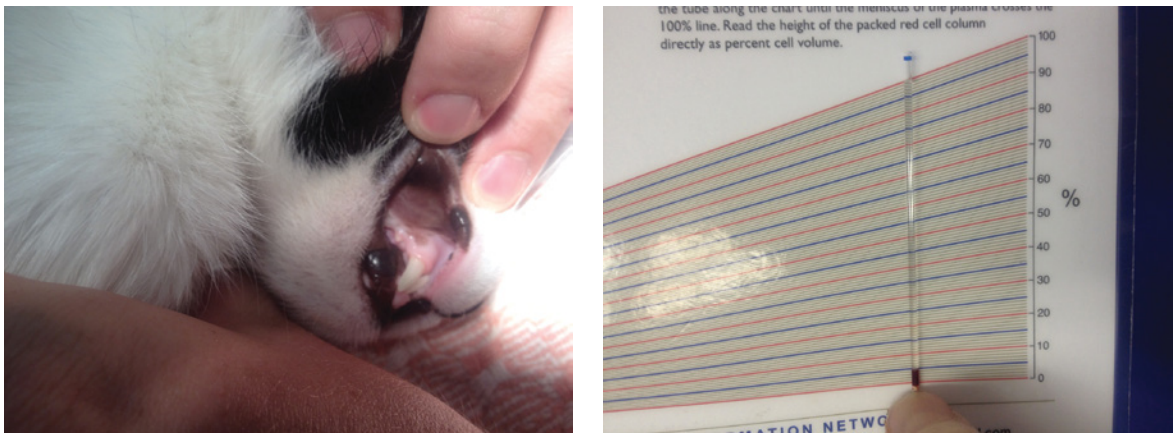


Figure 3.2 (a) Pale mucous membranes associated with anemia. (b) 5% PCV consistent with anemia. (Images courtesy Amy Johnson and Bel-Rea Institute of Animal Technology)

Figure 3.3 Anisocoria associated with central nervous system dysfunction. (Image courtesy Brandy Sprunger)

Table 3.2 FeLV/FIV laboratory work

Blood cell count changes	Leukopenia Anemia
PCV/TP	PCV decrease due to anemia Hemoconcentration due to vomiting/diarrhea
Blood chemistry	Hypoglycemia associated with vomiting/diarrhea/ wasting
Electrolytes	Imbalance due to dehydration and anorexia
Urine changes	Recurrent urinary tract infections

blood, but saliva or eye secretions are also possible samples. These tests will detect the virus but cannot differentiate between an acute or persistent infection. If a cat tests positive using one of these test kits, it is recommended to retest them in 3–4 months to see if the virus has been cleared from the body (Figure 3.4).

- IFA testing is accomplished by submitting a blood sample to the reference lab. This test will detect virus once the virus has made its way to the bone marrow and will establish the infection as a persistent one.

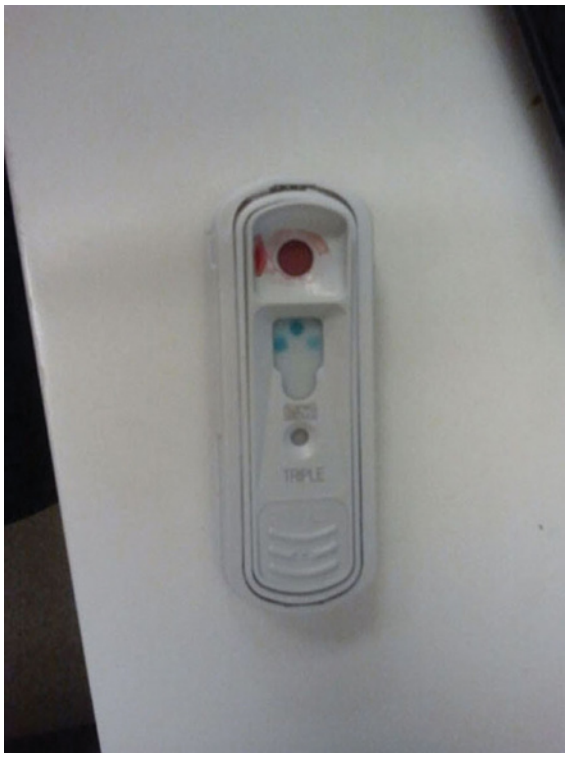


Figure 3.4 Positive IDEXX FeLV/FIV ELISA combo test. (Image courtesy Amy Johnson and Bel-Rea Institute of Animal Technology)

Treatment

- There is no cure for FeLV, especially with most cases being diagnosed at the point of persistent infection.
- Chemotherapy and antiviral therapies are being tested, but virus eradication is virtually impossible, especially in the advanced stages.
- Disease management includes keeping the cat stress free, indoors, and isolated from infections; avoiding raw meat diets; spaying or neutering; and maintaining appropriate vaccine schedules. A watchful eye should be kept on the cat for secondary infections. If any secondary infections arise the treatment should be longer and more aggressive than treatment for an FeLV-negative cat. Veterinary exams should be performed a minimum of twice yearly with routine laboratory work included.

Client Education and Technician Tips

TECH BOX 3.2: Client education is important with FeLV and FIV disease management. Client compliance is crucial.

- Methods for prevention of disease transmission include routine disinfection, frequent hand washing, avoiding the use of multidose vials, and testing all blood donors. It is also helpful to eliminate as many fomites as possible by using disposable clinic items whenever possible.
- Isolation of positive cats is important. Not only will this prevent the spread of the disease but it will also protect their fragile immune systems from secondary infections. When hospitalizing these cats they should not be kept in an isolation ward for fear of infecting them with diseases from the ward.
- Although the vaccine for FeLV is considered a non-core vaccine by the AAFP, vaccination of high-risk cats is recommended. Cats that should be vaccinated include those with access to the outdoors, feral cats, negative cats living in a household with infected cats, or cats with unknown infection status. Furthermore, cats in catteries, shelters, and multicat households are additionally at high risk. There are risks associated with the vaccine and these need to be discussed with the owner who is deciding whether to vaccinate or not. Although the possibility of sarcoma development is low, there has been an association made with the vaccine; this is a risk that needs to be discussed with owners.

TECH BOX 3.3: Cats with both stages of FeLV infections are immunocompromised and contagious.

- A positive test result does not mean euthanasia. After a positive test the cat may fight off the disease, and even if infection is persistent these cats can be managed and have a good-quality life for up to several years or more.
- Although there is no evidence of zoonosis, immune-compromised individuals are not recommended to own a positive cat due to the possibility of zoonotic secondary infections.

Feline Immunodeficiency Virus (FIV) or Feline AIDS

Description

Like FeLV, FIV is one of the leading causes of death in cats worldwide. The two viruses are closely related, with FIV being caused by a non-enveloped RNA lentivirus in the Retroviridae family. The virus will cause bone marrow suppression and immunodeficiency. Unlike FeLV, FIV is a virus the cat does not have the possibility of fighting off. This fatal infection is a lifelong disease. The virus, seen in domestic cats and a few wild felids, has an incubation period of months or even years and a long latent phase. FIV is a labile virus with survivability in the environment of only a couple of hours.

Transmission

- The most common transmission route is through saliva and bite wound inoculation. Once the virus enters the cat it reproduces in T-lymphocytes and spreads to other WBCs, lymph nodes, salivary gland cells, and CNS tissues.
- Mother-to-kitten transmission is rare but possible. Transmission can happen through the birth canal or nursing from the infected queen's milk.
- Other possible transmission routes include blood transfusion and sexual contact, although these methods of transmission are rare.
- Fomite transmission is unlikely and cats with casual contact have little risk of transmitting the disease.
- Infected cats will be positive for life, becoming the reservoir for the virus. Older males, feral cats, and free-roaming cats are at the highest risk for infection and transmitting the disease.

Clinical Signs

- There are three stages or phases of the disease. Clinical signs will vary depending on the stage of the cat.
- The acute stage occurs 4–6 weeks post-exposure but often goes unnoticed. Signs include lymphadenopathy, bacterial infections (often of the skin or gastrointestinal tract), fever, lymphopenia, and neutropenia. During this stage the virus is spreading through T-lymphocytes to all lymph nodes.
- During the latent stage of the disease most cats will recover from initial acute signs and will appear healthy, although a persistent lymphadenopathy is possible. This stage can last for months to years.
- The chronic stage is associated with severe immunosuppression, resulting in secondary infections of the respiratory tract, urinary tract, gastrointestinal tract, and skin. Clinical signs include gingivitis, stomatitis (Figure 3.5), diarrhea, wasting, and anemia.

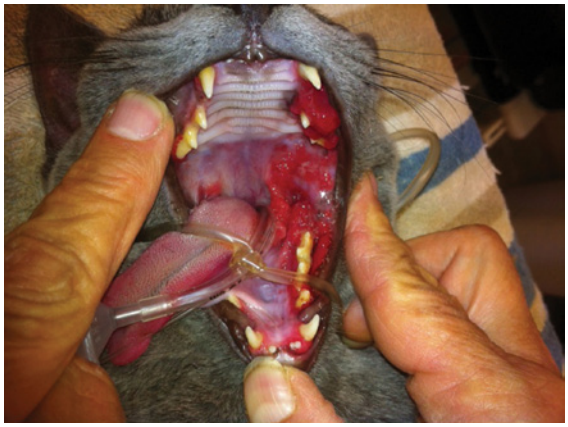


Figure 3.5 Severe stomatitis as seen with FIV. (Image courtesy Deanna Roberts)

- Encephalopathy can be seen in cats with the virus, especially kittens. CNS signs include seizures, circling, pacing, and aggression.
- FIV infections may also cause ocular dysfunction. Anterior uveitis, retinal hemorrhage or deterioration, and glaucoma may be seen.

Diagnosis

- Clinical signs, physical exam, and history in the advanced stage of the disease along with hematological and seriological work-up may help in diagnosis but are not considered definitive. (See Table 3.2)
- Common in-house ELISA tests will help with diagnosis. However, the shortfall of these tests is testing for antibodies. False positives are possible in kittens born to infected queens and vaccinated cats. Positive kittens should be retested after maternal antibodies are no longer circulating in the kitten's bloodstream (Figure 3.4).
- Reference lab tests include IFA, PCR, and western blot.
- Antibodies can take up to 12 weeks after the initial infection to form, and therefore negative cats should be retested up to 60 days after the negative test result.
- Cats with a very advanced form of the disease may test negative due to a lack of antibody formation.

Treatment

- There is no cure for FIV once the cat has become infected.
- Treatment is aimed at protecting the immune system. Keeping the cat stress free, indoors, and on a healthy diet are essential. Positive cats should be spayed or neutered and the appropriate vaccine schedules should be maintained. Secondary infections need to be closely monitored and may require antibiotic therapy. Veterinary exams should be performed a minimum of twice yearly with routine laboratory work.
- Currently there is little evidence to show antiviral drugs work on fighting the disease.
- Severe cases of recurrent stomatitis may require full mouth extractions.

Client Education and Technician Tips

- The best prevention for the disease is to keep cats indoors and away from positive cats. Owners will want to ensure new additions are tested prior to bringing them into a multiple cat household or cattery.
- There is a non-core vaccine available for FIV, but it is rarely used because it destroys the usefulness of testing.

Feline Infectious Peritonitis (FIP or FIPV)

Description

Feline infectious peritonitis is caused by a virus in the Coronavirus family and is highly fatal. Worldwide, FIP is seen more prominently in domestic cats and less in wild felids. Although

many cats are infected with coronaviruses, few will develop FIP. The disease is thought to occur due to a mutation from an enteric coronavirus or an abnormal immune response in the cat. Virulence will vary among strains of the disease. Survivability of the virus in the environment is 4–6 weeks, although this enveloped virus is easily killed by normal disinfection means.

Transmission

- Most often the virus enters the cat through ingestion or inhalation. The virus is shed in feces and saliva and spread through cat-to-cat contact or fomites.
- The virus starts replication in the intestinal epithelium and is then transported through WBCs to multiple body systems. Infected organs include the liver, spleen, kidney, brain, and lymph nodes.
- An extreme inflammatory process occurs within the blood vessels.

Clinical Signs

- Cats of all ages are at risk, but the highest prevalence of the disease is seen in cats less than 2 years of age. The disease is more common in purebred cats that come from catteries, with some breeds more susceptible than others.
- The acute disease may be asymptomatic, or mild signs of disease may occur including fever, respiratory disease, and diarrhea. Very few of these cats will develop clinical FIP weeks to years after exposure.
- The cat's immune response plays a role in the development of the disease and clinical signs. There are two forms of FIP: the effusive (wet) form and the non-effusive (dry) form. It is possible for cats to present with clinical signs from both forms.
- The effusive form develops more rapidly than the non-effusive form. Severe vasculitis causes leakage of plasma and plasma proteins into the body's cavities; fluid collects in the abdomen and pleural cavity. The cat will present with signs of a distended abdomen or dyspnea due to ascites (Figure 3.6).

TECH BOX 3.4: The effusive form of FIP progresses quicker and is diagnosed more frequently than the non-effusive form.

(a) (b)

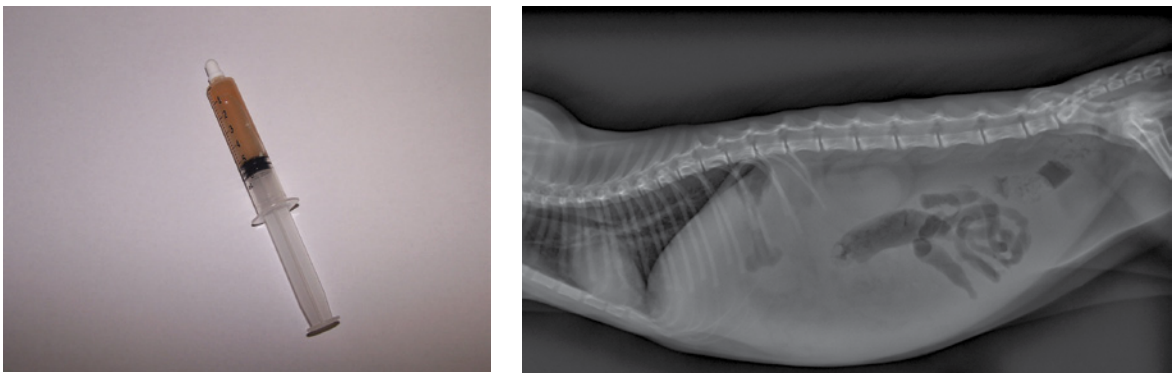


Figure 3.6 (a) Fluid from abdominocentesis on FIP-positive cat. (Image courtesy Amy Johnson and Bel-Rea Institute of Animal Technology) (b) Radiography of FIP cat with fluid in abdomen. (Image courtesy Kelly Melhorn)

- The non-effusive form of FIP tends to be more chronic, with non-specific clinical signs such as fever, lethargy, anorexia, and weight loss; ocular and CNS involvement are also possible. Furthermore, anterior uveitis, retinal hemorrhage, blindness, and seizures can be seen. With some dry forms only eye and brain disease will be noticed.
- Pyogranulomatous lesions develop in the abdomen because the immune system attempts to wall off the virus. These masses can obstruct gastrointestinal function.
- Adhesions between the liver and diaphragm, intestinal bowel loops, or other intestinal organ may develop. Nodules on internal organs or areas of focal necrosis may also be seen.

Diagnosis

- There is no reliable definitive test for FIP. The problem is the tests cannot differentiate between enteric coronaviruses and FIP antigen, or differentiate between the antibodies formed. A positive antibody titer or PCR does not mean the cat has clinical FIP. Often FIP is diagnosed based on presenting clinical signs, physical exam, and history and when all other differential diagnoses are ruled out.
- With the effusive form, fluid analysis can be performed. Fluid collected via abdominocentesis or thoracocentesis will be a viscous, straw- to yellow-colored fluid. The fluid may contain exudate that is white/gray in color. The fluid will have an increased specific gravity and protein level with mixed inflammatory cells (Figure 3.6).
- Laboratory work-ups are not definitive but will help with a developing diagnosis and may be helpful with the non-effusive form (Table 3.3).
- Exploratory surgery or necropsy with biopsy of pyogranulomatous lesions can diagnose FIP.

Table 3.3 FIP laboratory work

Blood cell count changes	Leukocytosis, especially neutrophils Non-regenerative anemia
PCV/TP	Increase in TP Decrease in PCV due to anemia
Blood chemistries	Liver, kidney, and pancreas dysfunction
Fluid analysis (effusive)	Sterile Viscous Straw to yellow in color Increase in specific gravity (1.015–1.050) Increase TP (5–12 g/dl) Mixed inflammatory cells

Treatment

- There is no cure for this highly fatal disease. The effusive form progresses more quickly than the non-effusive, with most cats dying within weeks to months after the onset of clinical signs. Cases of non-effusive are more insidious, progressing slowly over months to years.
- Anti-inflammatory drugs, antibiotics, and immunosuppressive therapy will make the cat more comfortable and possibly extend survival time slightly.
- Supportive therapy is directed at making the cat more comfortable. Draining fluid from the abdomen or thorax can provide temporary relief, but the fluid will accumulate again. Good nutrition, blood transfusions, and fluid therapy are also used supportively.
- Euthanasia should be considered when the cat's quality of life diminishes.

Client Education and Technician Tips

- There is a preventative vaccine for FIP, but it is rarely used due to its lack of effectiveness. The vaccination may be used in high-risk situations such as cats in catteries, shelters, or outdoor exposure.
- In multiple cat households and catteries sanitation and isolation of sick cats are important steps in prevention.

TECH BOX 3.5: FIP is a disease of purebred cats and catteries, although it can be diagnosed in domestic breed cats as well.

Feline Upper Respiratory Tract Infections

Description

Upper respiratory tract disease is a very broad classification of infections that cause ocular and or nasal discharge and sneezing in cats. There are multiple causative agents with dual infections being common. The two major viral causes are feline viral rhinotracheitis (FVR), a virus in the Herpesviridae family (FHV-1), and calicivirus (FCV), family Caliciviridae. Possible bacterial causes include *Chlamydophila felis* (chlamydia) and *Mycoplasma*. Survivability in the environment varies with the agent but most are typically fragile, lasting a few days at maximum. Incubation periods also vary between 2 and 10 days.

Transmission

- Unlike canine respiratory infections where the main route of transmission is aerosolization, feline respiratory diseases spread mainly through fomites. Although aerosolization of respiratory secretions is implicated in the spread of the disease, direct cat-to-cat contact is also a factor.

TECH BOX 3.6: The main route of transmission for feline upper respiratory infections is fomites.

- Many cats become carriers of respiratory disease for possibly years after recovery and act as reservoirs for the infection. Latent infections are also possible.
- These infections are common in crowded circumstances, such as shelters, catteries, or multiple cat households.

Clinical Signs

- Respiratory signs include nasal discharge, congestion, sneezing, lethargy, and fever (Figure 3.7).
- Typical signs of upper respiratory disease often include the eye in addition to the respiratory system. Ocular discharge, ulcerations, and conjunctivitis can be seen.
- Anorexia is common in these sick cats for two reasons: first, cats that cannot smell their food are unlikely to eat; second, oral ulcerations are also commonly seen with respiratory infections, especially FCV, and the pain associated with these ulcerations may contribute to anorexia. Cats with oral ulcerations may present with mouth open or hypersalivation (Figure 3.8).
- Severe cases can present with dyspnea, cyanosis, and open mouth breathing.
- Many cats will deal with chronic intermittent signs.

Diagnosis

- Most upper respiratory infections are diagnosed based on presenting clinical signs, physical exam, and history, although it can be difficult to differentiate between causative agents based solely on clinical signs.
- Confirmatory tests are available through reference labs but rarely used.

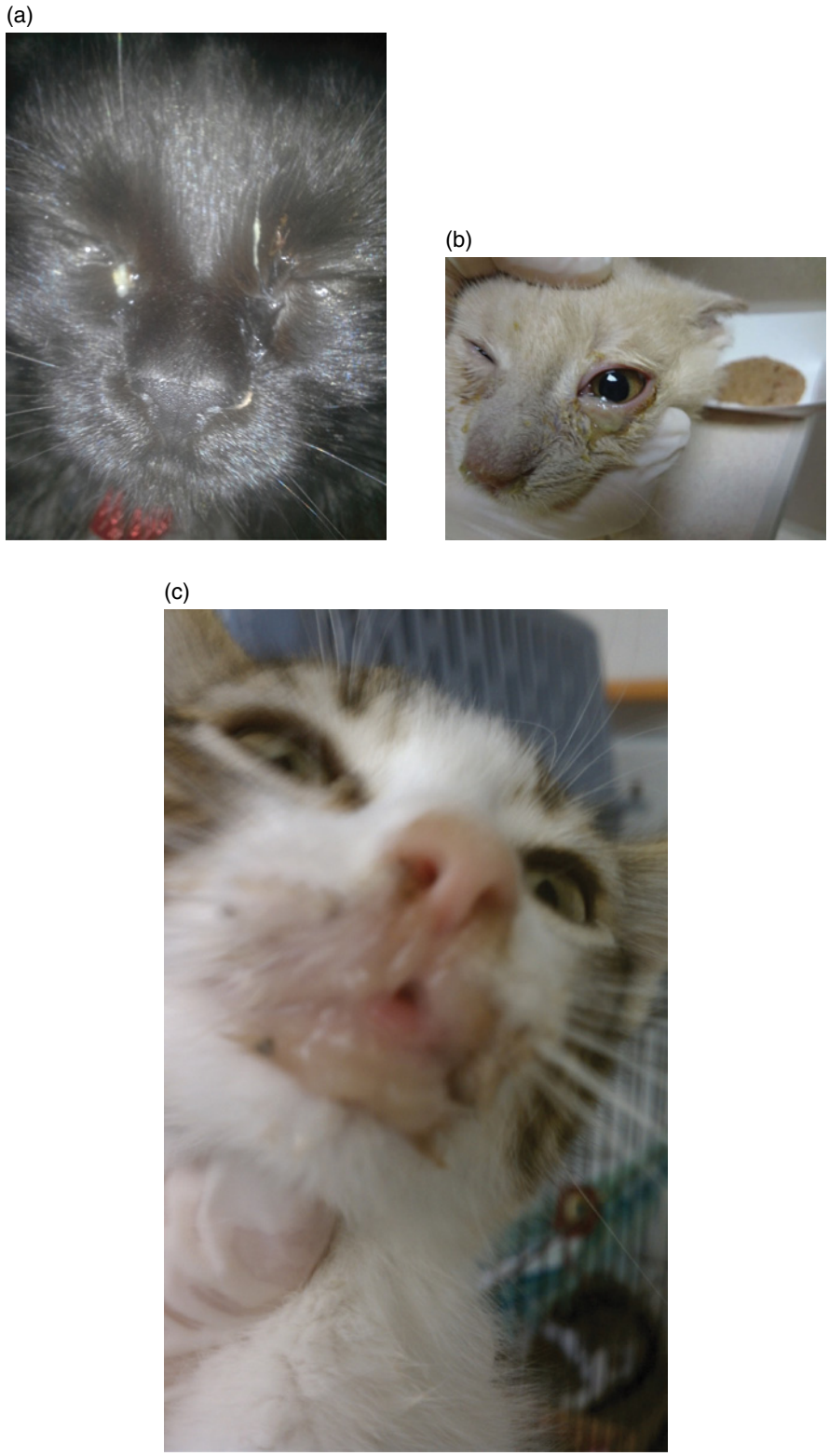


Figure 3.7 (a) Feline with FVR. (Image courtesy Kaylee John) (b) Cat with mucopurulent nasal and ocular discharge from upper respiratory infection. (Image courtesy Deanna Roberts) (c) Kitten with severe upper respiratory infection. (Image courtesy Ashlyn Witte)

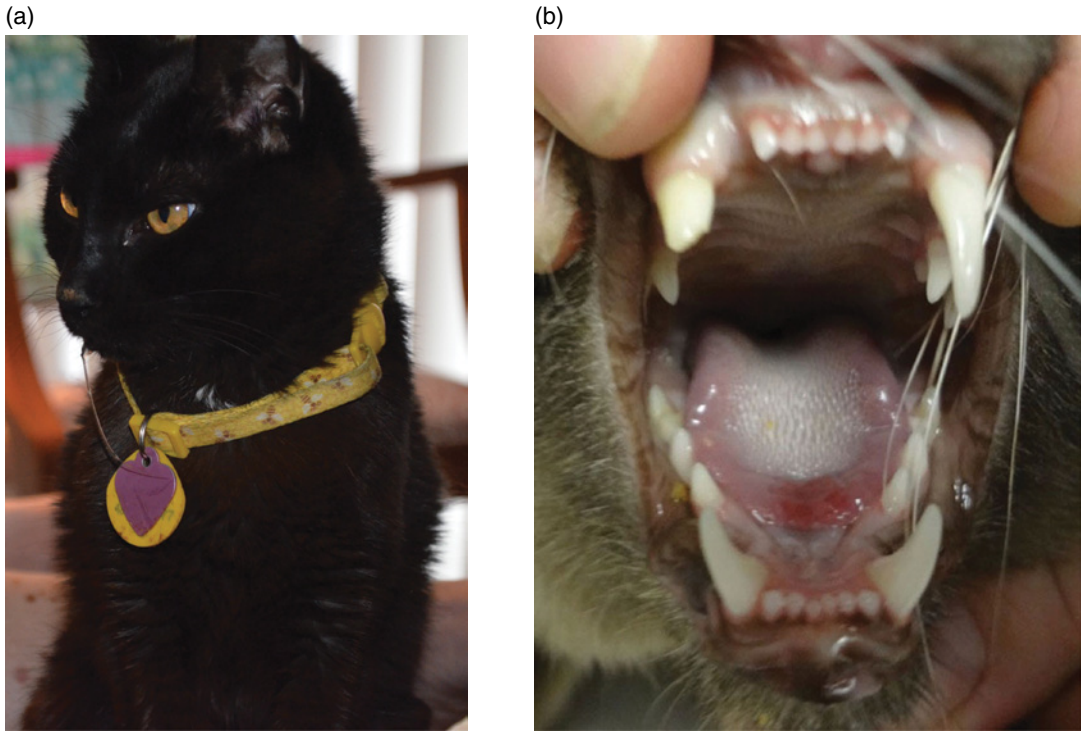


Figure 3.8 (a) FCV oral ulcer causing hypersalivation. (Image courtesy Amy McBurnie) (b) Feline oral ulceration as a result of upper respiratory infection. (Image courtesy Deanna Roberts)

- Fluorescein staining techniques can be used to confirm the presence of ocular ulceration; FVR ulcerations tend to have a distinctive dendritic appearance. Chlamydia is suspected in cases with ocular involvement in the absence of other respiratory signs.

Treatment

- Prognosis is good except in neonatal and geriatric patients.
- Oxygen therapy is required in severe cases.
- Supportive care measures include fluids to correct dehydration, antibiotics to treat or prevent secondary infections, cleaning discharge from eyes and nose, relieving congestion, and application of eye ointments. Good nutrition is imperative and a patient with anorexia may require force-feeding or placement of a nasogastric tube.
- The amino acid supplement L-lysine has become popular to help suppress clinical signs of herpes virus infections. The supplement has been used long term for chronically infected cats or at the time of outbreak with latent infections. The evidence is mixed, making this is an area of debate among veterinarians.

Technician Duty Box 3.2

In order to keep patients with upper respiratory infections comfortable and breathing as easily as possible, the mucus needs to be kept clear from their nose and eyes. This can be accomplished with warm water compresses and gentle wiping.

Client Education and Technician Tips

- Vaccinations are available for FVR and FCV and are considered part of the core vaccination series by the AAFP. A bacterin for *C. felis* is available. It is not considered a core vaccine but may be used in high-risk cats, such as those in multiple cat households or in environments where infections have been diagnosed.
- Other preventative measures include good ventilation, good sanitation, isolation of sick cats, and elimination of fomites when possible.
- *C. felis* infections can be zoonotic, causing conjunctivitis. Fastidious personal hygiene methods are necessary to prevent zoonotic infections.
- A novel mutation of calicivirus, virulent systemic calicivirus (VS-FCV), has been noted and is now contained in certain vaccines. Respiratory signs associated with VS calicivirus are more severe than the original virus, along with vasculitis, edema of face and lower extremities, alopecia, and ulcerative dermatitis. Pneumonia, hepatic necrosis, petechiation, and hemorrhage may also be seen. VS calicivirus carries a high morbidity and mortality rate and is currently diagnosed more frequently in adult cats than younger kittens.

Toxoplasmosis

Description

Toxoplasmosis is an infection caused by *Toxoplasma gondii*, a zoonotic protozoa seen worldwide. Felids, both wild and domestic, are the only definitive hosts of this parasite. This means that although other species will become infected, cats are the only ones that will shed infected oocysts in their feces. Cats rarely have clinical disease as a result of the protozoan, yet are important carriers of the disease with zoonotic risk to humans, especially pregnant women and people with severely immunocompromised conditions. *T. gondii* will cause congenital birth defects in babies born from mothers infected during pregnancy.

TECH BOX 3.7: Toxoplasmosis is one of the top parasitic diseases seen in animals and humans. Despite its high incidence rate, it rarely causes clinical disease in any species.

Transmission

- *T. gondii* is transmitted through consumption of tissue cysts in infected meat, dairy products, protozoan oocysts in cat feces, or transplacental transfer from mother to fetus.
- Cats become infected through consumption of uncooked meat, usually bird or rodent tissues, with oocysts later (3–10 days) released in their feces. These oocycts become infective a couple of days after entering the environment and will survive for several months to a year in the environment.
- In some cats the protozoa will translocate through the intestinal walls, making its way to other body tissues where it will cause tissue cysts and tissue damage.

Clinical Signs

- In cats infections rarely cause clinical disease. In immunocompromised cats or neonates clinical signs can be associated with enteritis, myocarditis, pneumonia, hepatic necrosis, lymphadenopathy, and encephalitis. Clinical signs include anorexia, lethargy, fever, diarrhea, dyspnea, cough, icterus, seizure, and possible death.
- Humans infected with *T. gondii* will show clinical signs involved with blindness, deafness, respiratory defects, and CNS disorders. In babies born with birth defects these may not be evident at birth but may manifest weeks, months, or years later.

Diagnosis

- Clinical signs of a toxoplasmosis infection are very vague and generic, making them not a factor in diagnosis.

- A *T. gondii* antibody titer test is available through reference labs in both human and veterinary medicine. Pregnant women can have their cats screened to ease their mind. Cats testing positive for antibodies without current infection are not likely at risk for transmitting the disease; negative cats should be handled with more caution. Women can also be screened. A positive antibody result without current infection means there has been a past infection and women are not at risk of transmitting the infection to their fetus.
- Tissues collected at the point of necropsy can be used for histopathology. Definitive diagnosis is determined with pathological tissue changes consistent with *T. gondii* and presence of the tachyzoite stage of the parasite in tissues.
- The feces of cats can be evaluated, looking for oocysts; yet this is often unreliable, as these look similar to other parasites.

Treatment

- In cats treatment is rarely necessary. If treatment is needed a combination of antibiotic and antiparasitic drug therapies will be used.
- Humans need to consult their physician for advice on treatment and supportive care options.

Client Education and Technician Tips

- Cats only transmit oocysts with their first exposure; subsequent exposures do not pose a threat, making the cats infective only once in their lifetime and only for a couple of days.
- Strictly indoor cats are at little risk of the disease, as they are not hunting the prey that transmits the disease.
- The feces of the cat is the only secretion that is infective. Pregnant women will not get the infection from casual contact with cats; it takes contact with the feces.
- Pregnant or immunocompromised individuals should avoid cleaning litter boxes or handling the feces of cats. If this cannot be avoided gloves should be worn, litter boxes should be cleaned daily, the boxes should be sanitized with bleach and/or boiling water, and hands should be washed immediately once the gloves come off.
- In North America people are more at risk for the infection by eating undercooked meat or drinking or eating unpasteurized dairy products or raw unwashed vegetables than directly from a cat.
- Other measures taken to protect humans from infection include keeping cats out of gardens and children's sand boxes, wearing gloves when gardening, and keeping cats indoors and away from wild prey.

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Rabies

chapter 4

Rabies Virus

Rabies is one of the more important diseases encountered in veterinary clinics. Rabies has a legally required vaccine and is zoonotic and fatal if not treated. Clients will often have questions about the disease, its transmission, and the legalities that surround the vaccine. Many of the misconceptions that surround the virus could easily be solved with public education, in which the veterinary technician will play a part. It is also vital that veterinary personnel know how to protect themselves and others from the infection.

Description

Rabies is a disease fatal to all mammals, caused by bullet-shaped RNA lyssaviruses in the Rhabdoviridae family. Rabies viruses are identified by variants specific to each reservoir species, although the variants can infect any species. Most human exposures in North America have been linked to bat variants (Figure 4.1). Reservoir species vary throughout the world. In North America bats, raccoons, wild canids, and skunks are implicated in the spread of most of the disease.

Saliva of infected species carries the virus, and it is the exposure to the saliva that results in infection. Once the virus enters the body it travels through peripheral nerves to the spinal cord and eventually to the brain. When in the brain the virus travels along the peripheral nerves back to the salivary glands. Once in the brain the virus will cause encephalitis and dysfunction of vital brain centers. The incubation period varies by species and can be between 1 week and 1 year in extreme cases.

Transmission

- The virus is spread through saliva, salivary glands, and nervous system tissues.
- Although bite exposures are most common, there are also non-bite methods of virus transmission. The virus can spread through saliva or nervous system tissue and come into contact with scratches, abrasions, open wounds, or mucous membranes. Although

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Figure 4.1 Rabid bat. (Image courtesy Kristin Hebertson)

rare, virus particles can potentially be aerosolized.
- Rabies virus will survive for up to 2 hours in dried saliva and 24–48 hours in nervous tissue at room temperature.
- The virus is not spread through the urine, feces, blood, skunk spray, or milk of the infected animal.

Clinical Signs

- Although there is variation from species to species, most rabies cases exhibit CNS dysfunction, behavioral changes, and progression to paralysis. There are three stages of the virus: prodromal, excitative/furious, and paralytic/dumb.
- During the prodromal stage of the disease animals will show the first signs of vague behavioral changes. The pupils will dilate and the nictitating membrane will become visible. Owners of these animals will know there is something wrong, with animals commonly labeled "Ain't doing right" or "ADR."
- During the furious (also called excitative) stage of the disease the animal becomes photophobic and hypersensitive to external stimuli and sounds. He or she becomes very aggressive and tends to roam. Animals in this stage will lose their fear of other animals and humans.
- The paralytic (also called dumb) stage occurs as the paralysis sets in. The animal may be ataxic, have an altered gait, and appear to be in a stupor. Paralysis of the masseter and throat muscles will drop the jaw of the animal and cause hypersalivation and the inability to swallow, making the animal look like it is choking (Figure 4.2).
- As a result of the progressive paralysis, death will occur within 7 days of exposure. Death is seen within a few hours of the initial paralysis onset.

Diagnosis

TECH BOX 4.1: You cannot accurately or legally test for rabies in a live animal patient. Diagnosis requires natural death or euthanasia.

- Animals that are to be euthanized need to be euthanized in a manner that leaves the brain intact for testing.
- The entire head should be removed and submitted to a laboratory specified by the state for rabies testing. Samples should never be frozen or chemically preserved, as this will interfere with the testing procedure. Suspect heads should be double bagged and placed in a leak-proof container. Shipping will be done

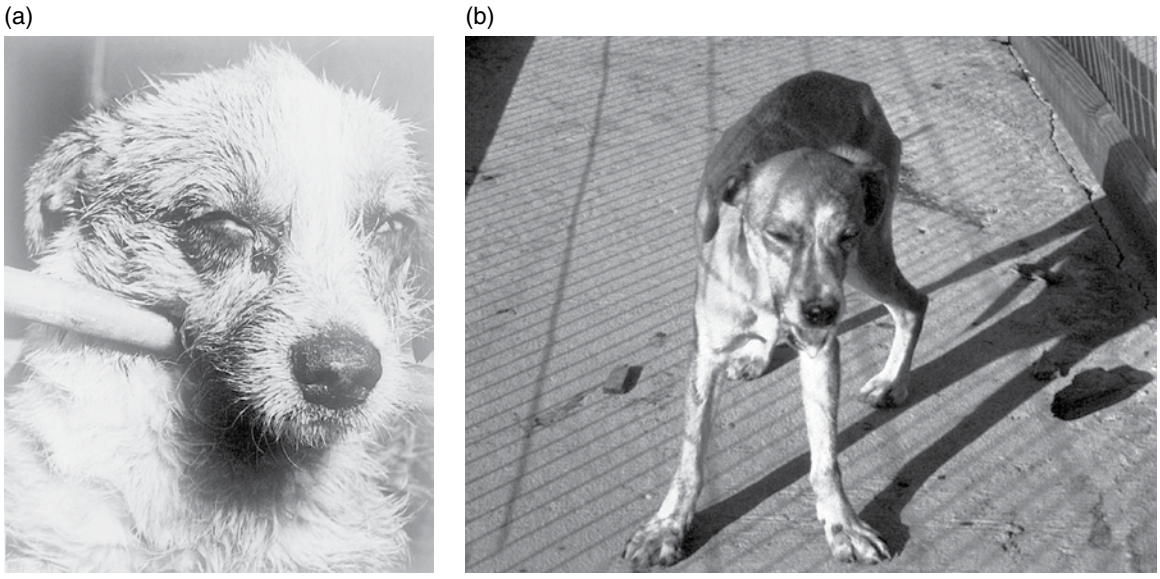

Figure 4.2 (a) and (b) Rabid dogs in later stages of disease. (Images courtesy Centers for Disease Control and Prevention's Public Health Image Library (www.cdc.gov))

through a courier set up by the laboratory with the box specifically labeled as "Biohazard/ Rabies Sample."

Technician Duty Box 4.1

Veterinary technicians will be involved in the collection and submission of rabies samples. Some clinics will make the collection of the suspect head a technician duty, while in others the veterinarian will be in charge of this with the technician assisting. Once collected, the head needs to be packaged properly and submitted to the appropriate state-approved laboratory.

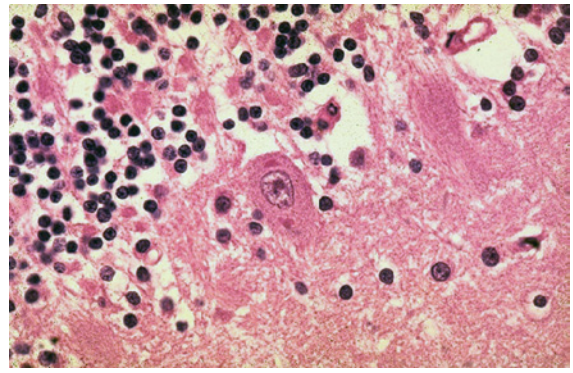

Figure 4.3 Negri bodies found in a neuron. Two darker inclusions seen in the neuron in the middle of the field. (Images courtesy Centers for Disease Control and Prevention's Public Health Image Library (www.cdc.gov))

- There are two methods of testing. IFA detects the rabies antigen in brain tissue. This is the preferred method, as there are fewer false negatives. An IFA test will test positive prior to clinical signs and will show positive in all positive species.
- Histopathology is another method of testing, where intracellular inclusions called negri bodies (Figure 4.3) are detected in the neurons of brain tissue. This method may produce false negatives, as negri bodies commonly form after clinical signs have

appeared, and not all species regularly form negri bodies.

Treatment

- There is no approved treatment for rabies in animals. Development of rabies will end in natural death or euthanasia.
- Treatment is available for humans with exposure to rabies virus. The treatment regimen is referred as "post-exposure prophylaxis" (PEP) and includes two components. The first, human diploid cell vaccine (HDCV), provides active immunity. The vaccines are given intramuscularly, usually in the arms, with seven injections over a month's time. The second component, rabies immunoglobulin (RIG), provides passive immunity and is injected into the bite sites and intravenously on the first visit.

Disease Prevention and Control

TECH BOX 4.2: Rabies cannot be treated in animal patients. Rabies exposures will result in death, unless the animal is vaccinated.

- Vaccination is the best thing that can be done to prevent rabies in domestic animals. Other disease prevention strategies include vaccination laws, licensing regulations, removal of stray and unwanted animals, regulation of interstate and international movement of animals, limiting exposure to wildlife, and good veterinary care.
- Pre-exposure vaccination laws will vary from location to location. Recommendations suggest that all dogs, cats, and ferrets be vaccinated. Horses must be vaccinated for interstate travel; however, vaccination is strongly recommended for all horses. Cattle and other livestock are vaccinated based on economics and public health significance.
- Vaccinations must be given per labeled instructions. No off-label usage is permitted for domestic animals. Parenteral vaccinations are not permitted for use in wild animals. There may be certain circumstances based on public health significance where the local authorities allow vaccines to be used off-label, although in the case of an exposure the off-label vaccinated animal cannot be considered protected.
- Regulations require vaccines be given by a veterinarian or someone under the direct supervision of a veterinarian. Vaccines are started between 12 and 16 weeks of age and a booster must be given 1 year later. Even mature animals must have the booster 1 year after the initial vaccine. In appropriate jurisdictions, after the first two vaccines are given a 3-year vaccine schedule can be established.
- Antibody levels are considered protective 28 days after the initial vaccine. After the booster antibodies respond immediately.
- Although local rabies statutes will vary, there are recommendations and guidelines set forth by groups including the World Health Organization (WHO), the Centers for Disease Control and Prevention (CDC), and the National Association of State Public Health Veterinarians (NASPHV).
- Rabies exposure in an area where infections occur is defined as any animal exposed to a wild, carnivorous mammal or bat. Even if the animal is not available for testing, it must be assumed to have rabies, and the encounter is labeled as an exposure to the disease.
- Recommendations suggest that an unvaccinated domestic animal exposed to rabies be euthanized. If the owner refuses, the animal needs to be confined as defined by the state for up to 6 months. During the confinement period the animal is watched closely for any clinical signs of rabies to develop. If any clinical signs are noted, the animal is euthanized and the head is sent for testing. If no

clinical disease develops, the animal is vaccinated 30 days prior to release.
- If the domestic animal exposed is currently vaccinated it should be revaccinated immediately and closely observed for 45 days.
- If a human is bitten, the first thing that should be done is a good cleansing of the bite, ideally with soap and water. The water will dilute the virus particles and disinfectants have been shown to kill the virus.
- If a human is bitten by a domestic animal, the protocol is based on the health of the animal and not vaccine status. If the animal is healthy and shows no sign of neurological dysfunction it should be observed for 10 days. If the animal is rabid, the bite is seen as a clinical sign of disease. The infected animal will be dead within the observation period. The animal is not immediately revaccinated/vaccinated because vaccine reactions can be confused with clinical signs of the disease.
- If the biter is a wild carnivorous mammal or bat, local health authorities and a doctor must be notified immediately. Authorities will make decisions on whether PEP is necessary based on factors such as the biting species, circumstances surrounding the bite, and prevalence of rabies in the area.

Client Education and Technician Tips

TECH BOX 4.3: Public education is key in dealing with the spread of rabies virus. The more the public understands about rabies, the easier it is to get compliance with regulations and stop the spread of the disease.

- Not all encounters result in an exposure. It is important to contact the appropriate authorities to determine how to proceed with the situation.
- Animals can be contagious before clinical signs of rabies appear. Variations are seen from species to species, but it may be up to 3 days prior to clinical signs.
- Animals in the prodromal stage of the disease pose the greatest risk to humans, especially veterinary personnel. The animal is known to be sick, but no pathognomonic signs of the disease are present.
- It is important to wear gloves during oral exams of unfamiliar animals, those that seem to be choking, or those exhibiting neurological signs.
- Although there is no feline variant of the virus, there are more cases reported every year in the United States in cats than dogs. This makes vaccination of cats crucial.
- Antibody titer testing cannot be used in substitution of a vaccine. Even animals with sufficient antibody titers are required to be vaccinated on schedule.

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Gastrointestinal Tract Disease

chapter 5

The role of the GI system is to acquire useable nutrients for the animal. The GI tract is a continuous tube of specialized organs running from the oral cavity to anus, with accessory organs aiding in the physical breakdown and chemical digestion of food. Diseases of the GI tract will result in an unsuccessful effort to get nutrients into the body and to the cells that need the nutrients. Common clinical signs associated with GI tract disease include anorexia, vomiting, regurgitation, diarrhea, dehydration, tenesmus, and abdominal pain; these signs will vary with the affected organ.

Oral Cavity

Periodontal Disease or Periodontitis

Description

Periodontal disease attacks the bone and gum surrounding the teeth. This is seen in both dogs and cats. Small breeds are more susceptible than large breeds to periodontitis. Food may be factor in causing the disease. Usually tartar buildup and gingivitis begins by age 2–4 years, and damage progresses to the periodontal ligament and bone.

Clinical Signs

- Increased pocket depth and loosening of the periodontal ligament to create tooth mobility.
- Other clinical signs include the buildup of tartar, gingivitis, gingival recession, possible bone loss, halitosis, and bleeding from the mouth (Figure 5.1).
- Animals with periodontal disease may be head shy and experience dysphagia, anorexia, and weight loss.
- If left untreated this disease can result in tooth loss, tooth root abscesses, and visible tooth furcation.

Diagnosis

- Presenting clinical signs, dental exam, dental radiographs, and measurement of increased pocket depth (normal in dogs is 1–3 mm

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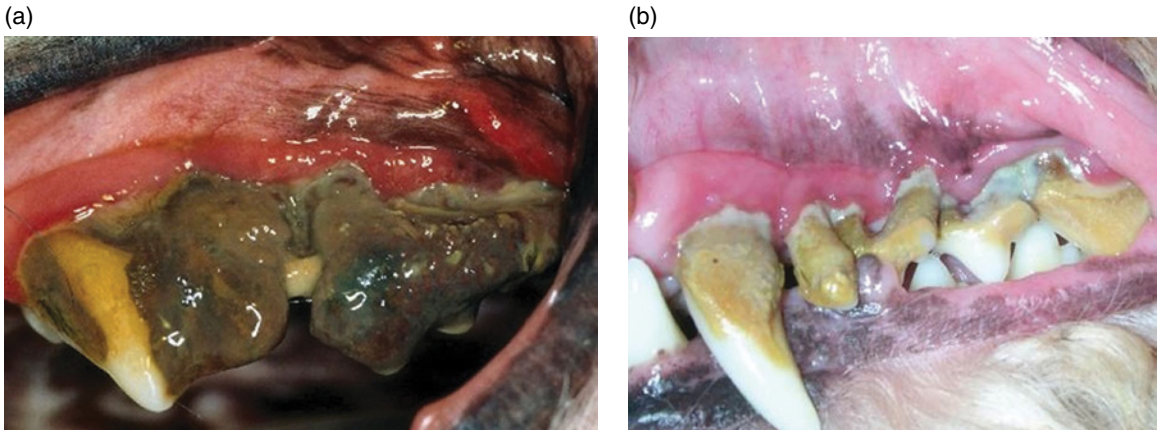

Figure 5.1 (a) and (b) Severe periodontal disease and tartar accumulation in dogs. (Images courtesy Deanna Roberts)

and in cats 0–1 mm) are used to diagnose periodontal disease.

Treatment

- Dental prophylaxis is important in treating and possibly reversing the disease.

Technician Duty Box 5.1

The veterinary technician routinely performs the dental prophylaxis and dental radiographs on patients with periodontal disease. More advanced procedures—for example, tooth extractions—will be the job of the veterinarian.

- Antibiotics may be prescribed if infection and inflammation are present.
- Measures such as tooth extraction and bone grafting may be considered based on the severity of disease.
- Analgesic drug therapy is an important part of treatment to keep the animal comfortable and able to eat, especially in severe cases.

Client Education and Technician Tips

- Prevention is key with any dental disease. Regular dental exams, cleanings, and plaque/tartar control techniques are important.

TECH BOX 5.1: Prevention is key in dealing with dental diseases.

Papilloma or Puppy Warts

Description

A condition of benign oral masses found in young dogs is known as papillomatosis. Papillomas are proliferative masses found on mucous membranes anywhere in the oral cavity and adjacent skin, caused by a single stranded DNA virus from the Papovaviridae family. The virus has an incubation period of 1–2 months.

Transmission

- Papillomas are spread through direct contact of mucous membranes, fomites, and dogs playing.

Clinical Signs

- Papillomas are wart-like masses found anywhere in the oral cavity.
- Depending on the location, papillomas may cause dysphagia and trauma may cause bleeding.

Diagnosis

- Most papillomas are diagnosed with oral exam, presenting clinical signs, and history.
- Definitive diagnosis is obtained via biopsy and histopathology.

Treatment

- This is a self-limiting virus, and no treatment is usually necessary, as the mass will regress on its own.
- Masses may be removed if causing problems with eating, chewing, and swallowing.

Epulis

Description

An epulis is the most common benign oral mass seen in dogs. The pedunculated mass grows from a gingival surface and is seen at any age and in any breed of dog, although Boxers are the most common breed to present with epuli. The cause of epulis is unknown. There are three forms based on tissue from which the mass originates. A fibromatous epulis originates from the margin of the gums and is smooth in texture. Ossifying epuli are more severe and involve the underlying bone. Acanthomatous epuli are ulcerative and found on the front part of the lower jaw and originate from the periodontal ligament.

Clinical Signs

- An epulis is a firm solid mass growing from the gingival surface that is usually not ulcerated, unless it is an acanthomatous mass. It may look like overgrowth of the gum tissue (Figure 5.2).
- Other possible clinical signs include pain, hypersalivation, bleeding, shifting in the alignment of the dog's teeth, anorexia, and weight loss.

Diagnosis

- Oral exam, presenting clinical signs, and history in addition to radiographs will aid in obtaining a presumptive diagnosis.

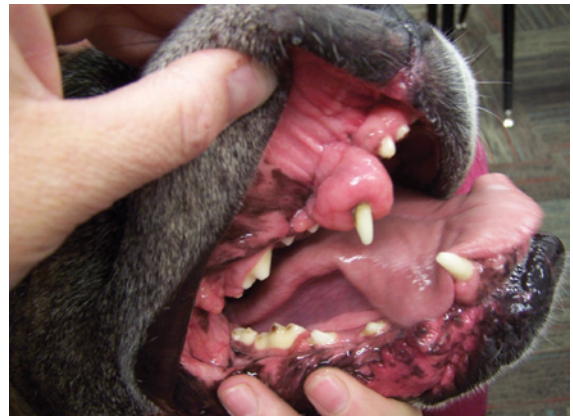

Figure 5.2 Epulis in a boxer. (Image courtesy Amy Johnson and Stephanie Aagaard)

- Definitive diagnosis is obtained via biopsy and histopathology.

Treatment

- Treatment involves surgical removal and possible reconstruction of the affected area. Removal of the underlying bone may be required, especially with an ossifying and acanthomatous epuli (Figure 5.3).
- Radiation therapy has been found to be successful with small masses, and chemotherapeutic agents may be used to stop the spread.

Client Education and Technician Tips

- Prognosis is usually good, although some types may be more difficult to manage than others (Figure 5.4).

Oral Melanoma

Description

Oral melanomas are tumors of the melanocytes of the mouth and recognizable by very rapid growth, local invasiveness, and early metastasis. Oral melanomas are the most common malignant oral tumor found in dogs, yet very rare in cats. These tumors are found more often in older dogs, and the incidence in males is greater than females. Dogs with

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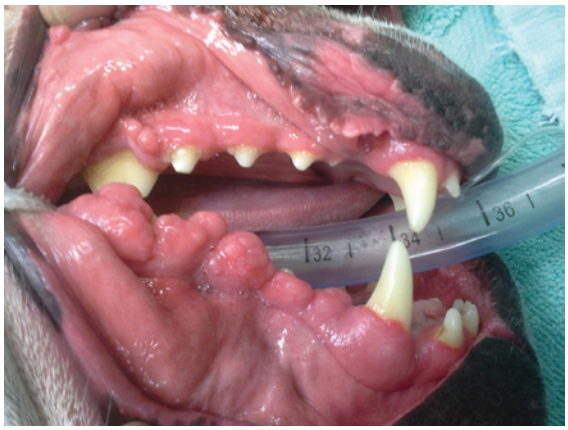

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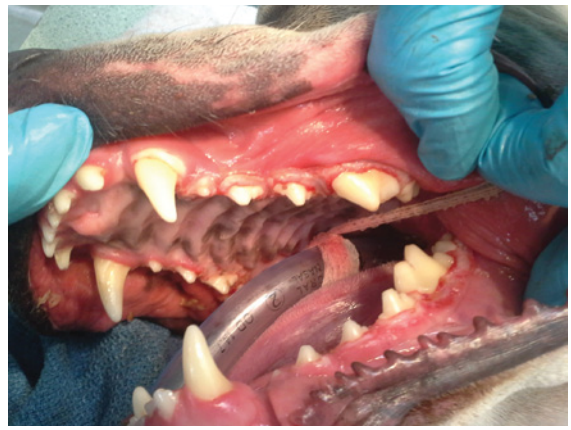
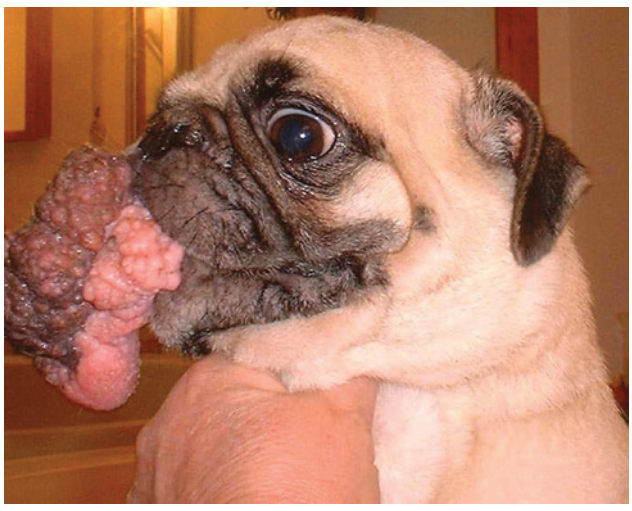


Figure 5.3 (a) and (b) Epulis before and after removal. (Image courtesy Kaylee John)

(a)



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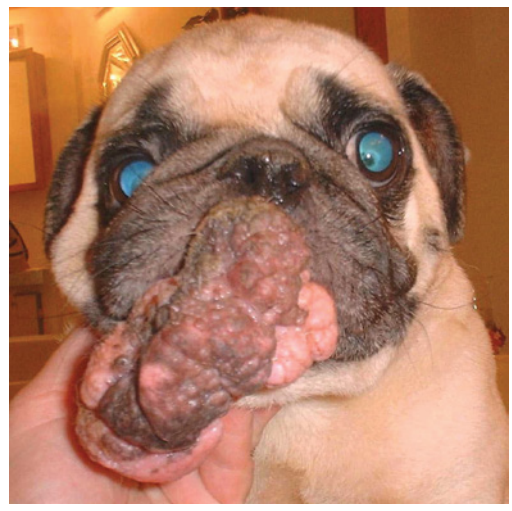

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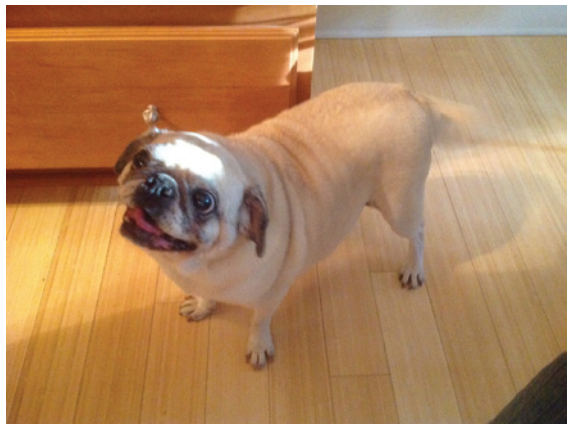

Figure 5.4 Severe epulis seen in a pug. (a) and (b) Before surgical removal. (c) After surgical removal. (Image courtesy Chris Hedrick/Pug Rescue of North Carolina)

more pigmentation to their oral mucosa are at greater risk. The cause of oral melanomas is not fully known, although genetics is a suggested cause.

TECH BOX 5.2: Melanoma is the number one oral neoplasia in dogs and carries the worst prognosis of all the oral tumors.

Clinical Signs

- Melanoma tumors are ulcerative, usually black masses, although some are amelanotic or without pigment. The mass may be ulcerated or necrotic (Figure 5.5).
- The masses are most often associated with the gingival tissue, although some can be found originating from the buccal or labial mucosa.

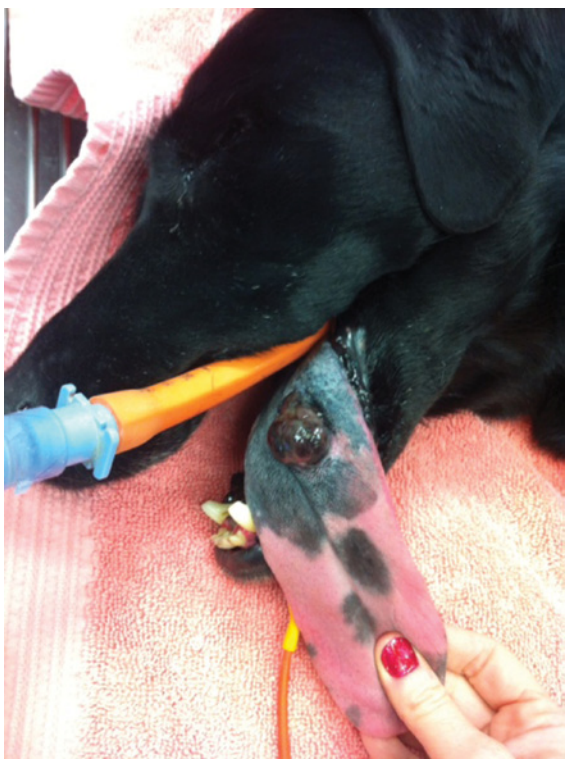

Figure 5.5 Lingual oral melanoma. (Image courtesy Deanna Roberts)

- Other clinical signs associated with melanoma include halitosis, anorexia, and hypersalivation.
- Clinical signs associated with metastasis include respiratory distress and swollen face or lymph nodes.

Diagnosis

- Oral exam and clinical presentation of a black mass provide presumptive diagnosis, although non-pigmented masses cannot be ruled out as melanomas.
- Radiographs or a CT scan of the jaw can help determine if the neoplasia has invaded the underlying bone.
- Definitive diagnosis is obtained via biopsy and histopathology (Figure 5.6).
- When looking for evidence of metastasis, regional lymph nodes should be aspirated and thoracic radiographs performed (Figure 5.6).

Treatment

- Dogs with melanoma have a very poor prognosis. Owners may choose not to treat and consider euthanasia when the quality of life is compromised.
- The mass can be surgically removed, making sure to get a wide excision to ensure complete removal. This often means mandibulectomy or maxillectomy.
- Chemotherapy, immunotherapy, or radiation therapy can be coupled with surgical removal to lengthen survival times.
- A new therapeutic vaccine has been conditionally approved for use in conjunction with other therapies. This vaccine is only currently available through veterinary oncology specialists.

