Common Reptilian Diseases

A.M. Timmerman, B.A., D.V.M.*
M. D. Doolen, D.V.M.**

Veterinarians are seeing more and more reptiles as they become increasingly popular pets. When taken from their natural habitat reptiles may succumb to disease that they may not otherwise be susceptible to. Disease may follow stress caused by changes in environment, poor husbandry, improper temperature, transportation, improper nutrition, or trauma. The intent of this paper is to familiarize veterinarians with three of the more common diseases seen in reptiles: pneumonia in snakes, metabolic bone disease in iguanas, and hypovitaminosis A in turtles.

Serpentine Pneumonia

The Burmese python *(Python molurus bivittatus)* and Boa Constrictor *(Boa constrictor)* are the most commonly seen snakes. Therefore, the following information is applicable to these members of the boid family. Snakes, as well as other reptiles, lack a diaphragm. Without a diaphragm snakes cannot cough, but they can hold their head at an angle so as to facilitate the drainage of sputum or mucous secretions.[1,2] Most snakes have only a single lung which is sac-like and highly reticulated. The trachea and main stem bronchi enter the hilus of the lungs more cranially than many other vertebrates and ciliated pseudostratified columnar epithelium lines only the larger airways.[1]

The incidence of pneumonia increases when a snake experiences stressors such as: overcrowding, excessive handling, inappropriate temperatures and humidity, inadequate nutrition, transportation, and parasites. These may allow bacterial overgrowth and subsequent disease. Opportunistic gram negative bacteria such as *Pseudomonas* and *Aeromonas* are the most common isolates in reptilian pneumonia.

* A .M. Timmerman is a 1994 graduate of the College of Veterinary Medicine, Iowa State University.

** M. D. Doolen is a 1991 graduate of the College of Veterinary Medicine, Iowa State University. He is currently practicing in New Jersey.

Pseudomonas is frequently found in healthy and sick reptiles. Others are: *Edwardsiella, Klebsiella, Pasteurella, Proteus, Staphylococcus, Streptococcus, Enterobacter, Citrobacter, Acinetobacter, Serratia,* and *Flavobacter.* [1,3] Marcus indicates that snake mites may be the primary transmitter of infectious pneumonia.[4]

The clinical signs of pneumonia are usually quite distinct in snakes. These signs include: gasping, wheezing, nasal and/or ocular mucous discharge (that may range from serous to thick, tenacious and yellow), open-mouth breathing, plugged internal and external nares, a raw rostrum from rubbing it on the cage or its body, anorexia, weight loss, and listlessness.[1,5,6,7,8] Snakes may also assume a posture that allows it to breathe easier. The head may be elevated by resting it on some object in the cage and the mouth may be open. This helps drain the mucus from the nares and trachea.

Other related diseases that may be seen in conjunction with pneumonia are: ulcerative stomatitis and necrotizing dermatitis ("scale rot, vesicular dermatitis, blister disease"). Often an organism causing a septicemia is the same causative agent of the pneumonia. [1,7,9,10]

Diagnosing pneumonia in snakes can be achieved by: noting clinical signs (listed above), performing a microscopic evaluation of the sputum looking for parasitic ova of lungworms, culture and sensitivity of a sample from the choana or trachea, radiology, and hematology. A post-mortem exam may be performed to confirm a diagnosis if the animal dies or is euthanatiized. Differential diagnoses should include ulcerative stomatitis, lungworm or pentastome infection, hypovitaminosis A, fungal infection, or a paramyxovirus infection. [7]

