INTRODUCTION
As the popularity of reptiles has grown, so has the demand for quality veterinary care. Today, reptile medicine represents a viable subset of companion animal practice. Reptiles are stoic and have evolved to mask signs of illness, which makes them a challenge to diagnose and treat. For veterinarians and technicians who are willing to become proficient, however, reptile practice offers many rewards.

SNAKE PROCEDURES
Capture and Restraint
Snakes are easy to hold because there is only one dangerous part to restrain. When approaching a snake you do not know, the head may first be covered with a towel. This has the dual effect of blocking his view of the handler and providing him a sense of security. Restrain by grasping behind the head with one hand and supporting the body with the other.

Snakes do not have complete tracheal rings, so use caution that asphyxiation does not occur. Some colubrid snakes will emit a musky paste from the scent glands as a defense mechanism. Snakes that are used to being handled are much less likely to bite. Bites from non-venomous species, if they occur, are generally harmless. Use the buddy system when dealing with giant or venomous species.

Basic Exam Skills
Weigh the patient (in grams) at every opportunity. A decline in weight may be the only clue that there is a problem developing. Assess body score by examining the dorsolateral musculature. Check for mites or ticks in the gular groove between the mandibles and under the edges of scales. Examine the heat pits and eyes for mites. Look for retained skin especially on the head and spectacles. During the oral exam, look for petechiation and abscessation. Check the tongue and glottis for swelling, symmetry, and inflammation. Observe snakes for righting reflex and positional nystagmus when the head is moved side to side. Palpate the abdomen for masses and ova.

Sexing via Hemipenal Probe
Use a lubricated blunt probe (e.g. ball-tipped feeding needle, urinary catheter). Pass the probe cranially under the lateral edge of the vent flap, then flip the probe to aim it caudally. Direct the probe into a longitudinal canal that runs paramedian along the tail. If the probe passes 3 caudal scales (into the scent gland) it is female, if it passes 7-10 caudal scales (into the hemipenal canal) it is a male.

Venipuncture
**Ventral tail vein:** caudal to the cloaca with the snake in dorsal recumbency. A 5/8-1”, 21-25ga needle is angled at 45° craniodorsally, between caudal scales. Apply slight negative pressure once you are under the skin. If the needle hits a vertebral body, withdraw slightly and redirect. (Best for large snakes)

**Cardiocentesis:** restrain the snake in dorsal recumbency, watch for heartbeat, and locate the heart ¼ to ⅓ of the way down the body. Steady the heart with fingers and advance a 5/8-1.5”, 21-25ga needle at a 45° angle craniodorsally, under a scute, into the apex/ventricle. Blood usually enters the syringe with each beat. (Best for small snakes)

**Palatine vein:** paired veins run along the roof of the mouth. Ideal for IV injections while under sedation. Use a 25 ga., 5/8” needle on a 1 cc syringe. Pre-bend the needle slightly for easier approach. Place pressure on the vein after withdrawing to avoid hematoma. (May require sedation)

**Tracheal Wash**
Tracheal wash is the preferred method for sampling of respiratory pathogens. With the snake’s head elevated, a sterile urinary catheter is placed through the glottis while the patient takes a breath. Then, 3-10 ml/kg of sterile saline is injected. The snake is quickly tilted downward, and the fluid is aspirated back into the syringe. Because of the simple anatomy of the reptile lung, samples collected in this manner may actually be lung washes. Wet
mount cytology of tracheal wash samples may reveal lung worm (e.g. *Rhabdias*, pentastomid) larvae or ova. Anaerobic and aerobic culture and sensitivity are also performed.

**Fluid Therapy**

Maintenance fluid rate for most reptiles is 15-25 ml/kg/day, and up to 5% of body weight may be given in a single dose if indicated. Subcutaneous or intracoelomic fluid administration is utilized in the majority of cases. In snakes, SQ fluids can be given along the lateral folds. ICo fluids are given ventrolaterally a short distance cranial to the vent, using care not to enter the caudal extent of the lungs.

IV catheters can be placed in most reptiles however a cut-down approach is usually necessary. The preferred site for snakes is the right jugular vein. A cut-down incision is made from 4-7 scutes cranial to the heart, at the junction of the ventral scutes and lateral body scales. After the vein is isolated, a catheter is placed in the usual manner and secured using tape, suture, and/or tissue adhesive.

Reptiles are slightly hypotonic when compared to birds and mammals. To prepare “Reptile Ringers Solution”, mix 2 parts Dextrose 2.5%/Saline 0.45% with 1 part lactated Ringer’s solution (or 1 part Dextrose 5%, 1 part Saline 0.9%, and 1 part Ringer’s).

**Tube-Feeding**

Snakes frequently present for lack of appetite. In some species (e.g. ball pythons) this can be considered a normal, seasonal occurrence. In others it may be attributed to stress or disease. Often, no abnormality can be found on physical examination or fecal testing. Force-feeding provides nutritional support for these patients while the clinician seeks to diagnose the problem and correct husbandry. In the majority of cases, force-feeding will stimulate a snake’s appetite. Medications (e.g. metronidazole, fenbendazole) are frequently added to the mixture to “shotgun” the problem before resorting to further diagnostic tests.

Oxbow Carnivore Care is used for this purpose. Mix to the desired consistency. Give 2.5-5% of body weight using a catheter-tipped syringe and lubricated 14-fr. red rubber catheter. A snake’s mouth can be opened with a rubber spatula or similar speculum. Its glottis will be located far cranially in the mouth and is easy to avoid. Gently massage the food caudally as it is given. After feeding, hold the snake’s head elevated for several minutes. Once he is moving forwards in a serpentine fashion it is ok to release him.

**CHELONIAN PROCEDURES**

**Capture and Restraint**

Turtles are generally easy to handle safely. Shy turtles may be encouraged to come out by pressing the rear limbs up into the inguinal fossae. Grasp the turtle’s head behind the jaws to keep it from retreating into the shell. This may be easiest to do from below: turtles are more wary of threats from above than from below. A box turtle can be preventing from closing its shell by keeping a forelimb pulled out. Another trick is to place a turtle on a pedestal, such as a jar or pill bottle, so that his plastron is above the table surface and feet cannot touch. This will often encourage the turtle to extend its neck, head, and limbs. A dental pick or similar instrument can be hooked under the beak in order to apply traction when pulling the head out of the shell. Tranquilization is often required to examine and perform diagnostics in larger species.

**Basic Exam Skills**

Weigh the patient in grams. An underweight turtle will “feel” lighter than it should. Evaluate scutes and skin for lesions. Examine each tympanum for ectoparasites or abscessation. Look for nasal discharge or ocular swelling. If an oral exam is possible, look for stomatitis.

**Sexing**

Many cheelionians exhibit secondary sex characteristics. Male box turtles have red irises; a female’s irises are brown. The plastron of most male land turtles is slightly concave, while the plastron of females is flat. In most chelonian species, the male’s vent is located near end of the tail, beyond the edge of the carapace. For females, the cloacal opening is near the tail base, even with the edge of the carapace. Male aquatic turtles have longer nails on the front feet and are smaller than females.

**Venipuncture**

*Dorsal tail vein*: a 5/8”, 23-25ga needle is placed as cranial as possible on the dorsal midline of the tail, angled at 45-90°, and advanced while maintaining slight negative pressure. If the needle hits a vertebral body, withdraw
slightly and redirect cranially or caudally. Lymphatic contamination is possible.

**Jugular vein:** position a 5/8”, 22-25ga needle laterally, caudal to the tympanum, and direct it caudally, midway down the neck. Best site for blood uncontaminated with lymph. Hold off site to avoid hematoma. The right jugular is larger than the left.

**Subcarapacial vein:** push the turtle’s head inside the shell, and place a 5/8-1.5”, 22-25ga needle on the midline, dorsal to the neck, through the skin just caudal to the cranial rim of the carapace. Direct the needle in a dorsal direction, aiming toward the junction of the cervical and thoracic vertebrae. Blood from this sinus is frequently mixed with lymph. Apply pressure to avoid a hematoma.

**Brachial vein:** using a 5/8-1”, 25-22ga needle, aim superficially in the sulcus behind the elbow joint (yes, it points backwards!) on the front limb. A similar approach is used behind the stifle joint to access the popliteal vein on the hind limb. Both approaches are blind sticks, and samples are frequently mixed with lymph. (Easiest on large turtles)

**Fluid Therapy**
Subcutaneous fluids can be administered into any accessible fold of skin (e.g. ventral neck fold, inguinal fold). Intracoelomic fluids can be given into the inguinal fossa, cranial to the rear leg. Use sterile technique and be careful not to inject the air sacs at the cranial extent of the fossa, or to hit blood vessels along the ventral extent of the fossa. The preferred site for placing an IV catheter in chelonians is the right jugular vein. An intraosseous catheter can be placed into the plastrocarapacial bridge. Radiographs will confirm proper placement.

**Nutritional Support**
Chelonians occasionally take food from a syringe, but are usually uncooperative. The glottis is found at the base of the tongue. To avoid aspiration, keep hand feeding formulas thick. Tube-feeding can also be used, but it can be very difficult to get the turtle’s head out and to open the beak. Anorexia in turtles usually requires days to weeks therapy to resolve. To avoid the stress and frustration associated with repeated treatments, pharyngostomy tubes are typically employed.

**Pharyngostomy tube placement:** Pre-measure tube length and mark the tube. Anesthetize the turtle, and place him in sternal recumbency. Place a curved hemostat into the pharyngeal cavity, and press the tip firmly outward against the right side of the neck. Make the exit site close to the edge of the carapace so that tube movement is minimal as the head moves in and out of the shell. Make a small incision over the hemostat tips. Push the hemostat tips through the incision and grasp the tip of the feeding tube. Pull the tube out the mouth, up to the premeasured mark. Lubricate the tube, redirect the tip of the tube in the hemostats, and feed it down the esophagus into the stomach. Secure the tube using a purse-string suture and a Chinese finger trap knot or tape butterfly/sutures (4-0 PDS). Cap the tube end, and secure the tube over the carapace with tape.

Chelonians are usually fed every 24-48 hrs. The volume to be fed may vary between 2.5-5% of body weight daily, depending upon the species, diet selection, and the patient’s condition. Oxbow Critical Care or Carnivore Care, liquid enteral formulas (e.g. Ensure, Sustecal), psittacine hand feeding formulas, and fruit, vegetable, or meat baby foods can be used.

**LIZARD PROCEDURES**
**Capture and Restraint**
Crocodilians can be safely handled once the mouth is taped shut. A speculum is usually taped in place if procedures such as tube-feeding or endoscopy are planned. The eyes may be covered in order to reduce stress. Iguanas and many other species can be restrained by applying firm digital pressure over the eye sockets (vaso-vagal response). Lizards become calm within seconds, and the heart rate and respiration become slower. Trim the nails prior to examination to avoid injuring staff. Large species may be handled using a towel. Grasp most species behind the head and at the tail base. Be careful not to induce tail shedding in lizards.

**Basic Exam Skills**
Always weigh the patient. To auscult the thorax, use a wet gauze pad over the diaphragm of your stethoscope. This trick absorbs much of the scratching associated with reptile scales. Place the bell directly between the forelimbs to listen to the heart. Use a warm water bath to stimulate defecation and obtain a fecal. Palpate the hemipenal bulges and examine the vent for retained hemipenal plugs. Note muscle tremors or tetany. Assess body score by
examining the tail base. Look for retained skin especially on the head and digits.

Sexing
Mature male green iguanas have larger femoral pores than females do. They can be probe-sexed in a manner similar to snakes. Male bearded dragons and leopard geckoes also have larger femoral pores than females do. These two species can also be sexed by “popping” the hemipenises: carefully exerting thumb pressure in a cranial direction along a hemipenal bulge. Crocodilians are sexed by examining the vent: the penis/elitoris is located on the ventroposterior surface of the cloaca near the vent.

Venipuncture
**Ventral tail vein:** with the lizard held in dorsal recumbency, place a 5/8-1.5”, 21-25ga needle on the ventral midline, approx. ¼-⅓ of the way down the tail. Advance the needle at a 45°- 90° angle craniodorsally, while maintaining slightly negative pressure. If the needle hits a vertebral body, withdraw slightly and redirect cranially or caudally. A lateral approach to this vessel can also be used, and has the advantage of keeping the patient in sternal recumbency.

**Ventral abdominal vein:** lies 1-2 mm within the coelomic cavity on ventral midline between the umbilical scar and the pelvic inlet. This vein can be visualized by transilluminating smaller species (leopard geckoes, fat-tailed geckoes, etc.). Obviously, there is a risk of iatrogenic injury to viscera when doing a blind stick.

**Supravertebral vessel:** located caudal to the occiput, dorsal to the spinal cord. Prep the area as for surgery. Use a 5/8-1.5”, 21-25ga needle. The approach is perpendicular, on the midline, caudal to the occiput. Apply suction and advance the needle until it enters the sinus and blood appears in the syringe.

Fluid Therapy
Subcutaneous fluids can be given into the lateral folds and over the shoulders. An IV catheter can be placed into the cephalic vein using a cut-down approach. This vein is located on the dorsomedial surface of the antebrachium. IO catheters can be placed into the femur, humerus, and proximal tibia.