Client Education and Technician Tips

- Unfortunately there is not a good prognosis for long-term survival. Prognosis is based on

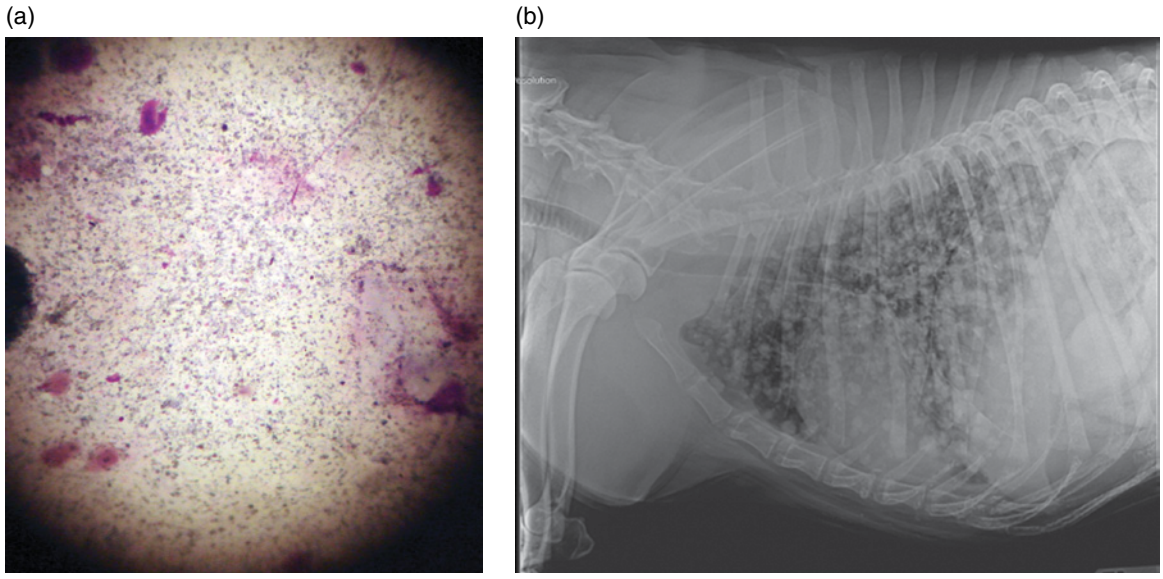

Figure 5.6 (a) Aspirate from a melanoma mass. (Image courtesy Amy Johnson and Bel-Rea Institute of Animal Technology) (b) Thoracic radiographs showing evidence of metastatic neoplasia. (Image courtesy Deanna Roberts)

size, location, and the stage of the tumor. Life span after a diagnosis of melanoma averages months to a year with treatment; some animals average as long as a couple of years with aggressive treatment.

Oral Squamous Cell Carcinoma (SCC)

Description

Squamous cell carcinomas are highly invasive oral tumors found in dogs and cats; they frequently involve the gingiva and tongue. Squamous cell carcinomas are most often ulcerative, inflamed, and involve necrosis. They are the most common oral tumor found in cats, usually older cats, and often go unnoticed until dental procedures. The cause of these tumors is unknown.

TECH BOX 5.3: Squamous cell carcinomas are the number one oral neoplasia diagnosed in cats.

Clinical Signs

- Cats with oral SCCs will often have an ulcerated lesion on the lateral tongue surface or gingiva, with some sublingual.
- With dogs the tumor is often found on the gingival surface and on one tonsil. This neoplasia is not easy to visualize on the tonsil, but owners will notice swallowing and breathing difficulties.
- Pain associated with the tumor will also cause anorexia, grooming difficulties, swallowing problems, halitosis, and hypersalivation.

Diagnosis

- Oral exam and clinical presentation of mass will aid in presumptive diagnosis.
- Definitive diagnosis is obtained via biopsy and histopathology.
- Diagnostic imaging, including radiographs, CT scans, or MRI will aid in determining the extent of the tumor involvement with the underlying bone.

Treatment

- Surgical removal with wide margins is possible, yet recurrence is common.
- Trials with different chemotherapeutic therapies, radiation, and anti-inflammatory drugs have shown little success.

Client Education and Technician Tips

- There is a very poor prognosis for cats with SCC and few options for treatment. Cats undergoing treatment have a survival time of less than a year, and even less without treatment. Euthanasia must be considered when the quality of life declines.
- Gingival masses in dogs are rarely metastatic, although the tonsillar masses often metastasize to regional lymph nodes.
- Prognosis for dogs depends on where the SCC is located, with the gingival masses offering a better survival time. With surgical removal survival time averages 1–2 years.

Oral Fibrosarcoma

Description

A fibrosarcoma is a malignant mass originating from the fibrous connective tissue in the mouth, seen in both dogs and cats. Fibrosarcomas are very locally invasive and the second most common oral tumor in cats. The mass is found more commonly in older cats and small to medium dogs. In large breed dogs the tumors are found more in younger dogs. The cause of this neoplasia is unknown.

Clinical Signs

- The tumor appears as an ulcerated mass on gingiva or palate. Swelling may be noted under the eye if associated with maxilla. This swelling can be confused with a tooth root abscess (Figure 5.7).

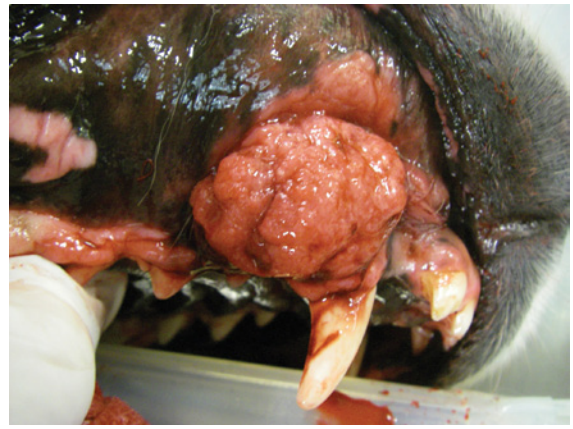

Figure 5.7 Canine fibrosarcoma. (Image courtesy Emma Worsham)

- Other clinical signs associated with this oral tumor include anorexia, weight loss, hemorrhage, halitosis, and dysphagia.

Diagnosis

- Oral exam and clinical presentation of mass will aid in determining a presumptive diagnosis.
- Definitive diagnosis is obtained via biopsy and histopathology.

TECH BOX 5.4: The best diagnosis of any oral tumor is through biopsy and histopathology.

- Diagnostic imaging techniques, including radiographs, CT scans, or MRI can be used to determine the invasiveness of the tumor and if there is involvement of the underlying bone.

Treatment

- Surgical removal of the tumor requires wide surgical excision, usually a hemimaxillectomy or hemimandibulectomy (Figure 5.8).
- Radiation therapy and chemotherapy may be coupled with surgery, but evidence shows little response to these therapies other than a short-term palliative response.

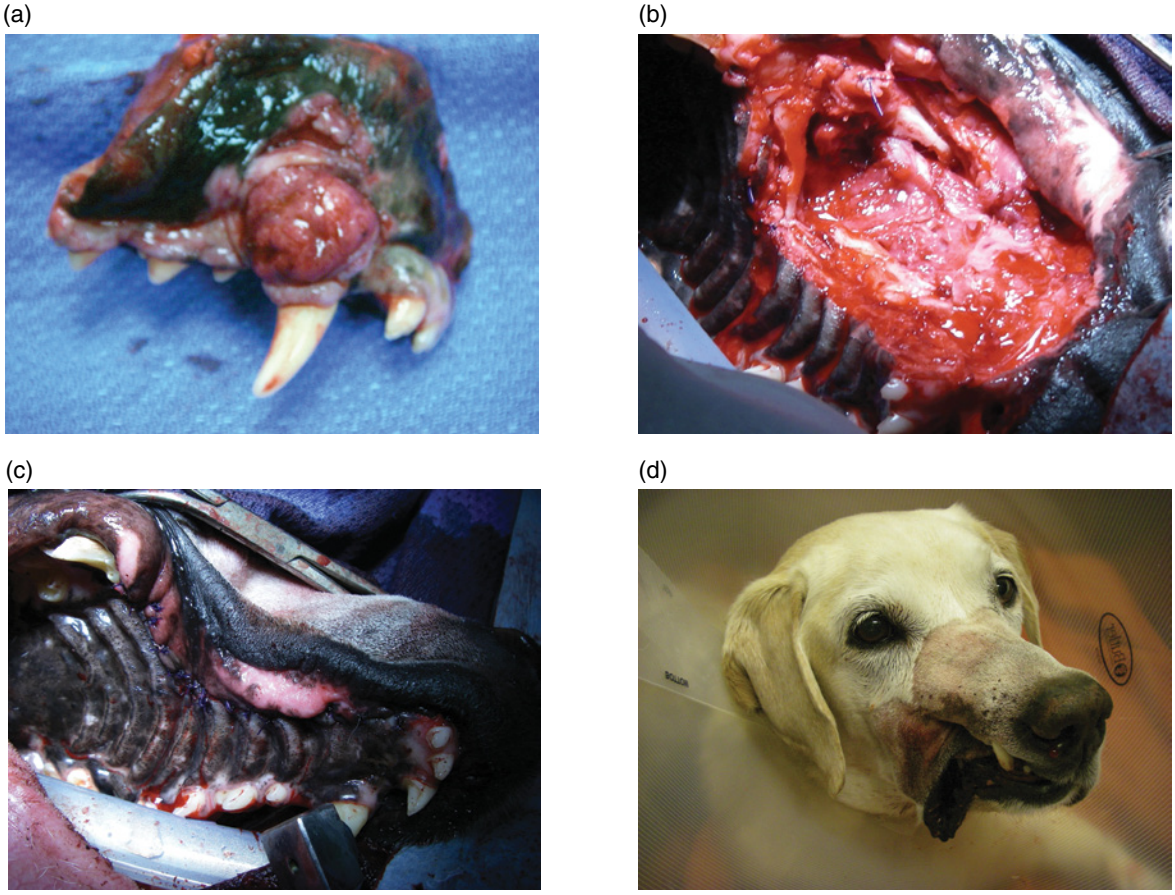

Figure 5.8 (a) Surgical excision of fibrosarcoma. (b) Surgical site post-removal. (c) Surgical site as finished. (d) Patient recovering. (Images courtesy Emma Worsham)

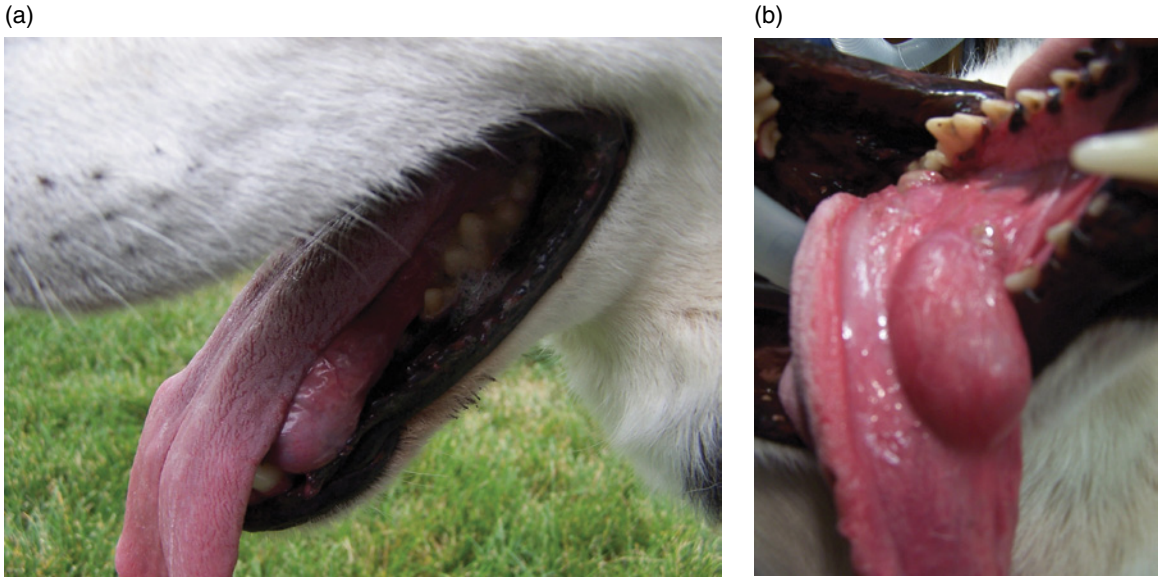

Figure 5.9 (a) and (b) Canine sublingual sialocele. (Image courtesy Amy Johnson and Bel-Rea Institute of Animal Technology)

Salivary Mucocele, Sialocele, or Salivary Gland Cyst

Description

A sialocele is an accumulation of saliva in a salivary gland, duct, or SQ tissues, caused by an obstruction to salivary flow. These cysts are most often associated with the sublingual salivary gland or duct. The condition is secondary to trauma, obstructive tumors, and can be spontaneous or idiopathic. These cysts can rupture or cause damage to the gland if not treated promptly.

Clinical Signs

- A sialocele is usually seen as a unilateral, nonpainful swelling under the neck or tongue (Figure 5.9).
- The mass is not always outwardly evident but may cause dysphagia.

Diagnosis

- Palpation of the mass with aspiration using an 18–20 gauge needle can be diagnostic. The aspirate is a thick, viscous, mucoid fluid possibly blood tinged. Microscopic evaluation reveals a few red blood cells but little else (Figure 5.10).

Treatment

- Treatment requires identification and removal of the initiating cause. This may includes anti-inflammatory drug therapy or tumor removal.
- If idiopathic or untreatable, periodic draining or surgical removal of the affected gland can be performed.

Client Education and Technician Tips

- These salivary glands sit in close association with the submandibular (mandibular) lymph

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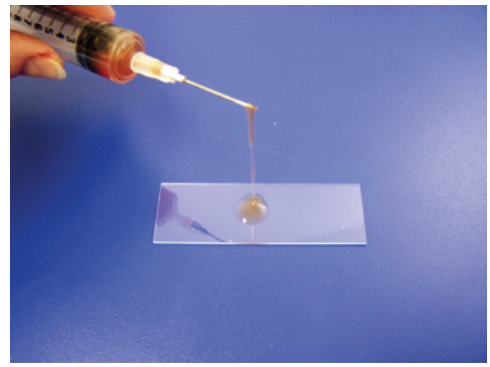

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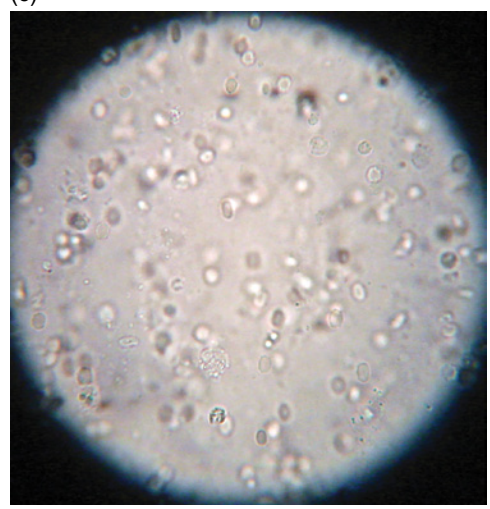

Figure 5.10 (a) Aspiration of sialocele. (b) Fluid removed from sialocele. (c) Microscopic examination of the fluid removed from the sialocele with RBCs and WBCs. (Images courtesy Amy Johnson and Bel-Rea Institute of Animal Technology)

nodes, making it important to differentiate between the two causes when there are problems.

TECH BOX 5.5: Salivary gland cysts can easily be confused with submandibular lymph node disease.

Esophagus

Megaesophagus or Acquired Paralysis

Description

The condition megaesophagus is caused by a hypomotility of the esophagus, leading to abnormal transport of ingesta. The accumulation of ingesta in the esophagus will cause a stretching and dilatation. This condition is seen in dogs and cats and may be congenital or acquired. There seems to be a genetic predisposition for the congenital form with Great Danes, Irish Setters, Newfoundlands, Miniature Schnauzers, German Shepherds, Shar-Peis, and Labrador Retrievers at the greatest risk. The disorder may segmental or generalized and mild to severe. The origin of the disease can be neurological or muscular or a disorder with the neuromuscular junction. The hypomotility is secondary to some other disorders, including myasthenia gravis (Figure 5.11), a neuromuscular disease, or trauma, but most often idiopathic.

TECH BOX 5.6: The severity of megaesophagus can vary greatly from patient to patient.

Clinical Signs

- In the congenital form, clinical signs first appear as the animal is being weaned and beginning to eat solid foods. Clinical signs with the acquired form can appear at any time.
- Regurgitation is the number one sign of megaesophagus. The regurgitated material returns as mucous-coated tubular casts with the food closely resembling the food that was eaten.

TECH BOX 5.7: Regurgitation is the number one sign of esophageal disease.

- Regurgitation increases the risk of aspiration pneumonia. Animals may present with signs of a respiratory infection, dyspnea, lethargy, and fever.
- Other clinical signs include stunted growth and a thin/malnourished animal despite a ravenous appetite.

Diagnosis

- Megaesophagus includes a history of regurgitation associated with meals.
- Physical exam may include small, thin animals with signs of aspiration pneumonia.
- With a megaesophagus patient, plain or contrast thoracic radiographs will show food, fluid, or air in the lumen of the esophagus that usually extends the length of the esophagus. Fluoroscopy and esophagoscopy can also be used to visualize the lumen of the esophagus and the material within (Figure 5.11).
- If a primary disease is suspected, testing for that primary cause is helpful.

Treatment

- If possible it is best to be able to identify and treat the primary cause.
- Esophageal surgery has not proven very successful but may be an option in some cases.

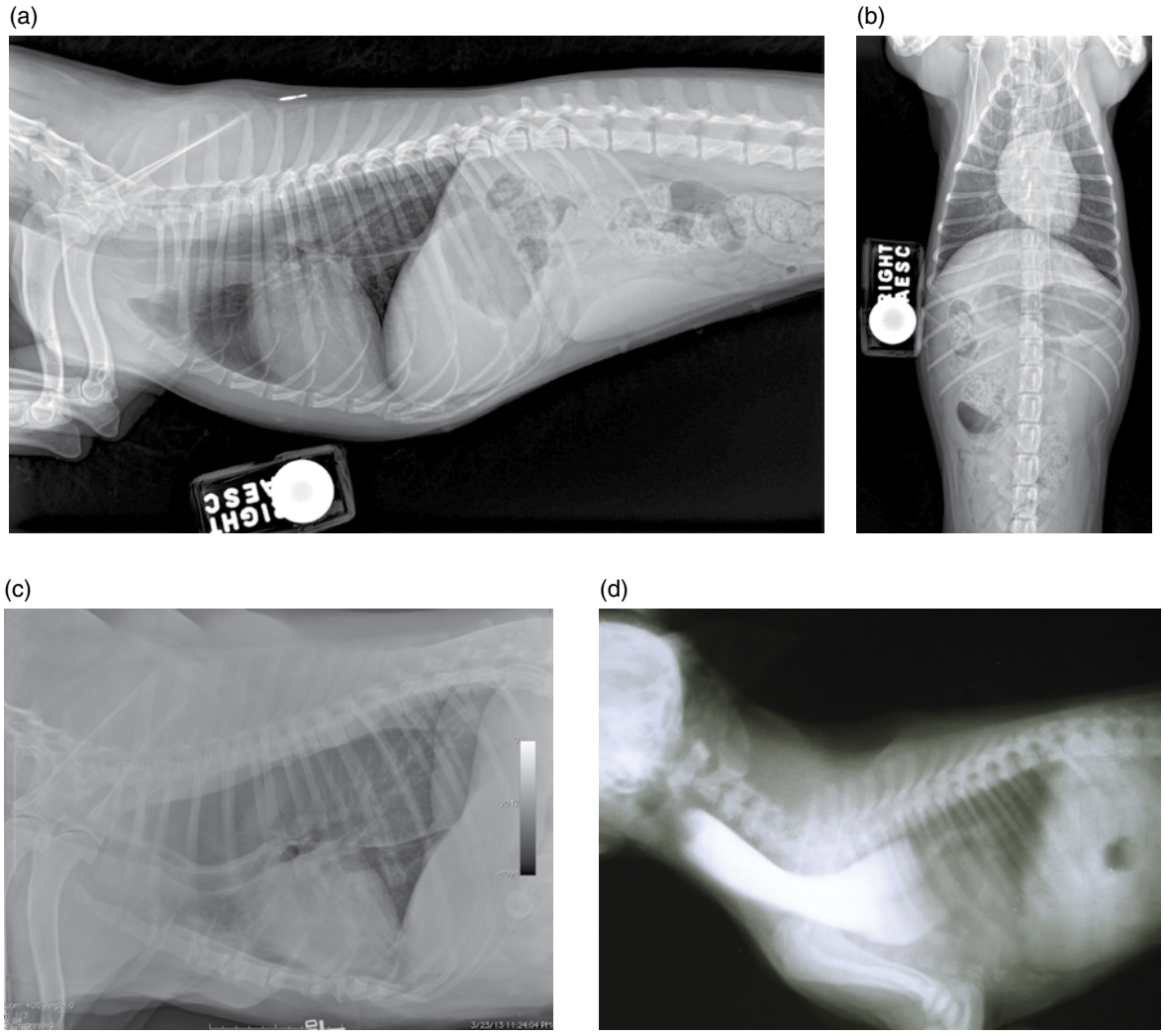

Figure 5.11 Radiographs of canine megaesophagus patient. (a) Lateral. (b) Ventrodorasl. (Images courtesy Dawn Martin) (c) Radiograph of canine megaesophagus secondary to myasthenia gravis. (Image courtesy Dr. Robert Roy/ Palm Beach Veterinary Specialists) (d) Contrast radiographs of canine megaesophagus. (Shutterstock image courtesy P. Fabian)

- Palliative treatment seems to have best results. The goal is to meet the animal's nutritional needs while avoiding aspiration pneumonia. This includes elevated feeding (Figure 5.12); small, frequent meals; and food consistency tailored to the animal.

Client Education and Technician Tips

- Prognosis depends on many factors, including the severity of the paralysis, nutritional state of the patient, and the patient's response to treatment.

(a) (b)

Figure 5.12 (a) and (b) Elevated feeding of a megaesophagus patient with a device called the "Bailey Chair." (Images courtesy Donna Koch. Photos taken by Andy Patterson. http://pets.groups.yahoo.com/group/megaesophagus/. http://www.baileychair.blogspot.com/)

- Aspiration pneumonia always remains a risk with this condition.
- Some cases may be so mild they go undiagnosed.

Esophageal Obstructions or Foreign Bodies

Description

An esophageal foreign body is the result of ingestion of foreign material that becomes lodged in the esophagus. Materials tend to get lodged in areas of little distension: thoracic inlet, base of the heart, and diaphragm hiatus. This condition is seen more frequently in dogs than cats.

TECH BOX 5.8: Esophageal obstructions tend to occur in areas of little distension.

Clinical Signs

- Clinical signs include regurgitation, gagging, hypersalivation, and difficulty swallowing.
- In some cases owners may see the object ingested.

Diagnosis

- Diagnostic imaging such as thoracic radiographs, with or without contrast, and esophagoscopy are used to visualize the foreign body.

Treatment

- Removal of the object is necessary. Endoscopic removal is best because it is the least invasive method. Surgery can be performed, but the prognosis is poor because of the healing process. Esophageal surgeries are often avoided, as the esophagus does not heal well and often scarring will cause a stricture of the esophagus.

Vascular Ring Anomaly (VRA) or Persistent Right Aortic Arch (PRAA)

Description

A VRA is a genetic defect animals are born with, caused by retention of an embryonic right aortic arch. The fetus will have both a right and left aortic arch with the right regressing shortly after birth, while the left will develop into the aorta. When the right arch is retained the esophagus becomes entrapped in a vascular ring that includes the right embryonic arch, aorta, pulmonary artery, and the base of the heart. As a result of this constriction the esophagus cannot dilate to accommodate the ingesta. As the ingesta reaches the narrowing of the esophagus it becomes retained. This condition is seen in dogs and cats but is seen more frequently in dogs.

TECH BOX 5.9: A vascular ring anomaly is a congenital condition, making clinical signs apparent early in life.

Clinical Signs

- Regurgitation is the number one sign of a VRA and it begins at the point of weaning. The regurgitated material is not digested and coated in mucous.
- Patients are thin and small despite ravenous appetites.
- Aspiration pneumonia is a risk because of the regurgitation, and a secondary megaesophagus may result from the hypomotility and retention of ingesta.

Diagnosis

- Diagnostic imaging, including plain or contrast radiographs, will show a constriction in the esophagus just cranial to the base of the heart. The ingesta is retained cranial to the constriction.

Treatment

- Palliative treatment includes elevated feeding; small, frequent meals; and liquid food.
- Surgery to ligate and resect the PRAA is the only curative treatment.

Client Education and Technician Tips

- Prognosis is varied and depends on the level to which the animal is malnourished and the development of secondary conditions, such as aspiration pneumonia and megaesophagus.
- Affected animals, their parents, and their siblings should not be bred.

Gastroesophageal Reflux

Description

Gastroesophageal reflux is a retrograde flow of gastric and duodenal contents into the esophagus resulting from an inadequate closure of the lower esophageal sphincter. Gastric acid, pepsin, bile salts, and other components of gastric juice are allowed to enter the esophagus, causing an esophagitis. Drugs, chronic vomiting, congenital anomalies, and hiatal hernia are among the common causes. The condition is seen most often in young dogs with developing sphincters and is a condition that they may outgrow.

Clinical Signs

- Regurgitation of partially digested food, possibly including bile and blood, is related to position. Animals lying down are more likely to regurgitate, making the regurgitation worse late at night or in the early morning hours. The loss of nutrients may result in weight loss.

TECH BOX 5.10: Unlike other esophageal conditions, gastroesophageal reflux regurgitation is associated with position and not meals.

- The related esophagitis will cause pain, discomfort, anorexia, and weight loss. Vomiting may also be present as a result of the discomfort of esophagitis.

Diagnosis

- Radiographs may show hiatal hernia or other primary causes but do not allow visualization of the sphincter or esophagitis.
- Endoscopy will allow visualization of the lumen and lining of the esophagus and sphincter.

Treatment

- Mild cases may respond to drug therapy, including antacids, antiemetics, and analgesics. Gastrointestinal prokinetic drugs will aid in increasing stomach emptying and will strengthen the sphincter muscle.
- Hiatal hernias will require surgical repair.
- Surgery to stabilize movement of stomach and esophagus may also aid in some cases.
- A diet change is a must with this condition. A diet of low-fat and low protein fed in small, frequent feedings will help the regurgitation.

Client Education and Technician Tips

- Some patients may grow out of this condition, but most will require lifelong management.

Stomach

Acute Gastritis

Description

Acute gastritis is the sudden inflammation of the stomach resulting from damage to the gastric mucosa. Damage to gastric mucosa allows hydrochloric acid to damage deeper tissues. This tissue damage also stimulates histamine release, which can worsen the damage to the stomach lining. Gastritis has many causes, with dietary indiscretion being the most common. Other causes include parasites, systemic viral diseases, toxins, poisons, food sensitivity, and drug administration, especially non-steroidal anti-inflammatory drugs (NSAIDs).

Clinical Signs

- Acute onset of vomiting is the most common clinical sign of acute gastritis. The vomitus may contain bile, blood, or mucous.
- Other clinical signs include dehydration, anorexia, abdominal pain, polydipsia, pica, hypersalivation, lethargy, and melena.
- Fever may be present based on the cause.

Diagnosis

- Owners may be able to provide a history of dietary indiscretion or drug therapy.
- Radiographs may show foreign body but will not show the tissue damage associated with gastritis.
- Testing for initiating causes includes fecal exam, tests for viral diseases, biopsies, and bacterial cultures.
- Last case option would involve an exploratory laparotomy.

Treatment

- Treatment involves removing the initiating cause and resting the stomach. The animal is taken off food and water for a period of time. Small amounts of water are provided, then small amounts of bland food. Bland diets are low in fat and fiber and easily digestible. Food is provided in small amounts and fed frequently at first.
- Other treatments include antacids, antihistamines, mucosal protectants, antiemetics, and antibiotics.

Client Education and Technician Tips

- Caution must be taken with the use of NSAIDs because of stomach irritation.

TECH BOX 5.11: NSAID therapy is a common cause of gastritis and gastric ulcers.

- Owners need to check with their veterinarian before using human over-the-counter drugs like Pepto-Bismol. Pepto-Bismol can be used with caution in dogs but cannot be given to cats.

Gastric Ulcers

Description

Gastric ulcers are erosions of gastric mucosa extending through deeper tissue layers. Hydrochloric acid and histamine promote further tissue damage. Ulcers present as circumscribed erosions with firm and raised margins giving them a crater-like appearance. Ulcers are a chronic condition that develops after damage to gastric mucosa. Causes may include chronic gastritis, bacterial infections, stress, liver or kidney disease, mast cell tumors resulting from histamine production, and certain drugs. The use of corticosteroid and NSAID therapy is a common cause. Bacteria and stress are not thought to be linked to the condition in animals as strongly as they are linked to human ulcers.

Clinical Signs

- Gastric ulcers are usually associated with chronic vomiting, often related to eating. The vomitus usually contains blood that may be frank or digested.
- Other clinical signs include abdominal pain, anorexia, weight loss, and melena.
- Pale mucous membranes can be a sign of anemia associated with gastric hemorrhage.

Diagnosis

- The patient's medical history, presenting clinical signs, and physical exam are a starting point in the diagnosis of gastric ulcers.
- Anemia and hypoproteinemia as seen on a CBC, packed cell volume (PCV), total protein (TP), and RBC count will indicate bleeding.
- Radiographs with barium will reveal a barium-lined crater that is seen as a white area on the radiograph.
- Endoscopy is the best diagnostic tool, as the ulcer can not only be visualized but a biopsy can also be obtained.

Treatment

- Treatment for gastric ulcers is best if the underlying cause can be identified and corrected.
- Drugs used to treat ulcers include antacids, antihistamines, and mucosal protectants.
- Animals may require surgical resection of the damaged part of the stomach.
- In cases of gastric perforation, immediate surgical intervention is required.
- Cases involving severe anemia may require blood transfusions.

Client Education and Technician Tips

- Prognosis is dependent on the underlying cause and the animal's response to treatment.

Gastrointestinal Obstructions

Description

Gastrointestinal obstructions are caused by foreign material retained in the stomach or intestines, most often material that has been ingested by the animal. This condition is seen more frequently in dogs than cats, and younger animals are at higher risk with their propensity for dietary indiscretion.

Clinical Signs

- Clinical signs vary based on duration of obstruction, type of foreign body, and the degree of obstruction.

- Vomiting is the most frequent clinical presentation and may be accompanied by diarrhea, abdominal pain, anorexia, polydipsia, and dehydration.
- Some foreign bodies like pennies or lead can cause systemic toxicity.

Diagnosis

- Laboratory blood work-up including CBC, blood chemistry profile, and electrolyte panel can be used to rule out other causes of vomiting.
- Radiography can be used to visualize the obstructing object. Radiolucent items will need contrast to be visualized.
- Endoscopy will allow for visualization of the object.
- Gastrointestinal obstructions are often diagnosed upon finding the object during exploratory laparotomy (Figure 5.13).

Treatment

- In some cases the object may be able to pass through the gastrointestinal tract on its own, making surgery unnecessary.

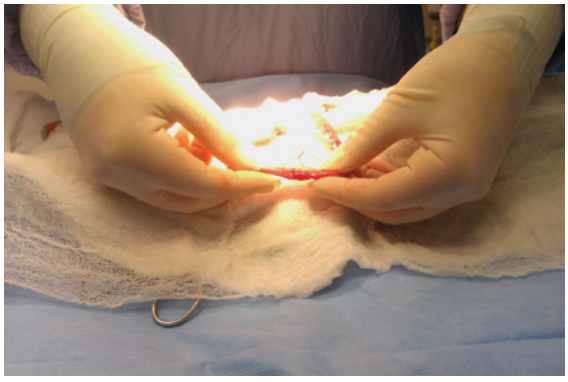

Figure 5.13 Gastrointestinal foreign body exploratory laparotomy. (Image courtesy Kristen Mutchler)

- As long as the object poses no danger by moving into the esophagus or oral cavity, vomiting can be induced to evacuate the object.
- Some objects may be retrievable via endoscope.
- Most cases will require laparotomy to find and remove the foreign object (Figure 5.14).

Pyloric Stenosis or Chronic Hypertrophic Gastropathy

Description

Pyloric stenosis is a condition of obstructive narrowing in the pyloric region of the stomach, causing gastric retention. The narrowing is secondary to hyrertrophic gastropathy or hormonal or neurologic dysfunction. It is a congenital condition most often seen in brachycephalic breeds.

Clinical Signs

- The most frequent clinical sign of pyloric stenosis is chronic vomiting that begins at the time of weaning. Vomiting occurs within a couple hours of eating and may be projectile vomiting. The vomitus includes undigested to partially digested food with no bile or blood.
- The animal is often hungry but is usually thin and small as a result of malnutrition.

Diagnosis

- Radiography will show delayed gastric emptying time. When barium is used on its own with radiography, the liquid will easily pass through the stomach. But when the barium is mixed with food, the images will revel a narrowing of the pylorus and gastric retention.
- Other imaging techniques, such as fluoroscopy and endoscopy, can be used to evaluate the extent and location of the narrowing.

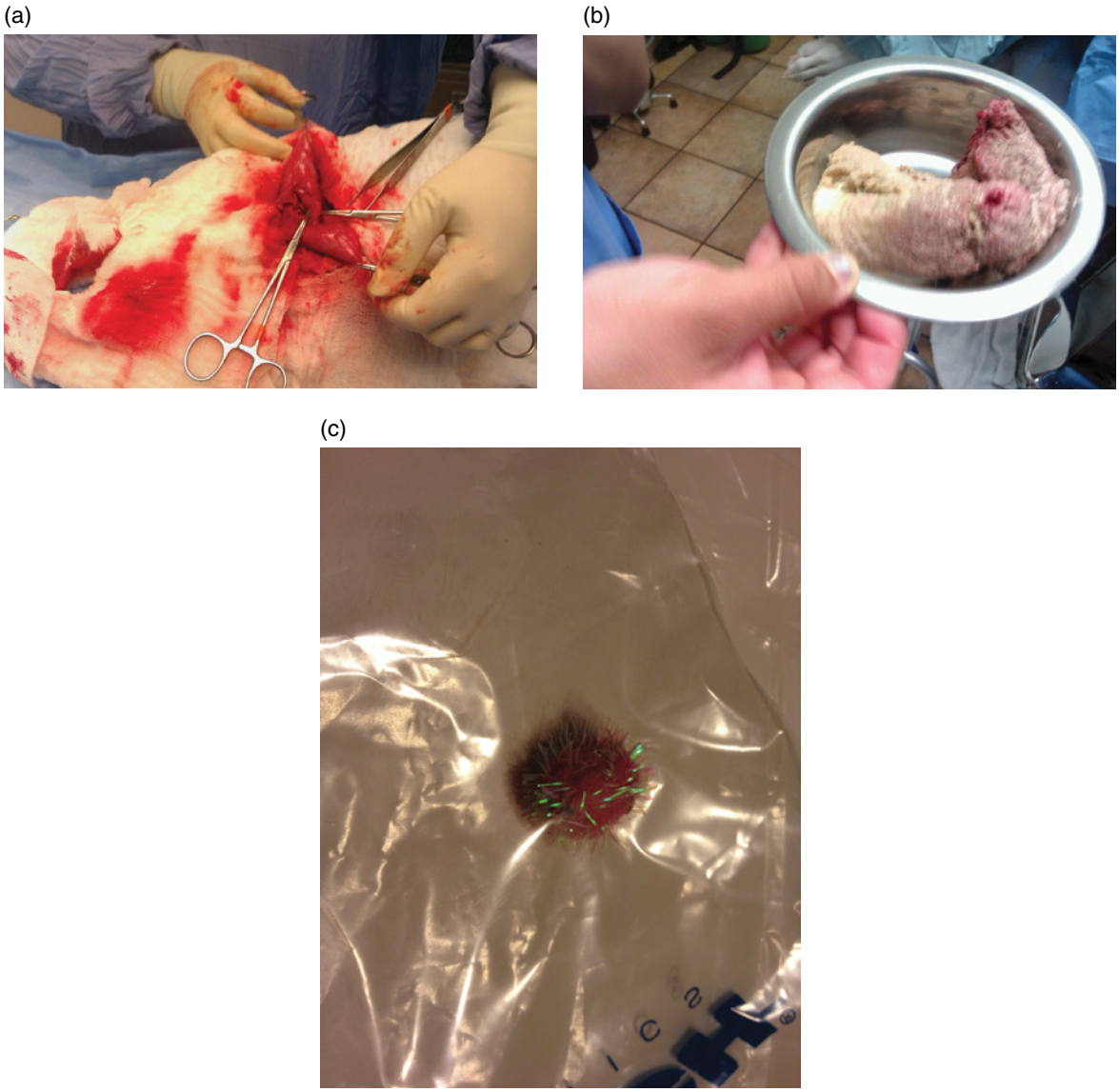

Figure 5.14 (a) Gastrotomy to remove foreign body. (b) Pillow stuffing removed from the stomach of canine patient. (Images courtesy Kristen Mutchler) (c) Small cat toy causing gastric obstruction in a kitten. (Image courtesy Amy Johnson)

Treatment

- Palliative therapy involves the feeding of the animal. Small, frequent, liquid meals are fed in an upright position to allow for better gastric emptying.
- Curative treatment requires surgical reconstruction of pylorus with a pyloroplasty or pylormyotomy.
- A highly digestible, low-fat diet is recommended with patients before and after surgery.

Client Education and Technician Tips

- Overall prognosis is good, especially if diagnosed and corrected early.

Gastric Dilatation and Volvulus (GDV)

Description

GDV is an acute life-threatening condition that requires immediate medical and surgical intervention in dogs. Although often referred to as bloat, there is a difference between the two conditions. Bloat is the accumulation of gas, food, and/or fluid in the stomach that cannot evacuate via esophagus or duodenum. A GDV includes torsion of the stomach that complicates the evacuation of gastric contents and obstructs blood flow. Gastric distension usually occurs first, and then the torsion follows. Pressure on the diaphragm can impair ventilation and cause a hypoxia. Venous blood return to the heart decreases, creating a hypovolemic shock that will lead to multiple organ dysfunction and death. GDV has a breed predisposition to large breed, deep-chested dogs, with the factors not fully understood.

TECH BOX 5.12: Although there is a breed predisposition, a GDV can occur in any breed of dog.

Clinical Signs

- A common GDV clinical sign is unsuccessful vomiting attempts. The dog may retch or gag but no vomit is produced. Hypersalivation also accompanies GDV.
- The abdomen is distended and tympanic or hollow sounding (Figure 5.15).
- GDV patients are uncomfortable, as seen with pacing, whining, changing positions, and stretching.
- Signs of shock include dyspnea or tachypnea, pale mucous membranes, weak pulse, rapid heart rate, and increased capillary refill time (CRT).
- The condition may lead to DIC and electrolyte and acid/base imbalances.

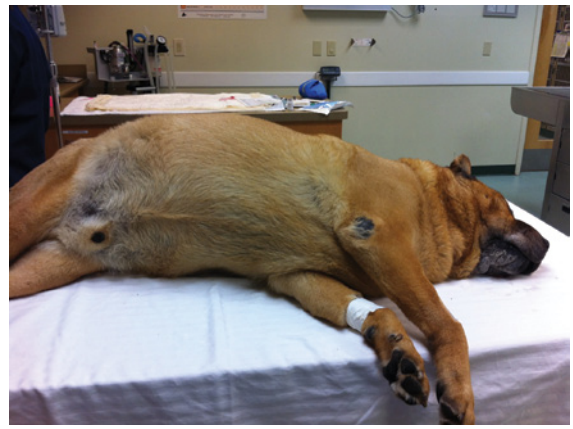

Figure 5.15 Distended abdomen in GDV patient. (Image courtesy Aran Gallagher)

Diagnosis

- Breed, history, and clinical presentation will aid in diagnosis.
- Radiographs will show gastric dilatation and possibly torsion (Figure 5.16).
- If passing a stomach tube is attempted, volvulus is suspected if the tube will not pass.

Treatment

- No matter what the order of treatment, the objectives remain the same. The goal is to correct circulatory shock, decompress the stomach, correct volvulus if necessary, and stabilize the patient.
- Decompression involves passing of a stomach tube (sedation may be required), temporary gastrotomy or trochar catheter, or surgical decompression.
- The stomach can be emptied through a stomach tube or surgically.
- Correction of volvulus requires a surgical fix. Additionally, if compromised a splenectomy can be performed during the surgical procedure.

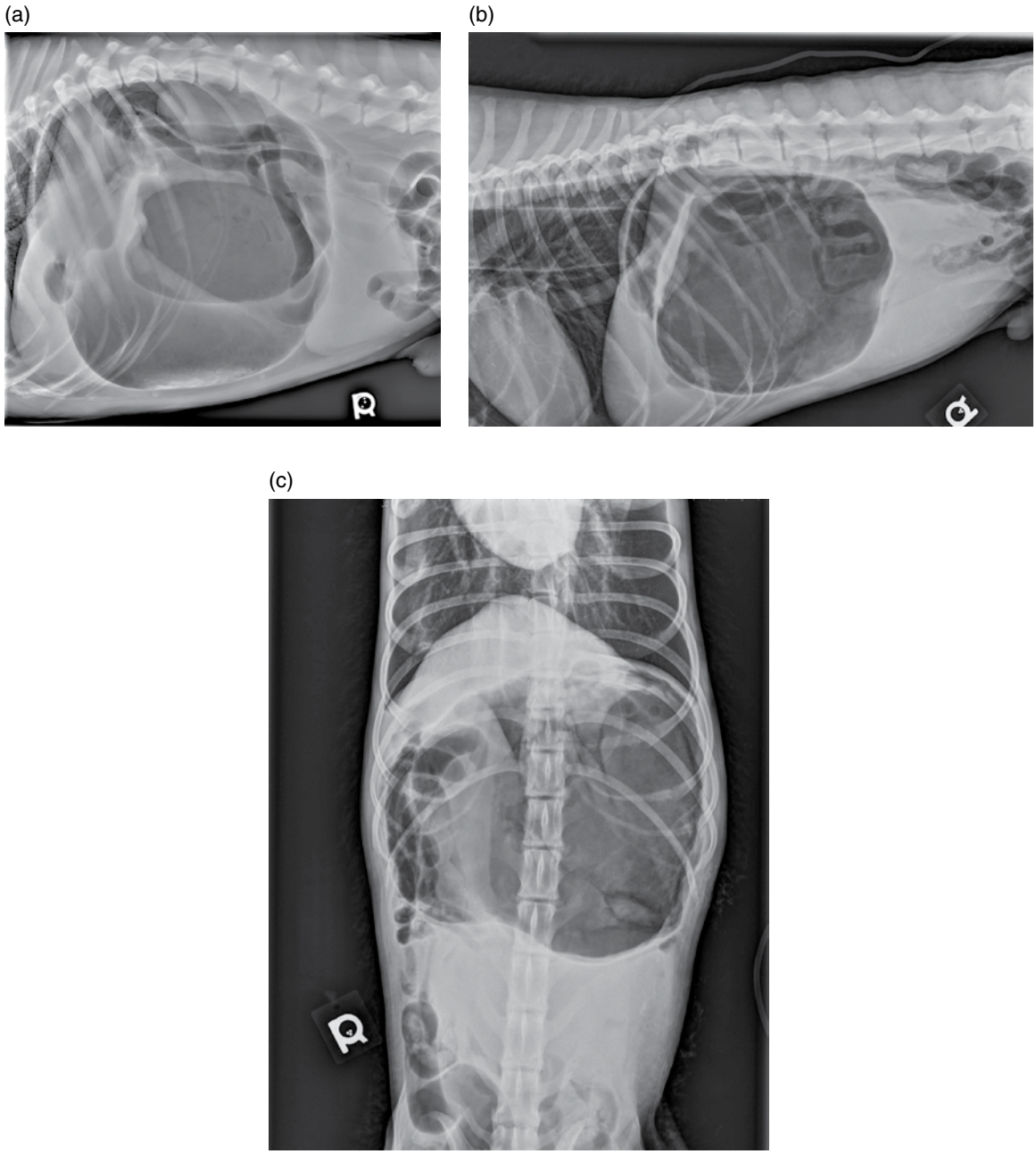

Figure 5.16 (a), (b), and (c) Radiographic images of GDV/bloat patients. (a) courtesy Mike Sagaert; (b) and (c) courtesy Brandy Sprunger.

- Performing a gastropexy, to prevent future volvulus, completes the surgery.
- Correction of circulatory shock and supportive therapy includes oxygen therapy, IV fluids, and drug therapy. Antibiotics for endotoxemia, antiarrhythmic drugs, and antacids are used with GDV patients. The patients are hospitalized for monitoring and recovery and are monitored for continued gastric distension.

Technician Duty Box 5.2

When dealing with emergencies, like a GDV, the veterinary technician needs to be able to triage the patient, get a quick physical exam, and begin diagnostics and treatment as soon as the veterinarian orders it. Once treatment is started the technician will need to be able to multitask, as time is of the essence.

Client Education and Technician Tips

- Predisposing factors include breed. Large breed, deep-chested dogs are at the highest risk. Although there is a breed predisposition, any breed of dog can suffer from a GDV.
- Ingestion of large amounts of water or food, especially when in association with exercise, is considered a risk factor for the condition.
- Other theories include altitude, stress, and the involvement of inflammatory bowel disease (IBD).
- Prevention includes prophylactic gastropexy in predisposed breeds, avoiding large meals, elevated feeding, avoiding large amounts of water to be ingested at once, and avoiding meals within a couple of hours of exercise.

TECH BOX 5.13: The difference between a GDV and "bloat" is the torsion that occurs in the case of GDV.

Intestines

Intussusception

Description

An intussusception is the inversion of one portion of intestines within another. Frequently the proximal portion telescopes into the more distal portion. With time the proximal portion can progress deeper into the distal portion. The condition is caused by inflammation and spasticity of the intestine or sudden diameter change. Inflammation and spasticity causes include foreign bodies, diarrhea, and parasites. Diameter changes are present at the ileocecal junction.

Clinical Signs

- Clinical signs vary based on where the intussusception occurs.
- Clinical signs include vomiting, anorexia, tenesmus, abdominal pain, diarrhea, and blood in the feces.

Diagnosis

- With abdominal palpation a cylindrical mass in mid-caudal abdomen may be felt commonly in cases of intussusception.
- Radiographs will show gas accumulation proximal to mass and usually an empty bowel distal to the mass. Contrast radiographs outline the obstruction.
- Ultrasound techniques can visualize the intussusception.

Treatment

- Some cases can be manually reduced externally through the abdomen.
- Some cases will spontaneously reduce themselves.
- Most cases will require surgical reduction. If the affected bowel is badly damaged, a resection of tissue will be necessary with an anastomosis.

Client Education and Technician Tips

- Prognosis is varied and is based on the integrity of the remaining bowel, where the intussusception occurred, the incidence of recurrence, and the duration of the condition.

Linear Foreign Bodies

Description

A linear foreign body is a type of foreign body caused by a long object, such as string, ribbons, or rubber bands. Part of the object becomes anchored in the proximal gastrointestinal tract, usually in the mouth under the tongue or between the teeth (Figure 5.17a). The rest of the object enters the intestines and the intestines will become bunched around the object. The tension will cause a cutting of the gastric and/or intestinal tissue. Gastric and intestinal perforations will lead to a life-threatening peritonitis. Because of the type of objects causing the obstructions, cats are more frequently diagnosed with this condition, although it can be seen in dogs.

Clinical Signs

- Common clinical signs of linear foreign bodies are similar to the clinical signs of any other foreign bodies: anorexia, vomiting, lethargy, abdominal pain, diarrhea, and dehydration.
- Perforations will result in severe abdominal pain, fever, vomiting, and collapse.

Diagnosis

- A string may be found anchored in the animal's mouth.
- Radiographs or ultrasound will show a plicated or accordion-like pleat to the intestines, with gas-filled loops of bowel.

Treatment

- Surgical removal of the foreign body and repair of the damaged tissue is required (Figure 5.17b).
- In cases of peritonitis, aggressive antibiotic therapy is necessary.

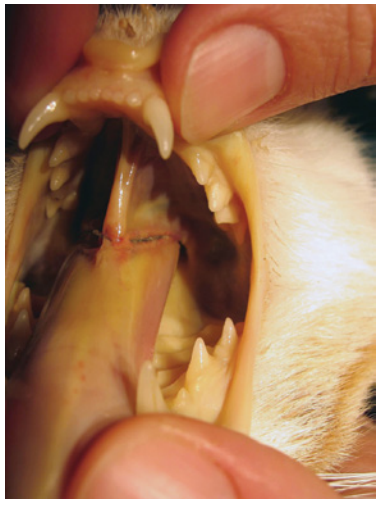

Figure 5.17a String linear foreign body sublingual laceration. (Image courtesy Kristen Mutchler)

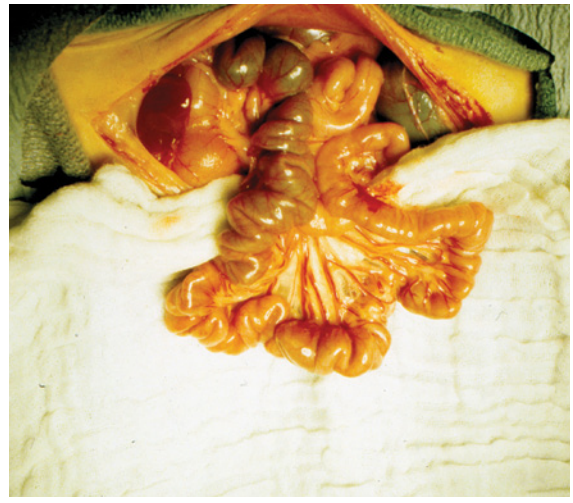

Figure 5.17b Linear foreign body surgical correction laparotomy. (Image courtesy Dr. Rucker, DVM/Bel-Rea Institute of Animal Technology)

Client Education and Technician Tips

- In the case of linear foreign bodies there is often a string visible in the mouth or protruding from the anus. It is imperative that this material not be pulled. The tension will lead to worsening damage in the animal's gastrointestinal tract.

TECH BOX 5.14: It is imperative that if there is any sign of a foreign body protruding from the animal, that it is not pulled. The resulting damage could be fatal.

Inflammatory Bowel Disease

Description

IBD is a chronic inflammatory condition of the stomach, small intestine, and/or large intestine. The disease is not very well understood and is thought to be idiopathic in most cases. Suspected factors include dietary sensitivity, autoimmune disease, drug allergies, infectious agents, and genetics.

Clinical Signs

- Frequent diarrhea is the most common sign of the disease.
- Other clinical signs include chronic vomiting, anorexia, weight loss, melena, and abdominal pain.

Diagnosis

- Thickened loops of the bowel may be palpable upon physical examination.
- Laboratory work-up is often unremarkable other than hypoproteinemia.
- Radiographs may show gas distension and the thickened loops of the bowel.
- Endoscopic visualization of the bowel endothelium will reveal inflamed tissue, granulation, erosion, and ulceration.
- Definitive diagnosis is determined through endoscopic intestinal biopsies, which will reveal inflammatory cell infiltration.

Treatment

- Treatment is aimed at reducing inflammation and reducing the immune response. The goal is to make the animal more comfortable, decrease clinical signs, decrease inflammation, and aid in weight gain.
- Antibiotics, in conjunction with a diet change, are often attempted first. The diet should consist of an easily digestible, hypoallergenic protein, low fat, and high fiber.
- Treatment of IBD often will involve corticosteroids to decrease inflammation and suppress the immune system with other immunosuppressive drugs used.
- Other medical treatments include antihistamines and analgesics.

TECH BOX 5.15: Treatment for IBD is mostly palliative, as the underlying cause is difficult to identify and correct.

Client Education and Technician Tips

- IBD is a condition that will require lifelong management.
- Prognosis depends on how well the animal responds to therapy.

Megacolon

Description

Megacolon is a condition seen in cats, usually middle-aged obese cats, where an abnormal dilatation of the colon is accompanied by colonic stasis. Megacolon will lead to constipation or obstipation, which can lead to a life-threatening emergency. Any disruption in colonic motility can cause complications. Motility changes lead to

dehydration, electrolyte abnormalities, shock, and organ dysfunction. Most cases are idiopathic, although contributing factors include obstructions like hairballs or pelvic strictures from a broken pelvis. Once a cat is diagnosed with a megacolon it is likely to recur.

Clinical Signs

- A cat with megacolon will be in and out of the litter box straining to defecate. It is important that tenesmus be differentiated from straining to urinate. If the cat is able to pass any feces it will be a small amount of liquid feces with blood and mucous. Inappropriate defecation may also be seen.

TECH BOX 5.16: The straining to defecate that results from megacolon must be differentiated from straining to urinate.

- Other clinical signs include discomfort, abdominal pain, lethargy, anorexia, and vomiting. In some cases the vomitus may contain fecal material.

Diagnosis

- History, physical examination, and clinical presentation will aid in diagnosis.
- Abdominal palpation will reveal a painful, hard fecal mass in the colon.
- Radiographs show a fecal mass in the colon, and the colon distended to a width more than the width of one lumbar vertebra (Figure 5.18).

Treatment

- First and foremost part of treatment is to stabilize patient. Fluid, electrolytes, and acid/base balances should be restored.
- An enema is needed to relieve the constipation or obstipation. Some patients may require sedation for this procedure.

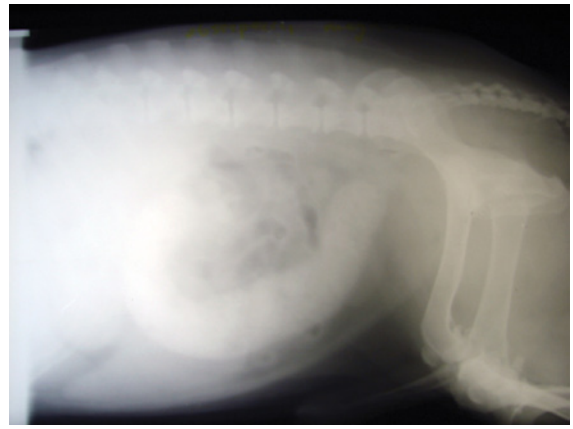

Figure 5.18 Megacolon radiograph. (Shutterstock image courtesy P. Fabian)

Technician Duty Box 5.3

Once a cat suffering from megacolon is stabilized, an enema is commonly the treatment of choice to relieve the constipation/obstipation. This is normally a two-person job (unless the cat is under anesthesia). The veterinary technician's role may be performing the enema or restraint of the cat.

- Severe cases may require surgical removal of fecal material.
- Surgical resection to remove the affected part of colon may be considered in some cases.
- It necessary to increase the time frames between recurrences. Goal of long-term care is to increase defecation frequency and make the stool softer and easier to pass. This is accomplished through SQ fluids given at home, soft foods, stool softeners, fiber, motility increasing drugs, and frequent enemas.

Client Education and Technician Tips

- Treatment and management are often lifelong.
- Close monitoring for fecal mats and skin irritation resulting from diarrhea is important in helping the cat defecate.

- When dealing with these painful cats in the clinic good restraint is a must.

Intestinal Neoplasia

Description

Although a small percent of all neoplasias are intestinal tumors, tumors can develop in the small intestine of cats and rectum and colon of dogs. Most have a tendency to be malignant. Lymphoma, lymphosarcoma, adenocarcinoma, mast cell tumors (MCTs), and leiomyosarcomas are diagnosed in dogs and cats. Lymphoma in cats may be linked to FeLV infections but is also found in FeLV-negative cats. There is also a possible link between severe IBD in cats and lymphosarcoma. Most patients diagnosed with intestinal neoplasias are older animals.

Clinical Signs

- Clinical signs are often vague and include abdominal pain, ADR, anemia/hypoproteinemia, V/D, anorexia, and weight loss.

Diagnosis

- Although clinical signs are usually nondescript, a history, clinical presentation, and thorough physical exam are a good starting point.
- Abdominal palpation with diagnostic imaging may be able to pinpoint masses in the abdomen.
- Definitive diagnosis is obtained via histologic evaluation of biopsies taken through endoscopy or laparotomy.

Treatment

- Surgical resection of the affected sections of the intestine.
- Chemotherapeutic regimens and radiation therapy may add to the success of treatment.

Liver

Cholangiohepatitis

Description

Cholangiohepatitis is a severe inflammation of the gallbladder, bile ducts, and liver. Cholangiohepatitis is more common in cats, although it is diagnosed in dogs, and is suppurative (infectious) or non-suppurative (non-infectious). There are multiple contributing factors, including parasites, ascending bacterial infection from the intestinal tract, gallstones obstructing the gallbladder, IBD, pancreatitis, dietary indiscretion, and neoplasias.

Clinical Signs

- Clinical signs include weight loss, anorexia, fever, icterus (Figure 5.19), V/D, dehydration, lethargy, and swollen, painful abdomen.
- The patient may be exhibiting signs of hepatoencephalopathy, including seizures, blindness, ataxia, and head pressing.

Diagnosis

- Blood chemistry panel, CBC, bile acids tests, and urinalysis will show changes consistent with liver disease and inflammation (Table 5.1).
- Diagnostic imaging, including radiographs and ultrasound, will be used to evaluate the liver, common bile duct, and gallbladder.
- Definitive diagnosis is obtained with the use of liver biopsies. These biopsies can be taken with the aid of ultrasound or may be taken at the time of exploratory surgery. The liver is often swollen and pale. The biopsies are also used to differentiate the type of cholangiohepatitis, as the treatment will differ dependent on type.

Treatment

- Most cases of suppurative cholangiohepatitis will respond to antibiotic treatment.

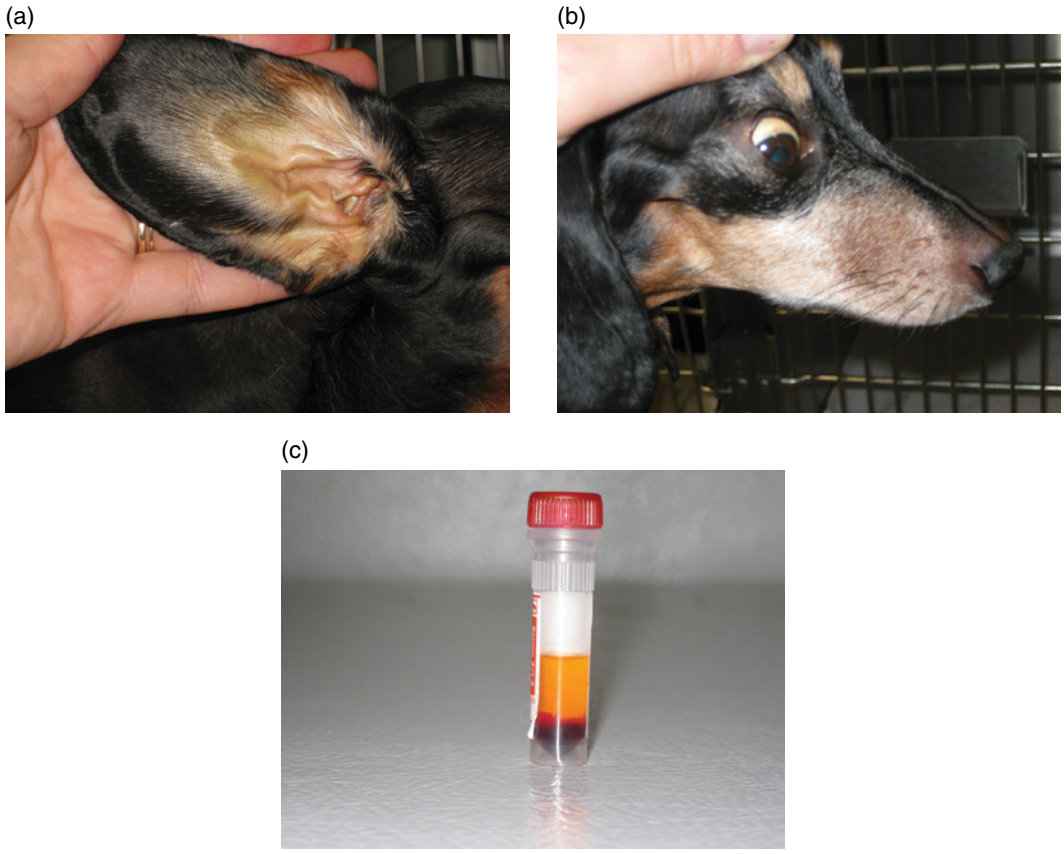

Figure 5.19 (a) Icteric pinna. (b) Icteric sclera. (c) Icteric serum. (Images courtesy A. K. Traylor, DVM, Microscopy Learning Systems)

Table 5.1 Liver disease laboratory work

	Cholangiohepatitis	Portal Systemic Shunt	Feline Hepatic Lipidosis
Blood cell count changes	Possible inflammatory leukogram	Non-regenerative anemia Poikilocytosis Target cells Leptocytes	Non-regenerative anemia Poikilocytosis Stress leukogram
PCV/TP	Hyperbilirubinemia		Hyperbilirubinuria
Blood chemistry	Increase in hepatic enzymes Increase in total bilirubin Increase in bile acids	Increase in ammonia Decrease in BUN Hypoalbunemia Hypoglycemia Hypocholesterolemia Decreased creatinine	Increase in hepatic enzymes Hypoalbuminemia Increase in ammonia
Urine changes	Bilirubinuria	Ammonium biurate crystals	Bilirubinuria

- Non-suppurative cases respond better to corticosteroid therapy.
- If the cause is a blockage, surgery may be needed to open up the blocked bile ducts.

