MASTER CLASS

HUSBANDRY, DISEASES AND VETERINARY CARE OF THE BEARDED DRAGON, *Pogona vitticeps*

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ABSTRACT: Bearded dragons are a popular reptile. This paper reviews natural history, husbandry, behavior, nutrition, and reproduction of the bearded dragon. In addition, important aspects of physical examination, diagnostic techniques, and common diseases are included.

KEY WORDS: bearded dragon

INTRODUCTION

The bearded dragon's (*Pogona vitticeps*) appearance, docility, captive hardiness, omnivorous diet, diurnal activity and manageable size (<20 in) have combined to make this reptile very popular (Christie, 1993). Several pairs were imported from Germany in the 1980's and were the founding stock of the captive population in the United States today. Bearded dragons consist of 8 species all of which are from the eastern and central (inland) parts of Australia (Weis, 2001). The "true" Rankin's dragon (*P. henrylawsoni*) is rarely available in the pet industry due to health issues associated with inbreeding and hybridization with *P. vitticeps*. The inland species has proven to be the most hardy and consistently reproductive in captivity. Assorted color varieties and sizes are available in the pet trade including sandfire and pastel color morphs and the so called "German giants." Most recently, there are dragons with reduced scale size (leatherbacks) and scaleless (silkbacks) mutations. Lack of genetic diversity may be a contributing factor for unthriftiness and susceptibility to disease.

NATURAL HISTORY

Bearded dragons belong to the family Agamidae. Agamids are the fourth largest family of lizards with some 300 species spread throughout Africa across Asia and into Australia. Agamids are absent from Madagascar. Examples of agamids are frilled dragons (*Chlamydosauras kingii*), spiny-tailed lizards (*Uromastyx* spp.), sailfin lizards (*Hydrosaurus* spp.), flying dragons (*Draco* spp.), water dragons (*Physignathus* spp.), mountain horned lizards (*Acanthosaura* spp.), toad-headed (Sinai) agamas (*Phrynocephalus mystaceus*) and butterfly agamas (*Leiolepis* spp.) (Rogner, 1997). Agamids are found in a variety of habitats including rain forests, wooded hillsides and deserts. These lizards possess round pupils, fleshy tongues and are not capable of caudal autotomy. Males have elaborate warning and courtship behaviors. Most, including the bearded dragon, are egg-layers. The natural habitat of bearded dragons consists of xeric

woodlands. In the wild, bearded dragons consume arthropods, worms, small rodents, lizards, greens, fruit and flowers.

HUSBANDRY AND BEHAVIOR

An appropriate enclosure should measure 4-6 ft long by 2 ft wide with a height of 2-3 ft. Most dragons are maintained in aquariums (45-55 gallons) with a screen top. Adults are kept alone or as sexed pairs. Adult males should not share the same cage. Arm waving by either gender in the presence of dominant conspecifics represents submissive behavior. Flattening of the body, erect stance, head bobbing, gaping, and an expanded black dewlap represent defensive behavior. A dominant male will usually stay on the highest perch in the cage. Recommended substrates are calciferous sand, beach sand, 2 inch pea gravel or carpet. The author recommends newspaper or paper towel for ease of cleaning and prevention of substrate ingestion. Climbing furniture such as branches or rocks should be provided to allow for thermoregulation. Hide areas are also recommended. Full spectrum lighting containing ultraviolet B (UVB; 290-320 nm) and a basking area of 95-105^{o}F are essential for normal skeletal development (Strimple and Strimple, 1998). The cooler part of the enclosure should be no less than 80^{o}F. A drop of 10-20^{o}F is permissible in the evening for adults. The author discourages the use of hot rocks; heating pads placed on the underside of the enclosure are more appropriate. A shallow water dish should be present at all times despite the fact that some dragons never seem to drink. Additionally, spraying the sides of the enclosure with tepid water every 24-48 hr. is recommended. Ecdysis occurs in multiple pieces; shedding frequency depends on growth rate. Breeding occurs September through March in the wild. Life expectancy in captivity is 5-10 yr. It is best to acquire a juvenile that is 2-3 mo of age.

NUTRITION

In captivity, bearded dragons consume insects (crickets, mealworms, superworms (*Zoophobas*) and assorted leafy vegetables (dandelion, Swiss chard, escarole, endive, romaine, spring mix, chicory, mustard, beet tops, bokchoy, etc.), carrots, squash and zucchini (Strimple and Strimple, 1998). Hatchlings and juveniles (< 4 mo. of age) can be offered 30% vegetables and 70% appropriately sized crickets (< width of dragon's head) (De Vosjoli and Mailloux, 1996). Proportions of diets can be approximately 50% insects and 50% vegetables for adults. Insects should be "gut-loaded" with a balanced commercial food (i.e., T Rex® Calcium Plus Food for Crickets). A Vitamin D-free calcium supplement should also be sprinkled on the feeder insects and vegetables immediately prior to presentation. Hatchlings <2 mo of age should be fed 2-3 times/day. Juveniles at 2-4 mo of age can be fed 2 times/day (Johnson, 2004). Animals from 4 mo of age to adulthood, can be fed daily to every 2 days (De Vosjoli and Mailloux, 1996). It is recommended to offer food in the mornings so that digestion occurs during the warmest part of day. A multivitamin powder that contains preformed Vitamin A can be provided once per month.