Cloacal Wash
A warm water soak will stimulate defecation in many lizards and land turtles. With care, fecal samples can be manually expressed from snakes. Alternatively, cloacal and colon washes can be performed in most reptiles. A large gauge catheter is inserted into the vent (and further, into the colon, if desired), 10 ml/kg sterile saline is injected, the abdomen is gently massaged, and then the fluid is withdrawn. This procedure will stimulate voiding in many cases. Sample is used for a wet mount, floatation, and culture if indicated.

Nutritional Support
Lizards are usually syringe fed. Some lizards will readily open their mouths if the commissures are stroked. If this does not work, then pressure can be applied over the orbits (vaso-vagal maneuver) while simultaneously pulling downward on the intermandibular skin. Beyond the glottis (located at the base of the tongue) is the large pharyngeal cavity. Lizards can also be tube fed. Feeding regimens are typically q12-24 hrs.

Herbivorous reptile species (tortoises, iguanas, Uromastyx, etc.) need a formula high in fiber, quality protein, and carbohydrates, but low in fat. A timothy hay-based high fiber gruel (Critical Care, Oxbow Animal Health, 800-249-0366), fruit or vegetable baby foods, and liquid enteral formulas (e.g. Ensure, Ensure Plus, Sustecal) should be used for nutritional support. For carnivorous reptiles, a gruel made from high quality kitten food and liquid enteral formula (“Duck Soup”) is used. Hill’s A/D can be used for this purpose, but it is formulated from liver and may promote gout in cases with dehydration or renal disease. Oxbow Carnivore Care is an egg and chicken-meal based powdered formula for syringe feeding. Omnivorous reptiles (e.g. box turtles, bearded dragons) are fed a combination of the ingredients listed above. The volume to be fed may vary between 2.5-5% of body weight daily, depending upon the species, diet selection, and the patient’s condition.

Injections
Because of the renal portal system, most injections are given in the front half of the body. Be aware that many commonly used injectable medications can cause tissue necrosis IM or SC. Intravenous injections are technically more demanding in reptiles. The same sites used for venipuncture can be used. Intracardiac is usually reserved for situations where there is no other option. Reptiles do not have much excess skin when compared to mammals. Give SC injections along the lateral fold in snakes and lizards, the shoulders in lizards, and by shallow needle-
insertion into the loose skin surrounding the limbs in chelonians. IM injections are made into the epaxial musculature of snakes, the limbs, shoulders, and epaxial muscles of lizards, and the limbs and pectoral muscles of chelonians. Pectoral muscles are accessed by inserting the needle parallel to the plastron under the leg. In all species, avoid drugs that require large injection volumes IM. ICo injections are given in the middle of the caudal quarter of the snake’s body. For lizards, ICo injections are given into the right caudal quadrant, cranial to the rear limb. Aspiration will help assure that you are not in the bladder. For chelonians ICo injections are given in the flank, near the junction of the skin with the shell, just cranial to the rear leg. Again, aspirate prior to injection in order to make sure you are not in the bladder.

REFERENCES
Harris, DJ, Johnson DH. Avian and Reptile Clinical Techniques Laboratory. Southeast Veterinary Conference, Myrtle Beach, SC 2004.

AVIAN MEDICAL PROCEDURES

INTRODUCTION
No matter your comfort level with other species, birds present special challenges owing to their unique anatomy and physiology. There are more similarities between birds and other companion species than differences, yet these differences can make birds difficult to treat. This section will provide an introduction to important management and therapeutic procedures commonly used in avian practice.

EXAMINATION/RESTRAINT
Avian physical exams must begin with a thorough history. Like most exotic animal problems, many bird diseases are husbandry related. Especially when dealing with novice bird owners, nothing can be assumed. Begin by asking how long the bird has been owned, where it was obtained, and what other pet species are in the home. Also ask for specifics about diet and cage environment.

Before the physical examination, observe the bird’s posture and behavior. Look for abnormal droppings. Follow a routine during the examination so that abnormalities are not missed. It is important to weigh birds at every opportunity. A digital scale (1gm increment) is best (Ricks Bird Supply: 414-461-6767). When examining birds, scale/perch combinations sometimes work; other times you may use a towel.

Birds that don’t step up on command may be caught using a towel. Dimming the lights will also help. It is easiest to catch a bird by using a towel, against the floor or side of the cage. Wrap the bird up and make a “birdie burrito”. Once restrained in the towel, the bird can be switched over to your hands if you wish. Using a towel for the entire physical is the author’s preference for larger psittacines. With the towel providing most of the restraint, pressure at only one or two spots is needed. By placing the bird in the dorsal recumbency, the towel wrap can be switched from side to side, exposing one wing, then the other. Still wrapped, the bird can be rolled over so that the sternum is faced down, and the tail and back may be examined.

When restraining the head, you may use an encircling grip or a side-to-side grip. The encircling grip is safe in birds because they have complete tracheal rings. Stretch the neck gently, and you may use your thumb or knuckle to lock underneath the lower beak. When using a side to side grip be careful not to exert too much pressure, particularly in macaws, so as not to bruise the delicate white tissue on the sides of the head. To examine the oral cavity loops of gauze, a wire speculum, or a bivalve nasal speculum and light source may be used.

GROOMING
Routine grooming procedures are a vital aspect of any veterinary practice. Wing, beak, and nail trimming give the veterinarian and staff regular contact with the patient and the owner. These appointments allow staff members to maintain proficiency and confidence in handling birds. They also provide for a cursory examination and a record of body weight, so signs of disease can be detected and discussed early in the course. Finally, they provide a source of
When performing a wing trim, use cat nail trimmers and cut the feather shaft near the wing just above the feathers, between the clear and white portions. This hides the cut-end, thereby lessening the likelihood of irritation to the bird. Cut the first 5-7 primaries on both sides. Alternatively, the first three primaries may be spared and the next 5-7 cut, leaving a more cosmetic appearance. Avoid cutting blood feathers. Be sure to forewarn your clients that their bird may still be able to fly and may need additional clipping. When performing a nail trim, use quick stop routinely on all nails, as some only begin to bleed after the bird has been released. Abnormal nail and beak overgrowth has been linked to nutritional, viral, or hepatic disease. Most birds do not need routine beak trimming. A Dremel tool may be employed. Use caution: overzealous beak trimming may result in bleeding and anorexia.

INJECTIONS
Subcutaneous injections are the preferred route for multiple or repeated injections such as boluses of fluids and certain drugs. For SQ injections, the patient is generally restrained in sternal recumbency or restrained upright with the ventrum exposed. Areas of loose skin can usually be found dorsally in the interscapular region, laterally on the flanks, and ventrally in the inguinal web. Smaller volumes can be injected into the patagium (wing web) or over the hips. Use caution to avoid accidental injection into air sacs found up near the neck and in the coelomic cavity. Some drugs (e.g. doxycycline) should not be given SQ, and others (e.g. enrofloxacin, calcium EDTA) should be diluted prior to SQ administration.

Intramuscular injections are the preferred route of administration for some medications (e.g. doxycycline, calcium gluconate), but multiple IM injections should be avoided. IM injections are painful and frequently unnecessary. Necropsy of birds that have received multiple IM injections of enrofloxacin, for example, will reveal extensive bruising and muscle necrosis. Intramuscular injections are usually administered in the pectoral muscles.

Intravenous injections are indicated for emergencies and some other situations. Vascular access points are the same as for venipuncture. Alcohol will help to expose the vein and make feathers lie flat. The jugular vein is the site most often utilized for venipuncture in parrots. It can be useful for IV administration, especially in smaller patients. The right jugular is much larger than the left jugular in the majority of individuals. The patient is restrained in left lateral recumbency, with the head pointing toward the phlebotomist. Bending the needle slightly at the hub will facilitate entry into the vein. After injection, return the bird to standing position, as the resistance to leakage of blood through the venipuncture site is greater than that for blood following its gravitational direction towards the heart. If the patient cannot stand, or if there is potential for a laceration of the vein, direct pressure over the venipuncture site is recommended.

The basilic vein is located on the underside of the wing, coursing over the elbow joint. This site is especially useful for IV access during general anesthesia and surgery. It is the preferred vascular access site for baby birds, for patients that cannot tolerate stress, and whenever aspiration secondary to regurgitation is likely. The patient is maintained in sternal recumbency, and the assistant elevates the wing closest to the phlebotomist. The area is wet with alcohol, and the vessel is occluded near the shoulder joint, and the vessel is accessed anywhere along its course. Hematoma formation at this site may be substantial, therefore direct pressure over the venipuncture site is recommended. Alternatively, when administering fluids, a small bolus of fluids can be placed perivascularly as the needle is withdrawn, thus collapsing the vein and providing prolonged pressure over the venipuncture site.

The medial metatarsal vein is the vascular access site of choice for many species, especially larger birds. The vessel is located medially above the tarsal joint. The feathers and skin are wet with alcohol, and several of the small feathers overlying the vein may be plucked. This vessel is located in a relatively non-expansile region, so the risk of hematoma formation is decreased. After an injection direct pressure over the vessel is advised since the leg lies below the level of the heart. A 25-28 ga needle is generally suitable for most injections in pet birds.

FLUID SUPPORT
Fluids are indicated to prevent and treat dehydration in birds, just as in other species. Critically ill or injured birds, those presenting for surgery, and birds with a HCT above 55% should receive fluid support. Isotonic fluids such as Ringer’s solution, 2.5% dextrose in 0.45% sodium chloride, and 0.09% normal saline can be administered SQ, IV, and IO. All fluids should be warmed to 100-102°F prior to administration.

Daily maintenance for most parrots is 40-60 ml/kg/day. Smaller species, such as finches, may consume up to 300 ml/kg/day. Fluid deficit (ml) may be estimated by multiplying body weight (g) by % dehydration. The author
typically gives fluids at 6-9% of BWt daily, in divided boluses. The subcutaneous route is used for routine cases, but is inappropriate in critically ill or severely dehydrated patients. IV or IO fluids should be used in those circumstances. One or two boluses of IV fluids frequently provide such an improvement that further IV administration is unnecessary. Orally administered fluids are indicated for maintenance and when the patient is minimally dehydrated, however oral fluids require normal GI function to be effective.

An IV catheter of the appropriate size and length can be placed in the jugular vein in a cardiac direction. It is important to place the catheter as near the thoracic inlet as possible to avoid kinking when the neck is in normal flexion. Use of a more rigid polypropylene catheter also minimizes the possibility of kinking. The catheter can be sutured in place or enclosed in a tape collar following installation. The tape collar actually increases the catheter’s stability but should be applied loosely to avoid constricting the crop and esophagus. A small diameter Teflon catheter can be placed in the basilic vein of larger birds as it crosses the medial aspect of the elbow. A section of tongue depressor may be taped alongside the catheter to provide stability. After the catheter is installed the wing should be placed in a figure “8” bandage to prevent dislodging.

Because of the difficulty in stabilizing an IV catheter and the small size of many patients, the intraosseous catheter has become popular among avian veterinarians. Two primary sites are used: the distal ulna and the proximal tibiotarsus. The technique simply involves installing a spinal needle through the end of the bone and into the marrow cavity. The femur and humerus are not used because of their pneumatic properties.

When utilizing the ulna, the most important landmark to identify is the dorsal condyle of the distal ulna. The carpus is flexed and the ulna is identified by palpation. Once the insertion site is located a surgical prep of the area is performed. The needle is directed under the dorsal ulnar condyle and proximally into the shaft of the ulna. Once the needle is placed the stylet is withdrawn and the needle is capped and secured with tape or stay sutures.

The approach to the tibiotarsus is similar to that for a normograde pinning of the bone. The cranial cnemial crest is identified on the anterior aspect of the proximal tibiotarsus between and just distal to the femoral condyles. The area is prepared for sterile technique and the spinal needle is directed into the tibial plateau just posterior to the cnemial crest and distally into the marrow cavity of the tibiotarsus. The stylet is withdrawn and the hub is then taped or sutured in place.

With either technique fluids should be administered especially slowly to avoid leakage (minimal with careful technique) and pain (significant with high pressure).

RESPIRATORY SUPPORT
Oxygen can be extremely beneficial in the early stages of critical care. Respiratory emergencies certainly require oxygen administration, and since many critically ill patients are acidotic their conditions can improve with oxygen supplementation. The method of administration depends on the primary problem. When possible, it is best to humidify and warm the oxygen prior to delivery to the patient. This is best accomplished by bubbling the gas through warmed isotonic or half-strength saline solution. A canister can be devised by using rigid tubing and an empty IV fluids bottle immersed in warm water.

CHAMBER
Any patient that can benefit from oxygen administration can initially be rested in an enclosed container into which oxygen is delivered. Although commercial chambers are available for this purpose, even a cardboard box will suffice. All that is required is that the oxygen be somewhat contained so as to increase its atmospheric concentration.

Commercially manufactured intensive care units offer the advantage of being able to supply heat and humidity in addition to oxygen. The unit can be kept in a “ready” configuration so the oxygen, heat, and humidity are immediately available in an emergency. In situations where extreme hypothermia is part of the presentation, increased humidity minimizes the risk of rebound hypovolemia caused by warming the periphery of the patient before the body core. Effective warming humidity can be provided by placing the bird on a grid over a pan of moderately hot (not scalding) water in the ICU.