Portal Systemic Shunt (PSS) or Portal Caval Shunt

Description

Portal systemic shunts are the most common circulatory anomaly in dogs and cats. Blood from the portal vein is shunted or bypasses the liver and returns to the vena cava and heart prior to filtration. Blood is returned to circulation before the liver is able to filter out toxins and nutrients. This condition is most often a congenital anomaly but can be acquired later in life as a result of chronic active hepatitis and cirrhosis. The anomaly vessel can be intrahepatic or extrahepatic. This blood vessel served a purpose of bypassing the liver in the fetus, yet should atrophy shortly after birth and allow blood through the liver in the neonate. Portosystemic shunts are seen in dogs and cats with congenital anomalies but seen more frequently in purebred dogs and male Himalayan and Persian cats. A PSS can, however, be diagnosed in mixed breed dogs and cats as well. Since the animal is born with the condition, it is usually diagnosed between 6 months and 1 year of age.

Clinical Signs

- The excess ammonia in the blood will cause a hepatoencephalopathy, especially postprandial, and is associated with high-protein meals. Clinical signs include seizures, head pressing, ataxia, stupor, and blindness.

TECH BOX 5.17: Hepatoencephalopathy is common with portal systemic shunts due to the excess ammonia in the blood.

- Animals with a congenital form of portosystemic shunts will tend to be small, underdeveloped, and thin as a result of malabsorption of nutrients meant for the liver. There is often a noticeable size difference between an animal with PSS and its littermates.
- These patients do not recover well from anesthesia, as the liver does not metabolize the drugs. It is therefore common to see a diagnosis of PSS after an ovariohysterectomy or orchiectomy.
- Other clinical signs include V/D, anorexia, pica, PU/PD in dogs (not common in cats,) hypersalivation, blindness, and excessive vocalization.

Diagnosis

- Blood chemistry panel, CBC, and urinalysis will show changes consistent with liver disease (Table 5.1; Figure 5.20).
- Radiography will reveal a small liver and enlarged kidneys.
- Definitive diagnosis is determined with the aid of digital imaging where blood flow patterns can be evaluated. The least invasive of these

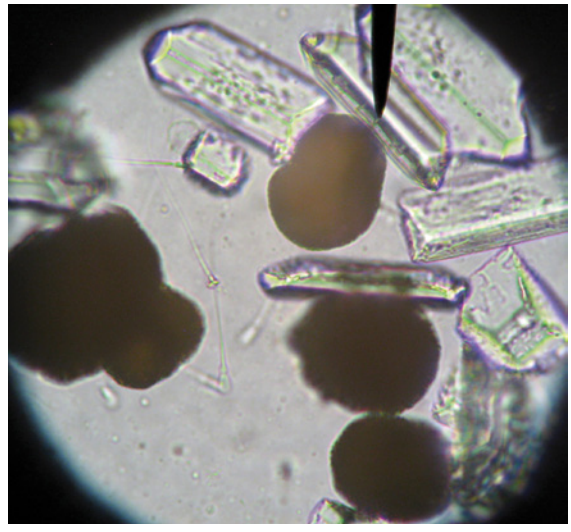

Figure 5.20 Ammonium biurate and struvite crystals seen in the urine of patients with a PSS. (Image courtesy Angela Taibo/Bel-Rea Institute of Animal Technology)

techniques is with an ultrasonography. Other diagnostic imaging techniques used include nuclear scintigraphy and portography.

Treatment

- Medical management can be used short term to stabilize the patient prior to surgery but is only palliative and temporary. This management method includes a low-protein diet to decrease the ammonia levels in the patient. Lactulose (a sugar solution) can be used to change the pH of the intestines, making the environment not conducive for normal intestinal bacteria. These bacteria are often associated with toxin production that the liver needs to filter. Antibiotics can also be used to decrease these bacteria if the lactulose and low-protein diets are not controlling the clinical signs.
- Surgical ligation of a shunt vessel is the treatment of choice and curative. Closely monitoring blood pressure during the surgery, and post-operatively, is essential, as occlusion of the shunt vessel can cause portal hypertension. Some patients may not be able to have their shunt fully closed because of hypertension. In some cases the clinical signs will resolve with partial ligation; others may require later surgeries to fully occlude the shunt. Other surgical complications include abdominal effusions, bloody V/D, abdominal pain, and ileus.

Technician Duty Box 5.4

One of the side effects of surgical ligation of a portal systemic shunt is portal hypertension. Veterinary technicians will be required to closely monitor the patient throughout the procedure and post-operatively.

Client Education and Technician Tips

- Prognosis with PSS is varied. Animals able to survive the post-operative recovery period and have full closure of the shunt have a good prognosis. Another factor in the recovery of the animal is the level of malnourishment at the time of diagnosis and treatment.

Feline Hepatic Lipidosis (FHL) or Fatty Liver Disease

Description

FHL is a condition seen in cats after a period of complete anorexia lasting days to weeks. It is the most common liver disease seen in cats. Starvation causes the body to mobilize fat and shift it to the liver for energy production, and cats do not metabolize fat well, thereby causing this condition; triglycerides infiltrate and accumulate in the liver. This condition is especially common in middle-aged, obese cats, as obese cats metabolize fat more readily than thinner cats. The accumulating fat leads to cholestasis and hepatic failure as the adipocytes cannot do the job of the hepatocytes. Hepatic lipidosis is life threatening if not treated. This condition is seen secondarily to anything that causes anorexia, including systemic diseases, stress, and neglect, but is often idiopathic.

TECH BOX 5.18: Hepatic lipidosis, the most common liver disease diagnosed in cats, is secondary to anything that causes anorexia in a cat.

Clinical Signs

- There has to be a history of anorexia in hepatic lipidosis patients.
- Clinical signs include V/D, melena, hepatoencephalopathy, icterus (Figure 5.21), rapid weight loss, lethargy, and hypersalivation.

Diagnosis

- History of anorexia and clinical presentation are important in developing a diagnosis.

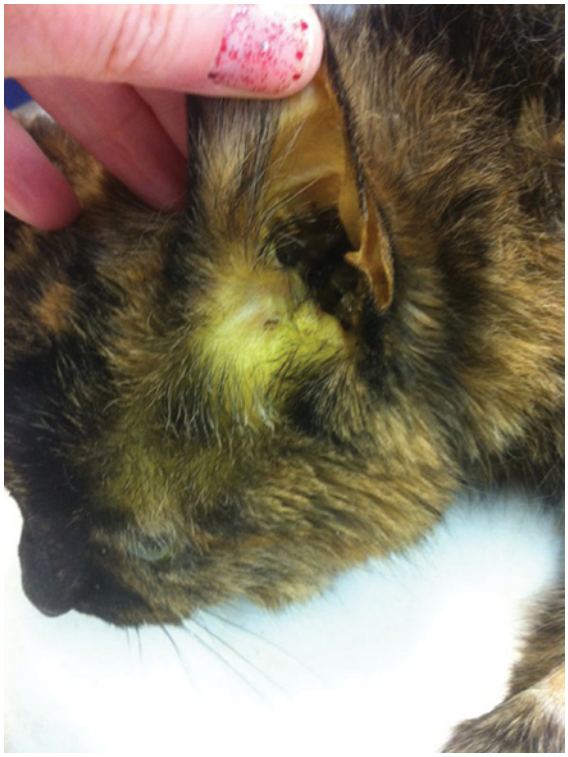

Figure 5.21 Icteric cat. (Image courtesy Deanna Roberts)

- Blood chemistry panel, CBC, and urinalysis are consistent with liver disease (Table 5.1).
- Radiographs and ultrasound will show an enlarged liver.
- Definitive diagnosis is obtained through biopsy. The biopsy will reveal adipocytes infiltrating the hepatocytes.

Treatment

- The key to treating this disease is to get the cat eating again. Fluid and nutrient support are important and may be needed long term. Force-feeding, often through external feeding devices, may be necessary. In some cases appetite stimulants will be prescribed for the cat.

Technician Duty Box 5.5

In order to see recovery in FHL patients the cat needs to receive much need nutrition and stimulation of his or her appetite. The veterinary technician will be experimenting with the cat's favorite foods and smelly foods. If this is unsuccessful the cat will need to be force-fed or fed through external feeding devices.

- Correction of electrolyte and acid/base imbalances is important.
- Diets low in fat and protein should be fed to the recovering cat.
- The vomiting seen with hepatic lipidosis can lead to gastritis. The treatment of gastritis will include antiemetics, antihistamines, antacids, and gastric mucosal protectants.

Client Education and Technician Tips

- Prognosis is good if diagnosed and treated early, although treatment can require long-term management. Once the cat is stabilized, at home the owner will perform nursing care, including force-feeding and SQ fluids.

Hepatic Neoplasia

Description

Primary hepatic tumors are not as common as tumors that have metastasized to the liver. There are many possible neoplasias found in the liver, some benign and some malignant. Primary tumors are found most frequently in older dogs and cats, and metastasis is more common in dogs than cats. Tumors include carcinoma, sarcoma, hepatocellular adenoma and carcinoma, biliary adenoma and carcinoma, and hemangiosarcoma, leiomyosarcoma, lymphoma, and lymphosarcoma. Neoplasias are likely to metastasize from the pancreas, thyroid, mammary glands, intestine, and bone. Tumors can be nodular, diffuse, or multifocal.

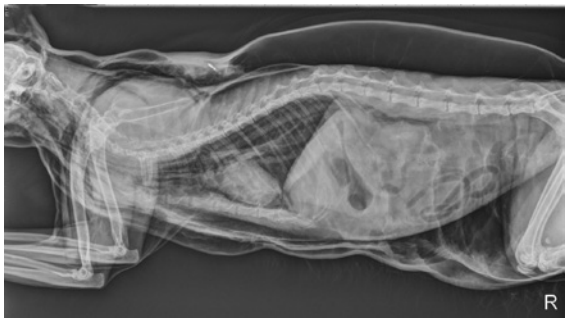

Figure 5.22 Radiograph of ascites seen in a liver disease patient. (Image courtesy Kristen Mutchler)

TECH BOX 5.19: Primary hepatic tumors are not as common as metastatic neoplasia.

Clinical Signs

- Clinical signs are very non-specific and will look like the clinical signs associated with any liver disease.
- Clinical signs include PU/PD, V/D, weight loss, anorexia, hepatoencephalopathy especially seizures, jaundice, hemorrhage, ascites (Figure 5.22), and hypoglycemia.

Diagnosis

- Laboratory values are consistent with liver disease.
- Diagnostic imaging, including radiographs and ultrasound, can be used to find the tumor, in addition to an exploratory laparotomy.
- A definitive diagnosis is obtained via biopsy and histopathology.

Treatment

- If only one lobe of the liver is involved, surgical removal would be indicated.
- If multiple lobes are involved or if a primary tumor, there is no effective treatment.
- Some tumors types will respond to chemotherapy.

Pancreas

Acute Pancreatitis

Description

Acute pancreatitis is characterized by abrupt inflammation that allows for an overproduction of digestive enzymes and resulting autodigestion of the pancreas and abdominal tissues. Tissue damage created by acute pancreatitis is generally reversible. Secondary complications include hepatitis, hepatobiliary obstruction, kidney failure, DIC, multiple organ failure, and death. With the seriousness of the complications, acute pancreatitis should be taken seriously and treated promptly. Pancreatitis is the most common exocrine pancreatic disease seen in both dogs and cats, although acute pancreatitis is more common in dogs. There is a breed predisposition in dogs including Schnauzers, Miniature Poodles, Cocker Spaniels, Yorkshire Terriers, Silky Terriers, and non-sporting breeds. Animals are usually middle-aged to older with risk factors including obesity, dietary indiscretion, idiopathic hyperlipidemia, high-fat diets, hypothyroidism, Cushing's disease, gastrointestinal infections, and certain medications.

TECH BOX 5.20: Acute pancreatitis can be life threatening and must be addressed promptly.

Clinical Signs

- Clinical signs include vomiting, diarrhea, anorexia, abdominal pain, lethargy, weakness, dehydration, and fever.

Diagnosis

- History of risk factors is important in identifying acute pancreatitis as a possible diagnosis.
- CBC and blood chemistry panel will aid a diagnosis but are not definitive. (See Table 5.2)

Table 5.2 Pancreas disease laboratory work

	Acute Pancreatitis	Chronic Pancreatitis
Blood cell count changes	Inflammatory leukogram Neutrophilia with left-shift Anemia Thrombocytopenia	Same as acute
PCV/TP	Decrease in PCV due to anemia Hyperbilirubinemia Hemoconcentration due to dehydration	Same as acute
Blood chemistry	Increase in pancreatic enzymes Likely increase in hepatic enzymes Azotemia Hypercholesterolemia Hypoglycemia or hyperglycemia	Variable Flare up increase in pancreatic and hepatic enzymes Late stage damage decrease in pancreatic enzymes
Electrolytes	Hypochloremia Hypophosphatemia Hyponatremia	Same as acute

- Increased amylase and lipase are suggestive of acute pancreatitis but not definitive. Levels should be three times above normal to consider pancreatitis.
- Radiographs are an important part of gastrointestinal work-up but not definitive for pancreatitis.
- Abdominal ultrasound is more useful than radiography. Acute pancreatitis can be diagnosed but not eliminated as a diagnosis.
- A canine/feline pancreatic lipase (cPL/fPL) ELISA is currently the test of choice, as this tests specifically for pancreatic lipase (Figure 5.23).
- An exploratory laparotomy will provide a definitive diagnosis, and especially if biopsies and cultures are taken, but the test is invasive.

Figure 5.23 Positive IDEXX cPL in a pancreatitis patient. Image courtesy Brandy Sprunger.

The pancreas will appear lumpy, nodular, cystic, inflamed, red, necrotic, or hemorrhagic.

Treatment

- The animal should be taken off of oral feeding (NPO) for the short term only, no longer than needed (24–48 hours maximum.) Early oral feeding helps the prognosis, but vomiting needs to be controlled to feed the patient and feeding tubes may be necessary.
- It is essential to stabilize the patient and restore fluid, electrolyte, and acid/base balances. The patient needs to be rehydrated with IV fluids.
- Pain control is an essential component of pancreatitis treatment.
- Other drugs used for treatment include anticholinergics, antibiotics, and antacids. Heparin and plasma may be used to prevent DIC, and insulin may be needed in cases of hyperglycemia.

Client Education and Technician Tips

- Long-term care will include low-fat diets and the management of obesity and other diseases. A cPL/fPL can be used to monitor the patient.

Chronic Pancreatitis

Description

Chronic pancreatitis is a continued inflammatory disease in which the pancreatic tissue damage becomes irreversible. Over time the damaged pancreatic tissue will lose function and fail to secrete an adequate amount of digestive enzymes. This condition stems from primary pancreatic disease or secondary to systemic diseases and can be seen in both dogs and cats. Secondary complications include exocrine pancreatic insufficiency (EPI), acute flare-ups, and diabetes mellitus (DM).

TECH BOX 5.21: Chronic pancreatitis often leads to other pancreatic conditions.

Clinical Signs

- Clinical signs are often intermittent, mild, and often go unnoticed, as acute pancreatitis is easier to recognize and diagnose.
- Acute flare-ups will present the same as animals with acute pancreatitis.
- As the disease continues and digestive enzymes decrease, the patient will present with clinical signs consistent with EPI.

Diagnosis

- History of acute pancreatitis or other pancreatitis risk factors is important in making a diagnosis, as clinical signs can be vague.
- Diagnostic tests will vary based on the extent of tissue damage, yet are consistent with either an acute pancreatitis diagnostics or EPI and DM testing (Table 5.2).

Treatment

- Treatment will vary based on the extent of tissue damage, and yet is consistent with the treatment for acute pancreatitis, EPI, or DM.
- Animals are managed with a diet change, specifically a low-fat diet, and should be monitored closely. Obesity should be managed and if drug therapy has led to pancreatitis an alternative should be found.

Client Education and Technician Tips

- Because the line between acute and chronic pancreatitis can be difficult to differentiate, it is not usually of significance. Animals are treated as they present.

Exocrine Pancreatic Insufficiency or Pancreatic Maldigestion

Description

EPI is characterized by an insufficient production of pancreatic digestive enzymes necessary to ensure adequate digestion and absorption of nutrients. Undigested food in the intestine leads to bacterial overgrowth that can further complicate the condition. This is a condition requiring lifelong management and more often seen in dogs than cats. EPI is diagnosed most frequently in German Shepherds and Rough Coated Collies. Causes linked include pancreatic hypoplasia, neoplasia, in dogs idiopathic acinar cell atrophy, and in cats chronic pancreatitis.

Clinical Signs

- The most common clinical sign of EPI is marked weight loss despite polyphagia. Animals are usually bright and alert otherwise.
- The feces of animals with EPI are excreted in large volumes, steatorrhic, loose, pale, and more odorous than normal. Patients may be coprophagic as a result of intense hunger.
- Animals with EPI tend to have skin conditions and a poor hair coat that may be attributed to malnutrition. Patients may also appear to be oily, especially in the perineal area, as the undigested fat will stain their skin and hair as the feces are passed.

Diagnosis

- There are fecal tests that can be run in-house that look for undigested nutrients and trypsin in the feces, but these are often unreliable.
- Definitive diagnosis is obtained via a reference laboratory blood test called a trypsin like immunoreactivity (TLI) test.

Treatment

- Animals with EPI need an enzyme substitute sprinkled on their food. The enzyme will replace the lacking enzymes and will digest the food for the animal. Most enzyme substitutes come from dried porcine pancreas, although plant substitutes exist as well.
- Animals with EPI should be placed on a low-fat diet.

Client Education and Technician Tips

- Enzymes must go on every meal, otherwise the animal will not absorb the nutrients from the meal. It is recommended the enzymes go on the food 20–30 minutes before the meal is fed.
- The enzyme substitutes come in several forms, and evidence suggests the powdered substitutes work best.
- Enzyme substitutes can be expensive and are a lifelong treatment.
- As a result of pancreatic damage, diabetes mellitus may be seen concurrently with EPI in patients.
- It is important to differentiate between maldigestion and malabsorption in the diagnosis process. Animals will present with similar clinical signs, but they are different conditions with different treatments and prognoses. Malabsorption is a condition where the patient has the enzymes needed for digestion but lacks the ability to absorb the products of digestion into the bloodstream.

Pancreatic Neoplasia

Description

Pancreatic neoplasias can be primary tumors or secondary tumors that have metastasized from other locations. Primary tumors include benign tumors such as pancreatic adenoma and pancreatic nodular hyperplasia, malignant pancreatic adenocarcinoma, or insulinoma (cats). Adenocarcinomas are the most common pancreatic neoplasm diagnosed in dogs and cats.

Clinical Signs

- Clinical signs are vague and often confused with clinical signs of pancreatitis. These clinical signs include fever, jaundice, weight loss, vomiting, and abdominal pain.
- Insulinomas will create a severe hypoglycemia causing sudden collapse, extreme weakness, and lethargy.

Diagnosis

- Chemistry panels and CBC are often nonspecific other than in the case of insulinomas, where hypoglycemia will be pronounced.
- Diagnostic imaging is a better tool, although radiographs are often of some help. Ultrasound is generally a more definitive tool. Ultrasound can also aid in obtaining biopsies.
- An exploratory laparotomy is most often the way to obtain a definitive diagnosis.
- Many cases are diagnosed at point of necropsy.

Treatment

- Pancreatic neoplasias often carry a grave prognosis, as there are limited treatments available.
- If possible the tumor should be surgically removed.
- Radiation and chemotherapy have shown little success.

- Antibiotics and anti-inflammatories can be used to reduce inflammation and the signs associated with it.
- Insulinomas respond best to steroid therapy coupled with chemotherapy. It is important to manage blood sugars.

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Urinary Tract Disease

chapter 6

The urinary tract includes the organs associated with urine production and elimination: the kidneys, ureters, urethra, and urinary bladder. These organs play a major role in the elimination of metabolic by-products and other waste products, regulation of water balance, regulation of red blood cell production, and regulation of blood pressure. Although some urinary tract diseases can be mild, many conditions will be life threatening when these important functions are compromised.

Bacterial Cystitis or Urinary Tract Infection

Description

Bacterial cystitis is inflammation and infection of the urinary bladder caused by a micro-organism.

Clinical Signs

- Signs include polyuria, pollakiuria, hematuria (Figure 6.1), and dysuria.
- Animals may also exhibit inappropriate urination, straining to urinate, and excessive licking of their urogenital area.
- Pain upon urination or caudal abdominal palpation can also be seen with the condition.

Diagnosis

- A urinalysis will show changes consistent with a urinary tract infection (Figure 6.2). (See Table 6.1)
- Changes may be seen on CBC. (See Table 6.1)
- Bacterial culture will help determine the specific bacterial agent.

Technician Duty Box 6.1

It will be anticipated that veterinary technicians have the ability to collect urine through catheterization, manual expression, cystocentesis, or free catch collection methods.

Small Animal Pathology for Veterinary Technicians, First Edition. Amy Johnson.

Companion website: www.wiley.com/go/johnsonvettechpath

Figure 6.1 Hematuria. (Image courtesy Amy Johnson and Bel-Rea Institute of Animal Technology)

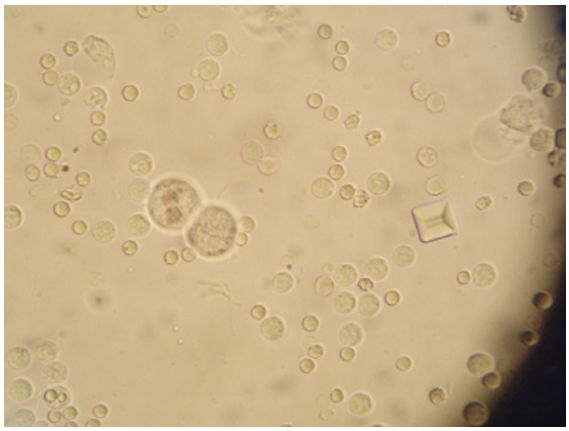


Figure 6.2 Urinary sediment with red and white blood cells. (Image courtesy Deborah Shaffer)

Table 6.1 Bacterial cystitis laboratory work

Blood cell count changes	Possible leukocytosis
Urine changes	Hematuria Increase in WBC Proteinuria Bacteriuria

Treatment

- Bacterial infections can be treated with antibiotics. A culture and sensitivity will determine the best antibiotic to use.
- Carnivore urine should be on the acidic end of the pH scale, but certain bacteria will thrive in more alkaline pH ranges. Modifying the pH of the urine with use of diet or urinary acidifiers may be needed in treatment of bacterial cystitis.

Client Education and Technician Tips

- Recurrent infections may be secondary to diabetes mellitus because the glucose present in the urine creates the ideal environment for bacteria to grow. Immunosuppressive diseases, hyperthyroidism, and hyperadrenocorticism can also be causes for secondary bacterial cystitis.

TECH BOX 6.1: Bacterial cystitis happens secondarily to many systemic conditions.

Pyelonephritis

Description

Pyelonephritis is a renal infection characterized by inflammation of the kidney and renal pelvis. It is usually as a result of an ascending bacterial infection, although some systemic bacterial or viral infections can spread to the kidneys via the bloodstream. Causes of ascending infections include impeded flow of urine and immunosuppressive conditions in older animals. Some cases are idiopathic.

Clinical Signs

- Clinical signs are usually less dramatic than those seen with acute kidney infections.

Table 6.2 Pyelonephritis laboratory work

Blood cell count changes	Leukcytosis Left-shift Anemia due to kidney failure
PCV/TP	Decrease in PCV due to anemia
Blood chemistry	Azotemia
Urine changes	Hematuria Increase in WBC Proteinuria

- Clinical signs include fever, kidney or flank pain, lethargy, vomiting, and PU/PD.
- CBC, blood chemistry, and urinalysis will show changes (Table 6.2).
- Urine culture and sensitivity may come back positive, although in many cases pyelonephritis cultures will be negative for the presence of bacteria.

Diagnosis

- Blood work, urinalysis, and clinical signs can be used for diagnosis.
- Renal ultrasound and pyelogram will show changes in size of kidney or size and shape of renal pelvis.

Treatment

- Antibiotics can be used to treat the infection as long as it is bacterial. These infections usually require higher and longer dosing than an animal with cystitis would need.

TECH BOX 6.2: Pyelonephritis needs to be treated more aggressively and longer than bacterial cystitis.

- Fluid therapy is an important part of therapy in many animals, as it may help prevent the infection from progressing to kidney failure. It will also help animals already in renal failure with better profusion and lessen uremia.
- With the worst case scenario, patients may require a nephrectomy.

Client Education and Technician Tips

- Reculture is important to see if antibiotic therapy is effective.
- Many animals are at risk for reoccurrence and should be monitored closely.

Urolithiasis (Urinary Calculi or Urinary Stones)

Description

Urolithiasis is the development of stones in the urinary tract. Stones can develop in the kidney, bladder, urethra, and ureter. These stones are composed of various minerals affected by pH similar to crystals. For the stones to form the patient must have increased mineral in the urine, adequate pH, and adequate time. A nidus may also be present to allow the stone to form.

Clinical Signs

- Clinical signs include dysuria, hematuria, and pain in caudal abdomen.
- Clinical signs become worse if urinary obstruction occurs.

Diagnosis

- Radiographs may allow for visualization of the stone. For radiolucent or small stones

Table 6.3 Urolithiasis laboratory work

Blood cell count changes	Possible leukocytosis
Urine changes	Hematuria Increase in WBC Crystaluria

cystoscopy, ultrasound, or exploratory cystotomy may be required.

- Microscopic urine evaluation can reveal crystaluria, although this is not definitive proof that stones have formed (Table 6.3).

Treatment

- Stones are either removed via cystotomy or in some cases dissolved with diet (Figure 6.3).

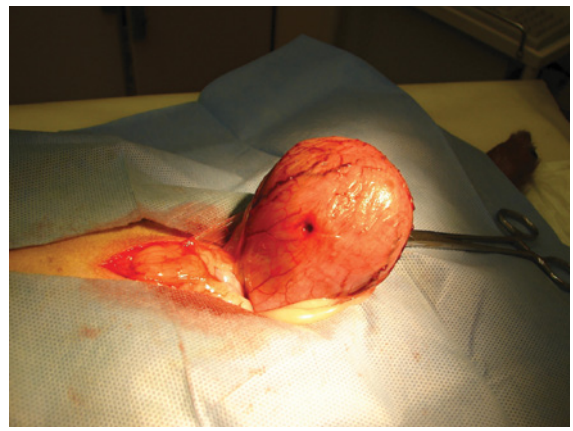
Client Education and Technician Tips

- It is important to determine the type of stone to correctly prevent reoccurrence. Once the stone is removed the type is determined either by an in-house stone analysis test kit or is sent off to a reference lab for determination (Figure 6.4).

TECH BOX 6.3: In order for appropriate treatment, stones need their composition analyzed.

- Prevention includes diet change to alter pH, allow the patient more access to water, and allow for more frequent urination. Increased water consumption will reduce the concentration of minerals in the urine and the time the urine is in the urinary bladder by increasing urinary frequency.

(a)



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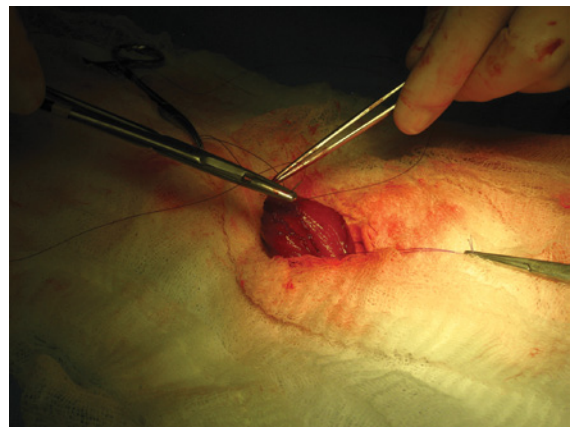

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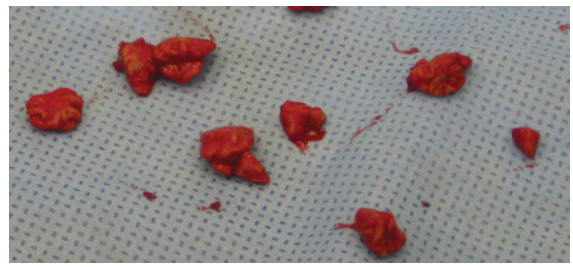

Figure 6.3 (a) Cystotomy with uroliths in urinary bladder. (Shutterstock photo courtesy P. Fabian) (b) Cystotomy. (c) Uroliths removed from the urinary bladder. (Images courtesy A. K. Traylor, DVM, Microscopy Learning Systems)

Figure 6.4 (a) Calcium oxalate dihydrate stones. (b) Miscellaneous uroliths. (c) Canine uroliths. (d) Canine struvite urolith. (Images courtesy Amy Johnson, Angela Taibo, and Bel-Rea Institute of Animal Technology)

Urinary Obstruction or Blocked Tom (Feline)

Description

Urinary obstruction is caused by factors obstructing the urethra and outflow of urine. Obstructions can be uroliths, crystalline debris, mucus, blood clots, or inflammation. This condition is more common in males than females, because of a narrower and longer urethra. An obstructed urethra will cause urine retention and will lead to an expanding bladder and buildup of toxins and other products the kidneys are responsible for eliminating. If the obstruction is not relieved, the animal will suffer from fluid and electrolyte imbalances, kidney failure, and possible bladder rupture. If not corrected this is a life-threatening condition.

TECH BOX 6.4: Urinary obstruction is a life-threatening emergency that requires prompt veterinary attention.

Clinical Signs

- Straining to urinate and frequent attempts to urinate are common clinical signs of this condition. Cats with obstructions will be in and out of the litter box, while dogs will frequently "want out" to urinate.
- Other clinical signs include painful attempts at urination signaled by crying or vocalization, hematuria, inappropriate urination, abdominal pain, anuria, and excessive licking of urogenital area.
- Anorexia, lethargy, vomiting, dehydration, coma, and death will mark later signs of the disease.
- Hydronephrosis will result from the urinary obstruction and further perpetuate the kidney damage.

Diagnosis

- A presumptive diagnosis is made on clinical signs and history, especially when abdominal palpation reveals a painful, hard, and distended urinary bladder.
- Radiographs or ultrasound will confirm a distended bladder and hydronephrosis.
- Urinalysis, CBC, and blood chemistry will show changes consistent with urinary retention (Table 6.4).

Table 6.4 Urinary obstruction laboratory work

Blood chemistry	Azotemia Metabolic acidosis
Electrolytes	Hyperkalemia
Urine changes	Increase in USG Hematuria Increase in WBC Possible crystaluria

Treatment

- Animals need to be unobstructed using a tomcat catheter and flushing stones out of the urethra with saline and a sterile water-based lube.
- An urethrotomy may be required if the flushing cannot dislodge the obstruction.
- Calculi flushed back into bladder may require a cystotomy to be removed, as it should not be excreted through the urethra again.
- A cystocentesis can temporarily relieve pressure, but is not curative.
- Fluids, analgesics, and anti-inflammatories should be used as well.

Technician Duty Box 6.2

Animals with urinary or renal dysfunction will require maintenance of urinary catheters and closed collection systems as well as quantification of urine output.

Client Education and Technician Tips

- Extreme caution should be used when palpating the bladder, as the fragile bladder may rupture.

TECH BOX 6.5: If a urinary obstruction is suspected, never attempt to express the urinary bladder.

- If uroliths or calculi are the cause of obstruction, analysis is necessary to determine the best treatment and prevention methods. Diet change or urinary acidifiers are commonly needed.
- A perineal urethrostomy may need to be performed in the case of recurrence.

Feline Urinary Tract Disease (FLUTD)

Description

FLUTD is a collections of conditions associated with the bladder and urethra in cats. FLUTD has many similarities to urinary obstructions but includes more complications and causes. Causes of FLUTD include feline idiopathic cystitis (FIC) or interstitial cystitis. Patients with FIC have the clinical signs of cystitis with no identifiable cause. Other causes of FLUTD include urinary calculi obstruction, urethral obstruction, bacterial cystitis, urinary tract neoplasia, or urinary tract infection.

Clinical Signs

- Although the syndrome of FLUTD has many possible causes, presenting clinical signs are all very similar.
- FLUTD includes clinical signs of hematuria, urinary straining, inappropriate urination, polyuria, pollikuria, anuria, urinary blockage (males), and excessive licking of urogenital area.
- Although FLUTD presents in all cats, the most common signalment is middle-aged, obese, indoor cats eating a dry diet. Other factors that may contribute to the condition are stress, multicat households, and changes in routine.

Diagnosis

- FLUTD is a collection of many bladder and urethral disorders, making it necessary to identify the underlying cause.
- Urinalysis is a starting point in diagnosis. If bacterial infection is present, a culture and sensitivity should be performed.
- Radiographs can be used to determine presence of calculi, uroliths, other neoplasia, or anatomical abnormalities.
- Many cases will have no identifiable cause.

Treatment

- Treatment is dependent on the cause, if the cause can be identified. Otherwise treatment is strictly symptomatic.
- Many companies have formulated urinary diets that can be fed, many of which will help with treatment and prevention of calculi and uroliths. Diets should also be consistent without a lot of change.
- Other treatments may include cystotomy, antibiotics, anti-inflammatory drug therapy, and fluid therapy.

Client Education and Technician Tips

- In some cases this may be a one-time condition, while unfortunately others will have recurrent problems.
- Owners can minimize problems by avoiding changes in routine, providing fresh water at all times, providing an adequate number of litter boxes, and keeping the litter boxes clean.

Acute Renal Failure (ARF)

Description

Acute renal failure is a sudden shutdown of nephrons resulting from major kidney damage. This quick decline in kidney function is life threatening. Some cases, with immediate correction, may be reversible. Causes include toxins and poisons, ischemia, nephrotoxic antibiotics, bacterial infections such as leptospirosis, hypoperfusion, snake venom, urinary obstruction, and other systemic diseases. This condition is divided into three phases: induction, maintenance, and recovery. The induction phase is characterized by kidney damage followed by a decrease in kidney function. The kidneys cannot concentrate the urine and azotemia is detectable. The maintenance phase is when irreversible kidney damage

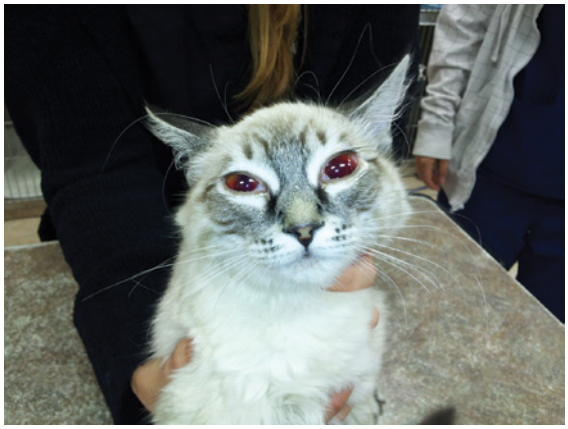

Figure 6.5 Uremic uveitis. (Image courtesy Brandy Sprunger)

occurs, and not all patients survive this phase. The recovery phase, if they survive, is marked by nephron repair. However, full recovery is not always seen.

Clinical Signs

- Decrease in urine output and kidney filtration leads to a buildup of toxins in the bloodstream, referred to as uremic poisoning. It is this poisoning that causes anorexia, oral ulceration, uveitis (Figure 6.5), lethargy, and vomiting/diarrhea, an odor of ammonia on the breath, and gastrointestinal bleeding.
- Other clinical signs include pain upon abdominal palpation, oliguria, and anemia.
- Fluid and electrolyte imbalances are seen because of declining kidney functioning.

Diagnosis

- History is an important factor in diagnosis. Finding a cause can help point to the diagnosis.
- Blood chemistry, electrolyte panel, CBC, and urinalysis will help develop a diagnosis (Table 6.5).

Table 6.5 Acute renal failure laboratory work

Blood cell count changes	Anemia
PCV/TP	Decrease in PCV due to anemia
Blood chemistry	Azotemia
Electrolytes	Hyperkalemia Hypernatremia
Urine changes	Initial decrease in USG

Technician Duty Box 6.3

Animals with urinary or kidney disorders will require frequent monitoring of urine specific gravity (USG), TP, and PCV.

Treatment

- Treatment is directed at correcting fluid, acid/base, and electrolyte imbalances, and IV fluids are an important part of the treatment. Diuretic therapy can help with the promotion of urine output.
- If possible the specific cause needs to be identified and corrected.
- Antiemetics can be used to combat the vomiting.

Client Education and Technician Tips

- The prognosis is poor but potentially reversible with aggressive and immediate therapy.
- Signs of disease are not apparent until two-thirds–three-fourths of the nephrons are nonfunctional.

TECH BOX 6.6: Acute renal failure may be reversible, while chronic renal failure is long-term permanent damage.

Chronic Renal Failure (CRF), Chronic Kidney Disease (CKD), or Chronic Renal Disease (CRD)

Description

Chronic renal failure is a result of progressive renal degeneration. It is commonly seen with age, although other causes include chronic bacterial or viral infections, inherited kidney malformation, hypertension, and immune mediated diseases. The disease has four stages. In stage I, kidney damage is present but there is no azotemia or clinical signs. In stage II azotemia is present without clinical signs. In stage III both azotemia and clinical signs are present. Finally, in stage IV severe azotemia accompanies clinical signs.

Clinical Signs

- Similar to acute renal failure, chronic renal failure leads to uremic poisoning. A decrease in urine filtration results in anorexia, lethargy, vomiting and diarrhea, dehydration, oral ulceration, gastrointestinal bleeding, abdominal pain, and an ammonia odor on the breath.
- An increase in urine output is seen because of the nephron's inability to concentrate urine.
- Hypertension associated with CRF can lead to blindness.
- Weak bones and pathological fractures may be seen as a result of calcium and phosphorous leaching from bones.

Diagnosis

- Blood chemistry, CBC, electrolyte panel, and urinalysis will show changes consistent with CRF (Table 6.6).
- Ultrasound or radiographs can be used to evaluate kidney size.

Table 6.6 Chronic renal failure laboratory work

Blood cell count changes	Anemia
PCV/TP	Decrease in PCV due to anemia
Blood chemistry	Azotemia
Electrolytes	Hyponatremia Hypokalemia Hyperphosphatemia
Urine changes	Decrease in USG

Treatment

- Treatment depends on the stage of CRF, and the patient may take weeks or months to respond. Treatment is strictly supportive and meant to improve the quality of life. CRF cannot be cured or reversed and will progressively get worse as more renal function is lost. Euthanasia should be considered when the quality of life declines.
- Patients need to be reevaluated on a regular basis. Blood pressure, electrolyte levels, acid/base balance, and packed cell volume all need to be monitored and corrected.
- Ash must be limited in the diet and animals fed low amounts of high-quality protein to decrease the strain put on the kidneys. CRF patients should have constant access to water.
- Fluids are an important part of therapy and can be given IV in the clinic, or the owners can be trained to give SQ fluids at home.
- Antacids and antiemetics can be used to help control vomiting.

Client Education and Technician Tips

- Clinical signs of kidney disease are not apparent until greater than 70% of the nephrons are non-functional.

TECH BOX 6.7: Once a patient is showing signs of kidney disease, more than 70% of kidney function has been permanently impaired.

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Reproductive Disease

chapter 7

Unlike other systems, the reproductive tract is significantly different in males and females. The anatomy, function, and hormonal influences are different in each gender, creating common disease variances in males and females. The function of the reproductive system in the female is production of the egg, maintenance of the pregnancy, and support of the neonate. In the male the reproductive function is production of sperm. Diseases of the reproductive tract are in most cases only seen in intact animals. This makes ovariohysterectomy (OHE) and orchiectomy important, not only as a treatment but also for prevention of diseases. If the animal is not being bred, it is in the best interests of their overall long-term health to be altered.

Vaginitis

Description

Vaginitis is defined as inflammation of the vagina usually due to micro-organism. Most commonly seen is bacterial vaginitis, although viral infection, foreign body, neoplasia, hyperplasia, and conformation abnormalities are other possible causes. This condition is seen in both dogs and cats, although more common in dogs. Vaginitis can be seen in any age of animal. In young dogs the condition is referred to as "puppy vaginitis." Adult onset vaginitis is more common in spayed animals than intact animals, and it is not common in breeding females.

Clinical Signs

- Clinical signs include vaginal discharge, excessive licking, visible inflammation, and dragging urogenital area on surfaces.
- Pollakiuria or urinary incontinence may also be seen.

Diagnosis

- Clinical history and vaginal exam are commonly used for presumptive diagnosis.
- Blood work may show inflammation with no other hematological signs. This will help

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differentiate between vaginitis and an open pyometra.
- Vaginal cytology will show the presence of excessive bacteria; a culture of the exudate can be helpful.
- Ultrasound and abdominal radiographs can be used to visualize any foreign bodies, confirmation of abnormalities, tumors, or to rule out pyometra. Additionally, vaginoscopy and digital examination can assist in determining cause.

Treatment

- Antibiotics will be prescribed in the case of bacterial infections. Cleaning of the vagina and vulva and douching with non-alcohol-based otic cleaners will help restore vaginal pH.
- Some cases will resolve with first heat cycle, therefore delaying the OHE may be recommended.

TECH BOX 7.1: Some cases of vaginitis may resolve on their own if the OHE is postponed.

- Vulvoplasty surgery may be indicated in cases of abnormal anatomy.

Pyometra

Description

Pyometra is a bacterial infection of the uterus that causes the uterus to fill with purulent material. The bacterial infection is secondary to hormonal linked hyperplasia of the endometrium. Thickened walls of the endometrium create a good environment for bacterial growth. Pyometras are most common in older (> 6 years) intact females. In breeding animals a pyometra will be seen 4–6 weeks post-partum.

This condition is more common in dogs than in cats and is diagnosed in one-fourth of intact females. An open pyometra is when the cervix remains open, allowing for drainage. A closed pyometra is a bigger concern, as the cervix is closed and purulent material accumulates in the uterus, similar to an abscess. Animals with a closed pyometra are most often more severely ill than those with an open pyometra.

TECH BOX 7.2: A pyometra is due to a secondary bacterial infection caused by hormonal dysfunction.

Clinical Signs

- A mucopurulent or serosanguineous vaginal discharge is seen with an open pyometra.
- Other clinical signs include PU/PD, fever, vomiting, anorexia, lethargy, and distended abdomen.
- Septicemia and shock will be seen in severe cases.

Diagnosis

- Ultrasound or abdominal radiographs will show an enlarged uterus. Radiographically it is difficult to differentiate the uterus from the small intestine if the diameter of the uterus is less than that of the small intestine.
- Cytology can be performed on the vaginal discharge of open pyometra patients. Discharge will typically show degenerated neutrophils and both intracellular and extracellular bacteria. Cytology of vaginitis patients will be similar, making it difficult to differentiate based on cytology alone.
- Hematologic findings show inflammation yet are non-specific for pyometra. Blood work may help rule out other causes for the clinical signs.

Treatment

- OHE treatment of choice. The animal should be stabilized by correcting fluid, electrolyte,

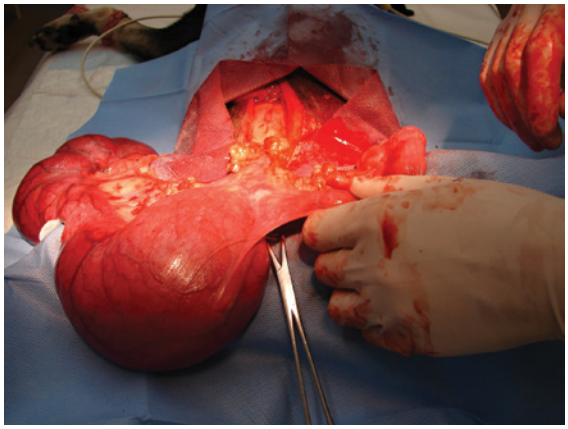

Figure 7.1 OHE of pyometra patient. (Shutterstock image courtesy P. Fabian)

and acid/base imbalances prior to surgery (Figure 7.1).
- Medical treatment should only be conducted when trying to save the uterus in breeding animals, and treatment includes antibiotics, hormone therapy, and fluids. Medical treatment always requires close monitoring, as it is not always successful.

TECH BOX 7.3: The treatment of choice with a pyometra is an OHE; medical treatment should only be attempted when breeding integrity is to be maintained.

Client Education and Technician Tips

- Pyometras are prevented by altering and should be done in all animals not used for breeding purposes.
- A pyometra is a fatal condition if not treated.
- Pyometra should be considered as a differential diagnosis with any of the clinical signs in an intact female dog; she is in need of immediate medical attention.

Dystocia

Description

Dystocia is defined as a difficult or abnormal birth. Dystocias involve maternal factors and/or fetal factors. Maternal factors include anatomical malformation like pelvic strictures and uterine stasis. Fetal factors involve size and presentation of fetus. Dystocias are seen in both dogs and cats and are more common in brachycephalic breeds, like Bulldogs.

Clinical Signs

- Dystocia should be considered in a pregnant female that has been in active labor with contractions and no birth within 1–2 hours.
- Other clinical signs include abnormal vaginal discharge, no contractions or birth within 24 hours of rectal temperature decrease, animals with history of dystocia, vocalizations, licking vulva excessively, and overdue (70 days after breeding).

Diagnosis

- Physical exam and history are an important starting point for diagnosis.
- Radiography and ultrasound can determine the cause of the dystocia, which is necessary for moving forward with treatment.

Treatment

- In the case of non-obstructive causes, medical therapy can be tried before going to cesarean section (C-section). Oxytocin or calcium gluconate can be used to promote uterine contractions.
- With an obstructive dystocia a C-section must be performed after the patient is stabilized.

Technician Duty Box 7.1

C-section procedures will require the help of the veterinary technician to receive the neonates and stimulate respiration. These newborn puppies or kittens will need mucus cleared from their airways, manual stimulation, and in some situations medical intervention.

Client Education and Technician Tips

- Previous dystocias should be monitored closely, as it is likely to recur with future pregnancies.
- Anesthesia needs to account for fetal absorption of drugs. Anesthetic protocols need to be adjusted for the safety of the unborn fetuses.
- Predisposed breeds may schedule C-section prior to natural birth occurring.

Mastitis

Description

Mastitis is inflammation of one or more of the mammary glands associated with an ascending infection via the teat canals. Mastitis is more common in dogs than cats and is usually seen postpartum; causes include poor sanitation, nursing trauma, and systemic infections. Normal gastrointestinal flora is a commonly associated opportunistic pathogen due to fecal contamination. Cases can lead to septic shock and can be life threatening.

TECH BOX 7.4: Good sanitation is a key factor in preventing mastitis.

Clinical Signs

- Clinical signs include red, painful, inflamed, firm mammary glands with possible discharge from the teats.
- There may be changes in milk color or consistency or lack of milk production.
- Fever, anorexia, dehydration, and lethargy can be seen with the patient.
- Puppies or kittens may be neglected leading to failure to thrive or death.

Diagnosis

- Clinical signs and history are important factors in determining a presumptive diagnosis.
- A microscopic exam of milk shows inflammatory cells and possibly bacteria. Culture and sensitivity will aid in diagnosis and treatment.
- Hematologic work-up will show changes consistent with inflammation and infection.

Treatment

- Antibiotics will be prescribed as determined by the culture and sensitivity.
- Hot packing the affected gland(s) will help with the discomfort and inflammation.

Technician Duty Box 7.2

Inflammatory conditions, including mastitis, will require hot packing of the affected area in order to speed recovery and make the animal more comfortable.

- Abscessed glands will require surgical lancing of the gland to allow for flushing and drainage.

Client Education and Technician Tips

- While patients are being treated, there may be a need for neonatal nutrient supplementation.

Mammary Neoplasia

Description

Neoplasia of the mammary glands is seen in both dogs and cats. Mammary tumors are more

frequent in dogs than cats but will see a higher malignancy rate in cats than dogs.

The causes of mammary tumors are unclear but hormones play a role, as well as genetics and nutrition. Tumors are classified as carcinomas, sarcomas, mixed tumors, and benign adenomas. Feline adenocarcinomas are most commonly present in the thoracic or anterior glands of older intact cats. In canine patients the two posterior glands are most commonly affected. In either species evidence suggests the younger an animal is spayed the less the risk of developing mammary neoplasia. OHE before the first estrus cycle decreases the risk greatly; there is some reduction after first estrus cycle.

TECH BOX 7.5: Canine mammary tumors are the most common neoplasm in intact female dogs.

Clinical Signs

- Palpable mass under the skin in abdominal area. A single mass or multiple nodules may be present. Although less common, inflammation and ulceration may be present (Figure 7.2).
- Other clinical signs include anorexia, weight loss, lethargy, and swelling in hind limbs.

Diagnosis

- Physical exam and palpation of mammary mass will point to mammary tumors, but definitive diagnosis requires histopathology.
- Thoracic radiographs and abdominal ultrasound will help determine if the tumor has metastasized.

Treatment

- Surgical removal is the treatment of choice (Figure 7.3).
- Chemotherapeutic regimens have questionable response.

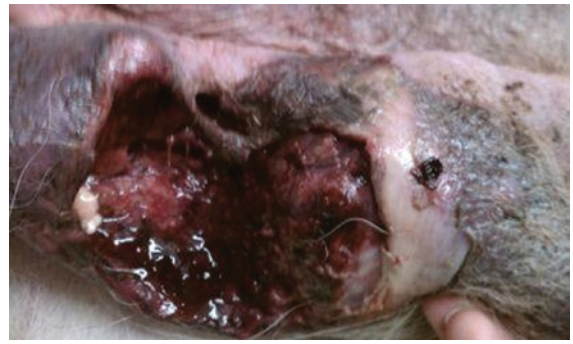

Figure 7.2 Necrotic mammary mass. (Image courtesy Kristen Mutchler)

Figure 7.3 Mammary mass excision surgical prep. (Shutterstock image courtesy Henk Vrieselaar)

Client Education and Technician Tips

- Prognosis is based on the type and size of the tumor, metastasis, and progression.

Prostate Disease

Description

Prostate diseases encompass multiple problems associated with the prostate gland in male animals causing enlargement and inflammation. Problems

with the prostate gland are more common in dogs than other species. Prostate problems include bacterial infections and abscesses, enlargement due to excessive hormone levels, and cysts. Benign prostatic hyperplasia (BPH) is seen in cases of excessive testosterone where squamous metaplasia is an estrogen induced enlargement. Cysts are obstruction of ducts emptying secretions to the urethra. Neoplasia of the prostate is also seen; this will be covered with neoplastic disease.

Clinical Signs

- Urinary complications are present with prostate disease because of the obstruction of the urethra. Straining to urinate; short, small stream of urine; hematuria; and bacterial kidney or bladder infections characterize urine outflow problems.
- Tenesmus will present, as the enlarging gland obstructs the emptying of the colon.
- Other clinical signs include pain in abdomen, anorexia, lethargy, and fever (with bacterial infections).

TECH BOX 7.6: Prostate disease will often cause problems with urination and defecation.

Diagnosis

- Abdominal and rectal palpation, ultrasound, and abdominal radiography can be used to check size, shape, and symmetry of the prostate gland. Pain should also be accessed with palpation.
- Prostate fluid normally moves into the urinary bladder, meaning bacterial prostatitis will result in a bacterial cystitis. Urinalysis will reveal presence of bacteria or inflammation.
- Cytology from the prostate is collected through urethral catheterization and milking the prostate, or through fine needle aspirate (FNA) or biopsy.

Treatment

- Although treatment is largely dependent on cause, neutering is the treatment of choice.
- Hormone therapy is used in cases brought on by hormonal imbalance.
- Bacterial infections and abscesses require aggressive antibiotic therapy, as it is difficult for antibiotics to penetrate into the gland.
- Prostate cysts will commonly require drainage.

Client Education and Technician Tips

- The best prevention for any prostate disease is orchiectomy.

Testicular Disease

Description

Testicular disease is defined as any disease of the testicles including bacterial, viral, or fungal infections, trauma, or torsion. Testicular problems are more common in dogs than cats and can involve any part of the testicle.

Clinical Signs

- Common clinical signs are pain and swelling of the testicles and licking of the scrotum.
- Anorexia, lethargy, and fever are also seen with testicular disease.

Diagnosis

- Palpation of testicles and ultrasound will aid in identification of location and structures involved.
- Semen evaluation and bacterial culture are performed to look for bacterial causative agents.
- Because brucellosis is a common cause of testicular disease a brucellosis test may be ordered.

- FNA or biopsy of tissue may be performed. Samples are often collected at time of neuter, if neutering is an option.

Treatment

- Treatment is dependent on cause, although neutering is the best treatment option.
- Cold packs can be used to decrease inflammation and discomfort.
- Antibiotics are used in cases of bacterial infections and are determined by culture and sensitivity.
- In cases of unilateral disease if the owner wishes to preserve breeding integrity, removal of the affected testicle can be performed, although with any testicular disease fertility prognosis is questionable.

Client Education and Technician Tips

- Prevention of testicular diseases is achieved through orchiectomy.

Male Reproductive Neoplasia

Description

Tumors of the testicle are more common in dogs than cats. Testicular neoplasia is usually seen in dogs over 4–6 years and is most commonly found in retained testicles rather than in descended ones. Half of all of retained testicles will become neoplastic. Leydig cell tumors (LCTs) or interstitial cell tumors (ICTs) are often benign and small and are commonly an incidental finding.

Seminomas are the second most common testicular tumor, developing in the testicular tissue, and are most often benign. Sertoli cell tumors are found in the seminiferous tubules and carry the greatest likelihood of metastasis. Sertoli cell tumors are the most common to develop in undescended testicles.

Prostatic adenocarcinoma is rare but highly invasive and likely to metastasize quickly. These tumors are also found more commonly in dogs than cats and develop in dogs over 9 years old.

TECH BOX 7.7: Male reproductive disease is more common in dogs than in cats.

Clinical Signs

- Clinical signs of a testicular tumor include hard/firm testicles, nodular-feeling testicles, or one testicle larger than the other one. With an undescended testicle a swelling or firm mass may be palpable in the inguinal canal.
- A sertoli cell tumor produces estrogen resulting in feminization of the male dog, including bilateral symmetrical hair loss, enlarged mammary glands, and in severe cases bone marrow suppression. Common with an estrogen-producing tumor is a red stripe of inflammation seen along the prepuce. Estrogen may also cause hyperplasia of the prostate gland.
- An LCT will cause a soft swelling with one or more round masses palpable.
- Seminomas are most often subclinical and rarely palpable, although signs of pain from the pressure of the growing tumor may be noticed.
- Clinical signs of a prostate tumor are very similar to signs of other prostate disorders. These signs include dysuria, pollakiuria, urinary incontinence, hematuria, and tenesmus. As the disease progresses lethargy, anorexia, and pain may be present.

Diagnosis

- Most testicular tumors can be palpated. Ultrasound can be used to confirm mass and location, and biopsy can be taken at time of tumor removal.
- Prostate tumor diagnosis is similar to any prostate disease, including cytology of prostate fluid, ultrasound, and abdominal and rectal palpation.

- Thoracic radiographs and ultrasound can be used to check for metastasis.

Treatment

- Neutering is the treatment of choice with testicular tumors and usually curative. If owners want to breed the healthy testicle can be left. It is important to neuter dogs with cryptorchid testicles as the incidence of cancer is so high. Cryptorchidism is genetic and these dogs should not be bred.
- Prostate tumors do not respond well to treatment, although neutering will help alleviate clinical signs. Surgical removal of the prostate is recommended if the cancer has not metastasized.

Client Education and Technician Tips

- The best prevention of either testicular or prostate tumors is orchiectomy.

TECH BOX 7.8: Most common treatment and prevention of any reproductive disease is spay or neuter.

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Endocrine Disease

chapter 8

The endocrine system is one of the systems involved in maintaining internal homeostasis. With the use of hormones the endocrine glands regulate functions such as growth, development, and metabolic activities. The pituitary gland and hypothalamus secrete hormones that target other glands in the body. The hormones regulate themselves through a negative feedback system; problems anywhere along the line can cause malfunctions.

Hyperthyroidism

Description

Hyperthyroidism is an excessive production of thyroid hormones T3 (triiodothyronine) and T4 (thyroxine), increasing the metabolic rate of animals with this condition. It is common in older cats and frequently caused by benign thyroid adenomas. While rare in dogs, the most common cause is a malignant thyroid adenocarcinoma.

TECH BOX 8.1: Hyperthyroidism is most often diagnosed in cats as a result of a benign thyroid adenoma.

Clinical Signs

- A palpable thyroid gland is a sign of disease, as a normal thyroid gland is not often palpable. The growing gland may also cause a change in vocalization.
- Increased thyroid hormone will cause a dramatic weight loss despite an increased appetite. Other clinical signs associated with the higher metabolic rate will include tachycardia, hypertension, systolic heart murmur, hyperactivity, and excessive grooming.
- Thyroxine has a diuretic effect causing PU/PD.
- Vomiting, anorexia, lethargy, and excessive vocalization may be seen as well.

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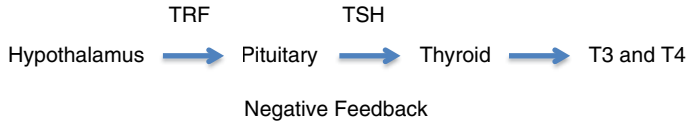

Figure 8.1 Thyroid function (TRF, thyrotropin-releasing factor; TSH, thyroid-stimulating hormone).

Diagnosis

- An increased blood level of T3/T4 may point to thyroid disease, although T4 is a better indicator. This is not definitive, as some cats with hyperthyroidism will have a normal T4.
- A T3 suppression test is a better indicator of thyroid disease in patients with a normal T4. A baseline T4 is obtained, T3 is injected, then a post-T4 is drawn. Normal animals should have a decreased T4 production, but hyperthyroid patients will have no suppression of T4 (Figure 8.1).

Treatment

- Radioactive iodine (I131) is the treatment of choice. An injection is used to destroy unhealthy thyroid tissue. One treatment is usually all that is necessary. The downfalls include the cost of treatment and the necessary hospitalization time. It can take up to 15 days in a treatment facility for the radioactive material to be excreted from the patient's body. Cats should also be screened for chronic renal failure prior to treatment, as hyperthyroidism can mask the signs of renal failure.

Technician Duty Box 8.1

Specialty clinics will perform radioactive iodine treatments on patients with hyperthyroidism. Veterinary technicians will aid in the treatment and monitoring of these cats and disposal of radioactive items.

- Oral administration of an antithyroid drug is a palliative method of treatment. Treatment with these drugs is fairly effective but comes with side effects that include vomiting, lethargy, and anorexia. Methimazole is a common drug used but may have to be discontinued, as it may cause excoriations of the head and neck. Another disadvantage is the daily administration of the drug for the rest of the animal's life. Drugs can be administered orally, rectally, or with a flavored paste. Regular monitoring with blood work is necessary to regulate dosage.
- The third option is a thyroidectomy: removal of the diseased thyroid tissue. Surgery is usually curative but comes with risks. The parathyroid glands sit in close association with the thyroid and an accidental parathyroidectomy must be avoided. The parathyroid glands regulate blood calcium levels, making it necessary to closely monitor blood calcium levels for hypocalcemia. Other risks include removing excessive thyroid tissue, causing an iatrogenic hypothyroidism, and the anesthetic risk factors to the patient.