Treatment of pneumonia should include antibiotics that are chosen based on culture and sensitivity. Some antibiotics used for reptilian pneumonia are listed in Table 1. Treatment should also include: 1) supplemental oxygen, 2) increased ambient temperature (to 98.6°F (37°C) or above), 3) nebulization with Amikacin: 25% solution of 50 mg/ml in saline, 4) fluid

therapy 50 mg/kg of LRS, intracoelomic, 5) vitamin supplement Injacom^{R}-100^{a} (Vitamin A - 100,000 I.U./ml, Vitamin D_3 - 10,000 I.U./ml, Vitamin E - 20 I.U./ml), 0.2 cc/kg IM or SQ once weekly, 6) restricted handling, 7) diuretics - the use of diuretics is controversial. The amount of dehydration and concurrent use of aminoglycoside antibiotics should be taken into consideration. Frye believes it to be helpful in drying edematous membranes while Ross believes it exacerbates the nephro-, oto-, and neurotoxic effects of aminoglycosides and should never be used to treat pneumonia.[1,13]

An optimal environment for a boid consists of appropriate temperature, housing, water, humidity, photoperiod, and nutrition.[14] Proper environment should include a cage that is large enough for the expected growth of the snake. A seclusion box should be offered for privacy, a dish large enough to soak in, and clean substrate such as newspaper, butcher paper, astroturf or indoor/outdoor carpet that won't harbor bacteria and can be easily cleaned. The temperature should be maintained around 85°F (29.4°C) for boids. At night the temperature should be lowered to no less than 75°F to simulate diurnal temperatures. Other species of snakes may require higher or lower maintenance temperatures. Sources of heat may come from heating tapes, cables, heating pads, or heat lamps. It is best to maintain a temperature gradient in the cage so the snake can move to where it feels most comfortable.[14,15] Humidity should be maintained around 85%. A photoperiod of 9-16 hours/day will fairly closely resemble that of the winter-summer season which boids are subjected to in the wild. Leaving a light on all the time is stressful to snakes. Adequate nutrition can include small rodents and birds, frogs, and lizards.[16] These supply the snake with the appropriate dietary vitamins and minerals. Also, proper cage cleaning can't be stressed enough. Cleaning will help keep the bacterial population down.

Reptilian pneumonia is a common occurrence and is observed most frequently when snakes have been transported and/or subjected to prolonged periods of sub-optimal temperatures. Antibiotics and intensive nursing care is needed as well as the correction of the snake's living environment. With appropriate care these snakes can live for many years.

Metabolic Bone Diseases in Iguanas

The common green iguana (*Iguana iguana)* is also becoming a very popular pet. Metabolic bone disease (MBD) is by far the most prevalent problem in iguanas kept as pets. Other names used are fibrous osteodystrophy and nutritional secondary hyperparathyroidism. It may be seen in any iguana at any age but young growing animals are the most commonly affected. [17] The two factors which predispose iguanas to metabolic bone disease are improper calcium to phosphorus ratios in the diet and lack of Vitamin D_3 due to inadequate UV exposure.[18]

Iguanas need calcium and vitamin D_3 which they would normally obtain from plants and UV light (ie. sunlight). Ideally, a calcium to phosphorus ratio in the diet should be approximately 1.2:1. Frequently pet owners feed only one type of plant material. Often the phosphorus far outweighs calcium in the diet. For example, iceberg lettuce is very low in calcium and should not be fed or only fed in very small amounts. Some plants such as rhubarb leaves contain oxalic acid which binds calcium. Spinach and broccoli also concentrate oxalic acid to a small degree but are high in calcium. They should be fed in moderation. [19,20]

Metabolic bone disease occurs as the calcium:phosphorus ratio becomes unbalanced in the body. As blood calcium decreases, the parathyroid gland releases parathormone (PTH) to conserve calcium concentration in the blood by causing resorption of calcium from bones. Over time the bones become demineralized and the cortical bone becomes thickened and spongy. Fibrous connective tissue is laid down to add stability. This gives the legs a thickened, muscular appearance. At this point bones are extremely prone to pathologic greenstick and folding fractures and these animals should be handled very carefully. [16,13]

Clinical signs of metabolic bone disease vary in severity. Burgmann describes the progression of signs in the following way:

"Symptoms may range from mild signs of weakness, poor digestion, slow growth, problems with reproduction, to neurological signs such as tetany, tremors, and convulsions. Paralysis due to fractured vertebrae, skeletal deformities such as bowed or swollen legs, irregular fibrous swellings of the mandibles, soft jaws, loose teeth, fractures of the long bones,

and stunted growth are common in prolonged or severe calcium deficiency."[1,21]

The mandible and maxilla will become rubbery and deformed and will bend if gentle pressure is applied from both sides. The iguana may not even be able to move or get up because it can't support its own body weight.