REPRODUCTION

Sexual maturity is attained by 18-24 mo of age. Brumation is achieved by cooling the adults for 4-6 wk. at 60-70°F range with no lighting during the late fall or early winter (Strimple and Strimple, 1998). Water should be provided during this time. The ambient temperature is then gradually increased to maintenance temperatures over a period of 2-3 wk. The lizards are fed and introduced to each other. Courtship consists of the male darkening its dewlap and circling the female. A receptive female flattens its body and permits copulation. After several days the male is separated from the female. When the female begins to show signs of gravidity, a nest box is provided that contains damp sand or potting soil at a depth of 10 in. After oviposition, the eggs are removed from the enclosure and incubated in vermiculate at a 1:1 ratio by weight with water. The incubator should have an ambient temperature of 82-86°F. Eggs typically hatch in 3-4 wk (Strimple and Strimple, 1998). Neonates possess an egg tooth that is used to facilitate hatching. Neonates should be housed separately and offered food of appropriate size within 24 hr. Hatchlings should be misted daily.

PHYSICAL EXAMINATION

The physical examination should begin with simple observation of the lizard in its carrier. Most bearded dragons are transported in small aquariums or transparent plastic containers. Observe its body mass, level of awareness, posture, skin color, injuries, locomotion, respiration, body conformation, etc. Bearded dragons are more comfortable when they are permitted to rest on a flat surface rather than manual restraint. Obtaining an accurate weight (gm) is very important.

Starting at the head, look for abrasions, jaw swelling, ocular lesions, patency of external nares and tympanic membrane anatomy. Bearded dragons possess a 3rd or parietal eye located between the eyes which enervates the pineal gland (Raftery, 2004). The pineal gland controls circadian rhythms, reproduction and behavior. The mouth is opened by gently depressing the tip of the mandible with an index finger. Bearded dragons possess acrodont dentition (Raftery, 2004). Acrodont teeth are ankylosed to bone and are not replaced if damaged. Color of the oral mucus membranes is normally pale yellow. The tip of the tongue is darker in color than the rest of the tongue. The glottis is situated on the floor of the mouth just caudal to the tongue. It is only visible when the mouth is opened widely. Normal skin color ranges from brown to orange depending on color morph, health status and body temperature. Look for areas of discoloration, swelling, dysecdysis (toes), scarring, etc. Palpate the limbs and joints including the vertebral column. The heart is ascultated with a doppler monitor. Heart rate is temperature dependent. Coelomic palpation is done to assess the presence of ingesta, masses, size of fat bodies and possible foreign bodies. Care must be taken when palpating adult females with suspected ovarian follicles. Check the ventral aspect of the body; pay particular attention to the vent and femoral glands. Males generally possess larger femoral glands than females (Raftery, 2004). In mature males, the paired hemipenes form bulges on either side of the tail base.

DIAGNOSTIC TECHNIQUES

A) Venipuncture: The preferred sample site is the ventral coccygeal vein (Heard et al., 2004). A tuberculin syringe with a 25 gauge needle is preheparinized prior to blood sampling. This vein can be accessed using either the ventral or lateral approach. Once the vertebral body is encountered, gentle aspiration is done. Sample volume (ml.) is calculated as 0.1 % of the body weight (gm). Other less desirable sites are the ventral abdominal vein, brachial plexus and heart. The ventral abdominal vein is located on the midline of the caudal coelom. There is the potential risk of uncontrolled hemorrhage and laceration of internal organs if the animal suddenly moves during sampling from this site. The brachial plexus is accessible from the axillary areas. The heart is located between the shoulders. Cardiocentesis should only be attempted with ultrasound guidance. Normal hematology/plasma biochemical values are included herein (International Species Information System, 2002; Table 1).

B) Fecal analysis: Fecal examinations are particularly important in bearded dragons. Endoparasitic infestations (coccidiosis, oxyuridiasis) are very common. Cestodiasis and amoebiasis are less common. Sometimes, cloacal contents can be gently "milked" through the vent. A cloacal lavage can be done by inserting a lubricated catheter, feeding tube or ball-tipped feeding needle and an appropriate volume (10 ml/kg) of saline is flushed into the cloaca (Klingenberg, 2004).

C) Urinalysis: Bearded dragons lack a urinary bladder. Urine is stored in the ureters and urodeum. Most of the urinary excreta is composed of semi-solid urates and some fluid. Urine specific gravity (1.003-1.014) of reptiles is normally lower than in mammals (Johnson, 2004). The presence of red blood cells is abnormal.

D) Non-invasive imaging: Radiography, Ultrasonography, Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) are routinely used for reptiles. Radiography is most useful for evaluating the skeletal, respiratory and reproductive systems (females). Common radiographic findings are metabolic bone disease, osteomyelitis, articular gout, fractures, lower respiratory tract infection, follicular development, gravidity and metastatic mineralization (Raiti, 2004). Ultrasonography is indicated for visualization of internal organs provided there is a soft tissue window. It is particularly useful for examination of the heart, liver and ovaries. CT, unlike radiography, is not limited by overlying structure such as bone. It provides transverse slices of all organs and is particularly useful for evaluation of bone and lungs. MRI is most useful for evaluation of all soft tissues and provides slice orientation (coronal, transverse and sagittal) views. CT and MRI require chemical immobilization.

