Common Emergencies in Exotic Pets
James K Morrisey, DVM, Dipl. ABVP (Avian)
Cornell University

Emergency medicine in exotic pets uses the same principals as in other species. Triage parameters are also very similar with priority being given to bleeding animals, animals in respiratory distress and animals in shock. It's important to be prepared for the emergency; have a designated area that with emergency supplies, drug dosage charts, and equipment that might be needed. Train your personnel in emergency procedures; from the front office staff to the techs to the animal caretakers. They should all know what signs constitute an emergency in exotic pets and where to bring the critical patient. This lecture will discuss some of the most common emergency presentations for birds, ferrets and rabbits. Initial treatment and stabilization options will be given for each condition.

BIRDS

Trauma/bite wounds

Fractures of the extremities should be bandaged until the patient is stabilized adequately for radiographs and definitive treatment of the fracture. Wing fractures may be stabilized in figure of 8 bandages for fractures distal to the elbow. Fractures of the humerus should be wrapped in a light figure of 8 bandage and then secured to the body to prevent should movement. Fractures of the coracoid or clavicle don't need to be bandaged unless the wing is drooping significantly that the bird is stepping on it or otherwise damaging it. A figure of 8 or body wrap can be used in those situations, depending on how much wing droop is present.

Fractures of the pelvic limbs most commonly involve the tibiotarsus because of the length of the bone and relatively light muscular covering. These fractures can be stabilized using an external coaptation and splinting. Femoral fractures are not amenable to external coaptation and need to be surgically repaired or allowed to heal by cage rest alone. Open fractures should be cleaned and flushed (if not pneumatic) and fixation should be attempted as soon as possible to minimize osteomyelitis and septicemia. Fracture repair methods will depend on several factors such as the location of the fracture, size of the patient, owner compliance, and clinician's preference. Stabilization of the patient will be determined by the animal's condition, but should include fluid support, analgesics, and antibiotics in open fractures.

Treatment of lacerations depends on the severity of the wound, time since occurrence, and the condition of the patient. Minor lacerations treated within 12-24 hours may be cleaned and closed surgically. Lacerations older than 24 hours should be cleaned and debrided and allowed to heal by second intention. Make sure to check for damage to underlying tissues in the area of the wound. Lacerations that penetrate the coelomic cavity should be gently cleaned but not flushed. Bandaging or suturing the body wall be done to protect the coelomic cavity from further contamination.

Birds sustaining bite wounds from dogs, cats, or other birds are frequently encountered. Bird bite wounds are usually of the crushing and tearing type and may require surgical repair or debridement. Mammal bites, especially cat bites, are true emergencies because of the pathogenic organisms, such as *Pastuerella multocida*, introduced into the tissues. Bite wounds should be flushed with warm saline or saline with chlorhexidine. Care should be taken when flushing holes that might communicate with the coelomic cavity. Birds should be placed on systemic antibiotics with activity against *P. multocida*. The penicillins (clavamox 125mg/kg po bid) or fluoroquinolones (enrofloxacin 20mg/kg po sid) are often used. Birds suffering from bite wounds may present in shock and should be treated as such with IV fluids, colloids and warmth. It may be difficult to locate bites wounds within the feathers. For this

reason, it is recommended to place any bird with potential dog or cat bite wounds on systemic antibiotics.

Crop burns occur most often in birds being hand fed formula that is too hot. The formula is often heated by a microwave oven, producing small pockets of formula that are very hot but not noticed when the food is tested. Cases of crop burn have also been seen in adult birds fed coffee and other hot foods. The burn affects the crop wall and overlying skin and causes initial erythema, which may not be noticed by the owner. As the crop wall and skin fuse and necrosis occurs, a fistula is formed. The first sign noted by most owners is the formula (or food) passing through the newly formed fistula.
Crop fistulas are not true emergencies unless the fistula is very large. Smaller, more frequent feeding may be used initially to maintain the bird's weight until the fistula is fully developed. Antibiotics can be used to prevent secondary infection (TMS 30mg/kg po bid). If the fistula is very large and oral feeding causes extensive leakage, the food may be placed directly into the proventriculus through the fistula using a soft red rubber catheter. Smaller amounts should then be used (approximately 1/2 of the volume of the crop). If the bird is thin, weak or appears otherwise malnourished, supportive care (hospitalization, fluids, gavage feeding, antibiotics, analgesics) should be initiated until the bird is capable of undergoing surgery. The surgical procedure involves initial separation of the crop from the skin then incorporates a two-layered closure of the crop. The skin is closed with a simple continuous pattern.

