

Diagnosis and Treatment of Avian Renal Disease

Christal Pollock, DVM, DABVP-Avian

*College of Veterinary Medicine, Kansas State University, 1800 Denison Avenue,
Manhattan, KS 66502, USA*

Significant causes of renal disease in the companion parrot include dehydration, hypovitaminosis A, excessive dietary vitamin D₃, heavy metal toxicity, bacterial nephritis secondary to systemic disease, and renal carcinoma. Additional important differentials include renal lipidosis in merlins and amyloidosis in waterfowl and songbirds. Diagnosis of renal disease may rely on the identification of consistent clinical signs, clinical pathology, survey radiographs, and laparoscopic evaluation and biopsy of the kidneys. Treatment of avian renal disease relies on supportive care such as fluid therapy and nutritional support. Other treatments vary with the underlying cause and the clinical picture but may include systemic antibiotics, diuretics, parenteral vitamin A, and agents to lower uric acid levels such as allopurinol. Reports on the incidence of renal disease in the avian patient vary, but renal disease is common in poultry and birds of prey [1,2]. Clinical renal disease is probably under-recognized in the companion bird, with the notable exception of renal tumors in the budgerigar (*Melopsittacus undulatus*).

Clinical signs of renal disease

Vague clinical signs such as weakness, anorexia, vomiting, or regurgitation often predominate in avian renal disease [3,4]. Early signs of mechanical compression or invasion of spinal nerves may include twitching and subtle signs of pain [3–8]. In rare instances, painful behavior may include feather picking or self-mutilation over the synsacrum [4,8,9]. As disease progresses, hematuria, unilateral or bilateral limb paresis, and disuse muscle atrophy may be observed [3,4,10,11]. Renomegaly may also lead to

E-mail address: cpollock@vet.k-state.edu

cloacal atony and constipation [4,7]. Metabolic abnormalities, particularly those caused by bacterial or viral nephritis, may cause persistent polydipsia/polyuria and, less commonly, oliguria, anuria, or seizure activity [3,4,6,8].

Important differentials for primary renal disease

Metabolic causes of renal disease

Dehydration is an important contributor to renal disease. Severe or persistent dehydration increases resorption of water causing a subsequent reduction in urine flow. As uric acid secretion decreases, urates may precipitate in renal tubules and ureters leading to impaction and potentially renal failure [2,12–15].

Deposition of lipid in renal tubules is an important problem of chicks, poults, and adult captive merlins (*Falco columbarius*) [16,17]. This condition has also been reported in the budgerigar parakeet and sulfur-crested cockatoo (*Cacatua galerita*) [7]. Renal lipidosis has been correlated with high-fat or low-protein diets, starvation, biotin deficiency, and chronic liver disease [2,15–17]. Poultry may exhibit acute onset of lethargy, followed by paralysis and death [16,17]. Merlins generally die acutely and are found in good flesh or slightly overweight [16].

Neoplasia of the avian kidney

In a study of 1203 budgerigar parakeets, 16% had tumors, and 23% of these tumors were renal [11]. The most common tumor in the bird is renal adenocarcinoma, which sometimes causes osteolysis and sclerosis of the ileum and synsacrum and potentially infiltrates nearby muscle and other surrounding tissue [7,15]. Distant metastasis to the skin, lung, liver, and oviduct is rare [15,18–20].

Nutritional causes of renal disease

Excess dietary protein or calcium, hypovitaminosis A, or hypervitaminosis D may lead to nephritis and other degenerative renal changes [21]. Profound vitamin A deficiency causes squamous metaplasia of ureteral mucosa and collecting ducts leading to blockage of the ureters and secondary hydro-nephrosis, hyperuricemia, and oliguric/anuric renal failure [2,4].

Excess vitamin D₃ promotes metastatic mineralization of viscera including the kidney [15,22,23]. This problem most commonly affects nestling parrots [15]. Clinical signs may include polyuria/polydipsia, anorexia, crop stasis, and weight loss [14,22]. The recommended level of vitamin D₃ for chickens is 300 IU/kg feed. Toxic effects reportedly occur with vitamin D₃ levels exceeding 1000 IU/kg feed [23].

Inflammatory causes of renal disease

Renal amyloidosis is most common in captive, adult waterfowl, shorebirds, cranes, flamingos, and songbirds [4,15,24]. Amyloidosis is often associated with chronic inflammatory conditions such as sepsis, gout, enteritis, and arthritis [24–26].

Infectious causes of renal disease

The absence of lymph nodes and the presence of renal and hepatic portal systems increase the risk of systemic or gastrointestinal microbes affecting the kidney [27,28].

Viral nephritis

Avian polyomavirus is the most important cause of viral nephritis in the companion psittacine bird. Up to 70% of affected non-budgerigar psittacines develop glomerulopathy characterized by immune complex deposition, but affected birds die acutely from other problems without showing signs of renal disease [4,15,26,29–31].

Other viruses with tropism for the avian kidney include infectious bronchitis virus, picornavirus, paramyxoviruses such as Newcastle disease virus, influenza virus, and togaviruses [12,15,27,32–34]. Infectious bronchitis virus is an important cause of renal disease and urolithiasis in galliforms [12,15,32]. Lymphoplasmacytic interstitial nephritis is common in birds infected with West Nile virus but only as a part of generalized disease [15,35].

Bacterial nephritis

Bacterial nephritis usually occurs when bacteria enter the kidney secondary to systemic disease through the renal arteries or the renal portal system [4,26]. Rarely, bacteria ascend the ureters secondary to conditions such as chronic cloacitis [4,23,26]. A wide range of bacteria has been reported to cause bacterial nephritis including Enterobacteriaceae, *Pasteurella* spp, *Pseudomonas* spp, *Streptococcus* spp, and *Staphylococcus* spp [4,15,26,27]. *Listeria monocytogenes* has been reported in raptors [15,26], whereas *Erysipelothrix rhusiopathiae* has been reported in quail and chicken [15,26,36]. *Mycobacterium avium* can, rarely, cause renal lesions [15,37].

Chlamydial nephritis

Chlamydial nephritis is poorly documented [4,38]. In a survey of 23 birds with psittacosis, 35% had renal congestion, bile pigment nephrosis, and glomerulopathy, but *Chlamydophila psittaci* could not be detected in renal tissue [26]. Identification of chlamydial organisms in the avian kidney has been reported in only two juvenile parrots [38].

Fungal nephritis

Fungi are a rare cause of renal disease [4,39]. Lesions may develop from fungal invasion of vessels or extension from air sacs [15].

Parasitic nephritis

Renal coccidiosis is the most important cause of parasitic nephritis. Disease caused by the coccidian *Eimeria* spp is most common in free-ranging, juvenile waterfowl [40,41]. Disease has also been described in the domestic goose (*Anser anser domesticus*) and aquatic birds such as the loon (*Gavia immer*), gull (*Larus argentatus*), puffin (*Fratercula arctica*), cormorant (*Phalacrocorax auritus*), woodcock (*Scolopax minor*), and penguin (*Eudyptula minor*) [14,15,42–45]. Renal coccidiosis is less commonly reported in raptors [15]. Although renal coccidiosis is often asymptomatic, emaciation, acute renal failure, and death may occur secondary to granulomatous interstitial nephritis [14,46,47].

