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Designed and published by
Veterinary Learning Systems,
780 Township Line Road,
Yardley, PA 19067

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Case Report

Management of Lymphoma in a Domestic Rat (*Rattus norvegicus*)

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MATERIALS AND METHODS

A 2½-year-old, 432-g male neutered rat (*Rattus norvegicus*) was evaluated for intermittent bilateral hindlimb lameness. Physical examination revealed porphyrin epiphora of the left eye. All other physical examination findings were within normal limits. No obvious crepitus was evident in the joints of the pelvic limb, and no lesions associated with the hindlimbs were noted. On palpation, no obvious pain was observed in the vertebral column or pelvic limbs. The rat was treated with meloxicam 0.2 mg/kg PO q24h (Metacam, 1.5 mg/mL oral suspension, Boehringer Ingelheim Vetmedica) for possible degenerative joint disease.

One month later, the rat developed hematuria and bilateral proprioceptive deficits in the pelvic limbs. The rat was alert and responsive, and auscultation of the heart and lungs was normal. Physical examination abnormalities included enophthalmia, blepharospasm, and chronic porphyrin epiphora of the left eye; left popliteal lymph node enlargement; and paresis in the rear limbs with bilateral scuffing of the dorsal surface of the pelvic feet on ambulation. Withdrawal reflexes were normal in both hindlimbs. Examination of the thoracic limbs and cranial nerve evaluation were unremarkable. There was no evi-

dence of muscle atrophy or pain on palpation of the vertebral column or pelvic limbs.

Initial diagnostic evaluation included a urinalysis of a voided sample, hematology, plasma biochemistry profile, and thoracic and abdominal radiographs. Abnormalities detected on the urinalysis included large amounts of blood, a pH of 6, and proteinuria (2+). Gram-negative rods were noted on cytologic examination of the sediment. Hematology revealed mild, regenerative anemia (34%, with 2–3+ polychromasia; reference range, 37%–49%*), mild leukocytosis ($13.1 \times 10^3/\mu\text{L}$; reference range, $4\text{--}10.2 \times 10^3/\mu\text{L}$) characterized by neutrophilia ($5.24 \times 10^3/\mu\text{L}$; reference range, $0.24\text{--}1.734 \times 10^3/\mu\text{L}$), lymphocytosis ($6.812 \times 10^3/\mu\text{L}$; reference range, $0.36\text{--}3.468 \times 10^3/\mu\text{L}$), monocytosis ($0.786 \times 10^3/\mu\text{L}$; reference range, $0\text{--}0.51 \times 10^3/\mu\text{L}$), and eosinophilia ($0.262 \times 10^3/\mu\text{L}$; reference range, $0\text{--}0.612 \times 10^3/\mu\text{L}$). Biochemical abnormalities included hyperglycemia (182 mg/dL; reference range, 74–163 mg/dL), hypokalemia (3.5 mmol/L; reference range,

*All reference ranges are taken from Williams BH, Weiss CA: Section four: Small rodents, basic anatomy, physiology, husbandry, and clinical techniques, in Quisenberry KE, Carpenter JW (eds): *Ferrets, Rabbits, and Rodents: Clinical Medicine and Surgery*, ed 2. Philadelphia, WB Saunders, 2004, pp 290–291.



Figure 1. Right lateral thoracic radiograph of a domestic rat demonstrating a soft tissue opacity within the thorax that silhouetted the heart and caused dorsal displacement of the trachea.



Figure 2. Right lateral thoracic radiograph of the same patient 6 months previously demonstrating a normal chest.

4.3–6.3 mmol/L), and hypomagnesemia (1.7 mg/dL; reference range, 2.6–3.2 mg/dL).

The patient was anesthetized by masking down with isoflurane in oxygen using a nonrebreathing circuit. Right lateral thoracic radiographs revealed increased soft tissue opacity within the thorax that silhouetted the heart and caused dorsal displacement of the trachea (Figure 1). This mass had not been evident on radiographs taken 6 months previously as part of the patient's diagnostic workup for respiratory disease (Figure 2). Abdominal radiographs revealed increased osteophyte production evident in both stifle joints, consistent with degenerative joint disease and vertebral spondylosis in the lumbar spine. Enrofloxacin 10 mg/kg PO q12h for 10 days (Baytril, 68-mg tablets [Bayer Animal Health] compounded in a 20-mg/mL suspension at the University of Georgia Veterinary Teaching Hospital) was prescribed for treatment of the urinary tract infection. Meloxicam was continued at 0.2 mg/kg PO q24h.

Reexamination 10 days after initial presentation indicated resolution of the hematuria and decreased porphyrin tearing. A weight loss of 9 g, tachycardia of 340 bpm, and tachypnea of 120 breaths/min were noted. Evaluation of the hindlimbs revealed delayed proprioception, absent postural thrust, severely decreased hopping in the right hindlimb, and mildly decreased hopping in the left hindlimb. The left popliteal lymph node remained enlarged.

Repeat urinalysis failed to indicate any evidence of bacterial infection. The patient was anesthetized as previously described, and thoracic ultrasonography revealed a 2.5 × 3-cm mass within the cranial mediastinal region. An ultrasound-guided aspirate of the mass revealed diffuse small, well-differentiated lymphocytes and large lymphoblasts, with mitotic figures frequently observed (Figure 3). The aspirate was consistent with a diagnosis of lymphoma.

Initial therapy for lymphoma consisted of L-asparaginase 400 IU/kg SC (Elspar, 5,000 IU/mL, Merck), lomus-

tine 1-mg capsule PO (CeeNU, Bristol Laboratories, Princeton, NJ), and prednisone 1.6 mg/kg PO q24h (Pediapred, 1-mg/mL syrup, Roxane Laboratories, Columbus, OH) (Table 1). Because of the difficulties in giving a lomustine capsule orally to a rat, the capsule was broken apart while the veterinarian was wearing double masks and rubber gloves, and its contents were mixed in a palatable liquid at each dosing. Any remaining drug and the capsule, masks, gloves, and syringe were discarded following a proper chemotherapy disposal protocol.

Although the rat initially appeared to respond to chemotherapy as evidenced by an initial weight gain of 5 g over 10 days and increased activity level, it was euthanized 66 days after the onset of therapy because of acute progressive dyspnea.

DISCUSSION

Based on cytologic findings, the provisional diagnosis was mediastinal lymphoma. A clinical pathologist did confirm this diagnosis; however, it was not confirmed at necropsy.

