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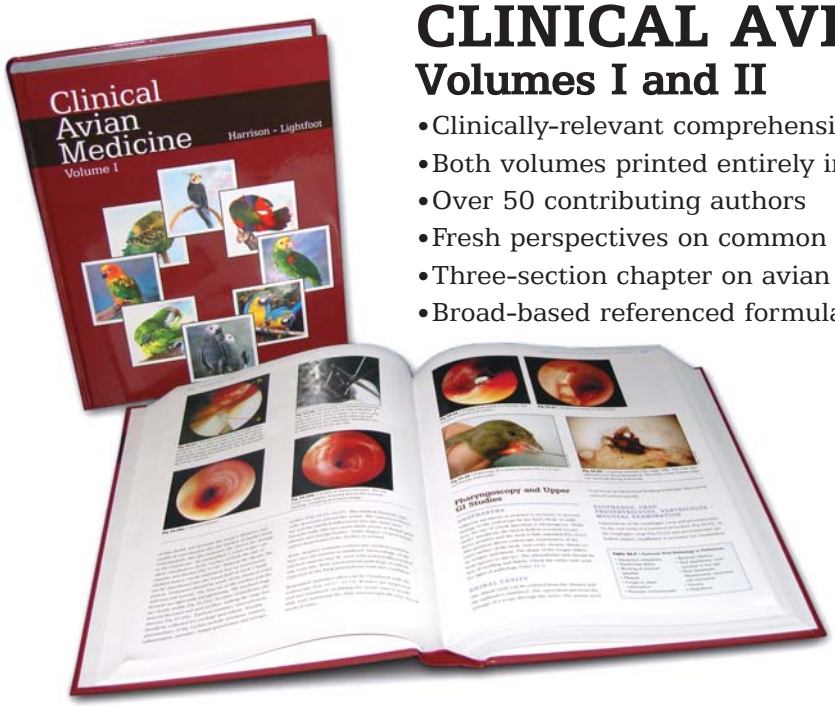

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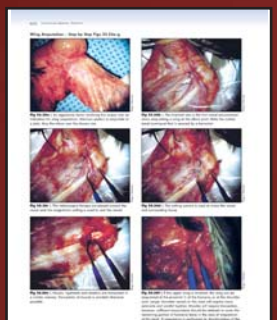

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A PRACTICAL RESOURCE FOR CLINICIANS

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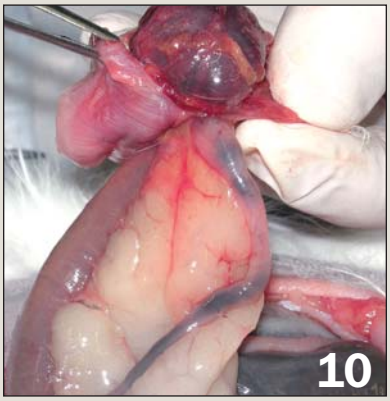

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Volume 11, Issue 3, September 2009

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Dr. Paul Gibbons is Named 2009 Exotic DVM of the Year

Steve Barten, DVM

Dr. Gbbons accepts his award from Dr. Kevin Wright, the 2008 Exotic DVM of the Year award.

Courtesy of Animal Emergency Center

August 14, 2009 - Dr. Paul Gibbons was selected by his peers as the recipient of the 2009 Exotic DVM of the Year award. The award was presented at the combined 30th Annual Association of Avian Veterinarians (AAV) Conference & Expo, 16th Annual Association of Reptilian and Amphibian Veterinarians (ARAV) Conference, and Association of Exotic Mammal Veterinarians (AEMV) Conference held in Milwaukee.

Dr. Gibbons received his DVM degree from the University of Illinois, College of Veterinary Medicine in 1994. He practiced in Minnesota, Wisconsin and Illinois before pursuing advanced training. At the University of California-Davis he completed a residency program in avian, reptile and exotic mammal medicine followed by a master's degree in comparative pathology. During his veterinary career he has worked with cattle, horses, sheep, llamas, goats, pigs, dogs, cats, rabbits, rodents, pet birds, ratites, raptors, waterfowl, reptiles, amphibians, fish, invertebrates and other exotic species. He has been involved with sled dog medicine for the past 6 years and currently serves as Head Veterinarian for the UP200 & Midnight Run Sled Dog Races in Michigan.

He is a Diplomate of the Avian Practice specialty of the American Board of Veterinary Practitioners (ABVP) and currently serves as chairperson of the Joint ARAV-ABVP Organizing Committee for Certification in Reptile and Amphibian Practice. He is an associate editor for the Journal of Herpetological Medicine and Surgery and is President-Elect of the ARAV. He served as Guest Editor of the July 2009 issue of the Journal of Exotic Pet Medicine and has published numerous articles and presented lectures on the husbandry, medicine and surgery of birds, reptiles, amphibians and exotic mammals. Currently he directs the Exotic Species Specialty Service at Animal Emergency Center & Specialty Services in Milwaukee.

Debbie Schouten

Dr. Sandy Smith Wins 2009 "Hollywood Goes Green" Backpack

Sandy Smith, DVM won the "Hollywood Goes Green" organic cotton backpack loaded with Harrisons' Pet Products goodies. It was the same one that environmentally conscious stars and their pets received in April, 2009 for Earth Day. Stars included: Jeremy Piven, Heather Graham, Justin Timberlake, Bill Maher, Adrian Grenier, Jake Gyllenhaal, Pink, Leonardo DiCaprio, Melissa Etheridge, Sheryl Crow, Eva Longoria, Tobey Maguire, Drew Barrymore, Ellen DeGeneres, Kathy Griffin, Tyra Banks, Bette Midler, Julia Louis-Dreyfus, Holly Robinson-Peete, Sting, Rosario Dawson, Tina Fey, Alicia Silverstone, Ricky Martin and Courteney Cox.

Katie Kersting (daughter of Dr. David Kersting of Bird Medicine and Surgery in St. Louis, Missouri) shows off her new "All My Pets Are Green" backpack at the Association of Avian Veterinarians 2009 Conference in Milwaukee. Ten-year-old Kersting writes a monthly column, "Just Hatched," for Bird Talk magazine.

AEMV Announces Research Fund

During its 2009 annual conference held in conjunction with AAV and ARAV in Milwaukee, the Association of Exotic Mammal Veterinarians (AEMV) announced the formation of its Research Fund. In a moment of levity, Dr. Dan Johnson, AEMV Treasurer, was challenged to shave his legs during the annual business meeting if the organization could raise $1,000 by the end of the scientific session day. Being a good sport, he agreed and the goal was readily surpassed.Harrisons' Pet Products donated $250 towards this goal. Information on the progressive growth of the account and the application process for funding is available at www.AEMV.org, according to Dr. Joerg Mayer, Research Fund Chair.

Dr. Nico Schoemaker

Dr. Dan Johnson, AEMV Treasurer, is shown before and after the Research Fund goal for the day was achieved.

Oprah Winfrey was among the celebrities who received a Harrisons' Pet Products gift bag loaded with certified organic pet goodies, including the new certified organic dog cookie mix as part of HollyWOOF 2009.

Harrisons' Pet Products Participates in HollyWOOF 2009

Celebrity pooches get spoiled in Hollywood! As the dog days of Summer fade into Fall, Distinctive Assets delivered special gift bags to 25 pet-loving stars. Every dog indeed has its day, especially in a town where diamond-adorned canines are nearly as famous as their celebrity owners! On August 28, 2009, Hollywood's premier celebrity gifting firm introduced fabulous, unique and useful pet products to stars and their loyal four-legged companions. A-list stars included: Oprah Winfrey, Tori Spelling, Alicia Silverstone, Miley Cyrus, Ricky Martin, Jai Rodriguez, Charlize Theron, Paris Hilton, Drew Barrymore, Mickey Rourke, Hayden Panettiere, Martha Stewart, Rachael Ray, Kathy Griffin, Katherine Heigl, Rachel Bilson, Amanda Bynes, Denise Richards, Mischa Barton, Nicole Richie, Fergie, Ellen DeGeneres, Glenn Close, Hilary Duff, Jessica Biel and Justin Timberlake. Distinctive Assets has a longstanding reputation for pampering the world's biggest stars.

Dr. Cathy Johnson-Delaney is Honored with 2009 Oxbow Mammal Health Award

Oxbow Animal Health has named Dr. Cathy Johnson-Delaney as the recipient of the 2009 Oxbow Exotic Mammal Health Award. Established in 2009, the Oxbow Exotic Mammal Health Award is an annual award presented to an animal health professional who advances the field of exotic mammal medicine and care. Dr. Johnson-Delaney, a veterinarian specializing in avian/exotic animal medicine, was honored with the award at the 2009 AEMV/AAV Conference in Milwaukee, Wisconsin on August 9, followed by a reception in her honor on August 11.

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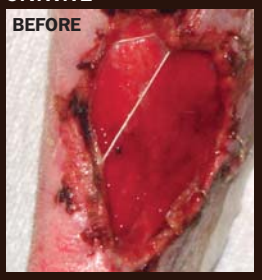

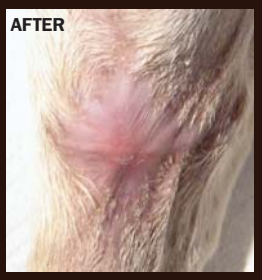

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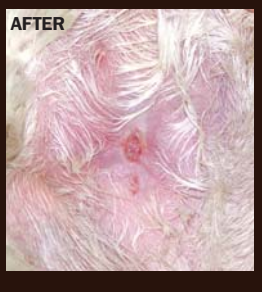

More information on these (and other) case studies is available at www.exoticdvm.com/clinicalcases

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A Case of Feline Infectious Peritonitis-like Disease in a Juvenile Ferret in Norway

Paula B. Brynildsen, DVM, Norwegian School of Veterinary Science, Laboratory Animal Unit, Oslo, Norway and
Olivia Kershaw, DVM, Dept of Veterinary Pathology, Freie Universität Berlin, Berlin, Germany

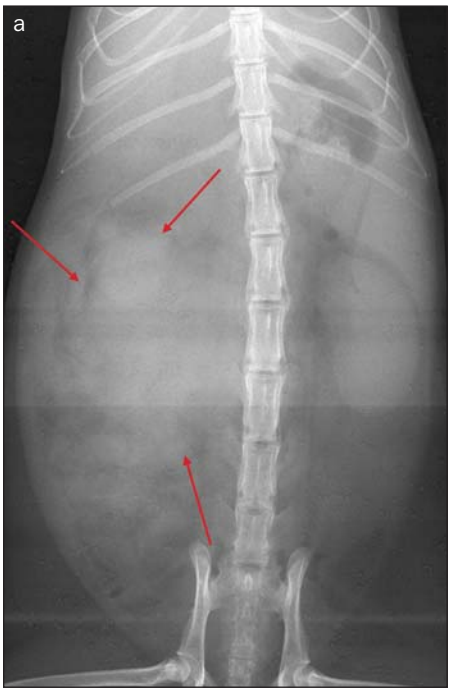
A juvenile intact female ferret (*Mustela putorius furo*), approximately 6 months of age and originally from the Netherlands, was referred by another veterinarian. It had experienced a 3-day duration of lethargy, anorexia, aggression, constipation and weight loss. Vomiting had started the night previous to presentation, and the owner described the vomitus as slimy and yellow. On physical examination, the referring veterinarian palpated an abdominal mass.

At presentation the animal showed marked aggression, which in the owner's view was completely out of character for the animal. Gradually, the ferret allowed gentle palpation of the abdomen and auscultation of the thorax. Palpation of the cranial abdomen revealed a large, firm and irregular mass that was primarily displaced to the right quadrant. Auscultation of the thorax was normal. Peripheral lymph nodes were normal. The mucous membranes were normal in color, with a capillary refill time of less than 1 second. The animal would not allow a rectal temperature to be taken, and the owner declined blood work. Due to the ferret's fractious behavior and its apparent pain, sedation and analgesia were recommended for radiography. At this point, differential diagnoses were lymphadenoma, lymphosarcoma or other neoplasia, abdominal abscess, foreign body or intussusception.

The ferret was sedated with butorphanol (0.1 mg/kg) in combination with medetomidine (0.08 mg/kg) SC.

Radiographs of the thorax and abdomen revealed a diffuse mass to the right cranial abdominal quadrant, corresponding to the height of the left kidney (Fig 1a,b).

Because the radiographs were



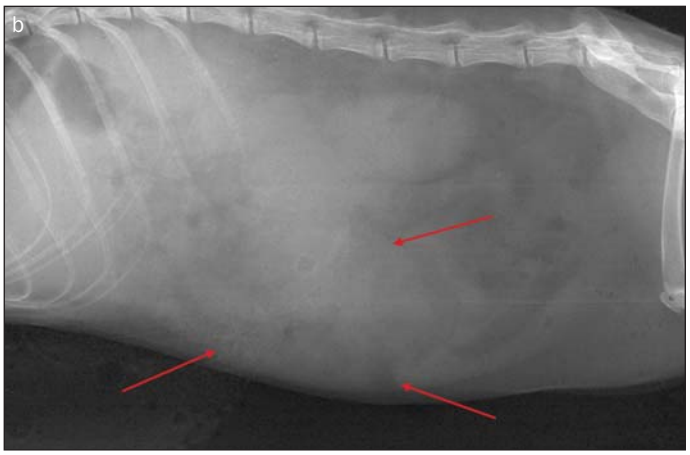


Fig 1a-b. In these ventrodorsal and lateral views, arrows indicate the easily palpable abdominal mass.

inconclusive and the owner declined ultrasonography, a laparotomy was immediately scheduled. The ferret was prepared for surgery while still sedated and anesthetized with sevoflurane via a mask. Anesthesia was maintained via mask using a semi-open circuit. A stable plane of anesthesia was reached between 4-5% sevoflurane and an oxygen flow of 0.7 L/min.

The incision site was blocked locally with 0.7 ml lidocaine/adrenalin, which corresponds to a dose of 14 mg lidocaine and 25.2 µg adrenalin. A long (14-cm) midline incision was performed, and exploration of the abdominal organs revealed a large mass of granulomatous-type tissue adherent to the cranial small intestine, pancreas and the visceral peritoneum (Fig 2).

The appearance, invasiveness and vascularity of the mass seemed too aggressive and difficult to excise, so the surgeon suggested the animal be humanely euthanized.

Samples from the mass and adjacent tissue were sent for histopathology. Based on the appearance of the mass, coronaviral infection and tuberculosis were added to the differential diagnosis list. Tissue samples were then submitted to the Dept. of Veterinary Pathology at the Freie Universität in Berlin, Germany, for immunohistochemistry. Suspected epizootic catarrhal enteritis (ECE), feline infectious peritonitis (FIP)-like disease or tuberculosis lesions were at the top of the differential list for this animal.

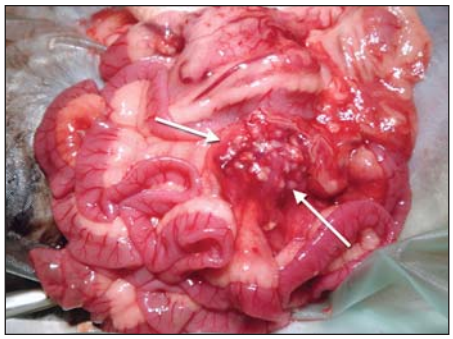


Fig 2. Shown are pyogranulomatous lesions (arrows) in the mesentery. Histopathology revealed granulomatous inflammation in several locations, including the pancreas, omentum and lymph nodes.

Immunohistochemical staining of the submitted adipose, pancreatic and lymphatic tissue and pyogranulomatous lesions confirmed the presence of coronaviral antigen.

Discussion

From 2002 to 2007, 23 domestic ferrets were diagnosed with a novel disease resembling the dry form of FIP.[1] The disease was characterized as progressive, highly fatal and mostly affecting young ferrets less than 3 years of age.[9]

The results of the immunohistochemical staining of the submitted tissue and the clinical signs at presentation confirmed that the ferret described here had a FIP-like disease clinically similar to the dry form of FIP in cats.[5] This is the first case report of this disorder in Norway. Antemortem diagnosis at this point is difficult without immunohistochemistry[1,3] due to the nature of the virus and the severity of the disease.

Blood samples from animals in the early stage of the disease have often showed non-regenerative anemia, hyperglobulinemia, decreased

albumin and thrombocytopenia. Hyperglobulinemia is often present in the serum and can be confirmed by electrophoresis of the serum proteins.[9] However, the differential diagnoses for hypergammaglobulinemia in ferrets should include Aleutian disease, inflammatory bowel disease, multiple myelomas and lymphoma/lymphosarcoma although the spikes for these diseases can be different (monoclonal vs. polyclonal). Aleutian disease parvovirus should be tested in order to rule out the disease.[6]

If early diagnosis and owner compliance are possible, treatment should include prednisone in relative high doses (2-4 mg/kg),[7] in order to suppress the humeral and cell-mediated immune response in the animals. Broad-spectrum antibiotics may also be used to avoid secondary infection due to the immunosupression, with doxycycline as the first choice. A high protein diet, such as Royal Canin Veterinary Diet Convalescence Support Instant Diet,® Hill's a/d® or Oxbow's Carnivore Care,® might help anorectic animals. A, D, E and B-vitamin complexes and other antioxidants should be administered as supportive care.[6,7] Nevertheless, all owners should be informed that the prognosis in most cases is grave.