The microsporidian, *Encephalitozoon hellem*, may also cause severe granulomatous nephritis [14,26,46,48–50], although the presence of microsporidians in the kidney or urine can be incidental [14,15,51]. Renal microsporidiosis is most commonly reported in the lovebird (*Agapornis* spp), particularly those positive for psittacine beak and feather disease [50,51]. There are also reports of renal microsporidiosis in the budgerigar, eclectus (*Eclectus roratus*), and red-bellied parrot (*Poicephalus rufiventris*) [51,52].

Traumatic causes of avian renal disease

In mammals, crush injuries and other conditions causing muscle necrosis are known to cause tubular changes, myoglobin cast formation, and renal failure, [14] Myoglobinuria has been reported in flamingo (*Phoenicopterus* sp) and ostrich (*Struthio camelus*) with capture myopathy, and there is one report of renal failure in an ostrich with extensive muscle necrosis and marked hyperuricemia [14,27].

Direct trauma is rare because the avian kidneys are so well protected by bone [7]. If a renal hematoma does develop, it can apply pressure to spinal nerves causing limb paresis [27]. Crushing of the kidney may also occur during dystocia [46].

Toxic nephropathies in the avian patient

Because of the presence of renal and hepatic portal systems, the avian kidney is frequently affected by toxins in the avian gut such as heavy metals, anti-inflammatory agents, and antibiotics [28]. Lead toxicity is associated with acute tubular necrosis or nephrosis and visceral gout [53].

The nonsteroidal anti-inflammatory agent flunixin has been implicated in presumptive nephrotoxicity of cranes and flamingos [54]. In northern bobwhite quail (*Colinus virginianus*), doses of flunixin as low as 0.1 mg/kg led to the development of gout [54]. Another nonsteroidal anti-inflammatory agent, diclofenac, has been linked to renal failure, visceral gout, and high death rates in vultures of the Indian subcontinent [55].

Most information regarding antibiotic nephrotoxicity is based on studies in mammals. For instance, renal tubules accumulate aminoglycoside potentially leading to nephrotoxicity in mammals [56]. Gentamicin may be more likely to cause nephrotoxicity in the bird because polyuria/polydipsia is often seen even at low doses [56]. Gentamicin (5 mg/kg intramuscularly every 12 hours for 7 days) led to profound polyuria/polydipsia in cockatoos (*Eolophus sp*) which persisted for 23 days after stopping treatment [57]. Loss of balance, impaired vision, and muscle spasms were described in two falcons (*Falco biarmicus*) given gentamicin (5 mg/kg/d for 4 days) [58]. Amikacin is considered the least nephrotoxic of the aminoglycosides, but transient polyuria/polydipsia may still occur [59].

A host of other drugs and toxins have been associated with renal lesions in birds, including dexamethasone, medroxyprogesterone, aflatoxins, mycotoxins, herbicides, and vitamin D₃-based rodenticides [15,46,60,61]. There are also reports of oak toxicity in a cassowary (*Casuaris casuaris*) [62] and of ethylene glycol poisoning in geese [63].

Postrenal disease

Conditions such as urolithiasis, dystocia, cloacal, or coelomic masses and, in rare instances, ureteral tumors may cause mechanical compression or obstruction of the avian ureter [7,64].

Urolithiasis and visceral gout

Urolithiasis and visceral gout are important causes of renal failure in pullets and caged laying hens. These conditions are seen only sporadically in companion birds [2,28]. Visceral gout is defined as the accumulation of uric acid topi on serosal surfaces of the pericardium, liver capsule, air sacs, and within the kidney but may be found any tissue [26]. Urolithiasis is simply the presence of urinary tract calculi.

The pathogenesis of gout is not completely understood, but gout is generally associated with conditions that reduce uric acid excretion or increase uric acid production [2,23,65]:

- Reduced uric acid excretion
- Increased uric acid production
- Dehydration
- Excess dietary calcium
- Renal tubular disease
- Excess dietary protein

Infectious renal disease
Hypovitaminosis A
Obstructive ureteral disease

Urolith development is most commonly associated with severe dehydration; other factors may include excess dietary calcium, dietary electrolyte imbalances, infectious bronchitis virus, *Mycoplasma synoviae* infection, mycotoxicosis, or shipping stress [12,13,66–68]. Excess dietary protein has also been correlated with increased production of uric acid, but even with very high levels of dietary protein (ie, 80%) gout develops only in genetically susceptible individuals [69,70]. Nevertheless, it is still theorized that long-term of high-protein feeding may induce hyperuricemia in granivorous or nectivorous birds [23,71].

The presence of uroliths in the kidney leads to compensatory hypertrophy of remaining renal tissue. Affected birds often appear normal until ureteral flow from the contralateral kidney is blocked, leading to lethargy, straining, and death [27,66,67]. Visceral gout is rarely diagnosed ante mortem, and birds are usually found dead [4].

Articular gout

Articular gout is defined as the accumulation of uric acid tophi in or around joints. Articular gout lesions are particularly common on the foot and hock [65]. Clinical signs of articular gout may include reluctance to move, shifting from leg to leg, lameness, and joint swelling [4].

Diagnosis

Early recognition and diagnosis of renal disease is extremely challenging, but an early definitive diagnosis provides the best opportunity for helping the patient [3].

Clinical pathology

In advanced renal disease, normocytic-normochromic anemia, hyperuricemia, uremia, and changes in plasma electrolyte, calcium, and phosphorus levels may be detected [72]. Uric acid excretion is largely independent of urine flow and therefore is unaffected by moderate changes in glomerular filtration [72]. Elevations in uric acid (up to 20 mg/dL) may be seen with severe dehydration [23,73,74], but uric acid does not increase significantly with renal disease unless there is extensive tubular damage [75]. Postprandial hyperuricemia may occur for up to 8 hours in carnivorous birds [76,77].

Urea nitrogen (BUN) has little value in the detection of renal disease in most birds [4,73], but BUN is a sensitive indicator of hydration. In the dehydrated bird, up to 99% of BUN is reabsorbed. A significant postprandial elevation in BUN has also been documented in healthy raptors [77].

The avian kidney cannot concentrate sodium or electrolytes much above normal levels [78]. Possible findings with renal failure may include hyponatremia, hyperkalemia, hypocalcemia, and hyperphosphatemia [27], although elevations in phosphorus are not commonly recognized in avian renal disease [72,74]. Alterations in these electrolytes have been inconsistently reported in active cases of avian renal disease. No definitive correlations between electrolyte abnormalities and renal disease in birds have been made.

Urinalysis

Urine flows from the ureters into the urodeum and then enters the colon and, in some species, the cecum or ileum, by reverse peristalsis (Fig. 1) [79,80]. Columnar epithelial cells lining the urodeum and colon modify ureteral urine through the absorption or secretion of water, electrolytes, and nitrogen [78]. Important indications for urinalysis include persistent biochemical or radiographic abnormalities consistent with renal disease or persistent polyuria (Fig. 2) [14]. Causes of polyuria are extensive and non-specific and include fluid therapy, renal disease, liver disease, gastrointestinal disease, diabetes mellitus, and pituitary tumors [4,14]. Polyuria may also occur with sepsis even when the pathogen does not directly affect the kidney, and psychogenic polydipsia has been reported in one African gray parrot (*Psittacus erithacus*) [14,81]. A common cause of polyuria and pollakiuria in the avian patient is stress [14,82].