Lymphoma is a proliferative cancer originating in lymphoid tissues involving any organ or tissue.^{1,2} Mediastinal lymphoma, which most commonly involves the anterior and posterior lymph nodes in the mediastinum, is generally associated with a poor prognosis.¹ Clinical signs vary with the location and extent of the disease.¹ Clinical signs of the mediastinal form include dyspnea, tachypnea, and uncompressible anterior mediastinum with dull heart and lung sounds.¹

Chemotherapy has played a role in cancer management for many years.³ Chemotherapeutic agents used in combination have a greater advantage over the use of a single chemotherapeutic agent.^{3,4} Combination therapy allows maximal killing of cells while maintaining an acceptable toxicity range, the expansion of the therapeutic range against the tumor cells, and the prevention or slowing of the development of resistant cells.⁵ In this case, the rat was treated

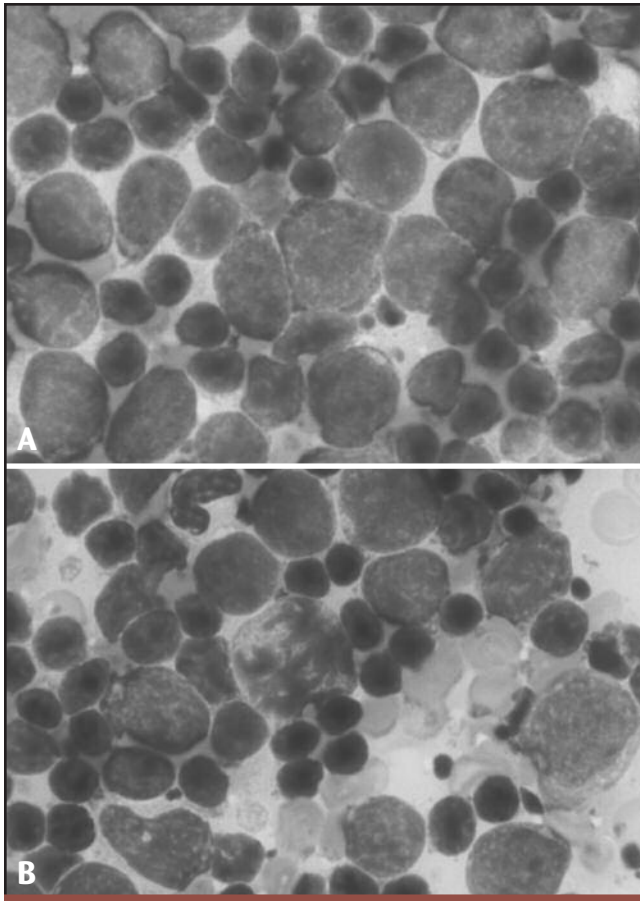


Figure 3. (A and B) Aspirate samples from the mediastinal lymph node revealing diffuse lymphocytes.

with a combination of L-asparaginase and lomustine, along with daily prednisone. L-asparaginase is a bacteria-derived enzyme that degrades the amino acid asparagine, deprives growing cells of asparagine, and causes an inhibition of protein synthesis. It is used in combination with other chemotherapeutic drugs for the treatment of lymphoma.⁶ Tumor cells may become resistant to the antitumor effects of L-asparaginase, and allergic reactions can develop following repeated administration because of the production of antibodies against the protein components of the drug.⁶ Lomustine is an alkylating chemotherapeutic agent used to treat resistant lymphoma in dogs.⁶ Primary side effects include myelosuppression and hepatotoxicity.^{4,6} Prednisone also has antitumor activity against lymphoma.⁶

Because cachexia is one of the most common paraneoplastic syndromes seen in veterinary cancer patients, it is important to begin nutritional supplementation early in the patient's treatment.⁷ It is often necessary to use a high-calorie, high-protein supplement as an adjunct therapy in cases of lymphoma.⁸ In this case, the rat received 3 to 5 mL a day of Ensure (Abbott Laboratories), along with various types of baby food to help it maintain weight and for easy delivery of daily medication.

TABLE 1. Chemotherapy Protocol Used in a Domestic Rat with Lymphoma

Week	Drug	Dose
1	L-asparaginase Prednisone Diphenhydramine (pre-med)	400 IU/kg SC 1.6 mg/kg PO q24h 2 mg/kg PO
2	Lomustine Prednisone	1 mg PO 1.6 mg/kg PO q24h
3	L-asparaginase Prednisone Diphenhydramine (pre-med)	400 IU/kg SC 1.6 mg/kg PO q24h 2 mg/kg PO
4	Prednisone	1.6 mg/kg PO q24h
5	Lomustine Prednisone	1 mg PO 1.6 mg/kg PO q24h
6	Prednisone	1.6 mg/kg PO q24h
7	Prednisone	1.6 mg/kg PO q24h
8	Lomustine Prednisone	1 mg PO 1.6 mg/kg PO q24h
9	Prednisone	1.6 mg/kg PO q24h
10	Prednisone	1.6 mg/kg PO q24h

CONCLUSION

With little information in the literature on the use of chemotherapeutic agents and the prognosis of cancers in exotic animals, the goal for this patient was to control the cancer and prolong survival while still maintaining a good quality of life.⁵ The chemotherapy was not designed as a cure for the patient's lymphoma but as a palliative therapy. While undergoing treatment, the rat maintained a good quality of life, and it is believed that the rat's life was extended by approximately 2 months. This is approximately one-tenth the total life span of a rat (with an average life span of 2 years). This is relatively comparable to the median remission time of 12 months in a dog (with an average life span of 10 years) being treated for lymphoma with chemotherapy.⁹

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Case Report

Dynamic Crossed-Pin Fixation of a Distal Femoral Growth Plate Fracture in a Domestic Rabbit (*Oryctolagus cuniculus*)

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INTRODUCTION

There is little peer-reviewed literature on the management of specific fractures in domestic rabbits, although multiple texts suggest that basic orthopedic techniques, such as those used for dogs and cats, can be applied.^{1–4}



Figure 1. (A) Craniocaudal and (B) lateral postoperative radiographs showing reduction of the Salter-Harris type II fracture of the distal femoral growth plate and fixation with two Kirschner pins.

HISTORY

An 11-week-old female domestic rabbit (*Oryctolagus cuniculus*), weighing 1.18 kg, was referred to the Exotic Animal, Wildlife, and Zoological Medicine Service of the Small Animal Veterinary Teaching Hospital at the University of Georgia with a Salter-Harris type II fracture of the distal right femur. The injury was suffered the previous day when the rabbit jumped out of its owner's arms.

THERAPY

The rabbit was premedicated with hydromorphone 0.2 mg/kg (ESI Lederle, Cherry Hill, NJ) and midazolam HCl 0.3 mg/kg IM (American Pharmaceutical Partners, Schaumburg, IL). Anesthesia was induced by mask with sevoflurane (Ultane, Abbott Laboratories) and maintained with 2.0%–2.5% sevoflurane in 100% oxygen via an uncuffed endotracheal tube.

A lateral parapatellar surgical approach was made to the right stifle and distal femur. The fracture was reduced and fixed with two 0.9-mm Kirschner pins (Imex Veterinary, Longview, TX) driven with a pneumatic drill (Mini-Driver, 3M Health Care) in antegrade fashion from the trochlear ridges, across the fracture site, and proximally into the medullary canal to the subtrochanteric region (Figure 1). The pins were cut short at the stifle, and the ends were countersunk below the level of the cartilage. The joint capsule and deep fascia were closed with simple interrupt-

ed sutures of monofilament absorbable suture (PDS II, Ethicon), and the subdermal fascia was closed with a simple continuous pattern using the same material. No external coaptation was used. The rabbit's recovery was uneventful.