Acknowledgements

The author wishes to thank the following people for assistance with this case: Steffi Deppenmeier at Laboklin GmbH; Prof. Dr. Achim Gruber, Department of Veterinary Pathology at De Freie Universität in Berlin; Fredrik Venold, DVM; and Dr. Estella Böhmer, Ludwig-Maximilians-Universität München.

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Umbilical Hernias in Rabbits

William G.V. Lewis, BVSc, CertZooMed, MRCVS, The Wylie Veterinary Centre, Essex, United Kingdom

Although umbilical hernias are rarely reported in the literature, the author has encountered at least 4 cases in the past 3 years, one of which was fatal. Anecdotally, a client reported that an umbilical hernia in his rabbit had been corrected during an ovariohysterectomy performed by another veterinarian.

Surviving Cases

A small umbilical hernia was discovered when a 6-month-old rabbit was presented for routine neutering. This was corrected using standard surgical procedures during the performance of the ovariohysterectomy.

In 2 other young rabbits (1 year and 2.75 years of age, respectively), an umbilical hernia was discovered in each during routine neutering. The former rabbit had previously given birth to a litter; it was assumed the umbilical hernia had developed subsequent to that (Fig 1a-c).

In all 3 cases above, no clinical signs other than swelling were noted. During the surgical correction, mesentery was noted protruding through the hernia and had adhered to the muscle. No organs were entrapped. All muscle edges were freshened with a scalpel blade, and the midline incision was closed with 3-0 Vicryl® in a continuous pattern. The skin was closed with intradermal Vicryl® and meloxicam (0.3 mg/kg PO q12h x 7d) was used as an analgesic (Fig 2a-d).

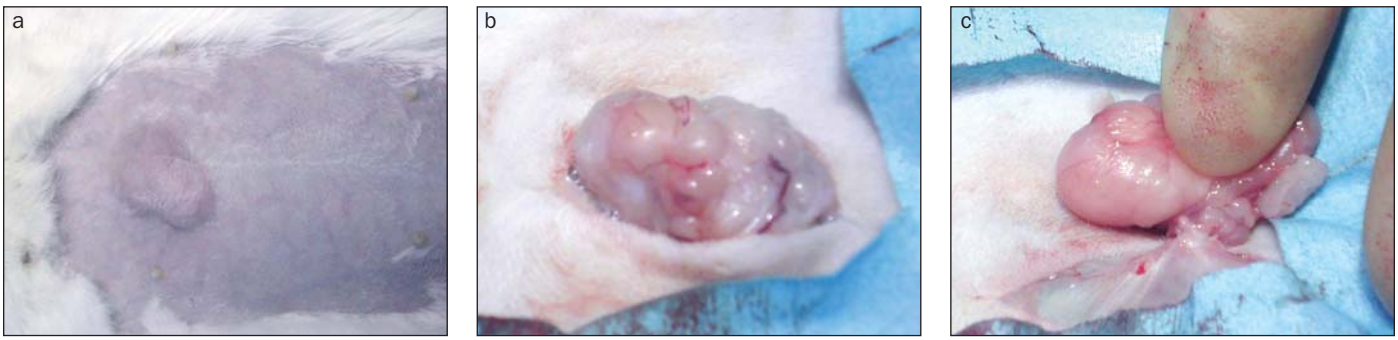
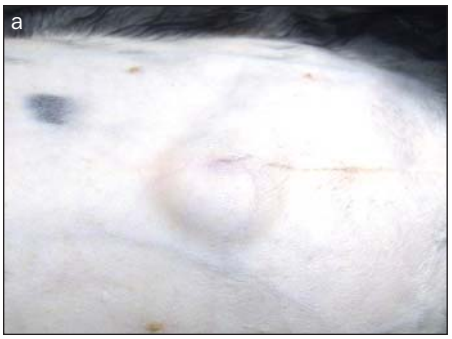
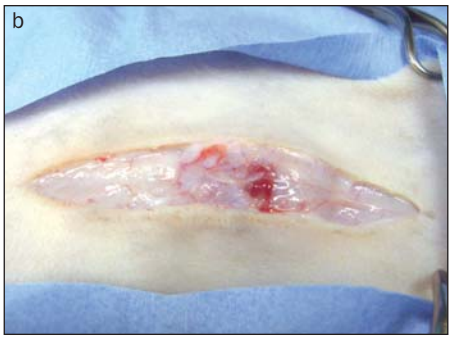
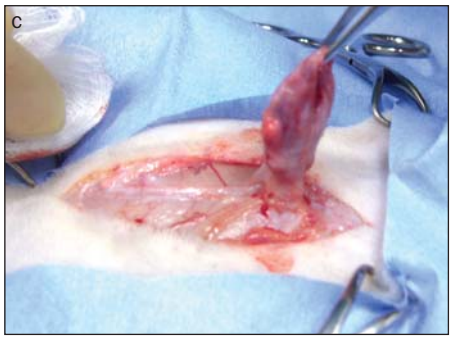
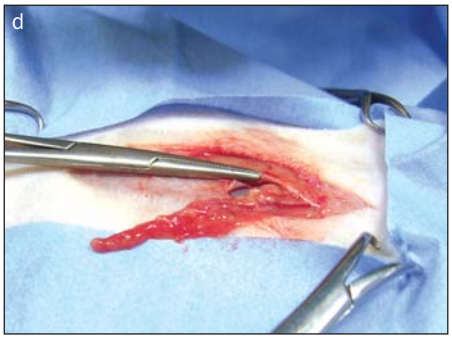


Fig 1a-c. This rabbit had no history of a hernia until after it had a litter. The hernia is visible through the skin. The fat protruding through the hernia was reduced.









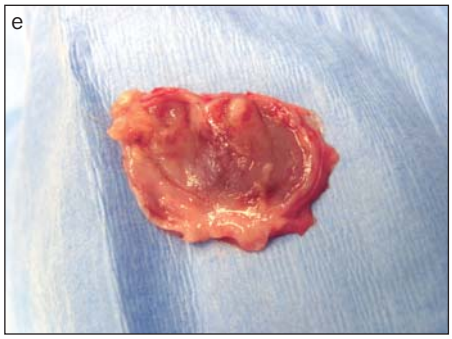


Fig 2a-e. Umbilical swelling is visible through the skin. Strangulated tissue, which appears to be fat, is seen protruding through the hernia. The tissue is dissected free. The protruding tissue was removed during surgical correction of the hernia.

Fatal Case

A 2.5-year-old rabbit was found dead in the hutch with no recent clinical signs. At necropsy, a swelling was palpable in the umbilical region. A postmortem confirmed a strangulated small intestine in the hernia. The intestine proximal to the herniation was dilated with fluid and gas, and the section distal to it was devoid of any contents, indicating a complete strangulation (Fig 3a-g). These findings led to a presumptive diagnosis of ischemic necrosis of the intestine, shock and subsequent death.

The rabbit had previously been spayed at 6 months of age, but the surgical incision was caudal to the umbilicus (Fig 4). One year later it had been presented straining as if having difficulty urinating. It made an uneventful recovery with symptomatic treatment (analgesics, antibiotics, supportive care) at that time, and 4 months later was presented dead.

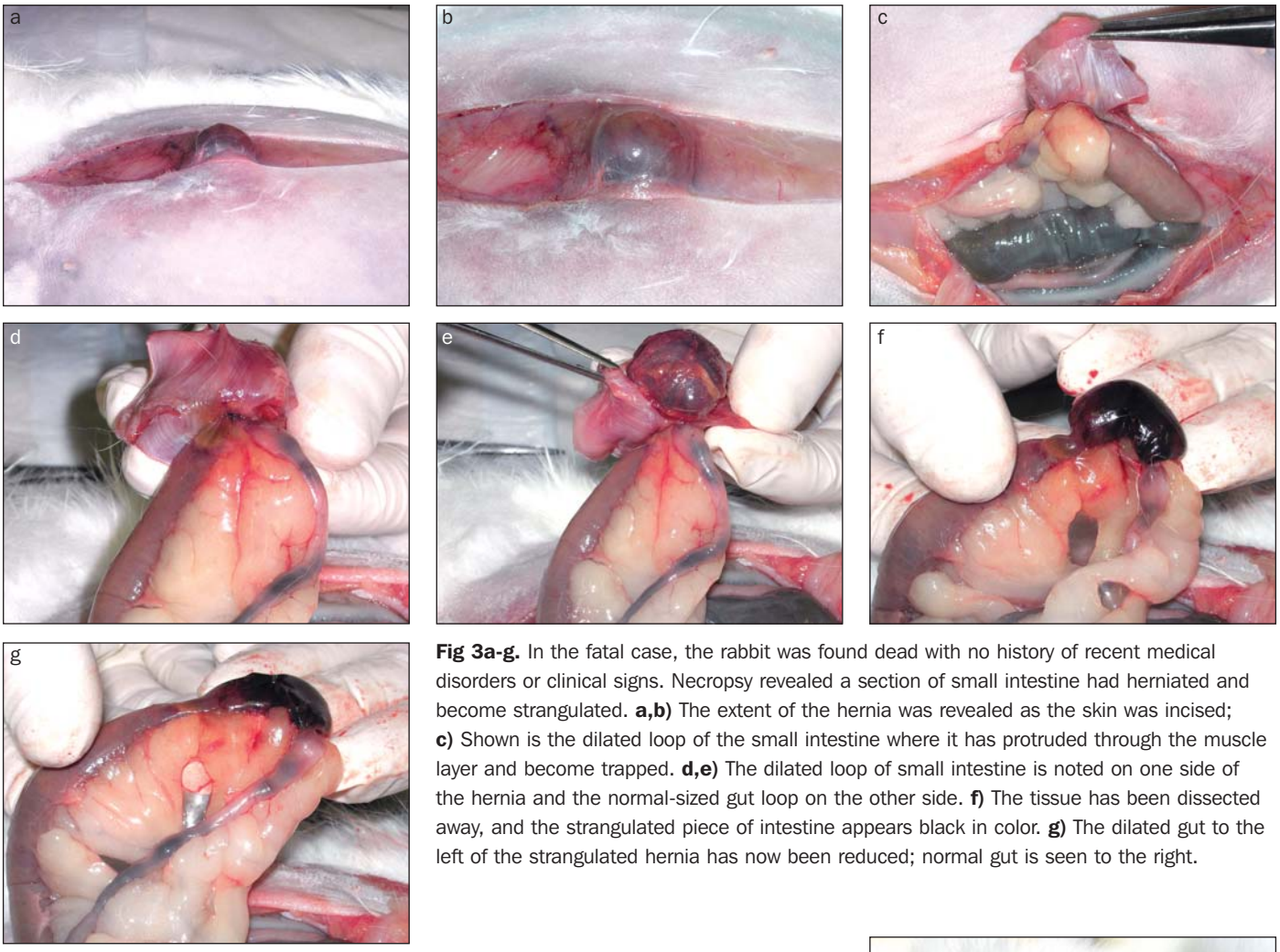


Fig 3a-g. In the fatal case, the rabbit was found dead with no history of recent medical disorders or clinical signs. Necropsy revealed a section of small intestine had herniated and become strangulated. **a,b)** The extent of the hernia was revealed as the skin was incised; **c)** Shown is the dilated loop of the small intestine where it has protruded through the muscle layer and become trapped. **d,e)** The dilated loop of small intestine is noted on one side of the hernia and the normal-sized gut loop on the other side. **f)** The tissue has been dissected away, and the strangulated piece of intestine appears black in color. **g)** The dilated gut to the left of the strangulated hernia has now been reduced; normal gut is seen to the right.

Fig 4. The rabbit was prepared for its previous ovariohysterectomy over 1 year prior to its death. The incision site was caudal to the umbilicus (arrow), suggesting the subsequent hernia was not iatrogenic.

Proventricular Intussusception in an Indian Peafowl

David Perpiñán, LV, MSc; Jamie N. Henningson, DVM and Douglas L. Armstrong, DVM

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Intestinal intussusception in avian species is infrequently reported and is most often associated with impaction, parasitism and enteritis.[1-4] Intussusception of the proventriculus is a rare condition in birds. Of the few cases described in the literature, it appears to be more common in young animals.[5-7] This paper presents a case of proventricular intussusception in an Indian peafowl (*Pavo cristatus*).

A free-ranging young female Indian peafowl was found moribund in a zoological collection. The bird was easily captured and elicited no response. It was placed in an incubator at 29°C (84°F) but died 2 hours later. At necropsy, the bird weighed 794 g, with a body condition score of 3/9 (1 = emaciated and 9 = obese). The most significant gross lesions were present in the gastrointestinal tract, where the ventriculus was severely distended. Upon incision, the ventricular wall was thin, and the koilin layer was brown. No grit or ingesta was present. A round ulcerated mass was present in the cranial part of the ventriculus, representing the completely invaginated proventriculus (Fig 1). The wall

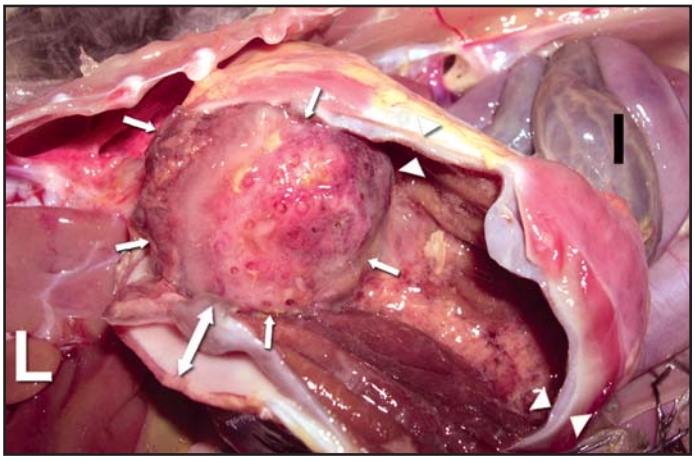

Fig 1. Intussusception of the proventriculus in an Indian peafowl. The ulcerated mucosa of the inverted proventriculus can be seen (long arrows). The wall of the ventriculus is thinned (arrow heads), and the koilin layer separates easily from the ventricular wall (double arrow). L = liver; I = intestines.

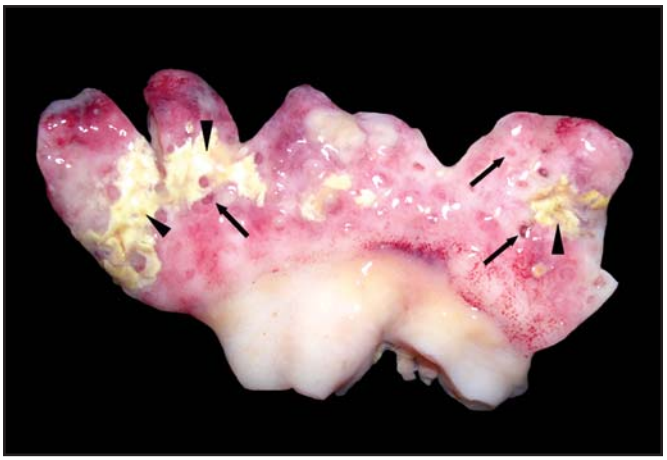

Fig 2. Mucosa of the intussuscepted proventriculus showing ulceration (long arrows) and deposits of fibrin (arrow heads).

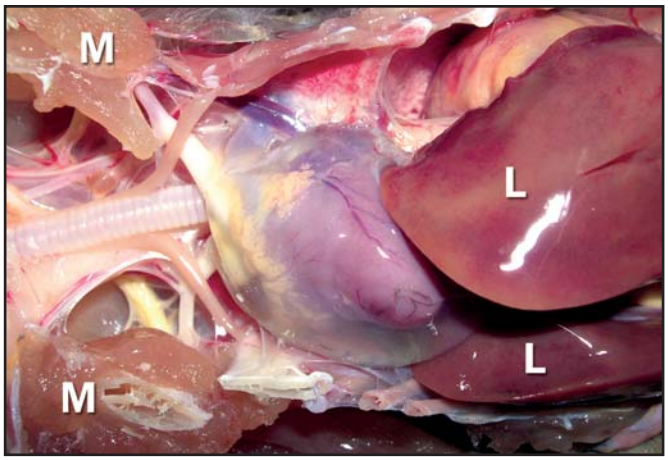

Fig 3. Hydropericardium (center of image) and generalized pallor affecting liver and muscles. L = liver; M = muscles.

of the proventriculus was thickened, and the mucosa was hyperemic and ulcerated with plaques of fibrin attached (Fig 2). Other gross lesions included hemorrhagic ceca, hydropericardium, and general pallor (Fig 3). A direct fresh smear of cecal contents was negative for parasites.

Histologically, the ventriculus was characterized by abnormal maturation of the koilin layer. Numerous varying-sized foci of a basophilic substance were observed in the keratinoid layer along with scattered degenerate heterophils. Severe ulceration was present in the proventriculus (Fig 4). Moderate numbers of heterophils and mats of condensed fibrin lined the ulcerated areas. In addition, severe diffuse fibrosis was noted in the areas of ulceration. There was also suppurative and diffuse inflammation of the crop, esophagus and intestine. Gomori methenamine silver stain was negative for fungal organisms, but Gram staining demonstrated gram-positive rods in ulcerated regions. No microbiological cultures were performed.