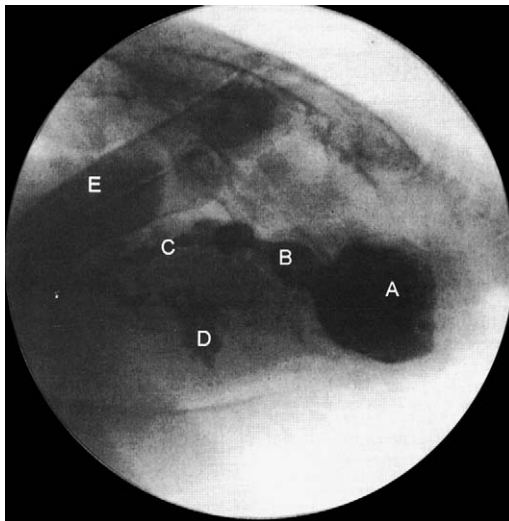


Fig. 1. Retroperistalsis of ureteral urine from cloaca (A) into avian large intestine (B, C, D). (E) the femurs. (From Brummermann M, Braun EJ. Effect of salt and water balance on colonic motility of white leghorn roosters. *Am J Physiol Regulatory Integrative Comp Physiol* 1995;268:690-8; with permission.)



Fig. 2. Polyuria in a bird dropping. Notice the large ring of urine around the feces and urates. (Courtesy of Ed Ramsay, DVM, DACZM.)

Although cloacal cannulation techniques have been described [83], free-catch urine samples are always collected from clinical patients. Obtain fresh urine samples free of urates and feces from clean, nonabsorbent surfaces such as wax paper [3,14,23,46]. A free-catch urine sample does not necessarily represent ureteral urine, and this fact should be taken into account when interpreting the results.

Birds possess a limited ability to concentrate urine, making avian urine isosmotic or slightly hyperosmotic. Urine specific gravity normally ranges from 1.005 to 1.020 g/mL but is highly variable among the different species. Urine specific gravity is not particularly useful unless values are consistently low [14,46].

Urine color

Pigments present in feces or newspaper can leach into urine and urates over time [14,23]. In the anorectic bird, concentrated bile pigments create emerald green or black feces that may stain urine even before droppings are passed [23]. Liver dysfunction or, in rare instances, hemolysis, may lead to biliverdinuria or lime-green, yellow, or, less commonly, orange urine and urates [14,23,46].

Red urine may be seen with hematuria, hemoglobinuria, or myoglobinuria. Hemoglobinuria may be seen in Amazon parrots (*Amazona* spp) with lead toxicosis producing dark red, pink, or tan/brown urates [4,23]. Hematuria may be associated with renal neoplasia, nephritis, or toxic nephropathy, although blood can also originate from the intestinal or reproductive tracts [4,23]. Transient wine-colored urine may occur in chicks, especially African gray and eclectus parrots. This condition may be correlated with hand-feeding animal protein-based diets [4,14,23].

Urine dipstick parameters

The pH of avian urine typically ranges from 6.0 to 8.0 [14,46,9]. Urine pH may be influenced by diet and cloacal contents [23,46], with urine more acidic in laying hens and more alkaline with bacterial metabolism [84]. Glucose levels in urine are normally zero to trace, although biliverdinuria may interfere with urine protein readings [23,46]. Normal avian urine is also free of ketones except during starvation or migration, when metabolism switches to beta-oxidation of fats [23,26]. Standard mammalian urine dipstick tests should be interpreted with caution, because these tests are not designed or calibrated for accuracy with avian species.

Urine sediment

Lane [46] recommends centrifugation of urine for 1 to 2 minutes. Normal sediment contains many squamous epithelial cells and amorphous urate, calcium oxalate, and sulfonamide crystals [46]. Low numbers of red and white cells (<3/high power field, $\times 40$) are present in avian urine. There should also be small numbers of bacteria present that are probably from fecal or cloacal contamination [14,23,46]. Normal bird urine contains no casts. Granular, hemoglobin, and other casts are reported in the literature and may be associated with renal disease [14,46].

Blood culture

To identify the cause of sepsis and bacterial nephritis, blood culture is a much better test than urine culture [14].

Radiographs

The avian kidney is difficult to evaluate radiographically because of its position within the synsacral fossa. Obscured by parenchyma on the ventrodorsal view, the kidneys are best viewed on the lateral projection. The most consistent radiographic sign of renomegaly is enlargement of the cranial renal division, which is best appreciated on the lateral view. Enlargement of this cranial renal division will also make the kidneys more apparent on the ventrodorsal view [3,7]. Renomegaly will also cause the wedge of air sac space separating the kidneys and coelomic viscera to become diminished in the lateral view. There may also be loss of the air sac diverticulum separating the dorsal renal surface from the ventral synsacrum [23]. Marked renomegaly may displace the ventriculus ventrally or caudoventrally [4]. Positive contrast radiography may make evaluation of the kidneys easier by helping outline coelomic structures [4,28]. Intravenous excretory urography may also provide more information on renal size, shape, and function [27,28].

Increased renal opacity may be associated with small kidney size, dehydration, or renal mineralization (Fig. 3) [3,4,23]. Urate tophi are not

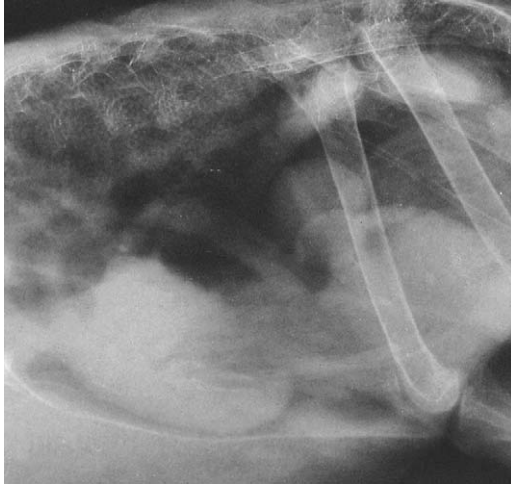


Fig. 3. Radiopacity of kidneys in a spectacled owl (*Pulsatrix perspicillata*) on the lateral view.

normally evident radiographically, but congestion of urates secondary to obstruction or gout may also lead to opacification [7]. Although the normal avian kidneys are more difficult to evaluate on the ventrodorsal view, they will become prominent with radiopacity (Fig. 4) [7,27].



Fig. 4. Radiopacity of kidneys in a spectacled owl (*Pulsatrix perspicillata*) on the ventrodorsal projection.

Ultrasonography

Because of the dorsal position of the avian kidney and the presence of air sacs, ultrasound is generally impossible in the normal bird [5,85]. Transcloacal ultrasound of the normal kidney has been described in large birds [86]. In smaller birds, organomegaly or ascites may compress the abdominal air sacs enough to create an acoustic window [5,23,85].

Alternate imaging

CT or MRI may prove helpful for evaluation of the renal system [87].