RESULTS

At hospital discharge 2 days postoperatively, the rabbit was ambulating well and had regained the use of the repaired limb. The owner was instructed to keep the rabbit confined to a cage. Limb function was normal 3 weeks later, and radiographs indicated that the fracture had healed with closure of the growth plate (Figure 2).

DISCUSSION

Anatomic realignment, stable fixation, and early restoration of joint motion are the prerequisites for the successful treatment of periarticular fractures. The anatomically reduced Salter-Harris type I or II fracture is somewhat stable because of its interdigitating conformation; however, internal fixation must be added to maintain reduction if the animal is to be permitted to use the limb during the healing process.

Several variations of intramedullary pinning have been described for fixation of these fractures in the dog and cat.⁵⁻⁷ Multiple (usually two) small pins driven in normo-grade fashion from the articular surface of the trochlea maximize the purchase on the short epiphyseal segment and enhance rotational stability better than a single pin. The direction of the pins can be slightly convergent and thus remain contained in the proximal medullary canal (so-called "dynamic" crossed pins or modified Rush pins) or can be somewhat more convergent and thus driven through the cortex of the diaphysis several centimeters proximal to the fracture (so-called "static" cross-pinning). Neither technique has been proven to be superior to the other.⁵ Regardless of the chosen technique, the distal ends of the pins are driven beneath the cartilage with a countersink to prevent intra-articular irritation and are left in place unless there is subsequent migration.⁷



Figure 2. (A) Craniocaudal and (B) lateral radiographs of the femur 3 weeks after fracture fixation showing healing with closure of the growth plate.

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Veterinary Anesthesia Drug Quick Reference

By Cheryl A. Blaze and Maria M. Glowaski
Published by Elsevier Saunders, 2004

Veterinary Anesthesia Drug Quick Reference is a concise, pocket-sized guide that addresses drug combinations for ferrets, rabbits, pigs, ruminants, dogs, and cats. There are also sections devoted to common exotic surgeries, namely ferret adrenalectomy and caprine urolithiasis. This small text includes precise photos for nerve-blocking techniques and addresses the effects of anesthesia on ECG, the use of specific multimodal anesthetic combinations (including advantages and disadvantages), blood gases and fluid selection, and drip rates for constant-rate infusion anesthetics. Included in the drug listings are the times of onset and durations of action. The quick-reference feature of this guide allows for fast decision making when devising anesthetic plans.

The authors present case management in a step-by-step manner. For example, the preanesthesia section addressing adrenalectomy in ferrets reminds readers about patient handling, relevant preoperative laboratory tests, and blood transfusions and offers catheter placement tips. The section on

induction presents various anesthesia protocols with their potential side effects. Pointers on ferret intubation are also included. The maintenance section details the anesthesia circuit and fluid therapy. The monitoring section addresses several criteria (anesthetic depth, blood glucose, ECG, body temperature, blood pressure, and blood loss) with recommendations on the indicated response to any changes in the patient's condition. Postoperative care, feeding, and pain management are also presented.

This same level of detail is given to case scenarios involving canine hit-by-car limb fractures, ophthalmic surgery, cesarean sections, and uroliths. Feline diabetes, hypertrophic cardiomyopathy, and urolithiasis are also addressed, and anesthetic management of seizing dogs and cats is outlined. In addition, there are similar sections regarding urolithiasis in goats and ruptured bladders in foals.

I highly recommend this user-friendly guide for clinicians and technicians in exotic and general practice.

Handbook of Veterinary Pain Management

By James S. Gaynor and William W. Muir
Published by Elsevier Mosby, 2002

Handbook of Veterinary Pain Management presents a forward-thinking overview on alleviating and preventing pain in veterinary patients. The book does not directly address exotic animal medicine; nonetheless, the concepts and philosophies described are applicable to all elements of veterinary practice. This book diverges from many traditional texts, as it reminds the reader of the veterinarian's obligation to alleviate pain. The book opens with a thought-provoking overview of the evolution of the profession's view on pain control in animals.

A novel feature of this book is the section on cancer pain, which presents a step-by-step planning guide, designed to adapt with the patient's level of pain and type of discomfort. The use of multimodal drug therapy targeted at the patient's specific source of pain is discussed. The dynamics of the analgesic plans rely on owner feedback, with adjustments in the treatment regimen based on the owner's

observations of his or her pet.

The book addresses clinical and technical aspects in great detail. The diagrams are precise, and the tables of drug-dosing regimens are comprehensive. Traditional pharmacologic treatments and alternative modalities are presented. Pain management and prevention are addressed on many practical levels, including chronic disease, surgical pain, and cancer pain. Alternative and complementary pain management modalities are explained, along with their clinical indications, contraindications, and limitations. The legal and ethical considerations regarding the uses of nontraditional modalities are also discussed.

Handbook of Veterinary Pain Management is unique, serving as both a clinical and a philosophical reference. Readers of this book will find practical recommendations for improving patient comfort in many (often overlooked) aspects of patient care.

Both books reviewed by **Andrew Silverstone, DVM**
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Natural History of the Sugar Glider (*Petaurus breviceps*)

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INTRODUCTION

The sugar glider is in the Petauridae family, with *Petaurus* meaning “rope dancer” and *breviceps* meaning “short headed.” There are seven subspecies of sugar gliders: *Petaurus breviceps* subsp *breviceps*, *P. breviceps* subsp *longicaudatus*, *P. breviceps* subsp *ariel*, *P. breviceps* subsp *flavidus*, *P. breviceps* subsp *tafa*, *P. breviceps* subsp *papuanus*, and *P. breviceps* subsp *biacensis*. Exotic animal veterinarians should become familiar with sugar gliders and other marsupials.

ANATOMY

Marsupials have some interesting anatomic differences when compared with placental mammals, the most important of which is the birth of a fetus, which must find its way to an external teat to complete development. All marsupials share this characteristic. Their most well-known physical characteristics include pouches and marsupial bones (pelvic ribs), although not all marsupials have either or both. The Petauridae do not have marsupial bones. The females have a pouch containing four teats. Sugar gliders also possess a patagium, or gliding membrane, that stretches between their front and hind legs (from the outside of the tip of the fifth finger to the inside of the first toe on each side). This membrane enables them to “fly” from tree to tree, covering distances up to 50 m (150 ft).

Although it is frequently reported in the literature that marsupials have a metabolism approximately two-thirds that of similarly sized placental mammals ($K = 70$ in mammals, $K = 49$ in marsupials), caloric and metabolic studies have shown that there are many additional factors to be considered. Marsupials do not always follow that guideline, and allometric scaling may not always be appropriate. The practitioner must be aware of variations in metabolism of various pharmaceuticals in different marsupials.

Sugar gliders have a gray coat with a dark stripe that runs dorsally from their nose to their lower back, a cream-colored underbelly, and black rings around the eyes that extend to the ears. They have a weakly prehensile tail, which is equal to or longer than the length of the rest of their body. They have five digits on each foot, with the second and third digits of each hind foot partially fused. All the digits are clawed except the large opposable first digit on the hind feet.

Sugar gliders have many scent glands used for marking territory and members of their social group. The main scent glands are on the forehead (frontal), on the chest (sternal), and alongside the cloaca (paracloacal). Scent glands are also located on the corners of the mouth, on the paws, and on the

inside surfaces of the ear pinnae. Oily secretions are normal and should not be confused with an abscess.