Discussion

Proventricular intussusception is an extremely rare condition in birds, with only a few reports dating from several decades ago.[5-7] The pathogenesis of proventricular intussusception remains unclear, but factors that may play a role in its development include changes in the thickness of the wall of the ventriculus and proventriculus and abnormal peristalsis secondary to local lesions, such as ulcers, scars, swelling or growths.[6,7]

The etiology of the case presented here is unknown, although the gram-positive bacterial infection may have produced ulcerative and fibrinonecrotic gastroenteritis leading to intussusception of the proventriculus. A series of 4 cases in chickens reported proventricular intussusception to be acute in duration;[7] however, some chronicity was observed in this case, as indicated by the presence of fibrosis in the proventriculus and esophagus. The hydropericardium may have been the result of wasting leading to hypoproteinemia,[8] as there was no ingesta present in the ventriculus. The ventricular wall may have been thin due to loss of body condition. These facts support the observation that death in this Indian peafowl may have been due to anorexia secondary to the proventricular intussusception or inflammation of the gastrointestinal tract. Another author also reported this condition to produce chronic clinical signs.[5] Although this condition is rare in birds, it is interesting to note that one of the reported cases also involved a young Indian peafowl with distension of the ventriculus and bacterial proventriculitis with fibrin and fibrosis.[6]

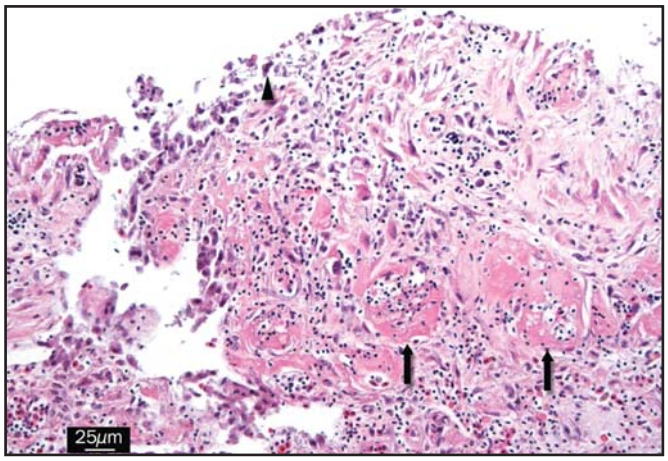

Fig 4. Ulcerated mucosa of the intussuscepted proventriculus in an Indian peafowl. Epithelial necrosis (arrow head), fibrin thrombi (long arrows), and diffuse heterophilic infiltrate and severe fibrosis are evident.

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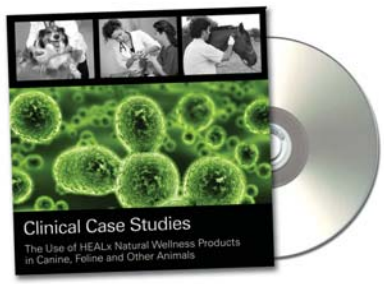

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Pulmonary Carcinoma in a Captive Fennec Fox

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A 7-year-old castrated male fennec fox (*Vulpes zerda*) was presented with a 10-day history of coughing and regurgitation immediately after eating. The owner also noted that the fox's water consumption was decreased and its urine appeared more concentrated. The fox had been neutered approximately 3 years prior and had no other medical history. The animal was not receiving any medications.

The fox was in adequate body condition (body weight 2.82 kg). It was lethargic on physical examination with tacky mucous membranes. Its temperature was elevated at 102.9°F (39.4°C).[6] No other abnormalities were noted. Complete blood count and serum biochemistry values were within reference ranges.

Thoracic radiographs revealed a mass in the cranial mediastinum causing dorsal deviation of the esophagus cranial to the heart. Bicavitary ultrasonographic examination revealed a right cranial lung lobe mass measuring 1.43 cm x 1.64 cm (Fig 1) with a normal abdomen and no evidence of metastasis. A fine needle aspirate of the mass yielded a caseous yellow material on gross examination. Cytology, bacterial culture and fungal culture were submitted. The patient was discharged with famotidine (1 mg/kg PO q12h),[8] amoxicillin clavulanate (Clavamox drops, 13.75 mg/kg PO q12h)[8] and instructions for elevated feedings.

Cytological examination of the aspirate did not provide a definitive diagnosis but was consistent with a cyst, an area of necrosis or an obstructed bronchus. Aerobic and anaerobic bacterial cultures of the aspirate yielded no growth.

The patient was re-examined 5 days later and found to be depressed with a persistently elevated temperature of 103.1°F (39.5°C). The owners reported that the fox continued to regurgitate and had started vomiting. Repeat thoracic radiographs (Fig 2) showed a persistent mass in the right cranial thorax. Due to the persistent regurgitation and vomiting, nondiagnostic aspirate sample and progression of clinical signs, neoplasia was suspected. Surgical excision was recommended.

A right intercostal thoracotomy was performed to remove the right cranial lung lobe. The fox was induced with IV propofol (13.5 mg) to effect. Cefazolin (22 mg/kg) and buprenorphine (0.01 mg/kg) were administered IV at induction. The fox was intubated and maintained on isoflurane (3%) using a nonrebreathing circuit.

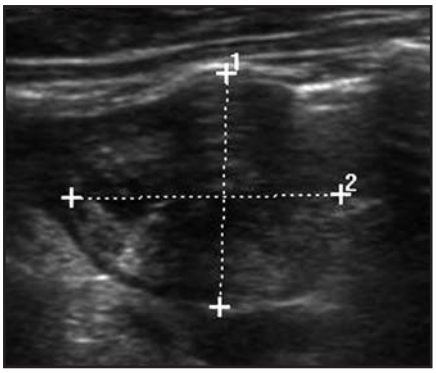

Fig 1. Sonograph of the right cranial thoracic mass. The mass measures 1.43 cm (1) x 1.64 cm (2).

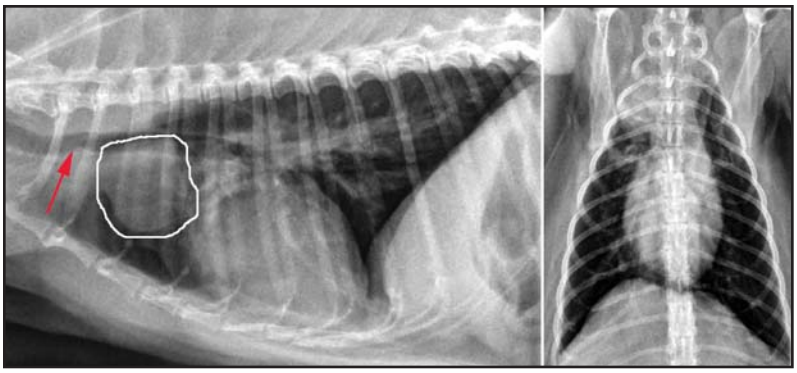

Fig 2. Right lateral and ventrodorsal thoracic radiographs of a fennec fox showing a single mass (circle) cranial to the heart base in the right lung field, causing dorsal deviation of the esophagus (arrow).

ECG, noninvasive blood pressure, pulse oximetry and capnography were monitored during surgery.

During the 4th intercostal thoracotomy approach, the 3rd, 4th and 5th ribs were fractured. The mass was soft and tan in coloration and measured 4 cm x 3 cm. It included the right cranial lung lobe hilus and was intimately associated with the great vessels of the heart. The local tumor invasion into the heart base caused the vasculature to be very friable. There was no evidence of metastasis within the thoracic cavity. Hemorrhage could not be controlled and humane euthanasia was recommended and elected. A full necropsy was not performed.

Histopathology of the mass was consistent with pulmonary carcinoma, pleocellular and neutrophilic inflammation. Fungal cultures from the fine needle aspirate had heavy growth of an *Aspergillus* sp.

Discussion

Wild canids, such as the fennec fox, are expected to have similar prevalence rates and types of neoplasia as the domestic dog.[7] Reports of neoplasia are limited, especially in fennec foxes.[3]

Nutritional and metabolic bone diseases are common in many exotic species.[11] Domestic dogs are unable to synthesize adequate amounts of vitamin D and therefore require a dietary source.[11] Both calcium and phosphorus levels in this patient were considered within normal limits[6] with an appropriate calcium-to-phosphorus ratio of 1:2.3. The diet of this fox, a commercial dog kibble, was also appropriate and should have been a nutritionally complete diet.[6] The fragility of this fennec fox's ribs may be normal for this species or may be secondary to pathology not detected by routine radiography and serology.

Primary pulmonary neoplasia is uncommon in domestic dogs, with metastatic pulmonary neoplasia being much more common.[1,12] Almost all primary pulmonary tumors are carcinomas.[12] In patients with solitary lesions, surgical excision is considered the most effective treatment.[1,5,12] Radiation therapy may be used but requires intensity-modulated radiation therapy[12] or CyberKnife® radiosurgery. Chemotherapy with single or multiple agents has also been used. Use of vinorelbine is promising in dogs with a 28.5% response rate for bronchoalveolar carcinoma.[9] Computerized tomography (CT) may be used prior to any treatment for staging and obtaining biopsy samples. The presence of an *Aspergillus* sp. may be similar to cases in humans with concurrent cancer and a canine case where actinomycosis was found concomitant with pulmonary neoplasia.[2] It is unknown if the fungal infection is clinically significant or if it can mask or delay a diagnosis of neoplasia. Other forms of pulmonary disease have been reported in the fennec fox, such as tuberculosis,[4] histoplasmosis[10] and pulmonary fibrosis.[3] To the authors' knowledge, this is the first documented case of pulmonary carcinoma in a fennec fox.

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Surgical Correction of a Rectal Prolapse in a Pet Skunk

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Angela Lennox is a 1989 graduate of Purdue University. She currently owns the Avian & Exotic Animal Clinic in Indianapolis. In 2005, she was presented the Exotic DVM of the Year Award. Dr. Lennox is a past president of the Association of Exotic Mammal Veterinarians. She was the editor for the Rabbit and Rodent Dentistry Handbook and Clinical Radiology of Exotic Companion Mammals.

Kevin Bowman

In many states, ownership of skunks as pets requires special permitting, regardless of whether they have been wild-caught or domestically raised. Owners acquire skunks through breeders, from pet stores and occasionally, as injured or orphaned wildlife. Domestically raised skunks have been bred to some degree for pet quality and unusual color patterns, and anecdotal reports indicate these generally are better pets than wild-caught animals.

Skunks sold in pet stores and most individuals offered by breeders are descented prior to purchase. However, in some cases, practitioners may be requested to descent them by performing a bilateral anal sacculectomy. While illegal in many parts of Europe, elected anal sacculectomy is considered a legitimate procedure in the United States.

Due to the relatively large size of the anal sacs even at an early age, the ductal approach is recommended. Complications include damage to the anal sphincter and resulting rectal prolapse.

Surgical Procedure

An 8-week-old female skunk weighing 3.5 kg was presented with prolapsed rectal mucosa. Surgical correction of the disorder was scheduled.

Preanesthesia was administered with diazepam (2 mg/kg SC). Induction of anesthesia was achieved with an IM combination of medetomidine (10 mcg/kg) + ketamine (10 mg/kg). Anesthesia was maintained with isoflurane (1-2%) delivered by endotracheal tube. Intraoperative pain control was achieved with carprofen (2 mg/kg IM) and maintained at 1 mg/kg q12h PO for the following 3 days.

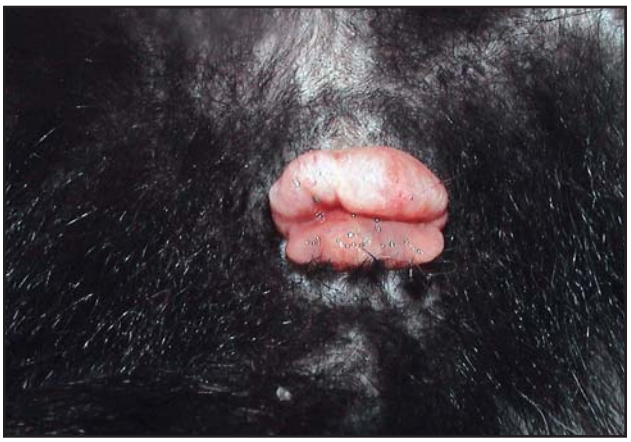

Fig 1. Appearance of prolapsed rectal mucosa in an 8-week-old female skunk previously descented using the ductal approach. Descenting had been performed prior to the sale from a pet shop; the age at the time of the surgery was unknown.

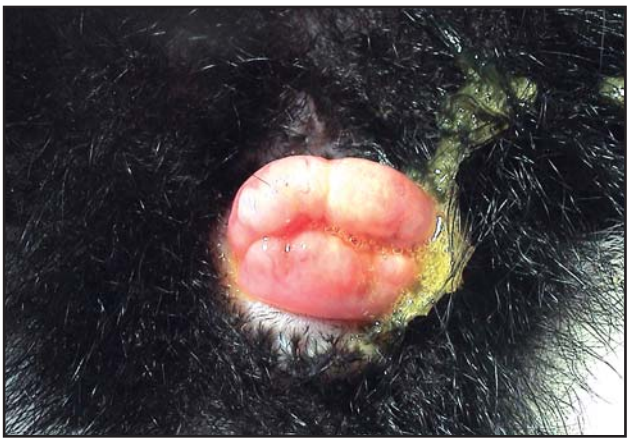

Fig 2. Defecation caused exacerbation of the prolapse. In this case, a recent meal of vegetables produced yellow-pigmented feces.

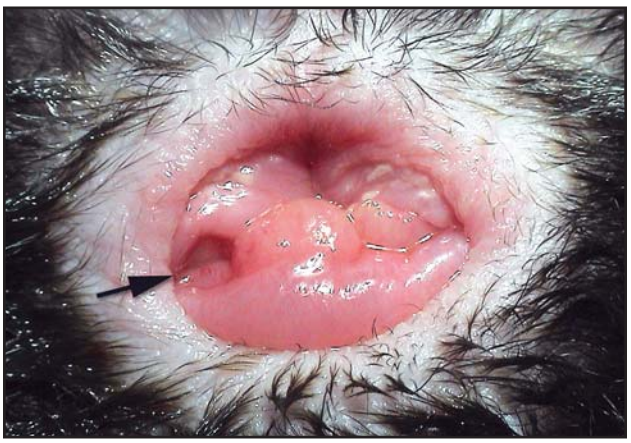

Fig 3. Closer inspection under anesthesia revealed two lesions of the internal sphincter muscle at the site of previous removal of the anal sacs and ducts. The lesion on the right (arrow) is more severe than that on the left. The skunk is positioned in dorsal recumbency.

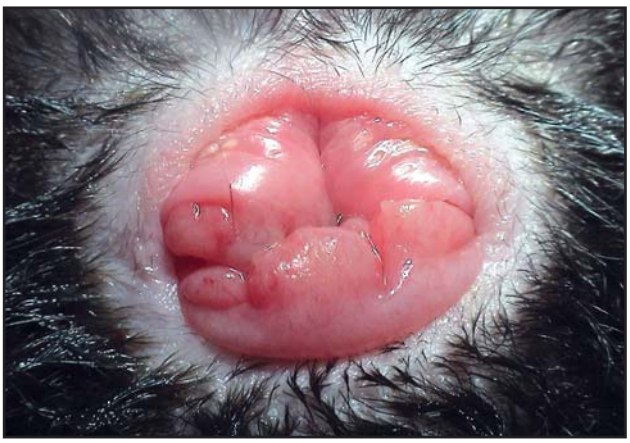

Fig 4. The goal of surgery is to debride and close the defects, restoring the integrity of the internal sphincter muscle. The mucosa of the internal sphincter is separated from the muscle to allow proper apposition of the muscle for suturing.

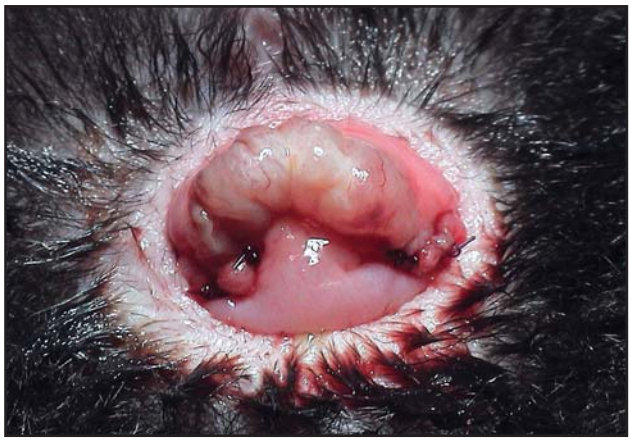

Fig 5. After debridement, two simple interrupted sutures using 3.0 absorbable monofilament are placed to close the defect of the muscle and mucosa.

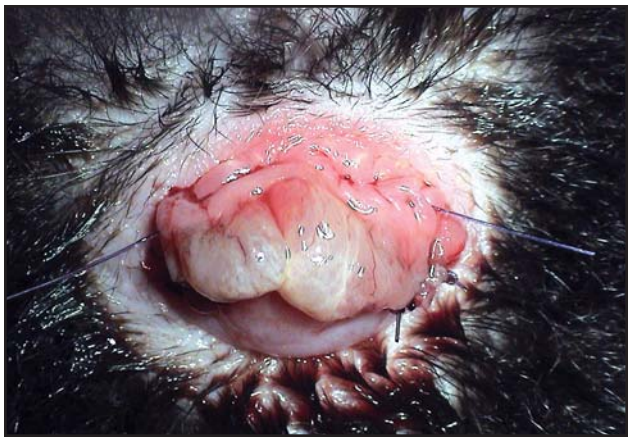

Fig 6. Despite correction, the prolapse is still evident, likely due to edema of the tissues.