MASK
Oxygen can be supplied via a typical anesthesia mask. The cone can be placed over the heads of large birds while small birds can be placed completely into the cone as though it were a chamber. Care should be taken that the
patient does not struggle and aggravate its already fragile condition.

AIR SAC CANNULA
Tracheal obstructions from foreign bodies, neoplasia, fungal granulomas, etc. initially require the creation of an alternate breathing passage. The existence of the air sac system in birds provides a means of ventilation not possible in mammals. Effective respiration can be achieved by intubating the caudal thoracic air sac. Anesthesia is helpful in birds that are capable of resisting restraint. Those which are severely dyspneic may offer little resistance and the urgency of establishing effective respiration may preclude anesthesia. It is critical that the patient be evaluated for the cause of the dyspnea if possible; air sac cannulation is life saving for tracheal causes of dyspnea but it is contraindicated for pulmonary causes.

The type of tube utilized depends on the size of the patient and the urgency of the situation. In small birds a 2-3 cm section of IV tubing will suffice. In larger birds a standard 3.0 mm ID cuffed endotracheal tube can be modified for abdominal installation. The tube is trimmed just above the airline thereby preserving the integrity of the cuff. A 1 X 3 cm strip of Elasticon is wrapped around the endotracheal tube 2-3 mm above the cuff. Inflation of the cuff after placement in the bird offers the advantage of securing the tube in place and more importantly expanding the air sac thereby improving the patency and effectiveness of the tube.

The breathing tube can be installed into the caudal thoracic air sac either between the last two ribs or just behind the last rib, just dorsal to the dorsal edge of the pectoral muscle. The patient is secured in lateral recumbency and the area is surgically prepped. The leg is flexed and abducted (not pulled cranially or caudally) to expose the last rib. A stab incision is made through the skin with the point of a #15 scalpel blade. A fine mosquito hemostat is used to bluntly dissect through the intercostal or abdominal muscles forming a hole barely large enough to insert the breathing tube. The tube is inserted and secured either by suturing or inflating the cuff, or both. Patency can be tested by holding a microscope slide at the opening to observe for breathing-induced fogging. Once secured, the bird can breathe freely through the tube or an airline can be connected for oxygen administration or anesthesia.

NEBULIZATION
Respiratory disease can be treated by nebulization of antibiotics, antifungals, mucolytics, bronchodilators, and other medications. Nebulizers use either an air compressor or ultrasonic energy to generate an ultra-fine particle mist of medication so it can be inhaled. In humans, it is necessary for droplet size to be 2-6 microns for deposition into the tracheobronchial area of the lung, and 0.5-2.5 micron for deposition in the alveoli. It is therefore desirable to provide a nebulizer which produces the desired droplet sizes, in the range 0.5-8 micron. The nebulizer usually connected to a chamber or ICU cage for administration. Presumably, the smaller the chamber the greater the dose actually inhaled by the patient. Birds are usually treated 2-3 times daily for 15-20 minutes until the patient is stable. The author has had excellent results with the PolyGreen ultrasonic portable nebulizer (BestMed, Golden, CO)

NUTRITIONAL SUPPORT
Maintenance of nutrient intake is critically important in avian patients. Due to their high metabolic rates, negative effects of starvation occur quickly. When a bird fails to eat due to illness or injury, it must be nutritionally supported by force-feeding directly per os, via a gavage tube, or through an indwelling alimentary catheter. Composition of supportive formulas is a matter of individual preference. Commercial products are available that provide calories and other nutrients for ill patients. Pelleted diets can be fine-ground and mixed with water or electrolyte solutions. Hand-feeding baby formulas can also be utilized. When determining a feeding schedule it is extremely important to consider and meet the patient’s fluid needs. Birds that have been domestically hand raised often accept warmed liquid foods orally. Even untamed birds will sometimes voluntarily accept oral feeding. Often however force-feeding is necessary and use of a gavage tube or indwelling feeding device becomes inevitable.

TUBE FEEDING
The simplest way to force feed is to employ a ball-tipped metal or rubber tube to deposit liquid food into the crop. Various commercial sources are available for tubes of both types specifically designed for this purpose. The size14 French red rubber feeding tube is an ideal size for most birds over 100 grams because it can be cut at any length at it will still fit snugly on a standard syringe tip. Birds are fed by sliding the tube over the tongue toward the right side of the bird’s neck (where the esophagus proceeds) and depositing formula in the crop. An oral speculum is used when the bird has enough strength to bite and damage the tube. A safe volume of formula for feeding directly into the crop is roughly 3-5% of the bird’s normal body weight. Feeding should always proceed slowly to avoid overfilling and reflux. Force-feeding should never be performed on birds that are recumbent as regurgitation may
occur leading to pulmonary aspiration and its consequences.

**ESOPHAGOSTOMY**

Certain situations mandate bypassing the crop and depositing food directly into the proventriculus or beyond. Babies suffering from crop burns or those with refractory crop dysfunction, and birds with severe beak injuries benefit greatly from an indwelling proventricular feeding tube installed via an esophagostomy. A 14 French red rubber feeding tube is passed down the esophagus of an anesthetized bird, manipulated through the crop and into the thoracic esophagus, and continued to the proventriculus until resistance is felt. At that time a 1 cm longitudinal incision is made on the right side of the neck over the feeding tube identified within the esophagus. The tube is isolated and transected beneath the incision, the oral end is removed completely, and the proventricular end is extracted 2-3 cm from the incision. A 1x5 cm strip of Elasticon is wrapped around the protruding end of the feeding tube and it is sutured in place on the neck. If the incision is large, it may be sutured, but typically suturing is not necessary. Since the ventriculus is smaller than the crop, feedings must be about half the size used for crop feeding, and twice as frequent. A male adapter plug can be used to cap the tube between feedings. This device has been left in place as long as seven weeks without complications. Removal is accomplished by simply cutting the stay sutures and extracting the tube. Debridement and surgical closure of the wound is not usually necessary.

**ABDOMINOCECTESIS**

Birds presenting with excessive fluid in the coelomic cavity may exhibit a pendulous abdomen and/or respiratory distress. Ascites can occur due to neoplasia, egg yolk peritonitis, and other causes. Abdominocentesis may be indicated to relieve distress, and to obtain samples for culture and cytology. The bird must be adequately restrained, either physically or chemically. A 22- to 27-ga needle is then inserted on the midline through aseptically prepared skin just caudal to the sternum. The author prefers to use a 25 ga butterfly catheter for this purpose. Direct the needle caudally and slightly to the right in order to avoid the ventriculus. As the sample is aspirated into the syringe, the needle may need to be rotated to free the bevel from the body wall or viscera. Cytology specimens should be prepared immediately in order to reduce artifact.

**CROP WASH**

A crop wash is useful for obtaining samples for cytology and culture. It is sometimes also performed to remove unwanted crop contents, as with toxin ingestion. The bird is restrained in an upright position and the neck is fully extended. An appropriately sized soft rubber or metal feeding tube is passed from the left side of the oral cavity into the cervical esophagus on the right side of the neck. The tube is advanced slowly and gently. No resistance should be encountered. If there is resistance, the process should be stopped to avoid perforating the thin-walled esophagus. When properly placed, the tube should be palpable within the esophagus and crop. The tube and the trachea should both be palpable as discrete structures before introducing any material into the crop. Sterile fluid (water or saline) may be infused into the crop (1.0-2.0 ml/100g BWt). The crop should then be gently massaged as the fluid is aspirated. Excessive negative pressure may result in aspiration of the crop wall into the lumen of the tube, causing injury to the crop.

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**GASTROINTESTINAL STASIS IN SMALL HERBIVORES**

**INTRODUCTION**

Rabbits, chinchillas, and guinea pigs are monogastric, hind-gut fermenters; all have a functional cecum and require a high-fiber diet. Fiber is broken down in the cecum by a variety of microorganisms which are nourished by a constant supply of water and nutrients from the stomach and small intestine. These same microorganisms produce volatile fatty acids (VFAs) which, in turn, affect appetite and gut motility. Any disturbance in this mutually beneficial relationship can result in gastrointestinal hypomotility—increased GI transit time characterized by decreased frequency of cecocolonic segmental contractions. In severe cases, this leads to ileus with little to no caudal movement of ingesta, known in practice as gastrointestinal stasis.

**PATHOPHYSIOLOGY**

Gastrointestinal stasis is not a disease but a symptom that one or more of the factors governing GI motility are out
of order. There are no breed or gender predilections for GI stasis, and it can occur at any age. Proper hind-gut fermentation and GI tract motility are dependent on the ingestion of large amounts of roughage, long-stemmed hay, and water. Diets that contain inadequate amounts of long-stemmed, coarse fiber predispose the patient to gastrointestinal stasis. Hypomotility of the GI tract can alter cecal fermentation, pH, and substrate production such that enteric microflora populations are altered. Diets that are low in coarse fiber typically contain high simple carbohydrate concentrations, which provide a ready source of fermentable products. This alters the large bowel ecology in a way that threatens favorable microorganisms and promotes bacterial pathogens (e.g. *E coli* and *Clostridium*), and toxin production. Bacterial dysbiosis can cause acute diarrhea, chronic intermittent diarrhea, enterotoxemia, ileus, or gas accumulation (“bloat”). As nausea and gastrointestinal discomfort lead to anorexia, fiber and water intake are further reduced, and the process becomes self-perpetuating. If prolonged, GI stasis often leads to hepatic lipidosis, dehydration, and other secondary complications.

Rabbits, guinea pigs, and chinchillas cannot vomit. In the healthy digestive tract, a moderate amount of gas is normally produced by the fermentation process and eliminated by peristalsis. With GI stasis, however, there can be excessive gas production and, without normal motility, the stomach and intestines can overfill with gas and become distended, a condition commonly called “bloat”.

**ETIOLOGY**

Gastrointestinal stasis is often the result of insufficient dietary fiber and/or excessive stress. It is most commonly associated with inappropriate diet. A diet high in roughage such as grasses and long-stemmed hay is ideal. Rabbits, guinea pigs, and chinchillas that do not receive enough fiber often suffer from subclinical hypomotility, and are thus more susceptible to other risk factors for GI stasis, whereas those that have adequate fiber intake are more resistant.

GI hypomotility is promoted by a diet consisting primarily of commercial pellets, especially those containing seeds, oats, or other high-carbohydrate treats. Feeding cereal products (bread, crackers, and breakfast cereals) and foods high in simple carbohydrates (fruits, yogurt drops, other treats) further increases the risk. Because intestinal microflora depend on a steady flow of water and nutrients, and the digestive tract depends on fiber for normal motility, any event leading to inappetence or anorexia (pre-surgical fasting, sudden changes in the diet, concurrent illness, starvation) or dehydration (sipper malfunction, bad tasting water, careless mistake) can trigger an episode of GI stasis.

Stressful conditions have a negative effect on gut motility. GI motility is regulated in part by the autonomic nervous system; stress increases the adrenal output of epinephrine and inhibits peristalsis. Common causes of stress include dental disease (malocclusion, molar elongation, odontogenic abscesses), metabolic disease (renal disease, liver disease), pain (oral, trauma, postoperative, urolithiasis), anxiety (dyspnea, fear, fighting, lack of hide box), neoplasia, infection, parasitism, and environmental changes (boarding, new pets, unfamiliar noises).

Other factors that can contribute to GI stasis include toxin ingestion, foreign material (scoopable cat litter, hair, carpet fiber), obesity, inactivity, confinement, and certain drugs (anesthetics, anticholinergics, opioids, antibiotics). Inappropriate antibiotics can damage enteric microflora, promote the growth of pathogens associated with GI stasis, and lead to antibiotic-associated enterotoxemia.

**PRESENTING SIGNS**

GI stasis is one of the most common presentations seen in small herbivores. Affected animals usually present for decreased appetite; patients will often initially stop eating pellets or hay, but will continue to eat treats, followed later by complete anorexia. Fecal pellets may have become scant, firm, or small in size; with complete GI stasis there may be no fecal production at all. In some cases there may be soft stools or diarrhea. Initially patients are bright and alert, but with prolonged stasis may present depressed, lethargic, or shocky. Signs of pain include bruxism, hunched posture, failure to groom, and reluctance to move. Affected animals may stretch out or roll in an attempt to relieve pain.

**DIAGNOSIS**

GI stasis should be suspected when there is decreased appetite and/or fecal production, and a recent history of inappropriate diet, illness or stressful event. Abdominal palpation may reveal small, hard fecal pellets or absence of fecal pellets palpable in the colon. The cecum may be filled with gas, fluid, or firm, dry contents depending on the
underlying cause. The normal stomach should be easily deformable, soft, and pliable; it should not remain pitted on compression. With complete GI stasis the stomach may be severely distended, hard, and nondeformable. The presence of firm ingesta in the stomach of a patient that has been anorectic for 1-3 days is compatible with the diagnosis of GI stasis. Differential diagnosis includes GI obstruction due to foreign body, volvulus, intussusception, or intestinal neoplasia, and any cause of anorexia and decreased fecal output (e.g. dental disease, metabolic disease, cardiac disease).