It is relatively easy to determine the gender of a sugar glider. Males have a bifurcated penis (located in the cloaca) and a pendulous cloacal scrotum. Females have a ventral abdominal pouch, two uteri, a bifurcated clitoris, and two long, thin lateral vaginal canals in addition to the central vaginal canal. Both males and females have a cloaca, which is the common opening to the urinary, reproductive, and gastrointestinal tracts.

Obesity is a common problem in captive gliders. The patagium should be thin and flexible on palpation and not rounded with fat. Gliders have enlarged lower incisors for bark chewing and a large cecum to enable fermentation of complex polysaccharides in sap and gum. Normal feces are elongated and firm and dark-brown to black in color.

HABITAT AND BEHAVIOR

The sugar glider is found in the wild in New Guinea, most of northern and eastern mainland Australia, and Tasmania. Their natural habitat consists of forests and woodlands, with colonies living in tree hollows. They are very vocal animals. Sugar gliders can tolerate a wide range of temperatures, between 18°C (64°F) and 31°C (88°F), with an ideal temperature range of 24°C to 27°C (75°F to 81°F) when kept in captivity. They are a nocturnal prey species with excellent night vision. Predators in the wild include cats, foxes, kookaburras, lace monitors, and owls.

Sugar gliders develop very strong social bonds and in the wild typically live in groups of five to 10 animals with one dominant male, multiple females, and subordinate males. The one dominant male is thought to breed all of the females. He also marks the group territory and other mem-

TABLE 1. Basic Parameters for Sugar Gliders

Average life span	4–5 years (wild), 10–15 years (captivity)
Average adult weight	140 g (male), 115 g (female)
Heart rate*	200–300 bpm
Respiratory rate*	16–40 breaths/min
Rectal temperature	96.5–97.9°F (35.8–36.6°C)
Cloacal temperature	89.6°F (32°C)

*Under isoflurane anesthesia.

Suggested Diets for Sugar Gliders

Choose one, and follow it exactly. Chop pieces together so gliders cannot pick out just their favorite item. No one commercial diet seems adequate, and long-term nutritional studies are being conducted. Leadbeater's mix is listed on the Internet by various sugar glider fanciers who have altered the recipe for palatability rather than nutritional content. The version listed below is the original.

Dr. Cathy Johnson-Delaney's Sugar Glider Diet

Leadbeater's mix (50% of diet): Major component of the diet, feed in evening. One glider portion is approximately 2 Tbsp.

- 150 mL warm water
- 150 mL honey
- 1 shelled, boiled egg
- 25 g high-protein baby cereal
- 1 tsp vitamin/mineral supplement
- An additional 100 mg of calcium carbonate can be added.

Mix warm water and honey. In a separate container, blend egg until homogenized and gradually add water/honey mixture. Then add vitamin powder and baby cereal, blending after each addition until smooth. Keep refrigerated until served. This can also be frozen in ice cube trays (one well is approximately one meal's worth).

Insectivore/carnivore diet (50% of diet): Example: Reliable Protein Products Insectivore Diet (760-321-7533, www.zoofood.com).

Treat foods: Fruit, various, chopped. Can add bee pollen, vitamin/mineral supplement, and live insects (calcium gut loaded for several days, adult insects preferred). Can

also dust insects before giving them to the gliders. Can give blooming eucalyptus branches when available. Commercial lorikeet nectar can also be offered several times a week. Have additional calcium added to gum arabic, mix it with fruit juice or lorikeet nectar, and use it smeared on branches, as an enrichment treat.

Chicago Zoological Park Diet (adapted from AAZK Animal Diet Notebook)

Recipe feeds one animal:

- 1 teaspoon-sized piece each, chopped: apple, carrot, sweet potato, banana
- 1 tsp leaf lettuce
- ½ hard-cooked egg yolk
- 1 Tbsp Nebraska Feline Diet (or other good-quality zoo feline diet such as ZuPreem or Mazuri)
- 12 meal worms

Sugar Glider and Squirrel Glider Diet (from Taronga Zoo, Sydney, Australia)

Recipe feeds two animals:

- 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 mix (see Dr. Cathy Johnson-Delaney's sugar glider diet)
- 4 g orange with skin
- 2 g pear
- 2 g rockmelon/melon/pawpaw (papaya)
- 3 g sweet potato

(continues on p 9)

bers of the group with his scent glands and will aggressively defend his territory.

In the wild, mating generally occurs in June or July. Most young are born between August and November, with two litters per breeding season common. Females are seasonally polyestrous, with an average estrus cycle of 29 days, and usually have two young (joeys) per litter. They have a short gestation period of 15 to 17 days. Joeys remain in the pouch until 60 to 70 days of age and wean at 110 to 120 days. Joeys weigh less than 0.2 g at birth and reach their adult weight by 12 months of age. They are sexually mature around 10 and 14 months of age for males and females, respectively. The young remain in the colony until 7 to 10 months of age, when they are forced to leave and join a new colony. Their average life span in the wild is 4 to 5 years, but in captivity they can live as long as 15 years.

HABITAT AND HOUSING RECOMMENDATIONS

Sugar gliders are not solitary animals; thus, they should be kept in groups of at least two or three gliders. Wire cages are recommended for adequate ventilation, with maximum wire spacing of 2.5 × 1.3 cm (1.0 × 0.5 inch). Gliders will chew on and can escape from cages made of wood. A small group of two to six gliders can be kept in a cage as small as 2.0 m³ (6.6 ft³), but larger is better. A habitat should include at least one nesting box large enough for all members of the colony to fit into, as well as numerous branches to make use of vertical space. Zinc, as well as newsprint, cedar, and pine bedding, have all been anecdotally reported to be toxic to gliders (Pye and Carpenter 1999). Heat sources need to be provided to maintain a daytime temperature of up to 31°C (88°F) and a nighttime temperature no lower than 20°C (68°F). Temperatures below 18°C to 20°C (64°F to 68°F) for

Suggested Diets for Sugar Gliders (continued from p 8)

- Once a week: feed day-old chick; when available, large insects or mealworms

Dr. Debra McDonald's* Sugar Glider Diet

Daily diet per animal:

- 1 piece of Eukanuba dog chow
- 6 g fruit, chopped (approx 1 Tbsp)
- 1 tsp nectar mix (commercial lorikeet nectar)
- 1 g fly pupae (1/4 tsp)
- 5 g corn (1/4 thin slice)
- 2 g sprouted seed
- 2 mealworms

Supplement:

- 5 pollen grains once a week
- 3 sultanas three to four times a week
- 2 sunflower seeds once a week
- 1 g (small cube) Pet Health Food (Australian product) once a week
- 1 almond once a week
- Insects (e.g., moths) three to four times a week
- Acacia, eucalyptus, other blossoms as available

Dr. Rosemary Booth's Sugar Glider Diet

Offer a total of 15% to 20% of body weight daily. Select one diet from each group each day, and do not skimp on

ingredients. Animals will benefit from a major effort to provide a regular supply of vitamin/mineral-enriched insects. It is necessary to provide a variety of insects.