Hypothyroidism

Description

Hypothyroidism is characterized by a decrease in thyroid hormone, which results in a reduction in metabolic rate. Hypothyroidism is rare in cats and is usually iatrogenic, resulting from overtreatment of hyperthyroidism. The condition is more common in dogs 2–6 years, with most cases resulting from primary thyroid disease. Lymphocytic thyroiditis is an autoimmune

condition implicated as a cause of primary thyroid disease, as well as idiopathic atrophy, where thyroid cells are replaced by adipocytes.

Hypothyroidism can be seen in any breed but is more often diagnosed in Labrador Retrievers, Golden Retrievers, Doberman Pinschers, Cocker Spaniels, and Boxers.

TECH BOX 8.2: Hypothyroidism is a disease more common in dogs than cats.

Clinical Signs

- The most common presentation of hypothyroidism is changes in the hair coat and skin. A poor, dry hair coat, thin breaking hairs, hair loss on the tail commonly called "rat tail," bilateral symmetrical alopecia, and excessive shedding are seen. The skin becomes thickened and hyperpigmentation of the skin is possible, especially on the ventral side of the abdomen and medial thighs.
- Other clinical signs include weight gain, PU/PD, lethargy, weakness, and exercise intolerance. Hypothermia will cause the animal to seek warm places.

Diagnosis

- T3/T4 may be decreased, although this is not specific as certain drugs and genetics can cause low numbers.
- Thyroid-stimulating hormone (TSH) levels will be increased in a hypothyroid patient.
- Hypothyroid patients will have decreased fat metabolism, causing a hyperlipidemia and hypercholesterolemia, as well as increased potassium levels because of decreased urinary excretion. Hypothyroid patients may also be anemic (Figure 8.1).
- TSH stimulation test is more definitive. A baseline T4 is obtained, after which TSH is injected, and a post-T4 is obtained. In normal patients T4 should double. In primary hypothyroid patients there is little or no increase (Figure 8.1).

Treatment

- Hypothyroidism is treated with a synthetic thyroid hormone (thyroxine).

Client Education and Technician Tips

- Hypothyroidism is one of most commonly overdiagnosed diseases because the signs are vague.

Hyperadrenocorticism or Cushing's Disease/Syndrome

Description

Cushing's disease is an increase in cortisol production from the adrenal glands. This condition is the most commonly diagnosed endocrine disease in adult dogs but is rare in cats. Most cases are pituitary-dependent hyperadrenocorticism (PDH) and attributable to a benign pituitary tumor that secretes excessive adrenocorticotropic hormone (ACTH). A smaller number of cases are caused by an adrenal tumor (AT) that secretes excessive cortisol and is seen more frequently in female dogs. Some cases are iatrogenic Cushing's resulting from prolonged steroid use or suddenly stopping steroid treatment. The cause must be determined to appropriately treat the disease.

Clinical Signs

- Cortisol suppresses the immune system, causing predisposition to bacterial infections, especially urinary tract infections.
- Cortisol causes a relaxation of skeletal muscles, and patients may be weak and have a distended abdomen or "pot-bellied" appearance (Figure 8.2).

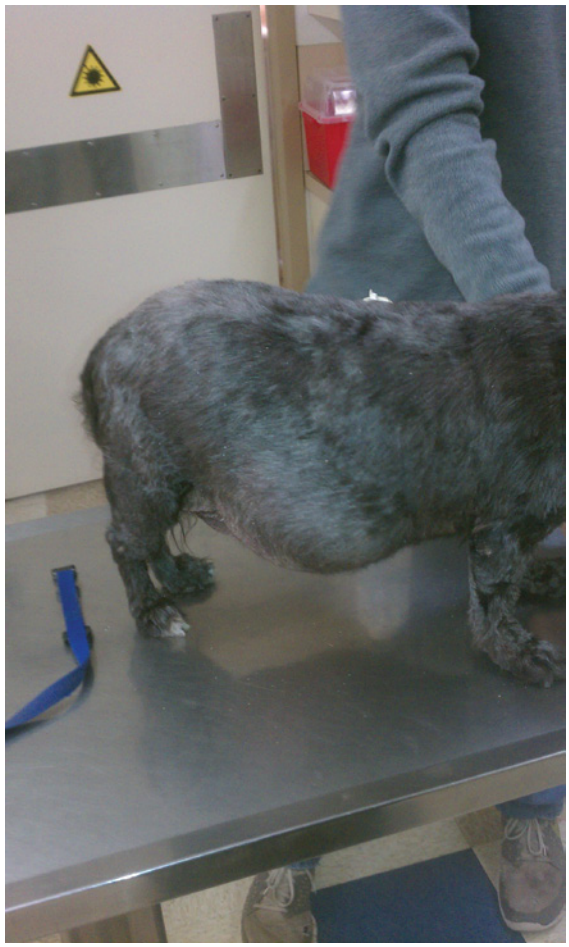

Figure 8.2 Distended abdomen in a Cushing's patient. (Image courtesy Hillary Price)

- Other clinical signs include PU/PD, polyphagia, weight gain, hair loss on body, thin skin, bruising, panting, and heat intolerance.

Diagnosis

- Radiographs or ultrasound will show a hepatomegaly, calcification of the adrenal glands, or enlarged adrenal glands. One enlarged gland is indicative of an adrenal tumor, while bilateral enlargement is more suggestive of a PDH.
- Blood chemistry will include an increased alkaline phosphatase (ALP) and alkaline amino transferase (ALT), cortisol, cholesterol, and blood glucose in addition to a decrease in blood urea nitrogen (BUN). CBC indicates a stress leukogram and regenerative anemia. Urinalysis consistent with Cushing's will have large amounts of dilute urine and urinary tract infection with inflammation, bacteria, and protein (Table 8.1).
- There are two steps involved when testing for Cushing's: one is to get a diagnosis and the second is to determine the source.

Table 8.1 Hyperadrenocorticism laboratory work.

Blood cell count changes	Stress leukogram Regenerative anemia
PCV/TP	Hemoconcentration if dehydrated
Blood chemistries and electrolytes	Increase in ALP, ALT, cortisol, cholesterol Hyperglycemia Decrease in BUN Hyponatremia Hyperkalemia
Urine changes	Decrease in USG Increase in WBC Bacteriuria Proteinuria

TECH BOX 8.3: It is important to determine the cause of Cushing's disease to be able to appropriately treat it.

- The two options for obtaining a diagnosis include an ACTH stimulation test and a

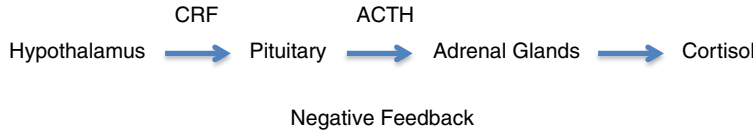

Figure 8.3 Adrenal function (CRF, corticotropin-releasing factor; ACTH, adrenocorticotropic hormone).

low-dose dexamethasone suppression (LDDS) test. An ACTH stimulation test is done by obtaining a baseline cortisol, injecting ACTH, and then measuring post-cortisol. A Cushing's patient will have a hyperresponse to the ACTH and the cortisol levels will be more than triple the levels of a normal patient. In the LDDS test a baseline cortisol is obtained, LDD is injected, and a post-cortisol is drawn. The LDD should suppress ACTH in a normal patient. If the condition is as a result of PDH, the tumor is resistant to the normal negative feedback system and no suppression will happen. If an AT is the cause, the tumor secretes cortisol autonomously and there is no suppression (Figure 8.3.)

- Differentiating causes is done with a high-dose dexamethasone suppression (HDDS) test. A baseline cortisol is measured, a much higher dose of dexamethasone is injected, and a post-cortisol is drawn. Suppression of cortisol is noted with a PDH as the dose is enough to suppress the tumor and therefore decreases ACTH. No suppression of cortisol is noted with an AT as the pituitary gland is already suppressed and the tumor is secreting autonomously.

Treatment

- Treatment is dependent on the cause.
- A PDH is treated with drugs that will cause selective destruction of adrenal cortex, therefore reducing the amount of cortisol produced.
- Adrenal tumors do not respond as well to medical treatment as a PDH. Adrenal tumors are surgically removed if possible, although complications involve developing a hypoadrenocorticism.

Client Education and Technician Tips

- One of the functions of cortisol is to keep blood glucose high. The increased blood sugar levels can cause diabetes mellitus to result secondarily.

Hypoadrenocorticism or Addison's Disease

Description

Adrenal failure will result in a failure to secrete adequate amounts of cortisol and aldosterone, known as hypoadrenocorticism or Addison's disease. Addison's disease is seen in young to middle-aged dogs and more often in females. Cases may be idiopathic, iatrogenic, or because of autoimmune disease, metastasis, hemorrhage, or infarction.

Clinical Signs

- Clinical signs of Addison's disease are very vague. Patients are often labeled as ADR, "Ain't doing right." Signs can be intermittent and vary in severity.
- Polyuria results from a lack of aldosterone production and will cause hyponatremia,

hypovolemia, and hypotension. Polyuric animals will be polydipsic to rehydrate.
- Renal excretion of potassium is inhibited, causing a hyperkalemia. This increase will attribute to bradycardia and electrocardiogram (ECG) abnormalities.
- Other clinical signs include anorexia, weight loss, vomiting, abdominal pain, anorexia, and lethargy.

Diagnosis

- There are many changes in the blood work of an Addison's patient. A CBC will show a lack of a stress leukogram that might normally be seen in a sick patient due to absence of cortisol, and anemia in the case of gastrointestinal bleeding. Electrolyte panels and chemistry screening will reveal hyperkalemia, hyponatremia, hypochloremia, hypoglycemia, and azotemia (Table 8.2).
- An ACTH stimulation test can be performed. A baseline cortisol is obtained, ACTH is injected, and a post-cortisol is measured. Addison's patients will have no increase in post-cortisol (Figure 8.3).

Table 8.2 Hypoadrenocorticism laboratory work

Blood cell count changes	No stress leukogram Anemia if GI bleeding
PCV/TP	Hemoconcentration if dehydrated
Blood chemistries and electrolytes	Azotemia Acidosis Hypoglycemia Hyponatremia Hyperkalemia
Urine changes	Decrease in USG

Treatment

- To stabilize a patient in an Addisonian crisis, fluids, electrolytes, and dextrose therapy may be necessary, and in addition steroids are needed to manage shock.
- Replacement of hormones including mineralocorticoids and glucocorticoids will be necessary in maintenance of the patience. Supplementation is available in a daily oral pill or an injection given approximately once monthly.
- Salting the food can be done to help replace the sodium lost in the urine.

Client Education and Technician Tips

- Cortisol helps animals deal with stress, and Addison's patients are unable to cope well with stress because of the lack of cortisol. Clinical signs may worsen when the animal is stressed. It is important to keep their environment as stress free as possible and to avoid sudden changes in routine.

TECH BOX 8.4: Addison's disease has very vague clinical signs, making pinpointing a diagnosis difficult.

Diabetes Mellitus or Sugar Diabetes

Description

Diabetes mellitus is a failure of the pancreas to secrete adequate amounts of insulin, causing an increase in blood glucose levels. Islet cell destruction leads to a decline or an absence of insulin production, and the excessive glucose will show in increased levels in the blood and urine. This condition is seen in dogs and cats. Animals are usually middle-aged to older animals, with obesity a factor in the condition. Following disease onset, it is a condition the animal and owners will deal with for life.

Clinical Signs

- There will be changes in the blood and the urine of the patient, most notably an increased level of glucose and ketones (Table 8.3).
- Glucosuria will lead to an increased likelihood of urinary tract infections, as the sugar in the urine creates an environment conducive for bacterial growth. The hypertonic glucosuria will create an osmotic diuresis, where water is pulled into the urine, resulting in PU/PD (Table 8.3).
- Hyperglycemia will also lead to polyphagia, weight loss, lethargy, and weakness.
- It is normal for the lens of the eye to absorb glucose from the blood. In diabetic patients the excess glucose absorbed will cause an increase in water pulled into the lens, creating bilateral cataracts. These cataracts can eventually lead to blindness in DM patients.
- Hepatomegaly is also seen as a result of lipid accumulation and fat mobilization.

Diagnosis

- Clinical signs, history, and physical exam can be used in conjunction with laboratory results to diagnose DM (Table 8.1).
- Chronic hyperglycemia and glycosuria, especially after fasting, should put DM on the rule out list. Ketonuria is another good indication of DM.

TECH BOX 8.5: Glucosuria and ketonuria are good indications an animal may have diabetes mellitus.

- In borderline cases or stressed cats fructosamine is a more valid indicator. Fructosamine is a blood protein that binds to glucose. It is able to provide a more accurate picture of an average glucose level over several weeks. It is also preferred in cats, as stress doesn't affect fructosamine as it does glucose.

Table 8.3 Diabetes mellitus laboratory work

PCV/TP	Hemoconcentration if dehydrated
Blood chemistries	Hyperglycemia
Urine changes	Decrease in USG Glycosuria Ketonuria

Treatment

- DM patients will need to be treated with insulin injections on a daily basis. These animals will need to be closely monitored to determine if the insulin dose is effective. Glucose curves and/or fructosamine tests will look at trends in the animal's blood glucose levels. The monitoring may be more frequent at first while the dose is adjusted to the animal.
- A weight loss plan needs to be put in place for obese animals. DM animals should be fed a low-fat, low-carb diet with complex carbohydrates instead of simple ones.

Client Education and Technician Tips

- Prognosis relies on the compliancy of the owner, as this disease requires frequent monitoring and lifelong insulin treatments. Insulin requires special handling and consistent administration, and it can be expensive.
- A common complication of DM is diabetes ketoacidosis (DKA). Ketones are acidic byproducts of fat cell metabolism; as they build up they cause the animal to become acidotic.

DKA is a life-threatening medical emergency requiring fluids, insulin, and electrolyte level maintenance. Clinical signs include PU/PD, panting, weakness, lethargy, depression, vomiting, and possibly a sweet smell on the breath of the animal.

- Insulin overdose resulting in hypoglycemia is also a serious complication. This occurs as a result of syringe reading errors or owners accidentally dosing the animal multiple times. Clinical signs include disorientation, lethargy, weakness, polyphagia or anorexia, and in severe cases coma or death. The overdosed animal will need food or sugar and prompt medical attention.

Diabetes Insipidus (DI), or Weak or Watery Diabetes

Description

Unlike diabetes mellitus, diabetes inspidius is a disease of the hypothalamus or pituitary gland characterized by a reduction in the secretion of antidiuretic hormone (ADH). The kidneys depend on ADH to reabsorb water. The condition is seen in dogs and infrequently in cats. Although many cases are idiopathic, central DI can be caused by hypothalmus or pituitary gland trauma, cysts, tumors, inflammation, or congenital defects. Nephrogenic DI is a result of kidney dysfunction where the kidneys do not respond appropriately to the ADH that is produced.

TECH BOX 8.6: Diabetes inspidius is not a disease of the pancreas but rather the hypothalamus or pituitary gland.

Clinical Signs

- A lack of ADH causes the kidneys to lose water, resulting in PU/PD. Large volumes of hypotonic urine are produced by the patient, and this results in polydipsia to replace the lost fluids.
- Patients who are not getting enough to drink will end up dehydrated with electrolyte imbalances.

Diagnosis

- A decreased urine specific gravity, clinical signs, and history can be used as a start to look at a diagnosis of DI.
- A water deprivation test can be performed. Water is withheld from the patient while the urine volume and USG are measured. With DI these values don't change with dehydration.
- An ADH response test is used to rule out other causes of the PU/PD like DM, renal failure, Cushing's, and hyperthyroidism. A USG is determined before the test is begun, then a synthetic ADH hormone (desmopressin) is administered. After the injection urine volume and USG are monitored. In the case of DI the kidneys will respond to the ADH and concentrate the urine.
- Diagnostic imaging, including ultrasound and CT scans, can be used to determine if there is a tumor or cyst and the location thereof.

Treatment

- Central DI is treated with a synthetic ADH hormone and is administered daily for the lifetime of the animal.

Client Education and Technician Tips

- It is very important not to restrict water in DI patients.
- Animals respond well to treatment, making the prognosis good.
- Without a diagnosis and treatment the animal will die from complications of dehydration and electrolyte imbalances.

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Ocular Disease

chapter 9

The eye is composed of many specialized structures that must work in conjunction with each other and the nervous system to convert light to visual images. If one of these structures is not functioning appropriately it can cause loss of sight and may be associated with pain. Ocular disorders may be congenital, immune-mediated, secondary to systemic complications, traumatic, or inflammatory in nature. It is imperative that ocular disorders be diagnosed early and treated aggressively if the patient's sight is to be preserved.

Conjunctivitis or Pink Eye

Description

Conjunctivitis is an inflammation of the conjunctiva of the eye caused by the presence of an irritant or infection. Causes include bacterial infections, allergenic irritation, ocular trauma, systemic disease such as distemper in dogs, ocular foreign body, or fur or eyelash irritation.

Clinical Signs

- Conjunctivitis is characterized by a red, inflamed conjunctiva. Chemosis, swelling of the conjunctiva and sclera because of edema, makes significant amounts of swelling possible.
- A serous to mucopurulent ocular discharge with crusting of material around the eye is possible.
- Signs of ocular pain include a prominent nictitating membrane, squinting, and blepharospasm (Figure 9.1).

Diagnosis

- Conjunctivitis is often diagnosed upon clinical signs, history, and complete ocular exam.
- Because there are so many causes of conjunctivitis, definitive diagnosis is dependent on finding the specific cause. Testing includes bacterial culture, testing for systemic diseases, or allergy testing.

Small Animal Pathology for Veterinary Technicians, First Edition. Amy Johnson.

Companion website: www.wiley.com/go/johnsonvettechpath

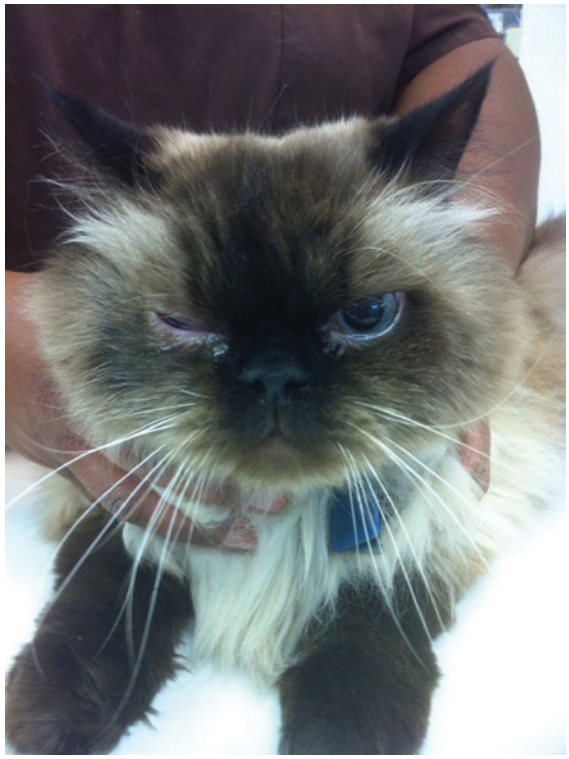


Figure 9.1 Blepharospasm in a cat. (Image courtesy Deanna Roberts)

Treatment

- Treatment begins with removal of the initiating cause.
- The eye should be rinsed with saline several times a day and kept clean of crusting.
- Antibiotic ointments are often prescribed. These ointments may include corticosteroids for their anti-inflammatory properties only if ocular ulcers have been ruled out. The use of steroids can affect the local immune response to the infection, so if mucopurulent discharge is present, steroids should not be used.

TECH BOX 9.1: The treatment of conjunctivitis is often dependent on identifying the initiating cause.

Epiphora

Description

Epiphora is an abnormal overflow of tears over the face caused by an obstructed nasolacrimal duct or the overproduction of tears. Obstructive causes vary but can include congenital eye deformities, infection causing inflammation of eye structures, trauma, foreign bodies, or tumors.

Clinical Signs

- Excessive watery discharge from eye, accumulation and drying of discharge along the eyelid, and tear staining on face below the eye area are seen with epiphora.
- Irritation of skin in the areas where the discharge has accumulated is also possible.
- Owners may note the rubbing of eyes or face on surfaces.

Diagnosis

- Physical exam including thorough eye exam, clinical signs, and history is used for presumptive diagnosis.
- Diagnostic imaging or surgical exploration of the nasal lacrimal duct may be needed to determine the cause of obstruction.
- Other tests include fluorescein eye stain, Schirmer tear test, or bacterial culture.

Treatment

- Treatment is reliant on identifying and correcting the underlying cause.
- Possible treatments may include flushing of nasal lacrimal ducts, placing a stent in the eye to keep the ducts open, surgical correction of deformity, or removal of irritant.

Client Education and Technician Tips

- Depending on the cause, this condition may require lifelong management, as recurrence is the most common complication.

Third Eyelid Prolapse or Cherry Eye

Description

A cherry eye is a prolapse of the gland in the nictitating membrane, or third eyelid, accompanied by inflammation and hypertrophy. This is a common condition in Cocker Spaniels, English Bulldogs, Boston Terriers, and Beagles, although it can be seen in any breed. Cherry eyes are rarely seen in cats. This is a condition with a genetic predisposition, but it can also be caused by ocular infection, irritation, or sun damage. The weakened connective tissue becomes mobile, irritating the gland and causing the inflammation and protrusion.

Clinical Signs

- With a cherry eye a red mass is seen protruding from the medial canthus of the eye accompanied by a mucopurulent discharge (Figure 9.2).
- Other signs of an irritated eye can be seen with a cherry eye: conjunctivitis, inflammation, excessive tearing, squinting, dry eye, or vision impairment. Pawing at the face or rubbing the face on surfaces is an indication of the irritation.
- The condition can be seen unilaterally or bilaterally.

Diagnosis

- Clinical signs and ocular exam are usually all that is necessary to make the diagnosis.

Treatment

- Topical treatments with antibiotic eye ointments and anti-inflammatories will help reduce the inflammation and treat secondary infections. However, these drugs alone are rarely enough to correct the condition.
- If possible the gland should be surgically replaced (Figure 9.3).
- Removal of the gland should be avoided if at all possible. The gland has an important function in tear production and its loss can predispose the animal to future ocular issues,

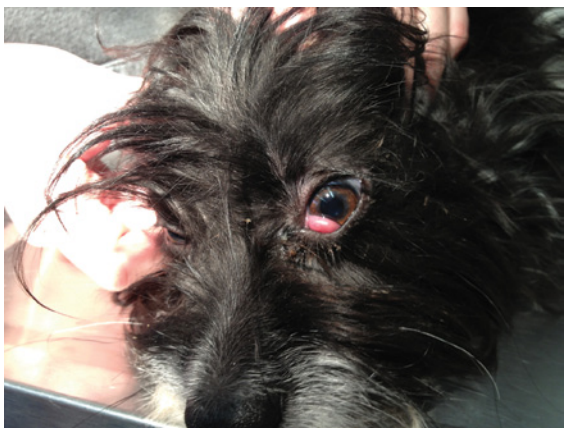


Figure 9.2 Cherry eye. (Image courtesy Amy Johnson and Bel-Rea Institute of Animal Technology)

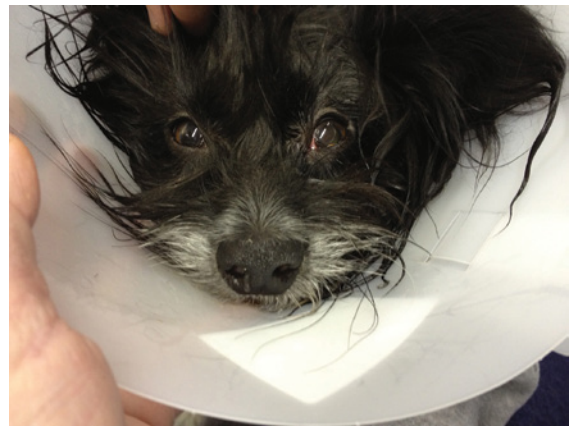


Figure 9.3 Cherry eye post-operative repair. (Image courtesy Amy Johnson and Bel-Rea Institute of Animal Technology)

including keratoconjunctivitis sicca (KCS), a common secondary condition if the gland is removed.

TECH BOX 9.2: It is imperative to try to preserve the gland and not remove it, as removal can lead to secondary KCS.

Client Education and Technician Tips

- Once this condition is seen in one eye, the second eye is likely to follow. The animal should be closely watched for development in the other eye.
- Because of the genetic link, animals with cherry eye should not be bred.

Entropion/Ectropion

Description

Entropion and ectropion are conformational abnormalities of the eyelid. An entropion is an inversion of the lid, while an ectropion is an outward turning of the lid.

Entropion is the most commonly seen eyelid deformity in dogs and is associated with severe pain. The inversion causes hairs, eyelashes, and cilia to create further eye damage. Corneal ulcerations and conjunctivitis are possible secondary conditions as a result of an entropion.

Ectropion is commonly observed bilaterally as "droopy eyes." The exposed conjunctiva creates chronic conjunctivitis, secondary bacterial infection, and irritant exposure for the animal.

TECH BOX 9.3: Eyelid conformational abnormalities are associated with severe pain and secondary complications.

Figure 9.4 Entropion. (Image courtesy A. K. Traylor, DVM, Microscopy Learning Systems)

Clinical Signs

- The clinical sign of these conditions is an inward inversion (entropion) or outward turning (ectropion) of the eyelid (Figure 9.4).
- The inversion with the eye seen with an entropion can cause painful ocular ulcerations. Squinting and blepharospasms will be seen as a result of the pain.
- Clinical signs of secondary conditions include conjunctivitis, corneal ulcerations, and ocular discharge.

Diagnosis

- Clinical signs and ocular examination are usually all that is necessary to diagnose the condition.
- Fluorescein eye stains can be used to determine the presence of an ocular ulceration.

Treatment

- Both eyelid abnormalities will require surgical repair of the eyelids to cure the condition (Figure 9.5).

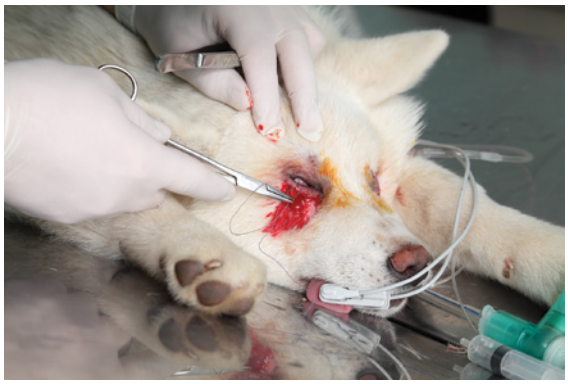


Figure 9.5 Eyelid repair surgery. (Shutterstock photo courtesy Sima)

- Ectropion may require antibiotic ointments with corticosteroids for control of infections and inflammation.

Glaucoma

Description

Glaucoma is characterized as an increased intraocular pressure (IOP). This pressure will results in optic disk and retinal damage and is associated with severe pain and loss of sight if not treated promptly. It is a condition seen in both dogs and cats, with many possible causes. Primary glaucoma is an inherited condition where the eye produces a fluid called aqueous humor faster than it can be removed. Secondary glaucoma results from other eye conditions that allow pressure to build in the eye. Possible secondary causes include anterior uveitis, ocular neoplasia (Figure 9.6), systemic infections, lens luxation, and other eye traumas. In dogs primary glaucoma is the most commonly seen with bilateral involvement. Cats tend to have more secondary cases.

Clinical Signs

- Clinical signs associated with the eye include blupthalmia, firm globe, greying or clouding of the eye, red bloodshot eye, corneal edema, dilated fixed pupil slow to respond to light changes, and lens displacement (Figure 9.7).
- Glaucoma is a very painful condition and may result in behavioral changes in the animal. The animal may become lethargic, temperamental, and head shy. Tearing and squinting may also be seen in response to pain.

Figure 9.6 Canine glaucoma due to ocular melanoma. (Image courtesy Amy Johnson)

TECH BOX 9.4: Increased intraocular pressures are associated with severe pain.

- The affected eye is blind, causing animals to bump into things and act lost in familiar environments. Loss of a menace reflex will also indicate a loss of sight.

Diagnosis

- Measuring IOP using a tonometer will determine if there is an increase in pressure.

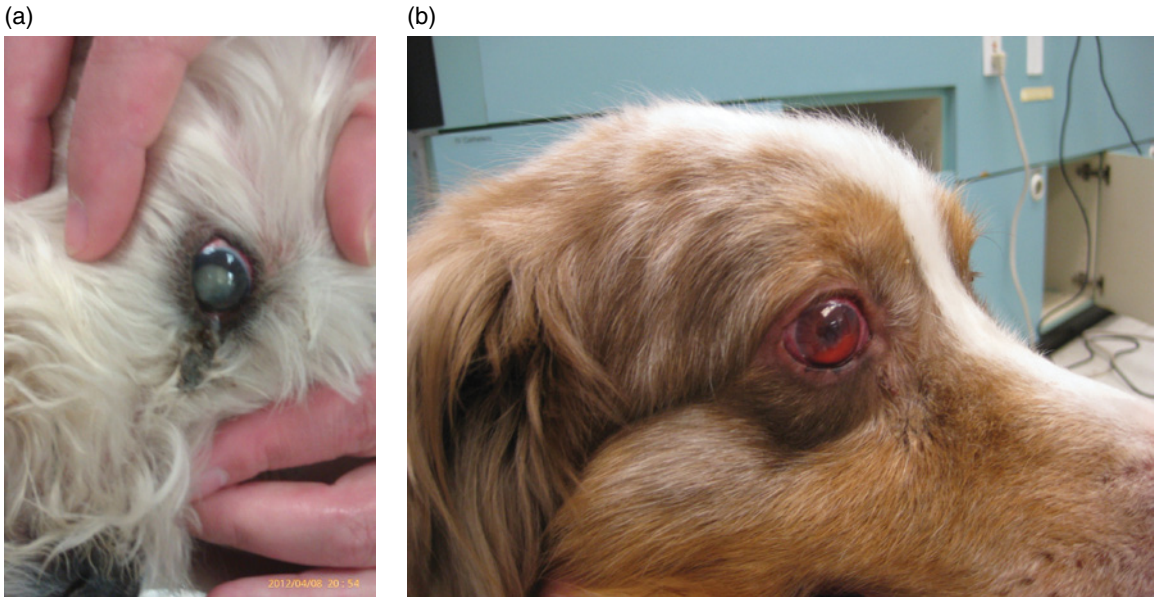


Figure 9.7 (a) Canine glaucoma. (Image courtesy Kristen Mutchler) (b) Canine glaucoma with hyphema. (Image courtesy A. K. Traylor, DVM, Microscopy Learning Systems)

Technician Duty Box 9.1

Patients with glaucoma will require frequent tonometry to measure IOP and gauge the effectiveness of treatments.

- Other methods of measuring IOP and evaluating the eye include slit lamp biomicroscopy, gonioscopy, and indirect ophthalmoscopy. These methods can help determine the cause of the increased IOP. Gonioscopy can be used to determine the risk of the other eye in developing glaucoma.

Treatment

- Treatment has a better prognosis if it is begun as soon as the first clinical signs are observed and a diagnosis is made. Often by the time owners notice a problem and begin treatment it can be too late to save the eye. Once the eyes are obviously enlarged the animal is permanently blind.
- Medical treatment involves the use of drugs to decrease IOP. These medications are used to increase osmotic pressure and draw aqueous humor out of the eye, block the production of aqueous humor with the use of enzymes, or constrict the pupil to increase fluid draining ability. Research shows a mixed response to antioxidant therapy meant to reduce oxidative damage to cells responsible for fluid drainage in the eye.
- The goal of surgical treatment is to reduce aqueous humor production in the eye.
- Some cases may require enucleation (Figure 9.8).

Client Education and Technician Tips

- Dogs with unilateral primary glaucoma should have the other eye closely monitored for the development of glaucoma in that eye.

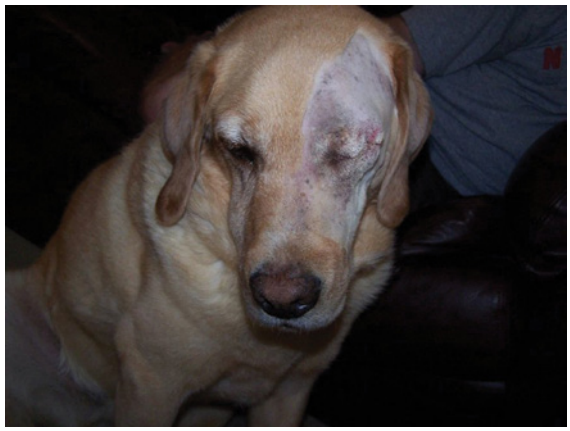


Figure 9.8 Glaucoma patient post-operative enucleation. (Image courtesy Amy Johnson)

- There is a genetic link, so affected dogs should not be bred.
- Dogs with glaucoma should not be walked on collars, rather with the use of harnesses, as evidence suggests pulling on the neck can increase intraocular pressure.

Corneal Ulcers

Description

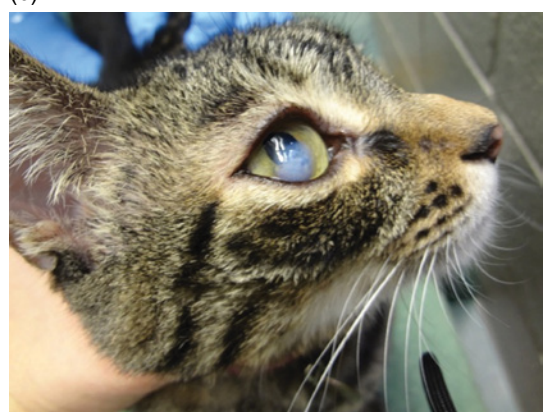
Corneal ulcers are defects in the corneal epithelium seen in dogs and cats and that are attributable to many causes including infections, corneal trauma, foreign bodies, eyelid conformational abnormalities, and keratorconjunctivitis sicca.

Clinical Signs

- Ulcerations on the cornea are very painful, as seen by squinting, tearing, a prominent nictitating membrane, pawing at the eye, and possible behavioral changes.
- There may be visual defects in the cornea, clouding of the eye, and conjunctivitis (Figure 9.9).

(a)

(b)



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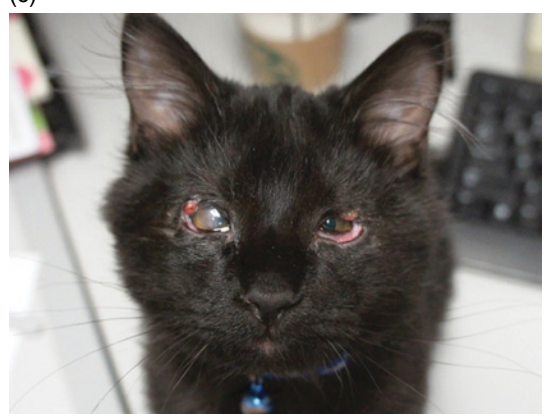

Figure 9.9 (a) Feline corneal ulcer. (Image courtesy A. K. Traylor, DVM, Microscopy Learning Systems) (b) Feline corneal ulcer. (c) Feline corneal ulcer and conjunctivitis. (Images courtesy Deanna Roberts)

Diagnosis

- Diagnosis is reached by patient history and clinical signs in conjunction with a thorough eye exam, fluorescein eye stain, and ophthalmoscopy.

Treatment

- The goal of medical treatment includes the control of inflammation, pain reduction, and removal of initiating cause. Often eye ointments or drops are used.
- In some cases surgery and corneal tissue grafts may be required.
- Animals dealing with corneal ulcers must not be able to traumatize their eye any further. These animals will require an Elizabethan collar (E-collar).

Chronic Superficial Keratitis or Pannus

Description

Pannus is a progressive bilateral, inflammatory condition of the cornea with multiple factors playing a role in the development thereof. There is a genetic predisposition in German Shepherds, Labradors Retrievers, Border Collies, Greyhounds, and Belgian Tervurens toward pannus, and it is thought to be immune-mediated. Additional factors include ultraviolet radiation and altitude.

Clinical Signs

- White, pink, or brown pigmentation to the cornea. Vascularization and opacification starting at the outside edge of the cornea and working inward can also be seen.
- The changes in the corneal surface will affect sight and may cause discomfort.

Diagnosis

- Diagnosis is made based on clinical signs, history, and a thorough eye exam.

Treatment

- The sooner treatment is started, the better the prognosis for the animal.
- There is no cure for pannus, but progression can be slowed with medical therapy.
- The main therapy is steroid therapy, including subconjunctival injections, ointments, or eye drops.
- Immunomodulary therapy can be used to suppress immune response.
- Other treatments include a superficial keratectomy, a surgical procedure excising part of the cornea, and radiation therapy, although these procedures are rarely used.
- Therapy is lifelong and cannot be stopped without the condition worsening or recurring.

Client Education and Technician Tips

- Animals with pannus require constant monitoring and reevaluations.
- Because ultraviolet radiation is thought to be a factor, animals with pannus should avoid time in the sun or should wear protective eyewear when outdoors.

TECH BOX 9.5: Pannus is associated with ultraviolet radiation, making it important to protect an animal's eyes from the sun.

Keratoconjunctivitis Sicca or Dry Eye

Description

KCS is a tear deficiency leading to chronic mucopurulent discharge, conjunctivitis, corneal ulcerations, and scarring. KCS is the most common cause of conjunctivitis in dogs and rarely seen in cats. In dogs causes include autoimmune disease, drug therapies, distemper, genetics, and trauma. In cats there is a link to chronic herpes viral infections.

Clinical Signs

- Clinical signs include red irritated eyes, thick mucopurulent discharge, crusting around the eyes, rubbing at the eyes, cloudy eyes, and possibly corneal ulcerations.

Diagnosis

- Clinical signs, patient history, and a thorough eye exam are used in conjunction with a Schirmer tear test to make a diagnosis of KCS. A normal Schirmer tear test is 18–24 mm, where a dog with KCS commonly measures less than 10 mm.

Technician Duty Box 9.2

Diagnosis and monitoring of certain eye conditions, including KCS, will require Schirmer tear tests to be performed.

Treatment

- Treatment is aimed at lubricating the eye, stimulating tear production, and decreasing damage to the cornea.
- Artificial tears and ointments with antibiotics and corticosteroids (only if no ulcers are present) can be used to lubricate the eyes.
- Lacrimogenic and cholinergic drug therapy is meant to increase the animal's tear production.
- Other drug therapies include immune-mediating drugs to suppress immune-mediated damage and mucus-dissolving eye drops.
- A surgical parotid duct transplantation may be indicated in some cases.

Anterior Uveitis, Iridocyclitis, or Soft Eye

Description

Inflammation of the anterior chamber of the eye, including the iris and ciliary body, is referred to as anterior uveitis. This is one of the more common eye conditions seen in dogs and cats. Causes include systemic diseases, trauma, irritants, cataract formation, neoplasia, and idiopathic cases.

Clinical Signs

- The eye has a cloudy appearance to it, with anterior uveitis resulting from increased protein in the cells of the anterior eye and inflammation.
- Pain is usually associated with this condition.
- Other clinical signs include miosis, decreased intraocular pressure, photophobia, and blepharospasm.

Diagnosis

- Clinical signs, patient history, and ophthalmoscopy.
- Measurement of IOP with tonometry can determine if pressures are decreased and rule out glaucoma.

Treatment

- Treatment is dependent on cause but is aimed at removing the initiating cause and reducing inflammation.
- Non-steroidal anti-inflammatory drugs or steroids can be used for inflammation. Steroid use should be avoided in cases stemming from infectious causes.
- Infectious causes are treated with antibiotics.
- Cycloplegic drugs can be used to control pain and mydriatic drugs to dilate the pupil.

Cataracts

Description

Cataracts are opacity of the lens of the eye. They can be seen in dogs and cats but are more

common in dogs. Cataracts will cause blurry vision, and dense large cataracts can cause blindness. Cataracts are classified by age of onset, location, cause, and the degree of severity. Causes include age, genetics, radiation, trauma, inflammation, and systemic diseases such as DM.

Clinical Signs

- A grey opacity to the eye is noted with cataracts.

Diagnosis

- Clinical signs, patient history, and ophthalmoscopy are used to diagnose cataracts. Ophthalmoscopy may require dilation of the eye.
- Slit lamp biomicroscopy gives the best visualization of the lens.

Treatment

- Surgical removal of the lens is the only curative treatment for cataracts.
- Some cataracts may spontaneously reabsorb, especially in young animals, and need no treatment.
- Corticosteroid treatment can be used if inflammation is suspected as the cause.
- Atropine eye drops can be used to keep the pupil dilated to control pain.

Client Education and Technician Tips

- Cataracts can lead to secondary uveitis and glaucoma. If left untreated the lens may luxate and block fluids in the eye.
- Cataracts should be differentiated from nuclear sclerosis, which is a normal clouding of a dog's eye resulting from aging. Nuclear sclerosis does not need treatment.

Progressive Retinal Atrophy (PRA) or Progressive Retinal Degeneration (PRD)

Description

PRA is a genetic disorder seen in dogs and cats where the retina atrophies and the animal goes blind. The condition is bilateral and non-painful, with clinical signs appearing in the first few years of life.

Clinical Signs

- The pupils are dilated with PRA and the owners will describe a "glow" or "shine" to the eye.
- Night blindness is seen first, then progresses to full blindness. Clinical signs of blindness include disorientation, fear of dark rooms, and getting lost in the dark.
- Cataract formation is common in late stages of the disease.

Diagnosis

- Ophthalmoscopy will show changes in the retinal blood vessel pattern, the optic nerve head, and the tapetum lucidum.
- Electroretinogram, a measurement of the retina's response to flashes of light, may be necessary for diagnosis and especially if cataract formation is present.

Treatment

- There is no real treatment for this disease.
- Some research suggests oral antioxidant therapy may delay the onset of blindness.

Client Education and Technician Tips

- Animals usually cope well with blindness, although owners need to keep consistency in

the house and help keep animals safe. Stairs should be blocked and dogs kept on leashes when outside.

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Integumentary Disease

chapter 10

The skin is the body's largest organ, with many functions including serving as a physical barrier against environmental insult, aiding in regulation of body temperature, and sensory perception. Breaking this barrier leaves the patient open for deeper or systemic disorders. Common clinical signs of dermatitis can be seen no matter what the cause and include pruritus, oily or dry skin, hyperpigmentation (Figure 10.1), skin thickening, erythema, and alopecia (Figure 10.2). A detailed patient history needs to be obtained and a thorough physical exam conducted because the clinical signs for each type of dermatitis are similar; diseases need to be differentiated through diagnostic testing.

Parasitic Skin Infections

Flea Allergy Dermatitis (FAD)

Description

Fleas are a troublesome ectoparasite of dogs and cats, causing dermatitis. Fleas can also transmit bacterial disease and parasites. Fleas are small insects visible with the naked eye, wingless, and have mouthparts adapted for sucking blood from the host. The most often seen fleas in dogs and cats in North America are *Ctenocephalides felis* (cat flea) and *C. canis* (dog flea) (Figure 10.3). One of the foremost flea-related problems, flea allergy dermatitis, is common in both dogs and cats and seen more frequently in the summer months, although in temperate climates can be seen year-round. When the feeding flea bites the host animal the saliva contains compounds that will irritate the skin and cause the formation of allergy-associated antibodies.

Clinical Signs

- Visible fleas or flea dirt (flea feces) on the animal or in the bedding associated with the animal are common in FAD patients.
- Clinical signs are variable from patient to patient. These clinical signs include dermatitis with pruritus and associated scratching,

Small Animal Pathology for Veterinary Technicians, First Edition. Amy Johnson.

Companion website: www.wiley.com/go/johnsonvettechpath

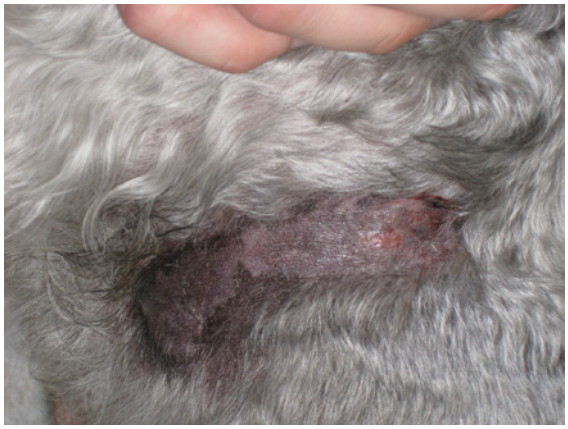

Figure 10.1 Hyperpigmentation, alopecia, and pyoderma in a canine patient. (Image courtesy A. K. Traylor, DVM, Microscopy Learning Systems)

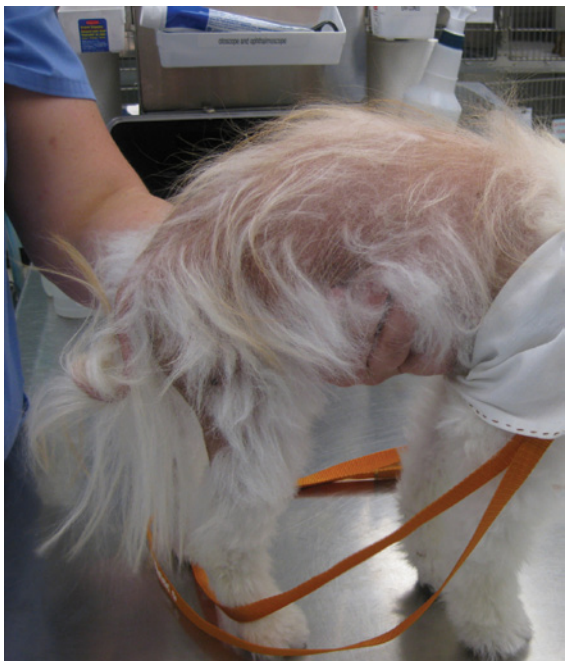

Figure 10.2 Alopecia in a canine patient. (Image courtesy A. K. Traylor, DVM, Microscopy Learning Systems)

biting, licking, and chewing at affected areas, saliva staining of fur, broken fur, alopecia, thickening of skin, hyperpigmentation, erythema, scaling, crusting, and pustules.

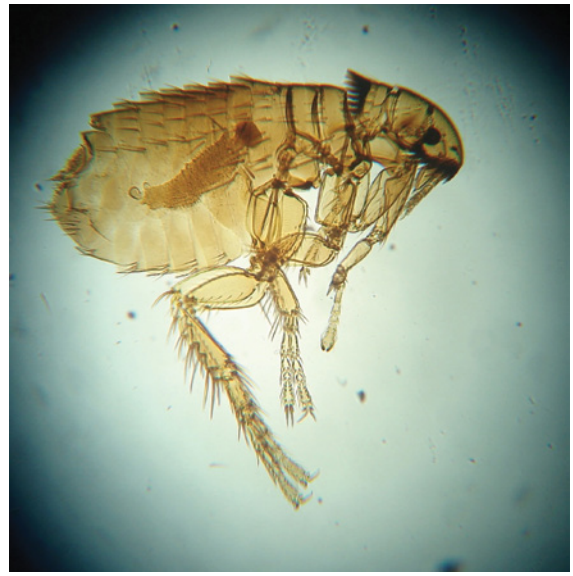

Figure 10.3 Microscopic image of a flea, *Ctenocephalides spp.* (Image courtesy Amy Johnson and Bel-Rea Institute of Animal Technology)

- Skin lesions are found on lower dorsal region, tail, and inner thighs.
- The intense irritation of the skin will cause the animal to be restless and uncomfortable.
- Secondary bacterial or yeast infections are common as a result of the self-traumatizing of the skin.

Diagnosis

- A thorough history in conjunction with clinical signs and physical exam will aid in a presumptive diagnosis. Exclusion of other dermatological diseases may be necessary, but the presence of fleas is helpful in making a diagnosis.
- A definitive diagnosis is determined through intradermal (ID) or serological testing.

Treatment

- Flea prevention and control is the key for treatment of patients. Antiparasitic drug therapies are available to keep fleas off the patient.

- Elimination of mature/adult fleas and the younger life cycles of fleas in the environment are necessary to prevent reoccurrence. The use of pesticides and regular washing of bedding and vacuuming will aid in accomplishing this.

TECH BOX 10.1: When treating pets for fleas, treatment of the environment is imperative to prevent recurrence.

- Palliative treatment when full flea elimination is not feasible includes glucocorticoids and medicated baths.
- Antibiotics may be necessary with secondary skin infections.
- Hyposensitization (allergy shots) may prove helpful in some situations, especially if the animal cannot be ridded of the fleas.

Ticks

Description

Ticks (Figure 10.4), small arachnids in the order *Ixodida*, are obligate ectoparasites that will feed on the blood of animals. Ticks are classified in two general categories: hard ticks and soft ticks, based on the presence of a hard scutum or shield. They are found to be a problem in both dogs and cats, although more often seen in dogs because dogs are outdoors and share the environment with ticks more frequently than cats. There are many tick species found throughout North America, and each species is responsible for transmitting a variety of infections including viruses, bacteria, and other parasites. Other tick-related disorders include tick paralysis, anemia, localized dermatitis, and toxicosis. Toxins in the saliva of the ticks can cause tick paralysis, a problem seen in dog hosts. These toxins are released into the animal's blood as the tick feeds, and the toxins affect the dog's central nervous system.

Figure 10.4 Microscopic image of a tick. (Image courtesy Amy Johnson and Bel-Rea Institute of Animal Technology)

Clinical Signs

- Visualization of the tick or evidence of tick attachments is often found by the owners or veterinary staff. A small raised nodule may be found where the tick was attached, with some animals having more of a reaction than others (Figure 10.5).
- A patient with tick paralysis will present with muscle relaxation, causing a paralysis or weakness. Clinical signs occur within 6–9 days after the tick has attached and include ataxia, hypertension, tachycardia, hind limb weakness, poor reflexes, dysphagia, hypersalivation, mydriasis, and vomiting. Megaesophagus, a condition secondary to muscle weakness, is associated with regurgitation and aspiration pneumonia.

Diagnosis

- Visualization of the tick is associated with presenting clinical signs, and a thorough physical exam and history will aid in diagnosis.
- Radiographs can be used to visualize a megaesophagus secondary to tick paralysis.

Figure 10.5 (a) Tick on a feline patient. (Shutterstock image courtesy Henrik Larsson) (b) Tick on a canine patient. (Shutterstock Image courtesy Eric Isselee)

Treatment

- Careful removal of the tick is a starting point. The tick should be removed with fine-tipped forceps and grasped as close to the tick's head as possible. Steady upward pressure without twisting the tick is important to remove as much of the tick's mouth parts as possible. Once removed the area should be thoroughly cleansed.
- Insecticide baths can be used for removal and preventing reattachment.
- Treatment is aimed at the animal's specific clinical signs and in severe cases of tick paralysis may include oxygen and ventilation.

Client Education and Technician Tips

- Tick control is imperative for prevention of tick-related problems. Tick prevention medications (acaricides) include topicals, dips, sprays, and shampoos.
- Restricting access to environments where ticks live, checking animals for ticks after being outdoors, and removing them immediately will also help in preventing disease.

Otodetic Mange or Ear Mites

Description

Ear mites, *Otodectes cynotis* (Figure 10.6), are seen in both dogs and cats, although more commonly in cats. These mites live in the ear canal and will pierce the skin to feed. They are a frequent cause of otitis externa.

TECH BOX 10.2: Ear mites are more frequently seen in cats than dogs. High-risk categories include cats with outdoor access and pet store cats housed together, especially kittens.

Clinical Signs

- Ear mite infections are often bilateral with clinical signs including thick "coffee ground" ceruminous discharge from the ears. Intense pruritus causes ear scratching, head shaking, and droopy ears.
- Severe cases may cause a rupture in the tympanic membrane and suppurative otitis.

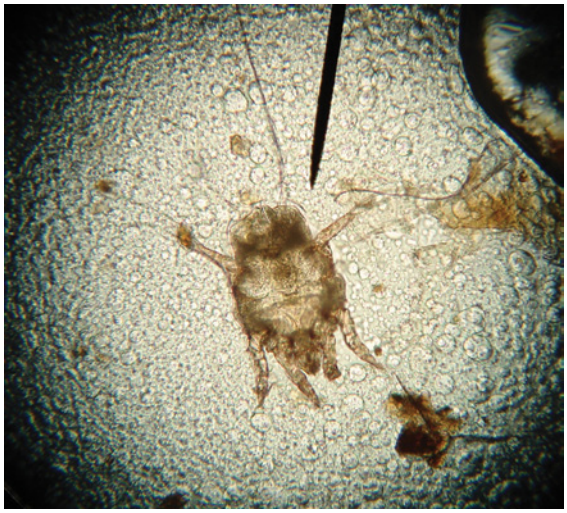

Figure 10.6 Microscopic image of an ear mite, *Otodectes cynotis*. (Image courtesy Bel-Rea Institute of Animal Technology)

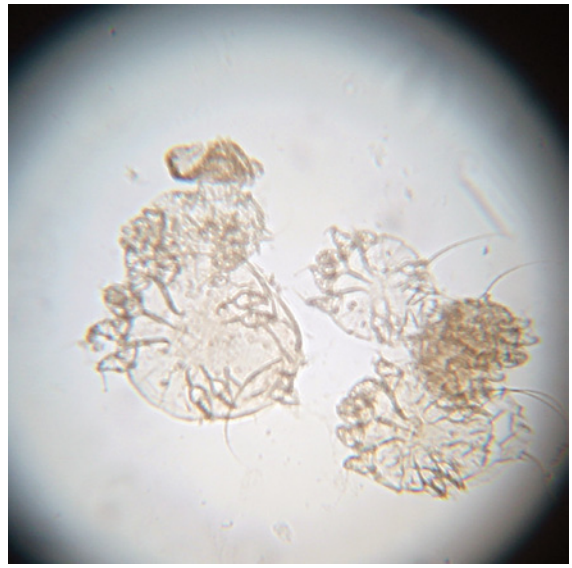

Figure 10.7 Microscopic image of a mite, *Sarcoptes scabiei*. (Image courtesy Bel-Rea Institute of Animal Technology)

Diagnosis

- Otoscopic examination of the ear, presenting clinical signs, and patient history will aid in a diagnosis.
- Collection of sample of ear discharge by swabbing the ear will further support the diagnosis. This discharge is microscopically examined by suspending in mineral oil on a slide; ear mites and eggs are visible.

Treatment

- Antiparasitic drug therapy will be administered in conjunction with ear cleansing agents. The medications are usually local agents in the form of eardrops.

Sarcoptes or Scabies or Sarcoptic Mange

Description

Sarcoptes mites, *Sarcoptes scabiei var canis* (Figure 10.7) in dogs, are highly contagious and zoonotic. These mites are microscopic, round in shape, and have a distinct head and four pairs of legs. They are usually host specific but can cross species lines and infect species other than that of origin, including humans. They are transmitted via direct contact and will cause dermatitis in hosts.

TECH BOX 10.3: Sarcoptes mites are highly contagious, zoonotic, and associated with intense pruritus.

Clinical Signs

- Sarcoptes are associated with acute intense pruritus causing self-trauma lesions.
- The lesions are common on the ventral side of the body, extremities, and ears. They are patches of crusty, scaly, and thickened skin.
- Secondary pyoderma is often seen.
- Infections can be generalized conditions and may cause lymphadenomegaly, emaciation, and death in severe cases.

Diagnosis

- Presenting clinical signs, physical exam, and history will aid in diagnosis. Sarcoptes is highly contagious so other animals and humans in the house may also have similar clinical signs.
- Superficial skin scraping and microscopic examination can aid in visualization of the mite. Multiple areas should be scraped, otherwise mites may be missed. Skin scrapings are not always successful, and sarcoptes cannot necessarily be ruled out based on a negative skin scrape.
- An antibody ELISA is available for further diagnosis.
- In some cases animals will be treated despite a definitive diagnosis to see if the animal will respond to treatment.

Treatment

- Treatments include topical or systemic antiparasitic drug therapies, medicated baths, and dips.

Client Education and Technician Tips

- Because these mites are so highly contagious, all dogs in the household or with direct contact with the animal should be treated.

Demodex or Demodetic Mange or Demodicosis

Description

Demodex mites are mites that inhabit the hair follicle and sebaceous glands of both dogs and cats, although they are less often found on cats. *Demodex canis* (Figure 10.8) is found on the dog and cats are found to have either *D. cati* or *D. gatoi*. Demodex mites are also called the cigar mite because of the morphology of the mite. They are microscopic mites with an elongated semitransparent body and four pairs of short legs. Infestation and infection are the result of numerous mites, as small numbers are the normal flora of dogs. Factors of infestation include age, immune system, and genetic predisposition. Both localized and general forms of the infection can be seen and may lead to other skin conditions and infections.

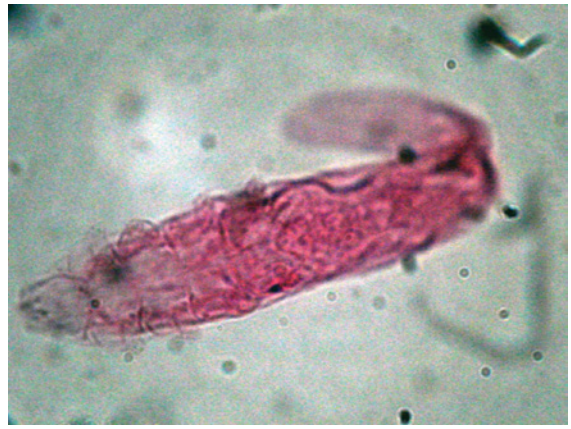

Figure 10.8 Microscopic image of a mite, *Demodex canis*. (Image courtesy Bel-Rea Institute of Animal Technology)

Clinical Signs

- Clinical signs include erythema, variable pruritus, alopecia, and hyperpigmentation of skin (Figure 10.9).
- Secondary pyoderma may be seen (Figure 10.10).
- Systemic infections may include fever, pododermatitis, lethargy, cellulitis, and lymphadenomegaly.

Diagnosis

- Presenting clinical signs, physical exam, history, and ruling out other causes of dermatitis will aid in diagnosis.
- Deep skin scraping may yield visualization of the mite. The collected samples are submerged in mineral oil and evaluated under the microscope.

Figure 10.9 Demodetic blepharitis. (Image courtesy Deanna Roberts)

Figure 10.10 Demodetic mange with severe pyoderma. (Image courtesy A. K. Traylor, DVM, Microscopy Learning Systems)

TECH BOX 10.4: The diagnosis of demodex requires deep skin scraping, as the mite lives in the hair follicle. Because small numbers of demodex are normal flora on dogs and cats, a positive diagnosis is made when identifying larger numbers associated with clinical signs.

Treatment

- Some cases will resolve on their own, yet most patients will be treated with medicated baths or dips and antiparasitic drug therapies.

Pediculosis or Lice

Description

Lice are obligate ectoparasites that live on the skin and fur of most animal species (Figure 10.11). Lice are spread by direct contact and most are species specific; the do not often cross species lines. There are two categories of lice seen in animals: chewing or biting lice (order Mallophaga) and sucking lice (order Anoplura). Lice are wingless flat insects usually 2–4 mm long, visible with the naked eye, and that survive by biting and chewing on dermal debris or sucking the blood of their animal hosts.

Clinical Signs

- Lice are visible with the naked eye, allowing owners or veterinary personnel to visualize them.
- Dermatitis with pruritus is common with infections involving lice. Hosts will show signs of scratching, rubbing, or biting at the skin and fur. This will lead to an ungroomed appearance, alopecia, or a rough, dry, matted coat.