CBC, biochemistry, and urinalysis are often normal, but may be used to identify underlying causes of GI hypomotility and anorexia. PCV and TS may be elevated with dehydration. Liver parameters may be elevated in cases with hepatic lipidosis. Inflammatory leukogram may be seen with intestinal perforation.

Gastric contents, including hair (rabbits), are normally present and visible radiographically, however, a distended stomach in spite of anorexia implies gastrointestinal stasis. A halo of gas can be observed around the inspissated stomach contents in some cases of GI stasis, and there can be moderate to severe gas distension throughout the digestive tract, including the cecum. Small fecal balls or the absence of fecal balls in the colon is highly suggestive of hypomotility. Severe distention of the stomach with fluid and/or gas is radiographic evidence of acute small intestinal obstruction, which constitutes a surgical emergency.

TREATMENT
Medical management of GI stasis centers on basic supportive care. Warmth, stress reduction, pain relief, fluid replacement and nutritional support are important first aid measures. The clinician should consider hospitalization in a quiet environment so that the patient can be observed, his progress monitored, and additional nursing care provided as needed. Provide thermal support through the use of incubators, heating pads, or radiant heat emitters; to avoid causing heat stress, ambient temperature should not exceed 80˚F. Small herbivores should be housed in a dark, quiet space away from natural predators’ noise and odors.

Anxiety can be safely reduced in most rabbits and rodents with injectable midazolam (0.25-0.5 mg/kg SQ or IM). Analgesics such as buprenorphine (0.02-0.05 mg/kg IM, SC q 8-12 hr) or meloxicam (0.2-0.3 mg/kg IM, SQ, PO q 24 hr) are essential in most cases as intestinal pain decreases appetite and impairs GI motility.

Parental fluids are typically given SQ at a rate of 25-35 ml/kg q 8 hrs. Warm all parental fluids prior to administration. Although the majority of cases can be treated effectively with subcutaneous fluids, shocky, azotemic, and critically ill patients should receive intravenous fluids. IV catheters can be placed in any site normally used for venipuncture, but the cephalic and saphenous veins are the most practical. In an emergency or when peripheral veins are collapsed, fluids can be administered intrasosseously. Provide additional fluid support orally with oral electrolyte solutions such as Rebound OES (Virbac) or Pedialyte (Abbott) at 15-20 ml/kg q 8 hrs. Fluids are continued and then gradually reduced until urine output and drinking return to normal.

Provide nutritional support by syringe-feeding gruel such as Oxbow Critical Care Formula. Additional options include vegetable baby foods and soaked, ground pellets. Feed approximately 20-30 ml/kg q 8 hrs. For prolonged nutritional support, a nasogastric tube can be placed in a manner similar to that used for cats (limited mostly to rabbits and large guinea pigs due to size constraints). Liquid enteral fluids (Ensure, Sustecal), Oxbow Critical Care Fine Grind formula, and diluted vegetable baby food can be passed through an 8fr or even a 5-fr NG tube. Patients should be tempted often with parsley and other fresh greens to see if they will eat on their own. Nutritional support is continued and then gradually reduced until fecal production and appetite return to normal.

Motility modifiers are indicated in cases of GI stasis, bloat, and reduced fecal output, provided intestinal obstruction and perforation have been ruled out. The author usually begins prokinetic therapy with injectable medication in severe cases. Metoclopramide 0.5 mg/kg PO, SC, IM, and cisapride 0.5 mg/kg PO are administered q 8-12 hr for 3-5 days, until appetite and fecal production return to normal. They may be used in alone or in conjunction. Simethicone 20 mg/kg PO q 8-12 hr may be indicated in cases of gas distention.

Antibiotics are suggested in cases where GI stasis leads to secondary bacterial overgrowth, as indicated by diarrhea, abnormal fecal cytology, or bloody stool. Broad-spectrum antibiotics such as trimethoprim sulfa (30 mg/kg PO q 12 hr) or enrofloxacin (15 mg/kg PO q 24 hr) should be selected. If antibiotic-associated enterotoxemia is suspected, it can be treated with metronidazole 20 mg/kg q12h (to treat Clostridium), and cholestyramine (binds bacterial toxins) 2 gm/20cc water, divided q 24 hr PO or by gavage. (Dose cited is for a typical rabbit). Probiotics (lactobacillus, yogurt) are of questionable efficacy, however transfaunation with cecotropes from a healthy individual
should be considered.

Because activity promotes GI motility, the patient should be allowed supervised time out of the cage to exercise. Access to a safe grazing area will provide additional fiber and enrichment.

**COMMENTS ON PARTICULAR SPECIES**

**Rabbit**

Rabbits have a normal gastrointestinal transit time (GITT) of only 4-6 hrs. Muscular contractions of the proximal colon separate fiber and non-fiber gut contents. Indigestible fiber is rapidly passed in the hard feces, while the soluble contents are retained in the cecum for fermentation. The cecum empties its contents periodically, and the fermentation products are consumed directly from the anus (called coprophagy or cecotrophy). This “pseudorumination” process for redigestion helps in the absorption of previously undigested nutrients and inoculates the gut with essential nutrients.

Gastrointestinal stasis in rabbits is commonly referred to as a “hairball”. As a result of their grooming habits, rabbits ingest a considerable amount of hair which dietary fiber normally removes. With GI stasis, fluid and fiber intake decrease and the gastric contents condense to form a semi-solid trichobezoar composed of hair and ingesta. Thus, the presence of a hairball is not a disease, but a consequence of chronic GI hypomotility. Surgery is not necessary in most cases. Fluid therapy and assisted feeding help to rehydrate the trichobezoar and facilitate its breakup. Note that papaya, pineapple juice, and proteolytic enzymes have been shown to be ineffective at dissolving hair.

Rabbits are unable to vomit because the cardia is well developed and the duodenum exits the stomach at an angle such that the pylorus is easily compressed. Gastric emptying may be prevented by gastric distention, compression due to trichobezoar, gas, or hepatomegaly, and lead to the development of bloat. Affected rabbits should receive analgesics and simethicone. Decompression by orogastric tube or transabdominal trocar may also be indicated.

**Guinea Pig**

The normal GITT for cavies is approximately 20 hours. Guinea pigs normally perform coprophagy or cecotrophy many times per day, either directly from the anus, or from the cage floor. As in the rabbit, coprophagy seems to be an important function, although its contribution to the nutritional needs of guinea pigs has not been fully characterized. Cavies require a dietary source of vitamin C (25mg/kg per day). When GI dysfunction leads to inappetence or anorexia, supplemental vitamin C should be given.

Guinea pigs do not adapt readily to changes in type, appearance, or presentation of their food or water. Sudden changes in the diet (including the brand of pellet food or hay) may result in serious GI upset or a self-imposed fast (risk factors for GI stasis). Any changes to the diet must be made very gradually.

Guinea pigs are especially susceptible to antibiotic-associated enterotoxemia. The normal gram-positive gastrointestinal flora are very sensitive to antibiotics. Certain antibiotics (penicillin, ampicillin, chlorotetracycline, clindamycin, erythromycin, lincomycin) will destroy the normal microbes and permit the overgrowth of *Clostridium* and elaboration of its toxins. Affected cavies exhibit anorexia, diarrhea, dehydration, and hypothermia. Treatment has been outlined previously, but may also include chloramphenicol 50 mg/kg PO q 8 hr to suppress further clostridial overgrowth, and refaunation with a slurry of feces from a normal individual.

**Chinchilla**

Chinchillas have a mean GITT of 12-15 hours. Unlike rabbits and guinea pigs, transit time in the chinchilla is not greatly impacted by a reduction in dietary fiber. Like rabbits and cavies, however, chinchillas produce two types of fecal pellets: nitrogen-rich cecropes, and nitrogen-poor fecal pellets.

Enterotoxemia from *Clostridium, E. coli, Proteus, or Pseudomonas* is common. Anorexia and decreased fecal output are early warning signs. Severe cases exhibit ileus, bloat, diarrhea, hair and fecal impaction, and rectal prolapse. Gastric trichobezoars in the chinchilla are associated with fur chewing rather than insufficient dietary fiber.

Chinchillas exhibit constipation more often than diarrhea. Affected individuals strain to defecate, and the few pellets they pass are thin, short, hard, and occasionally blood-stained. Constipation usually responds to the careful addition of fiber and fresh vegetables to the diet, along with mineral oil laxatives. Rectal prolapse, intestinal torsion, intussusception, or impaction of the cecum or colonic flexure can occur with chronic constipation, diarrhea,
or gastroenteritis. Impactions may respond to medical treatment (mineral oil enema), but prolapse, intussusception or torsion requires surgery.

Bloat (gastric tympani) is associated with feeding hay that has not matured or is rich in clover, sudden food changes (especially the addition of fresh greens and fruits), and GI inflammation. Affected animals are swollen, lie on their sides, hesitate to stir, and are dyspneic. Treat by decompression either by passing a stomach tube or using a transabdominal needle or trocar.

PROGNOSIS
The expected course and prognosis for GI stasis depends on severity and underlying cause. For mild cases with chronic symptoms due to inappropriate diet, the prognosis is generally excellent to good, and diet correction often leads to complete recovery. For moderately severe, acute cases which require hospitalization, the prognosis is good to fair; the patient usually improves after several days of intensive supportive care, and is discharged for additional care at home. In advanced cases—those that went unnoticed for several days prior to presentation—the prognosis is much worse. Hepatic lipidosis, shock, and other complications frequently make treatment unrewarding. Surgical correction of gastric trichobezoar, if indicated, carries a better prognosis than for surgical treatment of acute intestinal blockage by hair.

PREVENTION
Gastrointestinal hypomotility (and the myriad problems related to it) can be avoided through strict feeding of diets containing adequate amounts of indigestible coarse fiber (long-stemmed hay) and low simple carbohydrate content, along with access to fresh water. Allow small herbivores sufficient daily exercise, and prevent obesity. Minimize changes in the daily routine that might cause stress in small herbivores, and avoid sudden changes the diet. Be certain that clean water is available at all times, and is presented in a familiar manner (sipper vs. bowl).

Use only broad spectrum antibiotics such as trimethoprim-sulfas, florquinolones, chloramphenicol, aminoglycosides, azithromycin, and metronidazole. Discontinue antibiotics if soft stools or other gastrointestinal signs develop. Use drugs that can depress GI motility with caution in small herbivores. Avoid over-fasting patients prior to surgery, control perioperative pain and stress, and encourage patients to eat as soon as possible following surgery. Be certain that all postoperative patients are eating and passing feces prior to release.

Rabbits, guinea pigs, and chinchillas should receive an annual to semiannual complete physical exam. Weight the animal at every opportunity, and encourage owners to check weight monthly. Diet and husbandry should be reviewed, and regular fecal examinations should be performed. Routine screening for disease (CBC/chemistry, urinalysis, radiographs) establishes an individual baseline and aids in the early detection of disease.

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THE PNEUMONIAS OF SMALL MAMMALS

INTRODUCTION
Pneumonia is a common presenting complaint in exotic companion mammals. It usually occurs in young, sick, debilitated, or immunodeficient animals when natural defense mechanisms have been eroded. Pneumonia can be precipitated by stress such as shipment, overcrowding, or social conflict among cage mates. Improper environment and husbandry has a significant impact on an individual’s resistance to pneumonia. Extremes in temperature, humidity, exposure to waste, and poor nutrition all tend to increase one’s susceptibility to it. Likewise, the stress of concurrent infection, advanced age, dental disease, or other concurrent illness can lead to breakdown of the immune system and onset of pneumonia.

CLINICAL SIGNS
Patients with pneumonia may exhibit hunched posture, unkempt appearance, lethargy, unfocussed eyes, disinterest
in the surroundings, reduced appetite, weight loss, and/or diarrhea. Other clinical signs might include coughing, wheezing, or rapid respirations. Increased respiratory effort is usually manifested as pronounced abdominal movement when breathing. Discharge from the eyes or nose, and/or diarrhea may be present. A complete physical examination may reveal ocular or nasal discharge (found either on the face or the medial aspect of the front feet) and/or wheezing, and crackles, increased bronchovesicular sounds, or rales may be ausculted. There may be tachypnea or overt dyspnea, and possibly a fever.

DIAGNOSIS
Diagnosis of pneumonia in small mammals begins with thorough history and careful observation. The clinician should ask about diet, nutritional supplementation, the type of cage, bedding material, how often cage is cleaned, the presence of other animals, and any new animals that may have been introduced. Also ask about the routine and whether or not any changes to the routine (e.g. pet sitter, new diet, death of bonded cage mate) have occurred recently.

Workup for pneumonia generally begins with thoracic radiographs. In a normal small mammal the caudal lung lobes are large and well aerated. Evaluation of the cranial lung lobes may be difficult because in some species (e.g. guinea pigs) they are small. The classic radiographic appearance of bacterial pneumonia is an alveolar pattern, with air bronchograms in severe cases. Lesions can be diffuse, localized to a general area, or lobar in nature. If solitary masses are identified, differential diagnosis should include abscessation or consolidation of lung due to bacterial infection.