Diagnosis of metabolic bone disease is by diet history, physical exam findings, serum calcium levels, and radiographs. Radiographs may show lack of bone density and pathologic fractures (ie. greenstick or non-unionfractures). Radiographs may not be diagnostic if the disease is in an early stage or if the radiographic technique is poor. [19,23] A more complete evaluation may include: cbc, phosphorus, AST(SGOT), alkaline phosphatase, uric acid, bile acid, albumen, glucose, and a fecal examination for intestinal parasites. [21]

Differential diagnoses for MBD includes rickets or renal-associated fibrous osteodystrophy. Rickets can be closely associated with the osteomalacia seen in MBD. An iguana with rickets may have a normal calcium level but is deficient in vitamin D_3. The cartilage in these animals will overgrow or hypertrophy and become hyperplastic thus leading to poor bone mineralization. Renal-associated fibrous osteodystrophy also looks similar to MBD clinically but the underlying pathophysiology is different in that the loss of calcium is caused by a diseased kidney. Because the kidney is damaged, phosphorus is retained and increases in the blood. This triggers the parathyroid gland to secrete more parathormone and cause calcium resorption.[16]

One of the newer methods of treatment is calcitonin. Calcitonin is produced by the parafollicular cells of the thyroid gland in response to increased calcium or phosphorus levels in the blood. It acts to decrease the rate of bone resorption, pulls calcium and phosphate out of the blood, decreases osteoclastic activity, stimulates osteoblastic activity, and causes increased excretion of calcium in the urine.[18] Calcitonin can be given at:

1.5 IU/kg, SQ, three times daily[16] or
50 IU/kg, IM, weekly for two treatments.[21,25]

Before injecting calcitonin, it is important to ensure the iguana is normocalcemic, otherwise this treatment may cause a hypocalcemic tetany.[22] According to Frye, concurrent diuresis with physiologic saline or Ringer's solution should be given at:

10 ml/kg, IV or intracoelomic, once daily to prevent a rebound hypercalcemia.[16]

Also of primary importance in the treatment of MBD is parenteral calcium gluconate:

A 10% solution may be given intraperitoneally at 500 mg/kg.

This can be given weekly for two to six weeks. The injection may be given in the caudal abdomen, just in front of the femur about 1/3 the distance ventrally from the dorsal midline. The iguana is less likely to struggle when lying on its ventrum than if the injection is given into its ventral abdomen.[21,26] An oral calcium and vitamin D supplement should be added to the food twice daily or until signs resolve:

Oral: .25 tablet/5 lbs, Pet-Cal[R] [b], crumbled and mixed with food. once daily. Or a small pinch of vitamin/mineral supplement can be given to juveniles once daily. Older iguanas (>2 1/2 yrs) should be given one full pinch per 2 lbs two times a week or 1/8 teaspoon per 3-4 lbs once a week.

Parenteral: 100 IU vitamin D_3/kg, SQ, every 7 days.[22,27]

Additional therapy for metabolic bone disease consists of correcting the diet and educating the client about proper iguana maintenance. It is important to handle these iguanas very carefully during the recovery process. Fractures should be splinted if possible.