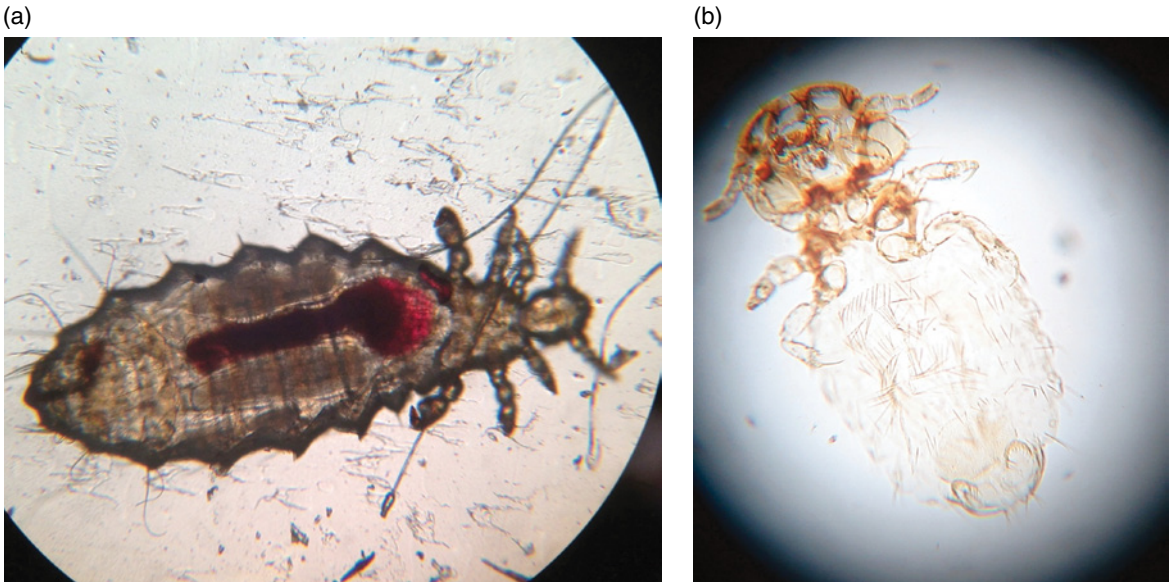

Figure 10.11 (a) Microscopic image of a biting louse. (Image courtesy Geoff Chevalier/Bel-Rea Institute of Animal Technology) (b) Microscopic image of a biting louse. (Image courtesy Bel-Rea Institute of Animal Technology)

- Sucking lice can cause anemia.
- Anxious hosts are common as a result of the constant irritation.

Diagnosis

- Presenting clinical signs, history, and physical exam will aid in diagnosis.
- Although the lice can be visualized without microscopic examination, looking at them under the microscope will allow for definitive differentiation between them and other parasites.

TECH BOX 10.5: Lice are one of the few ectoparasites visible with the naked eye, requiring little to no laboratory work for a diagnosis.

Treatment

- Antiparasitic drug therapies including topical sprays or applications, pour-on treatments, and medicated shampoos.
- Additionally, there are some systemic therapies that can be used.

Client Education and Technician Tips

- Grooming supplies and environment may spread lice, making sanitation an important part of stopping the transmission thereof. Animals will become re-infected if they, their bedding, and the environment are not cleaned.

Cuterebra Larvae or Botfly Larvae

Description

When it comes to flies, especially those categorized as botflies (*Cuterebra* spp.), adults can serve as a vector for disease but more troublesome are the larvae that develop in subcutaneous tissues of dogs, cats, ferrets, rodents, and rabbits. This condition of larvae developing in the tissues of host animals is referred to as "myiasis." These flies are usually host specific. The adult females lay their eggs in the environment of the host animals, allowing the animal to pick them up in

contaminated areas. The eggs hatch on the animal's skin as a result of the body heat, then enter the host through open wounds, nares, or mouth. They will then migrate to subcutaneous tissues, where they develop a breathing pore visible on the animal. After approximately 1 month the pupae exit the skin through the pores. Migrations through the respiratory system and brain will cause problems with those systems. These cuterebra are most commonly seen during summer months and are seen more frequently in free-roaming cats.

Clinical Signs

- Warbles are SQ swellings about 1 centimeter in diameter with visible breathing pore typically on head, neck, or trunk. Pain may be associated with these SQ masses. Hosts, especially cats, tend to excessively groom area. The site is open to secondary bacterial infection and purulent exudate may be seen.
- Neurologic and respiratory signs may be seen associated with the migration of the parasite. These signs include lethargy, upper respiratory infections, fever or hypothermia, seizure, blindness, abnormal mentation, ataxia, and vestibular disease.

Diagnosis

- Clinical signs and visible warble. Once the larva had been removed it can be identified.
- CT and MRI scans may aid in the process of observing migratory damage in cats.

Treatment

- The lesions should be explored using forceps to enlarge the pore and remove the larva in one piece if possible. Rupture may cause secondary infection or possible anaphylaxis in the host.
- Lesions should be flushed and allowed to heal.
- Antiparasitic drugs may be prescribed to kill any larvae in migration phase.

Client Education and Technician Tips

- Some cats may have lifelong neurological signs due to damage from the parasitic migration.

Facultative Myiasis-Producing Flies or Maggots

Description

Many flies fall into the category of facultative myiasis-producing flies; most common is blowflies from the family Calliphoridae. Larval stages of this fly are known as maggots and are associated with skin wounds in dogs and cats. Adult flies are attracted to the damaged flesh of the animal and lay their eggs on the flesh. Larvae will develop within 24 hours and move freely on the cutaneous tissue, known as strikes. The maggots do not usually ingest live tissue but instead the dead tissue, debris, and exudate. Maggots will, however, cause damage to newly developing tissue layers and tunnel through the skin, causing further tissue damage.

The tissue damage is a prime environment for more maggots. If this process is not stopped and treated it will become fatal. The host will die from shock, histolysis, or secondary infection. This condition is seen in animals housed outdoors, especially if the environment is moist.

TECH BOX 10.6: Maggots do not often ingest live tissue. Most animals infested by maggots have tissue damage prior to the maggots. Tissue injuries should be kept dry and animals indoors until the tissue has healed.

Clinical Signs

- The maggots will be visible; up to thousands of these small, thin, tubular worms the size of a grain of rice will be found. The tunnels in the tissue will also be seen on the animal.
- The host is known to have tissue damage prior to the infestation.

- The damaged flesh and maggots have a pungent and distinct odor to them.

Diagnosis

- Presenting clinical signs, physical exam, and history will provide a diagnosis. Visualization of the maggots and tissue damage make diagnosis straightforward.
- The species of fly can be determined with microscopic examination of the larva.

Treatment

- Treatment involves clipping the fur and removing the maggots. This will take time and sedation or anesthesia may be necessary to make the animal comfortable.

Technician Duty Box 10.1

Veterinary technicians will be given the duty of properly removing ectoparasites, including ticks and fly larvae.

- Supportive care is provided based on the patient's clinical signs and may include IV fluids and analgesics as the animal heals.

Client Education and Technician Tips

- Prevention includes close attention to skin wounds and keeping animals with damaged tissues in areas free of flies.
- Keeping fur free of urine and feces and the animal cleanly groomed will aid in avoidance of the flies, as they are attracted to urine and feces.
- The environment should be kept free of anything that will attract flies including standing water, garbage, and feces.
- Care should be taken to keep the animal's skin dry and the animal in a dry environment.

Fungal Skin Infections

Yeast

Description

Malassezia pachydermatis is a yeast found normally on the skin of dogs and cats, yet an overgrowth will cause dermatitis on skin and in ears. Causes for this overgrowth include seborrhea, allergies, congenital skin disorders, hormonal imbalance, and the appropriate temperatures and humidity. Dogs with long floppy ears are at risk of fungal ear infections, as air cannot circulate through the ear canal and traps in moisture, creating the perfect environment for growth.

TECH BOX 10.7: Yeast is a frequent cause of skin and ear infections, thriving in moist or oily areas.

Clinical Signs

- Inflammation and irritation of the skin associated with seborrhea, alopecia, pruritus, erythema, hyperpigmentation, thickening of the skin, and a distinctive odor of yeast.
- Ears will show a thick brown discharge and pruritus. Animals scratching at ears can cause auricular hematomas.

Diagnosis

- Presenting clinical signs, physical exam, and history will aid in obtaining a presumptive diagnosis.
- Microscopic cytologic evaluation of discharge from ear or skin surface collections. Samples are evaluated for purple staining (with Diff Quik or Gram's stain) budding oval structures (Figure 10.12).

Treatment

- Antifungal drug therapy is used in association with medicated shampoos and ear cleansing and drying agents.

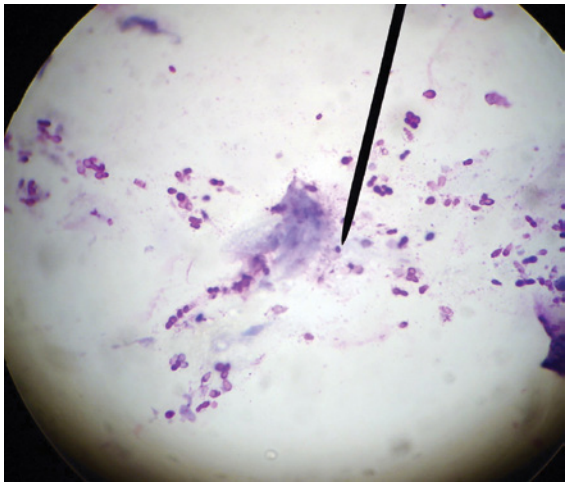

Figure 10.12 Microscopic image of yeast, *Malassezia pachydermatis*, from a canine ear cytology. (Image courtesy Bel-Rea Institute of Animal Technology)

- An important part of treatment and prevention includes keeping skin dry and non-oily as well as the ear canals dry.
- If the yeast overgrowth is secondary to an underlying condition, the primary disorder needs to be treated if possible.

Client Education and Technician Tips

- This yeast overgrowth is often a problem that will recur and needs to be monitored, with flare-ups treated as clinical signs arise.

Dermatophytosis or Ringworm

Description

Ringworm is an infection caused by a fungal mold growth, called dermatophytes, on the skin. The three species common in dogs and cats in North America include *Epidermophyton*, *Microsporum*, and *Trichophyton*. Ringworm infections are reported in many animal species including dogs and cats. *M. Canis* is most commonly diagnosed in dogs and cats and is zoonotic. Dermatophytes are transmitted by direct contact and fomites and will grow on keratinized tissues such as skin, fur, and nails. Ringworm is contagious, although not all animals exposed will develop infection. Factors in developing clinical disease are dependent on specific organism, immune system of host, age of host, and amount of organism exposure necessary.

Clinical Signs

- Clinical signs of a dermatophyte infection include broken hairs, alopecia, and scaly, crusty round-/ring-shaped lesions, papules and pustules. Erythema and pruritus are variable among species.
- Cats may be asymptomatic carriers of the infection.

Diagnosis

- Presenting clinical signs, physical exam, and history can help in a diagnosis.
- Fungal culture using Dermatophyte Testing Media (DTM) can obtain a diagnosis. Hair and scales from the patient's lesion are collected and placed on the medium, which is then incubated at either room temperature or 37°C. The media is checked daily for growth, which should appear within 3–7 days but in some cases takes longer. There will also be a color change in the medium, turning from yellow to red in the presence of a dermatophyte (Figure 10.13).
- Direct microscopic examination can be performed, looking for spores using lactophenol cotton blue (LPCB) stain. Hair or scales from the host's lesion are collected, usually using clear Scotch tape. A drop of stain is placed on the slide, and the tape is then placed on the slide in the drop of stain. The slide is scanned, looking for spores consistent with dermatophytes (Figure 10.14).
- Wood's lamp evaluation can also be performed. Dermatophyte infections may fluoresce an apple green color. This is not 100% effective so it is a great screening tool but not a definitive diagnosis. Animals lacking

(a)

(b)

Figure 10.13 (a) DTM prior to use. (b) Positive DTM exhibiting color change. (Images courtesy Bel-Rea Institute of Animal Technology)

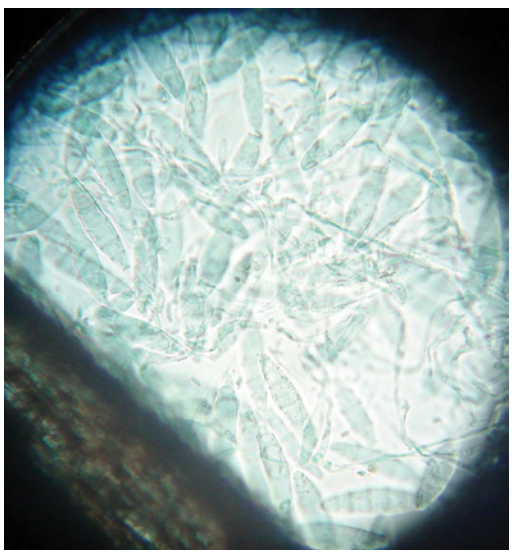

Figure 10.14 Microscopic image of ringworm, *Microsporum canis*, collected via Scotch tape prep-stained with lactophenol cotton blue. (Image courtesy Bel-Rea Institute of Animal Technology)

fluorescence cannot have dermatophyte infection ruled out.

Treatment

- The infection is self-limiting in some animals, especially those with short coats.
- Systemic or local antifungals can be prescribed. Topical antifungal creams, lyme/sulfur dips, and medicated antifungal shampoos may be used.

Client Education and Technician Tips

- The must environment must be treated as well, as recurrence is possible with a contaminated environment. The use of dilute bleach solution or other detergents labeled as killing dermatophytes may be used. Sunlight will also kill the organisms if items are left outside.

- Vacuuming is an important step in cleaning the environment, yet it may help in the spread of infectious spores. It is important to empty vacuum cleaner bags and bleach vacuum parts to avoid further contaminating the environment.

TECH BOX 10.8: Treating ringworm includes treatment of the environment; spores will remain in the environment, perpetuating the spread of the infection and increasing chance of recurrence.

Miscellaneous Skin Disorders

Pyoderma or Bacterial Folliculitis

Description

"Pyoderma" is a generic term for an infection of the skin, literally meaning pus on the skin. Causes of pyoderma include infection, neoplasia, and inflammation. The most common cause is bacterial infections, especially *Staphylococcus intermedius*, an opportunistic pathogen living on the skin of dogs and cats. Pyoderma is seen in dogs and rarely in cats. This condition can be primary or secondary and due to other underlying conditions including allergies, nutritional disorders, metabolic disorders, autoimmune disorders, parasites, seborrhea, feline acne, and immunosuppressive diseases (FIV or FeLV). Primary infections are due to skin infection without underlying disease conditions. Pyoderma can also be classified by depth of infection—superficial or deep. Warm, moist areas and pressure points are most commonly affected, as they are prime locations for bacterial growth.

Clinical Signs

- Clinical signs vary from patient to patient dependent on severity and cause. These signs include pruritus, crusting and pustules, scaling, alopecia, odor, purulent exudate, possible bleeding, pain, erythema, ulceration, and inflammation.

Diagnosis

- Presenting clinical signs, history, and thorough physical exam will aid in making a diagnosis but won't determine a specific cause.
- Determining the specific etiological agent requires testing for the underlying causes, impression smears, and bacterial culture. Bacterial sensitivity should be performed to determine best treatment course.

Treatment

- Treating underlying cause is necessary to fully treat the condition.
- Systemic and local antibiotic therapies are available for bacterial causes.
- Grooming and clearing lesions of long fur will allow for ease of treatment, keeping lesions clean, and keeping skin dry to allow for healing.

Technician Duty Box 10.2

Veterinary technicians will take on grooming duties when dealing with skin conditions. Many of these conditions will require medicated or therapeutic baths or medicated dips. Some patients may require clipping and cleaning of wounds and affected areas as well.

- Therapeutic bathing will make the animal more comfortable and clear skin of debris.

Client Education and Technician Tips

- May see recurrence depending on the underlying cause, especially if the condition is unable to be controlled.

Seborrhea

Description

Seborrhea is a condition characterized by abnormal keratinization of the skin, causing scaly, oily skin

or dry, flaky skin. There are both oily and dry cases of seborrhea, most animals having a combination of both. Seborrhea will cause the hair follicles to become obstructed, inflamed, and infected. This condition is seen in dogs more commonly than cats and can be primary or secondary. The secondary form is more common and is seen as a result of other underlying disease that predisposes the patient to oily, scaly skin. *Malassezia* (yeast), bacterial infections, alopecia, endocrine disorders, allergies, dietary deficiencies, and autoimmune conditions can contribute to seborrhea. Primary seborrhea is an inherited disorder. A family history of clinical signs is associated with disease in young animals, which progresses with age. In either case, the patient's oily or dry skin will predispose the animal to pathogens and self-trauma.

TECH BOX 10.9: There are two types of seborrhea: dry or oily. Most animals will have a combination of both oily and dry skin.

Clinical Signs

- Clinical signs include inflammation; pruritus; oily, crusty, scaly, and thickened skin; hyperpigmentation, papules, bad odor, and alopecia.
- Wounds from self-trauma will lead to the presence of secondary bacterial or fungal infections.

Diagnosis

- Physical exam, presenting clinical signs, and history will aid in a diagnosis.
- A superficial skin cytology can be used to identify yeast, bacteria, or inflammatory cells (neutrophils). Severe cases may require a skin biopsy.
- Age of onset, severity of clinical signs, and history of disease conditions may help in identifying a specific cause.
- Other disease conditions should be ruled out by performing a complete blood count, blood chemistry screening, and urinalysis.

Treatment

- Primary seborrhea cannot be cured; treatment is aimed at making the animal comfortable.
- Treating secondary conditions requires controlling the underlying cause.
- Palliative treatments include topical shampoos appropriate for the patient's skin, fatty acids, and vitamin and mineral supplements.
- Secondary conditions need to be addressed as they are diagnosed. Antibiotics can be used in the case of bacterial infection, antifungal drug therapy if yeast is present, and allergy medications with antihistamines if allergy conditions exist.

Client Education and Technician Tips

- If primary seborrhea is diagnosed, this condition will require lifelong management and veterinary care to keep the animal comfortable.
- Nutrition is an important key to maintenance.

Acute Moist Dermatitis or Traumatic Dermatitis or Hot Spots

Description

Hot spots are acute localized regions of dermatitis occurring in moist locations, seen in dogs and rarely in cats. The inflammation and infection will worsen as the animal licks, chews, and grooms the area. These lesions will occur secondarily to other skin problems and are most common in breeds with thick fur coats, allowing for moisture to become trapped on the epidermis. Other common causes include parasites and allergies, and hot spots may be seen due to boredom or stress as animals will groom excessively, especially paws and extremities.

Clinical Signs

- Circular patches of erythema, inflammation, alopecia, and salivary staining of fur that occur acutely.

Diagnosis

- Presenting clinical signs, thorough physical exam, and history are all that are necessary to make a diagnosis of a hot spot.
- Diagnostic work will be required to determine the etiologic agent if necessary.

Treatment

- Clip and clean areas, keeping them free of debris and fur as well as making medicating easier.
- Topical antibiotic/corticosteriod ointments can be used alone or in conjunction with systemic drug therapies.
- The animal must be restricted from licking, requiring an E-collar in most cases.
- The predisposing condition needs to be addressed to keep the hot spots from recurring.

Client Education and Technician Tips

- Prevention not only requires determining underlying cause but also keeping skin dry. Animals with thick, long fur coats may require grooming during hot, humid months.

TECH BOX 10.10: Boredom and stress are important causes of hot spots and should be addressed when treating the condition.

Atopy or Allergic Dermatitis

Description

Atopy is a condition that results from inhalant or skin absorption of environmental allergens, characterized by the animal's production of allergy associated antibodies (IgE). It is seen in both dogs and cats with a possible genetic predisposition, as purebred animals are at higher risk than mixed breed dogs or domestic breed cats, although all animals are at risk. Onset is usually within the first few months to years of life (1–3 years most common). Atopy may be seasonal or year-round dependent on the offending allergen. Seasonal allergies are worst in the spring and fall, as pollen counts are high. Indoor allergens are also possible as a result of dust, smoke, or grain.

TECH BOX 10.11: The term "atopy" is used in cases of contact or inhalant allergies. These allergies may be associated with outdoor allergens or something in the animal's indoor environment.

Clinical Signs

- Pruritus causes the animal to scratch at ears and underside of body, lick, chew, and rub their face.
- Other clinical signs of atopy include runny nose, watery eyes, sneezing, salivary staining of fur, erythema [Figure 10.15(a)], scaling, crusting, alopecia, hyperpigmentation and thickening of skin, and otitis.
- Seborrhea, pyoderma [Figure 10.15(b)], and self-trauma lesions complicate the condition.

Diagnosis

- Clinical presenting signs, physical exams, history, and rule out other causes of dermatitis are a starting point for a diagnosis.
- A definitive diagnosis can be gained with intradermal or serologic allergy testing.

Treatment

- The treatment of choice is immunotherapy or allergen exposure for hyposensitization that can be done using allergy vaccines. The patient is exposed to the specific allergens to desensitize the immune response.

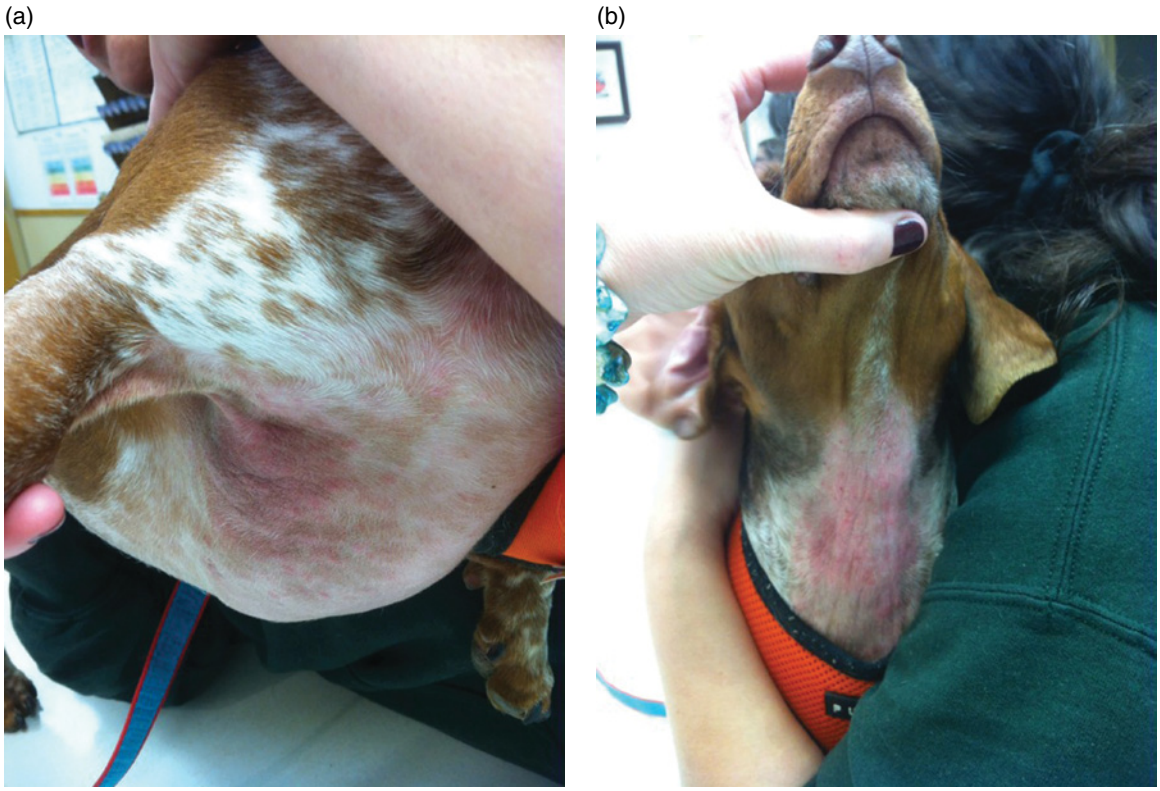

Figure 10.15 (a) Contact dermatitis after contact with carpet cleaner. (b) Contact dermatitis with mild pyoderma after contact with carpet cleaner. (Images courtesy Deanna Roberts)

Technician Duty Box 10.3

One of the treatment options for atopy is the administration of allergy injections for desensitization purposes. Commonly veterinary technicians will train owners to administer these injections, yet in some cases clients will bring their animals into the clinic for these injections to be administered by the staff.

- Palliative therapy includes antihistamines, corticosteroids, antibiotics, fatty acids, medicated shampoos, and immunosuppressive drugs (cyclosporin).
- Ideally, the animal's environment should be ridded of allergens, but this is nearly impossible to do. To help reduce clinical signs the animal should be kept indoors with windows kept shut during high pollen seasons, bedding should be washed frequently, and air ducts should be cleaned with high-quality filter systems.
- Most patients will respond best to a combination of therapies.

Client Education and Technician Tips

- This is a lifelong disease that will require long-term management and follow-up. Immunotherapy requires working long term with a veterinarian and the owner being willing to give injections to the animal. Treatment can get costly.

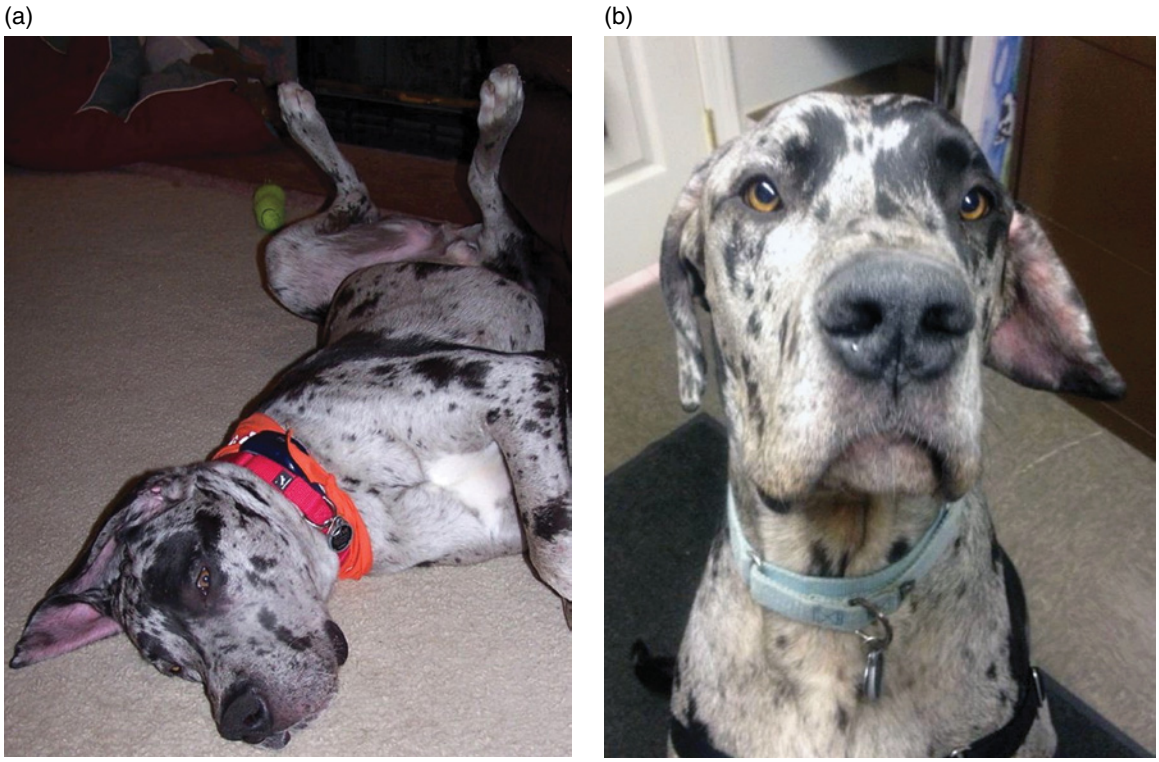

Figure 10.16 (a) Food allergy dermatitis with erythema of the ventral side of body and ears. (b) Food allergy dermatitis causing otitis and hematoma as a result of self-trauma. (Images courtesy Amy Johnson)

Food Allergy

Description

Food allergies are characterized by a reaction to an antigen in food, usually a carbohydrate or protein source. These are common allergies seen in both dogs and cats usually early in life. In adult onset cases the allergen has commonly been fed for more than 2 years. Food allergies will manifest themselves as both gastrointestinal and skin problems. Many animals with food allergies may also have inhalant/contact allergies.

Clinical Signs

- Clinical signs are very similar to those with atopy, other than they are non-seasonal. These signs include pruritus, erythema [Figure 10.16(a)], otitis [Figure 10.16(b)], seborrhea, pustules, self-traumatic skin lesions, and secondary bacterial and fungal skin and ear infections.

Diagnosis

- Presenting clinical signs, physical exam, history, and ruling out other causes of dermatitis will aid in diagnosis.
- A strict food elimination diet can aid in diagnosis and isolating the offending antigen.
- Serological and ID testing are available but with questionable reliability.

Treatment

- Feeding hypoallergenic diets avoiding the offending antigen will resolve clinical signs

(if there are not other allergies). Most animals respond well to grain-free, limited ingredient diets with a novel protein and carbohydrate source. Another option is feeding a hydrolyzed/denatured protein source.
- Palliative treatments include antihistamines, corticosteroids, and fatty acid supplements.
- Antibiotics may be necessary for secondary infections due to self-trauma.

Client Education and Technician Tips

- Food allergies are lifelong and animals may react to new ingredients over time and see a recurrence in clinical signs.
- Diets are very strict and involve restricting treats and table scraps.
- Not all diets are going to be allergen free. Owners need to be encouraged to read labels well. Although an offending allergen may not be first on the list of ingredients, there may be another source lower on the list. For example, a lamb diet may have chicken broth or fat as flavor.

TECH BOX 10.12: Limited ingredient diets need to be strictly followed. Owners need to restrict the offending allergen not only in the animal's main diet but also restrict treats, dietary supplements, and table scraps.

Epidermal Inclusion Cysts or Sebaceous Cysts

Description

Sebaceous cysts are benign cysts that occur as a result of a hair follicle or skin pore becoming clogged by dirt, sebum, scar tissue, or infection.

Clinical Signs

- The cyst will appear as a raised nodular cyst formation (Figure 10.17). If these cysts open they will discharge a thick waxy or fatty white/grey/brown/yellow discharge.

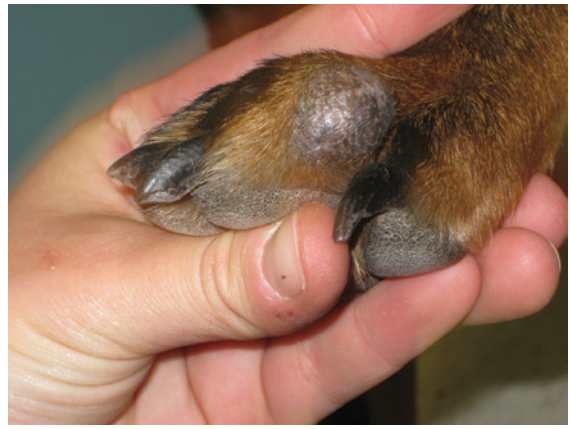

Figure 10.17 Sebaceous cyst. (Image courtesy A. K. Traylor, DVM, Microscopy Learning Systems)

Diagnosis

- Presenting clinical signs and physical exam will provide a presumptive diagnosis.
- A definitive diagnosis can possibly be obtained with an FNA, although they may require removal and biopsy.

Treatment

- Some sebaceous cysts will resolve on their own, but many will require surgical removal.
- Antibiotic or corticosteroid therapy to resolve secondary infection and inflammation if necessary.

Feline Acne

Description

Feline acne is a condition similar to human acne developing in the sebaceous glands under the chin and around the lips of cats. The condition results from a blockage of skin pores, usually with excess sebum and keratin production. Cases may be acute or chronic. Many cases are idiopathic but some may be linked to seborrhea, stress, bad grooming habits, and the use of plastic bowls for food or water.

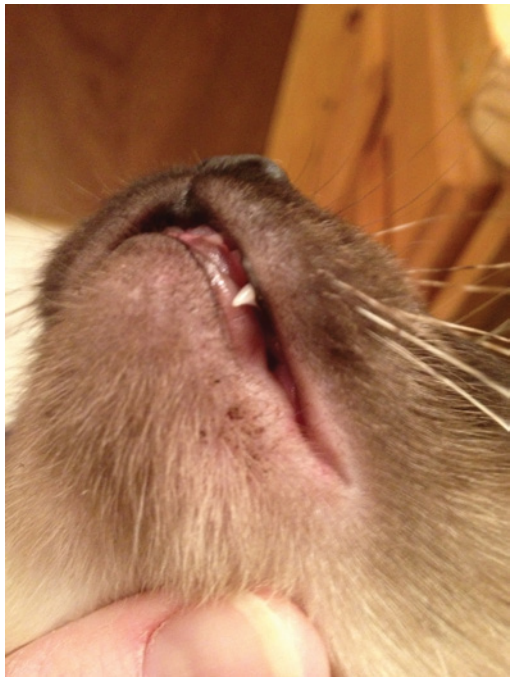

Figure 10.18 Mild feline acne. (Image courtesy Amy Johnson)

TECH BOX 10.13: There are varying degrees of feline acne. Many cats will have mild cases, while others will have open lesions and secondary complications.

Clinical Signs

- Cats will have visible blackheads (Figure 10.18) or pimple-like pustule lesions. These lesions may ulcerate and bleed or discharge purulent material.
- The lesions may be pruritic and cause excess scratching of the chin and alopecia.
- Self-trauma and open lesions may also cause a secondary pyoderma.

Diagnosis

- Clinical signs and physical exam are all that are necessary to determine a diagnosis.
- Cytology of the lesions can be performed if pyoderma is suspected.

Treatment

- Cleansing the chin with medicated scrub will clear most lesions. Long-haired cats may need their chins clipped to make the cleansing process easier.
- Medicated shampoos can be used in cases of seborrhea or pyoderma.
- Some cases may require antibiotics, especially if secondary bacterial infections suspected.
- Severe cases may require lancing and flushing of the lesions.

Client Education and Technician Tips

- Because of a possible link to plastic dishes, many cats will respond well with a switch to ceramic or stainless steel dishes.

Neoplasias Originating from the Skin and Associated Structures

Cutaneous Mast Cell Tumor

Description

Mast cells are found anywhere in the body but are highly concentrated in the skin. Tumors that arise from these mast cells are the most common potentially malignant tumor in dogs and the second most common cutaneous tumor in cats, although most feline MCTs are benign. These tumors can be located anywhere and found in any age of animal. MCTs are particularly harmful due to the release of damaging histamine from mast cells, which can cause further tissue damage and gastric problems.

TECH BOX 10.14: Mast cell tumors can be found anywhere on the body and will cause systemic complications due to the damaging histamine release.

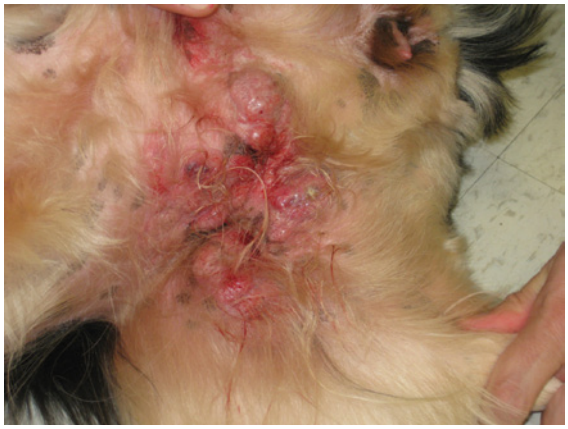

Figure 10.19 Mast cell tumor found on canine inguinal region. (Image courtesy A. K. Traylor, DVM, Microscopy Learning Systems)

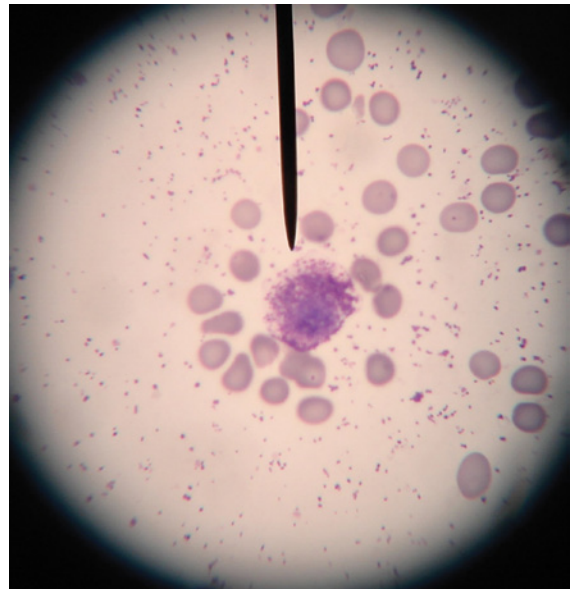

Figure 10.20 Microscopic image of a mast cell tumor aspirate, stained with Diff Quik. (Image courtesy Bel-Rea Institute of Animal Technology)

Clinical Signs

- An MCT will appear as a raised, nodular mass that may be soft or solid. There is wide variety in the appearance of the mass (Figure 10.19).
- Cats will tend to have a single nodule surrounded by alopecia found on the head and neck. Cats with an MCT are usually middle-aged and Siamese seem to be more predisposed than other breeds.
- Dogs more commonly have multiple masses found on the trunk, in interdigital spaces, and on the limbs. There is a genetic predisposition in Boxers, English Bulldogs, Boston Terriers, Shar-Peis, Labrador Retrievers, Golden Retrievers, Schnauzers, and Cocker Spaniels.

Diagnosis

- Presenting clinical signs, history, and physical exam will aid in diagnosis and eliminating other causes.
- A diagnosis can be obtained with an FNA (Figure 10.20).
- A biopsy will not only provide a definitive diagnosis but will stage the tumor. MCTs will be staged I–III, with III being the worst prognosis.

Treatment

- Tumors should be surgically removed with wide, deep margins. Cats may be good candidates for cryosurgical removal.
- Radiation, hyperthermia, and corticosteroid therapy are often used in conjunction with surgery, as a combination of multiple therapies is often used.
- Animals may also be treated with antihistamines to stop or slow the release of histamine from the mast cells.

Client Education and Technician Tips

- Prognosis is dependent on stage, but dogs tend to have a more guarded prognosis than cats.

- Cats usually have a good prognosis, unless the tumors are visceral.

Cutaneous Histiocytoma

Description

A histiocytooma is commonly a benign tumor that arises from histiocyte cells (Langerhans cells), cells that have an immune function. These tumors are seen in dogs, most commonly Boston Terriers. Although they can be seen at any age, most dogs are less than 3 years old.

Clinical Signs

- A histiocytoma is a solitary raised ulcerated nodule. They are fast growing and normally non-painful (Figure 10.21).
- The ulcerated lesion can cause a secondary bacterial infection.

Diagnosis

- These tumors are not easily diagnosed, as cytology can be confused with that of many other tumors.

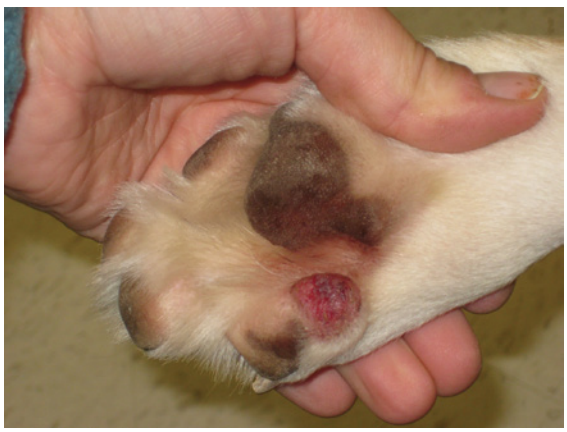

Figure 10.21 Cutaneous histiocytoma. (Image courtesy A. K. Traylor, DVM, Microscopy Learning Systems)

- Cytology or biopsy will be used to determine the diagnosis.

Treatment

- Most histiocytomas will resolve without treatment and are unlikely to recur.

TECH BOX 10.15: Histiocytomas are frequently benign and will often resolve without treatment necessary.

- If not resolving or if they are causing problems for the dog they can be removed surgically or through cryotherapy.
- Antibiotics will be used if there is a secondary infection.

Melanoma

Description

Melanomas are tumors that arise from melanocytes of the skin and are divided into two categories based on malignancy. A melanocytoma is a benign mass normally found on the head and forelimbs. Malignant melanomas are found on the lips, nail beds, and although not common they can be found on haired skin of the ventral abdomen and scrotum.

Both are more commonly found in older male dogs and very rarely in cats.

TECH BOX 10.16: Cutaneous melanomas are divided into two categories. A melanocytoma is a benign mass frequently cured with surgical excision. Malignant melanoma is a highly invasive neoplasia with a poor prognosis.

Clinical Signs

- Melanomas are raised ulcerative nodules usually melanotic, but amelonotic tumors can occur as well.

- If infiltrating the nail bed, the digit becomes inflamed and the patient may lose nail with underlying bone damage.

Diagnosis

- Presenting clinical signs, physical examination, and history can aid in making a diagnosis.
- Cytology from FNA or biopsy can more definitively diagnose melanoma.
- Radiographs can be used to determine bone damage and metastasis.

Treatment

- Surgical excision is recommended with either type of melanoma. With a melanocytoma, surgery is considered curative. Malignant melanomas require wide surgical excision and possible amputation due to the invasive nature of the tumor.
- Radiation and chemotherapy is not usually successful when it comes to malignant melanomas.
- A novel vaccine therapy has shown a potential therapeutic value.

Client Education and Technician Tips

- Malignant melanoma caries a poor prognosis, with few patients surviving even more than a year after treatment.

Cutaneous Squamous Cell Carcinoma

Description

Squamous cell carcinomas are one of the most common carcinomas arising from the skin. They are invasive tumors of the squamous epithelial cells seen in both dogs and cats as a result of prolonged exposure to sunlight, especially in pale-faced cats or white-skinned short-haired dog breeds. There may also be possible links to flea collars and cigarette smoke.

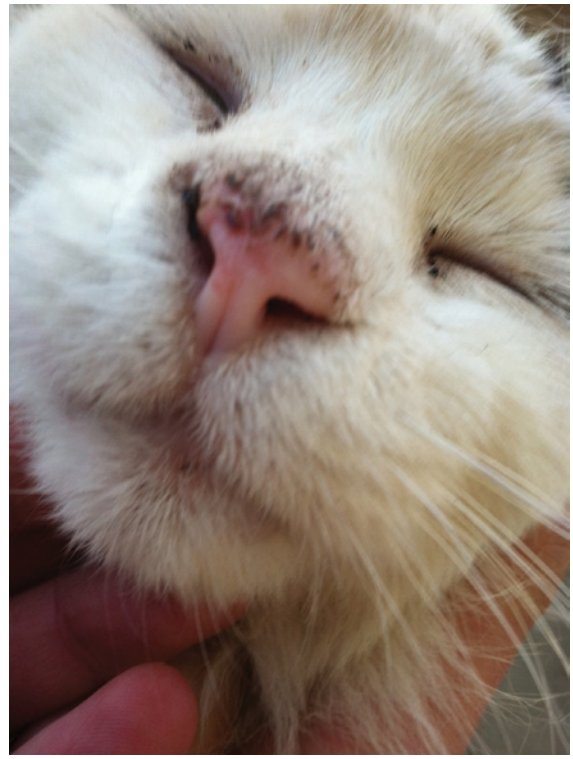

Figure 10.22 Feline SCC exhibiting an ulcerated, crusting lesion of the nose and nasal cavity changes. (Image of "Mr. Meow" courtesy Tammy Schneider)

Clinical Signs

- An SCC is a firm, raised, ulcerated plaque or nodule generally seen in older animals.
- Clinical signs include un-healing sores, crusting, white skin mass, thickened skin, hyperkeratinization, and erythema (Figure 10.22).
- The lesions in cats are most common on the lips, external nares, eyes, or ear pinna.
- In dogs the lesions are commonly found on the ventral side of the body and digits, limbs, anus, lips, nose, and scrotum. Lameness is seen if associated with limb or digit.

Diagnosis

- Presenting clinical signs, physical signs, and history will aid in a diagnosis.

- A definitive diagnosis is obtained via biopsy.
- Radiographs are necessary to determine if the tumor has metastasized.

Treatment

- Wide surgical excision and amputation are necessary due to the invasive nature of the tumor.
- Surgery can be coupled with radiation or chemotherapy.

Client Education and Technician Tips

- Limiting sun exposure will help to prevent SCC, especially in pale-skinned short-furred animals. Sunscreen may have success, but it is best to keep animals indoors during times of high sun.
- SCC is more common in high altitudes because of higher sun exposure, making it more important to watch sun exposure in these animals.

TECH BOX 10.17: SCC is a neoplasia with a strong link to sun damage. Prevention is key and includes protecting animals from sun exposure, especially at peak sun times.

Lipoma

Description

A lipoma is a benign tumor originating from adipocytes. They occur most frequently on the limbs, trunks, and ventral abdomen of obese female dogs but can be seen in older neutered dogs and Siamese cats.

Clinical Signs

- A lipoma is a soft, freely moving nodular mass under the skin. They are normally found during palpation, as they not always highly visible.
- Some animals are likely to develop multiple masses throughout the body.

Diagnosis

- Presenting clinical signs, physical examination, and palpation will aid in a diagnosis.
- Cytology from FNA or biopsy reveals adipocytes or fatty material. Alcohol stain fixatives will dissolve material on slide and masses most often float in formalin.

Treatment

- Masses can be surgically removed, although removal is not necessary unless it is inhibiting the animal's movement or making the animal uncomfortable.

TECH BOX 10.18: A lipoma is a benign mass and may not require treatment.

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Musculoskeletal Disease

chapter 11

The musculoskeletal system utilizes the bones, joints, cartilage, tendons, ligaments, and muscles to give the animal structure. This system, working in conjunction with the nervous system, allows for movement. The bones also have the added function of protecting vital internal organs. Disorders of the musculoskeletal system are often associated with pain, limit the animal's mobility, and can be seen with the bones, joints, or muscles.

Bone Fractures

Description

Commonly due to trauma, bone fractures can be a major problem as they may also involve complications with the blood, nerves, joints, or muscles. Broken bones can also potentially injure organs and local tissues as well as involve shock and infection. Other causes of bone fractures can be pathological, nutrient deficiencies, bone tumors, hormonal imbalance, or infection. Fractures are classified based on whether they are contained in the skin or not, the completeness of the break, and the pattern of the fracture line.

Fractures can be open or closed:

- Open fractures (also called compound) are fractures where the bone penetrates through the skin and is visible externally.
- Closed fractures (also called simple) are contained within the skin.

Fractures can also be complete or incomplete:

- A complete fracture is one where the fracture line is complete from one side of the bone to the other (Figure 11.1).
- An incomplete fracture has not broken all the way through.

Types of fractures based on the fracture line:

- Transverse fractures are complete where the fracture line is at a right angle to the bones axis.

Small Animal Pathology for Veterinary Technicians, First Edition. Amy Johnson.

Companion website: www.wiley.com/go/johnsonvettechpath

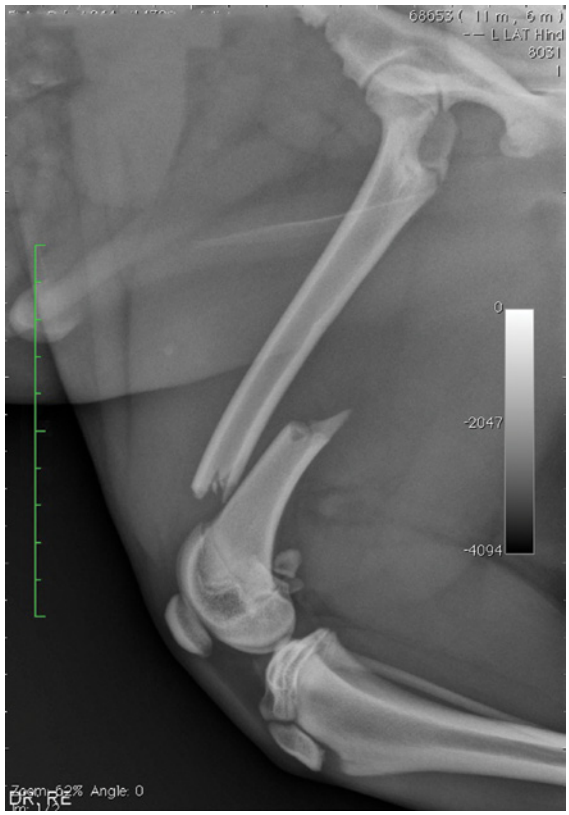

Figure 11.1 A complete fracture of the femur. (Image courtesy Dr. Robert Roy/Palm Beach Veterinary Specialists)

- Oblique fractures are complete where the angle of the fracture line is at an angle to the axis of the bone.
- Spiral fractures are complete where the angle of the fracture runs around the axis of the bone.
- Comminuted fractures are complete and involve multiple small bone fragments.
- Linear fractures (also called fissured fractures) are incomplete where the fraction line runs parallel to the axis of the bone.
- Greenstick fractures are incomplete where the bone is not separated but bent.
- Compression fractures are the collapse of a vertebra.
- Condylar fractures are fractures where the condyle of a bone is broken off. These are usually seen with the humerus, femur, or tibia.
- Avulsion fractures involve the loss of a section of bone and are generally due to muscle contractions (Figure 11.2).

Clinical Signs

- Fractures tend to be very painful, which can present as whining or other vocalizations, aggression, and inability to get comfortable.

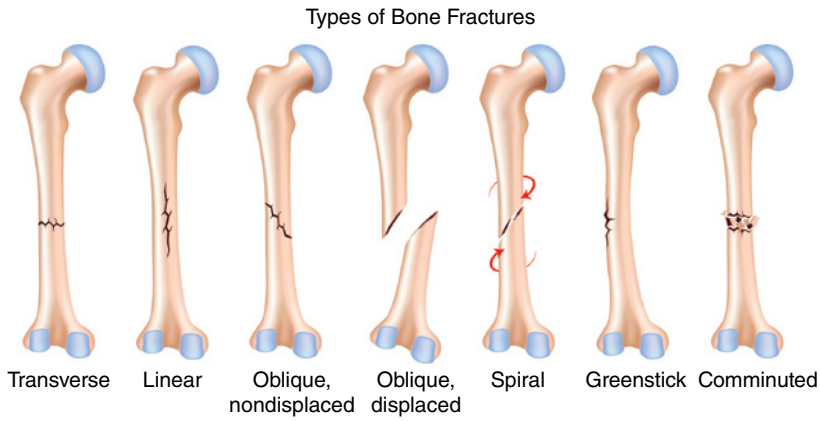

Figure 11.2 Common types of bone fractures. (Shutterstock image courtesy Alila Medical Images)

- Other clinical signs include lameness, possibly non-weight bearing, inflammation, or change in limb formation.
- Open fractures will have a visible bone outside of the skin.

Diagnosis

- Presenting clinical signs and history, especially if there is a history of trauma, can aid in diagnosis.
- Crepitus upon palpation may be noted.
- Radiographs will allow for visualization and classification of the fracture.

Treatment

- Many fractures can be treated with splints or casts and rest.
- Some fractures will require surgical repair including bone pins, plates, wires, and screws. External fixators are also a possibility for repair. Bone grafts can be used to help the healing bone (Figure 11.3).

Technician Duty Box 11.1

Orthopedic disorders requiring casts, splints, bandages, or external fixation devices will require extra care and maintenance. The veterinary technician will be given the task of training owners in how to care for their animal and their healing injury. Technicians will also be given the task of application of these devices, monitoring their condition, and changing of these devices.

- Antibiotics may be prescribed to avoid infection, especially in the case of open fractures or surgical repair.
- Analgesics are necessary to keep the animal comfortable.
- In some cases physical therapy and rehabilitation may be necessary to help the animal regain proper function and muscle strength.

Technician Duty Box 11.2

Patients with orthopedic disorders will in some cases require extra help with ambulation, physical therapy, and rehabilitation. There are many clinics specializing in physical therapy and rehabilitation that employ veterinary technicians to work alongside the veterinarians and physical therapists to aid in the patient's recovery.

Client Education and Technician Tips

- Fractures are most often associated with trauma, which means there may be more damage than just the fracture. Many animals after trauma may experience shock, internal organ damage, or internal bleeding. Often there is more to focus on than just the fracture.
- Casts and splints will necessitate special care requiring the owner to keep them clean and dry. Animals will also need to be monitored to make sure they are not trying to chew or bother the cast or splint.
- Post-operative wound/incision care is also an important key to avoid infections or further tissue trauma.
- Activity restriction is a must while the animal's bone is healing.
- Bones will take time to heal and will require follow-up exams and any necessary radiographs.

TECH BOX 11.1: No matter how they are corrected, fractures will take time to heal. These patients will require rest; good nutrition; maintenance of wounds, casts, or external fixators; and frequent follow-up visits to the veterinary clinic.

Osteosarcoma (OSA)

Description

OSA is the most common primary bone tumor in small animals and is seen in both dogs and cats,

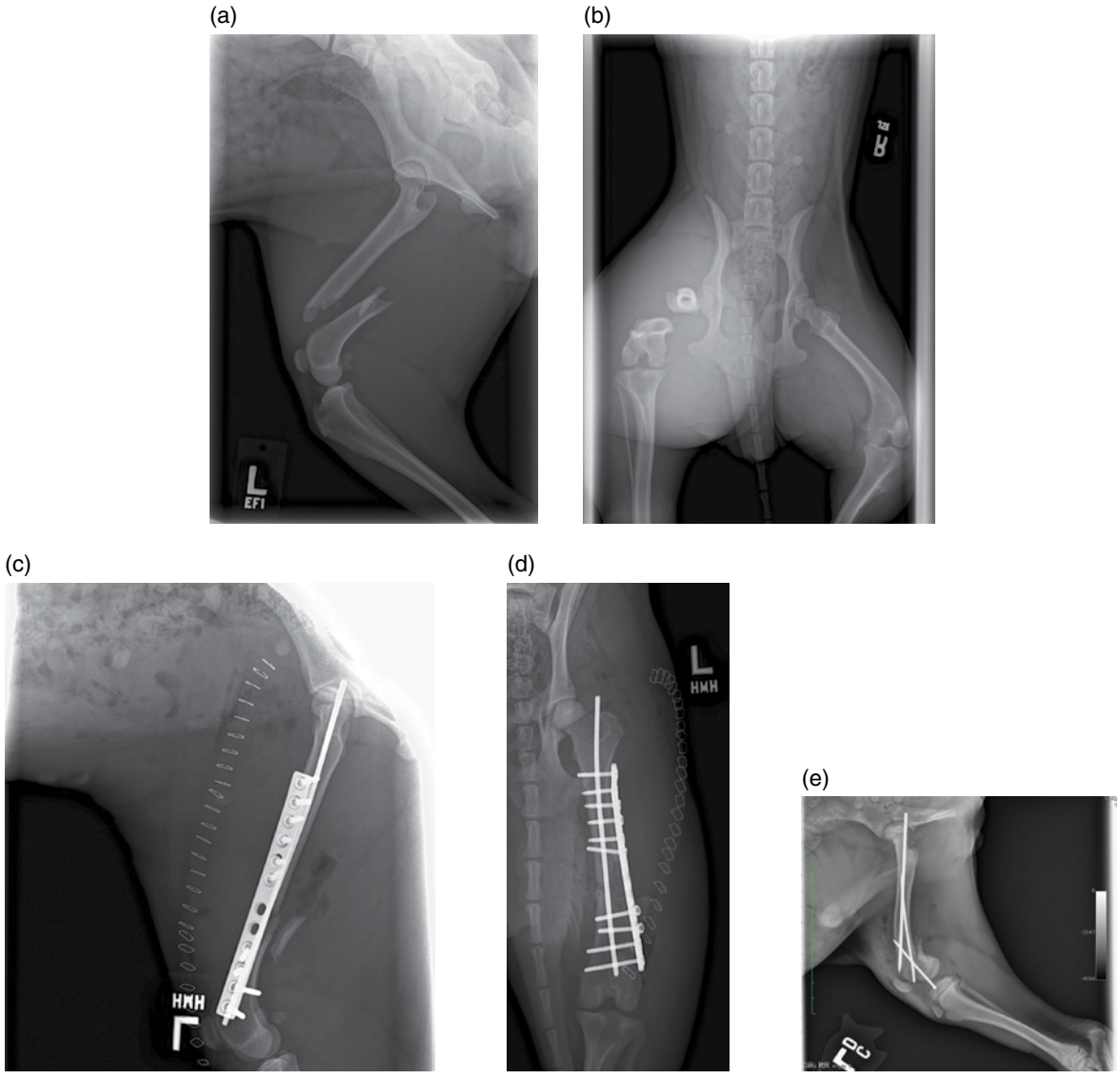

Figure 11.3 (a) and (b) Complete femur fracture prior to surgical repair. (c) and (d) Complete femur fracture post-surgical repair with bone plate, screws, and pin. (Images courtesy Jessie Gibbons) (e) Femur fracture repair with bone pins. (Image courtesy Dr. Robert Roy/Palm Beach Veterinary Specialists)

with an unknown cause. Large breed dogs seem to be more predisposed, and although these bone tumors can be seen in any age they are more common in older animals. The tumor starts in the center of the bone and grows outward, becoming more painful as it expands in size. The most common sites include the distal radius, proximal humerus, distal femur, or proximal tibia. These are locally invasive, highly metastatic tumors that do not cross the joint space.

TECH BOX 11.2: Osteosarcomas are the most common primary bone tumor diagnosed in small animals, carrying a guarded prognosis for long-term survival.

Clinical Signs

- Clinical signs include lameness that will worsen with time, inflammation, and non-traumatic bone fractures.

Diagnosis

- A presumptive diagnosis may be made based on clinical presentation and history.
- Radiographs will reveal osteolysis and proliferation (Figure 11.4).
- A definitive diagnosis is made based on a bone biopsy.
- Thoracic radiographs should be performed checking for metastasis.

Treatment

- The most common treatment for OSA is a combination of amputation, chemotherapy, and/or radiation therapy.
- Newer bone-sparing techniques are being tried with wide surgical excision of the mass, bone grafts, then plating the bone back together. As of now, there are many complications involved in this process.
- Analgesics need to be an important part of treatment in order to keep the animal comfortable.
- Euthanasia is a consideration, especially if there is evidence of metastasis.

Client Education and Technician Tips

- Prognosis is guarded; even with treatment average survival times range from months to 2 years.

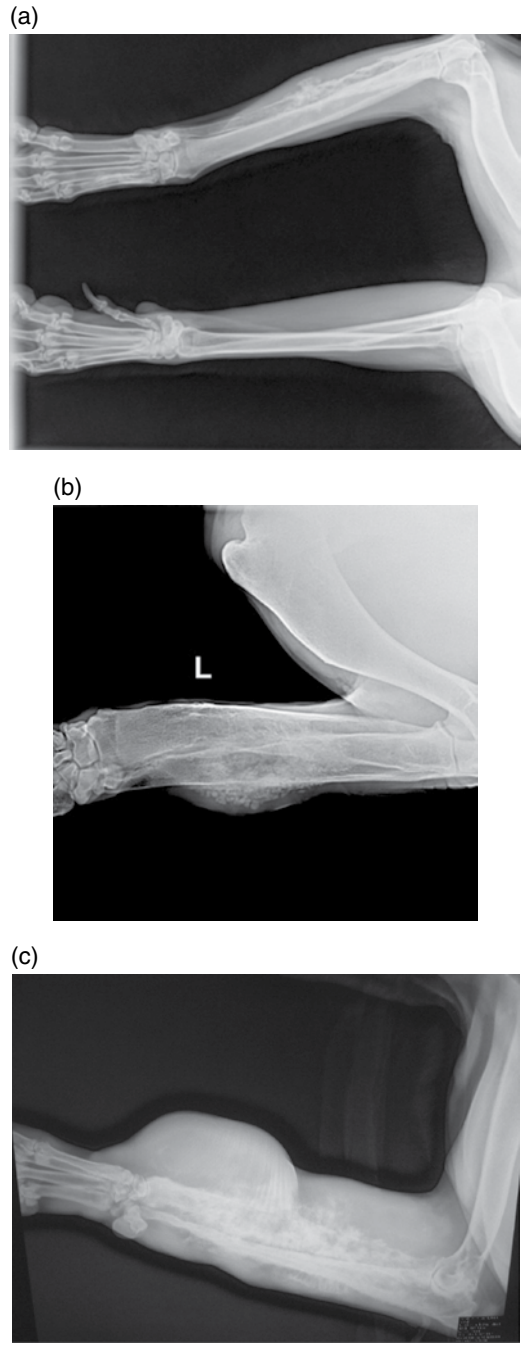

Figure 11.4 (a) and (b) Canine osteosarcoma lesion of the radius. (Image courtesy Deanna Roberts) (c) Canine osteosarcoma lesion of the radius/ulna. (Image courtesy Deborah Shaffer)

- Animals do very well with amputations and adjusting to ambulation on three limbs.

