

**Animal Emergency Center
Memorandum**

To: Referring Veterinary Hospitals

From: Rebecca Kirby, DVM

Date: March 12, 2007

Re: CE Fax Series – Renal Failure in Reptiles

I am pleased to present you with the next installment in the CE fax series provided by the specialists at the Animal Emergency Center. Dr. Paul Gibbons, specialist in avian medicine, had medical/surgical residency training in avian, reptiles, and small mammals. He has a particular interest in reptiles. He is providing you with a two part series on Renal Failure in Reptiles. Part 1 is attached, explaining the anatomical differences of the reptile renal system and pathophysiology of renal failure. Part 2 will follow next week. Please feel free to call Dr. Gibbons should you have any questions or comments regarding the CE material attached, or with any questions you may have about your avian, reptile or small mammal patients.

As always, we welcome your input and feedback regarding the CE Fax series or any other aspect of services provided to you and your clients by the staff of the Animal Emergency Center. Please contact Ms. Christina Matthews at 414-540-6710. This and other CE fax series articles can be found and downloaded from our website at www.animalemergencycenter.com.

Becky Kirby



RENAL FAILURE IN REPTILES

Part 1 of 2

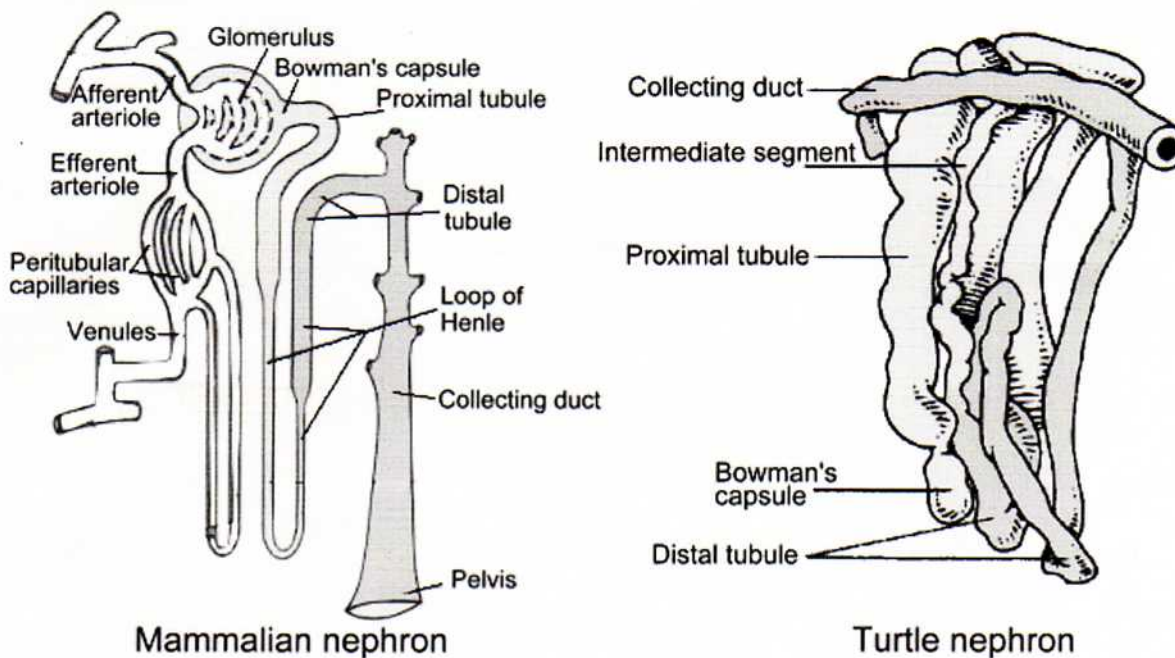
Paul M. Gibbons DVM, MS, DABVP (Avian)

Animal Emergency Center

Renal failure is very common in young to middle-aged lizards, turtles, and tortoises. It is usually a result of inappropriate environmental conditions or nutrition.