E) Endoscopy: This minimally invasive technique provides direct visualization of organs and permits biopsy sampling (Hernandez-Divers, 2004). General anesthesia is required. Celioscopy is useful for evaluation of coelomic organs. Cloacoscopy is useful for evaluation of the lower urogenital and intestinal tracts (Hernandez-Divers, 2004).

F) Necropsy: Necropsy is an invaluable tool that assists the clinician in improving diagnostic skills. Physical examination and surgical abilities are also enhanced. In many cases, necropsy and histology are required for a final diagnosis. It is recommended to archive pictures of normal organs to be used for comparison with diseased tissues.

DISEASES

A) Nutrional metabolic bone disease: This is the most common disorder of juvenile bearded dragons. Symptoms consist of anorexia, weakness, constipation, motor paralysis, tetany, skeletal deformities and pathologic fractures. It is characterized by poor skeletal calcification and hypocalcemia. NMBD is due to one or more of the following: deficiencies of dietary calcium (Ca), phosphorus, Vitamin D_3, and/or Vitamin A, lack of exposure to UVB and suboptimal basking temperatures (Calvert, 2004). Treatment depends on identification and correction of inappropriate husbandry practices. The author recommends oral Ca administration such as Ca glubionate syrup at 1ml/kg q 12 hr for critical patients until there is a positive clinical response. See Husbandry and Nutrition sections.

B) Trauma: Trauma is commonly seen in neonates and juvenile animals. These reptiles are commonly maintained in crowded, communal enclosures in pet shops or reptile shows. Wounds are typically found on the toes and tail. Adult males should not be kept together. Injuries can also occur during feeding. Treatment consists of surgical debridement and topical and systemic antimicrobials.

C) Endoparasitism

1) Intestinal Coccidiosis: Coccidiosis is extremely common in bearded dragons. Coccidiasis has been used to describe the presence of oocysts in the feces; however, the dragons are clinically normal (Greiner and Mader, 2006). Coccidiosis describes active infection and disease in affected animals which are usually neonate or juveniles (Greiner and Mader, 2006). Symptoms consist of anorexia, lethargy and diarrhea. It is likely that symptoms are due to concurrent infections such as adenovirus and microsporidiosis. Treatment is often unrewarding with traditional potentiated sulfas. The author recommends ponazuril at 30 mg/kg PO; repeat in 48 hr. (Bogoslavsky, 2007). Occasionally, the treatment needs to be repeated in 2 wk. Environmental decontamination is essential.

2) Microsporidiosis: (*Pleistophora* spp.) Microsporidiosis is considered to be very common in bearded dragons particularly in individuals originating from commercial breeding facilities. Microsporidia are intracellular protozoans that possess a dark staining polar cap. This organism causes nonspecific symptoms such as anorexia, cachexia and encephalopathy (Jacobson et al., 1998; Reavill and Schmidt, 2009). Clinically healthy animals may suffer acute death. The disease has been reported in dragons of all ages. Transmission occurs by the fecal-oral route from infective spores passed into the environment by infected dragons. Vertical transmission is also thought to occur. Feeder insects have been implicated in disseminating the disease; however, this has not been proven. Many affected animals have concurrent diseases such as adenovirus, coccidiosis and salmonellosis. Diagnosis is by histopathology. Multifocal granulomas surrounding infected macrophages or epithelial cells are commonly identified in the

kidneys, heart, spleen, intestine and brain (Reavill and Schmidt, 2009). There is no treatment for microsporidiosis.

3) Additional Endoparasites: Oxyurids (pinworms) are commonly identified in the feces of bearded dragons. These helminths inhabit the lower intestinal tract and in small numbers do not cause overt disease. In massive numbers, pinworms have been associated with unthriftiness, impaction and cloacitis (Klingenberg, 2004; Greiner and Mader, 2006). Fenbendazole at 100 mg/kg PO divided over 3 days and repeated in 2 wk is usually curative (Klingenberg, 2004). Cestodes are occasionally seen in fecal preparations. Treatment consists of praziquantel at 5-8 mg/kg PO or IM (Klingenberg, 2004). Entamoebiasis, although uncommon, is extremely pathogenic in bearded dragons. Hepatic and intestinal granulomas are associated with trophozoite migration (Klingenberg, 2004). Affected dragons are usually moribund at presentation. Diagnosis is by the presence of motile trophozoites or multinucleated cysts in direct fecal smears. Treatment can be attempted with metronidazole at 100 mg/kg PO q24hr for 2 doses and then q48 hr for 10 doses (Davies and Klingenberg, 2004). Flagellates are occasionally encountered in bearded dragons. Treatment consists of metronidazole at 100 mg/kg PO; repeat in 2 wk) (Klingenberg, 2004).