Panosteitis (Pano)

Description

Panosteitis is an acute, self-limiting condition associated with pain and inflammation in the long bones in quickly growing animals. This condition is seen most frequently in large and giant dog breeds, although it can be seen in any breed of dog and less commonly in cats. The cause of panosteitis is unknown, although genetics, stress, infection, or autoimmune factors are suspected.

TECH BOX 11.3: Panosteitis is frequently described as "growing pains" seen in young animals, especially large breed dogs. Animals will grow out of this condition with treatment aimed at making them more comfortable.

Clinical Signs

- The most common clinical sign is acute lameness due to bone pain in young dogs or cats. The lameness ranges from mild to severe and may be shifting and/or intermittent.
- Some animals will develop muscle atrophy.
- Other clinical signs include fever, anorexia, and lethargy.

Diagnosis

- A presumptive diagnosis is commonly based on presenting clinical signs, history, and signalment.
- Radiographs may reveal cloudiness in bone marrow cavity (Figure 11.5).
- Diagnosis may include ruling out other possible causes of lameness.

Treatment

- As animals will grow out of the condition, treatment is aimed at making the animal comfortable. This palliative treatment includes analgesics, steroids or other anti-inflammatories, and limiting activity.

Client Education and Technician Tips

- It is important to avoid excessive nutrients or dietary supplementation in young growing dogs. Excessive development contributes to the disorder.

Osteoarthritis or Degenerative Joint Disease (DJD)

Description

DJD is a progressive long-term deterioration of the joint cartilage, causing damage around the joint. This condition is seen more frequently in dogs but can present in cats as well. Causes of DJD include age and use, infection, trauma, developmental dystrophies, obesity, or autoimmune conditions.

TECH BOX 11.4: Osteoarthritis is a frequent condition seen in animals most often associated with age, yet can be seen secondarily to many conditions.

Clinical Signs

- Clinical signs include lameness, muscle atrophy, joint inflammation, crepitus, decrease in activity, and gait change.
- These clinical signs will become more prominent with exercise, weather changes, or inactivity.

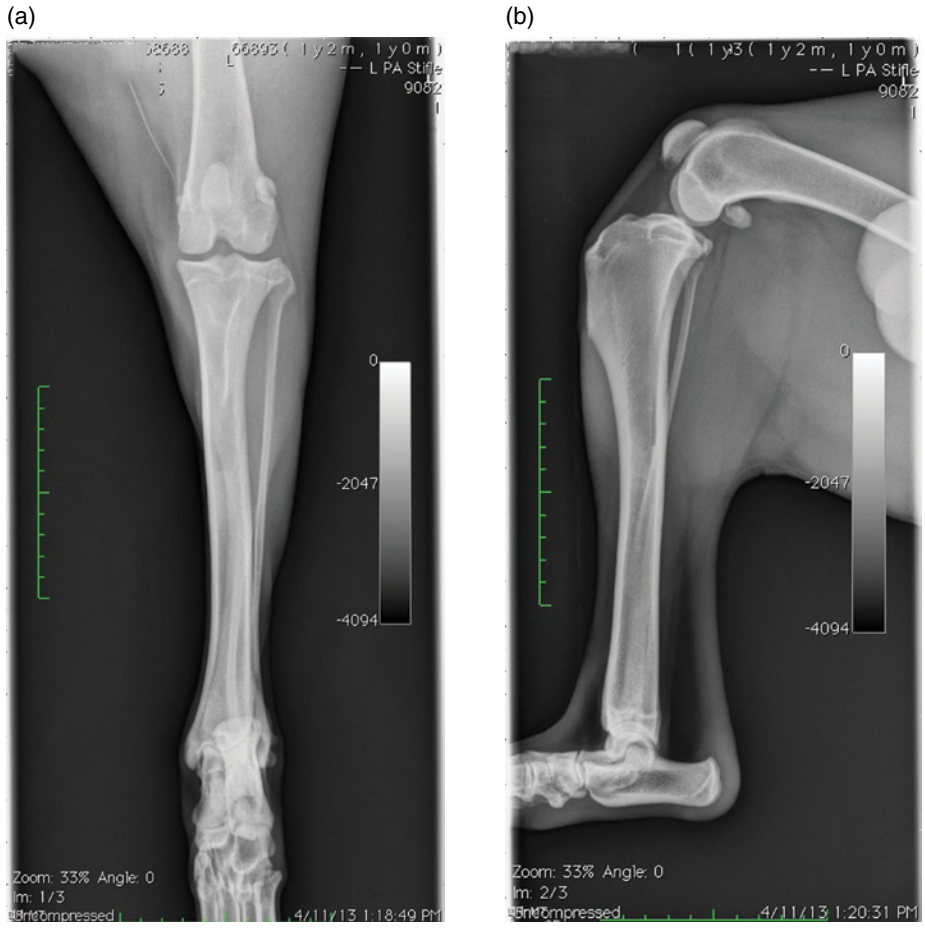

Figure 11.5 (a) and (b) Canine panosteitis radiographs. (Images courtesy Dr. Robert Roy/Palm Beach Veterinary Specialists)

Diagnosis

- A presumptive diagnosis can be made based on presenting clinical signs and patient history.
- Radiographs will show joint changes including narrowed joint space, bone sclerosis, osteophyte formation, subchondral cyst formation, and joint effusion, without inflammatory changes.
- An arthrocentesis may yield synovitis with generally very mild changes in clarity, cell counts, and color.

Technician Duty Box 11.3

Certain arthropathies are diagnosed with the help of synovial fluid evaluation. These tests can in many cases be performed in-house by veterinary technicians.

Treatment

- Medical treatment is palliative and includes weight loss and management, limiting exercise

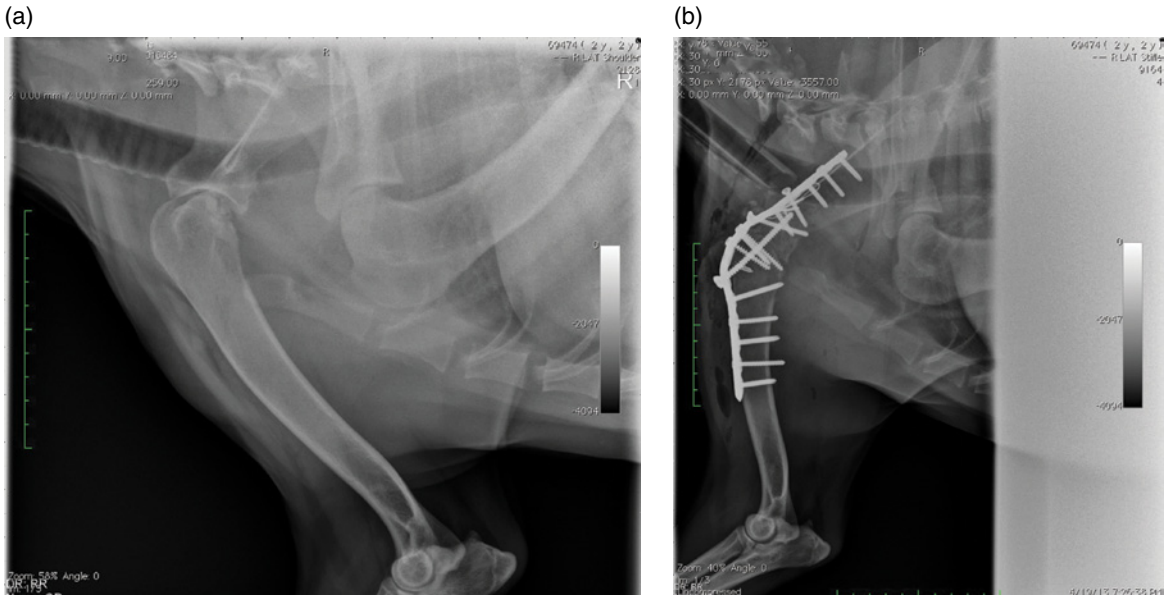

Figure 11.6 (a) Scapulohumeral joint preoperative radiograph exhibiting severe arthritic changes and decreased joint space. (b) Scapulohumoral joint arthrodesis post-operative radiograph. (Images courtesy Dr. Robert Roy/Palm Beach Veterinary Specialists)

(stairs, running, jumping), anti-inflammatories, analgesics, and a warm compress on affected joints. Many food companies now have specialized diets made for animals with joint problems that will include supplements to help with further degeneration of the joint surfaces.
- Physical therapy may aid in increasing joint mobility, muscle tone, and mass.
- Surgical treatments include arthrodesis (Figure 11.6), joint replacement, or joint excision.

Client Education and Technician Tips

- Many animals will be prescribed long-term NSAID therapy. These patients need continual monitoring of the liver and gastrointestinal system, as NSAIDs are linked to liver disease and gastric ulcers.
- Prognosis is questionable depending on age, severity of disease, joint(s) affected, and treatment of choice.

Hip Dysplasia

Description

Hip dysplasia, one of the most common skeletal disorders in dogs, occurs because of abnormal development of the coxofemoral joint. Hip dysplasia is associated with joint laxity and will lead to degenerative joint disease. Although it can be seen in any breed, it is most commonly seen in large and giant breed dogs. Causes of hip dysplasia include excessive quick growth linked to extra nutrients in the diet and genetics.

TECH BOX 11.5: Hip dysplasia, the most common skeletal disorder diagnosed in dogs, occurs due to unregulated growth and genetics. Because of the genetic link, animals with hip dysplasia should not be bred.

Clinical Signs

- Clinical signs will vary in degree of severity and will be worse after strenuous exercise.
- Clinical signs include lameness, an altered gait commonly referred to as "bunny hop" (moving hind legs together), reduced range of motion, pain, decreased activity, and crepitus.
- Animals with hip dysplasia will have difficulty getting up, running, jumping, and doing stairs.
- Muscle atrophy on hind limbs is common, which will cause hypertrophy on front limbs to compensate.

Diagnosis

- Presenting clinical signs, patient history, and physical exam will aid in a presumptive diagnosis.
- Radiographs will provide a definitive diagnosis by allowing visualization of the abnormal joint and arthritic changes (Figure 11.7). Radiographs should be taken based on protocols set forth by the Orthopedic Foundation for Animals (OFA), the University of Pennsylvania Hip Improvement Program (PennHIP), and Cornell's dorsolateral subluxation (DLS) test.

Treatment

- Medical treatment includes weight loss and management, physical therapy, anti-inflammatory drugs, and analgesics; although, similar to other chronic conditions, NSAID or steroid therapy needs to be closely monitored for side effects.
- Limited exercise is advised, although mild exercise is helpful. Non-slippery surfaces will also aid in the animal's mobility and comfort.
- There are several surgical options that range from modification of the joint to joint replacement, muscle modification, or removal of the femoral head.

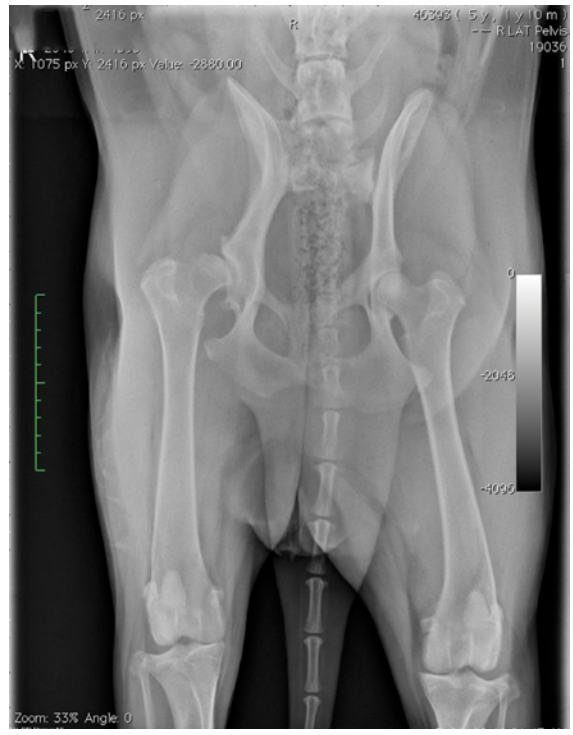

Figure 11.7 Canine hip dysplasia patient. Radiograph exhibits degenerative joint changes and abnormal positioning of the head of the femur and acetabulum. (Image courtesy Dr. Robert Roy/Palm Beach Veterinary Specialists)

- A *triple pelvic osteotomy (TPO)* is common in young dogs and modifies the shape of the acetabulum, allowing for better placement of the femoral head.
- A *total hip replacement (THR)* is more common in older dogs not responding to other therapies with DJD. In this procedure the acetabulum and femoral head are replaced with an artificial joint (Figure 11.8).
- A *femoral head osteotomy (FHO)* is the removal of the femoral head. This procedure eliminates the bone-on-bone contact and associated pain. The false joint becomes supported by the muscle mass.
- A *pectineal myotenectomy*, cutting of the pectineus muscle, will also relieve pain in hip dysplasia patients.

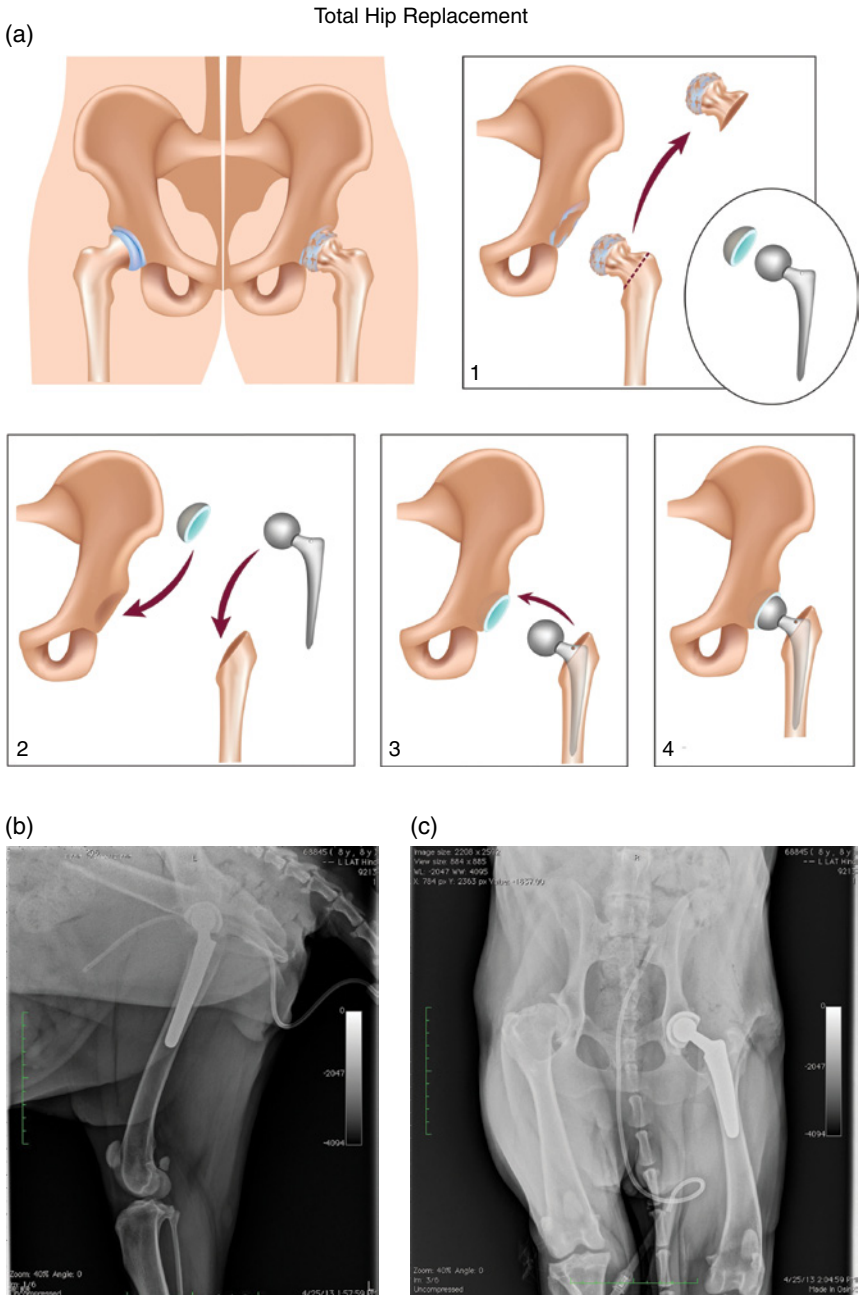

Figure 11.8 (a) Total hip replacement. (Shutterstock image courtesy Alila Sao Mai) (b) and (c) Post-operative THR radiographs. (Images courtesy Dr. Robert Roy/Palm Beach Veterinary Specialists)

TECH BOX 11.6: Owners have multiple options when determining how to treat their pet's hip dysplasia.

Client Education and Technician Tips

- Although hip dysplasia may not always be preventable, the signs can be lessened in predisposed breeds by maintaining an appropriate weight and not allowing an animal to grow too quickly by limiting nutrients.
- Due to the genetic nature of hip dysplasia, animals with the condition should not be bred.
- There are several organizations/programs, grading systems, and radiographic techniques for canine hips and hip dysplasia. These groups set standards for dogs based on age, breed, and conformation and set standard radiographic techniques.

Osteochondritis Dissecans (OCD)

Description

OCD is a condition that results from abnormal endochondral ossification of epiphyseal cartilage (abnormal bone formation from cartilage precursor), which leads to retention of excessive cartilage in the joint. The cartilage will form flaps and break off into the joint space. Joint debris leads to synovitis and osteoarthritis, further complicating the problem and adding to the arthralgia. OCD is seen in shoulder, elbow, stifle, and tarsal joints most commonly in large and giant breed dogs that are growing too rapidly. Causes include genetics, excessive nutrition and rapid growth, and joint trauma.

TECH BOX 11.7: OCD is a condition that may cause long-term joint damage.

Clinical Signs

- Clinical signs present most commonly in dogs 4–8 months of age and include lameness, arthralgia, inflammation, joint effusion, and muscle atrophy on affected limb. Dogs may not be able to bear weight on affected limb.
- Clinical signs will worsen after strenuous exercise.

Diagnosis

- Presumptive diagnosis can be made based on presenting clinical signs and history.
- Radiographs will show changes in the joint and bone (Figure 11.9), and "joint mice" (pieces of loose cartilage in the joint).
- Synovial fluid evaluation may indicate synovitis and help rule out infective processes.
- Arthrography and arthroscopy allows for better visualization of joint and cartilage.

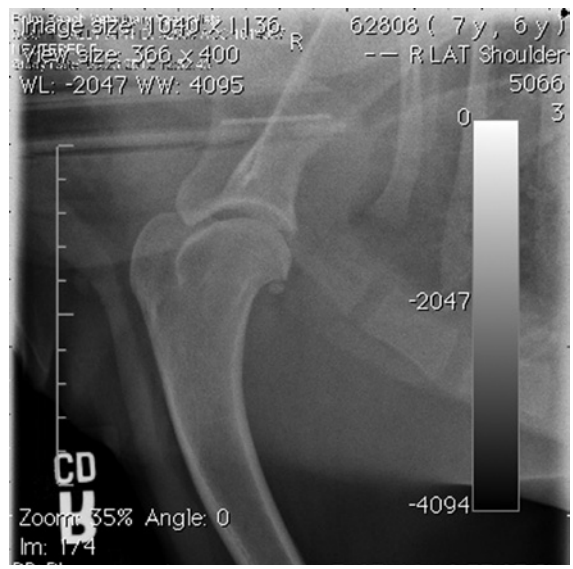

Figure 11.9 OCD radiograph of the scapulohumeral joint exhibiting bone and joint changes. (Image courtesy Dr. Robert Roy/Palm Beach Veterinary Specialists)

Treatment

- Surgical removal of joint debris and excision of cartilage flaps can be performed through arthroscopic or arthrotomy procedures. Curette of the bone edges will not only remove abnormal cartilage but stimulate fibrocartilage development.
- In cases where osteoarthritis has developed, anti-inflammatory drugs and analgesics will be necessary.
- Some minor cases may be resolved with limited exercise and drug therapy.

Client Education and Technician Tips

- Weight control and limited activity will be a necessary part of recovery.
- Prognosis is dependent on affected joint, as shoulder and stifle have a better chance of recovery than elbow or tarsus, and the degree of joint damage present.
- There is likely a genetic link so those animals affected should not be bred.

Patellar Luxation

Description

Patellar luxations involve the movement of the patella from the femoral (trochlear) groove where it should be located. The patella can move medially or laterally and the condition can be unilateral or bilateral. The disorder is often associated with other joint or bone disorders or limb deformities. Persistent problems can lead to osteoarthritis and long-term joint damage. Patellar luxations are seen in dogs and cats and are one of the most common stifle disorders in dogs. Toy and small breeds are more predisposed to patellar luxations, although they can happen in any breed. The most common cause seems to be genetics; trauma may also play a role.

Clinical Signs

- Animals with a hereditary luxation disorder will show clinical signs within the first few months of life, although some animals will develop clinical signs later in life.
- Animals will exhibit a "skipping lameness," where the affected limb will be held out behind them and they may possibly shake it. The animal may not bear weight on the affected limb, especially when the patella is out of the groove.
- An abnormal gait or conformation of the affected leg may also be noted. The animal will be bowlegged (medial luxation) or knock-kneed (lateral luxation).
- Pain is involved when the patella first moves out of place, but it is rarely painful once the patella is out of place.

TECH BOX 11.8: When dealing with a dog with a luxating patella, owners will notice the dog not bearing weight and possibly shaking the limb; but there is rarely pain associated with the condition, especially once the patella is out of its groove.

Diagnosis

- A complete orthopedic exam should be performed, and along with presenting clinical signs and history can aid in diagnosis. The stifle will feel unstable upon palpation.
- Radiographs will determine the degree of the disorder and limb changes. The disorder is graded based upon severity (Figure 11.10).

Treatment

- Surgical options include orthopedic or soft tissue options. The patella can be surgically fixed in place or the femoral groove can be deepened so the patella is less likely to luxate. Soft tissue surgeries include reconstruction of soft tissue surrounding the patella. The ligament on the side it luxates to is loosened, while the

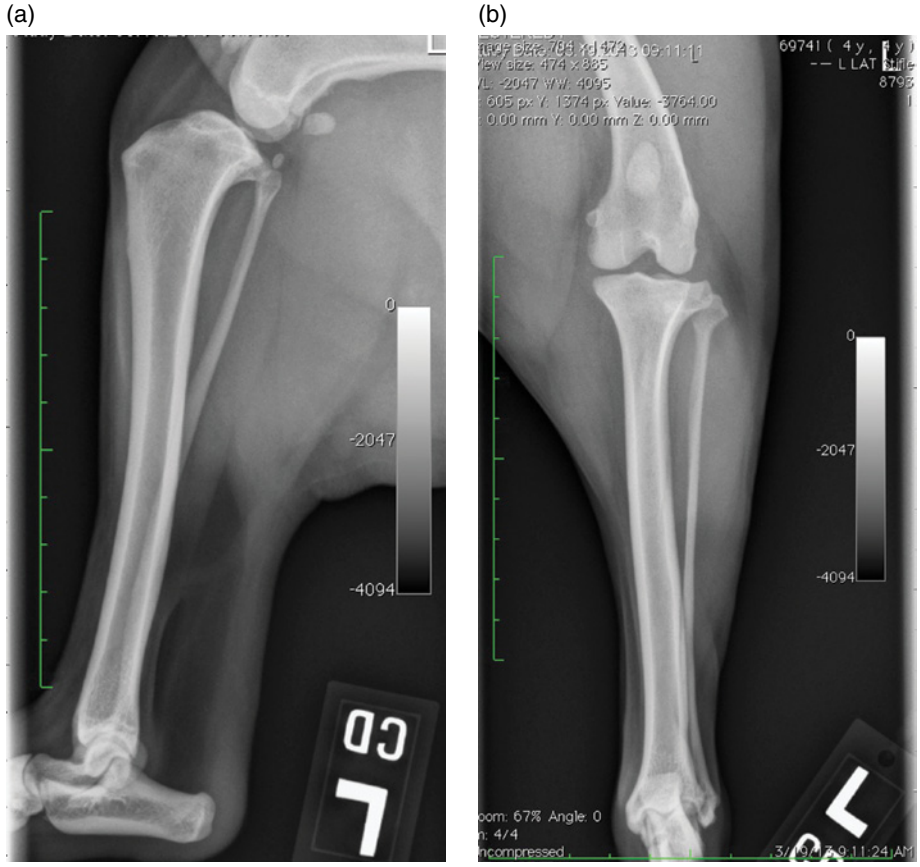

Figure 11.10 (a) and (b) Medially luxating patella preoperative radiography. (Images courtesy Dr. Robert Roy/Palm Beach Veterinary Specialists)

opposite side is tightened. Other surgical options include transposing of the tibial crest or correction of an abnormal femur. Severe cases may require arthrodesis, tibial osteotomy, or amputation (Figure 11.11).

Client Education and Technician Tips

- Because of genetic predisposition, animals with luxating patellas should not be bred.
- Problems may recur after surgery, but severity is often less than prior to surgery.
- Recovery requires exercise restrictions and limited activity.

Cranial or Anterior Cruciate Ligament (CCL or ACL) Rupture or Cranial Cruciate Ligament Disease (CCLD)

Description

ACL injuries, one of the most common reasons for hind limb lameness, involve a tear or rupture of the ligament, whose purpose is to stabilize the stifle. Commonly tears are in the middle of the ligament, although some may be at the point of origin and can be partial or complete. ACL damage occurs most commonly due to trauma but may be caused by a weakened ligament

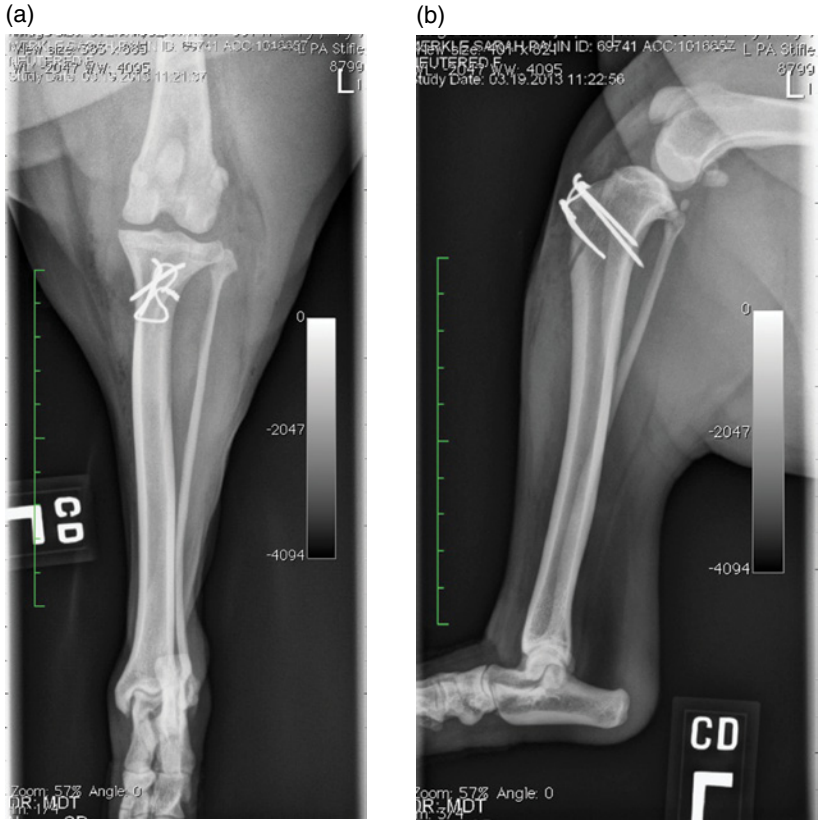

Figure 11.11 (a) and (b) Medially luxating patella post-operative tibial crest transposition (tibial osteotomy) radiography. (Images courtesy Dr. Robert Roy/Palm Beach Veterinary Specialists)

secondary to autoimmune disease, degeneration, or conformational deformities. Damage to ligament can lead to secondary osteoarthritis, injuries of the meniscus, and joint effusion.

TECH BOX 11.9: Cranial cruciate ligament injuries are the most common reason for hind limb lameness in dogs and should be on the rule out list anytime a dog comes in with unknown hind limb injuries.

Clinical Signs

- Clinical signs include lameness, non-weight bearing, pain, joint effusion, crepitus, abnormal gait, decreased activity, muscle atrophy, and decreased range of motion.
- Animals may experience difficulty with rising, running, or jumping or with stairs.
- A "drawer sign" (excessive cranial laxity of the tibia in relation to the femur) is often associated with ACL injuries. Increased tibial rotation may also be noted.
- Owners may notice animals with ACL injuries may sit abnormally. They will tend to put their legs out to the side of them instead of under them.

Diagnosis

- A thorough orthopedic exam should be performed, and along with presenting clinical

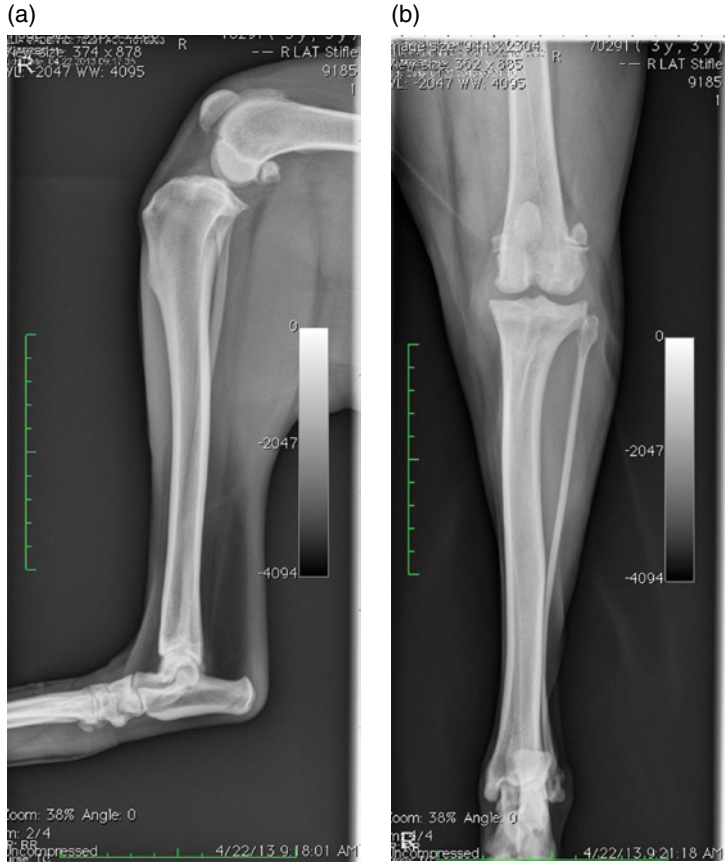

Figure 11.12 (a) and (b) Cranial cruciate ligament injury preoperative radiography. (Images courtesy Dr. Robert Roy/Palm Beach Veterinary Specialists)

signs and history will aid in diagnosis, especially if a drawer sign is noted.

- Arthrocentesis reveals inflammation and blood in the joint.
- Radiographs will show osteoarthritis and joint changes but not the ligament or soft tissue itself (Figure 11.12).

TECH BOX 11.10: Radiographs will not identify the ligament injury but will rule out other causes of hind limb lameness and help to evaluate joint changes.

- Arthroscopy and MRI will give a better picture of the state of the ligament.

Treatment

- Medical treatment includes weight loss and management, physical therapy, cage rest, anti-inflammatory drugs, analgesics, and possible joint supplements.
- There are several options for surgical repair of ACL injuries; the technique will vary based on the patient, complications, and surgeon's preference. The techniques are divided into osteotomy and suture techniques.
- Osteotomy techniques include tibial plateau leveling osteotomy (TPLO) and tibial tuberosity advancement (TTA). These techniques require a cutting of the bone. Stabilization of the stifle results not by replacing the ACL but

by changing the way the stifle works due to the orientation of the tibia and the quadriceps. These techniques are more common for large breed active dogs. The advantages of these techniques are that they will result in less progressive joint damage and a more stable joint as compared to the suture techniques.

- The *TPLO technique* changes the orientation of the tibia, resulting in a 90° attachment of the quadriceps (Figure 11.13).
- The *TTA technique* will advance the tibial tuberosity forward after a cut to the anterior aspect of the tibia, resulting in a 90° attachment of the quadriceps.
- The most common of the suturing techniques are extracapsular suture stabilization and a TightRope technique. These techniques work with the use of a strong suture material (often fishing line) outside the joint to stabilize the joint the way the ACL would. The advantages to these techniques are that they are less expensive and less traumatic, as no osteotomy is necessary, meaning less recovery time. The disadvantages include failure of the suture (common in large breed and active dogs) and long-term degenerative joint problems. *Extracapsular suture stabilization* has many variations, all using suture material in different ways to stabilize the stifle. The *TightRope technique* is a newer technique involving suture and toggles. This requires holes to be drilled in the bones but offers a stronger fixation for large active dogs than traditional suturing techniques. Despite the bone holes it is minimally invasive as compared to osteotomy techniques.

Client Education and Technician Tips

- Limited activity and cage rest during surgical recovery is imperative no matter what the technique. After the animal has had time to heal, light exercise will be necessary to build the muscle back up.

Intervertebral Disk Disease (IVDD)

Description

IVDD is characterized by a degeneration and protrusion of the disks located in between the vertebrae. The protrusion puts pressure on the spinal cord, causing CNS signs in patients. IVDD is a common spinal disorder in dogs and is rarely seen in cats. There is a genetic predisposition to dog breeds with chondrodystrophy (Dachshunds, Pekingese, Shih Tzus, Lhasa Apsos, Basset Hounds, Corgis, Beagles, and Cocker Spaniels) or fibroid degeneration (large breed dogs), although obesity and trauma can contribute to the disease. The disorder can be seen early in life but more commonly is acquired later in life. Common locations for disk problems are in the cervical and thoracolumbar spine.

Clinical Signs

- Clinical signs include neck and back pain, which will present as yelping, biting, trembling, and thoracolumbar kyphosis (arched back). Muscle spasms, rigid stance, or stiffness of head, neck, or back can be seen as well as lameness and lethargy.
- Because of the spinal cord compression the animals will have neurological deficits including ataxia, paraplegia or paralysis, urinary incontinence, toe knuckling, and abnormal reflexes.

Diagnosis

- Presenting clinical signs, history, physical and neurological exam, and breed will aid in a diagnosis.
- Radiographs, myelogram (Figure 11.14), MRI, and CT scans will further aid in diagnosis by allowing visualization of the ruptured disk, narrowed space, and spinal compression.

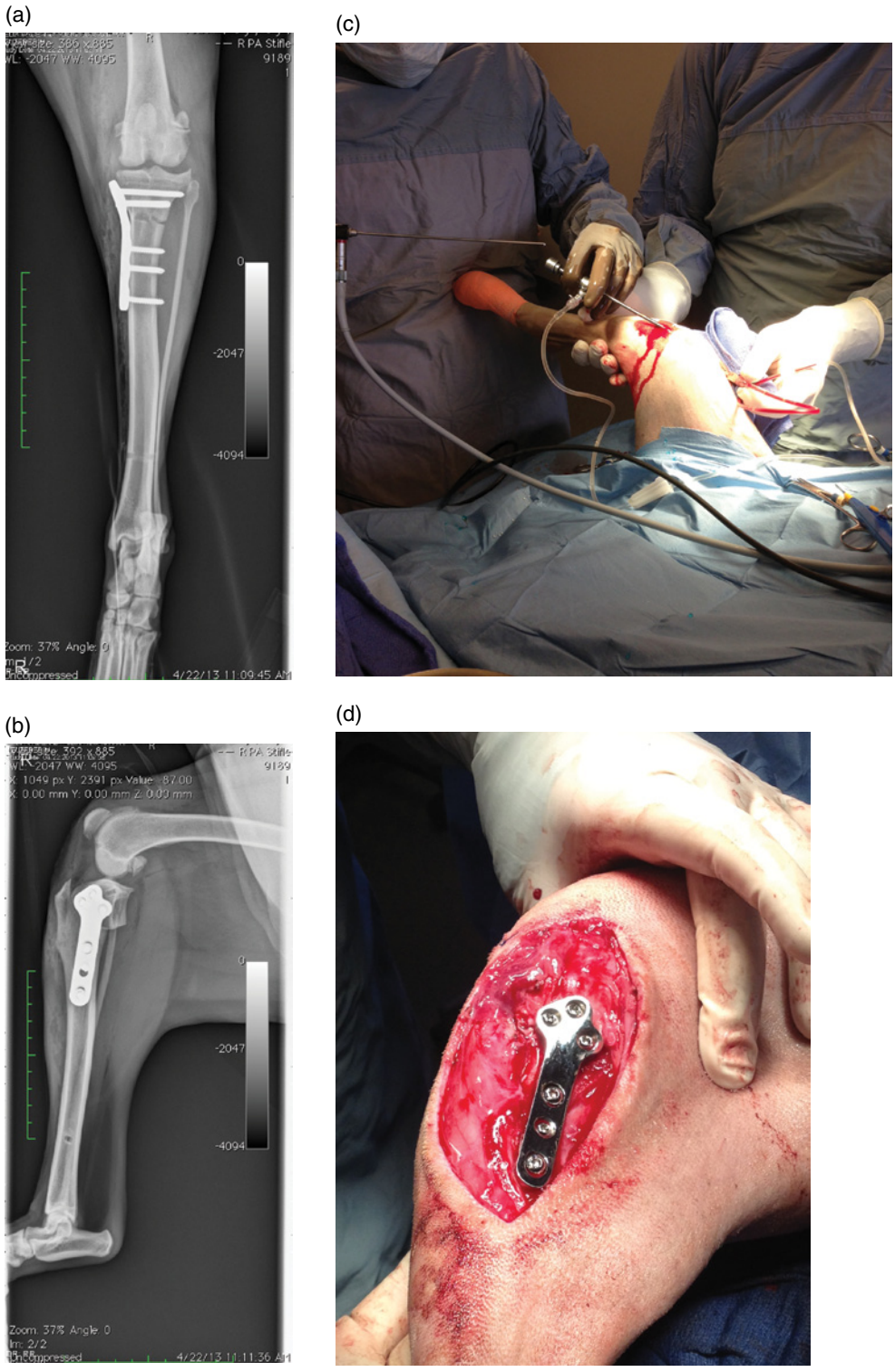

Figure 11.13 (a) and (b) Cranial cruciate ligament injury post-TPLO radiography. (Images courtesy Dr. Robert Roy/ Palm Beach Veterinary Specialists) (c) Arthroscopic TPLO procedure as a result of a partial tear in the cranial cruciate ligament. (d) Arthroscopic TPLO procedure showing the bone plate and screws. (Images courtesy Phillip Aughinbaugh, Dr. Robert Roy/ Palm Beach Veterinary Specialists)

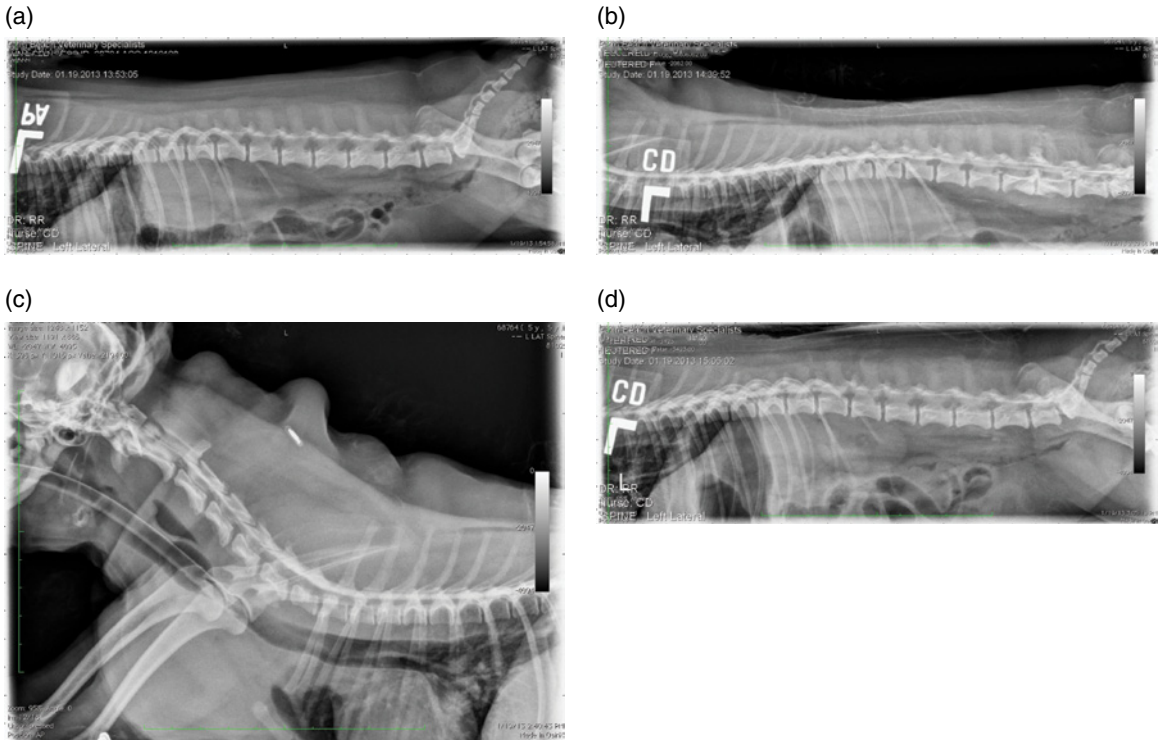

Figure 11.14 (a)–(d) Myelogram study performed to rule out IVDD. (a) Plain radiograph taken prior to injection of contrast material. (b) Contrast material running down the cord (bright white on dorsal aspect of spinal cord). (c) Contrast material running cranial, an indication that it can travel up and down the spinal cord without hitting resistance (disc compression). (d) Radiograph showing the contrast material has cleared. (Images courtesy Dr. Robert Roy/Palm Beach Veterinary Specialists)

Treatment

- It is imperative to treat promptly to avoid further compression of the spinal cord and worsening clinical signs.

TECH BOX 11.11: Although there are many factors involved in prognosis with IVDD, one of the most important factors is beginning treatment as soon as possible to prevent long-term neurologic damage.

- Patients with mild pain and neurological defects may respond to corticosteroids or other anti-inflammatory drugs, analgesics, and cage rest for several weeks. Clinical signs may recur with medical treatment only.
- Surgical options may be considered in severe cases, cases where cage rest has failed, or cases with recurrence. Surgery is aimed at decompressing the spinal cord by removal of disk material or removal of bone with a laminectomy.
- Some owners may elect to euthanize if prognosis is poor.

Client Education and Technician Tips

- Prognosis is dependent on severity of spinal cord compression, neurological deficit, and the time frame in which treatment has begun.

- Weight control is an important factor in prevention of the disorder.

Myasthenia Gravis

Description

Myasthenia gravis is a disorder characterized by a generalized muscle weakness and excessive fatigue as a result of a decreased number of acetylcholine (Ach) receptors at the neuromuscular junction. This disease affects both the nervous and muscular system and can be congenital or acquired. The acquired form of the disease is linked to autoimmune disease involving antibody destruction of the receptors and is believed to be of genetic origin. This condition is seen in both dogs and cats.

Clinical Signs

- Clinical signs include generalized weakness of skeletal muscles. Animals may collapse and experience exercise intolerance as a result of extreme fatigue. Weakened muscles may atrophy.
- Problems with other muscles include those associated with the esophagus, pharynx, and eye. Regurgitation and aspiration pneumonia are often seen and associated with megaesophagus secondary to myasthenia gravis. Animals may also experience a change in vocalization, sleeping with eyes open, and hypersalivation.
- A decrease in normal reflexes including blinking, gag, and spinal reflexes is also common with myasthenia gravis.

Diagnosis

- A physical exam and comprehensive neurological work-up, presenting clinical signs, and history will aid in diagnosis.
- Radiography or esophagoscopy can be used to diagnose megaesophagus (Figure 11.15).
- The current definitive test is immunologic testing ACh receptor antibody titers in serum.
- Also available is a test called a Tensilon test, which checks muscle response.

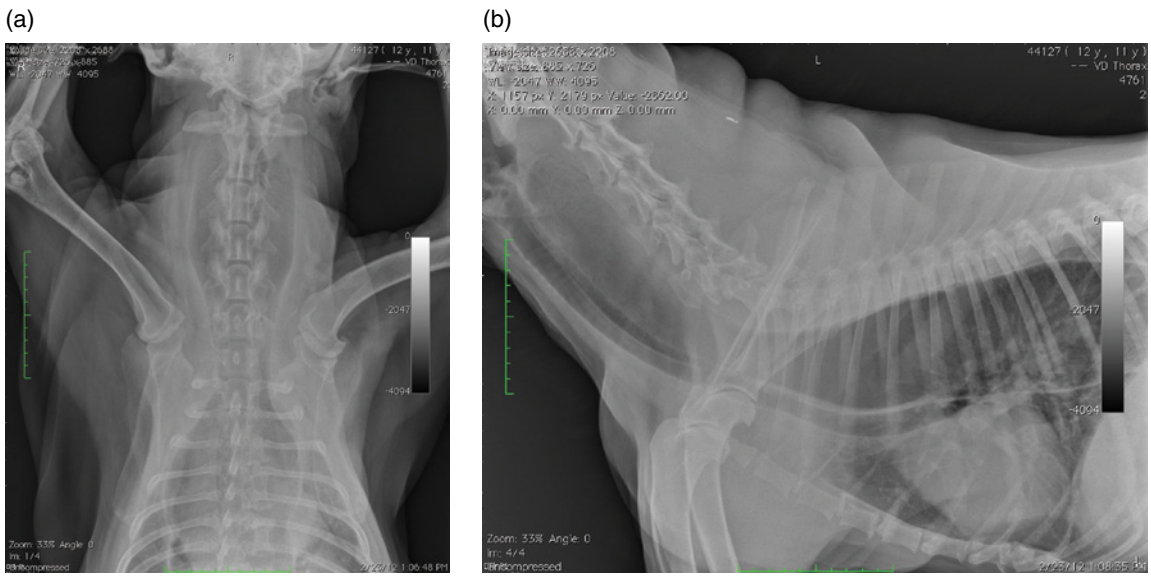

Figure 11.15 (a) and (b) Megaesophagus secondary to myasthenia gravis. (Images courtesy Dr. Robert Roy/Palm Beach Veterinary Specialists)

- Electrodiagnostic testing to look at action potentials after a muscle is stimulated is another option for testing.

Treatment

- Treatment is largely supportive and based on clinical signs and secondary issues the animal is experiencing. These treatments include treatment of aspiration pneumonia and nutrient therapy in cases of megaesophagus, IV fluids, and drugs to modify GI motility. If megaesophagus is present the animals need to be fed small, frequent, liquid meals from an elevated platform.
- Other treatments include anticholinesterase and immunosuppressive drugs.

Client Education and Technician Tips

- It is believed that vaccines may worsen this condition. Owners need to speak with their veterinarians and make decisions involving vaccination protocols.
- Many dogs will have spontaneous remissions throughout the illness.
- There is no prevention for this condition, although because there is a genetic link dogs with the condition should not be bred.

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Hematologic and Lymph Disease

chapter 12

The blood and lymphatic system are very important body systems, both for transportation and protection. The blood and its cells are responsible for the transport of oxygen and vital nutrients, protection from bleeding, and immunity. The lymph travels through lymphatic vessels with the primary function of disease protection and nutrient transport. Clinical signs of disease in these systems are associated with these functions.

Erythrocyte Disorders

Anemia

Description

Anemia, defined as a decrease in RBC numbers, is a condition measured by RBC count, PCV, and hemoglobin concentrations. There are three categories of anemia based on general cause. *Aplastic anemia* is caused by lack of erythropoiesis by the bone marrow, *hemolytic anemia* is characterized by RBC lysis, and *hemorrhagic anemia* occurs when there is a loss of blood. Anemia is also categorized by the bone marrow's response to the lack of RBCs. *Regenerative anemia* is seen when the bone marrow responds appropriately with erythropoiesis. Hemorrhagic and hemolytic anemia are often regenerative.

Non-regenerative anemia, when the bone marrow does not respond to the need for RBCs, occurs in cases of decreased erythropoietin (EPO) and bone marrow suppression. There are numerous causes of anemia including:

- Bacterial infection
- Viral infection
- Other infectious agents
- Toxins
- Autoimmune (covered separately)
- Renal failure
- Hemorrhage
- Hemolysis
- Nutritional deficiencies
- Neoplasia
- Liver disease
- Metabolic disorders

Small Animal Pathology for Veterinary Technicians, First Edition. Amy Johnson.

Companion website: www.wiley.com/go/johnsonvettechpath

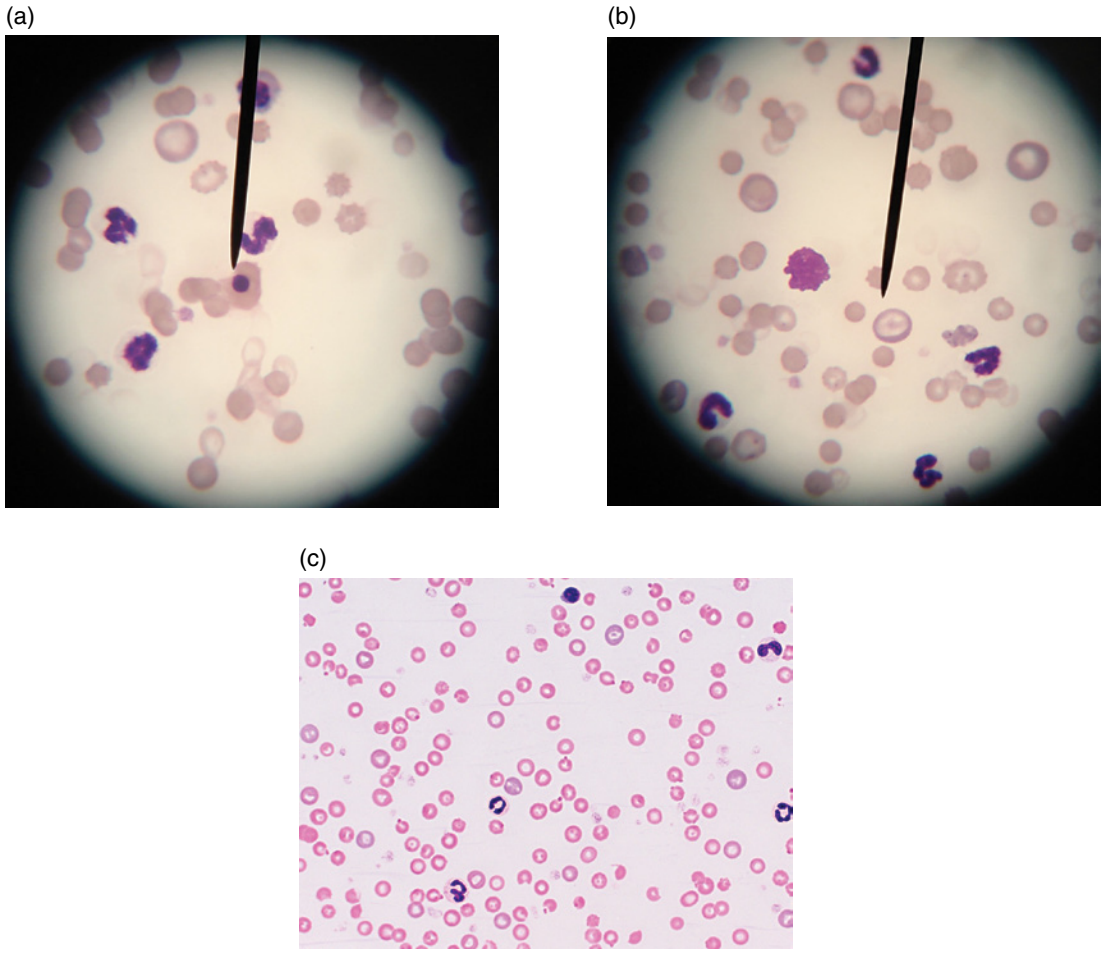

Figure 12.1 (a) Blood film from anemic patient; nucleated red blood cell (NRBC) seen at the end of pointer (Diff Quik). (b) Blood film from anemic patient; a polychromatophil seen at the end of pointer (Diff Quik). (Images courtesy Bel-Rea Institute of Animal Technology) (c) Blood film from anemic patient; Heinz bodies and polychromasia exhibited (Diff Quik). (Shutterstock image courtesy Vetpathologist)

Clinical Signs

- Clinical signs of anemia vary based on cause, degree of severity, and duration.
- Clinical signs include pale mucous membranes, tachycardia, weak pulses, hypotension, heart murmur, anorexia, lethargy, hypoxia, dyspnea, and tachypnea.
- Icterus will be seen in cases of hemolytic anemic as a result of the hemoglobin releasing bilirubin.
- In cases of hemorrhagic anemia obvious blood loss may be evident, but subtle internal bleeding may go unnoticed.

Diagnosis

- Presenting clinical signs, physical exam, and history will aid in developing diagnosis.
- Definitive diagnosis of anemia will require blood work including a CBC, PCV, and hemoglobin concentration evaluation, although this testing may not point to the primary cause (Figure 12.1; Table 12.1).
- Reticulocyte counts (Figure 12.2) and RBC indices can be used to determine if the anemia is regenerative or not.

Table 12.1 Anemia laboratory work

Morphology changes on blood film	Possible polychromasia, anisocytosis, spherocytes, ghost cells, Heinz bodies, NRBC Possible RBC parasite
Blood cell count changes	Decreased RBC count Decreased hemoglobin Possible thrombocytopenia
PCV/TP	Decreased PCV
Blood chemistry	Possible hyperbilirubinemia
Coagulation profile	Possible coagulation disorders
Reticulocyte count and RBC indices	Will determine regeneration

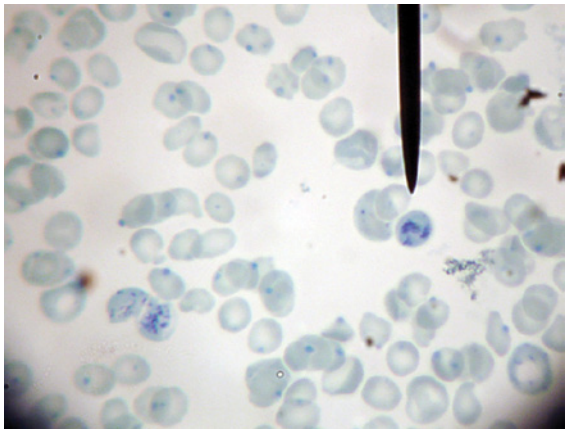

Figure 12.2 Reticulocyte slide from an anemic patient; reticulocyte seen at the end of the pointer with Heinz bodies throughout. (Image courtesy Bel-Rea Institute of Animal Technology)

- Blood film evaluation will aid in identifying certain RBC parasites as the cause of the anemia.
- Bone marrow aspiration and evaluation will be performed if it is suspected that the bone marrow is involved.

Treatment

- Diagnosis of primary cause is important in determining the treatment protocol, as treatment is based on the cause of the anemia. Some conditions can be treated; unfortunately others may be more difficult to treat.

TECH BOX 12.1: There are numerous causes of anemia. Identifying the underlying cause is essential in determining treatment and prognosis.

- Blood transfusions will replace RBCs and increase oxygen carrying capacity and blood volume, correcting circulatory shock. This may be a temporary solution, as the animal is still actively bleeding, undergoing hemolysis, or is not producing its own RBCs.

Technician Duty Box 12.1

Many hematological diseases will require blood transfusions. The recipient must be very closely monitored during the transmission. Veterinary technicians may also be involved in the collection of blood from donors, analyzing blood compatibility with the recipient, and appropriate management of blood bank products.

- Oxygen therapy may be necessary in cases of hypoxia, although the oxygen requires RBCs for delivery to the tissues (Figure 12.3).
- Fluids and other supportive measures will combat shock.
- Identification and correction of the primary cause will be necessary for long-term survival. EPO therapy can be used in cases where the patient has a decrease in this vital erythropoietic hormone; infections should be treated appropriately, or in the case of autoimmune disorders the immune system should be suppressed.
- Some cases may necessitate euthanasia if prognosis is poor and the animal is suffering.

Figure 12.3 Feline patient being treated in oxygen cage. (Shutterstock image courtesy Aspen Rock)

Client Education and Technician Tips

- Prognosis based on cause and effectiveness of therapy.

Immune-Mediated Hemolytic Anemia (IMHA)

Description

IMHA is a classification of anemia characterized by the body no longer recognizing the RBCs as its own and creating antibodies against them. With this disorder circulating RBCs are lysed by macrophages in the spleen and in some cases in the bone marrow. If the bone marrow is involved in the disorder the resulting anemia is non-regenerative; when there is no involvement of the bone marrow a regenerative anemia is seen. IMHA can be a primary or secondary disorder. Primary cases are idiopathic and most commonly seen in dogs. Cats are more likely to present with secondary cases caused by vaccines, drug therapies, neoplasia, or infections. Thrombocytopenia and pulmonary thromboemboli are commonly seen with IMHA, leading to further complications.

Clinical Signs

- As with other causes of anemia, patients with IMHA will have pale mucous membranes.
- Animals with IMHA will have general clinical signs including anorexia, lethargy, fever, vomiting, diarrhea, and PU/PD. Tachypnea, tachycardia, and heart murmurs may also be present with IMHA.
- Icterus is seen, as the lysed RBCs are releasing bilirubin as a part of hemoglobin.
- Animals with IMHA will normally have splenomegaly and hepatomegaly.
- Patients often have thrombocytopenia along with anemia, which will cause petechiation and other bleeding disorders.

