Non-infectious Diseases of Raptors

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This is Dr. Scott Ford's section of a 3-author paper presented for the Association of Avian Veterinarians Conference in 2011. Because of space limitations, references could not be included in the proceedings. Therefore, Dr. Scott Ford's section of the manuscript and all the references are presented in this document, available on Dr. Ford's website at www.alaskabirddoc.com. For the manuscripts of Drs. Jones and Chitty, please refer to the proceedings of the 2011 conference.

Scott Ford, DVM, Dipl ABVP (Avian), John Chitty, BVetMed, CertZooMed, MRCVS, and Michael Jones, DVM, Dipl ABVP (Avian)

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Affiliation: From Avian Specialty Veterinary Services, 13861 Hillcrest St NW, Poulsbo, WA 98370, USA (Ford); JC Exotic Pet Consultancy, Wombourne, Allington, Salisbury, Wilts SP4 0DD, UK (Chitty); and The University of Tennessee, CVM, Dept of SACS, 2407 River Dr, Room C247, Knoxville, TN 37996, USA (Jones).

Abstract: Birds of prey encounter a variety of non-infectious ailments including various types of intoxication, cardiovascular disease, neoplasia, urogenital disorders, and nutritional and metabolic disorders. These disorders will vary in presentation and frequency depending upon signalment and whether the affected bird is free-living, captive in a zoo, or used for falconry. This class will be co-taught by a veterinarian from each of these 3 care contexts. Emphasis will be upon practical recognition and treatment measures.

Key words: raptor, falconry, raptor rehabilitation, intoxication, neoplasia

Intoxicants of Clinical Interest in Raptors (Ford)

Heavy metals, barbiturates, cyanobacteria, anticoagulant rodenticides, avicides, and organophosphates are the most commonly encountered intoxicants with captive and wild raptors in a clinical setting.

Lead

Raptors achieve acute lead intoxication (plumbism) primarily through ingestion of prey or carrion containing lead ammunition fragments. Very small fragments of ingested lead can be life-threatening. Lead has a wide range of pathophysiologic effects in a variety of tissues. In summary, lead interferes with sulfhydryl-dependent enzyme function; mimics calcium and so interferes with neurologic function and mitochondrial respiration; and adversely affects DNA and RNA synthesis.1 Erythrocytes form lead-binding proteins in response to intoxication, and this may explain why red blood cells become fragile and rupture during severe intoxication.

Presentation may vary from mild ataxia and weakness to convulsions, seizures, paralysis, and death. A marked anemia is common, usually regenerative if sufficient time has passed for a bone marrow response. Radiographs may reveal metallic fragments in the gastrointestinal tract. Orthogonal views are important for ruling out peripheral bullet fragments which should not be

contributory to plumbism. Exceptions to this could include lead that is embedded in metaphyseal segments of long bones, particularly during egg-laying. Where metallic fragments are present, particularly when the fragments do not demonstrate deformation, consideration should still be given to whether the fragments are lead or some other non-toxic metal. Lead is a soft metal and will usually deform or fragment into irregular pieces upon impact although shotgun pellets may not always demonstrate this. The gold standard for diagnosis of plumbism is a whole blood lead measurement. Blood should not be collected in CaEDTA coagulants as the calcium will interfere with the testing procedure. Lithium heparin is acceptable. ESA manufactures a point-of-care lead analyzer (LeadCare, ESA, Chelmsford, MA, USA). The machine requires only a drop of blood and produces results in seconds vs. the usual wait time of 3–5 days from reference laboratories.

Treatment should first and foremost address physiologic support followed by elimination of absorbed lead from the blood and central nervous system (CNS) and of remaining pieces of lead from the gut. Supportive care will likely need to include parenteral fluids, possibly transfusion for severe anemia (eg, 12% PCV or less), thermal support, and careful reintroduction to alimentation.[2] For excretion of lead from the bloodstream, Calcium-Ethylene-diamine-tetra-acetic acid (CaEDTA) and Meso-2,3-dimercaptosuccinic acid (DMSA) are the mainstream options available in the United States. CaEDTA must be administered intramuscularly (30–50 mg/kg IM q12h PRN) as it is not well absorbed from the gastrointestinal tract.[3,4] Some authors have recommended rest periods of 2 days every 5 days of treatment. However, this appears to be unnecessary in raptors.[5] DMSA is administered orally (30 mg/kg PO q12h).[6]

Barbiturates

Barbiturate intoxication occurs in raptors that feed on animals euthanized with sodium pentobarbital.[7] The contaminated carcasses are typically pets, horses, or livestock that are not promptly buried or are left uncovered in public landfills. Symptoms will vary from an ataxic and drunken appearance to comatose and hypothermic to death. Other potential rule-outs include heavy metal intoxication, encephalomyelitis (eg, West Nile virus or algal toxin), or severe organophosphate poisoning. If the species involved is commonly known to eat carrion (eg, bald eagles, vultures, condors), there are several birds involved in a small setting, and/or the cases are occurring near livestock or landfills, then barbiturate exposure should be considered. Treatment consists of aggressive supportive care including parenteral fluids, thermal support, and oxygen or ventilator support in the worst cases. Several hours to a few days may be required for birds to recover consciousness at which point the long-term prognosis is generally good.