D) Ectoparasites: The snake mite (*Ophionyssus* spp.), lizard mite (*Hirstiella trombidiformis*) and chigger mite (Family: Trombiculidae) are occasionally seen on bearded dragons. Skin folds (i.e., lips, external ears and vent) should be checked carefully to visualize these ectoparasites. There are several treatment options: a) 1% injectable ivermectin at 5 mg/0.95 L of water sprayed topically on lizard q 7 days for 8 treatments; b) fipronil 0.29% sprayed topically on lizard; wipe away excess with dampened gauze; wash off drug with water after 5 min.; repeat in 7 days; c) 10% permethrin; dilute to 1% in tap water; apply topically on lizard in well ventilated room.; repeat in 10 days if necessary (Mader, 2006). Enclosures (without reptiles present) can be sprayed with the same insecticides and then washed with hot water (>120^{o} F).

E) Adenovirus: Infection causes nonspecific symptoms (anorexia, lethargy, diarrhea and encephalopathy) in affected bearded dragons with highest morbidity/mortality occurring in neonates and juveniles; however, dragons of all ages can be affected (Boyer and Frye, 2000; Reavill and Schmidt, 2009). Subclinical carriers can act as reservoirs. It is suspected the virus can be passed vertically, since neonates have expired from outbreaks despite being housed separately from the parents. Adenovirus infection typically leads to poor growth and mortality in young dragons and poor growth and failure to thrive in older animals. The liver, pancreas, kidneys and intestines are most commonly affected (Cranfield et al., 1996; Crocker et al., 2005). The virus infects enterocytes and is shed in the feces. Diagnosis up to this point has been by histopathology demonstrating the presence of basophilic intranuclear inclusions in hepatocytes, enterocytes and kidneys (Wagner et al., 2007). Currently, antemortum diagnosis is possible by either fecal-based polymerase chain reaction assay or negative staining electron microscopy (Ritter et al., 2009; Van Waeyenberghe et al., 2010). The advantage of the latter test is that it can also detect other viruses in the feces (i.e., calicivirus, dependovirus, coronavirus, parvovirus, rotavirus, etc.). There is no treatment for adenovirus.

Constipation: Constipation is usually due to sand ingestion. Symptoms are anorexia, lethargy, absence of feces and enlargement of the coelomic cavity. Tumors and organ enlargement are also

differentials to consider. Diagnosis is by palpation and radiography. Pica is commonly associated with poor husbandry (i.e., suboptimal temperature, lack of UVB, poor nutrition, dehydration, systemic disease, parasitism). Medical treatment consists of warm water soaks and mineral oil enemas.

Gout: In reptiles, hyperuricemia develops most commonly due to chronic renal disease. The inability of the kidneys to clear the blood of uric acid by tubular excretion leads to precipitation of urate crystals in major organs; hence, the term visceral gout (Johnson, 2004; Mader, 2006). Deposition of urates in and around joints is called articular gout. Predisposing factors are inappropriate diet (excessive animal protein), hypothermia and chronic dehydration (Mader, 2006). Diagnosis of articular gout is by radiography and arthrocentesis. Needle shaped crystals (urates) are readily visible microscopically. Hematology and biochemistries demonstrate renal disease (hyperproteinemia, hypernatremia, hyperkalemia, hyperphosphetemia and hyperuricemia). Management consists of correcting husbandry problems, reversal of renal disease and the use of xanthine oxidase inhibitors such as allopurinol at 20 mg/kg PO q 24 hr to lower uric acid production (Mader, 2006).

Chrysosporium* anamorph of *Nannizziopsis vriesii (CANV) : Also known as "yellow fungus disease," CANV is a primary fungal pathogen causing granulomatous dermatitis in snakes, lizards and crocodilians (Pare et al., 2001; Reavill and Schmidt, 2009). The CANV is not part of the normal dermal microbiota of reptiles and is capable of causing disease in healthy individuals (Pare et al., 2001). The bearded dragon appears to be particularly susceptible to CANV. Skin lesions appear as yellow to yellow-brown hyperkeratotic areas that can also affect the toenails. The CANV is capable of systemic invasion and has been identified in the lung, coelomic fat pads, liver, and spleen (Davies and Klingenberg, 2004; Reavill and Schmidt, 2009). Diagnosis is by histopathology and confirmed by fungal culture (Pare et al., 2001; Reavill and Schmidt, 2009). The CANV is considered contagious among bearded dragons and possibly zoonotic in immunosuppressed humans. In 7 bearded dragons, itraconizole (Sporonox oral solution, Ortho Biotech, Raritan, NJ, USA) at 5 mg/kg PO q24hr eliminated CANV by 27 days; however, there was 70% mortality presumably due to hepatic toxicity (Van Waeyenberghe et al., 2010). In the same study, 7 bearded dragons that were administered voriconizole (Voriconizole oral suspension, Pfizer Animal Health, Exton, PA, USA) at 10 mg/kg PO q24hr were cured of CANV by 47 days with 14% mortality. Plasma minimal inhibitory concentrations of itraconizole and voriconizole exceeded the mean inhibitory concentrations of both drugs during treatment. The authors concluded that voriconazole administered at a regimen of 10 mg/kg PO q24h seemed to be a safe and effective antimycotic drug to eliminate CANV infections in bearded dragons.

Fungal Stomatitis: *Emmonsia* sp. was identified as the causative agent of oral granulomas in a bearded dragon (Boyer and Garner, 2005). The granulomas involved the intermandibular space and portions of the upper jaw. *Emmonsia* are filamentous fungi found in soil and the lungs of burrowing rodents. The source of the infection was thought to be associated with poor husbandry of silkworm prey. The dragon died approximately 1 wk after presentation and 2 days after starting ketoconazole.