Hemogram with bacterial pneumonia may or may not reflect an increase in total white blood cell count; a relative neutrophilia and/or lymphopenia are more common. Leucopenia can result from overwhelming bacterial infection, viral infection, or inflammation.

Culture and sensitivity testing should be done before antimicrobial therapy is started. A tracheal wash is generally considered to be more accurate than a nasal swab for diagnosing pneumonia because it can provide material for cytologic exam and bacterial culture. Identification of degenerative neutrophils containing bacterial debris in a tracheal wash specimen is highly supportive of the diagnosis of bacterial pneumonia. While not a widespread practice, transthoracic needle aspiration is another way to obtain lung samples for cytology and culture.

Culture and sensitivity should test for both aerobic and anaerobic organisms, and empirical treatment with broad-spectrum antibiotics should start before results are available. In general, light growths of mixed bacterial populations are less important than the heavy growth of a single species in conjunction with a pathogenic response. Caution must be used when interpreting results; some labs will not report normal flora. However, in an immunocompromised individual, infection may result from normal flora that are opportunistic pathogens. In addition, the normal flora of many species still have not been established or are obscure. If culture can’t be done because antibiotics have already been started, then PCR should be considered. Bacterial PCR can also be helpful in identifying anaerobes (because these frequently do not survive the transport to the lab) and for getting results from an area where bacteria are likely to be dead (e.g. abscess, caseous pus). Where Chlamydophila is suspected (i.e. guinea pigs), a conjunctival scraping from affected individuals will contain intracytoplasmic, coccoid, basophilic organisms (i.e. Chlamydia elementary and reticulate bodies), or a PCR test is available for this infection, as well.

Necropsy findings from small mammals with pneumonia will vary by duration and severity of the disease, and by the organism(s) involved. Mild and acute cases will result in lung congestion, atelectasis. More severe and chronic cases develop suppurative lesions, fibrin adherions and fibrosis. Severely affected individuals can develop pulmonary abscesses, granulomas, and consolidation. There may also be other organ involvement: lymphadenitis, myocarditis, peritonitis, meningitis, septicemia, tracheitis, bronchitis, otitis media, etc.

There are a number of non-infections conditions that can cause respiratory distress in exotic companion mammals including heat stress, diaphragmatic hernia, pregnancy toxemia, and gastric torsion. Dyspnea and weakness may also be found with heart failure or pulmonary neoplasia.

TREATMENT
The ideal antibiotic treatment plan for pneumonia in small mammals will provide “four-quadrant” coverage, will be bactericidal, easy to administer, safe, and not cause gastrointestinal disease. Antibiotics with a post-antibiotic effect (e.g. aminoglycosides, fluoroquinolones) are preferred, so “pulse therapy” can be employed. Post-antibiotic effect
permits once a day dosing, which is less stressful to the patient and improves owner compliance. Aminoglycosides are potentially nephrotoxic, therefore supplemental fluids are advisable.

As hind gut fermenters, small herbivores rely on active cecal flora for digestion. If antibiotics with a gram-positive spectrum are given, dysbiosis and the overgrowth of pathogenic bacteria will occur. Thus, the antibiotics amoxicillin, ampicillin, clindamycin, and lincomycin are avoided when treating small herbivores. The antibiotics least likely to affect cecal microorganisms include trimethoprim-sulfamethoxazole, chloramphenicol, aminoglycosides, and metronidazole. Antibiotics that pose an intermediate risk include oral cephalosporins, tetracyclines, and erythromycins. In small mammals with a simple digestive tract (e.g., ferret), or minimal cecal fermentation (e.g., rat), the risk of antibiotic-associated gastrointestinal disease is minimal or greatly reduced.

Nebulization therapy is an important method of getting moisture and medication into the trachea, bronchi, and small airways. Nebulizers should secrete particles between 0.5 and 3.0 micrometers (a room humidifier will not suffice). Saline nebulization alone is helpful. When patients get dehydrated, the mucociliary escalator (ME) becomes impaired. The ME functions to trap particulates and bacteria and moves them cranially by the movement of cilia, to the oropharynx, where they can be coughed up and swallowed. The mucus layer is made up of two layers—the sol, which is watery, where the cilia move, and the gel lying on top, which traps particles. If the sol layer is depleted through dehydration, the cilia become trapped in the gel layer and movement is impeded, inhibiting the escalator. Systemic fluids and airway nebulization can contribute to the effective action of the mucociliary escalator by allowing the sol layer to perform as required. Nebulization can be used to deliver antibiotics directly to the airway surface, bypassing systemic circulation and minimizing side-effects. Sometimes other agents are added: mucolytics (N-acetylcysteine), or bronchodilators. Use caution, however, when nebulizing products that were not intended for that purpose (i.e., injectable antibiotics), because some animals may respond to the medication with bronchoconstriction.

There is a “blood-bronchus-barrier” which limits penetration of drugs into the airway secretions much the same way the “blood-brain-barrier” or the “blood-prostate-barrier” prevent drug penetration into those tissues. Penetration into airway secretions is important, since many bacterial airway infections remain largely on the luminal airway surface. Nebulization therapy bypasses the “blood bronchus barrier” and provides topical treatment to the airway. It is important to remember than the major function of the respiratory defenses is the removal of particulates, so nebulized drugs are efficiently removed, and only a very small portion of the administered medication reaches the lower airways. Therefore, it is also important to choose a systemic antibiotic that can penetrate the barrier, as well. Lipid-soluble compounds are better able to penetrate the barrier and reach adequate concentrations at the airway surface: metronidazole, chloramphenicol, azithromycin, tetracyclines (doxycycline), and fluoroquinolones penetrate barrier better than penicillin-based antimicrobials; cephalosporins and aminoglycosides have intermediate penetration. Cough suppressants should be avoided when treating pneumonia, since the goal of treatment is to break up and eliminate airway debris and mucus. Gentle coupage (physiotherapy), frequent turning of patients, short walks, and mild to moderate exercise may help to encourage the clearance of sputum.

There are few specific therapies for viral pneumonia. Most viral diseases of rodents do not cause severe disease, but can do so in very young animals or those carrying potential secondary bacterial pathogens. Polymicrobial infections are common and, as is the case with pneumonia in most other animals, in small mammals it can involve one or more bacteria or viruses. In many instances where bacteria and virus are both isolated, bacteria are secondary invaders. Provide antibiotics to prevent or treat secondary bacterial infection, and provide supportive care measures.

Good supportive care is important to treatment success. Exotic companion mammals often present in an advanced state of disease, and may not tolerate treatment unless initially stabilized with oxygen (if indicated), fluids, warmth, and nutritional support. Oxygen supplementation should be provided when there is detectable tachypnea, dyspnea, cyanosis, open-mouth breathing, or when pulse oximeter readings fall below 94%. Oxygen can be provided in an oxygen cage for small animals. It should be humidified prior to use, and is toxic over time. The maximum inspired oxygen concentration for long term use is 40%; higher levels may be used for two days or less.

Many exotic companion mammals are prey animals by nature, and are nervous in captivity. Many small mammals, especially guinea pigs, are creatures of habit and do not handle changes in routine as well as dogs and cats. Small mammals should be hospitalized in a quiet area away from cats, barking dogs, and other loud noises. Provide hide box, towel, or other shelter. Animals that are socialized and well-adapted to their environment tend to have a much better response to hospital care. Bonded pairs may need to be kept in the hospital together in order to reduce stress.
Probiotics such as lactobacillus supplements (e.g. Bene Bac) have not been proven to work, but may be of benefit when treating with antibiotics.

**COMMENTS ON PARTICULAR SPECIES AND ETIOLOGIES**

**Rabbits**

Respiratory disease in rabbits is often caused by bacterial agents: *Pasteurella multocida, Bordetella bronchiseptica, Staphylococcus aureus,* and many others. Reports of viral pneumonia in pet rabbits are rare. *Pasteurella* pneumonia can be chronic or acute; chronic disease is likely to take the form of pleuropneumonia or pericarditis, with abscesses developing in or around the lungs. Anorexia, weight loss, depression, and rapid fatigue are nonspecific signs, but in a rabbit should raise suspicion of lower respiratory tract disease. Rabbits often appear relatively normal, even with minimally functioning lungs.

*Bordetella* is a common inhabitant of the respiratory tract of rabbits. Its prevalence increases with age. *Bordetella bronchiseptica* infection risk is greatest in young rabbits and in hosts with compromised immune function. *Bordetella* has local-acting cytotoxic effects that impair host defenses, and may be an important predisposing factor in *Pasteurella* infections.

*Staphylococcus aureus* can be isolated from the respiratory tract of both healthy and diseased rabbits. While it is probably a secondary invader of compromised mucosa, *S. aureus* produces toxins that are able to block a number of host defenses. Like *Pasteurella,* disseminated infection can cause pneumonia and abscessation of the lungs and heart.

**Guinea Pigs**

Pneumonia is considered to be one of the most significant diseases affecting guinea pigs; it is the number one cause of death among guinea pigs in some surveys. Young guinea pigs are particularly at risk. Many of the organisms that cause pneumonia are acquired by guinea pigs as babies while they are still in the breeding colony, protected by maternal antibodies. Those with subclinical infection often continue to carry these pathogens, and only develop clinical disease later, when stress or concurrent illness occurs.

The course of pneumonia can vary from rapidly progressive and fatal, associated with respiratory failure and/or sepsis, or it can be a much milder presentation with lethargy subtle clinical signs. Guinea pigs are by nature very stoic and this combined with their natural tendency to hide disease means pneumonia is often advanced by the time clinical signs are noticed.

Pneumonia in guinea pigs is usually associated with opportunistic microbes. The two most important pathogens are the bacteria *Bordetella bronchiseptica* and *Streptococcus pneumoniae.* Other common bacteria include *Klebsiella pneumonia, Streptobacillus moniliformis, Staphylococcus aureus, E. coli, Pasteurella pneumotropica, Pasteurella multocida, Streptococcus zooepidemicus, Streptococcus pyogenes, Citrobacter freundii, Yersinia pseudotuberculosis,* and *Pseudomonas aeruginosa.* *Chlamydophila psittaci* can also cause pneumonia; however, in guinea pigs it usually causes a mild, self-limiting conjunctivitis.

Guinea pigs may also develop viral pneumonia. Guinea pig adenovirus is can cause severe, necrotizing bronchopneumonia with high mortality. Parainfluenzavirus has also been shown to cause disease. Pneumonia in guinea pigs also can be caused by atypical organisms including *Pneumocystis carinii* and nematode parasites. Vitamin C should be supplemented, initially by injection 100 mg/kg subcutaneously, and then provided through hand feeding and fresh vitamin-C rich vegetables and fruit. Guinea pigs can be vaccinated against *Bordetella* with commercially available vaccines.

**Chinchillas**

Pneumonia in chinchillas is relatively uncommon. Affected individuals usually are immunocompromised by age, nutritional status, or husbandry-related stress (overcrowding, high humidity, poor ventilation). Pneumonia in the chinchilla tends to be chronic, resulting in ocular or nasal discharge, lymphadenopathy, dyspnea, anorexia, depression, poor hair coat, and weight loss. Likely pathogens include *Pasteurella, Bordetella, Streptococcus, Klebsiella,* and *Pseudomonas.* Viral respiratory disease of chinchillas is not reported. Antibiotic combinations may be indicated. Prevention is by correcting husbandry and reducing stress.

**Ferrets**

Pneumonia is uncommon in ferrets. Viral causes in ferrets include canine distemper virus and influenza virus.
Bacterial pneumonia in ferrets is typically suppurative, affecting the bronchial airways, the lung lobes, or both. Reported primary bacterial that cause pneumonia in ferrets are *Streptococcus zooepidemicus*, *S. pneumoniae*, and groups C and G streptococci. Gram negative bacterial pneumonia has been documented with *Escherichia coli*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Bordetella bronchiseptica* and *Listeria monocytogenes*. The unicellular fungal organism *Pneumocystis carinii* is also known to infect the lung of ferrets, however pulmonary mycoses such as blastomycosis and coccidiomycosis are uncommon in pet ferrets.

**Rats**

Respiratory disease caused by infectious agents is the most common health problem in rats. Three major respiratory pathogens cause overt clinical disease: *Mycoplasma pulmonis*, *Streptococcus pneumoniae*, and *Corynebacterium kutscheri*. Two additional bacteria, cilia-associated respiratory (CAR) bacillus and *Haemophilus spp*, and three viruses, Sendai virus (a parainfluenza virus), pneumonia virus of mice (a paramyxovirus), and rat respiratory virus (a hantavirus), are minor respiratory pathogens that by themselves rarely cause overt disease. A coronavirus of rats, sialodacryoadenitis (SDA) virus, is highly infectious and causes overt disease confined to the eyes, ears, nose and throat. These minor respiratory pathogens interact synergistically as copathogens with the major respiratory pathogens to produce two major clinical syndromes: chronic respiratory disease (CRD) and bacterial pneumonia.