Blood feathers are actively growing new feathers that still maintain a blood supply as they grow. If the portion of the feather that is still growing is damaged it will bleed and can bleed profusely. This is often a frightening problem for the owner but birds rarely bleed to death from these. Classic treatment is to remove the feather at the base to stop the bleeding. The feather is grasped at the base and pulled sharply in the direction it is growing. This should stop the bleeding but may also damage the feather follicle, making new feathers more prone to malformation. Another option is to stop the bleeding at the point of trauma using hemostatic agents and direct pressure, leaving the growing feather intact. This usually maintains the viability of the follicle and stops the bleeding.

Unilateral lameness, especially in budgies and other small birds, is often the result of a renal or gonadal tumor placing pressure on the sciatic nerve as the nerve passes between the kidney and the pelvis. This change can often appear spontaneously but is the result of a chronic problem. The bird still maintains use of the femoral nerve so has an active withdrawal but is unable to move the rest of the leg or perch with the foot. Diagnosis is often based on history and PE findings but radiographs can occasionally show organ enlargement. Treatment is supportive and the prognosis is poor.

Egg-binding

Dystocia can be a serious event in birds. A bird presented for egg binding should be placed in a warm, humid environment while a history is taken. Pertinent information includes: age, previous egg laying activity, history of egg binding, and diet. Many birds presenting for egg binding are chronic egg-layers that have insufficient calcium stores necessary for uterine contractions. The calcium deficiency may be the result of continual egg production and/or poor dietary calcium intake. Another common presentation is the older bird that randomly decides to lay the first egg and the egg is too large to pass through the cloaca. Once a history is taken, the patient should be examined quickly to establish the presence and location of the egg, as well as general condition. If ascites is present, peritonitis or neoplasia should be suspected. If an egg is palpable within the pelvic canal, supportive therapy may be initiated. This includes calcium gluconate at 50-100mg/kg IM, SQ fluids, and tube feeding. Before using

hormones, the cloaca should be examined for strictures or other problems and lubricated to facilitate egg passage. Many cases of egg biding can be resolved with calcium therapy and supportive care.

If no egg is passed in a 8-12 hours, then radiographs are recommended to check for multiple eggs, egg size, and other factors that could change the treatment plan. Anesthetizing birds for radiographs also gives an opportunity to palpate the relaxed abdomen. If the bird is stable under anesthesia, this opportunity can be taken to remove the egg manually or via ovocentesis to collapse the egg. Manual expression involves gentle, slow manipulations of the egg through the oviduct and out the cloaca. Manual pressure should be intermittent to allow the bird to breath normally between pushes.

Ovocentesis should be done by directly visualizing the egg through the cloaca and oviductal opening. If pressure is applied on the egg towards the cloaca, direct visualization can usually be achieved. If the egg can be visualized through the oviduct, the contents can then be aspirated and the egg collapsed. A 22-20 gauge needle on a 6-12cc syringe is used to aspirate the egg contents. If the egg cannot be collapsed, larger needles can be used. Small cotton swabs should be used to lubricate around the egg before any attempt is made to remove it. If the egg cannot be directly visualized, the egg contents can be aspirated percutaneously. There is more risk with this method, therefore is should only be used if absolutely necessary. The egg should collapse after aspiration of the contents, if not, gentle pressure applied to two sides of the egg should cause it to collapse. There is some risk associated with the sharp edges created by ovocentesis, therefore, every effort should be made to remove the egg whole before attempting this procedure. Once the egg is collapsed, slide a Q-tip into the hole made by the needle and try to use this to pull the collapsed egg from the oviduct. If the egg is collapsed, but unable to be removed, the bird can be recovered and allowed 12-24 hours to pass the collapsed egg. If the egg is still not passed, abdominal surgery and salpingohysterectomy should be performed.

Respiratory Disease

Respiratory emergencies are common in avian practice and may be caused by a primary respiratory disease or secondary to other diseases. Primary respiratory diseases include infectious disease, inflammatory (allergic) problems, toxic insults, foreign bodies and neoplasia. Secondary respiratory disease is usually caused by abdominal distension, abdominal masses, and thyroid enlargement.

Upper respiratory diseases may occasionally present as a respiratory emergency because of open-mouthed breathing and increased respiratory rate. In these cases, remove any nasal plugs that occur and submit them for bacterial and fungal culture/sensitivity and histology. Cytology slides can be made for quick analysis of the type of bacteria and/or fungus present. Treat the bird with topical and systemic antibiotics and antifungals (if warranted) until the results of the cultures are obtained. Nasal flushes may be therapeutically and diagnostically beneficial. Collect the flushed solution in a sterile container for cytology and culture.