Mycobacteriosis: Mycobacteriosis is a potentially zoonotic disease that causes topical and systemic granulomatous lesions in reptiles. Tissue samples from affected sites should be acid-fast stained and if positive, cultured for microbiologic identification. In a retrospective study of 3,880 reptile accessions, 28 mycobacterial positive reptiles were identified (0.7%).[30] A bearded

dragon had oropharyngeal lesions from which *M. marinum* was identified (Reavill and Schmidt, 2010). Euthanasia of affected reptiles is recommended.

Salmonellosis: *Salmonella* spp. are considered part of the normal microflora of the gastrointestinal tract of reptiles and for the most part do not cause disease in healthy animals (Mitchell, 2006). These bacteria are capable of causing coelomitis, abscesses, pneumonia and sepsis in susceptible reptiles (Mitchell, 2006). All reptiles should be considered carriers of *Salmonella*. It is potentially zoonotic and owners should be advised to follow the guidelines on the handout developed by the Association of Reptilian and Amphibian Veterinarians and Centers for Disease Control.

Listeriosis: A moribund 2.5-yr-old male bearded dragon presented with anorexia and diarrhea of 3 days duration. Oral petechiation was noted on physical examination. Histology and microbiologic culture/sensitivity identified *Listeria monocytogenes* meningitis and septicemia (Girling and Fraser, 2004). Listeriosis is a zoonotic disease frequently associated with low environmental temperatures, such as is encountered in the refrigeration of human foodstuffs, or in soil contamination in livestock (Girling and Fraser, 2004). It was suspected that this lizard contacted the disease after being fed infected frozen and then defrosted mouse pups.

Cholelithiasis: A 3-yr-old male, captive-bred bearded dragon presented with a history of lethargy, anorexia and decreased fecal production of several days duration. Physical examination revealed a firm, spherical structure palpable within the coelom. A cholelith was surgically removed from the gall bladder (Ritzman and Garner, 2009). Histologically, there was transmural edema with focal acute ulceration in the gall bladder. Lesions in the liver included hepatic fibrosis and bile duct proliferation.

Periodontitis: Agamids, chameleons and tuataras are unique among lizards in that they possess acrodont dentition. Teeth are ankylosed to the crest of the mandibles and maxillae and are not continually replaced as are the teeth of other reptiles. The base of an acrodont tooth is covered only by a thin layer of epithelium instead of gingival tissue; hence, these lizards are prone to periodontal disease particularly when they are fed only soft food such as moistened processed food, mealworms and fruit (McCracken and Birch, 1994). Clinical signs include swelling, erythema and hyperplasia of the periodontal tissue, and accumulation of dental calculus (calcified plaque). Chronicity leads to osteomyelitis. Treatment consists of anesthesia, intraoral radiographs and debridement of periodontal pockets, microbiologic culture, antibiosis and changes in diet (Reavill and Schmidt, 2010).

Retained Eggs: In the author's experience, there is a much lower incidence of egg retention in bearded dragons than in green iguanas. The few cases that the author has seen, have responded well to oxytocin at 20 units/kg IM q12 hr coupled with provision of a suitable nest box.

Atherosclerosis and Pericardial Effusion: Atherosclerosis and pericardial effusion were diagnosed in a 2-yr-old lethargic male bearded dragon based on ultrasound visualization, necropsy and histology (Schilliger et al., 2010). The lizard's diet was totally insectivorous consisting of crickets (*Acheta domestica*), mealworms (*Tenebrio molitor*), kingworms (*Zoophobas morio*) and wax moth larvae (*Galleria mellonella*). Plasma biochemistries revealed mild hyperglycemia and mild hypercholesterolemia. Atherosclerosis is characterized by inflammation of arterial smooth muscle cells and formation of atherosclerotic plaques. This buildup leads to abnormalities of blood flow, and diminished oxygen supply to target organs (Schilliger et al., 2010). In mammals and birds, development of atherosclerosis may be the result

of a high concentration of plasma cholesterol, unbalanced diet, stress, hepatic lipidosis and/or lack of exercise (Schilliger et al., 2010).

Aneurysm: Several cases of aneurysms have been reported in bearded dragons (Barten et al., 2006; Sweet et al., 2009). The aneurysms usually arise either from an internal carotid artery or directly from an aorta. Affected lizards present with a large fluctuant or firm swelling up to 5 cm in diameter on the dorsolateral neck. In one bearded dragon, an aortic aneurysm was located entirely within the chest cavity just cranial to the cardiac apex. Clinical symptoms in the latter case were acute onset of listlessness, weakness, and pallor of the entire integument. The lizard suffered acute death several days after presentation due to rupture of the aneurysm. Histologic examination of the aneurysm revealed focal disruption and ulceration of the intimal layer of the aorta with pockets of lymphocytic inflammation within the disrupted intima (Sweet et al., 2009). Periodic acid–Schiff staining did not reveal any fungal elements. One report described successful removal of an aneurysm from the neck area (Barten et al., 2006). Blood loss during surgery required a transfusion from a donor bearded dragon. The lizard survived for 18 months. Another bearded dragon did not undergo treatment and survived for 8 months after diagnosis. The etiology of this syndrome is unknown. One possibility is that the carotid arteries, due to their shallow position in the dorsolateral pharynx, may be exposed to trauma during feeding. Hypertension and/or genetic predisposition is also possible.