Iguanas are generally herbivorous and will thrive on many types of plants. In their natural habitat iguanas will eat "leaves, fruit and flowers of select trees, vines and bushes in short frequent feeding bursts concentrated from late morning to early afternoon."[23] Table 2 shows the calcium to phosphorus ratio of some vegetables and fruits. Other foods high in calcium are: swiss chard, kale, beet greens, escarole, parsley, watercress, green beans, turnip greens, broccoli, tofu, figs and flowers such as roses, nasturtiums, carnations, hibiscus, rabbit pellets, leafy alfalfa hay, mustard greens, carrot tops, dandelion greens, and frozen mixed vegetables. [21,24] Treats such as crickets and mealworms can be given so long as it makes up only a small amount of the diet. A vitamin D_3 and calcium supplement should be added to the food on a regular basis if MBD is present and during diet conversion when the young iguana is learning to eat a variety of vegetables. It is important to note here that oversupplementation

can be just as devastating as the disease it was intended to prevent. Excess calcium in conjunction with vitamin D_3 (or UV radiation) may lead to a nephrotic syndrome and excess urate formation and deposition. Excess calcium may also cause other mineral imbalances. Hypervitaminosis D_3 will cause calcium resorption from bone and resulting calcification of soft tissues. [21]

Correct husbandry will help prevent disease. Iguanas, being arboreal, need a large enclosure with plenty of room to climb. Temperatures need to be kept no lower than 75°F (23.8°C) at night and around 90°F (32.2°C) during the day. A spot for basking should be available and should be between 95°F (35.0°C) and 100°F (37.7°C). A temperature gradient is needed in the cage so the iguana can go to where it feels most comfortable. Heat sources can consist of those mentioned for snakes but can also include using a space heater to keep the room between 75°F and 90°F or using 60-100 watt incandescent light bulbs. Heat sources that are kept outside the cage are generally much safer.[24]

An ultraviolet light which emits radiation in the 290 to 320 nanometer wavelength is needed to catalyze the production of cholecalciferol (vitamin D_3). The light should be kept two feet from the iguana. Make sure there is no glass or plastic between the animal and the UV light as it will filter out the needed wavelength of light. Because natural light is best, get the iguana outside if possible. A source of water should always be available. [22,24]

Iguanas with metabolic bone disease should be rechecked every four to eight weeks. At these times they should be re-radiographed to check for healing fractures and have blood calcium levels reevaluated. Other tests which may be run might include phosphorus, cbc, and uric acid levels. Because metabolic bone disease is preventable, client education can never be stressed enough. All clients should be aware of proper environmental conditions (space, temperature, water, climbing sources), nutrition (plants, fruits, vegetables, correctly-balanced vitamins and minerals), and how to detect symptoms of disease before it's too late.

Hypovitaminosis A in Box Turtles

A third common reptilian disease is hypovitaminosis A in the Box Turtle (*Terrapin spp)* . Turtles have very specific dietary needs. Hypovitaminosis A is a preventable disease. Good client education is imperative. Though there are many species of turtles kept as pets this section will focus on box turtles as they are the most common.

Hypovitaminosis A can be seen in many species and ages of turtles, but it may more severely affect the juvenile turtle. A diet lacking in vitamin A (ie. an all hamburger diet) is implicated. Turtles suffering from clinical signs usually show hyperkeratosis, puffiness of the eyes, and respiratory signs. Anasarca and squamous metaplasia of the urinary tract may be seen on post mortem exams. Multiple organs can be affected as the disease progresses.[16,20,26] The integument becomes thick and irregular and the eyes become swollen due to the keratinization of the ophthalmic (Harderian and lacrimal) glands. These are nasally and temporally located in the orbit of the eye, respectively. When vitamin A is lacking these glands will undergo a squamous metaplasia and keratinization that cause the eyes to bulge out. Blepharitis is a common sequela and one might see conjunctivitis or a mucopurulent discharge.[23] Respiratory signs such as wheezing, open mouth breathing, and nasal discharge may be due to the proliferation and accumulation of keratinous debris on mucous and glandular tissues throughout the oropharynx and lower airways.[16,20] This can lead to secondary bacterial infections, pneumonia, and middle ear infections. Normally the tympanic membranes have a concave shape. A middle ear infection can be detected by noticing a bulging out of the tympanic membranes. Other signs can include beak and toenail overgrowth sometimes to the point where the turtle can no longer prehend food.[29]

History of a diet that is poor in vitamin A and clinical signs of hyperkeratosis, puffy eyes, and renal problems are probably the most relevant diagnostic tools. Obtaining a thorough dietary history is important and can help narrow the differentials. Besides doing a visual exam, one can listen for respiratory abnormalities by placing a wet towel over the carapace (shell) and putting the stethoscope on top of the towel.