Birds with respiratory signs from abdominal distension usually show tachypnea with short, shallow respirations because of the loss of air sac volume. The distension may be due to abdominal masses, ascites or an egg. Treatment should include removing any fluid present for cytology and culture. This will often greatly ease the respiratory burden on the patient. The bird should be placed in an oxygen cage and kept quiet and calm. If an egg is present, stabilize the patient, anesthetize and remove it when the bird is stable. If a mass is present then surgical removal is often the best choice to relieve the respiratory distress.

Large air way disease is usually caused by tracheal foreign bodies, granulomas or thyroid enlargement. These birds usually present with a history of voice change, exaggerated respiratory click and open-mouthed breathing. These birds should be placed in oxygen as soon as possible until they are calm. Terbutaline (0.1mg/kg IM BID) may be of some use but often an air sac cannula must be placed. Tracheal or coelomic endoscopy and radiographs should be used to identify the problem and develop a treatment plan.

Small air way disease is usually caused by inhaled toxins, such as smoke or Teflon, or allergies. These birds are difficult to manage. They present open-mouthed breathing with a wide-based stance, wings abducted and an expiratory squeak. Oxygen and IM terbutaline should be administered. If allergic disease is suspected, also give diphenhydratmine (2mg/kg IM) and meloxicam (1mg/kg SC). If these do not relieve the distress, intubation and nebulization with terbutaline (01mg/kg in 9cc NaCl) via IPPV has been beneficial in some cases. Radiographs may show hyperinflated air sacs but are often normal.

Parenchymal disease can be caused by infections, pulmonary neoplasia or congestive heart failure. These birds present with increased respiratory rate and effort (as evidenced by tail bob) but are rarely open-mouthed breathing as this is often an acute manifestation of a chronic disease. Treat these birds with oxygen and other supportive care as deemed by a physical examination. A thorough history is important in determining the cause of the disease but radiographs and endoscopy with air sac and lung biopsies are usually warranted.

FERRETS

Insulinomas

Functional tumors of the pancreatic beta-cells in ferrets are common. These insulinomas may cause ferrets to present on emergency either at initial diagnosis or when there is an acute episode of tumor growth that results in an insulin surge. The resulting hypoglycemia causes ferrets to become weak, glassy-eyed, drool, exercise intolerant or have hypoglycemic seizures. The presumptive diagnosis can be made with a point-of-care glucometer. Less than 60mg/dl is highly suggestive of this problem, especially when no other cause of the hypoglycemia can be found. A definitive diagnosis requires concurrent insulin levels. Treatment is with prednisolone (0.25-1mg/kg initially, up to 2mg/kg long term) either IM or PO depending on the patient. Feeding the ferret a high fat/high protein meal will also help to improve the glucose level with a minimal insulin spike that occurs when oral dextrose is given. Severe hypoglycemia may require intravenous fluids with dextrose (2.5-5%). If the ferret is currently on medication then I usually recommend increasing by 25%, if the ferret is not currently on medication, I start with prednisolone at 0.25-0.5mg/kg PO BID. Diazoxide can be added if signs occur while already on a high dose of prednisolone.

Urinary Tract Disease

Ferrets with adrenal tumors may also present on emergency because of prostatic enlargement causing partial to complete urinary obstruction. These ferrets have a history of straining to urinate, frequent, small urinations that leads to lethargy and depression as azotemia develops. The onset is usually gradual if the owners are especially astute. Other signs of adrenal disease include symmetrical hair loss and aggression. A turgid, painful bladder may be palpated and the enlarged prostate is rarely palpable in the caudodorsal abdomen. Presumptive diagnosis is based on history and clinical signs. Ultrasound examination will show the enlarged prostate and enlarged adrenal gland. Prostatic cysts or abscesses can also occur with chronic disease. Initial treatment is to place a urinary catheter using a 3.5Fr red rubber catheter (use a sterile guitar string to pass this catheter by the *os penis*) or a Slippery Sam® catheter. The catheter should be sutured in place to avoid removal upon waking. The ferret should then

be given intravenous or subcutaneous fluids depending on the azotemia and antibiotics if an abscess is present. Treatment of the adrenal disease with leuprolide acetate (Lupron® 100ug IM monthly), deslorelin (Suprelorin F® implant yearly) or surgery.