Group 1

- Insects: 75% moths, crickets, beetles; 25% fly pupae, mealworms
- Meat mix: Commercial small carnivore or insectivore mix

Group 2

- Nectar mix: 1.5 cups fructose, 1.5 cups sucrose (brown sugar), 0.5 cup glucose made up to 2 L with warm water (Commercially available mixes have the advantage of some vitamin/mineral additives.)
- Dry lorikeet mix: 4 cups rolled oats, 1 cup wheat germ, 1 cup brown sugar, 0.5 cup glucose, 0.5 cup raisins or sultanas

Group 3

- Fruit and vegetables: Select from diced apples, nectarines, melons, grapes, raisins, sultanas, figs, tomatoes, sweet corn kernels, sweet potatoes, beans, shredded carrots, butternut squash
- Greens: Mixed sprouts, lettuce, broccoli, parsley, with a vitamin/mineral supplement

Note: Vionate is a good vitamin/mineral supplement. Use 1/8 tsp per glider per group 3 portion. Nursing/breeding: Add additional calcium carbonate into each group 3 portion.

*Former nutritionist from Healesville Sanctuary, Victoria, Australia.

more than 8 to 12 hours may place the gliders in a negative caloric balance.

NATURAL DIET AND CONSIDERATIONS FOR CAPTIVE NUTRITION

Sugar gliders are omnivorous. Their diet in the wild consists of pollen, nectar, insects, and sap. Captive sugar gliders thrive on a varied diet consisting of various fresh and dried fruits, vegetables, and nuts as well as some form of live foods, such as mealworms, crickets, and moths. Sugar gliders generally prefer to eat in high places, so food and water bowls should be placed in an elevated position and not on the cage floor. A good option is to feed a commercial diet (such as the Mazuri Insectivore Diet) with fresh items added for enrichment. Several highly recommended recipes for sugar glider diets have been used successfully.

CONCLUSIONS

Sugar gliders can make good pets as long as the owners understand that it is difficult to convert them to a diurnal

schedule and meet their housing and nutritional requirements. Sugar gliders benefit from a large amount of socialization; at least 2 hours a day is recommended. They can be playful and cuddly and can develop strong bonds with their human companions under the right conditions.

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Selected Abstracts on Exotic Mammal Medicine and Surgery

SPECIAL FOCUS: ZONOOSES

Human Deaths Associated with Rodent LCM Virus

CDC Factsheet (www.cdc.gov/ncidod/dvrd/spb/mnpages/dispages/lcmv/qa.htm). Updated July 29, 2005.

ABSTRACT 1

Lymphocytic choriomeningitis (LCM) is a viral infection carried by rodents that can cause meningitis, encephalitis, or meningoencephalitis in humans. In the family Arenaviridae, lymphocytic choriomeningitis virus (LCMV) is the causative agent of LCM. In addition to neurologic manifestations, LCMV may also cause pregnancy-related infections resulting in abortion, hydrocephalus, chorioretinitis, or mental retardation. The most common clinical manifestations of LCM in humans are asymptomatic infection or mild febrile illnesses. The incubation period of LCM is 8 to 13 days. The disease is characterized by a biphasic febrile illness, with the first phase typified by fever, malaise, muscle aches, headache, anorexia, nausea, vomiting, and various areas of pain throughout the body. The second phase occurs after a few days of remission and may involve symptoms of meningitis (e.g., fever, headache, stiff neck) or encephalitis (e.g., drowsiness, confusion, paralysis).

The common house mouse, *Mus musculus*, spreads LCMV. Infected mice may serve as chronic carriers, and it is possible for pregnant mice to transmit LCMV to their offspring. Transmission to humans occurs via inhalation of infected particles of rodent urine, feces, or saliva; ingestion of contaminated food; or contact with infected body fluids. Transmission to humans has also been reported from handling infected hamsters. Most infected humans recover completely from LCMV. Mortality is less than 1%.

Lymphocytic Choriomeningitis Virus Infection in Organ Transplant Recipients—Massachusetts, Rhode Island, 2005

CDC: *MMWR Morb Mortal Wkly Rep* 54(21):537–539, 2005.

ABSTRACT 2

In May 2005, four organ recipients were infected with LCMV from a common donor. The organ donor had a 1-week history of headache and stroke-related hemiplegia, followed by brain herniation and death within 3 days. There was no sign of infection in the donor. Within 3 weeks after the organs had been transplanted, solid organ recipients

began showing a variety of clinical signs such as fever, rash, diarrhea, thrombocytopenia, and kidney failure. Three of the four recipients died within 23 to 27 days posttransplant. The fourth recipient lived, most likely because of treatment with antibiotics and reduction of his immunosuppressive drug regimen. Preliminary investigation traced the source of LCMV to an infected hamster in the donor's home.

COMMENTARY (ABSTRACTS 1 AND 2)

Individuals who come in contact with urine, feces, saliva, or blood from LCMV-infected animals are at risk of developing LCM. Laboratory workers and owners of mice or hamsters may be at risk if the animals originate from colonies with LCMV or if the animals were exposed to infected wild mice. Proper hand hygiene and environmental cleaning can minimize the risk of infection from LCMV. Healthcare providers should be aware that LCMV could be transmitted through organ transplantation.

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Outbreak of Multidrug-Resistant *Salmonella typhimurium* Associated with Rodents Purchased at Retail Pet Stores—United States, December 2003–October 2004

CDC: *MMWR Morb Mortal Wkly Rep* 54(17):429–433, 2005.

ABSTRACT

In 2004, the Centers for Disease Control and Prevention (CDC) documented an outbreak of multidrug-resistant *Salmonella enterica* serotype *typhimurium* (*S. typhimurium*) from rodents and associated human cases. These were the first reported human cases to be linked to pet rodents. Both of the affected children had contact with a recently purchased pet hamster and mouse. Both rodents were ill and eventually died. Pulsed-field gel electrophoresis (PFGE) was performed on isolates from the mouse and children. The patterns of the isolates were indistinguishable from each other. Susceptibility testing indicated multidrug resistance. In August 2004, a significant portion of a large shipment of hamsters to a Minnesota pet distributor became ill. Many died or were euthanized, and those submitted for necropsy had *S. typhimurium* that was indistinguishable by PFGE from the other cases.

A national search was conducted, revealing 28 matching human isolates from 19 different states occurring from

December 2003 to October 2004. The majority of infected people had a history of rodent exposure within 8 days of clinical sign onset and, on PFGE testing, were found to have the same strain of *Salmonella*. Despite a thorough investigation, no common link or common rodent source was identified.

COMMENTARY

Veterinarians and public health practitioners must keep in mind that rodents are a possible source of Salmonella infection and that appropriate hygiene when handling pet rodents is crucial. Identifying rodents as a reservoir for multidrug-resistant Salmonella has significant public health implications.

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Epidemiological Aspects of the First Outbreak of *Baylisascaris procyonis* Larva Migrans in Rabbits in Japan

Sato H, Kamiya H, Furuoka H: *J Vet Med Sci* 65(4):453–457, 2003.