Differentials for hypovitaminosis A include: trauma or foreign bodies causing conjunctivitis, metabolic disturbances, parasitic infestation, and bacterial, viral, or mycotic infections.[26] A history of adequate vitamin A intake might lead one to take a closer look at other differentials.

Therapy consists of immediate vitamin A supplementation, correcting the diet, supportive therapy, and improvement of the housing if needed. Oral and/or injectable vitamin A can be utilized:

Oral (loading dose): 0.1cc/300 gm Box urtle, Injacom^{R}-100.[a] Then give 0.02cc/300 gm Box turtle/week for 2-3 more treatments.

OR

Parenteral: 2,000 IU/kg Aquasol A^{R} parenteral, SQ, in a front leg, twice weekly for 4-6 weeks.[26]

A multivitamin A, D_3, and E with calcium supplementation should be used at:

0.2 ml/kg, Injacom^{R}-100[a] (Vitamin A - 100,000 I.U./ml, Vitamin D_3 - 10,000 I.U./ml, Vitamin E - I.U./ml), IM or SQ at weekly intervals.[30]

It must be stressed that too much vitamin A can also create severe problems. Hypervitaminosis A can cause dry, flaky skin to severe skin lesions. Frye incriminates injectable vitamins as a source of skin loss.[16]

Supportive therapy for turtles may include ophthalmic antibiotic ointments if secondary bacterial infection is present. Gently remove any excess cellular debris around the eyes. Soak the turtle in shallow, warm (75-80°F (24-27°C)) water for 1-2 hours daily. [26]

Box turtles are omnivores. They should be fed a wide variety of foods such as: "land snails, beetles, sowbugs, millipedes, slugs, earthworms, spiders, carrion, small mammals, birds, crayfish, frogs, salamanders, lizards, snakes, smaller turtles and plant material such as mushrooms, strawberries, raspberries, mulberries, and tomatoes."[31] Other box turtles, such as the ornate box turtle *(Terrapene ornata ornata),* prefer diets mainly consisting of insects. Young turtles are mostly carnivorous. At least two-thirds of the diet should be animal-based. Most of the same fruits and vegetables listed for iguanas are also suitable for box turtles. There are several commercial turtle feeds listed by Frye that can also be used.[16,31]

Proper housing for box turtles is different from many other types of turtles. Box turtles are semi-aquatic and need access to water daily. Because box turtles cannot swim, the water bowl should be shallow enough for the turtle to get in and out. A large cage is preferable (at least a 20-gallon tank for a box turtle) and should be lined with newspaper, astroturf, medium woodchips, or other suitable material that won't harbor bacteria. Their environment can be set up much like that for snakes and iguanas. The daily ambient temperature should range from 70-80°F. Turtles need 12 to 14 hours of light per day. Frequent cleaning of the cage is of utmost importance especially the water dish. Box turtles will defecate in their water.[31]

Salmonella spp. are commonly isolated from the intestines of sick as well as healthy turtles. Young children should not be allowed to handle turtles as they may put the turtle in their mouth or stick their hands in their mouths after handling one. Children who are old enough should be taught to wash their hands after handling a turtle. This goes for adults as well. Also of concern is keeping the family dog away from the turtle. Turtles are fairly durable and can live a very long time with appropriate care.

The intention of this article is to give veterinarians a brief overview of some of the more common diseases seen in snakes, iguanas, and box turtles. It is important to be familiar with the proper husbandry and diet needed by each of these species. These diseases are easily preventable and good client education is the first step to better reptilian health.