Firefly (*Photinus* spp.) toxicosis: Lucibufagins protect fireflies from potential predators. These compounds are similar to bufotoxins causing gastrointestinal upset, cardiotoxicity and acute death (Glor et al., 1999). One firefly can kill an adult bearded dragon. To date there has been no effective treatment.

NEOPLASIA

A) Nerve Sheath Tumor (Schwanomma): Five subcutaneous masses were successfully removed from two adult bearded dragon clutchmates (Lemberger et al., 2003). Histologic, ultrastructural and immunohistochemical features were consistent with a peripheral nerve sheath origin. At 1 yr post excision, no local reoccurrence was noted; however, one of the individuals still had 3 masses ranging from 3-5 mm diameter on which no additional growth was observed.

B) Malignant Peripheral Nerve Sheath Tumor: An adult bearded dragon with an unresectable axillary mass was euthanized. Numerous metastases were present in the liver, heart, and lungs. Histologically, the mass was identified as a malignant peripheral nerve sheath tumor (Mikaelian, et al., 2001).

C) Metastatic Fibrosarcoma: A 9-yr-old, adult male bearded dragon with a 3 wk history of anorexia, malaise and weight loss had a large, firm, subcutaneous cervical mass on the right side that had been present for 2 months. The mass was identified as a fibrosarcoma with coelomic metastasis (Diaz-Figueroa et al., 2005). Fibrosarcomas commonly develop in subcutaneous tissue and then metastasize via the circulatory and lymphatic systems to visceral structures.

D) Malignant chromatophoroma: A 7-yr-old, male bearded dragon presented for left sided head swelling with increased yellow cutaneous pigmentation and two cutaneous masses on the

right side of the body (Strunk et al., 2009). Biopsies of the masses revealed malignant chromatophoroma. Necropsy revealed metastasis to the spleen, heart and liver.

E) Periocular Squamous Cell Carcinoma: In bearded dragons, this locally invasive malignant tumor is usually a solitary mass that is located adjacent to or behind the orbit (Garner, 2011). Despite its low rate of metastasis, complete excision is difficult to accomplish.

F) Gastric Endocrine Carcinoma: This is a highly malignant neoplastic entity in young bearded dragons (Ritter et al., 2009). Symptoms include anorexia, vomiting, hyperglycemia and anemia. The tumor originates from the pyloric mucosa, penetrates the gastric serosa and subsequently metastasizes to the liver and kidneys. These tumors are immunohistochemically positive for somatostatin suggesting they may be somatostatinomas.

G) Chronic Monocytic Leukemia: A 5-year-old, male bearded dragon presented for lethargy, weight loss and dehydration. Hematology revealed a nonregenerative anemia (12% hematocrit) and a monocytic leukocytosis (91.6 x 10^3 leukocytes /µl; 43 x 10^3 monocytes/µl). Histology revealed infiltration of the liver, small intestine, kidneys and subconjunctival tissues with neoplastic monocytes (Gregory and Latimer, 2004). Characterization of the monocytes was aided by the use of cytochemical and immunohistochemical stains.

REFERENCES

Barten S, Wyneken J, Mader D, Garner M. 2006. Aneurysm in the dorsolateral neck of two bearded dragons (*Pogona vitticeps*). Proc ARAV, 43-44.

Bogoslavsky B. 2007. The use of ponazuril to treat coccidiosis in eight inland bearded dragons (*Pogona vitticeps*). Proc ARAV, 8-9.

Boyer T, Frye F. 2000. Suspected adenoviral hepatitis transmission from juvenile to adult bearded dragons, *Pogona vitticeps*. Proc ARAV, 69-71.

Boyer T, Garner M. 2005. *Emmonsia* fungal stomatitis in a bearded dragon (*Pogona vitticeps*). Proc ARAV, 97-99.

Calvert I. Nutritional Problems. 2004. In Girling SJ, Raiti P (eds): BSAVA Manual of Reptiles. Wiley-Blackwell, Ames, IA, 289-308.

Christie B. 1993. Captive breeding and reproduction of the inland bearded dragon. Captive Breeding Magazine, Snake Bite Inc., Plymouth, MI, 1(2):20-23.

Cranfield M, Graczyk T, and Lodwick L. 1996. Adenovirus in the bearded dragon, *Pogona vitticeps*. Proc ARAV, 131-132.

Crocker C, Miller D, Styer E. 2005. Adenovirus in a bearded dragon with a note on potential disease dissemination issues related to reptiles. Proc ARAV, 87-882.

Davies RR, Klingenberg R. 2004. Therapeutics and Medication. In Girling SJ, Raiti P (eds): BSAVA Manual of Reptiles. Wiley-Blackwell, Ames, IA, 115-130.

De Vosjoli P, Mailloux R. 1996. A simple system for raising juvenile bearded dragons indoors. Vivarium Magazine, American Federation of Herpetoculturists, Escondido, CA, 7(6):42-55.

Diaz-Figueroa O, Patterson K, Mitchell M, Lomax L, David A, Zachariah T. 2005. Metastatic fibrosarcoma in a bearded dragon (*Pogona vitticeps*). Proc ARAV, 79-81.