ABSTRACT

Larva migrans caused by the common raccoon ascarid, *Baylisascaris procyonis*, is a zoonotic disease of critical importance in North America. The authors discuss the first proven outbreak of this disease in domestic rabbits (*Oryctolagus cuniculus*) in a wildlife park in Japan. Raccoons (*Procyon*

lotor) had been kept in this park for 9 years. A pet owner donated one raccoon to the park 8 weeks before the outbreak in rabbits. Of 12 raccoons, three (including the donated one) shed *B. procyonis* eggs in the feces, and two of the positive raccoons were kept in metal mesh cages on wooden pedestals 2 m (6.6 ft) from the rabbit enclosure. Circumstantial evidence indicated that the donated raccoon was the likely source of this outbreak.

All 12 enclosed raccoons were treated with an ascariocide, and the cages and the contaminated dirt floor of the park were decontaminated by extensive flaming. Three months after the control measures were instituted, recurrent ascarid infection was detected in three raccoons younger than 1½ years of age. The authors conclude that pet owners and public health workers in Japan need to consider the potential risk of serious zoonosis by *B. procyonis* as well as the difficulty of cleaning contaminated areas.

COMMENTARY

The outbreak of this familiar parasitic disease in Japan serves as a reminder of the dangers of larval migrans to rabbits and many other species commonly seen by exotic mammal veterinarians. Rabbits kept outdoors are in danger of exposure to B. procyonis. Another potential source of infection is feces-contaminated hay or other food items accessible to raccoons. Because pet ownership of raccoons is legal in many US states, exotic veterinarians should be aware of and prepared to educate owners about the dangers these animals pose to other household exotic pets.

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SPECIAL FOCUS: RABBIT HEALTH

Clinical Features of Skin Lesions in Rabbit Syphilis: A Retrospective Study of 63 Cases (1999–2003)

Saito K, Hasegawa A: *J Vet Med Sci* 66(10):1247–1249, 2004.

ABSTRACT

Skin lesions in rabbit syphilis are usually diagnostic, but it is occasionally difficult to differentiate these lesions from those of other dermatologic diseases. Skin lesions in 63 cases of rabbit syphilis were analyzed for early and accurate diagnosis. Lesions were found most frequently around the nose (55 cases), genitalia (22), lips (20), eyelids (12), and anus (10). Sneezing was observed in 33% of cases with nasal lesions. In cases of maternally acquired infection, lesions could be found mainly on the face initially. Rabbits should be examined carefully for facial lesions and lesions of the genitalia and anus, which are easily overlooked.

COMMENTARY

Rabbit syphilis, or venereal spirochetosis, is caused by the organism Treponema paraluis-cuniculi. This disease is transmitted by direct and venereal contact and is generally considered self-limiting. Asymptomatic animals may carry the organism and pass it to their young or to other rabbits during breeding. Stress may exacerbate lesions in infected animals. Typical skin lesions are highly suggestive, and many practitioners make this diagnosis based on the presence of lesions alone. This article points out the importance of a complete physical examination, including the perineum, to avoid missing lesions. Some early facial lesions are subtle. However, the presence of suspicious lesions around the nose and perineum should increase the index of suspicion for this disease.

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Hip Dysplasia in Rabbits: Association with Nest Box Flooring

Owiny JR, Vandewoude S, Painter JT, et al: *Comp Med* 51(1):85-88, 2001.

ABSTRACT

Hip dysplasia or splay leg is diagnosed occasionally in rabbits. Environmental factors have been implicated in the pathogenesis; however, specific causative sources have not been reported. This study describes the influence of flooring type along with the clinical and pathologic findings associated with splay leg in a colony of Dutch-belted rabbits. The effects of exposure to dichlorodiphenyltrichlorethane (DDT) and heredity on the incidence of the condition were also investigated.

The animals were part of a reproductive toxicologic study and housed in a facility accredited by the Association for Assessment and Accreditation of Laboratory Animal Care International (AAALAC). Adult does and bucks were kept in stainless-steel cages with slatted floors containing drop pans with aspen shavings. Dams were fed Prolab breeder diet (5P29, PMI Nutrition International, Brentwood, MO). Kits were fed Prolab Hi-Fiber pellets (5P25, PMI Feeds, St. Louis, MO) after being weaned from the dam.

After artificial insemination, pregnant does were exposed to either corn syrup (vehicle control) or to DDT (25 or 240 µmol/kg) in corn syrup daily from midgestation through 4 weeks postparturition. Does were given nest boxes with aspen shavings on day 27 of gestation.

Of the 95 kits that were given waxed cardboard nest boxes, 7% developed splay leg. Another 95 kits were given stainless-steel nest boxes with plexiglass floors; of these, 22% developed splay leg, valgus deformity, and patellar luxation in one or both hindlimbs. Body weights of affected and asymptomatic rabbits were similar. Because the plexiglass floors caused a higher incidence of splay leg than the cardboard floors, the investigators believed that the lack of traction could be the cause. Thus, they placed textured nonslip plastic strips (Safety-Walk, Medium Resilient Treads, 3M, St. Paul, MN) over the plexiglass for an additional 129 kits. None of these kits exhibited signs of splay leg.

Kits from all three groups were examined at 2 to 4 weeks of age for inability to adduct one or more limbs and then classified as normal or dysplastic. At 12 weeks of age, 12 affected kits (seven unilateral, five bilateral) and four normal kits raised on the plexiglass flooring without strips were given physical examinations. Eight of these kits received the control vehicle, and eight received DDT. Physical examinations did not reveal abnormalities other than splay leg with one or both hindlimbs extended laterad or caudad. Flexion of these limbs was markedly restricted. Three affected kits and one normal kit were also given complete neurologic examinations, including hindlimb nerve velocities, and pelvic radiographs. Neurologic examinations did not reveal abnor-

malities other than gait deficits due to an inability to adduct the hindlimbs. Nerve conduction velocities were similar for normal and affected limbs among both the affected and asymptomatic kits. Radiographic changes varied in severity from subluxation to luxation of the coxofemoral (CF) joint with bony proliferation and remodeling of affected acetabula. The affected acetabula were widened and shallow, measuring one-third to one-half the depth of the normal control.

Mild pathologic changes in affected kits included shallow acetabula, subluxation of the femoral head, and CF joint capsular thickening. Severe pathologic changes included markedly shallow to flattened acetabula, thickening and deformity of the femoral head and neck, prominent thickening of the CF joint capsule, and tibial bowing. Gross pathologic changes correlated with radiographic findings. Muscle atrophy or hypoplasia was not observed grossly; however, those changes were noted histologically in 25% of controls and 92% of affected animals. Sections of peripheral nerves associated with the muscle appeared normal in all animals. Histologic findings confirmed the thickening of the CF joint capsule with fibrocartilage formation, mild trabecular bone loss, bony sclerosis of the proximal femur, and adductor muscle hypoplasia.

The striking increase in incidence of splay leg concomitant with the change in flooring from smooth cardboard or plexiglass suggests traction as an important environmental factor in the development of hip dysplasia in rabbits. This theory is supported by the resolution of the condition once textured plastic strips were applied to improve traction. Although the DDT-treated kits had an increase in incidence of splay leg, continued studies using a variety of pesticides have not resulted in hip dysplasia in those rabbits that were kept in the altered nest box flooring. It is theorized that those particular kits may have been weaker or slower to develop, thereby enhancing the outcome of an unstable floor surface on musculoskeletal development.