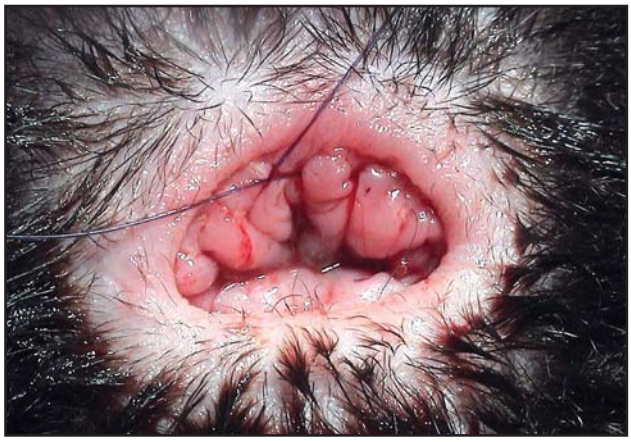

Fig 7. Placement of a 3.0 nylon purse string suture allows correction of the prolapse and time for the internal sphincter to heal.

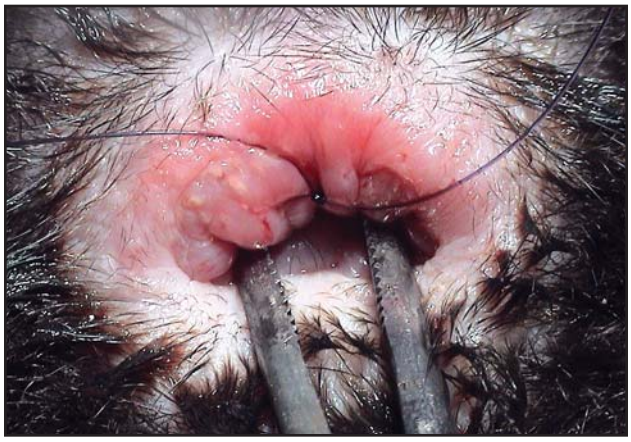

Fig 8. The suture is tightened to produce resolution of the prolapse yet still allow normal passage of feces.

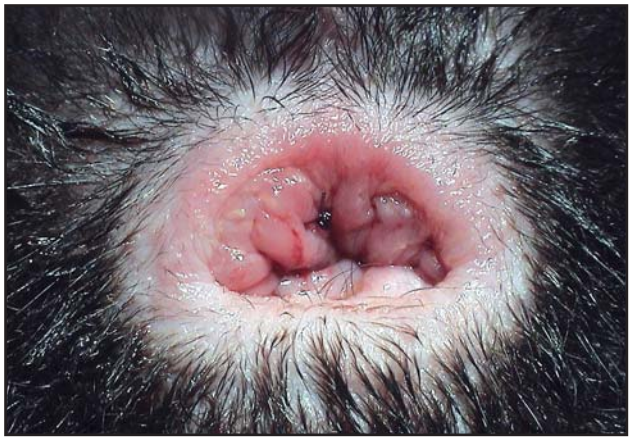

Fig 9. Shown is the postsurgical appearance following suture of the ductal incisions (not visible) and the purse string suture of the internal sphincter. Postoperative antibiotic treatment was provided by amoxicillin/clavulanate (10 mg/Kg q12h PO).

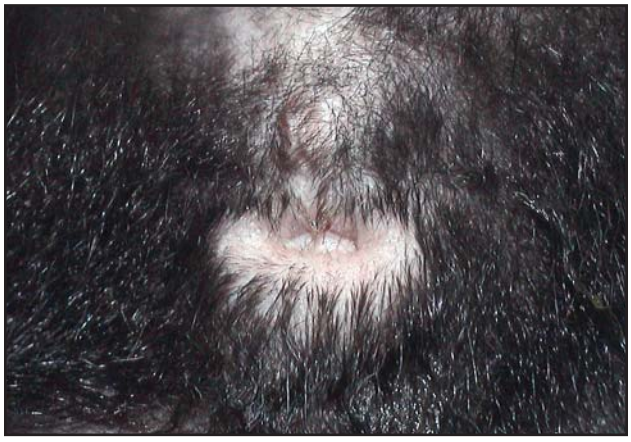

Fig 10. Follow-up 10 days post surgery and immediately after suture removal shows normal appearance of the anal sphincters.

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Ultrasound-guided Liver Biopsy in an Argentine Boa

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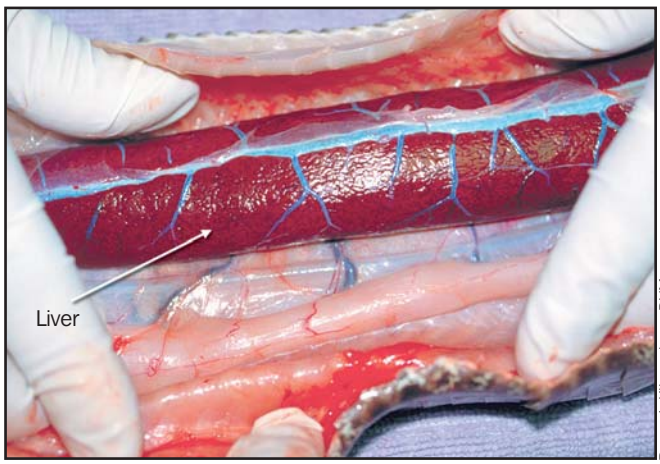
Daniel Gray received his DVM degree from Iowa State University in 2004 and spent his first 2 years practicing in Dubuque, Iowa. He is currently associated with the Gentle Vet Animal Hospital, where he pursues his interest in birds, reptiles, exotic mammals, zoo animals and wildlife medicine and surgery. He is consulting veterinarian to the Northeastern Wisconsin Zoo and the Bay Beach Wildlife Sanctuary as well as a Veterinary Medical Officer with the U.S. Department of Health and Human Services, which responds to federal and state disaster emergencies.

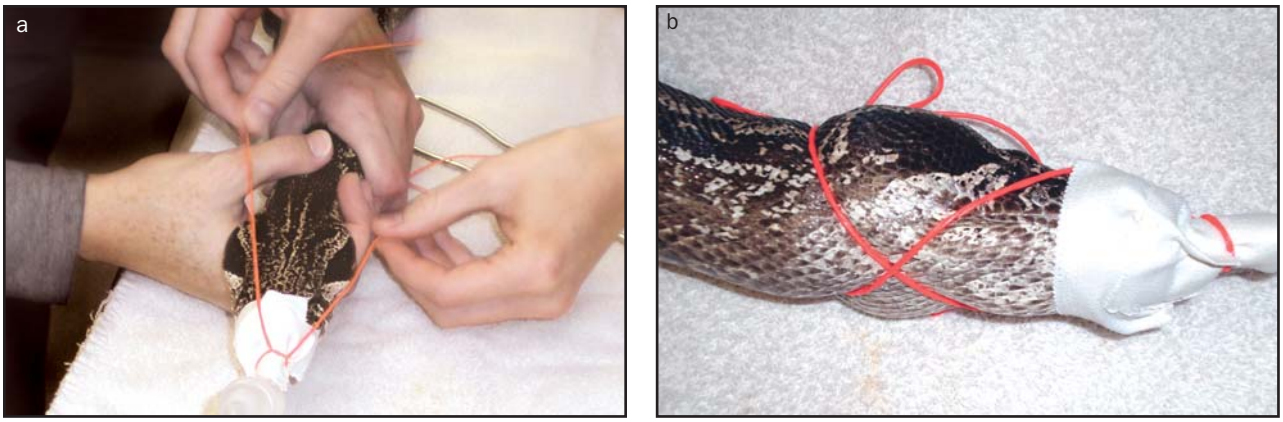
Inclusion body disease (IBD) is the most important viral disease of snakes that affects captive collections of boas and pythons. IBD has been associated with high morbidity and mortality rates. It can be a devastating and frustrating disease, especially to owners of rare breeding stock.

A female Argentine boa (*Boa constrictor occidentalis*) was presented after the sudden death of its mate with which it had produced a clutch of viable young. A necropsy of the male confirmed IBD via histopathology. The owner requested an IBD screening test of the female. Based on a review of the literature, it was determined that the best approach was evaluation of a liver biopsy. Due to the owner's concerns about a full excisional biopsy and related costs, the decision was made to obtain a core liver biopsy guided by ultrasonography.



Roger J. Klingenberg, DVM

In snakes, the liver lies on the right side of the body and is a large organ.



Figs 1a,b. To assure proper positioning, the snake was intubated while conscious with a 4-mm avian tracheal tube. Anesthesia was induced and maintained with isoflurane gas throughout the procedure. The tube was kept in place with white tape and orange ties.

Fig 2. Once the righting reflex was absent, the snake was placed in a ventrodorsal position for the ultrasound scan. The position of the liver was determined, and the approximate location was marked externally with cloth tape for reference.

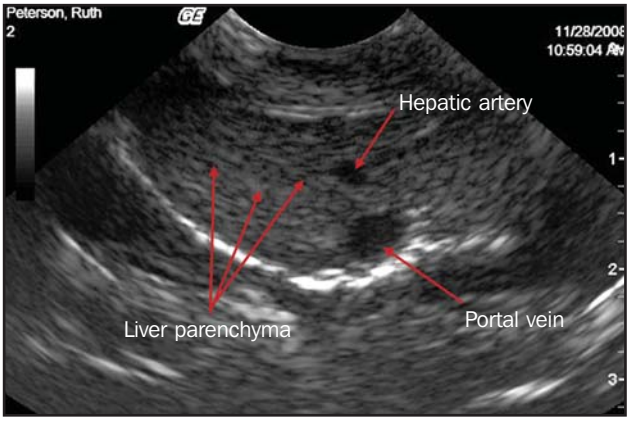


Fig 3. The organ was scanned with the ultrasound probe (LOGIQ Book XP variable megahertz probe, GE, www.gehealthcare.com). This ultrasound image represents the entire liver with its parenchyma and associated major vessels. The liver appeared to be of good symmetry and consistent echogenicity throughout. Care was taken to avoid inserting the biopsy needle close to the major vessels.

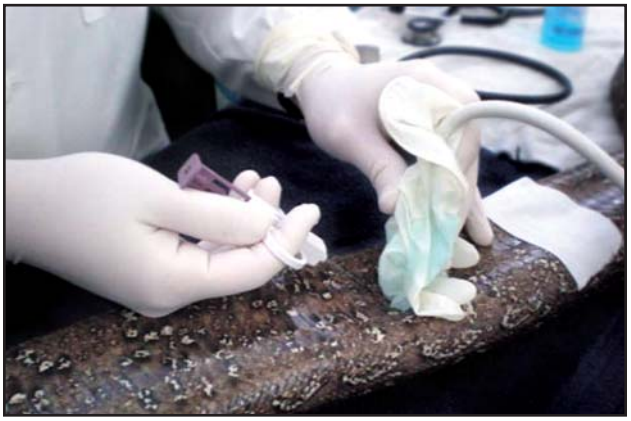


Fig 4. The needle insertion site was cleaned with 3 sets of alternating diluted chlorhexidine and alcohol scrubs. A sterile latex glove was filled with ultrasound gel and wrapped around the probe to prevent contamination of the biopsy site with the gel. A representative area of the liver was selected, and a SuperCore™ 16-ga x 9-cm biopsy needle (Jorgenson Laboratories, www.jorvet.com) was inserted between two scales into the liver.

A core sample of the liver was collected and extracted. The core was placed in formalin, and the procedure was repeated in a separate site to obtain a second representative sample. The samples were submitted for analysis (Northwest Zoo Path, www.zoopath.com). Although the biopsy results were negative for IBD, the snake may not be free of the disease. Boas with IBD do not always have viral inclusions in the liver. IBD is transmitted among boas through direct and indirect contact. Because this boa bred with the infected snake that died, it should be considered infected. The snake is currently in isolated quarantine.

References and Further Reading

1. Garner M, Raymond J: Methods for diagnosing inclusion body disease in snakes. Exotic DVM 6(3):90-92.
2. Klingenberg R: A fast and efficient liver biopsy technique in snakes. Exotic DVM 3(3):89-93.
3. Mader, DR (ed): Reptile Medicine and Surgery 2nd ed. Elsevier, 2006.

Forum Moderator: **Marc Kramer**, DVM, Miami, Florida, exoticdvm@mac.com

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Exotic DVM Online Forum

The Exotic DVM Online Forum was developed to provide an opportunity for a daily interaction among veterinarians and veterinary students regarding their experiences with exotic pet species. Occasionally some conversations are published in Exotic DVM veterinary magazine, but the real action takes place online. Here's what some of the members have to say about this ***free*** service.

"The Exotic DVM Forum is like 'Continuing Education' delivered directly to my INBOX. As a Forum member, I can read the questions and answers posted by 100s of exotic animal veterinarians from the US and abroad. Eavesdropping on these conversations provides me some of the most current information available. I learn about new drug protocols, emerging diseases, and other information months or even years before it appears in print. I am able to get assistance with difficult cases and share ideas with colleagues who have read journals and attended meetings that I haven't."
Dan Johnson, DVM

"I've been a member of the Exotic DVM Forum since its origin, and it keeps me on my toes with the constant influx of exotic medicine advances—information that I might otherwise not hear about until I receive a new journal volume or attend an upcoming conference. As a solo practitioner, it is great to be able to get quick perspectives on cases that are often not readily researchable within my clinic library or on-line."
Maxwell Conn, DVM

"I've been using the EDVM forum for many years, and find it a useful place to query colleagues on tough exotic animal cases. I get answers very quickly, and the archives allow quick access to subjects discussed in the past. Images and radiographs can be uploaded and posted for colleague comment. Overall the Forum has been a great resource."
Angela Lennox, DVM Dipl ABVP-Avian

"The Exotic DVM Forum provides a unique platform for the exchange of knowledge amongst veterinarians who treat exotics. Specialists discuss and share their experience and assist beginners who are always welcome. If you're an Exotic DVM, here is where you will meet your colleagues from all over the world."
Kornelis Biron, Dr med vet

Neurologic Damage to the Spinal Cord of a Rabbit

(In response to a question about a 2-year-old rabbit that suddenly stopped hopping and began dragging its back legs): A mild neurologic lesion of the spinal cord is relatively frequent in rabbits following many traumas (sometimes self-trauma) to the distal thoracic tract/lumbar tract of the spine. As in other mammal species, a tentative diagnosis and localization of the lesion may be performed with an accurate neurologic exam. Details of a neurologic examination are available in the following references, which can be accessed online:

- Osofsky A, LeCouteur RA, Vernau KM: Functional neuroanatomy of the domestic rabbit (*Oryctolagus cuniculus*). Vet Clin No Am Exot Anim Pract 10(3):713-730, 2007.
- Vernau KM, Osofsky A, LeCouteur RA: The neurological examination and lesion localization in the companion rabbit (*Oryctolagus cuniculus*). Vet Clin No Am Exot Anim Pract 10(3):731-758, 2007.

The alleged conscious pain perception of the hind limbs should be carefully checked several times and compared with the forelimbs. The unconscious spinal reflex in rabbits may be

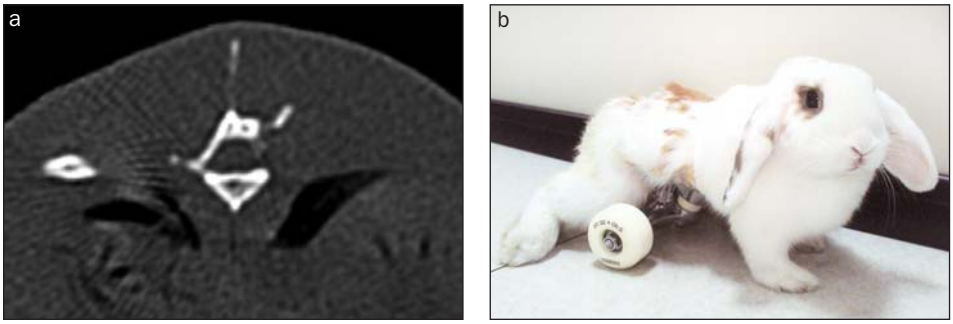

Fig 1a,b. A 5-month-old intact male rabbit suffered a partial fracture of T12 and was paraparetic. A homemade cart (skateboard wheels) was used with the animal before a surgical hemilaminectomy for decompression was scheduled. Eventually, the rabbit recovered the limb function. **a)** CT image (axial view) of the fracture of the left mammillary process of the 12th thoracic vertebra, resulting in compression of the spinal cord. These lesions are almost impossible to be diagnosed even with very good radiographs and require advanced diagnostic techniques. **b)** Shown is the rabbit on the cart. Note the abnormal neurologic posture of the right foot suggesting lack of proprioception.

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very evident, even in cases of severe paraparesis or paraplegia, misleading the veterinarian to a wrong diagnosis and a positive prognosis. In case of conscious pain perception, the rabbit must show clear pain (visible as a painful expression or "bothered" face), not just retract the limbs without showing any apparent interest in its toes being pinched.

Survey radiographs are frequently unremarkable (especially when the rabbit is paraparetic and not paraplegic), both because a bony lesion may not be present (just a spinal shock) or because it is technically impossible to see it, and advanced diagnostic techniques are needed.