Footnotes

a)Injacom^{R} - 100, Hoffmann-LaRoche, Nutley, NJ 07110.

b)Pet-Cal^{TM}, SmithKline-Beecham, Exton, PA 19341.

c)Aquasol A^{R} parenteral, Armour Pharmaceutical Co., Kankakee, IL 60901.

Table 1.

Amikacin sulfate - 2.5 mg/kg SQ or IM q. 72 hrs for 5-7 treatments. May be used with a beta-lactam antibiotic such as ampicillin, carbenicillin, piperacillin or cefotoxime.[11,12*]

Carbenicillin - 400 mg/kg IM for the first dose, then q 24 hrs.[11,12]

Ceftazidime - 20 mg/kg IM q 72 hrs.[12]

Chloramphenicol (in sodium succinate) - 20 mg/kg q 12 hrs IM or SQ for 2-3 weeks or 40 mg/kg SQ q 24 hrs.[11]

Gentamicin -2.5 mg/kg IM q 72 hrs.[12]

Piperacillin - 20-40 mg/kg IM q 12 hrs or 100 mg/kg, once daily. [11,25]

Tobramycin - 2.5 mg/kg IM q 12 hrs.[11]

*The 2.5 mg/kg dose of Amikacin is used when the beta-lactam antibiotics are used with it. The two drugs will potentiate each other so lower doses of each should be used.

Table 2. **Calcium and phosphorus contents of some vegetables and fruits.**[20]

	Calcium mg/100g	Phosphorus mg/100g	Ca:P ratio
Lettuce	25.9	30.2	0.86:1
Tomato	13.3	21.3	0.62:1
Cucumber	22.8	24.1	0.95:1
Broccoli tops	160	54.0	2.96:1
Cauliflower (boiled)	23.0	33.0	0.69:1
Carrots (boiled	36.9	16.7	2.21:1
Watercress	222	52.0	4.27:1
Raisins (dried)	60.6	32.8	1.85:1
Oranges	41.3	23.7	1.74:1
Dates	67.9	63.8	1.06:1
Melon (yellow)	13.8	8.7	1.59:1
Grapes (white)	19.1	21.9	0.87:1
Grapes (black)	4.2	16.1	0.26:1
Cherries	15.9	16.8	0.95:1
Dried Apricots	92.4	118	0.78:1
Pears	6.9	9.5	0.72:1
Apple	3.6	8.5	0.42:1
Banana	6.8	28.1	0.24:1

References

1. Frye FL. Infectious Diseases. In: Frye FL ed. *Reptile Care: An Atlas of Diseases and Treatment*. 1st ed. Vol 1. New Jersey: T.H.F. Publications, Inc., 1991; 120-124.

2. Davies PMC. Anatomy and Physiology. In: Cooper JE, Jackson OF, eds. *Diseases of Reptilia.* Vol 1. New York: Academic Press, 1981; 34-39.

3. Ross RA. The Epidemiology of Gram Negative Bacteria in Reptiles. In: Ross RA, ed. *The Bacterial Diseases in Reptiles.* California: Institute for Herpetological Research, 1984; 8-9.

4. Marcus LC. *Veterinary Biology and Medicine of Captive Amphibians and Reptiles.* Philadelphia: Lea and Febiger, 1981; 57,89.

5. Ross RA. The Epidemiology of Gram Negative Bacteria in Reptiles. In: Ross RA, ed. *The Bacterial Diseases in Reptiles.* California: Institute for Herpetological Research, 1984; 47-49.

6. Mattison C. Diseases. In: Mattison C, ed. *The Care of Reptiles and Amphibians in Captivity.* Poole, Dorset: Blandford Press, 1982; 86

7. Carpenter JW, Wilson SC. *Parasitic and Infectious Diseases of Reptiles.* 3rd Midwestern Exotic Animal Medicine Conference. 1993; 7-32.

8. Marcus LC. *Veterinary Biology and Medicine of Captive Amphibians and Reptiles.* Philadelphia: Lea and Febiger, 1981; 57,89.

9. Ross RA. The Epidemiology of Gram Negative Bacteria in Reptiles. In: Ross RA, ed. *The Bacterial Diseases in Reptiles.* California: Institute for Herpetological Research, 1984; 52-53.