It has been previously suggested that genetic factors play an important role in splay leg. In this study, the rabbits had unrelated parents; in three subsequent breeding trials using half of the does and pooled semen from at least half of the sires in the original study, no new cases of hip dysplasia were noted. All resulting kits were housed on the flooring with traction strips. Because of the design of this study, the role of a genetic component as well as other temporal factors cannot be ruled out.

COMMENTARY

When diagnosing hip dysplasia in young rabbits, it is important to get a thorough history to determine if the condition was caused by inappropriate substrate. If the condition is caught early, it may be possible to improve mobility by switching to flooring that provides traction.

*Melissa A. Kling, DVM
Secretary*

Association of Exotic Mammal Veterinarians

Chloramphenicol Treatment for Rabbit Syphilis

Saito K, Hasegawa A: *J Vet Med Sci* 66(10):1301–1304, 2004.

ABSTRACT 1

To establish a safe and efficient treatment for rabbit syphilis in companion rabbits, the efficacy of oral chloramphenicol was evaluated. All 39 cases clinically improved and recovered promptly. Fourteen of the cases (35.9%) relapsed, but most remained sensitive to chloramphenicol. Because safety is more important than efficacy in treating syphilis in domesticated rabbits, chloramphenicol should be chosen as a first-line agent. At the initial onset of disease, 3-week administration of chloramphenicol may be adequate. If relapse occurs repeatedly or if the owner cannot administer the medicine adequately, treatment with penicillin should be considered.

Rabbit Syphilis Diagnosed Clinically in Household Rabbits

Saito K, Tagawa M, Hasegawa A: *J Vet Med Sci* 65(5):637–639, 2003.

ABSTRACT 2

This paper deals with cases of clinically diagnosed rabbit syphilis presented from April to December 2001; the

rabbits showed distinct lesions around the nose and/or mouth, the rapid plasma reagin test was positive, and they responded to chemotherapy. Of 16 cases, 12 exhibited initial symptoms and four were relapses. Lesions around the genitalia and/or anus as well as the nose and/or mouth were seen in eight cases, and sneezing was observed in six cases. Fifteen cases were successfully treated with oral administration of chloramphenicol, and one was treated with long-acting penicillin by intramuscular injection. The mean age at onset was 8.8 months. Because none of these cases had any mating history, the authors concluded that the disease was likely to be maternally transmitted.

COMMENTARY (ABSTRACTS 1 AND 2)

Traditional treatment for rabbit syphilis is parenterally injected penicillin, which is associated with a high cure rate and a low rate of adverse side effects. The authors of these two papers suggest that the chance of untoward effects of penicillin makes the use of alternative drugs preferable for first-line use. Both papers present cases that were treated with chloramphenicol, and the findings from the cases support the efficacy of chloramphenicol. Use of this drug, however, should be balanced against the low rate of side effects of injectable penicillin along with the potential human hazards of contact with chloramphenicol.

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SPECIAL FOCUS: DIAGNOSTIC TESTING

Excretory Urography by Intraosseous Injection of Contrast Media in a Rabbit Model

Porzio P, Pharr JW, Allen AL: *Vet Radiol Ultrasound* 42(3):238–243, 2001.

ABSTRACT

There are many indications for an intravenous excretory urogram. However, where intravenous access is not available, the intraosseous route to the circulation may be an alternative. The authors found that safe and diagnostic excretory urograms could be obtained in rabbits following the injection of different contrast media via the intraosseous route. In fact, these excretory urograms were indistinguishable from ones obtained by the conventional intravenous route. The rabbits did not develop any abnormal clinical signs after the procedure; however, five of 22

(22.7%) tibiae receiving an intraosseous needle had post-mortem histologic lesions of osteochondrosis, whereas none of the 14 tibiae that did not receive an intraosseous needle had lesions. Further, the use of diatrizoate was associated with the development of osteochondrosis, whereas the use of iopamidol was not.

COMMENTARY

Because of the small size of many exotic mammal patients, certain diagnostic tests can be difficult to perform. Intraosseous fluids are commonly administered to sick patients. This article demonstrates the utility of intraosseous catheters for performing excretory urograms. Further studies are required to determine the utility of this procedure and the frequency of potential side effects, including osteochondrosis.

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Journal of Exotic Mammal Medicine and Surgery Partners with Seminars in Avian and Exotic Pet Medicine

We are pleased to announce that, beginning January 2006, the Association of Exotic Mammal Veterinarians will partner with Elsevier, a world-leading scientific and medical publisher, making *Seminars in Avian and Exotic Pet Medicine* an official publication of the AEMV. This decision has been considered very carefully, and it is the opinion of the board of directors and the overwhelming majority of members present at the annual membership meeting at ICE in May 2005 that this merger will be a significant benefit. The most immediate benefit is expansion of the publication from two issues of *JEMMS* to four issues of *Seminars*, which will be expanded to include original small mammal research and other peer-reviewed articles. Also included will be a section dedicated to AEMV news and events.

To summarize, membership in AEMV will now automatically include a subscription to *Seminars in Avian and Exotic Pet Medicine* and will represent an actual monetary savings over a subscription to *Seminars* alone. Members who already receive *Seminars* will have their subscriptions automatically converted to AEMV membership at the time of renewal.

Dr. Tom Tulley, an Editor-in-Chief of *Seminars*, remarked, "The Association of Exotic Mammal Veterinarians is one of the premier organizations dedicated to the study and practice of exotic animals. We at Elsevier are very pleased and proud to partner with AEMV and to support the Association in its mission to further advance veterinary care of exotic mammals."

Join the AEMV Yahoo Group

AEMV is growing rapidly, focusing on several important new projects and experiencing a number of exciting changes. To communicate more effectively, we would like to reactivate the AEMV Yahoo email group. This free service will allow us to update our members directly one to three times monthly, rather than having to wait for each edition of the journal. You may have already received an invitation to renew participation or join the group. Unfortunately, we do not have correct email addresses for many of you. If you did not receive a notice and wish to participate, please follow the instructions to join the Yahoo group:

1. Enter the following URL into your Web browser's address box: <http://groups.yahoo.com/group/AEMV/>.
2. Click on the "Join this Group" button in the upper right corner.
3. If you already have a Yahoo ID, please enter it. If you do not have a Yahoo ID, click on "Sign Up."

Because this is a members-only group, a request will be made to the Group Moderator to accept your request to join.

Exotic DVM of the Year Award Goes to Lennox

The 2005 Exotic DVM of the Year Award was given to our president, Dr. Angela Lennox, at this year's ICE. This award is presented annually to an individual who embodies the essence of stewardship of exotic companion animal species. AEMV extends congratulations to Dr. Lennox on this prestigious award and thanks her for her dedication to making our organization a success.