Stranguria or dysuria with blood in the urine is more likely to be from a urinary tract infection or stones. Urinary tract infections can occur with adrenal disease or stones and occasionally as a discrete entity. Gram negative bacteria are most common. Struvite stones occur in ferrets from using plant-based protein diets. Cystine stones have been occurring more frequently in ferrets and are thought to be metabolic in nature. Diagnosis is with palpation, radiographs or ultrasound and urinalysis. Treatment of bacterial cystitis is based on culture and urolithiasis is treated with surgery. Urinary acidifiers may work if there are only struvite crystals and high pH. Diuretics such as hydrochlorothiazide (2mg/kg po bid) can be used for cystine stones.

Anorexia and Vomiting

Gastric foreign bodies are common in ferrets because of their inquisitive nature. Foreign bodies are usually soft, rubber or plastic items, especially in young animals but trichobezoars are more common in older ferrets. Clinical signs include anorexia, bruxism, hypersalivation, cranial abdominal pain, diarrhea, and melena. Vomiting is more common with gastritis than with foreign bodies but can occur when there is complete obstruction by the foreign body. Diagnosis is with plain or contrast radiography or ultrasonography. Treatment is with surgical or endoscopic removal. Treatment for gastritis is recommended following removal of the foreign body as outlined below.

Gastritis and gastric ulcers are common in ferrets with a variety of diseases causing them to present on emergency. Ulcers may be secondary in a foreign body, due to infectious disease, toxin ingestion, and azotemia or induced by anti-inflammatories or stress. Any cause of anorexia in ferrets may cause gastritis as well due to the presence of Helicobacter. Clinical signs include lethargy, anorexia, bruxism, hypersalivation, vomiting and melena. The diagnosis is based on clinical signs and finding one of the causes listed above. Baseline diagnostics, such as radiographs, CBC and chemistry panel can help to elucidate many of these causes. Ultrasonography can show thickened stomach and GI tract as well as regional lymph node enlargement. FNA of the enlarged lymph nodes may reveal changes such as reactive nodes or the presence of eosinophils or plasma cells that help confirm the diagnosis. Definitive diagnosis requires endoscopic or surgical biopsy, but this disease is usually treated based on clinical suspicion. Treatment is aimed at relieving the hyperacidity with ranitidine(3.5mg/kg q12h), famotidine(0.5mg/kg q24h), cimetidine (10mg/kg q8h) or omeprazole (2.5-5mg/kg q24h). Gastric protectants, such as bismuth subsalicylate (1ml/kg q8h) or sucralfate (100-200mg/kg q12h) are also used. Supportive care, such as intravenous fluids and assist-feeding are also helpful. If ferrets are vomiting, they can be held off food for 12-24 hours. These ferrets should be kept on IV fluids with dextrose in case of concurrent insulinoma. Broad spectrum antibiotics, especially those that are effective against Helicobacter such as amoxicillin and metronidazole, are also used.

Diarrhea and Cachexia

Chronic or acute diarrhea may cause ferrets to present on emergency because of weakness and lethargy associated with electrolyte disturbances. Chronic disease can result in weight loss and cachexia that will cause a patient to present for emergent treatment. Acute diarrhea can be from bacterial enteritis caused by *Salmonella* spp., and *Campylobacter jejuni*. Clostridial organisms are often seen in low numbers in feces but have been associated with enteritis and colitis when present in large numbers and are sporulated. Salmonellosis is a contagious disease in ferrets characterized by fever, bloody diarrhea, and lethargy. The incidence in pet ferrets is low and the infection maybe associated with feeding

uncooked meats. *C. jejuni* has been associated with diarrhea and enterocolitis in many species, but can be found in the feces of normal ferrets, so the significance as a pathogen in ferrets is unknown. Diagnosis of bacterial enteritis is with fecal cultures. Treatment is with supportive care and broad-spectrum antibiotics.

Chronic diarrhea is usually the results of inflammatory bowel disease or viral infections. Inflammatory bowel disease is relatively common in adult ferrets and can progress to cause severe dehydration and lethargy. Clinical signs include loose stools, intermittent nausea, weight loss and occasionally vomiting. The cause is unknown but may be related to dietary intolerance, hypersensitivity reactions or aberrant immune response. The inflammation is usually lymphoplasmacytic but can also be eosinophilic enteritis. Definitive diagnosis requires gastric and intestinal biopsies, but a suspected diagnosis can be made based on clinical signs and history to rule out exposure to ECE, toxins, etc. Abdominal ultrasound may show thickened bowel and enlarged lymph nodes. Aspirates of these lymph nodes reveal inflammation (as in gastritis above). Routine blood work may reveal lymphocytosis, elevated liver enzymes and hyperglobulinemia. Treatment is aimed at controlling the immune response and dietary management. Prednisolone and azathiaprine are treatment options. Hypoallergenic diets (novel protein or hydrolyzed protein) used in cats are also indicated.