Garner M. 2011. Clinicopathologic features of select disease entities in the bearded dragon. N. Am. Vet. Conf. Orlando, FL.
Girling S, Fraser M. 2004. *Listeria monocytogenes* septicaemia in an inland bearded dragon, *Pogona vitticeps*. J Herpetol Med Surg, 14(3): 6-9.
Glor R, Means C, Weintraub MJ, Knight M, Glor R, Adler K, Eisner T. 1999. Two cases of firefly toxicosis in bearded dragons, (*Pogona* vitticep). Proc ARAV, 27-30.
Gregory C, Latimer K, Fontenot D, Lamberski N, Raymond JT, Campagnoli RP. 2004. Chronic monocytic leukemia in an inland bearded dragon, *Pogona vitticeps* J Herpetol Med Surg, 14(2): 12-16.
Greiner E, Mader D. 2006. Parasitology. In: Mader, D (ed): Reptile Medicine and Surgery. Saunders Elsevier Co., St. Louis, MO, 343-364.
Heard D, Harr K, Wellehan J. 2004. Diagnostic sampling and laboratory tests. In Girling SJ, Raiti P (eds): BSAVA Manual of Reptiles. Wiley-Blackwell, Ames, IA, 71-86.
Hernandez-Divers S. 2004. Diagnostic and surgical endoscopy. In Girling SJ, Raiti P (eds): BSAVA Manual of Reptiles. Wiley-Blackwell, Ames, IA, 103-114.
International Species Information System. 2002. Physiological Data Reference Values. CD Rom. ISIS. Apple Valley, MN.
Jacobson E, Green E, Undeen A, Cranfield M, and Vaughn K. 1998. Systemic microsporidiosis in inland bearded dragons *(Pogona vitticeps*). J. Zoo Wildl. Med, 29(3): 315-323.
Johnson J. 2004. Urogenital system. In Girling SJ, Raiti P (eds): BSAVA Manual of Reptiles. Wiley-Blackwell, Ames, IA, 261-272.
Klingenberg R. 2004. Parasitology. In Girling SJ, Raiti P (eds): BSAVA Manual of Reptiles. Wiley-Blackwell, Ames, IA, 319-329.
Lemberger K, Manharth A, Pessier A. 2003. Multicentric peripheral nerve sheath tumors in two related bearded dragons (*Pogona vitticeps*). Proc ARAV, 55-56.
Mader D. 2006. Acariasis. In: Mader, DR (ed): Reptile Medicine and Surgery. Saunders Elsevier Co., St. Louis, MO, 720-738.
Mader D. 2006. Gout. In: Mader, D (ed): Reptile Medicine and Surgery. Saunders Elsevier Co., St. Louis, MO, 793-800.
McCracken H, Birch C. 1994. Periodontal disease in lizards: A review of numerous cases. Proc ARAV, 108-114.
Mikaelian I, Levine B, Smith S, Harshbarger J, Wong J. 2001. Malignant peripheral nerve sheath tumor in a bearded dragon, *Pogona vitticeps*. J Herpetol Med Surg, 11(1):9-12.
Miller LA. Biological Electron Microscopy, Frederick Seitz Materials Research Laboratory, Room 125, 104 South Goodwin Ave Urbana, IL 61801 lamiller@illinois.edu
Mitchell M. 2006. Salmonella: Diagnostic methods for reptiles. In: Mader, D (ed): Reptile Medicine and Surgery. Saunders Elsevier Co., St. Louis, MO, 900-905.
Pare J, Sigler L, Rypien K, C Gibas C, and Hoffman T. 2001. Cutaneous fungal microflora of healthy squamate reptiles and prevalence of the *Chrysosporium* anamorph of *Nannizziopsis vriesi*. Proc ARAV, 43-49.
Raftery A. 2004. Clinical examination. In Girling SJ, Raiti P (eds): BSAVA Manual of Reptiles. Wiley-Blackwell, Ames, IA, 51-62.
Raiti P. 2004. Non-invasive imaging. In Girling SJ, Raiti P (eds): BSAVA Manual of Reptiles. Wiley-Blackwell, Ames, IA, 87-102.