I would recommend MRI, CT or both as soon as possible, in order to refine the clinical diagnosis and understand if decompressive surgery may be an option; otherwise medical treatment (with proper patient management) is the only option.

Symptomatic treatment should be directed according to the diagnosis and may include steroids in selected cases. Antibiotics are usually not helpful, apart from support when steroid therapy is administered.

Repair of Aural Hematoma in a Rabbit

Hematomas of the ear pinna in rabbits may be treated the same way as in dogs. Some different techniques are possible in order to keep pressure over the skin and cartilage (e.g., short pieces of plastic tubes, plastic patches, even small round pieces of radiographic films); all are stabilized with transfixing sutures. One must avoid the central artery of the ear pinna. Although rabbits tolerate these well, it is best to avoid tightening the suture too much, as this may result in discomfort. In lop ear breeds, the tips of the ears, rather than the entire pinna, can be taped together, which allows easy access for postoperative inspection of the surgical site. Sutures are removed in 7-10 days.

A possible complication is the same as in dogs: the base of the ear pinna may become someway "shrunk." This, associated with the narrower ear canal in this species and the unnatural position of the ear pinna, may be predisposing factors to chronic otitis.

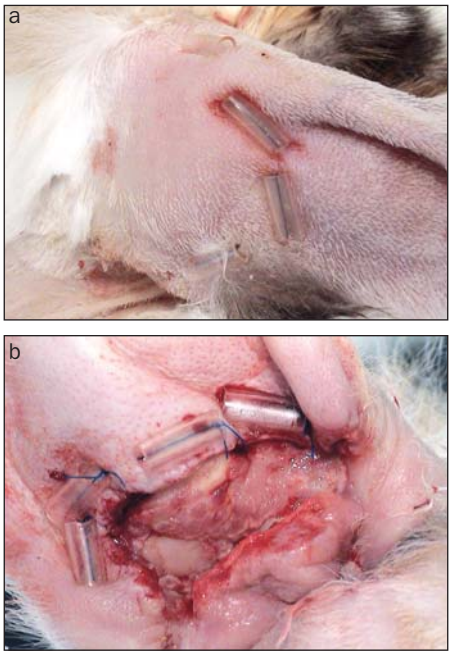

Fig 2a,b. Compression on the ear pinna of a lop eared rabbit is shown using small soft plastic tubes and transfixing them with 3.0 non-absorbable polypropylene suture. This rabbit underwent total ear canal ablation following bacterial otitis externa and media, but this technique can be applied to aural hematomas as well. **a)** Dorsal view of the ear pinna. **b)** Ventral view of the ear pinna.

Guinea Pig Intubation

Orotracheal intubation is significantly more difficult in guinea pigs than in rabbits. Both species are obligate nasal breathers, because the margin of the epiglottis lies beneath the margin of the soft palate. But the blind technique is not recommended for guinea pigs for the following reasons:

- Guinea pigs are generally smaller than rabbits—even when the weight is similar (approximately 1 kg), the rhinopharynx is smaller.
- Guinea pigs (plus other porcupine-like rodent species and the prairie dog) have a "pharyngeal ostium." The mucosa of the pharynx forms a type of "ring" ending ventrally with the most caudal portion of the tongue. The rhinopharynx (where the soft palate and the epiglottis meet) is beyond this ostium.
- Unless they have been completely anorexic for several days, guinea pigs always have food debris in their mouth. Food debris fouls the tip of the endoscope and can even be pushed into the trachea.

The steps for successful endoscope-guided intubation of guinea pigs (which require experience, time, skill and a good portion of luck) are:

1) Induction with injectable agents to a good anesthetic plane
2) Proper positioning on the table, use of a mouth gag and proper hyperextension of the head. This is critical, because when a rigid endoscope is used, it cannot follow the slight curve of this region, and visualization will be suboptimal.
3) Thorough cleaning of the mouth and the pharynx
4) Cleaning of the lens of the endoscope
5) Passing the pharyngeal ostium
6) Gently touching the soft palate to allow disengagement of the epiglottis and visualization of the laryngeal opening. One must see the laryngeal opening to avoid inserting the endotracheal tube into the esophagus.
7) Introduction of the 1.5 mm endotracheal tube (with the stylet) through the mouth. At this point, there is usually not enough room to keep both the tip of the endoscope and the end of the endotracheal tube in front of the larynx. Therefore, the endoscope is slightly retracted to exactly direct the endotracheal tube through the laryngeal opening.
8) When the laryngeal opening has been initially entered, the stylet must be retracted.

The over-the-endoscope technique has been described,* but this requires a dedicated semiflexible endoscope, and the endotracheal tube required for guinea pigs is usually too small to fit over it.

*Johnson DH: Over-the-endoscope intubation of small exotic mammals. Exotic DVM 7(2):18-23, 2005.

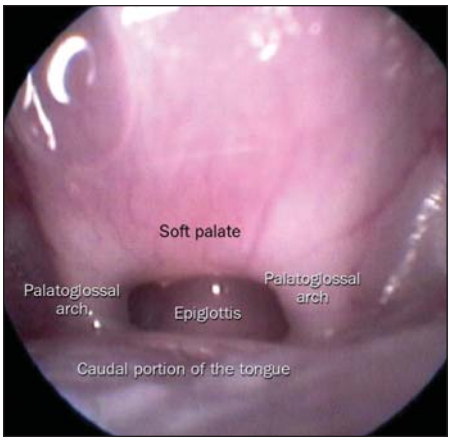

Fig 3. Beyond the narrow and long oral cavity typical of herbivore species, some rodent species (e.g., guinea pig, prairie dog, degu) have the intrapharyngeal ostium, made by the mucosa of the base of the tongue ventrally, the palatoglossal arch laterally, and the soft palate dorsally. The epiglottis is caudal to this ostium, and the tracheal opening is caudal to the epiglottis, making endotracheal intubation very difficult and, in the author's experience, impossible with the indirect ("blind") technique and without the endoscopic-guided technique. Shown is the intrapharyngeal opening (ostium) in the guinea pig.

Chinchilla Dental Health

All pet chinchillas develop some degree of acquired dental disease (ADD) during their lifetime, although it may be subclinical for many years. Chinchillas are peculiar, because (unlike rabbits and guinea pigs) they do not show detectable clinical signs before severe changes have occurred.

What we know about pathophysiology of chinchilla dental disorders comes from the excellent studies of David Crossley—improper nutrition (even when only partially improper) seems to be the reason. Underlying or predisposing factors, such as metabolic bone disease, have not been reported in the chinchilla and do not seem to be an issue. It would be simple and quick to say that pet chinchillas do not receive the same foodstuffs they have available in their natural environment.

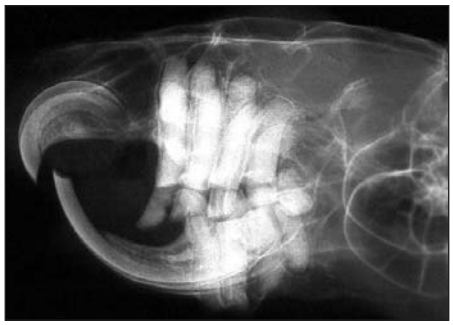

Fig 4. An oblique radiograph shows severely elongated upper cheek teeth in a chinchilla.

The physiology of the teeth and GI anatomy of chinchillas is similar to that of rabbits, but (according to their different natural environment), the nutrition is different. Rabbits are strict herbivores (in the natural environment they eat just grass). Chinchillas live above an altitude of 4,000 meters, where fresh grass is rarely present. Vegetables grow on mountainous terrains; therefore, the amount of silicates present and the biologic features of these foods are different.

The rule of thumb for nutrition of chinchillas is to provide a high-fiber diet. Alfalfa may be too high in calcium, but Timothy hay is basic. Chinchillas, unlike rabbits and guinea pigs, are used to eating far fewer fresh greens. Unfortunately, most of pet chinchillas do not eat enough hay for many reasons:

1) They were not trained when they were very young
2) They are fed too many treats and too many pelleted foods
3) They adapt well to a less than optimal nutrition.

A high quality pelleted food should be present in the diet of chinchillas but should not be offered ad lib. Because it is chewed more quickly and easily, chinchillas prefer it over hay. However, despite the good quality and proper amount of fiber, too much pelleted food will reduce the natural chewing movements, serving as a predisposing factor for ADD. Pellets that are too large for chinchillas will increase the vertical movements and pressures over the cheek teeth, also predisposing to ADD.

In summary, the best nutrition for chinchillas consists of good quality, high-fiber hay (ad lib); a small amount of high-quality, small-sized pelleted food (1 Tbsp or less) and a very small amount of fresh vegetables and treats (e.g., fibrous fruit, such as apple).

Small Mammal Suture Material

In small mammal surgery, proper instruments, proper suture material and proper surgeon's hands are as important as intraoperative local blocks and pre- and post-operative analgesia. The choice of suture material is vital to the postoperative outcome and may be one of the reasons for postoperative complications (e.g., edema, postoperative pain, self licking, self removal of stitches).

If we used suture sizes in small mammals in the same proportion as in larger pet carnivores, we would choose mostly 4.0 or 5.0. But these are more expensive and magnifying loupes may be needed. However, a larger suture applies more pressure on the soft tissues, especially softer tissues of herbivores. Braided sutures are more traumatic, because their surface is not smooth and they tend to become "swollen" when they absorb fluids (which then applies further pressure on tissues).

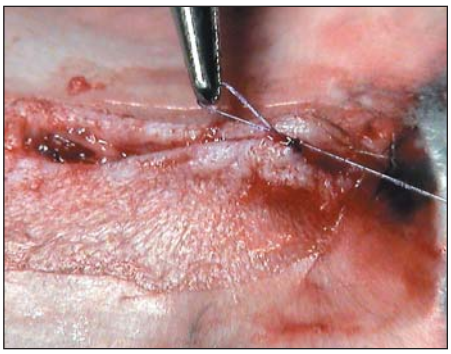

Fig 5. An ovariohysterectomy performed in a hamster using 5.0 absorbable subcuticular suture.

The Collapsing Ferret

Dr. Nico Schoemaker

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Dr. Johnson-Delaney, a 1980 graduate of Washington State University, practices avian and exotic animal medicine in Kirkland, Washington. She is a past president of the Association of Exotic Mammal Veterinarians and works closely with the Washington Ferret Rescue and Shelter. She has been committed to ferret health her entire career and has conducted research into ferret adrenal disease for many years.

As I've discussed before, one low blood sugar result on a collapsed ferret in the examination room should not be a definitive reason to diagnose "insulinoma" and start the ferret on prednisone or prednisolone, without even a thorough physical examination. And yet, I am presented with a lot of these ferrets. All have virtually the same history: collapsed or laying around (ferret people call it either "speed bumping or pelting" when the ferret all of a sudden goes "flat"). They may walk or play a little and then collapse again, sometimes with hypersalivation, sometimes with pawing at the mouth, sometimes with gagging or tremors, which seem to be progressive.

Questions to ask should always include: when did the ferret last eat before the collapse, what else was it doing (like play?), concurrent diseases and medical treatments, and has any pattern been noted. When most of these questions are answered, most cases are older ferrets, not really related to when they last ate, most haven't been playing or doing much anyway but sleeping. Yet the previous veterinarian had taken blood (with the blood glucose level at less than 90), so the ferret was put on corticosteroids. The clinical signs haven't changed, and for most, the ferret is getting worse.

We've discussed that diagnosing islet cell disease is best done by feeding the ferret, then taking blood for glucose and serum insulin levels at 2 hours, 3 hours and 4 hours. If the blood glucose level has dropped to 70-80 at the 2-hour interval, it is highly suspicious of islet cell disease with hyperinsulinemia, and the test should continue for one more hour. I send the serum for the insulin level at the point in time where the blood glucose is 60 or below. I run the blood glucose in the clinic using a Vetometer. (We recently compared the in-house Vetometer results with our local laboratory and found the blood glucose results within 1% of values obtained on blood samples that were immediately read or immediately serum-separated. The insulin levels are run at the University of Tennessee and may take a week to get back. The insulin level may be elevated or "within normal limits," but compared to the low blood glucose, it will be elevated.

Ferrets being assessed for islet cell disease should also have full CBC and serum chemistries done, as many do have concurrent gastrointestinal, liver, kidney or other endocrine

disorders. I like to do abdominal ultrasound and scout radiographs as part of the workup. Pending other health parameters, surgery to remove islet cell tumors will at least temporarily halt the clinical signs; however, there may be recurrence.

Back to the collapsing, exercise-intolerant ferret with dubious blood glucose problems: I start with the full physical examination and in many cases, I auscultate a marked cardiac arrhythmia. I then use an audio Doppler to explore the heart: each valve can be isolated, and the overall rhythm can even be heard by the owner. While once again we can jump on "heart block" as the diagnosis, without doing the next step of running an ECG, you really cannot assess what the heart is doing electrically.

I use a Vetronics Cardiostore (Devon UK) with the 2 mm 4 lead clips on awake ferrets. The recording is then downloaded immediately into the computer for the analysis. What I find in most of these cases amounts to the ferret collapsing at the point where the normal or slightly elevated heart rate suddenly drops to half. Many have a heart rate of 220-240 beats per minute that drops to less than 100 beats per minute for several beats; then the heart returns to the "normal" rate. On ECG, some of these ferrets throw ventricular escape beats, but all have some degree of heart block.

To follow are 3 ECGs from ferrets diagnosed as having insulinomas and put on prednisone on the basis of the clinical sign of collapse and one blood glucose test. It may be that islet cell disease is also present, but after treatment for the cardiac disease, the ferrets have done well and have had no collapsing episodes. Also, random blood glucose levels at the office were between 80-100, with owners noting it is usually over 2 hours since the ferret ate.

Along with ECG, ferrets with arrhythmias should have a full cardiac workup that includes radiographs, blood pressure and echocardiography, in addition to a general diagnostic blood panel and urinalysis. Treatment of heart disease depends on the cardiomyopathy found. In many cases what is really needed is a pacemaker. A pacemaker that does not have any wires needs to be developed, because the small canine units are not designed to have the chest compression and bending of the chest that ferrets do. In lieu of not placing a pacemaker, medical and nutritional therapy is currently what I rely on.

There is an excellent recent article on Ferret Cardiology by Robert Wagner in Vet Clin No Am Exot Anim Pract 12:115-134, 2009. It contains an excellent formulary. Treatment of severe heart block has not been very effective with either metaproterenol or isoproterenol as neither seem to be able to maintain the heart rate much above 80 beats per minute. I usually treat the ferret for the cardiomyopathy (usually it is dilated) with pimobendan (Vetmedin) at 0.3-0.5 mg/kg PO q12h and/or enalapril (enalapril maleate generic) at 0.25-0.5 mg/kg PO q24-48h. Furosemide at 1-2 mg/kg PO q8-12h is used if there is pulmonary edema and/or ascites present. The ferret is also placed on my "Cardiac Formula" nutraceutical combination (Table 1).

In severe dilated cardiomyopathies, I may increase the taurine levels to 2000 mg in the cardiac formula, as it seems there may be a relationship as there is in cats. I have seen some early cases of dilated cardiomyopathy in ferrets greatly correct using the cardiac formula with additional taurine. Studies need to be done in ferrets, as have been done in dogs and cats, to look at the cardiac nutrients and the effects on the heart.

In conclusion, one low blood glucose and a collapsed ferret should be given a thorough evaluation. It is easy to just prescribe prednisone and diagnose insulinoma, but in my experience, a good many of these ferrets have significant cardiac disease.

TABLE 1. CATHY JOHNSON-DELANEY'S CARDIAC FORMULA

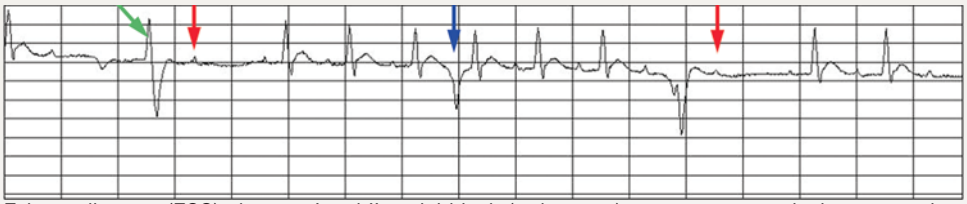
- Taurine (1000 mg)
- L-carnitine (1000 mg)
- CoQ_{10} (400 mg)
- Vitamin E (1000 IU)

Add above ingredients to 30 mL omega 3-6-9 fatty acid formula with omega 3-6 in a 3:1 ideal ratio - Optomega (USANA Health, www.usana.com)

Each ferret receives 0.5 mL PO q24h.

Case 1

A 5-year-old neutered male ferret presented because "the prednisone wasn't working," and the animal was collapsing more.



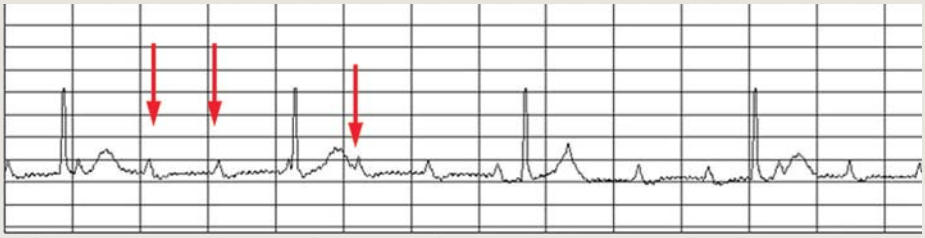
Echocardiogram (ECG) shows a Lead II: atrial block (red arrows), an escape ventricular contraction (green arrow), and a premature ventricular contraction (blue arrow).