CRD is also known as murine respiratory mycoplasmosis because *M. pulmonis* is the major component of the disease. Clinical signs are highly variable, and initial infection occurs without any clinical signs. Both upper and lower respiratory tracts are involved. Signs may include snuffling, nasal discharge, red tears, rapid respiration, weight loss, hunched posture, ruffled coat, and head tilt. CRD varies greatly in disease expression because of many environmental, host, and organismal factors that influence the host-pathogen relationship: cage ammonia levels, concurrent infection (Sendai virus, SDA virus, pneumonia virus of mice, rat respiratory virus, CAR bacillus), genetic susceptibility of host, virulence of *Mycoplasma* strain, and vitamin A or E deficiency. Antibiotic therapy (enrofloxacin/doxycycline combination, Azithromycin) will not cure CRD, but may alleviate clinical signs; affected animals typically have persistent *M. pulmonis* infection. Removing cage litter and replacing with clean paper daily may help reduce ammonia levels. Bronchodilators and short-acting corticosteroids are also sometimes helpful.

Bacterial pneumonia, the other major clinical syndrome, is nearly always caused by *Streptococcus pneumoniae*, usually with the help of *M. pulmonis*, Sendai virus, or CAR bacillus coinfection. *Corynebacterium kutscheri* causes pneumonia only in severely immunosuppressed individuals, and is rare in pet rats. Pneumonia caused by *S. pneumoniae* can be of sudden onset. Young rats are more severely affected than older ones, and the only symptom may be sudden death. Signs in mature rats include dyspnea, snuffling, abdominal breathing, and purulent nasal exudate (nares and front paws). Tentative diagnosis can be made by a Gram stain of this exudate (will reveal numerous gram-positive diplococci). Because severe bacteremia and multiorgan abscesses/infarctions are common; antibiotic treatment must be aggressive. Beta-lactamase-resistant penicillins are recommended.

**PROGNOSIS**

The prognosis for pneumonia varies by etiology and severity of the disease at presentation. Pneumonia is often difficult to reverse. Affected animals often have underlying immunodeficiency and tend to decline rather than recover. Prognosis becomes guarded once there is obvious respiratory distress.

**PREVENTION**

Many of the causes of pneumonia in small exotic mammals are husbandry related and corrections need to be made in order to prevent disease. Keeping a closed colony will ensure that new diseases are not introduced. New arrivals should go into quarantine, and they should not be mixed with the general population until there has been a reasonable quarantine period, usually 30-90 days.

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RABBIT AND RODENT DENTAL DISEASE

INTRODUCTION
Rabbits, guinea pigs, and chinchillas are all monogastric, hindgut-fermenting herbivores adapted to a course, high-fiber diet. They graze and browse almost continuously, chewing on plant material, gradually wearing their teeth down as a result. To compensate, the teeth (incisors and cheek teeth in these species) continue to erupt throughout life, at up to 2 mm per week. The rates of dental wear and tooth eruption are in equilibrium, and if they fall out of sync, problems occur. The dynamic process of mastication, tooth wear, and tooth growth can be thrown out of balance by improper husbandry, which is why dental disease is among the most common presenting complaints in exotic companion mammal practice.

PATHOPHYSIOLOGY
The possession of “open-rooted” teeth that continue to grow throughout life is the most important peculiarity of rabbits and rodents. This single factor underlies many of the diseases typical of these species, and is the primary reason why dental disease is so frequent as well. Lagomorphs and rodents are herbivorous, with highly specialized nutritional requirements and digestive physiology. This is the primary reason why dental disease is frequently followed by other secondary disease processes, which often actually produce the first clinical signs and symptoms noted by owners and veterinarians alike. As prey species, the small herbivores frequently minimize clinical symptoms, making early detection of dental disease a challenge. Small herbivores are highly dependent on normal dental function. As such, dental disease in rabbits, guinea pigs, and chinchillas often has a tremendous impact on health in general.

ETIOLOGY
The three main causes proposed for dental disease are congenital, nutritional, and metabolic bone disease. The primary congenital abnormality of teeth is incisor malocclusion, and this occurs mostly in dwarf rabbit breeds. Nutritional causes include insufficient dietary fiber and dental wear (all species), and vitamin C deficiency (guinea pigs). Metabolic bone disease can be due to nutritional deficiency of calcium or vitamin D, insufficient sunlight, or renal secondary hyperthyroidism (rabbits). Of course, dental disease can with any process that disrupts the teeth or bones of the skull (e.g. trauma, infection, or neoplasia).

TERMINOLOGY
That portion of a tooth extending above the gum line is the clinical crown; extending below the gum line is the reserve crown. Teeth that continue to erupt throughout life are termed elodont or aradicular. The “root” of an elodont tooth is more accurately termed the apex. Teeth that normally exhibit long clinical crowns are referred to as hypsodont (e.g. incisors and cheek teeth of rabbits, guinea pigs, and chinchillas); those with short crowns are termed brachyodont (e.g. cheek teeth of rat- and squirrel-like rodents). Visually indistinguishable aradicular hypsodont premolar and molar teeth are simply referred to as cheek teeth. The long space separating the incisors from the cheek teeth in rabbits and rodents is the diastema. Dental pathology other than congenital disease is called acquired dental disease (ADD).

PRESENTING SIGNS
Malocclusion of the incisor teeth is the most common clinical presentation of dental disease. While dental disease affecting the cheek teeth is a more frequent clinical diagnosis, diseases affecting the incisors are typically more obvious and apparent to owners.

Animals with dental disease tend to hypersalivate or drool. In mild cases, the animal may still be eating, but only selects certain food items from the previous menu. The animal may chew differently, hold its head at an odd angle, or otherwise indicate problems eating or drinking. It may go to food and show initial interest, but refrain from eating due to difficulty or pain. With advanced disease, dysphagia leads to anorexia. Odor from the mouth may be detected, and pus or blood may be evident in severe cases. Lumps or swellings may be evident on the jaw or skull. Exophthalmos, epiphora, or blepharospasm can also be signs. If asked, owners may report that there are fewer droppings than normal, that droppings are smaller than normal, or softer, etc. Weight loss, lethargy, and unkempt appearance are also common. There may also be vocalization or erratic behavior due to discomfort.

DIAGNOSIS
Dental disease can usually be confirmed during physical examination. A bivalve nasal speculum (illuminated
attachment for Welch Allyn otoscope/ophthalmoscope handle) makes oral examination much easier and provides a far better field of view than a penlight or otoscope. Malocclusion or malposition of incisors and/or cheek teeth will indicate disease. Sharp edges and spurs that form along the edges of teeth can cause lesions on adjacent lips, buccal mucosa, gingival, or the tongue. The buccal aspect of upper cheek teeth and the lingual aspect of lower cheek teeth are trouble spots to examine closely.

Examination under general anesthesia provides a more thorough examination. Loose teeth, pus, blood, and oral ulcers all indicate dental disease. A light source with magnification (i.e. illuminated loupé, rigid endoscope) is extremely helpful for identifying subtle lesions. Skull radiographs (V/D, lateral, and oblique views) are important for assessing tooth apexes and reserve crowns, and for characterizing odontogenic abscesses. CT and MRI imaging may be necessary in more complex cases.

**EQUIPMENT**

To treat the gambit of dental lesions encountered with small herbivores, a number of specialized instruments should be considered. The basic setup for removing dental spurs will include a Lempert rongeur, diamond file, rabbit/rodent mouth gag, cheek dilator, and tongue spatula. For elevating and removing teeth there is the Crossley incisor luxator, the Crossley cheek tooth luxator, feline notched elevators (concave- and convex-cutting), and cheek tooth extraction forceps. To probe the distal aspect of the clinical crown on the last cheek tooth of each quadrant, the author uses Billeau flexible wire ear loop curette. A straight dental hand piece or Dremel tool is also useful for reducing crowns or sharp edges. A rodent/rabbit dental table that serves as work station, patient positioner, and mouth gag is also available.

**ANESTHESIA**

Most small herbivores do not regurgitate or vomit. They frequently hold food material in their oral cavities (especially guinea pigs), which could result in aspiration. Fasting for an hour prior to dental procedures will allow the oral cavity to empty. Atropine or glycopyrrolate will reduce salivation. Isoflurane and sevoflurane are the anesthetics of choice. Premedication with midazolam (0.25-0.50 mg/kg IM) and buprenorphine (0.02-0.05 mg/kg SQ or IM) will reduce the level of anesthetic gas required. Induce by mask or chamber, and usually maintain by mask. These species are “obligate nasal-breathers”, therefore the mask only needs to cover the nose. For certain dental procedures it will be necessary to intubate the patient. This can be a challenge in rabbits and rodents because in these species the glottis is small, the tongue is so shaped that it hides the larynx, and the larynx is smaller than the trachea. Especially in the rabbit, the vocal cords are strong and can easily deflect the tip of the endotracheal tube into the esophagus. Visualization of the larynx is aided by hyperextension of the head and neck. A small-bladed laryngoscope (e.g. Miller 0 neonatal blade) is used to depress the tongue and elevate the soft palate. A canine otoscope can be used instead of a laryngoscope in smaller patients. Direct visualization of the trachea can also be achieved using an endoscope. The endoscope is positioned so the larynx is in view, and an endotracheal tube is passed parallel to the scope and into the trachea. Further, with some scopes it is possible to put endoscope directly inside the tube like a stylet, and to visually guide the scope/tube assembly into the trachea.

Use a circulating water heat pad intraoperatively to prevent hypothermia; provide a warm area for recovery as well. For prolonged procedures, hospitalization and post-operative support is strongly advised. Observation is continued until the animal is once again eating on its own.

**TREATMENT**

**Incisors**

Overgrown incisors can be trimmed with wire cutters, however this method runs the risk of fracturing the tooth. A better option is to cut the tooth down to the desired level with a Dremel cutting wheel or dental burr, being mindful of the heat generated and careful not to overheat the tooth. The author usually attempts to restore a beveled cutting edge to the occlusal surface of the incisor after reduction.

Removal usually involves extraction of all incisors. The incisor is first elevated on its mesial and distal aspects using a Crossley incisor luxator, then on the labial and lingual surfaces using the curved feline notched elevators. While the periodontal ligament is being luxated, the tooth is loosened using *only the fingers* so as not to accidentally break the tooth. The tooth is alternately rotated clockwise and counterclockwise while alternately placing traction and *apical pressure* on the tooth. Once the operator feels as though the incisor could be pulled out, the tooth is firmly twisted and driven apically so as to cause permanent damage the apical germinal tissue. Finally, the incisor is extracted using only the fingers. The defect can be flushed with sterile saline and closed or left open.
to heal, but packed with hemostatic sponge to prevent food from entering the cavity.

**Cheek Teeth**
Premolar/molar points and spurs are rongeured, and then filed smooth with a diamond hand file, dental hand piece, or Dremel rotary bit. The operator must use caution with a rotary tool, however, that excessive trauma does not occur at either the rotating bit or along the spinning shaft. A safer alternative for filing cheek teeth is the Dremel oscillating engraver, the kind of engraver used to personalize valuables. The sharp engraving tip is removable, and has the same shaft diameter as the rotary bits. We replaced the engraving tip with a ball-shaped diamond bit. The result is very effective vibrating bit that will grind teeth but not hurt soft tissue. Clinical crowns may be reduced in similar fashion.

Extraction of cheek teeth is usually easier than incisor removal because cheek teeth are usually not removed unless they are already diseased and loose. The periodontal ligament of the mesial and distal tooth surfaces are luxated with the transverse blade of the Crossley cheek tooth luxator, and the buccal and lingual aspects are luxated with the sagittal blade. The process is continued until the tooth is sufficiently loose to be extracted with cheek tooth forceps. The remaining cavity is packed with hemostatic sponge to prevent food packing, and left to heal by secondary intention.

**COMMENTS ON PARTICULAR SPECIES**

**Rabbit**
Domestic rabbits are descended from the European rabbit, *Oryctolagus cuniculus*, and belong to the order *Lagomorpha*. The lagomorphs (rabbits, hares, and pikas) are characterized by the presence of a second small pair of upper incisors or “peg teeth”. The relationship of rabbits to rodents is controversial; lagomorphs were once considered to be a suborder of the *Rodentia*. Wild European rabbits eat a diet of graze and browse which is high in fiber and low in energy. A large quantity of poorly digestible fiber is essential for normal peristalsis and dental wear.

The nasolacrimal duct of the rabbit courses from a single ventral lachrymal punctum, through the maxilla, into the nasal cavity. It courses very near the apexes of the first cheek tooth and the incisor, and may be occluded by dental disease of either. The dental formula of the rabbit is: 2(I 2/1, C 0/0, P 3/2, M 3/3). Because rabbits are obligate nasal-breathers, and mouth-breathing is a poor prognostic sign. Rabbits are notoriously prone to laryngospasm. Insertion of an endotracheal tube therefore has to occur during inspiration, when the vocal cords are open. Endotracheal intubation is easiest in large breeds.

**Dental Malocclusion:** Rabbits have elodont, hypsodont incisors and cheek teeth. Without the regular wear that a course, fibrous diet provides, cheek teeth overgrow and develop sharp edges and points. These may cause buccal ulcerations along the upper arcades, and lingual ulcers along the lower arcades. Apical elongation into the mandible and maxillary bone also occurs, as a result of increased occlusal contact. Treatment is by trimming or filing molar points, and/or shortening overgrown cheek teeth to allow the mouth to close and reduce occlusal contact. Recurrence is likely, and routine follow-ups every 4-6 weeks may be necessary. Providing timothy grass hay ad lib helps to prevent this problem.