Diagnosis

- Presenting clinical signs, physical exam, and history can aid in diagnosing anemia but not necessarily IMHA.
- A CBC, PCV, and urinalysis (UA) will give a definitive diagnosis of anemia. Agglutination microscopically (Figure 12.4) or macroscopically on a slide agglutination test is often

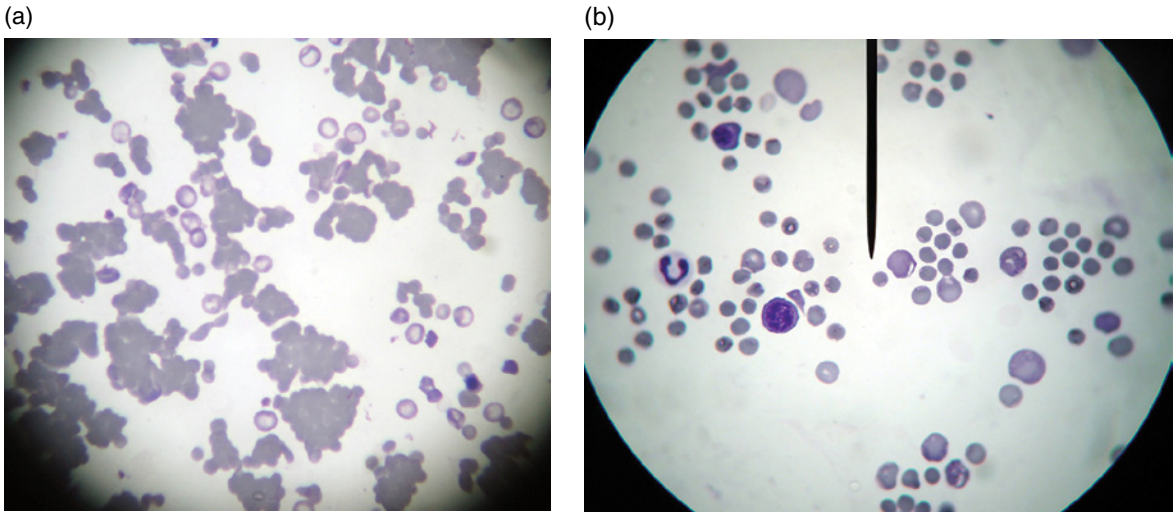

Figure 12.4 (a) Blood film of an IMHA patient exhibiting agglutination, NRBC, and polychromasia. (b) Blood film of an IMHA patient exhibiting spherocytes (at the end of pointer), NRBC, and polychromasia. (Images courtesy Bel-Rea Institute of Animal Technology)

Table 12.2 IMHA laboratory work

Morphology changes on blood film	Spherocytes, agglutination Possible Heinz bodies, NRBC, polychromasia, anisocytosis, ghost cells
Blood cell count changes	Leukocytosis with left-shift and toxic segs Possible thrombocytopenia
PCV/TP	Decreased PCV
Blood chemistry	Hyperbilirubinemia Hepatic dysfunction due to hypoxia, inflammation, or necrosis
Coagulation profile	Possible coagulation disorders
Urine changes	Hyperbilirubinuria

seen with IMHA. Blood chemistry analysis may show organ dysfunction due to hypoxia, necrosis, or inflammation (Figure 12.4; Table 12.2).

- A leukemoid reaction can be seen with IMHA, where WBC production is stimulated, causing a dramatic leukocytosis.
- Abdominal and thoracic radiographs can be used to evaluate abnormalities in internal organs including hepatomegaly, splenomegaly, and signs of thromboemboli as well as being used to identify underlying causes.
- A definitive diagnosis of IMHA can be obtained with a positive Coombs test, which will detect antibodies or complement (proteins) on RBC surface. Flow cytometry is another option to detect antibodies bound to the RBC.
- Some cases are diagnosed by ruling out other causes of anemia.
- Reticulocyte count and RBC indices will indicate whether the anemia is regenerative or not.

Treatment

- Treatment is aimed at stopping the RBC destruction, treating hypoxia, and stopping thromboemboli.
- Blood transfusions will replace lost RBCs and aid in carrying oxygen to hypoxic tissues, yet this is a temporary solution as long as the immune system is still attacking RBCs.

TECH BOX 12.2: It is essential to control the destruction of RBCs if blood transfusions are to be successful in treating IMHA.

- Immunosuppressive drugs are necessary to stop the destruction of RBCs. Commonly used drugs include corticosteroids, azathioprine, and cyclosporine.
- Pulmonary thromboemboli will require blood thinners and anticoagulant therapy.
- A splenectomy can be performed but only in a worst-case scenario. Removing the spleen will take away the source of the macrophages responsible for destroying the RBCs.

Absolute Erythrocytosis or Polycythemia

Description

Polycythemia, a condition of elevated RBC numbers, is the opposite of anemia. This disorder occurs in both dogs and cats and can be a primary or secondary condition. Primary polycythemia, called polycythemia vera, is caused by a myeloproliferation of RBCs with no known cause. With polycythemia vera, RBC production is increased despite low to low-normal levels of EPO. Secondary polycythemia develops due to increased levels of EPO associated with systemic hypoxia, EPO-secreting tumors, or other hormones that stimulate erythropoiesis. Hyperviscosity of the blood occurs with the increased RBC number and will decrease circulation and can lead to heart failure and death.

Clinical Signs

- Clinical signs include erythema of mucous membranes and skin, PU/PD, dilated blood vessels (especially noted in the retinal vessels), weakness, lethargy, and anorexia.
- Bleeding disorders occur commonly with polycythemia due to hyperviscosity of the blood. Clinical signs include epistaxis, hematuria, hemoglobinuria, hematemesis, bloody stool, or hemorrhage.
- CNS signs can be seen due to hyperviscosity of the blood as well. Seizures, ataxia, weakness, and blindness are possible.

TECH BOX 12.3: Hyperviscosity syndrome is a life-threatening condition seen with polycythemia, causing many complications.

Diagnosis

- Presenting clinical signs, physical exam including ophthalmic exam, and history can aid in a diagnosis.
- Erythrocytosis can be relative due to hemoconcentration; this relative increase needs to be ruled out as the cause of polycythemia.
- Laboratory tests include CBC and PCV, which will reveal an increased RBC count PCV (65–75%).
- Serum EPO levels can be used to differentiate between primary and secondary polycythemia.

Treatment

- Treatment is aimed at reducing RBC numbers and decreasing viscosity of the blood. Phlebotomy is used to remove RBCs from circulation and drug therapy (hydroxyurea) to slow erythropoiesis. Fluids should be given with phlebotomy to replace fluid volume.
- Identification of underlying cause in secondary cases is necessary for determining the most appropriate treatment protocols.

Client Education and Technician Tips

- Patients will require monitoring with frequent CBCs to evaluate RBC number and PCV. Drug therapies are myelosuppressive and it is necessary to make sure the RBC values are not declining into an anemic state.

Leukocyte and Lymph Disorders

Malignant Lymphoma or Lymphosarcoma (LSA)

Description

LSA is a progressive fatal disorder characterized by unregulated growth of malignant lymphocytes. The tumors tend to form in lymphoid tissues including thymus, lymph nodes, spleen, and bone marrow but can also occur in skin, liver, gastrointestinal tract, eye, CNS, and bone. The disorder is seen in both dogs and cats and is the most common hemopoietic neoplasm in dogs. There are four recognized forms of LSA based on location in dogs: multicentric (most common), alimentary, medistinal, and extranodal. Cats tend to have more atypical locations, with young cats commonly showing masses in their chest and older cats in the abdomen.

TECH BOX 12.4: Lymphosarcoma is a fatal disorder. Although it responds well to chemotherapy, prognosis for long-term survival is poor.

Clinical Signs

- Common clinical presentation of LSA includes generalized lymphadenopathy that is rapid and non-painful, anorexia, lethargy, fever, and dehydration. Cats do not normally present with lymphadenomegaly.
- The alimentary form may present similar to a gastrointestinal obstruction with V/D, weight loss as a result of malabsorption or maldigestion, abdominal pain, and constipation.
- The mediastinal form will present like a respiratory disorder with dyspnea.
- With extranodal form's clinical signs are associated with the specific site. Small raised ulcerations and scaly lesions occur with cutaneous LSA, pathological fractures with bone involvement, kidney lesions will cause renal failure, and blindness is associated with lesions in the eye.

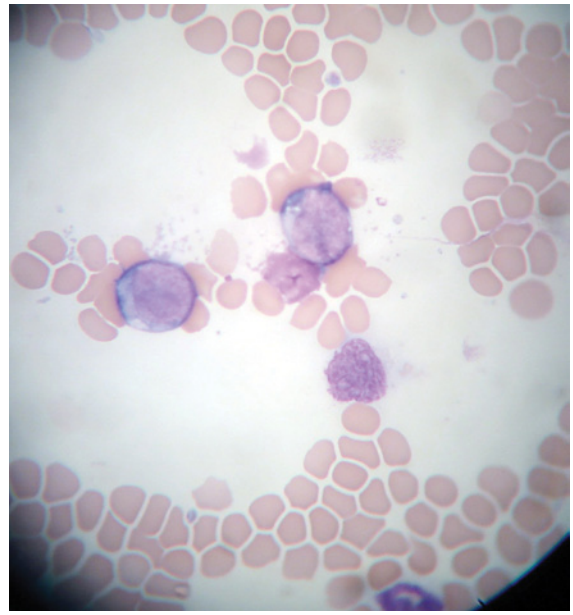

Figure 12.5 Malignant lymphocytes from a canine patient with lymphosarcoma (Diff Quik). (Image courtesy Bel-Rea Institute of Animal Technology)

Diagnosis

- Presenting clinical signs, physical exam, and history can aid in determining a diagnosis.
- Diagnosis is straightforward with cytology/histopathology from FNA or biopsy revealing malignant lymphocytes (Figure 12.5) found in lymph nodes and other tissues throughout the body.
- PCR can be performed if cytology/histopathology fails to confirm a diagnosis. PCR evaluation can determine if lymphocytes are malignant.
- Diagnostic imaging techniques can be used to determine body systems involved.

Treatment

- LSA is treated with chemotherapy, with the multicentric form responding to treatment better than other forms of LSA.
- Analgesic drugs can be used to keep the animal comfortable.

Client Education and Technician Tips

- LSA has one of the best response rates to chemotherapy in veterinary medicine, yet survival times average less than 1 year.
- Prognosis is better for dogs than cats. Many cats will go into remission, but despite this the survival time averages 6 months.

Multiple Myeloma (Plasma Cell Tumor)

Description

Plasma cell tumors arise from plasma cells (a type of WBC) in the bone marrow becoming malignant and multiplying abnormally. Plasma cells secrete immunoglobulins (proteins) that give rise to gammopathies, increased immunoglobulin in serum. Multiple myelomas are the most common type of plasma cell tumor, seen in older dogs and less frequently in cats. Plasma cell tumors are more common in flat bones and may be linked to genetics, exposure to carcinogens, or viral infections.

Clinical Signs

- Multiple myelomas cause bone damage, including pathological fractures, bone lysis, and osteoporosis. These animals present with lameness, spinal disorders, and pain.
- The gammopathies cause a hyperproteinemia resulting in hyperviscosity syndrome or a thickening of the blood. Bleeding occurs as a result of the highly viscous blood interfering with coagulation pathways.

TECH BOX 12.5: Multiple myelomas will cause hyperviscosity syndrome due to the increased proteins in the blood, which will cause potentially life-threatening complications.

- Renal disease occurs as a result of protein accumulation in the kidney and amyloidosis, which lead to hypoperfusion and hypercalcemia.
- Severe cases involve seizures, coma, ophthalmic abnormalties (as a result of ocular bleeding), and immunodeficiency.

Diagnosis

- Presenting clinical signs, physical exam, and history will aid in diagnosis, but laboratory diagnostic tests will be required, as clinical signs can be vague.
- Bone marrow aspiration will show malignant plasma cells that are large, round, and mitotic. Plasma cell numbers will also be increased (normal 5%).
- Diagnostic imaging techniques including radiographs and MRI bone scans will show bony lesions of lysis and osteoporosis and pathological fractures. Biopsy of these bone lytic regions will aid in further determining a plasma cell tumor as the culprit.
- Laboratory tests including CBC, PCV, and reticulocyte count will reveal non-regenerative cell deficiencies and hyperviscosity of the blood. Coagulation profiles will further evaluate coagulation disorders and blood chemistry profile will identify renal perfusion dysfunction and failure (Table 12.3).

Table 12.3 Multiple myeloma laboratory work

Blood cell count changes	Non-regenerative anemia Thrombocytopenia Leukopenia
PCV/TP	Decreased PCV Hyperproteinemia
Blood chemistry	Hypercalcemia Renal dysfunction
Coagulation profile	Coagulation dysfunction
Urine changes	Hyperproteinuria

Treatment

- Chemotherapy is the treatment of choice, aiding in bone healing and reduction of immunoglobulins.
- Surgical removal is generally not done due to the complicated nature of these tumors.
- Drug therapies will include analgesics, antibiotics, and corticosteroids.
- It is important to treat secondary complications.
- Fluid diuresis will help reduce viscosity and treat hypercalcemia. Treatment for hyperviscosity syndrome also includes plasmapheresis. This process involves removing the blood plasma and cleaning the plasma of immunoglobulins, with the plasma then being returned to circulation.

Client Education and Technician Tips

- Multiple myelomas are very complex tumors, making long-term survival generally poor.

Chylothorax

Description

Chylothorax is a condition characterized by accumulation of chyle, a lymphatic fluid, in the thorax. Chyle is a milky-looking fluid filled with small fat molecules and other nutrients destined for the cranial vena cava from the intestinal lymphatic system. This condition seen in dogs and more often in cats occurs as a result of a rupture or abnormality of thoracic (lymph) duct (TD). The cause may be due to systemic disease or trauma, but many cases are idiopathic, with the cause not commonly found. The fluid accumulation in the thoracic cavity prevents lungs from fully expanding, causing dyspnea. Chyle is an irritant that will lead to inflammation. Chyle is also the main carrier of WBCs, proteins, and vitamins in the body. A leakage will lead to weakness, metabolic disorders, and immune system compromise.

TECH BOX 12.6: Chyle is identified upon thoracocentesis as a white, milky fluid, filled with fat, WBCs, and nutrients.

Clinical Signs

- Clinical signs manifest as a result of fluid accumulation in the thoracic cavity and include respiratory distress, dyspnea, shallow breathing, cyanosis, weakness, cough, and anorexia.

Diagnosis

- Presenting clinical signs and physical exam with auscultation of the heart and lungs will reveal muffled heart sounds due to the fluid accumulation in the thorax.
- Radiographs will be used to confirm the presence of fluid in the thoracic cavity.
- A thoracocentesis will identify chyle, a milky-looking fluid containing fat molecules, in the chest. Laboratory evaluation can confirm presence of triglycerides in the fluid. The triglyceride level of chyle should be higher than the triglyceride level in the blood.
- Ultrasound or CT scans may aid in determining the primary cause of TD rupture.

Treatment

- Thoracocentesis can be performed to remove fluid from the thoracic cavity to allow for more comfortable breathing.
- A low-fat diet will reduce the amount of chyle produced by the body. There are also nutraceuticals that may provide benefit in controlling protein levels in chyle.
- Surgery used to prevent leakage from the TD or to remove the abdominal reservoir for chyle are also treatment options.

Client Education and Technician Tips

- Treatment is not always successful, especially if the underlying cause is not determined or treatable.

Thrombocyte and Coagulation Disorders

Primary Immune-Mediated Thrombocytopenia (PIMT) or Idiopathic Thrombocytopenia

Description

PIMT is a disorder involving immune-mediated destruction of circulating platelets, or less likely megakaryocytes platelet precursors. This life-threatening condition causes bleeding that leads to anemia. The disorder is seen in dogs and less frequently in cats. Female dogs are at higher risk, although there is no sex predisposition in cats. German Shepherds, Old English Sheepdogs, Poodles, and Cocker Spaniels are at highest risk of this disorder, although a genetic link has not been proven.

TECH BOX 12.7: PIMT is an autoimmune condition where the immune system destroys circulating platelets and possible platelet precursors in the bone marrow.

Clinical Signs

- Clinical signs are related to bleeding and include petechiation, bleeding gingiva, epistaxis, melena, hematemesis, hematuria, and hemoptysis.
- Dyspnea, anorexia, and lethargy may also be seen with PIMT.

Diagnosis

- Presenting clinical signs, physical exam, and history can aid in pointing to a bleeding disorder.
- Low platelet concentrations are determined by looking at a quantitative platelet count, usually less than 10,000–50,000 platelets/microliter (normal is 200,000–500,000 platelets/microliter). Platelets can also be estimated on a blood film evaluation, but this is less accurate than the quantitative counts.
- Bleeding will lead to anemia that can be measured with a CBC, RBC count, and PCV.
- Bone marrow aspirates can be used to evaluate if megakaryocytes are the target of the destructive antibodies.
- ELISA or IFA can detect antibodies attached to the platelets or megakaryocytes. The IFA test is not the most reliable. A negative ELISA result can rule the disorder out, but a positive result cannot differentiate between primary and secondary platelet disorders.
- Ruling out other causes of platelet deficiencies can help in making a presumptive diagnosis.

Treatment

- Treatments include activity restriction and corticosteroids or other immunosuppressants to suppress the immune destruction.
- Blood transfusion should be used to correct anemia but is not generally very helpful, as circulating platelets will be destroyed within a couple of hours.

Client Education and Technician Tips

- Relapses are common and patients need frequent monitoring of platelet and RBC values.
- Although a genetic link has not been proven, animals diagnosed with PIMT should not be bred.

Hemophilia

Description

Hemophilia is a bleeding disorder caused as a result of protein clotting factor deficiencies involved with the intrinsic coagulation pathways. There are two types, based on the particular factor, of hemophilias seen in dogs and cats. Hemophilia A, a factor VIII deficiency, is the

most common inherited bleeding disorder seen in both dogs and cats. Hemophilia B is a factor IX deficiency and is not as common as type A. Hemophilias are X-linked chromosome disorders where females are carriers but the males are affected. Both types have the same clinical presentation, testing, and treatment.

TECH BOX 12.8: Hemophilia A is the most common inherited bleeding disorder in dogs and cats, although hemophilia B is relatively rare in these species. Unfortunately neither is curable.

Clinical Signs

- Clinical signs associated with hemophilia include increased bleeding times with procedures, tooth eruption, or umbilical vessels at birth.
- Also seen with hemophilia are lameness as a result of hemarthrosis, hematoma formation, and hemorrhagic body cavity effusions.
- Animals with some amounts of factor VIII or IX will not have severe clinical signs but will have prolonged bleeding with surgery or trauma.
- Animals with little to no factors VIII or IX will spontaneously bleed.
- Cats will rarely show evidence of spontaneous hemorrhage despite the concentration of clotting factors present.

Diagnosis

- Presenting clinical signs, physical exam, and history will aid in diagnosing a bleeding disorder but not specifically which one.
- Clotting factor concentrations in the blood can be measured. Patients with hemophilia will have deficient concentrations of either factor VIII or IX.
- Animals with hemophilia will have prolonged activated partial thromboplastin time (APTT) and activated clotting time (ACT), detecting problems with the intrinsic and common coagulation pathways, but will have normal von Willebrand factor (vWF).

Treatment

- This condition cannot be cured. The only treatment that can be offered to these patients is transfusions of cryoprecipitate, plasma, or whole blood during a bleeding event. Whole blood should be avoided due to blood type reactions, unless anemia results from the bleeding.
- Transfusions should also be considered prophylactically when surgery or procedures are necessary that may cause bleeding.

Client Education and Technician Tips

- This is an inherited disease and animals diagnosed with hemophilias or those determined to be carriers should not be bred.

von Willebrand's Disease

Description

von Willebrand's disease is a congenital inherited insufficiency of vWF, a crucial protein involved in primary hemostasis. This protein factor mediates platelet adhesion to the endothelium beginning the clot formation. von Willebrand's disease is the most common inherited bleeding disorder in dogs and is rarely seen in cats. This condition is divided into three categories. Type I is the most common with mild to moderate clinical signs and low vWF concentrations. Type II shows moderate to severe clinical signs and low vWF concentrations. Type III shows severe clinical signs as a result of a complete absence of vWF. Several breeds are commonly diagnosed with von Willebrand's disease, including Doberman Pinschers, Pembroke Welsh Corgis, Airedales, Shetland Sheepdogs, and Scottish Terriers.

TECH BOX 12.9: von Willebrand's disease is a common inherited bleeding disorder in dogs, yet very rare in cats. There are varying degrees of severity based on how deficient the vWF is.

Clinical Signs

- Clinical signs are associated with excessive bleeding including gingival bleeding, epistaxis, hematuria, blood in feces, bruising, and anemia.
- Excessive bleeding is noted after medical procedures or blood collection.

Diagnosis

- Presenting clinical signs, physical exam, and history will aid in diagnosing a bleeding disorder but not specifically which one.
- Coagulation tests reveal normal APTT, prothrombin time (PT), and ACT. Some patients will experience a deficiency in factor VIII; if this is the case APTT and ACT may be prolonged.
- Patients will also have normal platelet counts.
- Prolonged buccal mucosal bleeding time (BMBT) is also associated with lack of vWF.
- There is a diagnostic test for von Willebrand's disease that will test concentrations of vWF.

Treatment

- There is no cure for this condition, making treatment aimed at controlling bleeding events.
- Transfusions of whole blood, cryoprecipitate, or plasma are necessary during bleeding events or prior to surgery.
- Desmopressin therapy is used to stimulate release of vWF from stores in endothelial cells. This therapy is not a long-term solution and does not work in patients with type III, but it is helpful prior to surgery or medical procedures that will cause bleeding.

Client Education and Technician Tips

- Patient's activities need to be restricted to avoid bleeding events.

Disseminated Intravascular Coagulopathy

Description

DIC is an acute, life-threatening, uncontrolled inflammatory response characterized by inappropriate coagulation and fibrinolysis, which are initiated in the patient's blood vessels. This will lead to thrombosis and hemorrhage throughout the body, as well as organ failure. DIC is always a secondary disorder that is triggered by exposure to or release of tissue factor that occurs as a result of numerous underlying causes including electrical shock, heat stroke, sepsis, intravascular hemolysis, neoplasia, burns, and pancreatitis. DIC is seen in both dogs and cats.

TECH BOX 12.10: DIC carries a very poor prognosis, with very few patients surviving the event.

Clinical Signs

- DIC is often subclinical.
- Patients with DIC will exhibit signs of hemostatic defects. Dogs are more likely to hemorrhage, while cats will exhibit more diffuse thrombosis.
- Because this is a secondary condition, DIC will only be seen with underlying disease.

Diagnosis

- There is not one test that can be run to evaluate DIC in patients. The general rule is that patients with four or more abnormal test results have DIC. Coagulation profiles, CBC, blood chemistry profile, and urinalysis are

Table 12.4 DIC laboratory work

Morphology on blood film	Schistocytes
Blood cell count changes	Hemolytic anemia Thrombocytopenia Left-shift neutrophilia
Electrolyte profile	Metabolic acidosis
Blood chemistry	Azotemia Renal dysfunction Hepatic dysfunction Hemoglobinemia Hyperbilirubinemia
Coagulation profile	Increased APTT, PT, fibrinogen
Urine changes	Hemoglobinuria Bilirubinuria

common laboratory tests performed on these patients (Table 12.4).
- Patients must show underlying disease and evidence of thrombosis or hemorrhage and have lab values consistent with DIC.
- DIC can be diagnosed on necropsy.

Treatment

- DIC is a very rapidly progressing condition that requires immediate aggressive treatment or euthanasia.
- Treatment measures include heparin or other blood thinners, blood or plasma transfusions, antibiotic therapy, fluid therapy to profuse tissues, oxygen therapy if required, and correction of electrolyte imbalances.
- Because DIC is a secondary condition, the primary cause must be identified and corrected.

Client Education and Technician Tips

- Prognosis is very poor/grave because the condition progresses so quickly. Unfortunately, very few patients survive.

Rodenticide Toxicity

Description

Rodenticide toxicity is the most common poisoning seen in dogs and cats and results from an ingestion of rodenticides, which cause coagulopathy as a result of vitamin K dependent factors. These poisons inhibit the recycling of vitamin K, which is a necessary cofactor in activating clotting factors II, VII, IX, and X. Without these factors animals cannot convert prothrombin to thrombin. These poisons are designed to taste good, making them attract animals. Dogs and cats either directly ingest the poison or indirectly by ingesting another dead animal with the toxin in its system.

TECH BOX 12.11: Rodenticide toxicity is the most common poisoning seen in dogs and cats, due to ingestion of the poison or an animal carcass. These substances should be avoided in areas with dogs and cats.

Clinical Signs

- Clinical signs appear 3–5 days post-ingestion, as the active clotting factors are depleted and not replaced.
- These clinical signs vary but are all due to coagulopathy and include hematomas especially at pressure points, deep and superficial bruising, petechiation, ecchymosis, hemorrhage, anemia, melena, hyphema, dyspnea because of hemothorax, hemoabdomen, epistaxis, hematuria, hemoptysis, blood in vomit or diarrhea, lameness as a result of hemarthrosis, and ataxia or seizures associated with bleeding in the brain.
- Also seen are anorexia and lethargy.

Diagnosis

- Presenting clinical signs, physical exam, and history will aid in a diagnosis but are not definitive except in the cases of know ingestion.

- APTT, ACT, PT times are increased, with the PT being the first value to test abnormal.
- Proteins induced by vitamin K1 absence or antagonism (PIVKA) is the most specific test available for diagnosis. A three-fold increase in PT or PIVKA is highly suggestive of rodenticide poisoning.
- Animals with rodenticide toxicity will often have regenerative anemia.
- Radiographs can be used to evaluate bleeding.
- Necropsy tissue evaluation can reveal rodenticides in tissues.
- Positive response to vitamin K therapy suggestive of this toxicosis.

Treatment

- If the ingestion is recent, emetics, activated charcoal or other absorbents, and a cathartic will be the treatment of choice.
- All patients suspected of rodenticide ingestion will receive vitamin K therapy through injectable or oral route. The length of treatment is dependent on type of rodenticide ingested.
- Fresh frozen plasma or whole blood transfusions are necessary in cases of bleeding events.
- Fluids, oxygen therapy, and other supportive measures may be necessary based on severity of clinical signs.

Client Education and Technician Tips

- Prognosis depends on type of rodenticide ingested and at what point treatment is initiated. Animals with underlying disease, neonates, and geriatrics are more at risk of death.
- Vitamin K absorption is aided by feeding with a fatty food, as it increases bioavailability. Vitamin K should be given with a high-fat canned food.
- Coagulation profile values need to be monitored through therapy and for several weeks post-treatment.
- Prevention includes keeping animals from roaming and not using rodenticides in the house or on property with dogs and cats present.

Feline Aortic Thromboembolism (FATE) or Feline Saddle Thrombus

Description

FATE is a life-threatening, painful, strictly feline condition where a thromboemboli is lodged at the saddle, the area where the aorta branches at the pelvis into right and left iliac arteries. This emboli originates from the left atrium of the heart and once lodged in the saddle region obstructs blood flow to the rear legs. This causes an inflammatory cascade and metabolic abnormalities, which can lead to circulatory shock. This will cause tissue, blood vessel, and nerve damage. Most cats with a saddle thrombus have some sort of heart disease causing turbulent blood flow, which can result in clots. Hypertrophic cardiomyopathy is a common cause, which is secondary to hyperthyroidism. In other patients neoplasia may be an underlying cause.

TECH BOX 12.12: A saddle thrombus in a cat is an extremely painful condition, requiring analgesics as a necessary part of the treatment protocol.

Clinical Signs

- Most cats will have both rear legs affected, but some may have just one. Legs will become hard, cold, and paralyzed, with pads turning blue.
- This is an extremely painful condition resulting in vocalization, panting, and tachypnea.
- Severe cases will lead to systemic shock.

Diagnosis

- Presenting clinical signs, physical exam, and history will aid in determining a diagnosis.
- Thoracic radiographs will reveal fluid around lungs, and an enlarged heart; signs of congestive heart failure (CHF).
- ECG or ultrasound can be used to evaluate the heart; US is more definitive.
- CT scans will allow for blood vessel evaluation and visualization of the thromboemboli.
- Blood chemistry profile will be used to evaluate organ function.

Treatment

- Some owners will opt to euthanize due to prognosis and discomfort of the cat.
- Analgesics are a necessity to make the cat more comfortable.
- Correction of circulatory shock, including IV fluids, needs to take place immediately if the cat is to survive.
- Blood thinners will be used to dissolve clot, although the disorder will recur if the underlying heart disease is not controlled.
- Surgery to remove clot can be performed only if it is caught immediately, there are no other clots in the heart, and patient is not in CHF.
- Limb amputation may be necessary due to compromised tissues as a result of blood flow disruption.

Client Education and Technician Tips

- Body temperature is a good indicator of prognosis. If the rectal temperature is above 98.9°F, the cat will have a much better prognosis than those below 98.9°F.
- If heart disease is not controlled, recurrence is common.
- Vigilant nursing care of the cat is required until the cat is able to walk again.
- Cats will require anti-clot therapy long term.

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Diseases of Rabbits, Guinea Pigs, and Chinchillas

chapter 13

Chinchillas, rabbits, and guinea pigs are popular pets and will therefore be seen frequently in veterinary clinics. Guinea pigs and chinchillas are very closely related, and although rabbits are not as closely related, there are many similarities between species. Common problems in the three species include gastrointestinal diseases, urinary tract disease, foot problems, oral malocclusions, hyperthermia, and infectious agents.

Urolithiasis/Bladder Sludge

Description

A common condition in guinea pigs and rabbits is the formation of urinary calculi that can result in stones and "bladder sludge." This is also known as hypercalcinuria or hypercalciuria. Bladder sludge is more common in rabbits and is composed of calcium salts and crystals that have not formed into stones. The consistency is that of chalk or fine sand (Figure 13.1). In both guinea pigs and rabbits the composition of uroliths is usually calcium phosphate, calcium carbonate, and triple phosphate precipitating from the urine. Guinea pigs also form oxalate stones. Normal urine pH of rabbits and guinea pigs is 8.0–8.5, and crystals and stones tend to form when the pH increases. Other factors include nutritional imbalance, especially increased calcium, genetics, decreased water intake, and metabolic disorders.

Clinical Signs

- Clinical signs associated with uroliths include hematuria, blood spots in the cage, dysuria, anuria, pollakiuria, abdominal pain, lethargy, and anorexia.
- Rabbits with bladder sludge tend to spray urine or inappropriately urinate. Thick urine sticking to the perineal area of the rabbit is also common.
- With guinea pigs signs are harder to pinpoint, as guinea pigs are very stoic and the signs are more subtle.

Small Animal Pathology for Veterinary Technicians, First Edition. Amy Johnson.

Companion website: www.wiley.com/go/johnsonvettechpath

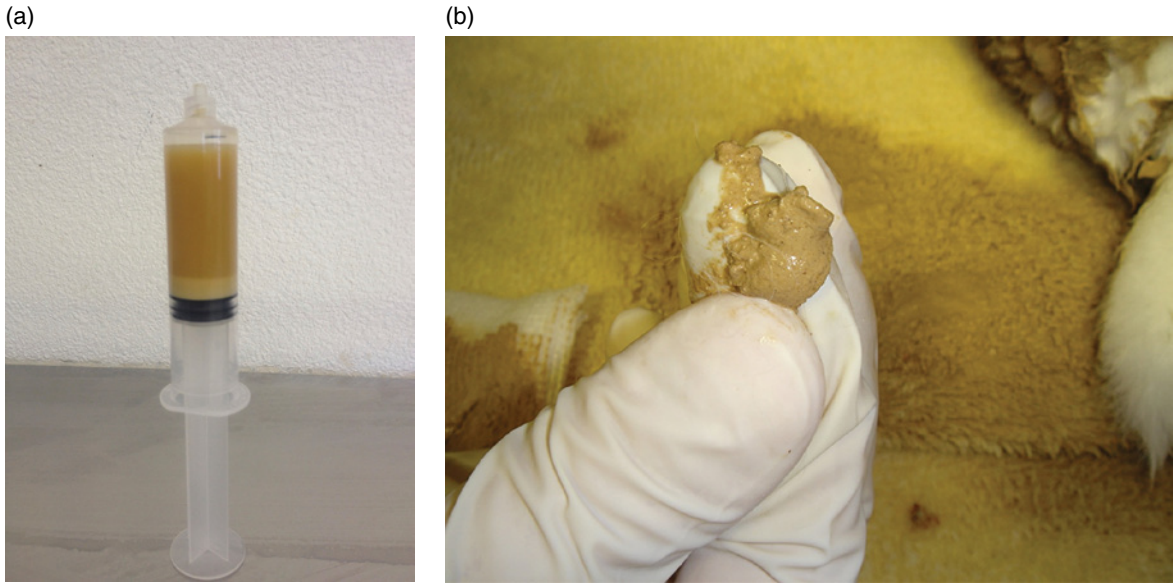


Figure 13.1 (a) Rabbit urine. (Image courtesy Amy Johnson and Bel-Rea Institute of Animal Technology) (b) Thick bladder sludge from a rabbit. (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

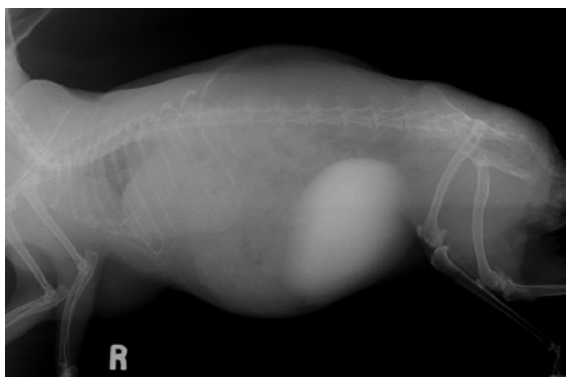


Figure 13.2 Radiograph showing bladder sludge in a rabbit prior to flushing. (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

Diagnosis

- The patient's history and physical exam will point to a presumptive diagnosis of uroliths.
- Urinalysis will show hematuria and possibly crystals in the urine.
- Radiographs or ultrasound may reveal the stones or sludge in the bladder (Figure 13.2).
- An intravenous pyelogram (IVP) will be used in cases of stones in the kidney.

Treatment

- Medical treatment that attempts to dissolve stones in guinea pigs and rabbits has been met with very little success.
- Stones may need to be surgically removed with a cystotomy (Figure 13.3).
- Bladder sludge will require catheterization and flushing. It may be productive to agitate the bladder first, allowing a mixing of the sand-like material (Figure 13.4).
- Diuresis will flush more fluids through the bladder, aiding in flushing any material out.
- Reduction of calcium in the diet is an important step in treatment and prevention.
- Urinary acidifiers are a controversial part of treatment. Caution must be used not to acidify the urine too much.

Client Education and Technician Tips

- Exercising of rabbits will mix the sludge in bladder and make the rabbit more likely to excrete the precipitate.
- Alfalfa hay should be avoided in adult rabbits and guinea pigs because of increased calcium content.

TECH BOX 13.1: Alfalfa pellets, hay, and treats should be avoided with adult rabbits and guinea pigs due to the calcium content.

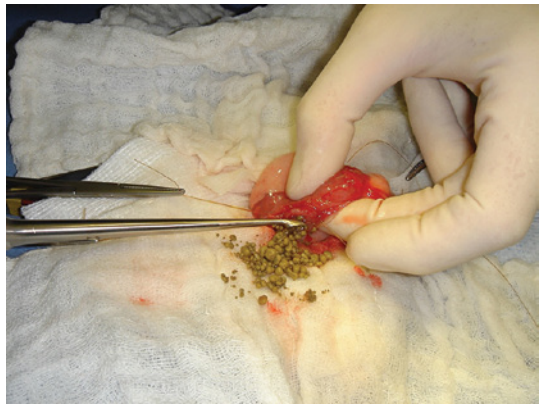


Figure 13.3 Rabbit cystotomy with stone removal. (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

- Increasing water consumption is an important step in increasing urination frequency.

Gastric Stasis

Description

Gastric stasis is a condition seen in rabbits, guinea pigs, and chinchillas and characterized by loss of gastric and intestinal motility. Predisposing factors include stress, pain, and diets high in pellets (carbohydrates) and low in fiber (hay). Gastric stasis can be fatal if not treated promptly.

TECH BOX 13.2: Gastric stasis can be a fatal condition in herbivores if not identified and treated promptly.

Clinical Signs

- Gastric stasis is commonly accompanied by a history of anorexia.
- Clinical signs include a decreased fecal production, hard dry feces, dehydration from diminished water intake, abdominal discomfort, lethargy, and decreased or absent gut sounds upon auscultation of abdomen.

(a) (b)

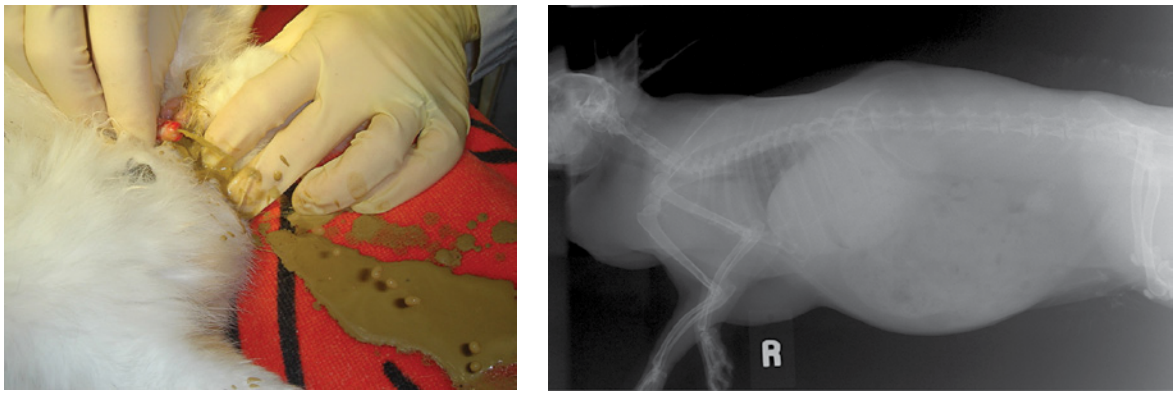


Figure 13.4 (a) In the process of flushing a rabbit urinary bladder. (b) Radiograph of rabbit urinary bladder post-flushing. (Images courtesy Dan Johnson, DVM, www.avianandexotic.com)

Diagnosis

- Physical exam, history, and clinical signs aid in diagnosis.
- Auscultation of abdomen reveals decreased or absent gut sounds, or in cases of gas accumulation loud gurgling.
- Radiographs may show gas, an empty bowel, or underlying conditions.

Treatment

- Treatment includes pain management with the use of analgesics and stress reductions.
- Fluids to correct dehydration and help to hydrate gastrointestinal contents.
- Force-feeding and gastric motility modifying drugs are important to start the gastrointestinal tract moving again.

Technician Duty Box 13.1

Patients with GI stasis will require force-feeding and fluid therapy for recovery.

- It is important to identify and correct the underlying cause.

Client Education and Technician Tips

- This condition can be seen post-operatively, making it important to keep the animal eating and to control any pain.

Ulcerative Pododermatitis, Bumblefoot, or Sore Hock

Description

Ulcerative pododermatitis is a condition seen in guinea pigs and rabbits where chronic inflammation and ulceration of the plantar surface of the foot is left open to secondary bacterial infections. Although there are several bacterial organisms implicated in the condition, *Staphylococcus aureus* is the most common. Predisposing factors include obesity, poor sanitation, and incorrect flooring or bedding that creates pressure sores. With rabbits stress, nerves, foot "stomping" trauma, paralysis from broken backs, and genetic predisposition may play a role.

Clinical Signs

- Patients with ulcerative pododermatitis will have red, inflamed, sore paws. Hyperkeratinazation, abscesses, and ulcerations may be also be seen (Figure 13.5).
- Pain may make the animal reluctant to move.
- Rabbits will sit in a position to take the weight off the back of the plantar surface and may look like they are "tiptoeing" when they walk.

Diagnosis

- Diagnosis is simply maintained with a physical exam and clinical signs.
- Culturing of wounds will determine the bacterial agent causing the infection.
- In severe cases radiographs may be necessary to determine if there is bone involvement.

Treatment

- An important aspect of treatment is to switch the animal to smooth flooring and comfortable bedding.
- The feet can be soaked in disinfecting solutions, with antibiotic ointment applied, and possibly bandaged.
- Analgesics are necessary to control pain.
- Some severe cases may require debriding of necrotic tissue and oral antibiotics.

(a) (b)

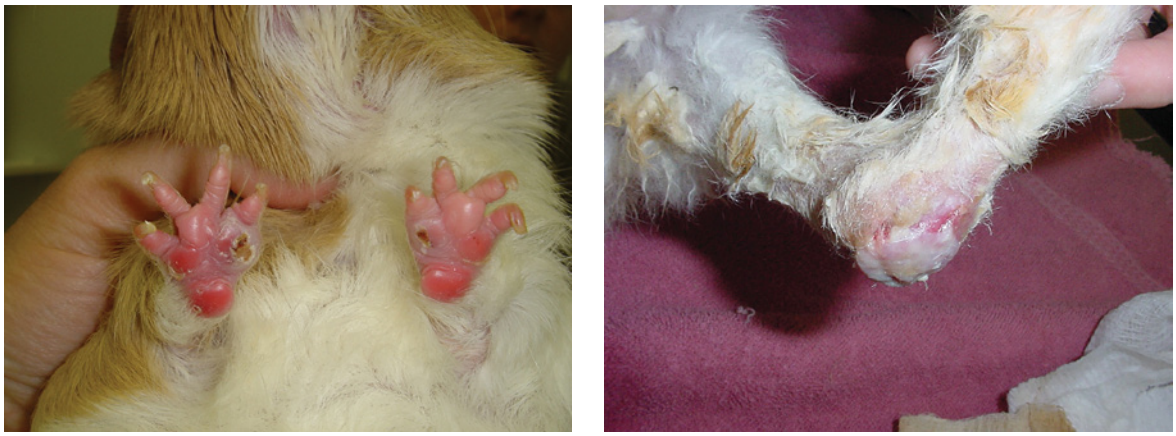


Figure 13.5 (a) Bumblefoot in a guinea pig. (b) Sore hock in a rabbit. (Images courtesy Dan Johnson, DVM, www.avianandexotic.com)

Client Education and Technician Tips

- Proper flooring and a dry surface are key to prevention of this condition.

TECH BOX 13.3: Ulcerative pododermatitis is commonly caused by improper flooring or animals sitting on surfaces covered in urine or feces.

Malocclusion or Slobbers

Description

Rodents and rabbits have open-rooted (continually growing) teeth. Malocclusions occur when the teeth grow too long and prevent the mouth from closing appropriately. This condition can impede eating and drinking, leading to dehydration and malnutrition. The main predisposing factors are thought to be genetics and nutritional deficiencies that cause jaw deformities. Other factors include inappropriate diet, broken jaws, and cage chewing. Rabbits and chinchillas tend to have problems with incisors and occasionally with cheek teeth, while guinea pigs have the most problems with their premolars and anterior molars.

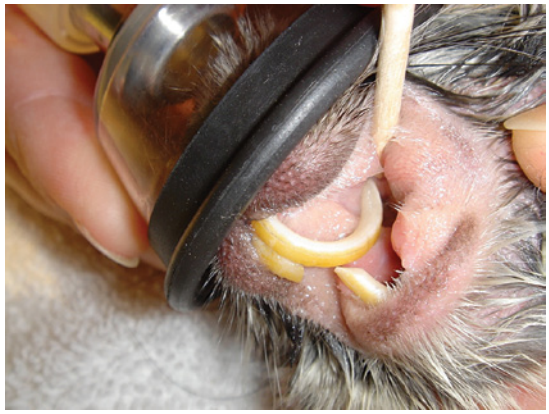
Clinical Signs

- Long teeth that keep the mouth from closing appropriately and cause hypersalivation and wet matted fur around the mouth and chin. This can lead to alopecia and inflammation in those areas (Figure 13.6).
- Other clinical signs include anorexia, weight loss, dehydration, oral bleeding, and an unkempt appearance.
- Problems with the cheek teeth will cause buccal and tongue lesions (Figure 13.7).
- Malnutrition and dehydration can lead to seizures, coma, and death.

Diagnosis

- Physical exam and clinical signs are enough for a diagnosis.
- Dental radiographs can show teeth, roots, and alignment to aid diagnosis and treatment.

(a)



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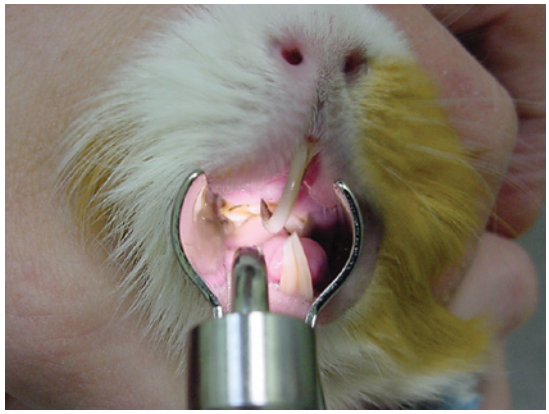


(c)

Figure 13.6 (a) Incisor malocclusion in a chinchilla. (b) Incisor malocclusion in a guinea pig. (c) Incisor malocclusion in a rabbit. (Images courtesy Dan Johnson, DVM, www.avianandexotic.com)

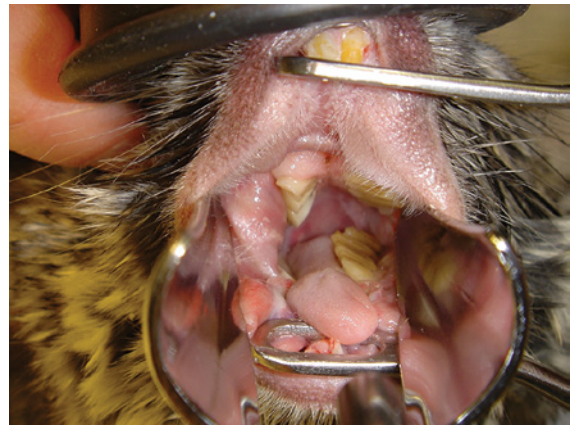


Figure 13.7 Cheek teeth malocclusion in a chinchilla. (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

Treatment

- The teeth will require trimming, but caution must be used not to cut into the pulp cavity.

Technician Duty Box 13.2

Patients suffering from a malocclusion will require their teeth to be trimmed or filed, allowing them to appropriately close their mouth and eat and drink.

- Change of diet to include a hard pellet to file teeth and adequate nutrition to avoid deficiencies.
- Provide objects to gnaw on to allow for teeth filing.

Client Education and Technician Tips

- There is a genetic link in all animals and those affected should not be bred.

Heat Stroke

Description

Heat stroke is seen in guinea pigs, rabbits, and chinchillas when their body temperatures increase into hyperthermic ranges. High environmental temperatures, high humidity, cage ventilation, and restraint are all contributing factors.

Clinical Signs

- Clinical signs include panting, hypersalivation, tachypnea, and increased body temperature.

Diagnosis

- A diagnosis is made based on clinical signs, physical exam, and an elevated body temperature.

Treatment

- The animal needs to be cooled with a cool water bath and IV fluids.
- Steroids are also a common part of treatment.

Client Education and Technician Tips

- Prognosis in these animals is generally poor.
- Caution should be used when restraining, choosing housing, and cage placement. Cages should not be placed in direct sunlight or under/over heat vents and should have adequate ventilation.
- Rabbits housed outdoors need shade, and on hot days a cool area should be provided.

TECH BOX 13.4: Animals housed outdoors are much more susceptible to hyperthermia than hypothermia.

Respiratory Infection

Description

Respiratory infections in these species are most often caused by bacterial agents, although viruses may be a cause as well. In rabbits *Pasteurella multocida* is the main implicated agent and referred to as "snuffles". In guinea pigs *Bordetella bronchiseptica* or *Streptococcus pneumoniae* are the main causes. In chinchillas *Pasteurella multocida*, *Streptococcus pneumoniae, Bordetella bronchiseptica*, and *Pseudomonas aeruginosa* are the main bacterial agents. Predisposing factors include crowding, poor ventilation, high humidity, and stress.

Transmission

- These infections are spread through direct contact, fomites, and aerosolization.

Clinical Signs

- Many animals will be asymptomatic and will hide their signs of illness until they are severely ill, resulting in death.
- Clinical signs include anorexia, weight loss, lethargy, nasal and ocular discharge (Figure 13.8), dyspnea, sneezing, rough hair coat, otitis, head tilt, pneumonia, and discharge found on the medial aspect of forelegs from grooming.
- Rabbits with *Pasteurella* may have abscesses throughout the respiratory tract, genital lesions, and septicemia.

Diagnosis

- Presenting clinical signs and physical exam will provide a diagnosis.
- Bacterial culture can determine the causative agent.
- In rabbits, if *Pasteurella* is suspected an ELISA or PCR assay can confirm the diagnosis.

(a) (b)

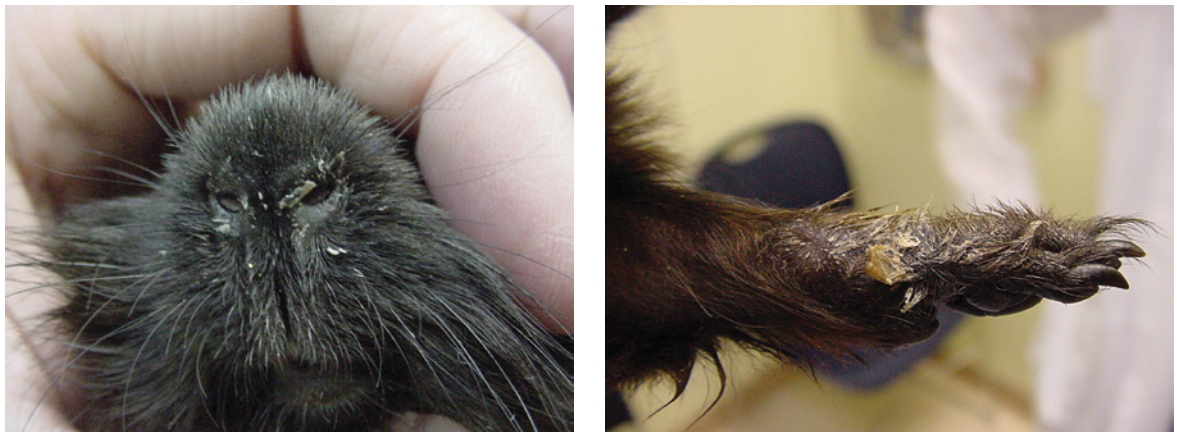


Figure 13.8 (a) Guinea pig with nasal exudate from respiratory infection. (b) Guinea pig with nasal exudate dried on feet as a result of grooming. (Images courtesy Dan Johnson, DVM, www.avianandexotic.com)

Treatment

- Mostly antibiotic treatment is used in conjunction with supportive care, including force-feeding and fluids.

Client Education and Technician Tips

- Many respiratory infections in these species are chronic and carry a poor prognosis.
- Humans may transmit the infections, especially in cases of *Streptococcus pneumoniae*.

Mastitis

Description

Mastitis is common in rabbits and guinea pigs and may be seen occasionally in chinchillas. The inflammation and infection associated with the mammary glands are attributed to *Staphylococcus aureus, Streptococcus* spp., *Klebsiella* spp., and *Pasteurella multocida*. In rabbits mastitis can be seen in lactating animals and those with pseudopregnancies. The introduction of bacteria can be caused by trauma from nesting boxes, teeth, or unsanitary housing conditions.

Clinical Signs

- Clinical signs include changes in mammary gland and teat color, hot inflamed mammary glands, hyperemia, and mucopurulent discharge from teat.
- A change in milk consistency or color may be noted, as well as blood in the milk.
- Neonates that were thriving and are now deteriorating can also be seen with mastitis.

Diagnosis

- Physical exam and presenting clinical signs will aid in diagnosis.
- Bacterial culture of discharge or milk will identify the bacterial organism associated with the mastitis.

Treatment

- Antibiotics will be prescribed along with hot packing of the glands.
- Lancing, flushing, and draining abscessed glands may be necessary.
- Analgesics can be prescribed to keep the animal comfortable.

- The neonates may have to be hand-reared during the treatment.

Client Education and Technician Tips

- Sanitation is an important key in preventing mastitis.

Rabbit Hairballs or Trichobezoar

Description

Trichobezoars are a problem in rabbits, as the rabbit grooms and ingests fur. Rabbits cannot vomit, making it essential for the fur to make its way through the gastrointestinal tract. This is normally not a problem if there is food in the rabbit's stomach to push the fur through. It is only in anorexic rabbits or those that ingest large amounts of fur from excessive self-grooming (from boredom) or hair chewing as a result of low-fiber diets that the inability to vomit becomes a problem.

TECH BOX 13.5: Hairballs are often a result of anorexia; if the stomach is empty gastrointestinal motility slows.

Clinical Signs

- Clinical signs include anorexia, lethargy, dehydration, and no fecal production. These rabbits are usually healthy otherwise.

Diagnosis

- Trichobezoars should be considered in all rabbits with a history of sudden anorexia and decreased or absent water consumption.
- With abdominal palpation a gastric mass may be able to be palpated.
- Radiographs or ultrasound will provide a visualization of the mass.

Treatment

- Medical management can be attempted using fluids, administration of mineral oil through a stomach tube, gentle massage of stomach, and gastric motility modifiers. Papain and bromelin are enzymes found in pineapple and papaya that can be administered to break down the hair in the stomach.
- Surgical removal of the mass may be necessary if medical treatment cannot force the fur through the gastrointestinal tract (Figure 13.9).

(a) (b)

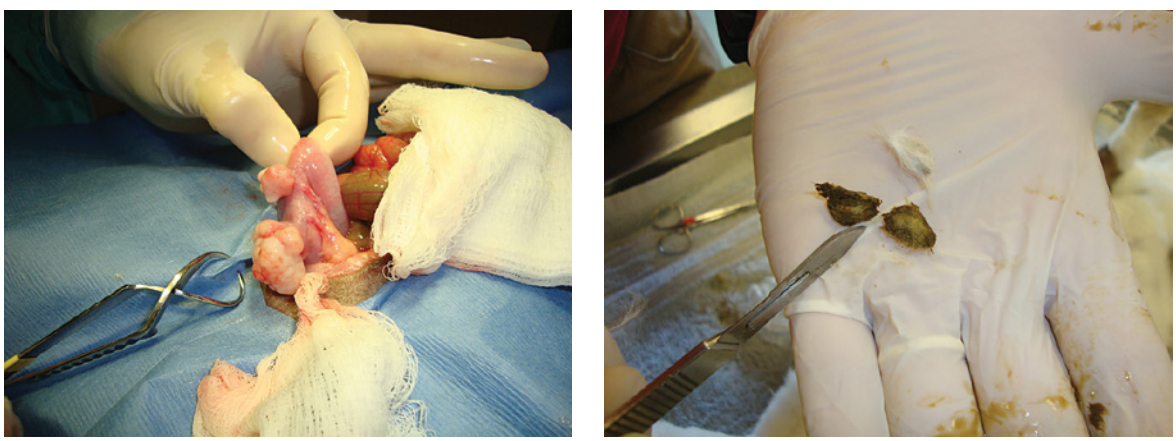


Figure 13.9 (a) Laparotomy on a rabbit removing a dried hair mat the rabbit ingested. (b) Hair mass removed from rabbit. (Images courtesy Dan Johnson, DVM, www.avianandexotic.com)

- The normal flora in the gut will be disrupted and must be restored. This can be accomplished by the use of probiotics or cecotropes from healthy rabbits.

Client Education and Technician Tips

- High-fiber diets and fruits with proteolytic enzymes, like pineapple and papaya, should be fed to prevent hairballs. Fresh fruits and vegetables contain fiber and should always be a part of the rabbit's diet.
- Rabbits should be brushed when shedding to avoid ingestion of excess fur.
- Stress and obesity are factors that can be prevented.

Rabbit Buphthalmia

Description

Buphthalmia is an inherited form of glaucoma that is common in rabbits. With this condition there is an abnormal production and removal of aqueous fluid in the eye. The condition can be unilateral or bilateral.

Clinical Signs

- Increased intraocular pressure and the increased size of the globe are common clinical signs of buphthalmia.
- Other clinical signs include opacity of the cornea, pain, corneal edema, and epiphora.
- As the vision is affected, a mild head tilt will be noted in the animals.

Diagnosis

- History and presenting clinical signs will aid in a presumptive diagnosis. Since this is a congenital condition, clinical signs usually occur early in life.
- IOP will be measured and increased in animals with buphthalmia.
- MRI or CT scan can better aid in visualization of the ocular structures.

Treatment

- Treatment is not necessary in all cases. Rabbits with IOP under a level where pain is associated may be fine with close monitoring.
- There are surgical procedures that will reduce fluid production and increase the removal of the fluid in the eye.
- Medical management can be attempted with the drugs normally used in glaucoma cases. These drugs can be administered locally or systemically, depending on the severity of the case.
- Chemical ablation is accomplished through an injection into the eye causing the death of the cells that produce the fluid.
- Enucleation is another option if the IOP causes the eye to become painful and other treatments are not pursued.

Client Education and Technician Tips

- Genetic condition, so affected animals should not breed.

Rabbit Uterine Adenocarcinoma

Description

Uterine adenocarcinoma is the most common neoplasia diagnosed in intact female rabbits over 5 years old. The adenocarcinoma is usually multiple tumors that will metastasize to other organs.

Clinical Signs

- A decline of fertility is one of the first clinical signs associated with uterine adenocarcinomas, but this may or may not be noted by owners.
- Other clinical signs include a bloody vaginal discharge, hematuria, masses may be palpable upon abdominal palpation, cysts in the mammary glands, behavioral changes especially

aggressiveness, pale mucous membranes, lethargy, and anorexia.

Diagnosis

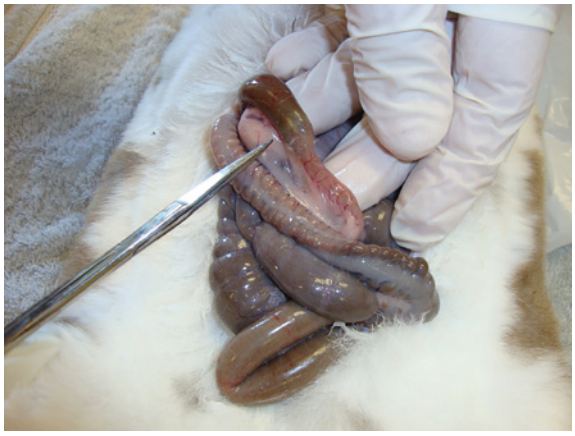
- Presenting clinical signs associated with the history of an adult intact female rabbit will put uterine adenocarcinoma on the rule out list.
- Radiographs and ultrasound will aid in diagnosis.
- Definitive diagnosis is obtained via biopsy and histopathology.

Treatment

- An OHE is the best treatment option. Prognosis is improved if the uterus is removed prior to metastasis (Figure 13.10).
- Chemotheraputic agents may be used in cases of metastasis.

TECH BOX 13.6: Rabbits not meant for breeding should be spayed.

(a)



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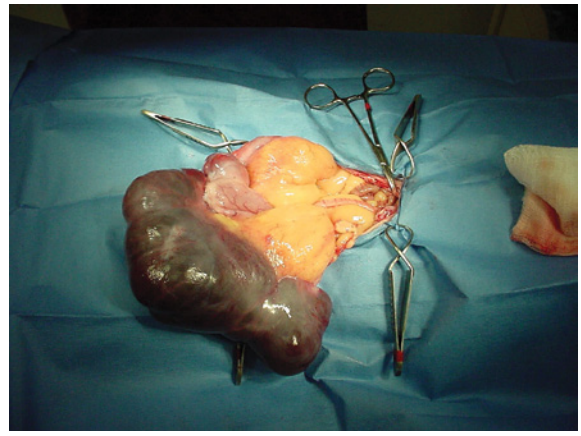

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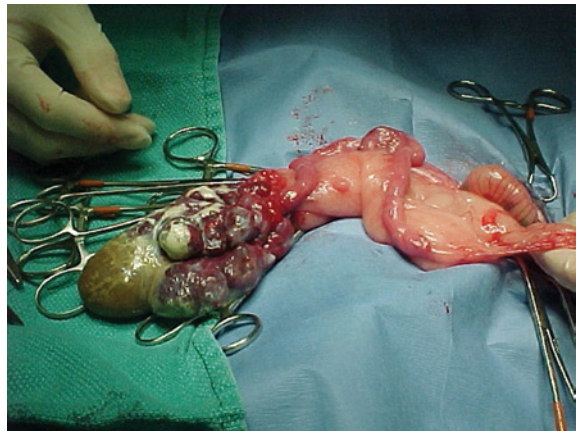

Figure 13.10 (a)–(c) Surgery to remove uterine tumor in rabbit. (Images courtesy Dan Johnson, DVM, www.avianandexotic.com)

Scurvy

Description

Scurvy is a condition seen in guinea pigs resulting from a lack of vitamin C. Guinea pigs lack the enzyme needed to synthesize vitamin C so it must be provided in the diet. Without vitamin C, defects in collagen synthesis and blood clotting ability are noted. Guinea pigs fed inappropriate diets such as rabbit food, expired food, or improperly stored food are at risk for scurvy.