Laparoscopy

Laparoscopic renal biopsy is the best ante mortem diagnostic test for avian renal disease [4]. Indications for renal biopsy include persistent polyuria/polydipsia and serum biochemical abnormalities, gout, radiographic abnormalities of the kidneys, or abnormal urinalysis results, particularly the presence of casts [1,4,8]. Renal biopsy should be avoided in patients with a single kidney, cystic kidneys, renal abscesses, or hydronephrosis [1,3].

The standard entry site for laparoscopy is the caudal thoracic air sac [3,28]. A caudal entry site dorsal to the pubis and caudal to the ischium allows better access and visualization of the caudal renal division, but this site should be avoided in raptors because the presence of large tail muscles increases the risk of hemorrhage [28].

There are conflicting recommendations on where to biopsy the avian kidney. Muller [1] has recommended the cranial renal division because of its size and visibility, but the middle or caudal divisions may be safer sites. The cranial renal artery reportedly lies more superficial and therefore is more easily lacerated or torn during biopsy [28,88].

Diagnosis of gout

Cytologic evaluation of gouty lesions reveals uric acid crystals and inflammatory cells. The murexide test can be used to confirm the presence of urates. Nitric acid is mixed with crystals on a slide that is slowly flame dried. If red or purple color appears after ammonia is added, urates are present [4]. Histologically, urates are demonstrated by using alcohol fixation and special stains [28].

Renal scintigraphy

Given the challenges of obtaining a nonmodified ureteral urine sample, scintigraphy is a potentially useful method to evaluate renal function [89]. Methods have been described in the chicken, pigeon, and cockatiel (*Nymphicus hollandicus*) [7,89,90].

Systemic arterial blood pressure

Currently, blood pressure is not easily or routinely measured in the bird. In chickens, glomerular filtration is maintained over pressures ranging from 40 to 110 mm Hg [91].

Therapeutics

Management of renal failure should focus on delaying or halting progression of disease and treatment of sequelae. If the cause of the renal failure is known, specific therapy should be instituted. Although severe loss of renal tissue is permanently disabling, survival for extended periods is possible with only a small proportion of normal renal tissue.

Supportive care

Treat dehydration rapidly to prevent exacerbation of renal disease [28]. In renal failure, provide aggressive fluid therapy. Maintenance fluid requirements are estimated as 40 to 50 mL/kg/d in many species. Providing twice the required maintenance volume of fluids is a good initial goal for many patients. Replace only insensible fluid loss in anuric or oliguric birds (20–25 mL/kg/d) [28,78]. Weigh all birds receiving fluids twice daily.

Provide nutrition high in fat and carbohydrates and low in protein, potassium, phosphorus, and sodium in mild to moderate renal failure in granivorous and nectivorous species [4,28]. Products such as psittacine hand-feeding formula and low-protein products have been recommended [4,28]. Dietary restriction of protein may relieve some clinical signs of renal failure such as nausea. A diet restricted in protein and minerals also reduces the serum phosphorus levels, which may slow the progression of renal disease. Never the less, low-protein diets may lead to malnutrition, and low-protein diets are contraindicated in patients with advanced renal failure [92].

In the dog, research has suggested that omega-3 polyunsaturated fatty acid (PUFA) supplementation may preserve renal function and delay the progression of renal disease [93]. Omega-3 PUFA are abundant in fish oil [93,94]. Although the precise mechanism is not fully understood [93], omega-3 PUFA may work particularly well in animal models of renal disease that involve an immune component [94], a form of renal disease which is rare in the avian patient [15]. Administer omega-3 fatty acids for 6 to 12 months (S. Echols, personal communication, 2005).

Medical management

Drugs used to treat avian renal disease are listed in Table 1.

If dietary restriction of protein is unsuccessful in maintaining a normal level of serum phosphorus, hyperphosphatemia may be managed with oral

phosphate binders. Hypocalcemia may require calcium supplementation. Depending on the underlying cause of disease, other drugs that may be indicated include anti-inflammatory agents in amyloidosis and parenteral vitamin A in individuals with a deficient diet [4,8,28].

Volume overload is best prevented rather than treated, but diuretics such as furosemide or mannitol or renal vasodilators such as dopamine may be indicated if the animal is well hydrated and urine production is poor [4,92,96]. In confirmed or suspected cases of bacterial nephritis, choose antibiotics that achieve adequate renal tissue levels such as fluoroquinolones [28,56,97]. Antibiotics should also ideally be bactericidal. Cephalosporins are considered an excellent choice for urinary tract disease in the mammal, but the degree of biotransformation and routes of excretion are unknown in the bird [97]. Avoid nephrotoxic medications such as aminoglycoside antibiotics and other potential renal toxins (Box 1). Sulfa drugs should be avoided in dehydrated patients because sulfonamides possess a low water solubility and may precipitate in mammalian kidneys [56]. Differences in the organization of collecting ducts and ureteral branching in the bird may predispose certain avian species to obstructive nephropathies resulting from drug precipitation [56]. Administer antibiotics for at least 4 to 6 weeks (S. Echols, personal communication).

Hyperuricemia may respond to allopurinol or colchicine, although allopurinol can induce hyperuricemia and gout in the red-tailed hawk (*Buteo jamaicensis*) [95]. The recombinant enzyme, urate oxidase, shows great potential for the treatment of hyperuricemia in pigeons and red-tailed hawks [98]. Continue therapy with colchicine or allopurinol until signs of gout are gone and the patient is well stabilized (S. Echols, personal communication).

Administration of a histamine-receptor antagonist such as cimetidine or famotidine decreases gastric acidity and vomiting. Multiple B-vitamin preparations should be given to compensate for urinary losses of water-soluble vitamins [92].

Treatment for anemia may include iron supplementation and anabolic steroids such as nandrolone to stimulate erythrocyte production. Recombinant erythropoietin may also be effective in stimulating red blood cell production in birds showing clinical signs of anemia. Anti-erythropoietin antibodies are known to develop in a significant percentage of mammals, leading to refractory anemia [92].

In mammals, peritoneal dialysis or hemodialysis is ideally initiated when signs of renal disease are present and are not treatable by other forms of medical management. The use of dialysis has not been described in the avian patient.

Management of renal tumors

Nephrectomy is the treatment of choice for unilateral renal tumors in the dog [92]. Unfortunately, renal tumors are exceedingly difficult to manage