Reavill D, Schmidt R. 2009. Review: Pathology of the reptile integument. Proc ARAV, 93-106.
Reavill D, Schmidt R. 2009. A retrospective review of the diseases in family Agamidae (agamas, bearded dragons, frilled dragons, water dragons). Proc ARAV, 111-116.
Reavill D, Schmidt R. 2010. Mycobacterial infections in reptiles. Proc ARAV, 16.
Ritter J, Garner M, Chilton J, Jacobson E, Kiupel M. 2009. Gastric neuroendocrine carcinomas in bearded dragons (*Pogona vitticeps*). Vet. Pathol, 46: 1109-1116.
Ritzman T, Garner M. 2009. Cholelithiasis and surgical cholelith removal in a bearded dragon (*Pogona vitticeps*). Proc ARAV, 117.
Rogner M. 1997. Lizards Volume 1, Krieger Publishing Co., Malabar, FL, 119-170.
Schilliger L, Lemberger K, Chai N, Bourgeois A, Charpentier M. 2010. First case of atherosclerosis associated with pericardial effusion in a bearded dragon (*Pogona vitticeps*). Proc ARAV, 70-73.
Strimple P, Strimple J. 1998. Bearded dragons: A beginner's guide to captive husbandry and reproduction. Reptiles USA Annual Magazine, Fancy Publications Inc., Mission Viejo, CA, 28-45.
Strunk A, Haney S, Reavill D. 2009. Malignant chromatophoroma in a bearded dragon (*Pogona vitticeps*). Proc ARAV, 73-75.
Sweet C, Linnetz E, Golden E, Mayer J. 2009. What Is Your Diagnosis? JAVMA, 234(10):1259-1260.
Van Waeyenberghe L, Baert K, Pasmans F, van Rooij P, Hellebuyck T, Beernaert L, de Backer P, Haesebrouck F, Martel A. 2010. Voriconazole, a safe alternative for treating infections caused by the *Chrysosporium* anamorph of *Nannizziopsis vriesii* in bearded dragons (*Pogona vitticeps*). Med Mycol, 48(6):880-885.
Wagner R, Dahlhausen R, Klein E. 2007. Detection of adenovirus in bearded dragons with PCR. Proc ARAV, 19-22.
Weis P. 2001. The bearded dragon: Australia's goodwill ambassador. Reptiles USA Magazine, Fancy Publications Inc., Mission Viejo, CA, 11-16.

Table 1. International Species Information System Reference Ranges for Physiologic Data Values: Inland bearded dragon, *Pogona vitticeps.* Physiologic reference ranges calculated for both sexes combined, all ages combined. Conventional U.S.A. Units. Sample results submitted by 24 member institutions. © I.S.I.S. - March 2002.

Test	Units	Mean	St. Dev.	Minimum Value	Maximum Value	Sample Size[a]	Animals[b]
White blood cell count	*10^3/µl	9.069	5.425	2.200	29.70	57	41
Red blood cell count	*10^6/µl	1.05	0.19	0.73	1.55	29	21
Hemoglobin	g/dl	10.1	1.5	8.0	13.0	21	16
Hematocrit	%	30.0	5.8	18.5	41.5	72	53
MCV	fL	297.2	45.6	228.6	373.7	28	20
MCH	pg/cell	104.1	19.5	72.7	142.5	17	12
MCHC	g/dl	35.4	8.0	25.0	59.1	21	16
Nucleated red blood cells	/100 WBC	0	0	0	0	2	2
Heterophils	*10^3/µl	3.070	2.132	0.350	9.220	57	41
Lymphocytes	*10^3/µl	4.612	3.601	0.240	17.00	57	41
Monocytes	*10^3/µl	0.780	1.114	0.050	5.940	36	29
Eosinophils	*10^3/µl	0.356	0.412	0.052	1.440	11	9
Basophils	*10^3/µl	0.481	0.457	0.032	2.509	48	37
Azurophils	*10^3/µl	0.573	0.598	0.000	2.312	30	22
Calcium	mg/dl	16.5	11.5	7.1	56.0	58	42
Phosphorus	mg/dl	5.5	2.4	2.7	15.1	52	38
Sodium	mEq/L	157	12	137	186	37	29
Potassium	mEq/L	3.7	1.1	1.3	6.3	38	30
Chloride	mEq/L	127	16	104	163	21	17
Bicarbonate	mEq/L	24.0	2.8	22.0	26.0	2	2
Carbon dioxide	mEq/L	16.7	3.6	12.7	20.0	7	7
Osmolarity	mOsmol/L	297	0	297	297	1	1
Blood urea nitrogen	mg/dl	2	1	0	4	21	18
Creatinine	mg/dl	0.2	0.2	0.0	0.6	12	10
Uric acid	mg/dl	4.4	2.6	0.0	11.5	61	47
Total bilirubin	mg/dl	0.5	0.9	0.0	3.7	18	18
Direct bilirubin	mg/dl	0.0	0.0	0.0	0.0	4	4
Indirect bilirubin	mg/dl	0.0	0.0	0.0	0.0	4	4
Glucose	mg/dl	205	53	118	416	51	38
Cholesterol	mg/dl	436	199	160	900	24	20
Triglyceride	mg/dl	310	172	93	459	4	4
Creatine phosphokinase	IU/L	1159	1664	10	7000	37	26
Lactate dehydrogenase	IU/L	300	196	35	628	19	14
Alkaline phosphatase	IU/L	158	132	15	447	35	30
Alanine aminotransferase	IU/L	10	5	1	20	17	16
Aspartate aminotransferase	IU/L	27	25	0	92	46	37
Gamma glutamyltransferase	IU/L	18	29	0	81	8	7
Amylase	U/L	823	461	497	1149	2	2
Total protein (colorimetry)	g/dl	5.1	1.4	3.1	8.5	40	33
Globulin (colorimetry)	g/dl	2.2	0.9	1.0	4.4	25	22
Albumin (colorimetry)	g/dl	2.6	0.8	1.3	4.6	34	26
Fibrinogen	mg/dl	180	110	0	300	5	5

[a]Number of samples used to calculate the reference range.
[b]Number of different individuals contributing to the reference values.
Source: International Species Information System 12101 Johnny Cake Ridge Road, Apple Valley, MN 55124, USA. www.isis.org