- P amplitude (amp) 0.18 millivolts (mV)
- P-duration 10 milliseconds (mS)
- P-R interval (int) 53 mS - some have ventricular premature contractions overriding; some have missing QRS
- R amp 1.09 mV
- QRS duration 37 mS, when appear fairly normal
- S amp 0.29 mV
- T amp 0.31 mV
- R-R int varies due to block, ventricular escapes, premature ventricular contractions

Clinically, the heart rate would drop to half, and the ferret would lay down; then it would bounce up quickly when the rate came up to above 200, only to collapse again when it dropped below 100.

Case 2

A 6-year-old neutered male ferret presented due to collapsing ("Prednisone didn't seem to be doing anything.")

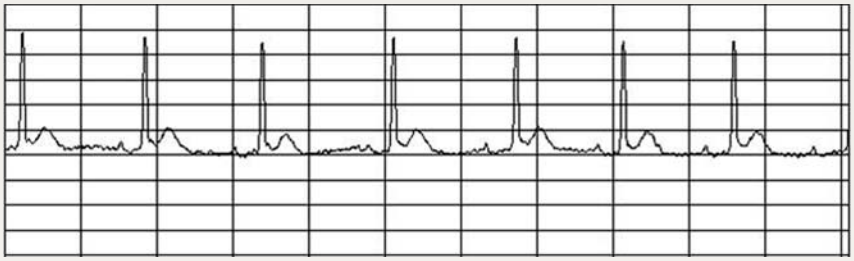


ECG shows P waves (red arrows) without QRS following. The third one is nearly on top of a T wave. Ventricles beating 90 beats per minute (bpm); atria beating 310 bpm; verified on ECG, also dilated cardiomyopathy.

- P amp 0.47 mV
- P duration 27 mS
- R amp 1.85 mV
- QRS duration 37 mS
- T amp 0.51 mV

Case 3

A 5-year-old spayed female collapsing ferret; referring vet had diagnosed atrial tachycardia, congestive heart failure and insulinoma. Blood glucose was 120 in the office 4 hours after a meal. Prednisone had been discontinued by the owner after only a few days due to the hyperactivity it seemed to be causing.



Heart rate 173 BPM

- P amp 0.14 mV
- P duration 10 mS
- P-R Int 53 mS
- R amp 2.2 mV
- QRS duration 27 mS
- QT int 13 mS
- T amp 0.53 mV
- R-R int 347 mS

On echocardiograph, marked dilated cardiomyopathy with atria barely moving. The ferret has responded well to pimobendan at 0.3 mg/kg q12h plus the cardiac nutraceutical formula and no longer has collapsing episodes.

Normal values for ECG in ferrets are published (Wagner, 2009); however most ferrets in this study are sedated with ketamine/xylazine.

TABLE 2. PUBLISHED VALUES FOR NON-SEDATED FERRETS

- P amplitude (mV) ≤0.2
- P duration (mS) 10-30
- PR interval (mS) 30-60
- QRS duration (mS) 20-50
- R amplitude (mV) 1 ± 2.8
- QT interval (mS) 60-160

Heart rate beats/min 200-280 is considered awake normal.

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David Brust received his DVM degree from Texas A&M University in 1984 and has been a practicing exotic animal veterinarian since that time. He is a past president of the West Houston Veterinary Medical Association and host of the radio talk show, "Ask the Vet." He is the current president of the Association of Sugar Glider Veterinarians,™ www.asgv.org. Dr. Brust is the author of *"Sugar Gliders: A Complete Veterinary Care Guide,"* and has filmed over 30 educational documentaries for the public regarding proper sugar glider care and husbandry.

All photos courtesy of ASGV™ and www.asgv.org.

WHAT EVERY VETERINARIAN NEEDS TO KNOW ABOUT

Sugar Gliders

Sugar gliders (*Petaurus breviceps*), also known as sugar bears, are small marsupials similar in appearance to small flying squirrels that are native to Australia, Indonesia and New Guinea. They were first brought to the United States in 1993 and have rapidly grown in popularity as companion pets. Most domestic sugar gliders in U.S. are the smaller New Guinean subspecies. Although they are legal companion pets in 46 of the contiguous states (with the exception of California and Pennsylvania), breeding and sales are strictly regulated by the USDA.

Suitability as Pets

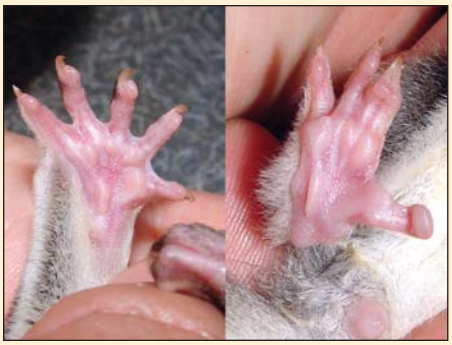
Young sugar gliders are best human-socialized between 8 and 12 weeks out of the pouch. The bonding process may take several weeks to complete. Human socializing for taming and handling may be difficult in sexually mature adults who were not socialized as youngsters.

Sugar gliders are colony animals, therefore it is strongly recommended

Table 1. Vital Statistics

Physiological		
Life span		12-15 years
Head/body length		13-19 cm (5.0-7.5 in)
Weight	Male	113-170 g (4-6 oz)
	Female	85-142 g (3-5 oz)
Heart rate		200-300 beats/minute
Respiratory rate		16-40 breaths/minute
Food consumption		15-20% BWt
Base metabolic rate		2.54 W/kg
Avg. basal metabolism		46.2 kJ/d (130 g animal)
Avg. active metabolism		84-126 kJ/d
Cloacal temperature		89.6°F (32°C)
Rectal temperature		97.3°F +/- 0.7°F (36.3°C)
Thermoneutral zone		75-88°F (24-31°C)
Reproductive		
Breeding cycle		Year round in captivity
Estrous cycle		Polyestrous - 29 days
Gestation		5-17 days, after migration, fetus will remain in pouch 50-75 days.
Litters per year		1-2
Incidence of multiple births		Twins 80% of the time; triplets are documented
Weaning		35-60 days out of pouch

**Adapted from Brust DM: Sugar Gliders: A Complete Veterinary Care Guide. Association of Sugar Glider Veterinarians,™ 2009, www.asgv.org*

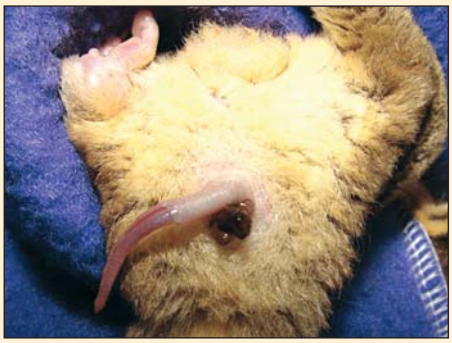


One of the most distinguishing features about sugar gliders is that they have 4 hands. Each hand has 4 fingers and an opposable thumb.

A sugar glider's nails may become sharp. Nails may be filed but not clipped; clipping nails may reduce the animal's ability to firmly grasp its surroundings, allowing it to fall.

While the patagium is similar in appearance to that of a flying squirrel, sugar gliders exhibit muscular control over it and can steer themselves to their target.

Males have 2 scent glands—a diamond-shaped "bald spot" on the forehead and a smaller sternal spot in the center of the chest. Females do not have either of these characteristics.



Males have a bifurcated penis with a preputial covering; the scrotum is anterior to the cloaca.

they should be housed in groups of two or more whenever possible. If housed alone, owners must be advised to spend a minimum of 2 hours per day interacting with the animal to provide necessary companionship and prevent malaise. Sugar gliders may self-mutilate if not given enough social stimulation.

Behavior

Although nocturnal by nature, sugar gliders are able to adjust to any schedule that allows maximum interaction with their owners. They enjoy playing outside their enclosure; however, careful supervision is strongly recommended to prevent encounters with common household hazards, such as floor or halogen lamps, metal venetian blinds and houseplants.

When properly trained, they may exhibit behavior similar to many dogs, e.g., expressing affection, recognizing their name, coming on command. With training, they will ride around in the owner's pocket for hours without restraint.

Common vocalizations include "crabbing" (when frightened), barking (lonely or playing), purring/chirping (contentment) and sneezing/hissing (grooming or playing).

Aggression is rare in well-acclimated animals and is typically limited to young joeys or unsocialized adults. When threatened, a sugar glider will stand on its back legs and charge at the threat, feigning strikes and making loud sounds similar to a locust.

Physiological Characteristics

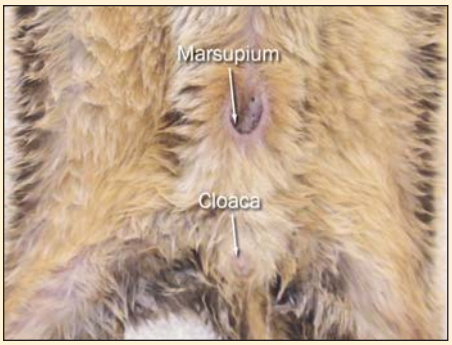
- Each of the 4 hands possesses sharp, scimitar-like claws and opposable thumbs.
- Teeth do not continually grow like rodents and should not be routinely trimmed unless presenting serious issues.
- Sugar gliders exhibit exceptional muscular control over the gliding membrane (patagium), allowing the animal to glide up to 50 m.
- The semi-prehensile tail is primarily used for steering when gliding.

Sexing

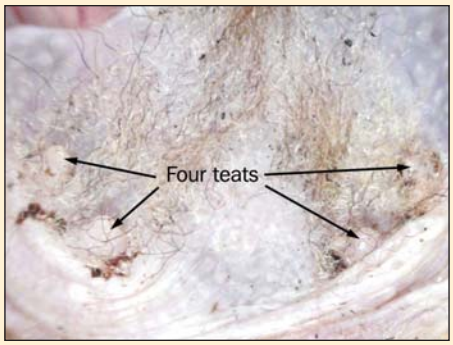
Males have a large pendulous scrotum and a bifurcated penis. Prominent scent glands are visible on the forehead and chest. Females exhibit a ventral pouch (marsupium) with 4 internal teats.

Growth of Joey

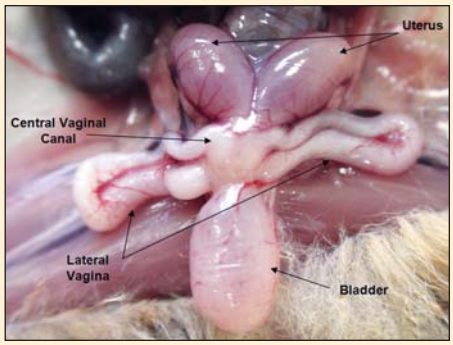
A study of the offspring from 30 breeding pairs was conducted to observe and record the developmental weight and characteristics of young sugar gliders over the first 8 weeks out of the pouch. The study concluded that certain developmental markers were more reliable than size and weight in estimating the age of joeys (Table 1).



Female sugar gliders have a prominent, mid-abdominal pouch (marsupium) where they carry their young.

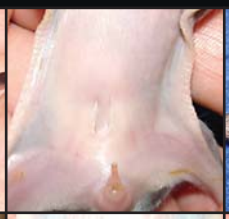
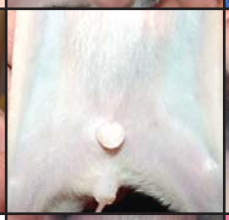
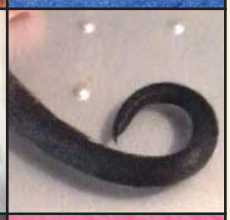
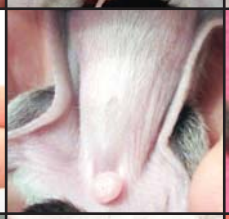


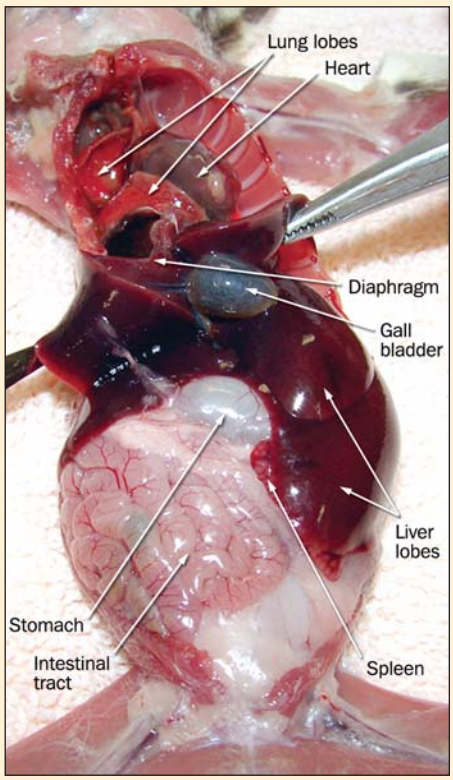
Females have four teats.



Female sugar gliders have 2 uteri and 2 elongated lateral vaginae that open into a single cul-de-sac divided by a septum.

Table 1. Growth Stages and Distinguishing Characteristics

GROWTH OF JOEY			
WEEK 1 8-18 g body weight; no fur, eyes closed			
WEEK 2 12-22 g body weight; very fine fur, eyes closed			
WEEK 4 18-35 g body weight; fur-becoming more prominent; tail starting to fluff out; animal becoming weaned			
WEEK 8 23-75 g body weight; self-sufficient; very active at night			



Normal internal organ placement.

Dietary Recommendations

Free-ranging sugar gliders' diet consists primarily of pollens, arthropods and plant and insect exudates; however, their diets can vary greatly by season, location and climate conditions. Attempts to replicate this type of diet for domesticated animals may be impractical in non-clinical settings. Sugar gliders should not be presented with a wide selection of high-sugar, high-fat items as they will almost always eat these foods to the exclusion of other more nutritious foods. Inappropriate feeding practices and inadequate homemade diets are believed to be a substantial contributing factor to many illnesses seen by practitioners and reduces the animal's life span.

Although some homemade diets may be adequately designed, they are rarely practical for the average owner because it is often more difficult for them to secure necessary ingredients and maintain precise feeding ratios.

Fresh portions should be fed in the evening. Preservatives, pesticides and excessive fat should be avoided in the diet. Acceptable treats include small portions of fruit (e.g., melons, peaches, mangos, blueberries, papaya), yogurt

Selected Sugar Glider Diets

SUGAR GLIDER DIET 1

(Recommended by author, see www.asgv.org for additional information)

The ideal daily diet for a domesticated sugar glider should equal approximately 15-20% of its body weight and consist of the following 4 components:

- **Nutritionally-balanced kibble*** (approx. 75% of daily intake). This equates to 1-2 oz per animal and should be available free choice in the enclosure at all times.
- **Sliced fresh fruits and vegetables** (approx 25% of daily intake). This equates to approximately one-eighth of an apple per animal and should be placed in the enclosure at night and removed each morning. Items should not be diced or chopped to maintain moisture content.
- **A calcium-based multivitamin*** should be sprinkled over fresh fruits or vegetables 3-4 times per week.

***Special consideration:** Kibble and multi-vitamin products should be designed specifically for sugar gliders and formulated to work in tandem with each other. Mixing products made for other animals is generally not recommended.

The following feeding programs are published in Johnson-Delaney C: Exotic Companion Medicine Handbook for Veterinarians. Zoological Education Network, 2000.

Owners electing to use any of the following diets should be advised to mix the ingredients precisely as outlined in order to maintain nutritional consistency and efficacy.

SUGAR GLIDER DIET 2**

- **50% Leadbeater's Mixture**
- **50% insectivore/carnivore diet**

Leadbeater's Mixture:

- 150 ml warm water
- 150 ml honey
- 1 shelled hard-boiled egg
- 25 g high protein baby cereal
- 1 tsp vitamin/mineral supplement

Mix warm water and honey. In separate container, blend egg until homogenized; gradually add honey/water, then vitamin powder, then baby cereal, blending after each addition until smooth. Refrigerate.

**Based on research and consultation with Australian zookeepers, veterinarians, and naturalists

SUGAR GLIDER DIET 3

(one daily portion)

- Include equal amounts of: chopped apple, grapes or mango, carrot, sweet potato, hard-cooked egg yolk, zoo formula insectivore or exotic feline diet, plus 1 Tbsp volume of pet industry-raised insects
- Pet industry-raised insects that have been fed a commercial cricket diet or enriched feed
- Or, owner can dust all insects, fruits and moist foods with a complete vitamin/mineral powder
- Insects include mealworms, crickets, waxworms, moths
- 1 Tbsp insects (2 small mealworms or 4 small and 2 large or 2 waxworms)
- Nectars formulated for lories/lorikeets can be given as a fruit-portion substitute or as a treat
- Foods should be "chopped together" to decrease the ability of the glider to pick out only the favorite parts

SUGAR GLIDER DIET 4†

(feeds 1 sugar glider)

- 1 tsp-sized piece each, chopped: apple, carrot, sweet potato, banana
- 1 tsp leaf lettuce
- 1/2 hard-cooked egg yolk
- 1 Tbsp good quality zoo feline diet
- 1 dozen mealworms

† Chicago Zoological Park adapted from AAZK Animal Diet Notebook

SUGAR GLIDER DIET 5#

(feeds 2 sugar gliders)

- 3 g apple
- 3 g banana/corn
- 1.5 g dog kibble
- 1 tsp fly pupae
- 3 g grapes/kiwi fruit
- 2 tsp Leadbeater's mixture (see previous Diet 2)
- 4 g orange with skin
- 2 g pear
- 2 g cantaloupe/melon/papaya
- 3 g sweet potato
- On Wednesdays: feed day-old chick; when available, large insects (mealworms)

Taronga Zoo, Sydney Australia

and applesauce. Owners should be cautioned against feeding fatty, nutrient-deficient insects as treats because sugar gliders will often hold out and refuse to eat anything else once they become accustomed to insects. Treats should be no more than 5% of daily intake. Filtered spring or drinking water (not unfiltered tap water) should be available at all times.