Table 1
Drugs used in avian renal disease

| Drug | Dosage | Indications | Comments |
|-------------------------|------------------------------|---------------------------------|--|
| Allopurinol | 10–15 mg/kg PO q 12 h | Hyperuricemia | Do not give to red-tailed hawks and possibly other birds of prey; maintain hydration; use with amoxicillin/clavulanate or aspirin is contraindicated |
| Aluminum hydroxide | 30–90 mg/kg PO q 12 h | Phosphate binder | Compounds containing aluminum may interfere with fluoroquinolone absorption |
| Amoxicillin | 20–100 mg/kg PO q 12–24 h | Bacterial nephritis | Use with allopurinol is contraindicated |
| Amoxicillin/clavulanate | 125 mg/kg PO q 8 h | Bacterial nephritis | |
| Butorphanol | 0.5–4.0 mg/kg IM q 4–6 h | Analgesia, renal tumors | |
| Calcium glubionate | 25–150 mg/kg PO q 12–24 h | Hypocalcemia, hyperphosphatemia | |
| Calcium gluconate (10%) | 25–100 mg/kg SC, IM q 12 h | Hypocalcemia, hyperphosphatemia | If the calcium × phosphorus (Ca × P) product exceeds 70, metastatic mineralization is likely to occur as in mammals; compounds containing calcium may interfere with tetracycline and fluoroquinolone absorption |
| Cefotaxime | 75–100 mg/kg IM, IV q 4–8 h | Bacterial nephritis | Administration q 4 h recommended in cockatiels based on pharmacokinetic data |
| Cefoxitin | 50–100 mg/kg IM, IV q 6–12 h | Bacterial nephritis | |
| Ceftazidime | 50–100 mg/kg IM, IV q 4–8 h | Bacterial nephritis | |
| Ceftiofur | 10 mg/kg IM q 4–12 h | Bacterial nephritis | |
| | 50–100 mg/kg IM q 4–8 h | | |
| Ceftriaxone | 100 mg/kg IM q 4 h | Bacterial nephritis | Gradually increase dose to q 12 h; may exacerbate gout in some cases |
| Cimetidine | 5 mg/kg PO, IM q 8–12 h | Nausea | |
| Ciprofloxacin | 50 mg/kg PO, IV 12h | Bacterial nephritis | |
| Colchicine | 0.01–0.04 mg/kg PO q 12–24 h | Hyperuricemia | |
| | | | |

| | | | |
|----------------------------|--|--|---|
| Enrofloxacin | 10–15 mg/kg PO, SC, IM q 12 h | Bacterial nephritis | Compounds containing calcium, aluminum interfere with absorption |
| Furosemide | 0.1–2.0 mg/kg PO, SC, IM, IV q 6–12 h 1–4 mg/kg PO, SC, IM, IV q 6–12 h | Volume overload | Lower dosage range recommended for raptors and nectivorous birds |
| Iron dextran | 10 mg/kg IM, repeat in 7–10 d | Anemia | Use cautiously in species in which iron storage disease is common (toucans, mynahs) |
| Mannitol | 0.25–2.0 mg/kg q 24 h IV (slow bolus) | Volume overload | Osmotic diuretic |
| Meloxicam | 0.5 mg/kg q 1 h PO, SC | Analgesia in the palliative treatment of renal tumors; amyloidosis | Potentially the least nephrotoxic of the nonsteroidal agents |
| Metoclopramide | 0.5 mg/kg PO, IM, IV q 8 h | Gastrointestinal ileus; crop stasis | |
| Methylprednisolone acetate | 0.5–1.0 mg/kg PO, IM | Renal tumor, palliative | |
| Nandrolone laurate | 0.2–2.0 mg/kg SC, IM once or q 3 wk | Anemia; chronic renal failure | |
| Norflaxacin | 8–10 mg/kg PO q 24 h | Bacterial nephritis | |
| Omega-3 fatty acids | 0.1–0.2 mL/kg flaxseed oil: corn oil mixed at a ratio of 1:4 PO SID or added to food | Glomerulopathy | Consider vitamin E supplementation with long-term use |
| Potassium chloride | 20–40 mEq/L fluids | Diuresis, hypokalemia | |
| Urate oxidase | 100–200 IU/kg IM q 24 h | Hyperuricemia | Currently very expensive |
| Vitamin A | 2000–5000 IU/kg IM once or q 24 h × 14 d, followed by 250–1000 IU/kg q 24 h PO | Hypovitaminosis A | Chronic use may lead to vitamin A toxicity. |
| Vitamin B complex | 1–2 mL/L fluids | Renal failure, supportive care | |

Data from Pollock CG, Carpenter JW, Antinoff N. Birds. In: Carpenter JW, editor. Exotic animal formulary. 3rd edition. St. Louis (MO): Elsevier Saunders; 2005. p. 135–344; Lumeij JT, Redig PT. Hyperuricemia and visceral gout induced by allopurinol in red-tailed hawks (*Buteo jamaicensis*). In: Proceedings Tagung der Fachgruppe Geflügelkrankheiten, Giessen, Germany: Deutsche Veterinärmedizinische Gesellschaft; 1992.

Box 1. Drugs with known potential for nephrotoxicity^a

Aminoglycosides particularly gentamicin
 Amphotericin B^b
 Calcium EDTA
 Chloramphenicol
 Cisplatin
 Deferoxamine
 Enalapril
 Flunixin meglumine and other non-steroidal anti-inflammatory agents
 Nystatin^c
 Paramomycin^d
 Polymyxin B
 Sulfanomides^e
 Tetracyclines^f

^a If potentially nephrotoxic agents must be used, monitor patients closely for clinical signs of nephrotoxicity such as polyuria/polydipsia, monitor serum/plasma uric acid levels and maintain hydration. Most knowledge regarding nephrotoxicity is based upon information gained from mammals; the drugs listed are not the only potentially nephrotoxic agents available.

^b Amphotericin B is highly nephrotoxic in mammals, however nephrotoxicity has not been documented in birds with even long-term treatment.

^c Nephrotoxicity may occur if nystatin is systemically absorbed due to the presence of erosions and ulcers lining the gastrointestinal tract.

^d Nephrotoxicity may occur if ulcerative bowel lesions are present and systemic absorption occurs.

^e Sulfa drugs are known to possess low water solubility, and may precipitate in renal tubules in the face of dehydration.

^f High doses of tetracycline or outdated tetracycline may cause acute tubular nephrosis.

Data from Refs. [1,56,59].

surgically in the bird because of the kidney's dorsal location, the vascular nature of these tumors, and the likelihood of regional invasion into nearby tissues [4,10].

Palliative treatment is more commonly chosen for management of renal tumors and may include analgesics and steroids such as methylprednisolone [4,99]. In mammals, chemotherapy has not been shown to be effective against renal tumors other than lymphosarcoma [92], and the use of chemotherapy has been little evaluated for avian renal tumors. Use of carboplatin (5 mg/kg intravenously) in a parakeet dramatically improved limb use, although the mass continued to enlarge [100].

Treatment of urolithiasis

Treatment of urolithiasis is generally not attempted in domestic fowl, the type of bird most frequently affected by this condition. The primary goal in the treatment of obstructive uropathy is to relieve blockage of urine flow. There is one description of surgical removal of ureteroliths in a companion parrot and one report on the use of lithotripsy for urolithiasis in a Magellanic penguin (*Spheniscus magellanicus*) [64,101].

Fluid therapy, ideally administered by an intravenous or intraosseous route, improves renal function and corrects electrolyte abnormalities after the obstruction has been relieved. Normal saline is the fluid of choice. Large quantities of fluids may be required because postobstructive diuresis may occur for 1 to 5 days. Carefully monitor urine output, body weight, serum electrolytes, hematocrit, and total protein levels [92].

Summary

Renal disease in the avian patient is probably under-recognized. An important reason may be the subtle nature of clinical signs until disease is quite advanced. Common diagnostic tests performed in the diagnosis of renal disease include a complete blood cell count, chemistry panel, urinalysis, survey radiographs, and laparoscopic evaluation and biopsy of the kidneys. Depending on the patient's signs, history, and physical examination findings, additional diagnostic tests may include heavy metal blood levels, fecal flotation, blood culture, and viral serologic tests. Important underlying causes of renal disease in the avian patient include renal coccidiosis in waterfowl, dehydration, toxicosis, systemic bacterial infection, and amyloidosis. Primary renal tumors are relatively uncommon in birds with the notable exception of the budgerigar parakeet. When gout is present, it should generally be considered as a clinical manifestation of severe renal dysfunction [4,78]. The mainstay of treatment for renal disease in the bird is supportive care such as fluid therapy and nutritional support. Additional therapy should ideally be tailored to the underlying pathogenesis of disease and specific sequelae.