Mandibular prognathism (lower incisors projecting in front of the upper incisors) is analogous to brachycephalic syndrome in dogs, and is most common in dwarf rabbits. Incisors normally grow 2-2.5mm per week. Unopposed, the upper incisors curve inward and grow up toward the roof of the mouth. The lower incisors tend to grow outward and upward, occasionally through the upper lips or nose. These teeth can be trimmed periodically (once every 4-6 weeks) or surgically extracted. Extracted incisors occasionally regrow, requiring a second attempt at removal.

**Odontogenic Abscesses:** Skull and jaw abscesses typically stem from apical tooth infections. Prognosis has historically been poor, however new approaches show great promise. In most cases, the affected tooth must be removed. One method involves weekly surgical debridement, followed by wound packing with antibiotic soaked synthetic gauze, and then temporary wound closure. The process is repeated until the abscess shrinks and dead space is eliminated. After three “cleanup” cycles, the fourth treatment involves implanting antibiotic impregnated PMMA beads and permanent wound closure. An alternative method is to marsupialize the abscess and aggressively flush, debride, and pack the abscess with an antimicrobial agent (e.g. sugar, honey, povidone iodine) daily for up to several weeks. The patient is kept on systemic antibiotics throughout abscess treatment, and antibiotic selection is based upon culture and sensitivity testing. Anaerobic coverage is important.
**Metabolic Bone Disease:** There is strong evidence for metabolic bone disease as a cause of ADD in rabbits. Harcourt-Brown has demonstrated that affected rabbits exhibit osteopenic bone in comparison with unaffected rabbits, and that progressive osteodystrophic disease affects the teeth and bones of the skull. The visual and radiographic features are typical of nutritional secondary hyperparathyroidism. The dietary calcium intake of rabbits offered an inappropriate diet mixture of pellets, seeds, and cereal grains has been show to fall short of the minimum required for mineralization of bone. Vitamin D, parathyroid hormone, and calcium derangements have been documented in rabbits housed indoors versus those housed outside. If dietary calcium levels are corrected in the early stages of dental disease, horizontal ridges in the incisor enamel will grow out, and normal dental appearance will be restored. ADD is uncommon in laboratory rabbits (usually fed a balanced pelleted ration), wild rabbits, and pet rabbits housed outdoors.

**Guinea Pig**
Guinea pigs have elodont incisors and cheek teeth. They have large tongues and small, narrow oral cavities. The dental formula is: 1/1 incisors, 0/0 canines, 1/1 premolars, 3/3 molars. The incisors are generally white in color.

**Scurvy:** Guinea pigs cannot metabolize endogenous vitamin C. They require 15-25 mg per day in their diet. Ascorbic acid is necessary for collagen synthesis. A lack of vitamin C results in defective collagen, including that which is necessary to anchor teeth tightly in their sockets. Without it, teeth loosen and malocclusion results.

**Dental Malocclusion:** Over time, sharp edges and points may develop on cheek teeth. Ulcerations, and in severe cases, entrapment of the tongue commonly occur. With insufficient wear, the molar crowns may elongate and, if the mouth cannot fully close, the incisors may elongate as a result. Treatment is by trimming or filing molar points, and/or shortening cheek teeth to allow the mouth to close. Primary incisor malocclusion is rare, and incisor trimming alone is rarely indicated. Particularly with guinea pigs, incisor malocclusion suggests cheek tooth abnormalities. Providing timothy grass hay ad lib and adequate vitamin C helps to prevent this problem. Recurrence is likely, but prognosis is good with regular recheck and tooth trims in most cases.

**Chinchilla**
Chinchillas have elodont incisors and cheek teeth. The dental formula is the same as for the guinea pig. The incisors are usually orange-colored. Chinchillas have vestigial cheek pouches.

**Dental Malocclusion:** Also called “slobbers”, as chinchillas are particularly prone to developing a wet chin, face, chest, and forepaws. Molar and incisor malocclusion is common: Crossley detected dental abnormalities in 35% of apparently healthy chinchillas presenting for clinical examination, and incisor abnormalities in 55% of those presenting with signs of illness. Uneven wear can cause sharp edges and points on cheek teeth, resulting in oral ulcerations. The buccal aspect of upper molars and the lingual aspect of lower molars are the trouble spots.

**Tooth Elongation:** The natural diet of chinchillas is tough, fibrous, and low in energy content. As a result, wild chinchillas must chew a lot of food to meet their energy requirements and provide adequate tooth wear. Low roughage diets prevent natural tooth wear from occurring. As cheek teeth elongate, increased occlusal contact occurs. Affected teeth are forced apically and begin to curve; remodeling of the surrounding tissues results. The tooth apex begins to intrude through the jaw bone. Lesions can be palpated along the ventrolateral surface of the mandible. Radiographically, they can be seen extending dorsally into the orbits, nasal cavity, and sinuses as well. Exophthalmos and damage to the lachrymal ducts may result. Winking and squinting can indicate dental disease beneath the affected orbit. Tooth root dysplasia leads to increased curvature of teeth, inability to chew properly, spike and point formation, and secondary infection. Unfortunately, there is no cure. Severely diseased teeth can be removed. Opposing teeth may also need to be pulled. Frequent rechecks and repeated cheek tooth trimming are indicated. Long term pain relief and syringe feeding may be necessary. The long term prognosis is poor, but a good quality of life can be offered with regular tooth trims and monitoring.

**SUPPORTIVE CARE**
Supportive care measures are often the key to a good outcome following dental procedures. The clinician should consider hospitalization so that the patient can be observed, his progress can be monitored, and additional nursing can be provided if necessary. In spite of their thick fur, small herbivores can lose body heat quickly. When sick or anesthetized, thermal support should be provided through the use of incubators and thermal pads. In order to avoid causing heat stress, do not let ambient temperatures exceed 80˚F. Provide nutritional support to by syringe-feeding gruel such as Oxbow Critical Care Formula. Additional options include vegetable baby foods and soaked, ground
pellets. Feed approximately 20-30 ml/kg q 8 hrs. This is continued and then gradually reduced until fecal production and appetite return to normal.

Fluid rate is 75-150 ml/kg/day, depending on severity. Fluid support can be provided by both oral and injectable routes. Provide fluid support orally with oral electrolyte solutions such as Rebound OES (Virbac) or Pedialyte (Abbott) at 15-20 ml/kg q 8 hr; parental fluids are typically given SQ at a rate of 25-35 ml/kg q 8 hrs. Warm all fluids to body temperature prior to administration. Fluids are continued and then gradually reduced until urine output and drinking return to normal. Gradually reduce fluid therapy as urine output and drinking return to normal.

Motility modifiers are indicated in cases of GI stasis, bloat, and reduced fecal output, whenever intestinal obstruction or perforation has been ruled out. Metoclopramide 0.5 mg/kg PO, SC, IM, and cisapride 0.5 mg/kg PO are administered q 8-12 hr for 3-5 days, until appetite and fecal production return to normal. Prokinetics may be used in alone or in combination.

Avoid the temptation to give antibiotics “just in case”. Especially in small herbivores, antibiotics can have negative effects on appetite and digestion. Routine trims (even those with ulcerations) do not usually require antibiotics. Antibiotics are indicated for those cases in which infection is present or contamination has occurred. Antibiotics are selected based upon the most likely pathogen and the safety with regard to affecting cecal flora. Until the results of culture and sensitivity testing are known, an antibiotic combination that provides broad-spectrum coverage against both aerobic and anaerobic bacteria should be chosen. Penicillin G benzathine/penicillin G procaine combination has been widely used in the rabbit (at 80,000 IU/kg q48h SQ), but should NOT be used in guinea pigs or chinchillas. Chloramphenicol is safe in rabbits and rodents at 50 mg/kg q12h PO. Azithromycin is safe and effective orally in rabbits and guinea pigs at 15-30 mg/kg q24h PO, however owners must be advised to discontinue the drug if anorexia or diarrhea occurs; the author has no experience with its use in chinchillas. Enrofloxacin 5-10 mg/kg PO 12 hr, and trimethoprim/sulfa 30 mg/kg PO q 12 hr are poor choices for infected teeth and odontogenic abscesses alone, but either antibiotic may be useful in conjunction with metronidazole 20 mg/kg PO q 24 hr. Systemic antibiotic therapy may be necessary for up to 4 weeks post-operatively in with severe infections.

Powdered bark of the Slippery Elm tree is an effective demulcent, forming a soothing film over mucous membranes, and relieving minor pain. It can be used to help alleviate oral ulceration, such as occur in small herbivores from dental malocclusion, molar spurs, and iatrogenic causes. The powder is sold in health food stores. A dilute paste is made using tap water (one 250 mg capsule into 15 cc water) and administered after hand feeding. Treatment may be applied directly to lesions (i.e. 0.50cc into each buccal cavity).

Small herbivores are more stoic and less communicative than their larger counterparts with regards to showing pain. At the same time, most are prey species, hard-wired for “flight”, and are therefore more susceptible to the negative effects of pain. In our practice, buprenorphine and meloxicam have proven to be universally safe and effective in small exotic mammals. Buprenorphine is usually administered as preemptive analgesic 30 min prior to surgery, 0.02-0.05mg/kg SQ or IM, depending on severity and species. The dose may be repeated in 6-12 hrs. Meloxicam is typically dosed at 0.2-0.3mg/kg q 24 hr for 5-7 days for post-operative pain.

**PROGNOSIS**

The prognosis for patients with dental disease varies by the severity of the lesion. Incisors malocclusion frequently requires repeat trimming, however removal of the incisors is practical in the species presented here. ADD of the cheek teeth is generally considered progressive and non-reversible; the prognosis for return to normal is poor. Mild cases can be managed by intermittent trimming and filing of the teeth. With advanced ADD, removal of loose or infected teeth, and treatment of odontogenic abscesses are frequently indicated. The goal of treatment is to provide the animal with a good quality of life by minimizing discomfort, maintaining body condition, and enabling the pet to eat on its own if possible.

**PREVENTION**

Parents who produce prognathic young should be removed from the breeding program (particularly rabbits). High-carbohydrate, high-sugar diets meet the caloric needs of herbivores before adequate fiber intake and dental wear has occurred. Therefore a coarse diet, high in fiber and low in simple-carbohydrates, is recommended. Long-stem grass hay intake should be offered ad lib, and a moderate amount of pellet and greens should be offered daily. Treats such as dried fruit and yogurt drops should be minimized or eliminated. A complete physical exam and laboratory database are recommended every 6-12 months. Any suspicion of dental disease should indicate a closer oral
examination while sedated. In addition, skull radiographs will aid in early detection of dental disease.

REFERENCES

ADRENOCORTICAL DISEASE IN FERRETS

INTRODUCTION
Adrenocortical disease (ACD) affects middle-aged to older ferrets with a reported prevalence of up to 25%. Ferret adrenal disease is different from human, canine, and feline Cushing’s disease because in ferrets adrenal sex hormones are overproduced instead of cortisol. Estradiol, 17-hydroxyprogesterone, or one or more of the plasma androgens may be increased as a result of adrenocortical hyperplasia, adenoma, or adenocarcinoma. Neutering, photoperiod, and genetics are believed to play a role in the pathology of adrenocortical disease.

CLINICAL SIGNS
Adrenal disease typically causes dorsocaudal alopecia in either gender. Pruritis, sexual or aggressive behavior, and a noticeable increase in musky odor can also occur. Affected females may develop vulvar enlargement, and males may present with stranguria or urinary obstruction secondary to prostatic hyperplasia, prostatic cysts, prostatic abscesses, or prostatitis. Additional clinical signs may include estrogen-induced bone marrow toxicity, mammary gland hyperplasia, cystitis, paraurethral or paraprostatic cysts, muscle atrophy, and lethargy. Clinical signs vary depending on which sex hormones are elevated; however, clinical signs do not always correlate with the size of the affected gland or degree of adrenal pathology.

ETIOLOGY
Evidence suggests that neutering promotes ACD by removing sex hormonal negative feedback on the hypothalamus and leads to overproduction of gonadotropin-releasing hormone (GnRH). In response, the pituitary maintains persistently elevated luteinizing hormone (LH) and follicle-stimulating hormone (FSH). LH binds with functional receptors on the adrenal gland, causing it to overproduce sex hormones and inducing hyperplastic and/or neoplastic adrenocortical enlargement. Exposure to a relatively uniform, long photoperiod (such as that encountered by indoor ferrets) may play role in the development of ACD. Genetics could also play a role: one or more tumor suppressor gene aberrances are believed to exist in the US ferret population.

DIAGNOSIS
Physical exam findings of symmetrical alopecia, swollen vulva, or palpable mass cranial to the kidney are suggestive of ACD. CBC may reveal anemia in cases with elevated estradiol. Blood chemistry frequently reflects an elevated ALT. Elevations in plasma estradiol, androstenedione, and 17-OH-progesterone are diagnostic. Ultrasonography can demonstrate adrenal gland enlargement and distinguish right versus left ACD for surgical planning. Laparotomy permits biopsy with histopathology.