Exotic DVM Paper of the Year Award Goes to Capello

The 2005 Exotic DVM Paper of the Year was awarded to AEMV member Dr. Vittorio Capello. The award was announced at this year's ICE. His paper, "Extraction of Cheek Teeth and Dental Disease," was selected for its overall excellence and direct applicability to exotic animal practitioners. Dr. Capello was AEMV's featured instructor for our Rabbit and Rodent Dental Disease wet lab held in conjunction with ICE. Congratulations!

Important Notes from the Secretary

Please be sure to check your listing on the AEMV homepage. If it is incorrect or absent, please send an email to info@aemv.org to have the listing corrected. If you renewed or joined using PayPal before April 2005, you will need to go into your PayPal account and cancel the subscription. If you do not, your credit card will automatically be charged on the anniversary date. For obvious reasons, this has proven problematic but has been corrected for the future. If you need assistance in this matter, please email our Secretary, Dr. Melissa Kling, at mkn.dvm.uga@att.net.

Dear Editor,

I read with interest Dr. Johnson-Delaney's article titled, "Melatonin Study with Four Intact Male Ferrets and Two Female Ferrets with Adrenal Disease" (Vol. 3.1, May 2005) and was surprised to read the cost of Lupron quoted as "\$5.50/100 µg of the 7.5-mg Lupron . . . for 3 months would be \$16.50. For ferrets receiving 200 µg of Lupron monthly, my cost for 3 months' therapy is \$33." According to the peer-reviewed literature, the only formulation of Lupron that has been US Food and Drug Administration (FDA) approved (and hence tested for purity and efficacy) and has been shown to be efficacious in the treatment of ferret hyperadrenocorticism is the 30-day 3.75-mg Lupron Depot produced by TAP Pharmaceuticals Products (Lake Forest, IL).¹ The listed price for this drug is \$445.52 for 3.75 mg, which equates to \$11.88 per 100-µg dose (\$35.64 for a 3-month course or \$71.28 for a 3-month 200-µg course). Even if using the 7.5-mg Lupron, the costs are \$21.44 and \$42.88 for 3-month courses of 100 µg and 200 µg, respectively. These figures are significantly higher than those quoted in the article, and both sets of data assume that every single microgram of drug is used for therapy whereas, in reality, compounding and storage are likely to result in 5% to 10% wastage, and hence 5% to 10% higher pricing. Shipping costs and practice markup must also be taken into consideration for final drug pricing. Therefore, it would seem that the figures quoted for Lupron therapy were significantly underestimated and in fact would appear to be comparable to, if not greater than, the melatonin implants.

Of perhaps even greater concern is that the compounded formulations of Lupron by various compounding pharmacies have never been tested or proven by FDA standards to be effective, pure, and efficacious. If a compounding pharmacy can supply Lupron at a cheaper price than the manufacturer, then I would urge colleagues to be very wary of the quality of the product being prescribed. According to pharmacists at the University of Georgia, a chemical-grade (not pharmaceutical-grade) product may be cheaper to obtain and compound, but purity and efficacy are unproven. It is wise to always remember that you do not know the efficacy of a drug after it has been compounded by a nonregulated third party.

Finally, there appears to be some controversy surrounding the efficacy of implants. Recent peer-reviewed papers detailing the use of implants (melatonin and deslorelin) appear to indicate that they are effective to some degree.²⁻³ However, the recent article by Dr. Johnson-Delaney casts serious doubt on the effectiveness of melatonin implants. While I agree that the evidence in support of melatonin is not as well documented as it is for deslorelin, this issue is made even more unclear by trying to compare two papers of differing stature in the same issue of *JEMMS*.^{2,4} One paper was clearly marked peer reviewed ("Melatonin Implants: An Option for Use in the Treatment of Adrenal Disease in Ferrets" by Dr. Jerry Murray), but the other (by Dr. Johnson-Delaney) was uncategorized and presumably not peer reviewed.

It seems clear from the continued clinical concerns and debates on ferret adrenocorticism and the increasing number of potential therapies that there is no single, infallible treatment. From a veterinarian's perspective, I hope we will strive to spend more resources in trying to prevent this disease, and from a welfare perspective (and 7 years in England), I hope we can better advise our small mammal clients on which species make better pet alternatives to ferrets.

Best wishes and kindest regards,

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4. Johnson-Delaney C: Melatonin study with four intact adult male ferrets and two female ferrets with adrenal disease. *JEMMS* 3(1):7-9, 2005.

Author's Response

I would like to thank Dr. Hernandez-Divers for his thoughtful comments. With regard to formulations of Lupron, the 3.75-mg and the 7.5-mg depot 30-day microsphere are identical and are simply labeled differently for use in human men (7.5 mg) and women (3.75 mg) for different medical applications. Therefore, 100 µg pulled from a kit of 3.75 mg or 7.5 mg is identical. I have discussed the formulations with TAP Pharmaceuticals, so the "validation" point is moot. The prices I quoted are exactly what I pay for use in three different facilities in the Seattle area. We order the 7.5-mg kit from Canada legally in my state, and I dilute it and freeze it in aliquots. There is very little waste. I get 37.5 doses of 100 µg from the 3.75-mg kit and 75 doses from the 7.5-mg kit.

During the initial studies conducted in collaboration with Dr. Jack Oliver and the University of Tennessee, we started out using the 3.75-mg package for a few months and then discovered it was much more economical to use the 7.5-mg package. I worked closely with TAP, providing them with all the information on our use of the product, and their safety and marketing groups approved of how we mixed and froze the product. In the early phase of our first Lupron study, we did quality control checking of the product we

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compounded ourselves and were convinced that our mixing technique resulted in the correct dosage.

With regard to compounding and product efficacy, I agree with Dr. Hernandez-Divers' comments and would like to add that anyone purchasing medications, especially injectable medications, from compounders should periodically submit a batch or vial to the laboratory for analysis. I have been appalled at the lack of quality control from many compounders, which typically do not submit their finished batch for spectrophotometer/chemical analysis to guarantee the product they sell is as promised. The paper I wrote for the May 2005 *JEMMS* was peer reviewed and included not to increase confusion, but to make clear how much more work there is to do on this subject. Melatonin, Lupron, or newer drugs such as deslorelin are not curing the disease, as Dr. Hernandez-Divers points out, but are helping buy the ferret a normal life span. I believe that until the major suppliers change their procedures, the problems of early spaying and neutering are not going to go away. To this end, Dr. Michelle Hawkins of the University of California, Davis, and I have submitted projects to try to identify tumor-suppressor genes, which we believe are at the very root of the problem. If we solve this issue and change breeders' neutering protocols, perhaps in the future this disease will become

a thing of the past. But until these studies are completed and published, proving that adrenal disease is linked to genetic propensity coupled with husbandry practices, the major producers will not change their ways. Perhaps pressure from major pet store chains and the ferret-buying public could help as well.

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Editor's Note

Drug costs for Lupron in our clinic are more similar to those reported by Dr. Hernandez-Divers, with the approximate cost of 100 µg being \$8.13. Close examination of the final product after reconstitution and division by a technician fortunately revealed very little waste. Final drug costs obviously will depend on the original cost of the drug plus reasonable markup and any cost associated with administering the drug. Because owners are able to pay for Lupron once a month, and many learn to safely give injections at home, we have found the acceptance rate of Lupron therapy to be very high.

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