Coronavirus can cause severe diarrhea, especially in younger and older ferrets. This disease is highly transmissible and is often brought into a group of ferrets by an asymptomatic juvenile animal. Clinical signs begin 2-14 days after introduction of the new ferret or exposure through fomites and include anorexia, vomiting, green or mucoid diarrhea, melena, dehydration, lethargy and weight loss. The virus causes blunting of the intestinal villi and consequent maldigestion and malabsorption. Definitive diagnosis is requires PCR of feces or blood (Michigan State University) but a presumptive diagnosis can be made from history and clinical signs. Treatment is supportive with fluids, nutritional support, broad spectrum antibiotics, and gastrointestinal protectants.

Lawsonia intracellularis can cause a proliferative bowel disease, especially in younger ferrets. Signs include diarrhea, weight loss, and rectal prolapse. Definitive diagnosis requires culture of a rectal biopsy. Treatment is with chloramphenicol (25mg/kg po q12h) for 14-21 days.

RABBITS

Anorexia

Dental disease is a common cause of anorexia in rabbits. All teeth in rabbits are open-rooted and grow 10-12cm/year. Incisor malocclusion is usually noted by the owners earlier and is not often an emergency presentation but malocclusion of the cheek teeth is harder to recognize. These rabbits may appear hungry but drop food or stop chewing quickly. Points occur most commonly on the buccal surface of the upper arcades and lingual surface of the lower arcades because of the jaw alignment. The cheek teeth may be examined using an otoscope or speculum in many awake rabbits but sedation or anesthesia may be required for a thorough view. Treatment is by occlusal adjustment with a low-speed dental drill or hand files. Points may be removed with ronguers. The rabbits are then supported with meloxicam, fluids and assist-feeding until eating on their own.

The most common problem associated with anorexia in rabbits is gastric stasis which is often related to diets low in indigestible fiber or high in carbohydrates. Rabbits may also develop stasis secondary to stresses such as changes in the diet or environment or dental disease. Other clinical signs include dehydration and the formation of small, hard fecal pellets. The rabbits are typically bright and alert despite these problems, unless the stasis has been prolonged. A firm, dough-like stomach may be

palpable on physical examination. Radiographs usually show a soft tissue mass in the stomach surrounded by a halo of gas and excess gas in the intestines. Treatment for gastric stasis is rehydration, force-feeding, and motility modifying drugs. Rehydration of the patients as well as the stomach contents is important in the treatment of gastric stasis. Subcutaneous fluids (50-100 ml/kg/d) can be administered in the hospital or at home by the owner. Oral fluids, such as water or electrolyte replacement mixture, should also be given using a syringe. This helps to rehydrate the stomach material for easier passage. Force-feeding the patient a high-fiber diet, such as Critical Care Formula (Oxbow Products), mixed vegetable baby food, canned pumpkin, or soaked alfalfa pellets will help to stimulate gastric motility and place the animal in a positive nitrogen balance. Additionally, fresh greens and timothy or grass hay should be available for the rabbit to eat on its own. Metoclopramide (0.5 mg/kg SC q 8 hr) or cisapride (0.5 mg/kg PO q 12 hr) are used to stimulate gastric and intestinal motility. Radiographs are recommended before using these drugs to rule-out gastric obstruction. Most rabbits respond to this medical care in 1-2 days, although occasionally rabbits may take longer to return to normal food intake. Surgical treatment for gastric stasis is rarely indicated.