Housing Recommendations

The recommended enclosure size for 1 or 2 adult animals over 5 months of age is: 36 inches (91 cm) wide by 24 inches (61 cm) deep by 40 inches (102 cm) high. Large aviary cages are the most practical option for adult sugar gliders. Additional height is the primary consideration.

The ideal enclosure size for 1-2 babies or juveniles younger than 5 months out of the pouch is: 18-20 inches (46-51 cm) wide and deep and 24-30 inches (61-76 cm) high.

PVC-coated wire is preferred over epoxy, paint, powder-coated or galvanized wire due to potential health and safety hazards. Rectangular openings should be no larger than ½" x 1" (1.25-2.5 cm). Enclosures consist-

Due to an instinctual fear of falling, sugar gliders will become significantly less active when they cannot firmly grasp their surroundings.

A smaller, "starter" cage is more conducive to the well-being of joeys under 5 months out of pouch. The ideal cage size for 1-2 joeys is 18-20 inches (46-51 cm) deep x 24-30 inches (61-76 cm) high.

A nesting cloth, loosely draped over a heat rock is recommended rather than traditional nesting boxes or hanging pouches, as a sleeping area, especially for young joeys. This combination reduces stress on the glider and promotes the bonding process with owners.

ing primarily of vertical bars (e.g., bird cages) are not recommended for babies or juveniles, as soft tissue tears may develop between the digits from sliding down the bars.

A removable plastic waste tray should be at least 1" (2.5 cm) from the floor of the enclosure. Paper lining is preferred over wood shavings. Daily removal of feces and soiled papers and general cleaning of an enclosure and all supplies are recommended as well as a quarterly sterilization of the housing and accessories.

Important considerations for placement of the enclosure in the home include environmental temperature, noise levels, odor, lighting and the social nature of the animal.

The ideal temperature range in the home for a healthy animal is 75-80°F (24-27°C). Nighttime temperature should not drop below 70°F (21°C). The use of a supplemental heat source is strongly recommended. A conventional heat rock is preferable to a heat lamp or UV lighting, especially during the bonding period, as it allows the animal to efficiently self-thermoregulate throughout the day regardless of temperature variations. Alternatively, ceramic heat emitters positioned with a linen towel or surgical huck towel can be used for regulating the heat.

Food and water bowls and food items may be placed inside an enclosed dining area to avoid contamination and unnecessary waste. The use of both a conventional water bottle and a weighted secondary water dish is recommended.

Environmental Enrichment

Sugar gliders enjoy most traditional pet toys. Any item with loose strings or wires that could entangle the animal should be avoided. Solid exercise wheels provide an important opportunity for necessary exercise. Traditional hamster or rodent wheels should be avoided due to hazards associated with the prehensile tail. Rope and/or wooden toys should be replaced every 3-4 months.

Plants and branches are recommended to promote leaping and climbing. Quality artificial plants are preferred to natural fauna due to health and sanitary considerations. Varied sizes of branches of nontoxic trees can be used (for a list of safe plants go to www.asgv.org). These should be removed and cleaned every 2-3 weeks and must be thoroughly rinsed. A preferable alternative to foliage is 1-inch plastic chain, available at home improvement centers.

Grooming

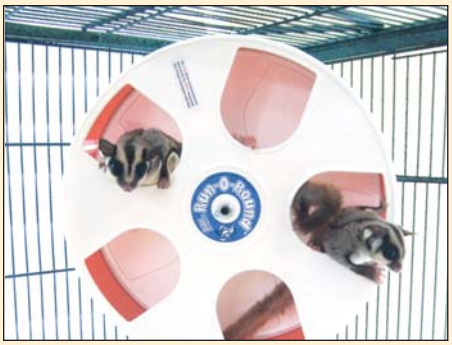
Bathing is not required. Sugar gliders will routinely groom themselves and each other. Effective topical sprays and waste tray additives are commercially available.

Restraint

Manual restraint is best accomplished using either a surgical huck towel or fleece bonding pouch as a glove. These animals should not be scruffed or held by the tail. Sugar gliders can be transported to the clinic in a zippered, fleece pouch.

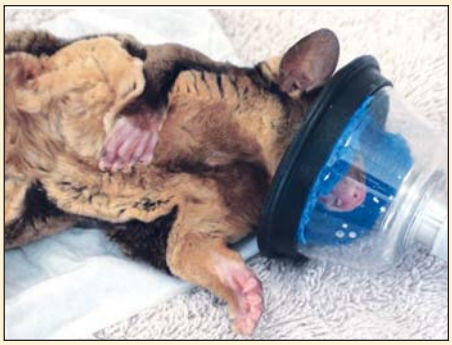
Sedation

Sedation is usually required for a clinical examination or diagnostic sampling. Isoflurane may be used at 5% for induction, using a large face mask as an induction chamber, and 1-3% for maintenance with a small face mask. A non-rebreathing circuit should be used for both induction and maintenance. If isoflurane is used for induction, application of a topical methylcellulose eye lubricant should be used. Alternatively, sugar gliders may be sedated first with administration of an anxiolytic, such as midazolam (0.3-0.5 mg/kg IM). The use of an electrocardiogram (ECG) may be useful to help monitor the animal if it is



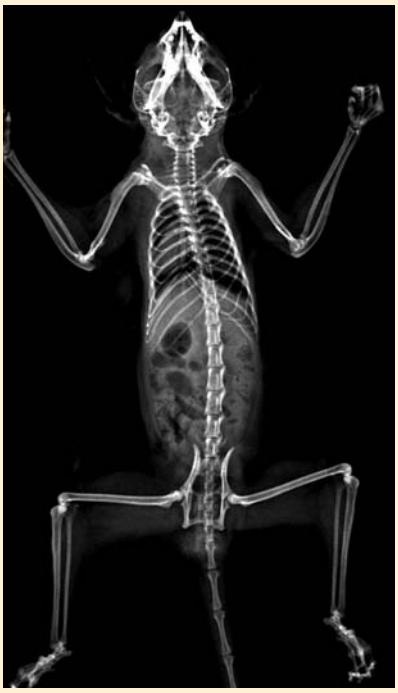
Solid-construction (not wire mesh) exercise wheels provide a good source of environmental enrichment and exercise for sugar gliders.

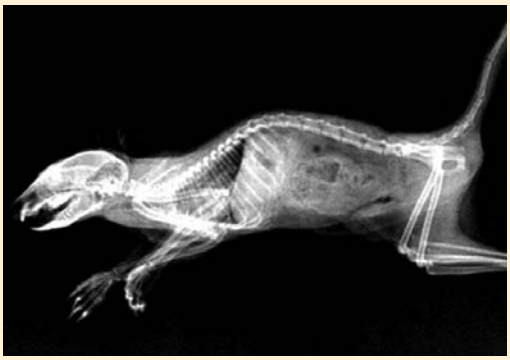
The least stressful method of sedation is achieved by using a large face mask as an induction chamber while 5% isoflurane is inhaled.



Once induced, 1-3% isoflurane is delivered for maintenance using either a small face mask or 1 mm Cook endotracheal tube.

A sugar glider can be safely restrained by placing the thumb under the jaw and the index finger on top of the head.





Normal radiographs, dorsoventral and lateral views

anesthetized longer than 5-10 minutes. Fluid therapy is required to maintain homeostasis.

Veterinary Visits

The initial consultation and annual examination should include:

- Careful analysis of all aspects of the diet and husbandry (directly related to most clinical presentations)
- Physical examination
- Stool flotation/smear for abnormal protozoa/parasite levels (a fecal sample is usually obtained by simply picking up or restraining the animal)
- Dental examination
- Other diagnostics
  - CBC/chemistry tests
  - Radiographs to assess bone density
- Males should be neutered whenever possible to avoid anti-social behaviors and self-mutilation.

Blood Collection

Only small volumes of blood may safely be drawn, up to a maximum of 1% of the animal's body weight in grams. A 1-mL tuberculin (or 0.5-mL insulin) syringe, with a 25- to 29-gauge needle, is recommended for most diagnostic sampling, depending on the site selected.

The cranial vena cava may be accessed at the thoracic inlet by directing the needle caudally at 30° off midline toward the contralateral hind limb. To avoid inadvertent cardiac puncture, insert the needle halfway of its length as the vessel is superficial in location. (View instructional collection videos at www.asgv.org.) With practice, blood collection at this site is usually the most successful regardless of the animal's size or condition.

The medial tibial artery is highly mobile and easiest to access just distal to the stifle using a 29-gauge needle. As much as 0.5 ml blood may be

Table 2. Hematologic Reference Ranges for Domestic Sugar Gliders

Parameter	Reference range	Sample size
Basophills	29.50-62.75 x $10^3/\mu L$	8
Eosinophills	92.02-281.18 x $10^3/\mu L$	10
HCT	51.29-54.49%	62
HGB	15.83-16.86 g/dL	53
Lymphocytes	3693.98-7157.15 x $10^3/\mu L$	62
MCH	18.79-19.39 pg	53
MCHC	30.63-30.99 g/dL	53
MCV	60.17-68.05 fL	54
Monocytes	112.55-170.69 x $10^3/\mu L$	45
Neutrophills	1461.03-2204.57 x $10^3/\mu L$	61
Platelets	292.18-400.32 x $10^3/\mu L$	53
RBC	8.31-8.83 x $10^6/\mu L$	53
WBC	5.49-9.31 x $10^3/\mu L$	62

Values shown are the 95% confidence intervals after outliers were removed. Blood was collected from the cranial vena cava. Statistically, 90% of the population should have values within these limits.

**Glucose levels measured immediately after collection.

Table 3. Biochemistry Reference Ranges for Domestic Sugar Gliders

Parameter	Reference range	Sample size
Albumin	3.12-4.64 g/dL	99
Alk phos	89.37-115.04 IU/L	75
ALT	96.76-136.60 IU/L	81
Amylase	2117.18-3350.82 IU/L	8
AST	54.42-99.79 IU/L	38
BUN	15.07-18.07 mg/dL	100
Calcium	8.53-8.85 mg/dL	97
Chloride	105.97-108.64 mEq/L	94
Cholesterol	111.70-123.99 mg/dL	78
CPK	1080.78-1636.71 IU/L	47
Creatinine	0.47-0.59 mg/dL	100
Globulin	2.9-3.1 g/dL	92
Glucose**	152.70-171.89 mg/dL	85
Magnesium	1.63-2.14 mEq/L	13
Phosphorus	4.35-6.12 mg/dL	62
Potassium	4.60-5.53 mEq/L	93
Sodium	138.76-143.06 mEq/L	92
Total bilirubin	0.12-0.70 mg/dL	72
Total protein	6.74-7.01 g/dL	92

Larger samples (up to 1 mL) may be obtained from the cranial vena cava. Notice the needle is not completely inserted.

The techniques used for administering fluids in sugar gliders are the same as in other small mammals; however, care should be taken not to administer fluids laterally, as they can pool in the patagium, resulting in slow absorption and discomfort to the patient.

Sugar gliders are particularly prone to obesity, especially when not exercised frequently or when fed inadequate diets that are too high in fat or sweets.

obtained from each side.

Cardiac puncture and orbital bleeding are not recommended.

Injection Sites

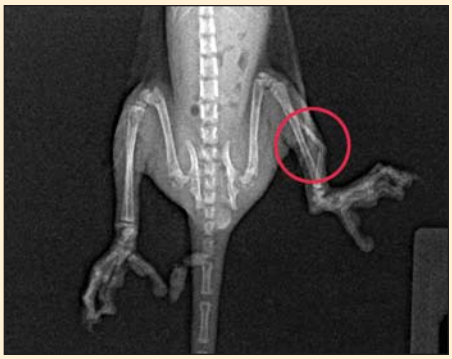
- **Intravenous:** Cephalic or lateral saphenous veins, using a 25-gauge needle
- **Intramuscular:** Quadriceps, epaxial muscles of the neck and biceps/triceps, using a 25-gauge needle. Avoid the use of medications that sting.
- **Subcutaneous:** Dorsal midline of the thorax, using a 25-gauge needle. Check for pooling in the patagium.

Most Common Disorders

- **Malnutrition,** which may be expressed as hind-limb paralysis, blindness, dehydration, cataracts, metabolic bone disease and seizures
- **Obesity**
- **Intestinal parasites**
- **Hair loss,** typically resulting from poor nutrition and vitamin intake
- **Pneumonia,** including discharge from the eyes/nose
- **Diarrhea,** resulting from a change in diet, inappropriate diet containing too high sugar content, bacterial overgrowths, *Giardia*, Cryptosporidia or *Clostridium* sp.
- **Stress-related diseases,** including self-mutilation (particularly solitary animals), cannibalism of young and eating disorders

Hair loss in an obese sugar glider.

Cataract causes may include: genetics, vitamin A deficiency, pouch infections and an improper diet too rich in sugars or fat.



Radiograph shows warped bone development in the rear leg (circle) consistent with metabolic bone disease.

- **Endocrine disorders**
- **Trauma** (fractures, burns)
- **Dental Disease**
- **Neoplasia**

Zoonotic Potential

There are no records of sugar gliders being susceptible to any specific pathogen or infectious disease. In over 15 years of widespread domestication and practical observation, *Clostridium piliforme* infections have been the most common diagnosis, and no documented cases of zoonotic transfer have been recorded.

Like most other mammals, it is believed that sugar gliders naturally host trace levels of various bacteria and flagellates in their digestive tracts. Under normal presentation, no treatment is typically required. During periods of abnormally high stress (e.g., adoption, transportation, introduction of new foods, change of diet), the immune system often becomes compromised, and bacteria/flagellate levels increase. The most common presentation is diarrhea, with *Giardia* often suspected as the causative agent in chronic cases.

To date, no documented case has confirmed a *Giardia* transfer from

What Every Owner Should Know About Sugar Gliders

- The most common mistake owners make is to feed the animal things it "likes." This is problematic due to the animal's overwhelming predilection for sweets and fats. Treats should consist of small pieces of fruit, yogurt or applesauce, not to exceed 5% of the total daily intake.
- Kibble or supplements designed for cats, primates or reptiles should not be fed to sugar gliders.
- Uneaten fruits/vegetables should be removed from the cage each morning.
- Owners should thoroughly wash their hands, including under their fingernails, before handling animals in order to avoid accidental transfer of toxins or bacteria.
- Sugar gliders are susceptible to toxicosis and a wide range of household hazards due to their keen senses and highly inquisitive nature. They should be protected from access to:
  - open containers of fluids, such as toilets, sinks, bathtubs, or buckets
  - stovetops, light bulbs, toasters, coffee pots
  - fruit-scented air fresheners/cleaners, insect or rodent baits, pesticides sprayed in rooms or on foods, residues left on hands or under fingernails and chemicals in tap water used as drinking water
  - chocolate or caffeinated drinks
  - toxic houseplants or holiday decorations
  - overheated non-stick cookware and other kitchen hazards

sugar gliders to humans. It is believed that some genotypes of *Giardia* may be host-adapted and endemic to marsupials and under normal circumstances do not appear to cause clinical signs.

Web Resources

Updated veterinary-oriented resources, including an online veterinary care guide, procedural videos and extensive client education materials are available at the Association of Sugar Glider Veterinarians™ website, www.asgv.org. Due to an educational grant, first-year memberships are free for a limited time.

RESOURCES

A client education brochure on sugar gliders is available from Zoological Education Network - 800-946-4782 www.exoticdvm.com

Table 4. Formulary for Sugar Gliders*

Although every effort has been made to ensure the accuracy of the information presented herein (particularly doses), in all cases the clinician is responsible for the use of any pharmaceuticals. Most drugs used in exotic companion species are considered extra-label, and few pharmacokinetic studies have been conducted; therefore, the clinician must critically evaluate the information provided and stay informed of recommendations in the literature.