10. Cooper JE. Bacteria. In: Cooper JE, Jackson OF, eds. *Diseases of Reptilia.* Vol 1. New York: Academic Press, 1981; 179.

11. Jenkins JR: *Drugs and Dosages for Reptiles and Amphibians.* 3rd Midwestern Exotic Animal Medicine Conference. 1993; 3-5.

12. Mader DR. Antibiotic Therapy. In: Frye FL, ed. *Reptile Care: An Atlas of Diseases and Treatments*. 1st ed. Vol 2. New Publications, Inc., 1991; 621-633.

13 Ross RA. The Epidemiology of Gram Negative Bacteria in Reptiles. In: Ross RA, ed. *The Bacterial Diseases in Reptiles.* California: Institute for Herpetological Research, 1984; 64-71.

14. Frye FL. Captive Husbandry. In: Frye FL ed. *Reptile Care: An Atlas of Diseases and Treatments*. 1st ed. Vol 1. New Jersey: T.F.H. Publications, Inc., 1991; 64-71.

15. Eggleston DW. Boas and Pythons: The Big Guys. *Reptiles*. 1994; 1: 36-51.

16. Frye FL. Nutrition: A Practical Guide for Feeding Captive Reptiles. In: Frye FL, ed. *Reptile Care: An Atlas of Diseases and Treatment. 1st ed.* Vol 1. New Jersey: T.F.H. Publications, Inc., 1991; 48-49.

17. Boyer TH. Common Problems and Treatment of Green Iguanas (Iquana). *Bull Assoc Amphib and Rep Vet*. 1991; 1: 68-71.

18. Wissman M, Parsons B. Metabolic Bone Disease in Green Iguanas. *Reptiles*. 1994; 1: 68-71.

19. Jackson OF, Cooper JE. Nutritional Diseases. In: Cooper JE, Jackson OF, eds. *Diseases of Reptilia.* 1st ed. Vol 2. New York: Academic Press, 1981; 422-425.

20. Frye FL. Common Pathologic Lesions and Disease Processes. In: Frye FL, ed. *Reptile Care: An Atlas of Diseases and Treatments*. 1st ed. Vol 2. New Jersey: T.F.H. Publications, Inc., 1991; 529-617.

21. Burgmann PM, McFarlen J, Thiesenhausen K. Causes of hypocalcemia and metabolic bone disease in Iguana. *J Sm Exotic An Med*. 1993; 2: 63-68.

22. Mader DR. Use of Calcitonin in Green Iguanas, Iquana, with Metabolic Bone Disease. *Bull Assoc Amphib Rep Vet*. 1993; 3: 5.

23. Boyer TH. Green Iguana Care. *Bull Assoc Amphib Rep Vet*. 1991; 1: 12-14.

24. Jenkins JR. The Green Iguana. *J Sm Exotic An Med.* Vol 1. 1992; 138.

25. Doolen MD. Personal communication.

260 Boyer TH. Common Problems of Box Turtles (Terrapene spp) in Captivity. *Bull Assoc Amphib Rep Vet.* 1992; 2: 9-14.

27. de Vosjoli P. *The Green Iguana Manual.* California: Advanced Vivarium Systems, 1992; 21-22.

28. Marcus LC. *Veterinary Biology and Medicine of Captive Amphibians and Reptiles.* Philadelphia: Lea and Febiger, 1981; 42,44,176

29. Breitweiser BA. Hyperkeratosis and Vitamin A Deficiency in Terrapins. *Captive Breeding.* 1993; 1: 28-30.

30. Holt PE. Drugs and Dosages. In: Cooper JE, Jackson OF, eds. *Diseases of Reptilia.* Vol 2. New York; Academic Press, 1981; 570.

31. Boyer TH. Box Turtle Care. *Bull Assoc Amphib Rep Vet.* 1992; 2: 14-17.

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"Chester" by Nancy McGuire