TECH BOX 13.7: Guinea pigs must have vitamin C supplied in their food. Close attention must be paid to pellet expiration dates and storage conditions to ensure that animals are getting adequate amounts of the vitamin.

Clinical Signs

- Clinical signs appear 1–2 weeks after the vitamin C deficiency occurs.
- Animals with scurvy are reluctant to move, show painful movement, have inflamed joints, and exhibit lameness.
- Other clinical signs include anemia, opportunistic infections, anorexia, hemorrhages, weight loss, an ill-groomed or unkempt appearance, and sudden death.
- Hemorrhage around the muscle and periosteum of joints and epiphyseal enlargement are also noted in scurvy patients.

Diagnosis

- Clinical signs and history of vitamin C deficiency will provide a presumptive diagnosis for scurvy.
- Observation of lesions at time of necropsy will provide a definitive diagnosis.

Treatment

- If the condition is diagnosed soon enough, the treatment of choice is the daily administration of vitamin C for 1–2 weeks.

Client Education and Technician Tips

- Vitamin C is a very unstable vitamin and foods are usually only good for 90 days.
- Food should be stored in cool, dry, and dark locations.
- Vitamin C can also be supplemented with fresh fruits and vegetables containing vitamin C, treats, and vitamin supplements.

Antibiotic-Associated Enterotoxemia

Description

Antibiotic-associated enterotoxemia is a condition seen in rabbits, guinea pigs, and chinchillas prescribed certain antibiotics. Administration of antibiotics, and especially Gram- positive specific antibiotics, in the herbivore species will cause a disruption of normal flora, as their normal flora consists of mostly Gram-positive bacteria. This disruption allows for overgrowth of clostridial organisms (Gram-negative) that release toxins, which results in diarrhea and blood toxicity.

Clinical Signs

- Clinical signs begin within hours to 2 days after antibiotic administration is begun.
- Clinical signs include diarrhea, lethargy, anorexia, dehydration, and electrolyte imbalances.

Diagnosis

- History of antibiotic administration and clinical signs provide a presumptive diagnosis.

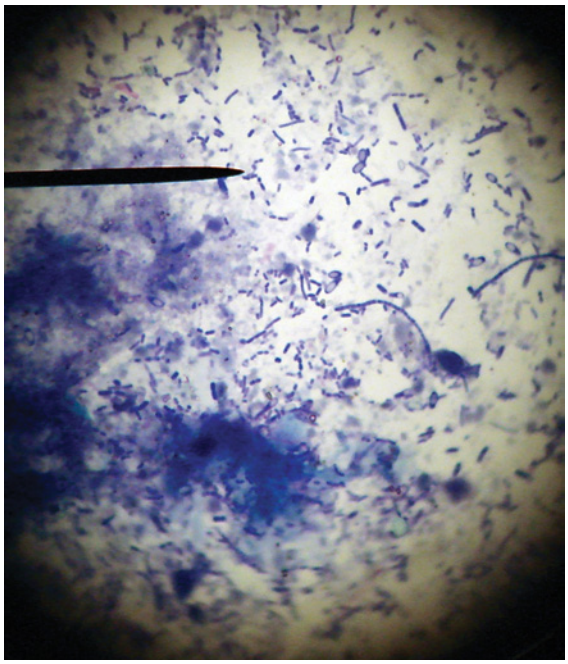

Figure 13.11 Clostridium found on a fecal cytology (stained with Diff Quik). Oval rods with clear center and purple outline. (Image courtesy Amy Johnson and Bel-Rea Institute of Animal Technology)

- A fecal cytology revealing increased *Clostridium* spp. will provide a definitive diagnosis (Figure 13.11).

Treatment

- This condition is often fatal, making prevention, rather than treatment, imperative.
- Antibiotic treatment must be ceased immediately.
- Fluid and electrolyte therapy is key in restoring fluid, electrolyte, and acid/base balances.
- Analgesics will make the patient more comfortable.
- Other treatments include probiotic therapy to restore bacterial balance, force-feeding, and drugs to bind the enterotoxins.

Client Education and Technician Tips

- Antibiotics should be chosen and prescribed carefully in these species. Penicillins and cephalosporins including ampicillin, amoxicillin, clindamycin, streptomycin, and erythromycin should be avoided. Safer choices include tetracycline, sulfa drugs, fluoroquinolones, and chloramphenicol.

TECH BOX 13.8: Guinea pigs can be given antibiotics, but the drug of choice should be chosen cautiously.

Streptococcal Lymphadenitis, Cervical Lymphadenitis, or Lumps

Description

Lumps is a condition seen in guinea pigs and characterized by abscesses of the cervical lymph nodes. These abscesses are caused by streptococcal organisms, and most commonly by *Streptococcus zooepidemicus*. The bacterium is allowed entrance to the body through the oral cavity lesions or through the respiratory tract.

Clinical Signs

- Inflammation of the ventral cervical lymph nodes will result in swelling under the chin.
- Initially the lymph nodes are firm, but later they fill with a thick caseous exudate resulting in a soft feel to the lymph node.
- Although some animals will develop a fatal septicemia, most seem healthy other than the infected lymph nodes.

Diagnosis

- Physical exam, including palpation of the ventral cervical lymph nodes, and clinical signs will provide a presumptive diagnosis.
- A culture of the lymph nodes will identify the organism.

Treatment

- Oral antibiotics will be prescribed, exerting caution not to prescribe an antibiotic that will cause a toxemia.
- Lancing, flushing, and draining of the abscessed lymph nodes may be required.

Cavian Cytomegalovirus (CMV)

Description

CMV is a common infection in guinea pigs caused by a virus from the Herpes virus family. Clinical signs are rare unless the animal is stressed or immunocompromised.

Transmission

- Transmission is from the virus shed in saliva or urine and transmitted in utero from sow to piglets.

Clinical Signs

- Inflammation and tenderness of salivary glands is noted in animals showing clinical signs of CMV.

Diagnosis

- Diagnosis is obtained via cytology of the salivary glands. The virus has characteristic inclusions seen in the cytoplasm and nucleus of cells of the salivary gland.

Treatment

- Although there is experimentation with antiviral therapy, there is limited treatment, other than supportive care, available.

TECH BOX 13.9: Both CMV and cavian leukemia virus will persist in the body of guinea pigs for years before clinical signs will become evident.

Cavian Leukemia/Lymphosarcoma

Description

Cavian leukemia/lymphosarcoma is a B-lymphocyte neoplasia caused by a virus in the Retroviridae family and similar to that of FeLV. The virus affects the blood cell production of infected animals. Most guinea pigs acquire the disease in utero and are born with the virus, which can live dormant for years before clinical signs appear.

Clinical Signs

- Clinical signs include pale mucous membranes, unkempt/disheveled appearance, anorexia, lethargy, icterus, and an ascending paralysis.
- Lymphadenopathy, splenomegaly, and hepatomegaly are also common with the virus.

Diagnosis

- History and clinical signs will put cavian leukemia on the rule out list.
- The CBC of infected animals reveals anemia and leukocytosis.
- Aspiration of lymph nodes will aid in diagnosis, along with radiographs or ultrasound showing the enlargement of lymph nodes, spleen, and liver.

Treatment

- There is no treatment for this virus, which will prove fatal for the guinea pig.

Guinea Pig Dystocia

Description

Dystocia is defined as a difficult birth and is common in guinea pigs with obesity, pelvic symphysis fusion, large fetuses, or abnormal fetal presentation. A dystocia can also be caused by uterine inertia.

Clinical Signs

- Clinical signs include bloody or greenish-brown vaginal discharge, abdominal pain, or an animal past the gestation time frame.
- Guinea pigs should normally deliver all fetuses within 1 hour of labor beginning. Prolonged labor without presentation of a fetus is common in dystocias. Part of a fetus may be seen in the birth canal and appear to look "stuck."

Diagnosis

- Breeding history, physical exam, and clinical signs will provide a diagnosis.
- Radiographs can be used to evaluate the number, size, and presentation of fetuses as well as the pelvic symphysis.

Treatment

- If the cause is attributed to a uterine inertia, hormone injections can be used to stimulate contractions.
- A C-section can be performed, although sows rarely survive the surgery.

Client Education and Technician Tips

- Guinea pigs should be bred for the first time before the pelvic symphysis fuses between 7 and 9 months. Once initially bred, the pelvis will not fuse.

TECH BOX 13.10: Guinea pigs not meant for breeding should be spayed or not housed with males.

Chinchilla Fur Slip and Fur Chewing

Description

Chinchillas with fur chewing conditions will chew on the fur of the lower body because of boredom, poor nutrition or husbandry techniques, and stress. Fur slip is the loss of fur in patches as a result of mishandling, fighting, or agitation from the release of adrenalin.

Clinical Signs

- Fur chewing chinchillas will have a "lion's mane" appearance, as the fur is chewed short on the lower half of the body where they can reach.
- Fur slip in chinchillas will cause hair loss in patches with the underlying skin appearing normal (Figure 13.12).

Figure 13.12 Chinchilla fur slip. (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

Diagnosis

- Both conditions are diagnosed on clinical signs, history, and physical exam.
- Animals that chew their fur will have fur in their stomachs upon necropsy, laparotomy, or radiography.

Treatment

- For fur chewing animals stimulation as well as chew toys should be provided. Another important factor to lessen chewing is stress reduction.
- Fur slip cannot be treated other than letting the fur grow back, which may take up to 5 months. Prevention is key and is accomplished by stress reduction and proper handling techniques.

Chinchilla Gastric Tympany (Bloat)

Description

Bloat in chinchillas is characterized by abdominal distension from gas accumulation in the stomach. Common causes include diet change, overeating, or hypocalcaemia in lactating females.

Clinical Signs

- Clinical signs include lethargy, dyspnea, and distended abdomen.
- Abdominal pain will cause the chinchilla to repeatedly roll and stretch.

Diagnosis

- A diagnosis is made using clinical signs, history, and physical exam.
- Radiographs will show a distended stomach filled with gas.

Treatment

- Passing of a stomach tube will relieve distension from gas.
- Lactating females respond well to calcium supplementation, usually given with IV.

Client Education and Technician Tips

- Owners should use caution when making diet or feeding schedule changes.
- Meals should be measured to avoid overfeeding.

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Diseases of Ferrets

chapter 14

Ferrets are carnivores that are closely related to dogs and cats. Their popularity as pets is bringing them into veterinary clinics more frequently, although they are illegal to own as pets in several states. Ferrets are very inquisitive, playful animals that will effortlessly find themselves in trouble. They will steal, chew, and ingest many objects around the house, making them very susceptible to gastrointestinal foreign bodies. Ferrets are susceptible to many infectious diseases, not only of ferrets but also human influenza and canine distemper. The most common diseases seen in ferrets include insulinomas, adrenal gland disease, and lymphoma.

Pancreatic Beta Cell Tumor or Insulinoma

Description

Insulinoma, the most common neoplasia seen in ferrets, is a tumor of the pancreatic beta cells. These tumors produce excess insulin, overdosing the patient, which results in hypoglycemia. Insulinomas are common in ferrets older than 2.

TECH BOX 14.1: Pancreatic beta cell tumors are the most common neoplasia seen in ferrets.

Clinical Signs

- Clinical signs of insulinomas in ferrets are commonly associated with hypoglycemia (Figure 14.1). These signs include weakness, lethargy, hind limb weakness, ptyalism, difficulty waking, weight loss, and ataxia.
- Bruxism (grinding of teeth and clenching of the jaw) and pawing at the face are also seen with insulinomas, possibly because of nausea.
- Seizures and coma may be seen in severe cases.

Diagnosis

- Blood chemistries on ferrets with insulinomas will reveal hypoglycemia with normal or

Small Animal Pathology for Veterinary Technicians, First Edition. Amy Johnson.

Companion website: www.wiley.com/go/johnsonvettechpath

Figure 14.1 Ferret exhibiting chronic weight loss, hypersalivation, and acute collapse due to hypoglycemic episode. (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

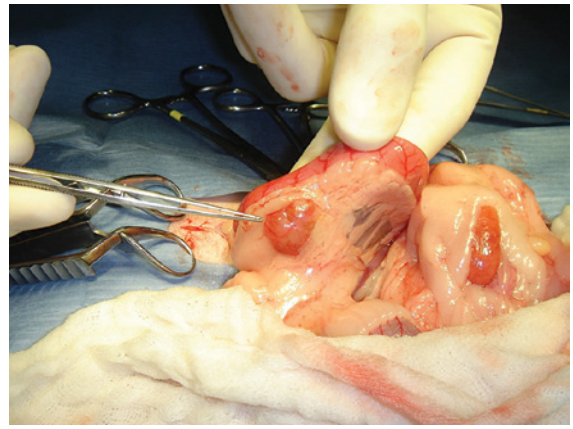


Figure 14.2 Exceptionally large pancreatic islet beta cell tumor, more commonly known as insulinoma. (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

elevated insulin levels, although insulin levels alone are not definitive.
- Ultrasound can be used to visualize pancreatic mass.

Treatment

- There is no cure for an insulinoma.
- Medical treatments include corticosteroids and insulin inhibitors.
- Some cases will involve removal of the mass, although insulinomas tend to metastasize within the pancreas, making full removal difficult (Figure 14.2).

Technician Duty Box 14.1

Ferrets suffering from an insulinoma will require frequent blood collection for monitoring of their blood glucose levels.

- High-carbohydrate diets and treats should be avoided, especially those with simple sugars. Simple sugars will enter the bloodstream quicker than more complex ones, causing spikes in blood glucose levels.

Client Education and Technician Tips

- Once a diagnosis is made, medical treatment is lifelong.
- Post-operative prognosis is guarded, as in many cases the insulinomas reoccur.
- This condition requires constant glucose monitoring to manage patients appropriately.

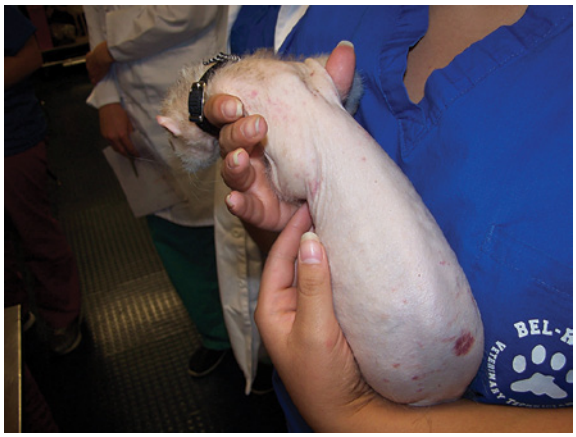
Adrenal Disease or Hyperadrenocorticism

Description

Unlike adrenal disease in dogs, adrenal disease in ferrets involves excess secretion of sex hormones (progesterone, testosterone, and estrogen). Hyperadrenocorticism, the second most common neoplasia in ferrets, is diagnosed in ferrets as young as 2, but the average age of onset is 4 years. This condition is caused by adrenal adenoma, adenocarcinoma, or in rare cases adrenal hyperplasia.

TECH BOX 14.2: Hyperadrenocorticism in ferrets is the not the same as Cushing's in dogs, as the disease in ferrets is caused by an oversecretion of sex hormones and not cortisol.

(a)



(b)

Figure 14.3 (a) Dorsal alopecia in a ferret with adrenal disease. (b) Ventral alopecia in a ferret with adrenal disease. (Images courtesy Amy Johnson, AnDee Groditski, and Bel-Rea Institute of Animal Technology)

Figure 14.4 Swollen vulva typical for adrenocortical disease in a female ferret. (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

Clinical Signs

- Hair loss is one of the common signs of adrenal disease in ferrets. Bilateral, symmetrical alopecia is seen on the dorsal side including flank, rump, and tail. The hair loss starts at the tail and ascends. Pruritus may also accompany the alopecia (Figure 14.3).
- Spayed females will have a swollen vulva, similar to an intact female in heat (Figure 14.4).
- Males will show signs of aggression and a return to mounting behavior. Straining to urinate is a sign of an enlarged prostate gland.
- Bone marrow suppression is seen as a result of excess estrogen production. This suppression will cause anemia, immune system suppression, and thrombocytopenia.

Diagnosis

- History, presenting signs, and physical exam are enough to make a presumptive diagnosis.
- Complete blood count will show anemia, leukocytopenia, and thrombocytopenia.
- Enlarged adrenal glands may be palpable.
- Although radiographs usually will not aid in the diagnosis, ultrasound will detect the enlarged glands.

- Definitive diagnosis requires a sex hormone panel or surgical biopsy of the adrenal glands.

Treatment

- Medical treatment is palliative, involving gonadotropin-releasing hormone (GnRH) blocker, androgen receptor blockers, antiandrogen drugs, aromatase inhibitors, and melatonin. Melatonin can be used to treat alopecia and reduces secretion of GnRH.
- Surgical treatment is the treatment of choice. Removal of adrenal glands can be curative, but the disease still recurs in about half of patients. Caution must be taken with surgery, as it can cause iatrogenic hypoadrenocorticism, requiring hormone supplementation.

Client Education and Technician Tips

- Once the disease is diagnosed, the treatment is lifelong and the patient requires constant monitoring.
- Theories of causes include a result of early spay or neuter (although it is seen in intact animals), a deviation from an archetypal diet, and genetics.

Aplastic Anemia/Estrogen Toxicity

Description

Aplastic anemia in ferrets results from an excess production of estrogen, which has toxic effects in the bone marrow. Female ferrets are induced ovulators—that is, not ovulating unless bred—and this causes prolonged secretion of estrogen. The bone marrow suppression results in the absence of RBC, WBC, and platelet production. These ferrets are susceptible to anemia, bleeding disorders, and secondary infections that can be fatal.

TECH BOX 14.3: Non-breeding ferrets should be altered.

Clinical Signs

- Bone marrow suppression and anemia will lead to pale mucous membranes, lethargy, dyspnea, anorexia, and hind limb weakness.
- A swollen vulva is noted due to the estrogen.
- Females in prolonged estrus will experience alopecia. The hair loss starts at the base of the tail and the medial surfaces of the legs and will eventually extend throughout the body.

Diagnosis

- Clinical signs in an unspayed female are enough to make a presumptive diagnosis.
- A CBC will show non-regenerative anemia and low PCV, WBC, and platelet counts.
- High estrogen levels in an intact female on a blood hormone panel are indicative of the condition.

Treatment

- The treatment of choice in females not meant for breeding is an OHE.
- If breeding is intended the ferret can be cycled out of estrus with hormone injections.
- In severe cases blood transfusions and iron supplementation may be required.
- Supportive care includes fluids, force-feeding, and steroids.

Client Education and Technician Tips

- Laws throughout the United States restrict the sale of unaltered ferrets to avoid conditions like these in pet ferrets.

- Death results from bleeding disorders attributable to thrombocytopenia or from secondary infections caused by immune system compromise.

Lymphoma/Lymphosarcoma

Description

Lymphoma and lymphosarcoma are common neoplasias in ferrets that affect many body systems and lymph nodes, including the spleen, liver, heart, thymus, and kidneys (Figure 14.5). These acute conditions in young ferrets are referred to as "juvenile" lymphosarcoma and are seen in ferrets younger than 2 years old. Chronic conditions in older ferrets are referred to as "classic" lymphosarcoma. A viral cause is suspected, as reports of the disease in multiferret households are increased.

Clinical Signs

- Enlarged lymph nodes are a common clinical sign in ferrets with "classic" lymphosarcoma. "Juvenile" lymphosarcoma is not always accompanied by lymphadenopathy.
- Other clinical signs are more non-specific, including anorexia, lethargy, weight loss, V/D, distended abdomen, and skin masses.
- Animals with a thymic mass will experience dyspnea due to the pressure of the mass on the lungs.

Diagnosis

- History, presenting clinical signs, and blood work with an elevated lymphocyte count are suggestive of the disease.
- Ultrasound and radiographs can be used to evaluate the size of lymph nodes and visualize any visceral masses.
- A definitive diagnosis is obtained with an aspirate of the bone marrow and the suspected lymph nodes (Figure 14.6).

Treatment

- Treatment includes removal of affected lymph nodes and chemotherapy or radiation therapy.
- Supportive care involves fluids, force-feeding, and corticosteroids.

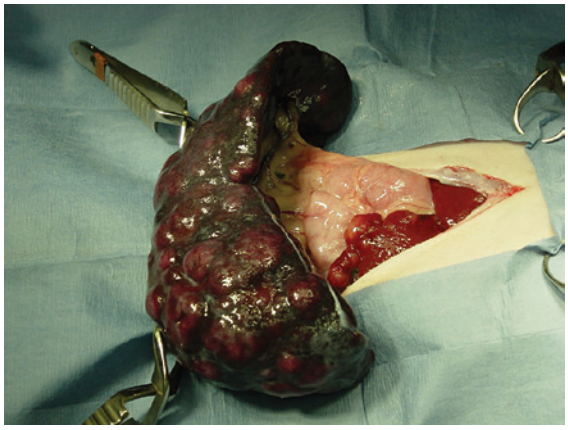


Figure 14.5 Nodules on spleen and liver due to Stage 4 lymphoma in a ferret. (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

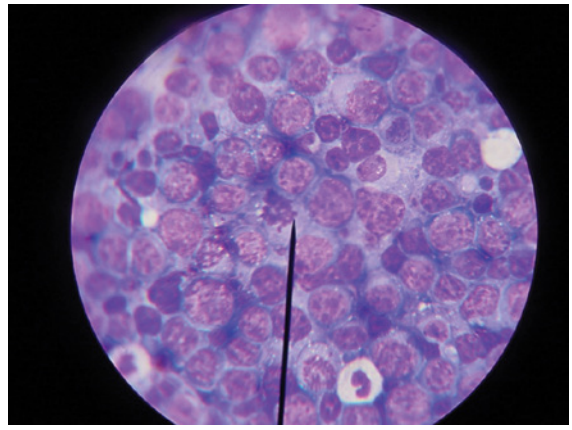


Figure 14.6 1,000X impression of lymphoma nodule, Wright-Giemsa stain (Diff Quik). (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

Client Education and Technician Tips

- Prognosis is questionable and based on progression of disease at the point of diagnosis. The more aggressive the treatment is the better the prognosis.

Influenza

Description

Ferrets are susceptible to many human flu viruses. Young ferrets are generally at a greater risk due to secondary infections. These viruses are zoonotic as well as inverse zoonotic and spread through aerosolization, direct contact, and fomites.

TECH BOX 14.4: Flu viruses are zoonotic as well as inverse zoonotic.

Clinical Signs

- Clinical signs are generally mild and include sneezing, coughing, serous discharge from the nose and eyes, conjunctivitis, lethargy, fever, anorexia, and rarely V/D.
- Clinical signs usually persist 1–2 weeks.

Diagnosis

- A presumptive diagnosis is usually made based on physical exam, presenting clinical signs, and history of illness or exposure.
- A definitive diagnosis can be obtained via reference lab tests, although this is rarely done.

Treatment

- This infection is generally self-limiting, with no treatment required.
- Antibiotics for prevention and treatment of secondary bacterial infections may be used, along with antihistamines and cough suppressants.
- Supportive care includes fluids and good nutrition.

Client Education and Technician Tips

- Influenza infections can be confused with the early signs of distemper. Distemper has a thicker green nasal and ocular discharge and a high fever.
- To avoid illness, sick owners and animal handlers should avoid ferrets during their illness or wear a mask and wash their hands.

Epizootic Catarrhal Enteritis (ECE) or Green Slime Diarrhea

Description

ECE is a highly contagious gastrointestinal disease seen in many ferrets, especially from shelters, rescues, pet stores, and areas of high ferret concentration. The cause is suspected to be a virus from the Coronavirus family with an incubation of 2–4 days. Although highly contagious, the disease has a low mortality.

TECH BOX 14.5: ECE is a highly contagious disease transmitted by introduction of new ferrets into the household.

Transmission

- The infection is transmitted through the fecal-oral route with fomites or introduction of a new ferret into the household.

Clinical Signs

- Younger ferrets show few, very mild clinical signs, but the older the ferret the worse the disease condition. Clinical signs usually last 2–14 days.

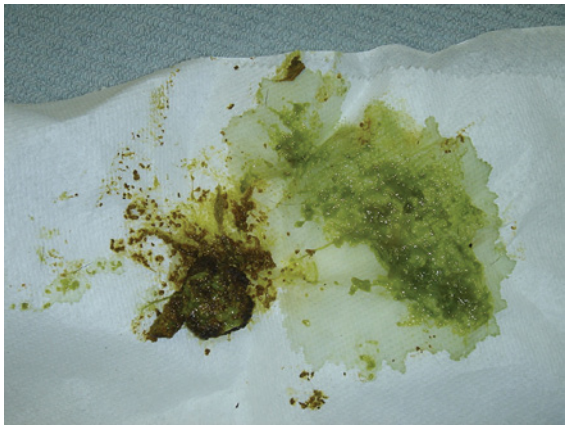


Figure 14.7 "Green slime" diarrhea in a ferret. (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

- Clinical signs include anorexia, weight loss, lethargy, dehydration, and vomiting.
- Persistent diarrhea is common, with the feces changing throughout the progression of the disease. In the early stages green mucoid diarrhea is seen (Figure 14.7). In later stages the lining of the intestine is affected, stopping the absorption of nutrients and fluids; this will cause the feces to take on a "bird seed" appearance (Figure 14.8).

Diagnosis

- History of exposure to new young ferrets and clinical signs are enough to make a presumptive diagnosis.
- A definitive diagnosis is difficult to obtain. Electron microscopy of the feces may identify the cause as a coronavirus.

Treatment

- Treatment is strictly supportive and will include easily digestible food, fluid therapy, antibiotics, and gastrointestinal protectants.

Client Education and Technician Tips

- Good sanitation is imperative in stopping the transmission of the disease.

Figure 14.8 "Green slime" diarrhea in a ferret. (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

- Most recovered animals become persistent carriers of the disease for 6 months. All animals should be isolated for 6–12 months from uninfected animals.

Ferret Systemic Coronavirus (FRSCV) or Ferret FIP

Description

FRSCV is a newly recognized, emerging disease in ferrets and is similar to FIP in cats. This disease is seen in young ferrets, commonly less than 1 1/2 years old, and is usually fatal.

Transmission

- The disease is spread through contact with infected ferrets. Although the exact route of transmission is unknown, the fecal-oral route is strongly suspected.

Clinical Signs

- Clinical signs include diarrhea, anorexia, weight loss, lethargy, vomiting, fever, muscle wasting, bruxism, green urine, inflamed rectum, prolapsed rectum, abdominal masses, splenomegaly, enlarged kidneys, and heart murmurs.
- Central nervous signs include hind limb weakness, ataxia, tremors, seizures, and head tilt.

Diagnosis

- History, presenting clinical signs, blood work, radiographs, and ultrasound will aid in a presumptive diagnosis.
- A blood chemistry panel will reveal changes in abdominal organs and CBC will show a nonregenerative anemia and thrombocytopenia.
- Radiographs and ultrasound will allow evaluation of abdominal organs and abdominal masses.
- Histopathology of lesions showing lesions consistent with FRSCV is highly suggestive of the disease.
- PCR and immunohistology are assays that will give a definitive diagnosis.

Treatment

- Prognosis is very poor as most cases are fatal. Most commonly ferrets with FRSCV are euthanized.
- Treatment if attempted is symptomatic and supportive and includes fluid and nutrient support. Euthanasia needs to be considered when the quality of life declines.

Canine Distemper

Description

Ferrets are highly susceptible to canine distemper, a virus that carries a high mortality in ferrets.

TECH BOX 14.6: Ferrets are susceptible to canine distemper, which is most often fatal. Ferrets and dogs housed together should both be vaccinated for the disease.

Transmission

- Ferrets are exposed to the virus through exposure to aerosolized secretions, fomites, and infected animals, usually dogs.

Clinical Signs

- The early signs of the disease are characterized by dermatitis on the chin and in the inguinal area, which will progress to a thick brown crusting on the eyelids and nose. Hyperkeratinization of the footpads is also common.
- As the disease progresses, fever, anorexia, red mucous membranes, mucopurulent ocular and nasal discharge, pneumonia, and rectal prolapse occur.
- During the end stages of the disease CNS signs including hypersalivation, coma, ataxia, and seizures become common. These CNS signs are referred to as "screaming fits."

Diagnosis

- A history of exposure in an unvaccinated ferret and presenting clinical signs are enough to make a presumptive diagnosis.
- IFA, histopathology, or antibody titer will provide a definitive diagnosis.

Treatment

- There is no treatment other than supportive. Distemper carries up to a 100% mortality rate with ferrets dying 10–14 days post-clinical signs.

Client Education and Technician Tips

- Ferrets can be vaccinated against canine distemper. There is one vaccine on the market for use in ferrets, although vaccine reactions are always a risk in ferrets.
- Owners and caretakers with dogs, especially young puppies, should use caution with handling ferrets.

Gastric Foreign Bodies

Description

Ferrets are very inquisitive, curious animals, especially young kits, and have a tendency to ingest foreign materials that become retained in their stomach or cause intestinal obstruction. These items are usually rubbery and spongy toys and objects and bedding (Figure 14.9) but can also be hairballs. Gastric foreign bodies are a common diagnosis in young anorexic ferrets.

TEXT BOX 14.7: Gastric foreign bodies are the most common GI disease in ferrets under 1 year.

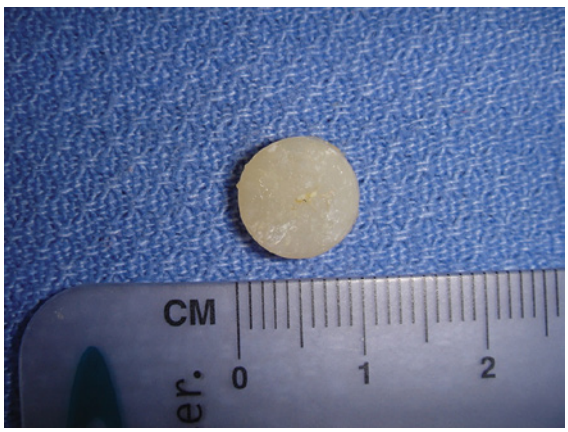


Figure 14.9 Rubbery gastric foreign body in a ferret. (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

Clinical Signs

- Clinical signs include vomiting, anorexia, bruxism, and abdominal pain.

Diagnosis

- History of anorexia and clinical signs in a ferret with the tendency to ingest foreign objects puts gastric foreign bodies at the top of the rule out list.
- The foreign object may be palpable with abdominal palpation.
- Definitive diagnosis is obtained via radiographs or endoscopy where the object can be visualized (Figure 14.10).

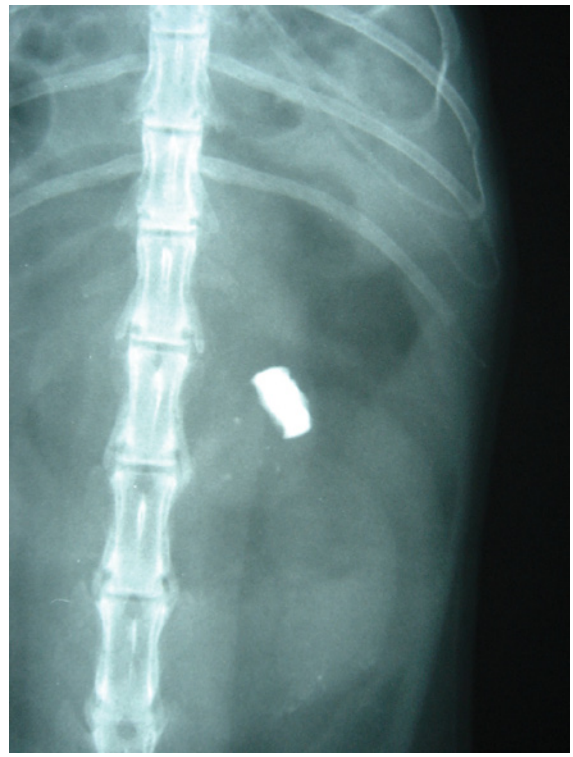


Figure 14.10 Radiograph of a dental crown foreign body in a ferret. (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

Treatment

- Some objects may be removable via endoscopy depending on size, shape, and location.
- Laparotomy to find and remove the foreign body.

Client Education and Technician Tips

- These ferrets have a good prognosis.
- In the case of hairballs, ferret laxative or petroleum jelly can be used for prevention.
- Owners must be vigilant to keep the ferret out of harm and appropriate toys should be chosen.

TEXT BOX 14.8: Soft, rubber, or chewy toys should be avoided with ferrets.

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Diseases of Hamsters, Gerbils, and Rats

chapter 15

Small rodents are often kept as pets for young children, school classrooms, and people living in places that do not allow dogs and cats. Their advantages include being contained in a cage, the ease of cleaning and feeding, and the low cost of maintenance. The major disadvantage of these species is the short life span. Some diseases are common throughout the three species, although each species has its own unique conditions. The most frequently seen diseases in these species are infectious agents.

Malocclusions

Description

Malocclusions are seen in rodents as a result of open-rooted/continually growing teeth and when the teeth grow too long to allow for proper mouth occlusion. This condition is often caused by incorrect diet, resulting in a lack of filing of teeth, but can also be attributed to jaw malformation resulting from genetics or nutrient deficiencies.

TEXT BOX 15.1: Rodents have continually growing teeth that should have their length monitored on a regular basis.

Clinical Signs

- Often long teeth growing outside of the mouth or penetrating into the palate and/or sinus will cause ptyalism, wet fur around mouth, oral or nasal bleeding, anorexia, weight loss, decreased or absent water intake, and dehydration (Figure 15.1).

Diagnosis

- Diagnosis is made based on clinical signs and physical exam.
- Dental radiographs can be used to evaluate the extent of the damage.

Small Animal Pathology for Veterinary Technicians, First Edition. Amy Johnson.

Companion website: www.wiley.com/go/johnsonvettechpath

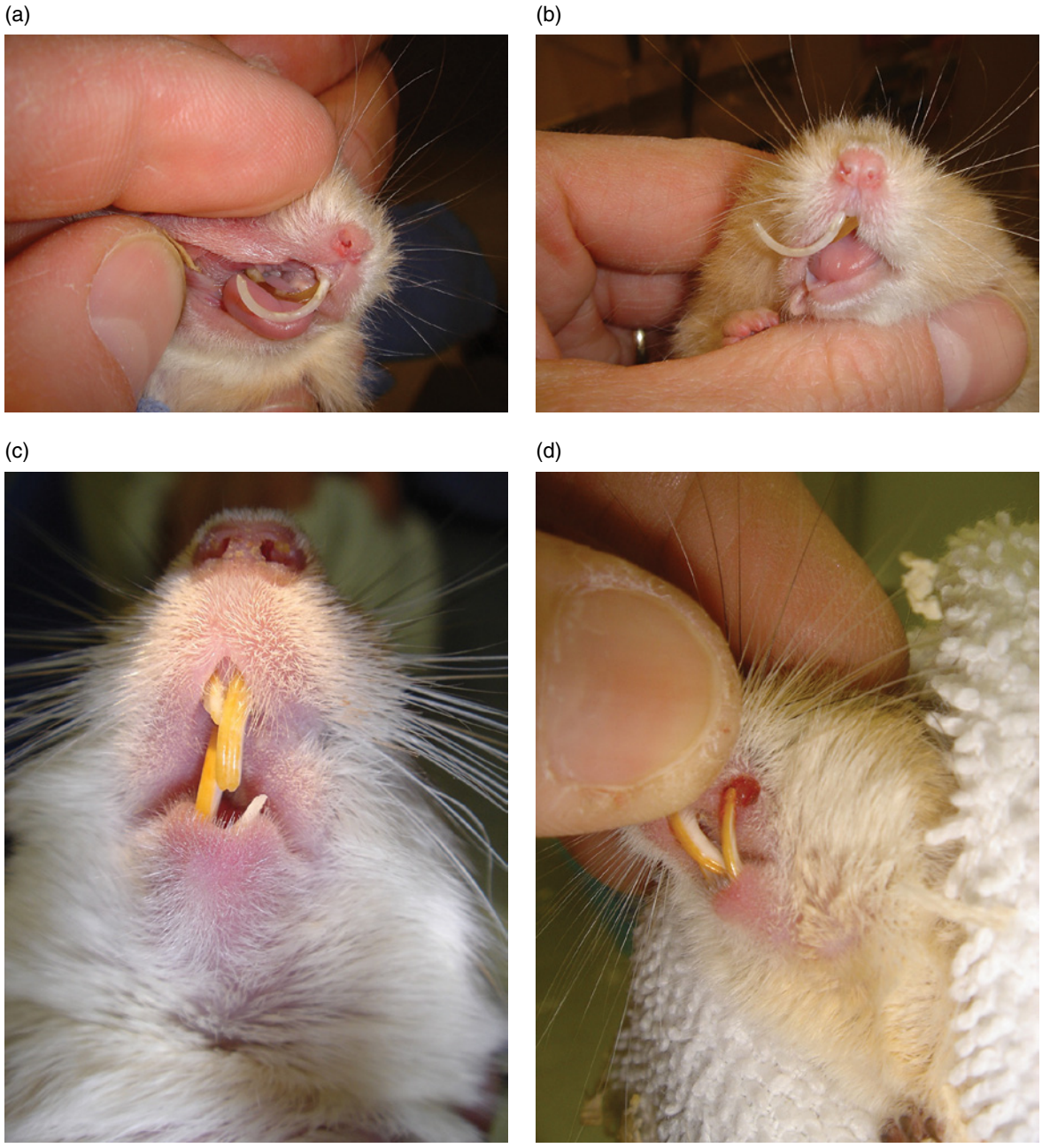

Figure 15.1 (a) Incisor malocclusion in a hamster. (b) Incisor malocclusion in a hamster. (c) Incisor malocclusion in a gerbil. (d) Incisor malocclusion in a gerbil resulting in a soft tissue injury. (Images courtesy Dan Johnson, DVM, www.avianandexotic.com)

Treatment

- Treatment involves cutting or filing the teeth using caution not to enter the pulp cavity or cut the teeth too short to allow for proper chewing.
- Supportive care is necessary for debilitated animals. Fluid and nutrient supplementation is required if the animal is unable to eat or drink on its own.

Client Education and Technician Tips

- A hard pellet diet is necessary to keep teeth short, and chew toys should be provided.

Proliferative Ileitis, Proliferative Enteritis, or Wet Tail

Description

Proliferative ileitis is a general term used to describe a diarrheal disease caused by a bacterial infection. Multiple organisms are to blame, with *Lawsonia intracellularis* the main organism, yet *Escherichia coli*, *Campylobacter jejuni*, and others have been isolated. This condition is seen in all rodents but is most commonly associated with hamsters. The disease carries a high morbidity and high mortality rate. Contributing factors include stress, transport, overcrowding, surgery, or dietary changes.

TEXT BOX 15.2: Proliferative ileitis is a general term used for bacterial diarrheal diseases in hamsters.

Transmission

- The bacteria are spread via the fecal-oral route.

Clinical Signs

- Diarrhea found in the cage or accumulating on the ventral side of body and perineal area, causing the fur to take on a wet look (Figure 15.2).
- Other clinical signs include anorexia, lethargy, and dehydration.

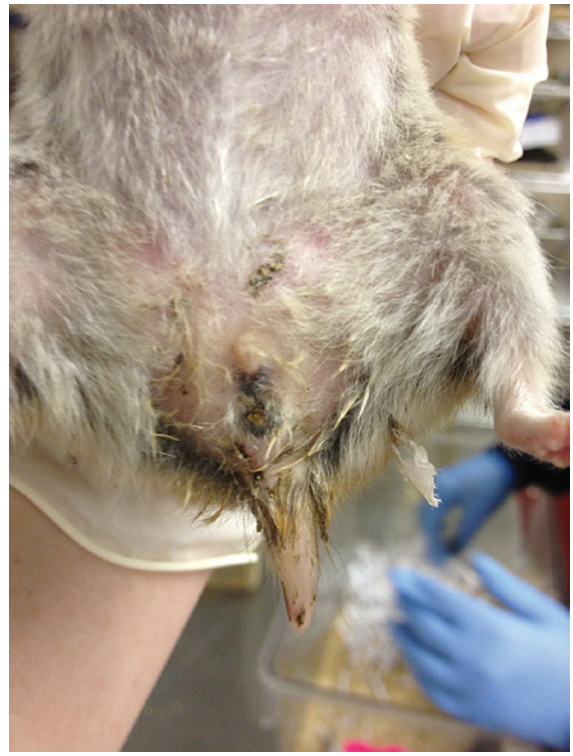

Figure 15.2 "Wet tail" in a hamster. (Image courtesy Amy Johnson, Jennifer Koester, and Bel-Rea Institute of Animal Technology)

Diagnosis

- Clinical signs, history, and physical exam will aid in the general diagnosis.
- Identification of specific bacterial organism requires histopathology, culturing of lesions, or PCR at the point of necropsy.

Treatment

- Supportive care includes fluids and electrolyte replacement.

- Antibiotics will be prescribed but must be cautiously chosen so as not to cause antibiotic toxicity.

Client Education and Technician Tips

- Isolation, good sanitation, stress reduction, and preventing overcrowding are important keys in the prevention of this condition.

Antibiotic-Associated Enterotoxemia or Clostridial Enteropathy

Description

Similar to other herbivore species, this condition results from Gram-positive specific antibiotic causing an overgrowth of clostridial (Gram-negative) organisms. This condition carries a high mortality rate.

TEXT BOX 15.3: Antibiotic-associated enterotoxemia is often a fatal condition in rodents that is easier to prevent rather than treat. Antibiotics should be prescribed cautiously.

Clinical Signs

- Clinical signs include profuse diarrhea, anorexia, weight loss, and dehydration in association with antibiotic administration.

Diagnosis

- Clinical signs, a history of antibiotic administration, and physical exam will aid in diagnosis.
- Fecal cytology or anaerobic cultures provide a definitive diagnosis (Figure 15.3).

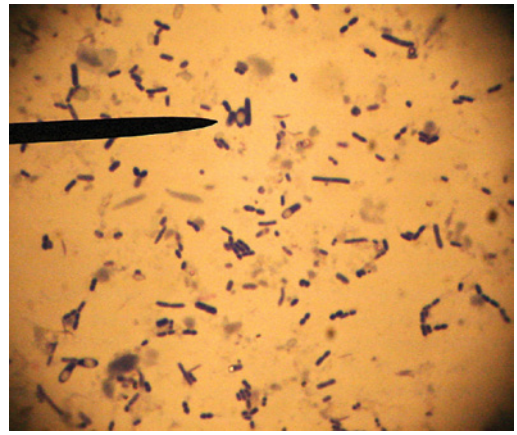

Figure 15.3 *Clostridium* in a fecal cytology (Diff Quik). (Image courtesy Amy Johnson and Bel-Rea Institute of Animal Technology)

Treatment

- Supportive therapy includes fluid, nutrition, and electrolyte replacement.
- Oral administration of feces from healthy animals will help reestablish normal flora balance.

Tyzzer's Disease or *Clostridium piliforme*

Description

Tyzzer's disease, reported in rats, gerbils, and occasionally in hamsters, causes a disease similar to wet tail. The infection is caused by *C. piliforme*, a spore-forming Gram-negative rod bacteria.

Transmission

- Tyzzer's is transmitted through the fecal-oral route.

Clinical Signs

- Clinical signs include anorexia, weight loss, dehydration, often foamy and yellow diarrhea, and sudden death.

Diagnosis

- Presumptive diagnosis is based on clinical signs and history.
- Definitive diagnosis is determined via Gram-negative organisms found on histology of intestine, liver, and heart tissue or PCR and ELSIA assays available through reference labs.

Treatment

- Antibiotics can be prescribed to fight this bacterial infection.
- Supportive care includes fluid and nutrient support.

Client Education and Technician Tips

- *C. pilliforme* is a spore-forming bacteria. These spores are difficult to kill so vigilant sanitation is important.

Respiratory Infections

Description

Infections of the respiratory tract are common in rats and hamsters, although rarely seen in gerbils. There are many causes associated with these infections, both bacterial and viral, including murine respiratory mycoplasmosis (MRM), cilia-associated respiratory (CAR) bacillus, *Streptococcus* spp., *Staphylococcus* spp., *Pasteurella* spp., Sendai virus, *Klebsiella* spp., *Bordetella* spp., and *Corynebacterium* spp. Often these are opportunistic infections from normal flora bacteria.

Acute and chronic respiratory infections are possible, with concurrent infections being common.

Transmission

- Most infections are spread via aerosol, direct contact, and fomites.
- Some organisms may be transmitted in utero and through sexual transmission.

Clinical Signs

- Respiratory infections in some animals are often subclinical, or animals will show signs of infection after stress has compromised their immune systems.
- Clinical signs include sneezing, nasal and ocular discharge (Figure 15.4), dyspnea, hunched posture, tooth grinding, head tilt, unkempt

(a)

(b)

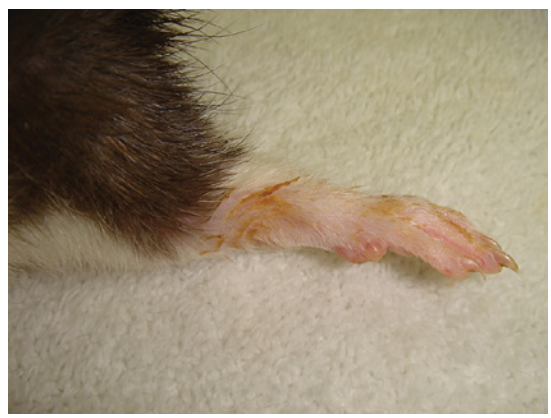

Figure 15.4 (a) Porphyrin and mucus accumulation on the face associated with respiratory infection in a rat. (b) Porphyrin and mucus accumulation on the forepaws associated with respiratory infection in a rat. (Images courtesy Dan Johnson, DVM, www.avianandexotic.com)

appearance, and loud breathing sounds called "snuffles."

Diagnosis

- Clinical signs, history, and physical exam will provide a diagnosis of respiratory infection.
- Histopathology at time of necropsy, ELISA, or PCR can be used to identify the specific causative agent(s).

Treatment

- Antibiotics are often prescribed but in many cases they will just suppress the clinical signs and not actually clear the infection.

TEXT BOX 15.4: Antibiotic therapy may help suppress respiratory infections but not rid the body of the infection.

- Supportive care may be necessary in severe cases, including fluid and nutrient supplementation.

Client Education and Technician Tips

- Good sanitation and isolation of sick animals are important steps in the prevention of the infections.

Neoplasia

Description

Neoplasias are more common in rats than any other rodent species, although occasionally some tumors will be diagnosed in older gerbils.

In rats mammary tumors are the most common tumor, as mammary tissue is dispersed throughout the body. These tumors are usually a benign fibroadenoma and can grow very large. Pituitary adenomas are also common, especially in female rats. A possible correlation has been made with these tumors and high-calorie diets. Large granular lymphocytic leukemia, a blood cancer, is a common cause of death in older rats.

TEXT BOX 15.5: Mammary fibroadenomas are the most common reproductive tumor found in both male and female rats.

Although tumors are rare in gerbils, they can occasionally be seen in older animals. Squamous cell carcinomas associated with the ventral scent gland are thought to develop secondary to bacterial infections of the skin or gland. These scent gland tumors rarely metastasize. Squamous cell carcinomas and melanomas of the foot and ear may occasionally develop.

Clinical Signs

- Mammary fibroadenomas will appear as a mass beneath the skin, usually freely movable under the skin, and may ulcerate (Figure 15.5).
- Pituitary adenomas commonly secrete prolactin, causing mammary changes, including lactation. These tumors can put pressure on the

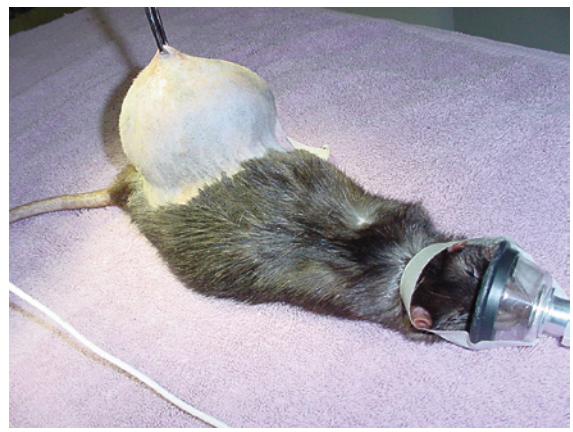

Figure 15.5 Mammary gland fibroadenoma in a rat. (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

Figure 15.6 Neoplasia of the abdominal scent gland in a gerbil. (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

brain and brain stem, causing hydrocephalus and CNS signs including head tilt, seizures, paralysis, circling, ataxia, weakness, and anisocoria.

- Large granular lymphocytic leukemia in rats causes leukocytosis, weight loss, anemia, jaundice, and lethargy. Lymphadenomegaly, splenomegaly, and hepatomegaly will also be noted with this cancer.
- Gerbils with squamous cell carcinomas of the ventral scent glands will have an ulcerated mass found in association with the ventral scent gland. These masses may start as a small scabby rough lesion that the animal self-mutilates (Figure 15.6).
- Gerbils with squamous cell carcinomas or melanomas will have an ulcerated or black mass found on the foot or the ear margin (Figure 15.7).

Figure 15.7 Melanoma surgically excised from the rear paw of a gerbil. (Image courtesy Amy Johnson and Bel-Rea Institute of Animal Technology)

Diagnosis

- Diagnosis can be obtained via palpation of the mass or radiographs and other diagnostic imaging techniques.
- Histopathology will determine the definitive type of the neoplasia.

Treatment

- Surgical removal of the mass, if possible, is ideal. The prognosis depends on the mass type and whether it has metastasized (Figure 15.8).
- Euthanasia should be considered when the quality of life is compromised.

Ulcerative Pododermatitis or Bumblefoot

Description

Bumblefoot is inflammation of the plantar surface of the foot seen in rats, gerbils, and hamsters. The condition starts as a wound and

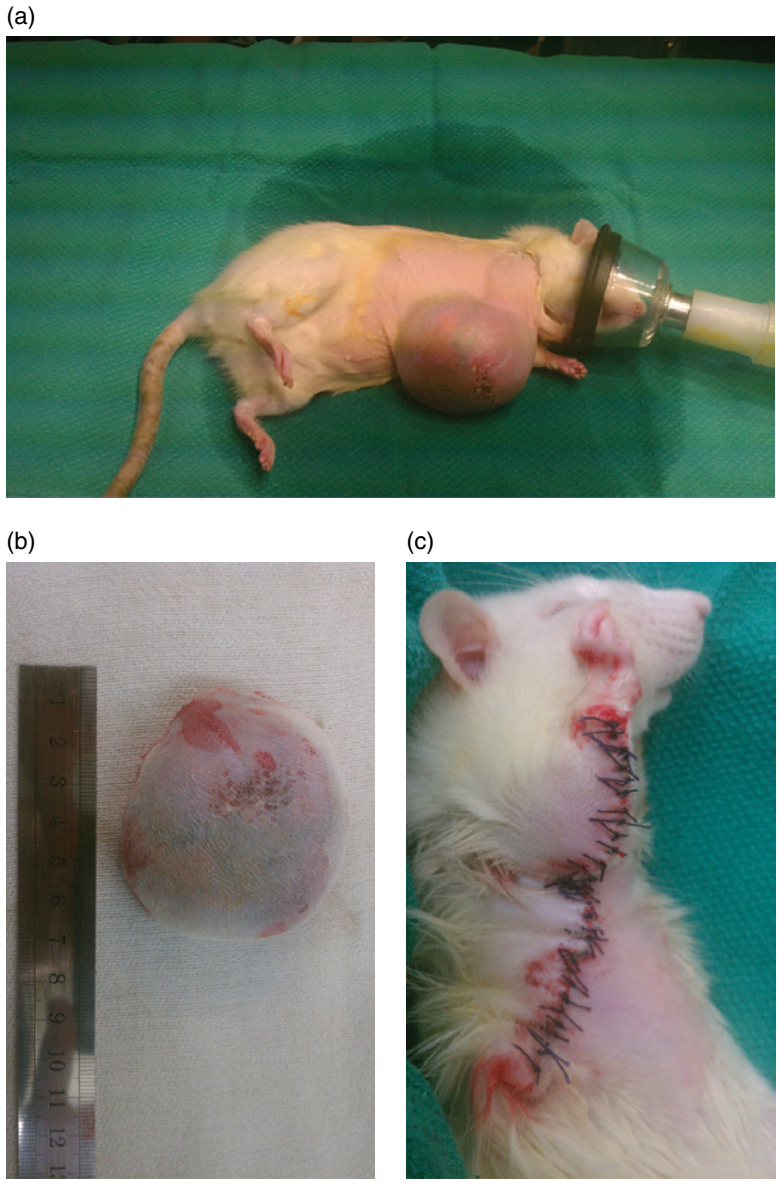

Figure 15.8 (a) Surgical excision of mammary tumor in a rat. (b) Tumor post-excision. (c) Patient sutured post-operatively. (Images courtesy Hillary Price)

becomes infected as urine, feces, and contaminated bedding comes into contact with the wound. Although many organisms can be linked to the condition, the most common organism isolated from these wounds is *Staphylococcus aureus*. Uncomfortable bedding or cage flooring, obesity, and genetics are all predisposing factors.

Clinical Signs

- Animals with bumblefoot have unhealthy plantar surfaces of their feet, including inflammation, ulcerations, and abscesses.

Diagnosis

- Clinical signs, history, and physical exam are enough to make a diagnosis of bumblefoot.
- Culturing can provide a causative agent.

Treatment

- The feet should be soaked in disinfecting agents with antibiotic ointments applied topically.

Technician Duty Box 15.1

Soaking the feet of patients with ulcerative pododermatitis is an important part of their recovery. The veterinary technician will be given the duty of soaking the feet of these animals and application of topical antibiotic ointments.

- More severe cases may need oral antibiotics and the feet bandaged.
- Animals should be moved to more comfortable bedding and solid surfaces. Cage flooring should be kept sanitary.

Chromodacryorrhea or Red Tears

Description

Chromodacryorrhea is a condition seen in rats and gerbils caused by a secretion of porphyrins (pigments) from the Harderian gland located behind the eye. These pigments are released as a result of stress or illness.

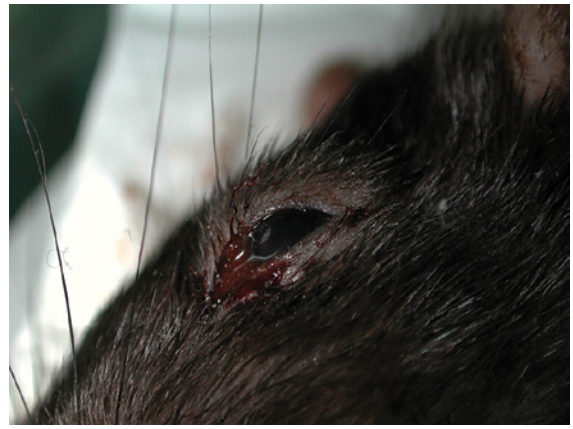

Figure 15.9 Chromodacryorrhea in a rat. (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

Clinical Signs

- A red-colored pigment is released into the eye and drains through the nasolacrimal duct. This causes red tears and a red crusting around the eyes and nose of the affected animal (Figure 15.9).
- This pigment is commonly confused with blood and may alarm the owner.

Diagnosis

- Clinical signs, physical exam, and history will provide a diagnosis.
- Porphyrins can be differentiated from blood with the use of a Wood's lamp, as porphryin fluoresces under the Wood's lamp while blood does not.

Treatment

- No treatment is necessary.

TEXT BOX 15.6: Chromodacryorrhea is not a condition that requires treatment, yet the animal should be evaluated to find the underlying condition.

Client Education and Technician Tips

- This condition is often the first sign of illness or stress in these animals. Animals should be examined by a veterinarian to determine the underlying cause of the condition.

Arteriolar Nephrosclerosis or Hamster Nephrosis or Renal Failure

Description

Nephrosis is a degenerative renal disease seen in old hamsters and is a common cause of death in geriatric animals. This condition is diagnosed more commonly in females and may be secondary to viral infections, hypertension, and the aging process.

TEXT BOX 15.7: Renal failure in a common cause of death in geriatric hamsters.

Clinical Signs

- Clinical signs include anorexia, lethargy, weight loss, and PU/PD.
- Many animals will experience sudden death in the absence of clinical signs.

Diagnosis

- Clinical signs and history will add nephrosis to the rule out list.
- Blood work will reveal uremia and proteinuria. Urinalysis will show a low specific gravity.
- Histological changes of kidneys at the point of necropsy will give a definitive diagnosis.

Treatment

- There is no treatment for this condition.
- Euthanasia needs to be considered when the quality of life declines.

Lymphocytic Choriomeningitis Virus (LCMV)

Description

LCMV is a zoonotic infection found in hamsters and caused by an *Arenavirus*. The natural carriers of this infection are wild mice, but the virus can be transmitted to hamsters. Hamsters are often subclinical carriers, causing an influenza-like disease in humans. The virus can be linked to miscarriages in pregnant females, and a fatal encephalitis/meningitis may be seen in severely immunocompromised people.

TEXT BOX 15.8: LCMV is a zoonotic disease linked to influenza-like disease, miscarriages, and in rare cases meningitis and encephalitis. In severely immunocompromised people this virus can be fatal.

Transmission

- The disease is transmitted from animals to humans through saliva, bites, or contact with urine, feces, or infected tissues.
- Hamsters with persistent infections shed large amounts of virus in the urine.

Clinical Signs

- Hamsters are almost always subclinical.
- In hamsters showing clinical signs a chronic wasting disease is evident, including anorexia, weight loss, seizures, and decreased fertility in females.

Diagnosis

- Definitive diagnosis is made through pathology and a serological ELISA antibody test.

Treatment

- No treatment is needed in animals, as they are asymptomatic and known positives should be euthanized due to the zoonotic potential.

Client Education and Technician Tips

- Sanitation, good personal hygiene, and limiting exposure to wild reservoirs are important keys to prevention of the disease.

Gerbil Epileptiform Seizures

Description

Gerbils may experience a form of spontaneous epileptic seizures due to stress, handling, or new environments.

Clinical Signs

- Gerbils may appear hypnotic and nonresponsive in minor cases, may experience muscle twitching, or may experience grand mal seizures.
- These seizures are typically brief, only lasting a couple of minutes at most.

Diagnosis

- With clinical signs and history a diagnosis can be made.

Treatment

- No treatment is usually required, as the gerbil will come out of the seizure on its own accord.
- Antiseizure medications can be tried in severe cases but should be used with caution.

Client Education and Technician Tips

- Frequent handling of animals, especially young ones, will lessen stress associated with handling.

Gerbil Tail Slip or Tail Degloving

Description

Tail slip in a gerbil is a degloving injury of the tail and most commonly as a result of mishandling. Other causes may include cage mate fighting or getting tail wedged in the cage. Gerbils have little connective tissue holding the skin to the tail, making the injury common.

TEXT BOX 15.9: To avoid degloving injuries, gerbils should never be handled by the tip of their tail.

Clinical Signs

- Degloving of the skin on the tail leaving muscle, nerves, and vertebrae exposed (Figure 15.10).

Figure 15.10 Tail slip in a degu (a close relative to the gerbil). (Image courtesy Dan Johnson, DVM, www.avianandexotic.com)

Diagnosis

- A diagnosis can be made based on clinical signs.

Treatment

- The injury involves pain and may result in self-mutilation and infection. Because of this the tail should be amputated at the point of the injury, with the skin sutured closed.
- Analgesics will keep the animal comfortable after the injury and surgery.

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