Dosages listed in this formulary are anecdotal as reported in the literature except where noted. Sugar gliders have carnivore/omnivore gastrointestinal tracts; therefore, carnivore guidelines should be followed for antibiotic choice. Pain resulting from an injection can cause the rapid induction of shock in small species of marsupials.[9,15]

Compounding note: *Due to sugar gliders' overwhelming predilection for sweets, most sugar gliders respond favorably to medications that are compounded with a fruity flavor. Tutti-fruity typically works best, although other fruit flavors, such as apple, peach have also been used with favorable results.*

DRUG	ROUTE	DOSAGE	COMMENTS	REFS
Acepromazine (A) + ketamine (K)	SC	(A) 1 mg/kg + (K) 10 mg/kg	Postoperative analgesia and sedation to prevent self-trauma to incision site	6, 20
Acepromazine (A) + butorphanol (B)	PO	(A) 1.7 mg/kg + (B) 1.7 mg/kg	Tranquilization, analgesia post op	6, 20
Alfaxalone-alfadolone acetate (Saffan, Glaxo, London)	IV	0.1-0.2 ml/kg	Immobilization sedation	9,22
	IM	0.25-0.5 ml/kg		
	SC	15 mg/kg		
Amikacin (A) + Penicillin G (P)	SC	(A) 3 mg/kg q12h + (P) 25,000 IU/kg q12h (with fluid support)	Respiratory infection	11
Amoxicillin	PO, IM	30 mg/kg q24h x 14d	Dermatitis, general	6,10
Amoxicillin/clavulanic acid	SC	12.5 mg/kg q24h	Inject form not avail US	6
Atropine	IM, IV, SC	0.02-0.04 mg/kg	Control salivation during sedation	9,22
Bismuth subsalicylate	PO	1 ml/kg q8-12h x 5-7d	Diarrhea related to *Giardia*, coccidia	11
Buprenorphine	IM	0.01-0.03 mg/kg	Analgesic	11
Butorphanol	IM, SC, PO	0.1-0.5 mg/kg q6-8h prn	Analgesic	6,12,15,20
Butorphanol (B) + acepromazine (A)	PO	(B) 1.7 mg/kg + (A) 1.7 mg/kg; dilute with saline to administer	Post-op sedative, analgesic	6
Calcium glubionate	PO	150 mg/kg q24h long term	Calcium deficiency/nutritional osteodystrophy	6
Calcium gluconate	SC	100 mg/kg q12h x 3-5d (diluted in saline to 10 mg/ml)	Calcium deficiency/nutritional osteodystrophy	11
Calcium glycerophosphate/ lactate	IM	7 mg/kg IM	Calcium deficiency/nutritional osteodystrophy	6
Carbaryl powder (5%)	Topical	Sparingly, also in nest boxes	Ectoparasites	6,11,20
Cephalexin	SC	30 mg/kg q24h		6
Ciprofloxacin	PO	10 mg/kg q12h x 7-10d	Similar use as enrofloxacin	6
Cisapride	PO, IM	0.25 mg/kg q8-24h	GI motility enhancer	6,15
Dexamethasone	IV, IM, SC	0.2 mg/kg q12-24h	Antiinflammatory; higher dosages for shock	6,15
Diazepam	IM, PO, IV	0.5-2.0 mg/kg	Calming, higher dosages IV for seizures	6,15,22
Doxapram	IV	2 mg/kg	General CNS stimulant, especially respiration	17
Enalapril	PO	0.5 mg/kg	Vasodialator in the treatment of heart failure and hypertension	17
Enrofloxacin	PO, IM, SC	2.5-5.0 mg/kg q12-24h	Antibiotic; note: may cause tissue necrosis SC	6,15
Epinephrine	IV	0.003 mg/kg	Stimulates heart, antagonizes effects of histamine, raises blood sugar	17
Fenbendazole	PO	20-50 mg/kg q24h x 3d repeat in 14d	Anthelmintic	2,6,11
Fluoxetine	PO	1-5 mg/kg q8h	Self-mutilation	11,16
Furosemide	SC, IM	1-4 mg/kg q6-8h	Diuretic	15,17

DRUG	ROUTE	DOSAGE	COMMENTS	REFS
Furosemide	PO	1-5 mg/kg q12h	Diuretic	15
Glycopyrrolate	IM, IV, SC	0.01-0.02 mg/kg	Control salivation during sedation	6,22
Griseofulvin	PO	20 mg/kg q24h x 30-60d	Antifungal, *Trichophyton* spp.	26
Hyaluronidase	Fluids	(150 IU/ml) 0.5-1.0 ml/L	Speeds fluid absorption	11
Isoflurane		5% induction/1-3% maintenance	Preferred anesthesia	6
Itraconazole	PO	5-10 mg/kg q12h	Fungal treatment	1,6
Ivermectin	PO, SC	0.2 mg/kg once, repeat in 10-14d	Anthelmintic	2,6,11
Ketamine	IM	20 mg/kg	Follow with isoflurane	6
Ketamine (K) + medetomidine (M)	IM	(K) 2-3 mg/kg + (M) 0.05-0.1 mg/kg	Immobilization. Reverse medetomidine with atipamezole 0.05-0.4 mg/kg IV	22
Lactulose	PO	0.2 ml q24h	Constipation	6
Lincomycin	IM	30 mg/kg q24h x 7d	Dermatitis	10
Meloxicam	PO	0.1-0.2 mg/kg q12h; Use 0.5 mg/ml concentration	Analgesia and nonsteroidal antiinflammatory; animals like sweet taste	11
Metoclopramide	IV, IM, SC, PO	0.05-0.1 mg/kg q6-12h prn	GI motility enhancer	6,15
Metronidazole	PO	80 mg/kg q24h	Infection	10
Midazolam	IM	0.25-0.5 mg/kg	Anxiolytic, preanesthetic sedation	14
Oxfendazole	PO	5 mg/kg once	Anthelmintic	2,6
Piperazine	PO	100 mg/kg	Anthelmintic	26
Prednisolone	IM, SC, PO	0.1-0.2 mg/kg q24h	Corticosteroid	15
Pyrantel pamoate (P) / Febantel (F)	PO	(P) 14.4 mg/kg + (F) 15 mg/kg	Roundworms, strongyles	6
Pyrethrin powder	Topical	Product safe for kittens, same dosage, frequency	Ectoparasites	6
Selamectin	Topical	6-18 mg/kg repeat in 30d	Ectoparasites	11
Sevoflurane		1-5% to effect	Anesthesia	6
Sulfadimethoxine	PO	5-10 mg/kg q12-24h x7-10d	Antibiotic; make sure well hydrated	6,11,15
Trimethoprim / sulfa	IM, PO, SC	10-20 mg/kg q12-24h x7-10d	Antibiotic; make sure well hydrated; SC may cause necrosis	6,15
	PO	50-57 mg/kg q24h		6
Vitamin A		500-5000 IU/kg	Skin disorders	6
Vitamin B complex	IM	0.01-0.02 ml/kg	Vitamin; be very careful of "sting"; administer under anesthetic or dilute	15
Vitamin E	PO	25 mg/animal/day	Vitamin	26
Yohimbine	IV	0.2 mg/kg	Reverse xylazine	22

*Adapted from Association of Sugar Glider Veterinarians'™ web site, www.asgv.org ©2009 by David Brust, DVM and Marsupial Formulary ©2002 by Cathy Johnson-Delaney, DVM, Dipl ABVP (Avian)

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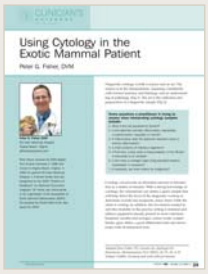

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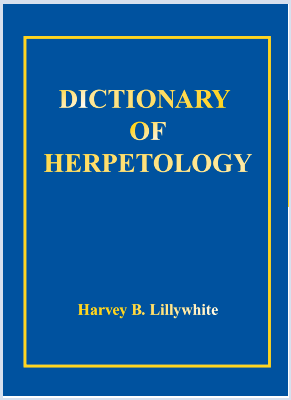

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Dictionary of Herpetology

By **Harvey B. Lillywhite**

One of the author's hopes for this book is "that it will be useful to a breadth of persons who might be interested in herpetology for one reason or another." I had a discussion with several exotic animal veterinarians, all of whom have a strong interest in reptiles and amphibians, and each admitted to little or no reading of herpetological periodicals, such as Journal of Herpetology and Copeia. Some of the reasons offered were time constraints, lack of overlap with species they see in their practice, and an unfamiliarity with the terms that were used in the herpetological literature. With that in mind, I put this book to the test, picking several words from recent articles in primary herpetological literature to see if they were covered in the book and if they were easy to understand.

On the Plus Side

I started with "sympatric" and "allopatric" from an article on lungless salamanders. The definitions are in straightforward language:

- Sympatric = "living in the same geographic location, with reference to the overlap in geographic range of two closely related species, which remain otherwise distinct"
- Allopatric = "a condition of geographic distribution referring to species or populations of organisms that occur in non-overlapping, but usually adjacent, areas"

If you don't understand these terms, any discussion of the lungless salamanders of the Appalachian region is going to leave you hopelessly lost. Okay, while on this subject, does the phrase "lungless salamanders: exist in the dictionary? Yes, and it is defined as "collective vernacular name for the salamanders belonging to the family *Plethodontidae*." How about the acronym POTZ which pops up frequently? The dictionary refers me to preferred optimum temperature zone, which is "the range of temperatures within which a particular ectothermic species functions optimally overall."

Other terms that were encountered in my random search were monimostyly, indicator species, pith, squirt gland, epiplastron, chi-squared test, gular disc, guild, and she-male (no, not what you're thinking but a "male within a mating ball that releases a pheromone attracting other males, as though the snake was a female. Because this distracts other nearby males, the phenomenon gives the 'she-male' a competitive advantage in courtship…").

I tried over 50 words and found them in this book. The 41 black and white illustrations are extremely helpful in understanding certain concepts (such as the venom delivery system of a viper), the names of the superficial anatomic features (such as scales and scutes) and some internal features (such as gonads).

On the Minus Side

This is not a dictionary for all terms biological, and the author has been selective about what gets included. For example, Dr. Lillywhite acknowledges that while molecular studies have clearly classified birds as reptiles, ornithological terms lay outside this text.

Add to Bookshelf?

I am a herpetological nerd who finds it increasingly hard to fit the primary literature into my reading schedule, and this book has proven itself as a useful tool when I find myself in the middle of an article with unfamiliar terms. Sadly, I fear the time of the printed and bound dictionary is past, as typing into Wikipedia, Google or various other search engines often is easier than flipping through a dictionary. Of course, you can't do that when the power is out.

I think that Dr. Lillywhite has succeeded in his hope that this book will be useful to people with more than just a passing interest in amphibians and reptiles. If you are a veterinarian who sees amphibians and reptiles but really just wants to know how to keep it and treat it, the *Dictionary of Herpetology* is not going to be used all that much. However, if you are a veterinarian with an avid interest in amphibians and reptiles who enjoys reading the scientific literature covering the broad field of herpetology, buy this book.

Reviewed by **Kevin Wright, DVM**
Arizona Exotic Animal Hospital
Mesa, Arizona

Diseases of the Goat, Third Edition

By **John Matthews, BSc, BVMS, MRCVS**

This is a comprehensive reference that provides a lot of relevant information in an easily portable, soft-covered book.

On the Plus Side

The index itemizes all the chapters by subgroups of topics based on clinical signs (lameness, infertility and diarrhea) or techniques (anesthesia and surgical procedures). The presentations and outlines of the chapters make it quick and easy to locate a specific topic and the associated subtopic of interest.

Chapters are divided into basic information regarding the problem, initial assessment, clinical examination/signs, recommended diagnostics (further assessment) and treatment recommendations. Further breakdowns include etiology, epidemiology, diagnosis and control. Recommendations for further reading (references) are also provided.

Numerous tables provide quick guides to assessment and disease processes. The photo plates in the center of the book help to outline problems or disease processes described in the text.

The Appendix provides values for the normal goat, including weights, physiologic values, reproductive data and blood and cerebral spinal fluid values. Additional appendices provide information on drug dosages, reportable diseases (in the UK only) and the most common differential for 4 of the largest concerns in this species.

On the Minus Side

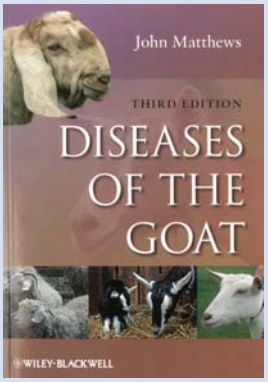
The author of this book is from the United Kingdom (UK), so some of the diseases in Europe are not appropriate for the United States (US) and Canada. Other diseases are more prevalent in the Western hemisphere and are not addressed in detail. Also, some terms that are standard in the UK are not used or are spelled differently in the US—"aetiology," for example. However, these are minor issues in the usefulness of the text.

Several of the drugs mentioned in the text are not available in the US and may not be appropriate for treatment in that region. Practitioners are recommended to consult with Food Animal Residue Avoidance Databank (FARAD) regarding appropriate dosages and withdrawal times.

Add to Bookshelf?

If you are seeing goats at all, it is a great addition to your library. It provides easy access to many different syndromes with a quick flip of the page. However, additional references may be needed in the US and Canada for certain syndromes not seen in Europe.

Reviewed by **Ariana Finkelstein, DVM**
All Species Veterinary Services
San Antonio, Texas



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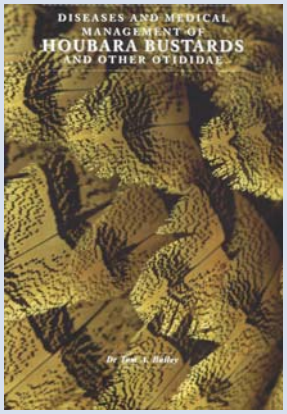

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Diseases and Medical Management of Houbara Bustards and Other Otididae

Edited by **Dr. Tom A. Bailey**

Dr. Bailey has compiled an impressive collection of information on Otididae species, in particular the Houbara or Macqueen's bustard. The editor's extensive work and collaboration on bustard projects, including his PhD project, provides an excellent background to oversee the submissions to this book for completeness and medical importance. Otididae species are significant in conservation efforts and falconry, especially in the Middle East.

The first section, which covers veterinary considerations of bustard captive breeding, rehabilitation, restoration programs and important aspects of managing captive bustards, is especially important to the novice overseeing or treating bustards in a wildlife conservation program or zoological collection. The "Clinical Workup of Bustards" section includes chapters on examination, biochemistry and hematology, cytology, radiology, endoscopy and postmortem examination. Additional sections review bustard medicine and diseases and the health of free-living bustards.

On the Plus Side

Particularly helpful features of the text are diagnostic protocols as they relate to sample collection; microbial, viral and cytological testing that may be performed on the collected biological specimens; how to transport the sample in order to preserve its integrity; and culture media used. Endoscopy is important for diagnostic evaluations within the coelomic cavity of birds; this chapter contains a number of excellent color images of endoscopic views of different anatomical regions of bustard patients. Approximately 7 tables per chapter aid the reader in understanding important information.

I enjoyed the introductory chapters detailing the Otididae species, veterinary considerations of captive bustards and important aspects of managing bustards in captivity. Dietary considerations, reproductive management, biosecurity issues and housing are described in detail and highlighted with appropriate tables and images. Six appendices include a formulary, biosecurity protocol, normal hematology and biochemistry values, and field work protocols. The biosecurity protocol as well as the form for recording history and physical examination results is complete and easy to copy for use in a veterinary clinic or wildlife facility.

On the Minus Side

When a book is published on a specific group of birds in which there is no other published work to compare, it is difficult for a reviewer to find fault. The one aspect of this book that may have been improved upon is the image size. Most images are rather small in relation to the quality of the book. This is a minor complaint as all images are focused and in color. Also readers may find it unusual to have all the references listed in a single chapter at the end of the book; however, the references are in alphabetical order and should be easy to find. All of the positive attributes mentioned previously far outweigh any faults one may find when using this reference source.

Add to Bookshelf?

Dr. Tom Bailey and the contributing authors are to be commended on publishing this excellent book. The text will be of great benefit to veterinarians in which bustards are involved as part of their practice, veterinarians who oversee bustard collections in zoological parks, and wildlife conservationists who work with Otididae species. As the veterinary reference to bustards it will also be of interest to veterinarians studying for avian and zoological medicine specialty examinations. For the practicing veterinarian, wildlife biologist, and/or veterinary nurse, Diseases and Medical Management of Houbara Bustards and Other Otididae contains most if not all of the information needed to properly care for this group of birds.

Review by
Thomas N. Tully, Jr., DVM, MS,
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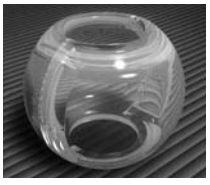

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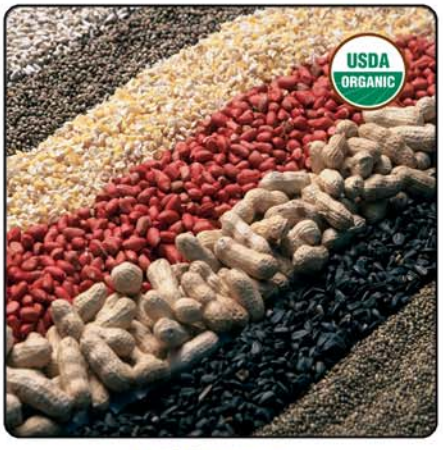

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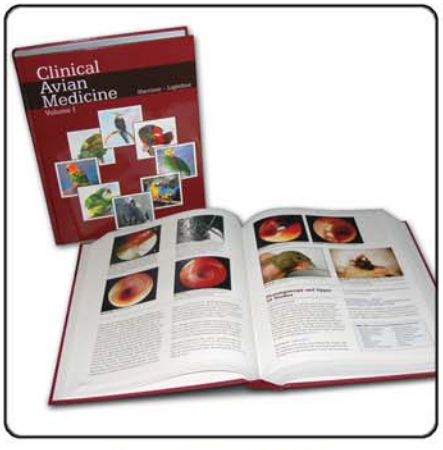

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