TREATMENT
The goals of management are to reduce sex steroid production and to provide a normal life span and a good quality of life for the patient. Surgery aims to remove or debulk the tumor (adrenalectomy), whereas medical treatment (hormonal suppression) does not affect the tumor and is not curative. Treatment choice depends on which gland is affected (left vs. right), surgeon’s experience, severity of clinical signs, age of the animal, concurrent diseases, and owner finances.
Surgical
Adrenalectomy is the treatment of choice for ferrets that are healthy enough for surgery. Both adrenal glands should be observed and palpated. An adrenal gland lies at the cranial pole of each kidney, often embedded in fat. Not all diseased adrenal glands are enlarged, thus palpation alone is not sufficient to evaluate for disease. If the entire gland is not visible the thin layer of peritoneum and fat surrounding the gland should be carefully dissected. The right adrenal gland can usually be found on the dorsal surface of the vena cava where it enters the liver. Cysts, yellow-brown discoloration, irregular texture, and enlargement are indications for removal.

It has been reported that approximately 85% of ferrets with hyperadrenocorticism have enlargement of one adrenal gland, and that the other 15% have the disease bilaterally. Often the left adrenal is completely removed and the right adrenal is debulked by placing hemostatic clips across it to allow for 50-75% removal. Alternatively, the capsule is incised and the glandular contents are shelled out. If the right adrenal gland is debulked (less risky than complete removal) signs of ACD may return, necessitating repeat surgery or medical therapy.

Removal of the left adrenal gland is usually uncomplicated. The phrenicoabdominal (adrenolumbar) vein is identified and ligated at the lateral aspect of the gland, the gland is elevated and gently undermined, and the phrenicoabdominal vein is traced to the vena cava and inspected for tumor invasion. If no tumor invasion is detected, the vein is ligated and the gland is removed.

Right adrenalectomy is often more difficult because of adherence of the gland to the wall of the vena cava and the greater potential for vascular invasion. Magnifying loupes, microsurgical instruments, and vascular clamps are often needed. If the tumor is small, often it can be almost completely freed from the wall of the vena cava with gentle dissection. If so, place hemostatic clips between the gland and the cava and resect the gland. However, frequently the gland cannot be freed from the vena cava because of tumor invasion, or it is located mostly on the dorsal aspect of the vessel. For these cases, more advanced surgical techniques may be needed.

Cryosurgery of the adrenal gland and vena cava is safe and effective. Freezing kills tumor tissue and blood vessel endothelium, but vessel wall integrity remains intact thanks to the elastic structure of the vessel wall. After cellular death by freezing, this elastic collagen serves as the matrix for new endothelial growth. Some controversy exists about cryosurgery of the adrenal gland in ferrets, however, as recent work suggests that cryosurgery of the adrenal gland is associated with shorter post-op survival time than surgical debuking alone.

Medical
The GnRH analog leuprolide acetate (Lupron) is a potent inhibitor of LH and FSH. The dose of the 30-day depot formulation is 100-250 micrograms/kg IM q4w until signs resolve, then q4-8w as needed, lifelong. Side effects include dyspnea, lethargy, and irritation at the injection site. Another GnRH analogue, deslorelin acetate (Suprelorin, Peptech Animal Health, North Ryde, Australia), is commercially available for non-surgical contraception of male dogs in Australia, New Zealand, and some European countries. Deslorelin slow-release implants have been evaluated for use in ferrets with ACD, and results are positive. Deslorelin implants may be legally imported in compliance with FDA guidelines.

Melatonin helps to regulate the ferret’s natural breeding season. Melatonin 0.5-1.0 mg/animal every 24 hour by mouth, administered 7-9 hours after sunrise, has been shown to be effective in alleviating clinical signs of alopecia, aggressive behavior, vulvar swelling, and prostatomegaly. The implant form of the drug is approved by the USDA for use in mink, and is commercially available for ferrets in a 5.4mg constant-release implant that releases melatonin over a 3-4 month period. Possible side effects include lethargy and weight gain. Melatonin can be used alone or in combination with other therapies.

RECOVERY
In most ferrets, clinical signs ACD resolve after adrenal sex hormone levels return to normal. Improvement with medical therapy may be more likely in patients with adrenal hyperplasia or adenoma; less likely in patients with adenocarcinoma. Reduction in vulvar swelling, paraurethral cysts, or prostatic size may be expected within several days. Hair coat typically returns to normal within 2-4 months.

PROGNOSIS
Prognosis for all treatments varies and depends on tumor type, age of animal, presence of concurrent disease, and
mode of treatment. The 1- and 2-year survival rates for ACD are reported to be 98% and 88% respectively. Following unilateral adrenalectomy or subtotal adrenalectomy, monitor for the return of clinical signs; tumor recurrence is common. The reported recurrence rate with development of disease on the contralateral gland following unilateral adrenalectomy is 17%, whereas recurrence following subtotal bilateral adrenalectomy is 15% 7-22 months after surgery.

PREVENTION
Neutering prevents fatal estrogen-induced bone marrow suppression in jills, reduces aggression in hobs, and decreases the musky odor associated with all ferrets, making them better pets. Alternatives to surgical neutering need to be developed that accomplish the same medical and behavioral goals without predisposing ferrets to ACD. The best hope for such a treatment appears to be deslorelin acetate, which, as of this writing, has yet to be approved in the US.

INSULINOMA IN FERRETS

INTRODUCTION
Pancreatic islet beta cell tumors secrete high levels of insulin and cause hypoglycemia. Clinical signs include lethargy, weight loss, weakness, ptyalism, bruxism, seizures, and death. Treatment modalities include medical therapy, chemotherapy, surgery, and dietary changes. The etiology of insulinoma is unknown, however a nutritional hypothesis has recently been offered along with a prevention strategy based upon feeding a more natural, archetypal diet.

EPIDEMIOLOGY
Insulinoma is by far the most common neoplasm in middle-aged to older ferrets, with a reported incidence of 21.7% to 25% of neoplasms diagnosed. Most ferrets begin exhibiting clinical signs around 4-6 years of age. It is likely that islet cell tumors exist subclinically for months or years before symptoms occur.

Insulinomas are common in North America, where the majority of ferrets are fed dry kibble containing 10-45% carbohydrate, and many are fed treats that contain sugar. Insulinomas are uncommon in Europe, New Zealand and Australia, where ferrets are fed a low-carbohydrate diet of meat scraps, poultry scraps, and/or fish scraps, and sugary treats are not provided.

Domestic pet ferrets in the U.S. have limited genetic diversity, suggesting that insulinomas may have a genetic component. Finally, keeping ferrets indoors may contribute to the propensity of endocrinopathies in ferrets in the United States.

PATHOPHYSIOLOGY
Insulin is released when levels of glucose, amino acids, and free fatty acids are increased in the blood. Insulin then causes a rapid uptake and storage of glucose by cells, inhibits hepatic gluconeogenesis and glycogenolysis, and promotes the conversion of excess glucose into fatty acids. The net effect of all these processes is a decrease in blood glucose levels. Insulinomas secrete indiscriminately and are not responsive to inhibitory stimuli such as hypoglycemia or hyperinsulinemia. Although local tumor recurrence is common, metastasis is not.

CLINICAL SIGNS
Onset of symptoms may be gradual or sudden. Appetite may be normal or decreased, and weight loss may be noted. Advanced insulinomas result in hypoglycemia, causing weakness, anorexia, lethargy, stupor, and seizures. The ferret’s eyes may appear “glazed” during episodes. Hypersalivation and pawing at the mouth (presumably due to nausea, numbness, or tingling) also occur. Affected ferrets may present with hind limb paresis or ataxia, as if nerve damage has occurred. Corticosteroid injection, which raises blood glucose levels, may temporarily improve symptoms.

DIAGNOSIS
In patients were insulinoma is suspected but blood glucose is within normal limits (80-120 mg/dL), a carefully monitored 3- to 4-hour fast may be required to confirm hypoglycemia. A presumptive diagnosis is made when ferrets demonstrate fasting blood glucose of less than 60 mg/dL in the presence of clinical signs of insulinoma, and these signs cease after a feeding or intravenous administration of glucose (Whipple’s triad). Other causes of hypoglycemia, such as sepsis, starvation, hepatic disease, and lab artifact, should be ruled out.
Elevated insulin level with concurrent hypoglycemia is consistent with hyperinsulinism and supports the diagnosis of insulinoma. However, a low or normal insulin level does not necessarily rule out the presence of insulinoma, and could indicate erratic insulin production.

Histopathology of surgical biopsy is required for definitive diagnosis. Tumors may be described as hyperplasia, adenomas, or carcinomas, and a specific tumor may have a combination of any of these processes. Immunohistochemistry has been used to further characterize pancreatic islet cell tumors and their metastasis in distant organs.

**TREATMENT**

**Surgery**
Surgical resection of pancreatic nodules or partial pancreatectomy is considered the treatment of choice for greater clinical resolution and longer survival. Pancreatic nodules may be removed individually or, in the case of multiple nodules, a partial pancreatectomy can be performed. In one study, ferrets with partial pancreatectomy had longer survival times than those with nodulectomies. Because of likely recurrence, owners should be advised that surgery is not curative but rather it may temporally stop or slow the progression of disease and provide a longer disease-free interval than medical therapy alone. In one study, nodulectomy combined with partial pancreatectomy had a significantly longer median survival time (668 days), compared with nodulectomy alone (456 days) or medical treatment only (186 days).

**Symptomatic Therapy**
Glucocorticoids increase blood glucose levels by inhibiting cellular uptake, promoting hepatic gluconeogenesis, and inhibiting insulin binding to insulin receptors. Prednisolone (Pediapred Liquid) 0.5-2 mg/kg q 12 hr PO can usually control mild to moderate clinical signs. Begin at lowest dosage and gradually increase as needed to control symptoms.

Diazoxide is added when clinical signs cannot be controlled with prednisone alone. At the same time, the dosage of prednisone may be lowered. Diazoxide inhibits insulin secretion, and stimulates the release of epinephrine. It promotes hepatic gluconeogenesis and glycogenolysis, and decreases cellular insulin uptake. Begin diazoxide at 5-10 mg/kg q 12 hr PO; the dose can be increased gradually to 30 mg/kg q 12 hr if lower doses are inadequate.

Decreases in blood glucose in the ferret often lead to increases histamine release and stomach acid production. Famotidine 0.5 mg/kg PO q 12-24 hr) will help to alleviate the pain, nausea and inappetence associated with insulinoma.

**Chemotherapy**
Doxorubicin chemotherapy for insulinoma has direct toxic effects on pancreatic beta cells. Pre-treatment workup should include CBC, blood chemistry, ECG, and chest radiographs. The patient is pretreated with diphenhydramine 5 mg IM, and doxorubicin is infused at 30 mg/m^2^ (approximately 1 mg/kg) IV. The regimen is repeated q 3 weeks for 4 doses.

**Diet Modification**
Diet is important in the management of insulinomas. High-sugar treats should be discontinued as these can induce a rebound insulin release and trigger a hypoglycemic episode. Diet should be changed to a high-protein, low-carbohydrate formulation. Food should be available at all times; it may need to be placed in multiple areas to ensure easy access. Offering food to ferrets (and hand feeding them, when necessary) every 4 hours, especially right before playing, will help to prevent hypoglycemic episodes.

**Management of a Hypoglycemic Episode**
If clinical signs are noted, the ferret should immediately be fed a high-protein, easily digestible diet (Oxbow Carnivore Care) to abate symptoms. If the ferret presents comatose or exhibiting seizures, corn syrup or sugar solution can be applied to the oral mucous membranes to provide temporary relief. Blood glucose level should be quickly assessed for hypoglycemia, and an intravenous catheter should be placed immediately for a slow bolus of 50% dextrose (0.25-2 mL), titrated to effect. Once seizures have ceased, the patient should be placed on maintenance fluids with 5% dextrose. If the ferret continues to seizure despite IV glucose, diazepam (1 mg/kg IV) may be required to stop the seizures.
**PREVENTION**

Ferrets are obligate carnivores. A growing number of experts are recommending that ferrets be fed a diet that more closely mimics natural diet: raw meat and bone. Finkler advocates a nutritional approach to insulinoma prevention, and recommends a diet low in carbohydrates, and high in fat and protein. Simple sugars should be completely avoided. He recommends a diet containing 42-55% protein, 18-30% fat, only 8-15% carbohydrates, and 1-3% fiber.

Wild polecats consume whole, small prey such as rodents, lagomorphs, and birds. Their natural diet is high in proteins and fat and low in carbohydrates and fiber. Some advocate the feeding of prey items such as mice and chicks. The nutrient analysis of a rat carcass, for example, is 55% protein, 38.1% fat, 1.2% carbohydrate, and 0.55% fiber. Commercially available archetypal diets that have been designed to mimic the nutrient profile of whole prey include Ferret Archetypal (Wysong), Natural Gold (Pretty Pets), and Evo Ferret Diet (Innova). Ancestral diets may someday be found to aid in the prevention of insulinoma.

**REFERENCES**