References

- [1] Muller K, Gobel T, Muller S, et al. Use of endoscopy and renal biopsy for the diagnosis of kidney disease in free-living birds of prey and owls. *Vet Rec* 2004;155(11):326–9.
- [2] Siller WG. Renal pathology of the fowl- a review. *Avian Pathol* 1981;10:187–262.
- [3] Murray MJ, Taylor M. Avian renal disease: endoscopic applications. *Seminars in Avian and Exotic Pet Medicine* 1999;8(3):115–21.
- [4] Speer BL. Diseases of the urogenital system. In: Altman RB, Clubb SL, Dorrestein GM, et al, editors. *Avian medicine and surgery*. Philadelphia: WB Saunders; 1997. p. 625–44.
- [5] Canny C. Gross anatomy and imaging of the avian and reptilian urinary system. *Seminars in Avian and Exotic Pet Medicine* 1998;7(2):72–80.

- [6] Gevaert D, Nelis J, Verhaeghe B. Plasma chemistry and urine analysis in Salmonella-induced polyuria in racing pigeons (*Columbia livia*). *Avian Pathol* 1991;20:379–86.
- [7] McMillan MC. Imaging of avian urogenital disorders. *AAV Today* 1988;2(2):74–82.
- [8] Echols MS. Antemortem diagnosis and management of avian renal disease. In: Association of Avian Veterinarians Annual Conference Proceedings. St. Paul (MN): 1998. p. 83–90.
- [9] Van Toor AJ, Zwart P, et al. Adenocarcinoma of the kidney in two budgerigars. *Avian Pathol* 1984;13:145–50.
- [10] Freeman KP, Hahn KA, Jones MP, et al. Right leg muscle atrophy and osteopenia caused by renal adenocarcinoma in a cockatiel (*Melopsittacus undulatus*). *Vet Radiol Ultrasound* 1999;40(2):144–7.
- [11] Neuman U, Kummerfeld N. Neoplasms in budgerigars (*Melopsittacus undulatus*): clinical, pathomorphological and serological findings with special consideration of kidney tumours. *Avian Pathol* 1983;12:353–62.
- [12] Cowen BS, Wideman RF, Rothenbacher H. An outbreak of avian urolithiasis on a large commercial egg farm. *Avian Dis* 1987;31:392–7.
- [13] Julian R. Water deprivation as a cause of renal disease of chickens. *Avian Pathol* 1982;11: 615–7.
- [14] Phalen D. Avian renal disorders. In: Fudge AM, editor. *Laboratory medicine avian and exotic pets*. Philadelphia: WB Saunders; 2000. p. 61–8.
- [15] Schmidt RE, Reavill DR, Phalen DN. Urinary system. In: *Pathology of pet and aviary birds*. Ames (IA): Iowa State Press; 2003. p. 95–107.
- [16] Forbes NA, Cooper JE. Fatty liver-kidney syndrome of merlins. In: Redig PT, Cooper JE, Remple D, et al, editors. *Raptor biomedicine*. Minneapolis (MN): University of Minnesota Press; 1993. p. 45–8.
- [17] Pearce J, Balnave D. A review of biotin deficiency and fatty liver and kidney syndrome in poultry. *Br Vet J* 1978;134(6):598–608.
- [18] Howerth EW, Schorr LF, Nettles VF. Neoplasia in free-flying ruffed grouse (*Bonasa umbellus*). *Avian Dis* 1986;30(1):238–40.
- [19] Hubbard GB. Renal carcinoma in a captive Edwards lorry (*Trichoglossus haematodus capistratus*). *J Wildl Dis* 1983;19(2):160–1.
- [20] Latimer KS, Ritchie BW, Campagnoli RP, et al. Metastatic renal carcinoma in an African grey parrot (*Psittacus erithacus erithacus*). *J Vet Diagn Invest* 1996;8:261–4.
- [21] Chandra M, Singh B, Soni GL, et al. Renal and biochemical changes produced in broilers by high-protein, high-calcium, urea-containing, and vitamin-A-deficient diets. *Avian Dis* 1984;8(1):1–11.
- [22] Schoemaker NJ, Lumeij JT, Beynen AC. Polyuria and polydipsia due to vitamin and mineral oversupplementation of the diet of a salmon crested cockatoo (*Cacatua moluccensis*) and blue and gold macaw (*Ara ararauna*). *Avian Pathol* 1997;26:201–9.
- [23] Styles DK, Phalen DN. Clinical avian urology. *Seminars in Avian and Exotic Pet Medicine* 1998;7(2):104–13.
- [24] Schneider RR, Hunter DB, Waltner-Toews D, et al. A descriptive study of mortality at the Kortright waterfowl park 1982–1986. *Can Vet J* 1988;29:911–4.
- [25] Nakamura K, Tanaka H, Kodama Y, et al. Systemic amyloidosis in laying Japanese quail. *Avian Dis* 1998;42(1):209–14.
- [26] Phalen DN, Ambrus S, Graham DL. The avian urinary system: form, function, diseases. In: Association of Avian Veterinarians Annual Conference Proceedings. Boca Raton (FL): Association of Avian Veterinarians; 1990. p. 44–57.
- [27] Lierz M. Avian renal disease: pathogenesis, diagnosis, and therapy. *Vet Clin North Am Exotic Am Pract* 2003;6:29–55.
- [28] Lumeij JT. Pathophysiology, diagnosis and treatment of renal disorders in birds of prey. In: Lumeij JT, Remple D, Redig PT, et al, editors. *Raptor biomedicine III*. Lake Worth (FL): Zoological Education Network, Inc; 2000. p. 169–78.