Acute gastrointestinal obstruction can occur in rabbits and can be caused by a hairball, foreign body, or pyloric disease. These rabbits usually present extremely depressed, weak, and may be in shock (T<100F). Treatment of the metabolic condition and decompression of the stomach are necessary if these rabbits are to be saved. The stomach tube is passed to remove excess gas and fluid and then flush the stomach. A large red rubber tube or other large bore tube works well for this. The rabbit should be sedated or anesthetized (usually via mask with isoflurane or sevoflurane) unless the animal is severely recumbent. Be prepared for a drop in heart rate while passing the tube over the glottis, this usually resolves once you get past this area. Once the tube enters the stomach, remove as much gas and fluid as possible with gentle palpation and gentle suction. Then flush the stomach with warm (100^{o}F) water until it is clean or blood is noted. This often dislodges the pyloric/duodenal blockage and no further treatment of the GI tract is needed. Metabolic disturbances caused by the blockage still need to be treated aggressively with IV fluids, colloids and monitoring and correcting blood gas and electrolyte abnormalities and blood pressure changes. Radiographs and ultrasonography are useful in making this diagnosis and determining the presence of foreign body or pyloric disease. Syringe feeding small amounts (3-5ml every 2 hours) of high-fiber diets have been shown to be helpful in the medical aspect of GI obstruction. Keep the rabbit on IV fluids with dextrose, then slowly increase the feeding amount and decrease the frequency. The prognosis is guarded with 50-60% survival. Surgical treatment with removal of the foreign body have been used after gastric flushing with about 50% overall success rate. A more recent paper used subcutaneous fluids, subcutaneous metoclopramide and feeding 2-3ml every two hours with an 80% success rate. We have tried this on a few rabbits with success.

Torticollis

Acute head tilt in rabbits can be caused by peripheral or central vestibular disease. Peripheral disease is usually caused by head trauma or otitis media from bacteria such as *Pastuerella, Staphylococcus* and *Streptococcus* that travel through the Eustachian tube. Central disease can be caused by *E. cuniculi*, cerebral larval migrans, and trauma. Clinical signs are similar and include head tilt, circling, rolling and nystagamus. The nystagamus is generally vertical with central disease and horizontal with peripheral disease. Diagnosis of peripheral disease can be with radiographs or CT scan. Central disease is more difficult to definitively diagnose although a negative *E. cuniculi* titer can help rule that disease out. Treatment is with antibiotics for otitis, usually enrofloxacin (20mg/kg PO SID) or penicillin (60,000 units SC EOD). *E.cuniculi* is treated with albendazole or oxybendazole (20mg/kg PO SID). Cerebral larval migrans are treated with ivermectin. Anti-inflammatories are helpful in all these cases. Usually meloxicam (1mg/kg PO SID) but in severe cases prednisolone (1mg/kg PO BID, tapering dose) has been

used. Meclizine (12.5-25mg per rabbit PO BID) can help with rolling and nausea. A solid flooring with good traction and padding on the sides of the cage can help rabbits that are rolling. Syringe feeding may be necessary initially. Prognosis is guarded if rolling continues. Many rabbits will have a permanent head tilt but learn to walk normally.

Respiratory Disease

Rabbits with respiratory disease may present on emergency with either upper or lower respiratory disease. URD is commonly called "snuffles" because of the sonorous respirations heard over the nasal cavity and trachea. Affected rabbits may be lethargic and inappetant. Mucopurulent discharge may also be found on the nose or medal aspect of the forearm as the rabbit grooms its face with the forepaws. Conjunctivitis may occur if the organism travels up through the nasal-lacrimal duct. A serous nasal discharge can occur with allergies and irritations but is not usually mucopurulent. Treatment is based on deep nasal culture and sensitivity with topical and systemic antibiotics. Ophthalmic drops can be used intra-nasally or placed in the eye to travel through the nasolacrimal duct into the nasal passages. Nebulization of antibiotics and mucolytics may be helpful in severe cases.

Pneumonia has few distinct clinical signs in the early stages of the disease. Typically, affected rabbits initially exhibit non-specific signs such as weight loss, anorexia, and lethargy. Dyspnea and tachypnea are not typically seen until there is severe loss of respiratory function. Rabbits showing dyspnea generally have a poor prognosis unless managed intensively. Auscultation may reveal areas in which lung sounds are absent because of lung consolidation or abscesses. Radiographs are diagnostic. Lung aspirates for culture and sensitivity can be performed. Treatment is with systemic antibiotics and nebulization of antibiotics, mucolytics and potentially bronchodilators. Long-term therapy is often required.

Thymomas can occasionally cause signs of lower respiratory disease because of the space-occupying affect. More typical signs include hiccups and bulging eyes. Palpation of the cranial chest cavity can show decreased compliance but radiographs are most helpful in showing the cranial mediastinal mass. Ultrasound-guided aspirates can show lymphocytes and epithelial cells to help confirm the diagnosis. Removal of cystic fluid can be beneficial to the patient. Treatment is with radiation therapy or surgical removal. Prednisolone (1mg/kg po bid) has been used palliatively.