BACKGROUND

Reptile kidneys have some major similarities and some major differences when compared to mammalian kidneys. Important similarities in blood supply include afferent arterioles, glomeruli, efferent arterioles, peritubular capillaries, and renal veins. Important anatomical similarities in the tubular system include the renal (Bowman's) capsule, proximal convoluted tubule, distal tubule, collecting duct, and ureter. Reptiles lack the loop of Henle and instead, have a short intermediate segment. In addition, reptiles have a renal portal system that mixes venous blood from caudal parts of the body into that of the efferent arterioles at the start of the peritubular capillaries. A reptile kidney contains a few thousand nephrons, whereas a human kidney has about one million.



Several important physiological differences between mammalian and reptile kidneys also exist. Reptile nephrons cannot increase the solute concentration of urine above that of plasma because they do not have a loop of Henle. Urea (BUN) is the major end product of mammalian protein metabolism. In contrast, reptiles form urea, uric acid, and/or ammonia in varying quantities depending upon their ecological habitat type. Desert reptiles form mostly uric acid. Aquatic

reptiles form ammonia and urea in proportions that vary among species. Urea is formed in very small quantities by reptiles that live on land. Urea and ammonia are highly water-soluble and need a lot of water for excretion. Uric acid is poorly soluble in water, is suspended in renal tubular urine, and requires relatively little water for excretion. Uric acid is formed in the liver, is freely filtered by glomeruli, and is actively secreted from blood into proximal tubule cells. It then passively diffuses down the concentration gradient from inside tubule cells into the proximal tubule lumen.

Reptiles reabsorb filtered water not only in the renal tubules, but also in the colon, cloaca, and, when present, the bladder. Like mammals, reptile renal tubules contribute to regulation of sodium, potassium, hydrogen ions, calcium, and phosphorus. Unlike mammals, the reptile urinary bladder plays a major role in regulation of solutes, and many reptiles also utilize nasal salt glands. Reptile kidneys also synthesize vitamin C, convert vitamin D to its active form, and produce erythropoietin.

PATHOPHYSIOLOGY AND ETIOLOGY

Glomerular filtration rate (GFR) decreases with dehydration or salt excess and increases with water load. In mammals, decreased GFR leads to cessation of blood flow to renal tubules, which places tubule cells at risk of ischemic necrosis. In reptiles, renal portal blood continues to perfuse the peritubular capillaries, which can keep the cells alive during episodes of dehydration.

Uric acid is viscous and requires hydrostatic pressure to move from the proximal tubules to the collecting ducts. Water deprivation, salt excess, or chronic low environmental humidity can cause decreased GFR. Over time, this can lead to build up of uric acid within the lumen of renal tubules. Increased intraluminal concentration prevents urate from diffusing out of renal tubule cells. At high concentrations inside tubule cells, uric acid will crystallize and can form gout tophi that may expand into the tubule lumen and surrounding parenchyma. Renal gout causes severe nephritis, tubule cell necrosis, peritubular cell necrosis, and can lead to renal fibrosis. This can result in hyperphosphatemia, decreased vitamin C production, decreased vitamin D conversion, decreased erythropoietin, and eventually, hyperuricemia.

Excess or inappropriate protein can also cause elevated uric acid concentrations that can precipitate the same cascade of events as occurs with decreased GFR. Herbivorous reptiles, including green iguanas and many tortoises, require relatively low levels of protein in their diet, and should only be fed plant material. Carnivorous reptiles should be fed whole animal prey rather than organ meats such as beef liver.

Other causes of renal failure in reptiles include nephrocalcinosis secondary to nutritional secondary hyperparathyroidism; amyloid deposition; hypercholesterolemia with xanthomatosis; hypovitaminosis A with squamous metaplasia and blockage of renal collecting ducts; toxins such as aminoglycoside antibiotics and organophosphate insecticides; and infectious agents. Parasites include trematodes, strongyloid nematodes, *Entamoeba invadens*, intranuclear coccidia, microsporidia, flagellated protozoa, and myxosporidia. Bacterial and fungal infections of the kidney usually result from systemic infection and are uncommon. Viral inclusion body disease of pythons, boas, and vipers can affect epithelial cells of the kidneys.

Next week: Part 2, Diagnosis & Current Therapy of renal failure in reptiles . . .