- [29] Gerlach H, Enders F, Casares M, et al. Membranous glomerulopathy as an indicator of avian polyomavirus infection in Psittaciformes. *J Avian Med Surg* 1998;12(4): 248–54.
- [30] Lafferty SL, Fudge AM, Schmidt RE, et al. Avian polyomavirus infection and disease in a green aracaris (*Pteroglossus viridis*). *Avian Dis* 1999;43(3):577–85.
- [31] Phalen DN, Wilson VG, Graham DL. Characterization of the avian polyomavirus-associated glomerulopathy of nestling parrots. *Avian Dis* 1996;40(1):140–9.
- [32] Lee CW, Brown C, Hilt DA, et al. Nephropathogenesis of chickens experimentally infected with various strains of infectious bronchitis virus. *J Vet Med Sci* 2004;66(7): 835–40.
- [33] Swayne DE, Radin MJ, Hoepf TM, et al. Acute renal failure as the cause of death in chickens following intravenous inoculation with avian influenza virus A/chicken/Alabama/7395/75 (H4N8). *Avian Dis* 1994;38(1):151–7.
- [34] Ziegler AF, Ladman BS, Dunn PA, et al. Nephropathogenic infectious bronchitis in Pennsylvania chickens 1997–2000. *Avian Dis* 2002;46(4):847–58.
- [35] Kramer LD, Bernard KA. West Nile virus infection in birds and mammals. *Ann N Y Acad Sci* 2001;951:84–93.
- [36] Mutalib A, Keirs R, Austin F. Erysipelas in quail and suspected erysipeloid in processing plant employees. *Avian Dis* 1995;39(1):191–3.
- [37] Sato Y, Aoyagi T, Matsuura S, et al. An occurrence of avian tuberculosis in hooded merganser (*Lophodytes cucullatus*). *Avian Dis* 1996;40(4):941–4.
- [38] Shivaprasad HL, Crespo R, Woolcock PR, et al. Unusual cases of Chlamydiosis in psittacines. In: Association of Avian Veterinarians Annual Conference Proceedings. Monterey (CA): 2002, p. 205–7.
- [39] Tham VL, Purcell DA, Schultz DJ. Fungal nephritis in a grey-headed albatross. *J Wildl Dis* 1974;10:306–9.
- [40] Gajadhar AA, Cawthorn RJ, Wobeser GA, et al. Prevalence of renal coccidia in wild waterfowl in Saskatchewan. *Can J Zool* 1983;61:2631–3.
- [41] Leighton FA, Gajadhar AA. *Eimeria fraterculae* sp. n. in the kidneys of Atlantic puffins (*Fratercula arctica*) from Newfoundland, Canada: species description and lesions. *J Wildl Dis* 1986;22(4):520–6.
- [42] Gajadhar AA, Leighton FA. *Eimeria wobeseri* sp. n. and *Eimeria goelandi* sp. n. (Protozoa: Apicomplexa) in the kidneys of herring gulls (*Larus argentatus*). *J Wildl Dis* 1988;24(3): 538–46.
- [43] Montgomery RD, Novilla MN, Shillinger RB. Renal coccidiosis caused by *Eimeria gavia* n. sp. in a common loon (*Gavia immer*). *Avian Dis* 1978;22(4):809–14.
- [44] Obendorf DL, McColl K. Mortality in little penguins (*Eudyptula minor*) along the coast of Victoria, Australia. *J Wildl Dis* 1989;16(2):251–9.
- [45] Yabsley MJ, Gottdenker NL, Fischer JR. Description of a new *Eimeria* sp. and associated lesions in the kidneys of double-crested cormorants (*Phalacrocorax auritus*). *J Parasitol* 2002;88(6):1230–3.
- [46] Lane RA. Avian urinalysis a practical guide to analysis and interpretation. In: Roskopf WJ, Woerpel RW, editors. Diseases of cage and aviary birds. 3rd edition. Baltimore (MD): Williams and Wilkins; 1996. p. 783–94.
- [47] Skirnisson K. Mortality associated with renal and intestinal coccidiosis in juvenile eiders in Iceland. *Parasitologia* 1997;39(4):325–30.
- [48] Lowenstein LJ, Petrak ML. Microsporidiosis in two peach-faced lovebirds. In: Midgaki G, Montali RJ, editors. The comparative pathology of zoo animals. Washington (DC): Smithsonian Institution; 1980. p. 365–8.
- [49] Poonacha KB, William PD, Stamper RD. Encephalitozoonosis in a parrot. *J Am Vet Med Assoc* 1985;186(7):700–2.
- [50] Randall CJ, Higgins RJ, Harcourt-Brown NH. Microsporidian infection in lovebirds (*Agapornis* sp.). *Avian Pathol* 1986;15(2):223–31.

- [51] Barton CE, Phalen DN, Snowden KF. Prevalence of microsporidian spores shed by asymptomatic lovebirds: evidence for a potential emerging zoonosis. *J Avian Med Surg* 2003; 17(4):197–202.
- [52] Pulparampil N, Graham D, Phalen D. *Encephalitozoon hellem* in two eclectus parrots (*Eclectus roratus*): identification from archival tissues. *J Eukaryot Microbiol* 1998;45(6): 651–5.
- [53] Degernes LA. Toxicities in waterfowl. *Seminars in Avian and Exotic Pet Medicine* 1995;4(1):15–22.
- [54] Klein PN, Charmatz K, Langenberg J. The effect of flunixin meglumine (Banamine) on the renal function in northern bobwhite quail (*Colinus virginianus*): an avian model. In: Association of Avian Veterinarians Annual Conference Proceedings. Boca Raton (FL): Association of Avian Veterinarians; 1994. p. 128–31.
- [55] O'Rourke K. Veterinary drug kills vultures abroad. *J Am Vet Med Assoc* 2004;224(8):1238, 1240.
- [56] Frazier DL, Jones MP, Orosz SE. Pharmacokinetic considerations of the renal system in birds: part II. Review of drugs excreted by renal pathways. *J Avian Med Surg* 1995;9(2): 104–21.
- [57] Flammer K, Clark CH, Drewes LA, et al. Adverse effects of gentamicin in scarlet macaws and galahs. *Am J Vet Res* 1990;51(3):404–7.
- [58] Fernandez-Repollet E, Rowley J, Schwartz A. Renal damage in gentamicin-treated lanner falcons. *J Am Vet Med Assoc* 1987;181(11):1392–4.
- [59] Pollock CG, Carpenter JW, Antinoff N. Birds. In: Carpenter JW, editor. *Exotic animal formulary*. 3rd edition. St. Louis (MO): Elsevier Saunders; 2005. p. 135–344.
- [60] Mollenhauer HH, Corrier DE, Huff WE, et al. Ultrastructure of hepatic and renal lesions in chickens fed aflatoxin. *Am J Vet Res* 1989;50(5):771–7.
- [61] Morgulis MS, Oliveira GH, Dagli ML, et al. Acute 2,4-dichlorophenoxyacetic acid intoxication in broiler chicks. *Poult Sci* 1998;77(4):509–15.
- [62] Kinde H. A fatal case of oak poisoning in a Double-Wattled Cassowary (*Casuarus casuarus*). *Avian Dis* 1988;32(4):849–51.
- [63] Petrak ML. Poisoning and other causalities. In: Petrak ML, editor. *Diseases of cage and aviary birds*. Philadelphia: Lea & Febiger; 1982. p. 646–52.
- [64] Dennis PM, Bennett RA. Ureterotomy for removal of two ureteroliths in a parrot. *J Am Vet Med Assoc* 2000;217(6):865–8.
- [65] Austic RE, Cole RK. Impaired renal clearance of uric acid in chickens having hyperuricemia and articular gout. *Am J Physiol* 1972;223(3):525–30.
- [66] Mallinson ET, Rothenbacher H, Wideman RF, et al. Epizootiology, pathology, and microbiology of an outbreak of urolithiasis in chickens. *Avian Dis* 1983;28(1):25–43.
- [67] Niznik RA, Wideman RF, Cowen BS, et al. Induction of urolithiasis in single comb white Leghorn pullets: effect on glomerular number. *Poult Sci* 1985;64(8):1430–7.
- [68] Wideman RF Jr, Closser JA, Roush WB, et al. Urolithiasis in pullets and laying hens: role of dietary calcium and phosphorus. *Poult Sci* 1985;64(12):2300–7.
- [69] Kamphues J, Otte W, Wolf P. Effects of increasing protein intake on various parameters of nitrogen metabolism in grey parrots (*Psittacus erithacus erithacus*). Abstracts of the First International Symposium on Pet Bird Nutrition. Hannover (Germany): 1997. p. 118.
- [70] Pegram RA, Wyatt RD. Avian gout caused by oosporein, a mycotoxin produced by *Chaetomium trilaterale*. *Poult Sci* 1981;60(11):2429–40.
- [71] Siller WG. Avian nephritis and visceral gout. *Lab Invest* 1959;8:1319–57.
- [72] Chandra M, Singh B, Gupta PP, et al. Clinicopathological, hematological, and biochemical studies in some outbreaks of nephritis in poultry. *Avian Dis* 1985;29(3): 590–600.
- [73] Lumeij JT. Plasma urea, creatinine and uric acid concentrations in response to dehydration in racing pigeons. *Avian Pathol* 1987;16:377–82.

- [74] Fudge AM. Avian clinical pathology—hematology and chemistry. In: Altman RB, Clubb SL, Dorrestein GM, et al, editors. Avian medicine and surgery. Philadelphia: WB Saunders; 1997. p. 142–57.
- [75] Lumeij JT, Wolfswinkel J. Tissue enzyme profiles of the budgerigar (*Melopsittacus undulatus*) [PhD thesis]. In: Lumeij JT, editor. A contribution to clinical investigative methods for birds with special reference to the racing pigeon (*Columbia livia domestica*). Utrecht: University of Utrecht; 1987. p. 71–7.
- [76] Kolmstetter CM, Ramsay EC. Effects of feeding on plasma uric acid and urea concentrations in blackfooted penguins (*Spheniscus demersus*). *J Avian Med Surg* 2000;14(3):177–9.
- [77] Lumeij JT, Remple JD, Remple CJ, et al. Plasma chemistry in Peregrine Falcons (*Falco peregrinus*): reference values and physiologic variations of importance for interpretation. *Avian Pathol* 1998b;27:129–32.
- [78] Dorrestein GM. Physiology of the urogenital system. In: Altman RB, Clubb SL, Dorrestein GM, et al, editors. Avian medicine and surgery. Philadelphia: WB Saunders; 1997. p. 622–5.
- [79] Goldstein DL, Skadhauge E. Renal and extrarenal regulation of body fluid composition. In: Whittow GC, editor. Sturkie's avian physiology. 5th edition. San Diego (CA): Academic Press; 2000. p. 265–97.
- [80] Braun EJ. Integration of renal and gastrointestinal function. *J Exp Zool* 1999;283(4–5):495–9.
- [81] Lumeij JT, Westerhof I. The use of water deprivation test for the diagnosis of apparent psychogenic polydipsia in a socially deprived African grey parrot (*Psittacus erithacus erithacus*). *Avian Pathol* 1988;17(4):875–8.
- [82] Palmore WP, Fregly MJ, Simpson CE. Catecholamine-induced diuresis in turkeys. *Proc Soc Exp Bio Med* 1981;167:1–5.
- [83] Halsema WB, Alberts H, De Bruijnen JJ, et al. Collection and analysis of urine in racing pigeons (*Columba livia domestica*). *Avian Pathol* 1988;17(1):221–5.
- [84] Hochleitner M. Biochemistries: Urinalysis. In: Ritchie BW, Harrison GJ, Harrison LR, editors. Avian medicine: principles and applications. Lake Worth (FL): Wingers Publishing Inc.; 1994. p. 242–4.
- [85] Hofbauer H, Krautwald-Junghanns M-E. Transcutaneous ultrasonography of the avian urogenital tract. *Vet Radiol Ultrasound* 1999;40(1):58–64.
- [86] Hildebrandt TH, Pitra C, Göritz F. Transintestinal ultrasonographic sexing. In: European Association of Avian Veterinarians Annual Conference Proceedings. Jerusalem (Israel): 1995. p. 37–41.
- [87] Romagnano A, Heard DJ, Johnson RD, et al. Magnetic resonance imaging of the brain and coelomic cavity of the domestic pigeon (*Columba livia domestica*). *Vet Radiol Ultrasound* 1996;37(6):431–40.
- [88] Speer BL, Harris D, Murray M, et al. Round table discussion: endoscopic renal biopsy. *J Avian Med Surg* 1997;11(4):273–8.
- [89] Marshall KL, Craig LE, Jones MP, et al. Quantitative renal scintigraphy in domestic pigeons (*Columba livia domestica*) exposed to toxic doses of gentamicin. *Am J Vet Res* 2003;64(4):453–62.
- [90] Radin MJ, Hoepf TM, Swayne DE. Use of a single injection solute-clearance method for determination of glomerular filtration rate and effective renal plasma flow in chickens. *Lab Anim Sci* 1993;43(6):594–6.
- [91] Wideman RF, Gregg CM. Model for evaluating avian hemodynamics and glomerular filtration rate autoregulation. *Am J Physiol* 1988;254(6 Part 2):R925–32.
- [92] Brown S, Sandersen SL. Urinary system. In: Kahn CM, Line S, editors. The Merck veterinary manual. Non-infectious diseases of the urinary system in small animals. 9th edition. Summerset: John Wiley & Sons; 2005. p. 1249–88.
- [93] Brown SA, Finco DR, Brown CA. Is there a role for dietary polyunsaturated fatty acid supplementation in canine renal disease? *J Nutr* 1998;128:2765S–7S.

- [94] Bauer JE, Markwell PJ, Rawlings JM, et al. Effects of dietary fat and polyunsaturated fatty acids in dogs with naturally developing chronic renal failure. *J Am Vet Med Assoc* 1999; 215(11):1588–91.
- [95] Lumeij JT, Redig PT. Hyperuricemia and visceral gout induced by allopurinol in red-tailed hawks (*Buteo jamaicensis*). In: Proceedings Tagung der Fachgruppe Gefluengelkrankheiten, Giessen, Germany: Deutsche Veterinaermedizinische Gesellschaft; 1992.
- [96] Braun EJ. Comparative renal function in reptiles, birds and mammals. *Seminars in Avian and Exotic Pet Medicine* 1998;7(2):62–71.
- [97] Anadón A, Martinez-Larranaga MR, et al. Pharmacokinetics and residues of ciprofloxacin and its metabolites in broiler chickens. *Res Vet Sci* 2001;71(2):101–9.
- [98] Poffers J, Lumeij JT, Redig PT. Investigations into the uricolytic properties of urate oxidase in a granivorous (*Columba livia domestica*) and in a carnivorous (*Buteo jamaicensis*) avian species. *Avian Pathol* 2002;31(6):573–9.
- [99] Bauck L. A clinical approach to neoplastic disease in the pet bird. *Seminars in Avian and Exotic Pet Medicine* 1994;1(2):65–72.
- [100] Macwhirter P, Pyke D, Wayne J. Use of carboplatin in the treatment of renal adenocarcinoma in a budgerigar. *Exotic DVM* 2002;4(2):11–2.
- [101] Machado C, Mihm F, Buckley DN, et al. Disintegration of kidney stones by extracorporeal shockwave lithotripsy in a penguin. In: Proceedings of the First International Conference on Zoological and Avian Medicine. Oahu (Hawaii): 1987. p. 343–9.