Cyanobacterial-induced avian vacuolar myelinopathy (AVM)

Avian Vacuolar myelinopathy is a lethal disease of herbivorous waterfowl and their avian predators. It was first recorded in bald eagles and American coots in 1994.[8] Occurrence appears to be increasing though the range of incidence appears to be confined to wintertime in lakes in the south to southeastern United States from Texas to the North Carolina. Where large numbers of waterfowl and eagles overwinter, the resultant die-offs can be spectacular.[8] A publication in 2005 identified a link between hydrilla (*Hydrilla verticillata*), an exotic aquatic weed, and a relatively rare epiphytic cyanobacterial species that is currently unnamed (assigned to the order Sitgonematales).[9] The disease has been replicated in waterfowl experimentally and is not found in lakes that lack this specific cyanobacterium. Incidence and range of the disease is expected to increase with the inevitable spread of hydrilla and its associated cyanobacteria.[8] Cyanotoxins are formed form secondary metabolites of the cyanobacteria and cause widespread vacuolation of the white matter of the brain and spinal cord. Affected birds (primarily described in waterfowl) may be reluctant to fly, wobble in flight, and exhibit ataxia on land.[10] Eagles are frequently found dead or close to death. Complete blood count and serum biochemistry results have been documented as normal. The disease is confirmed by histopathology of the brain or spinal cord. Lead intoxication

and pasteurellosis can produce similar presentations and also occur with some frequency in the wintertime. A complete blood count, hematocrit, and lead level are indicated to help rule-out these two conditions. History suggestive of ill eagles found in close association with concentrations of waterfowl may also be supportive of AVM. There is no known treatment. Because other diseases may cause similar symptoms, treatment should begin with aggressive supportive care, possibly adjunct chelation therapy, antimicrobials, and isolation of the affected bird.

Anticoagulant rodenticides

There are chronic (death occurs 1–2 weeks following lethal dose), single-dose (second-generation), and multiple-dose (first generation) rodenticides.[11] These poisons are mostly derived from coumarin and function primarily by interrupting the vitamin K cycle leading to failure to synthesize clotting factors I, II, VII, IX, and X) in the liver. Resistance to first-generation anticoagulant rodenticides (warfarin, pindone, coumafuryl, coumachlor, isovaleryl indanedione) began to be seen in the 1970s, so second-generation anticoagulant rodenticides (SGARs: brodifacoum, bromdiolone) were created that had 100–1000 times the potency and longer liver half-lives.[12] These newer poisons worked well because only a single feeding was needed and enough time elapsed (up to 8 days) post-consumption that rodents did not associate illness with the bait. Both generations present risk to secondary consumers and second-generation products have been found to be particularly likely to poison certain species of owls.[13,14] Symptoms in raptors include hemorrhage, pale mucous membranes, peripheral vascular collapse, shock, and weakness.[15] Normal platelet counts in the face of prolonged bleeding or clotting times and petechial hemorrhages is highly suggestive of anticoagulant rodenticide intoxication. Prothrombin time would be helpful for diagnosis but is generally not available for birds. Anticoagulant rodenticide screening tests are available and practical for use in raptors, but the risk of encouraging further hemorrhage from venipuncture must be considered. [15] Treatment consists of supportive care, blood transfusion in severe cases (author uses a PCV <15% as a guideline), and administration of vitamin K_1 (0.2–2.2 mg/kg IM q4–8h until stable, then q24h PO/IM for 14–28 days).[4,11,15]

Strychnine

In the US, strychnine use is strictly limited by the Environmental Protection Agency to below-ground control of pocket gophers. However, it has a history of use as an avicide and is still available outside the US.[16] Because of its fast mode of action and rapid excretion, it is not considered a likely secondary intoxicant in applications for underground rodents.[13, 17] However, when used as an avicide, target species may be sufficiently incapacitated to be easy prey and still contain unabsorbed quantities of strychnine in their gastrointestinal tract.[18] Signs include hyperexcitability and convulsions. Sublethal doses may cause changes in foraging behavior.[16]

Avicides

Starlicide (synonyms: DRC-1339, 3-chloro-p-toluidine HCl, 3-chloro-4-methylaniline HCl, 3-chloro-4-methylbenzenamine HCl): This is a slow-acting avicide used to control ravens, starlings, crows, pigeons, cowbirds, grackles, magpies, and gulls.[19,20] It is the only avicide currently licensed by the EPA and is authorized for use only by trained US Department of Agriculture (USDA) personnel. Its risk to predators and scavengers is recognized by the EPA.[21] In target species, death occurs 1–3 days post-ingestion by irreversible kidney and heart damage (from methemoglobinemia) and CNS depression (cardiac and respiratory arrest).[20,22,23] Risk to raptors has been considered low because of its milder mode of action (primarily CNS depression), with the exception of owls.[20] It is rapidly (90% in 2 hours) metabolized and excreted in all species.[20] No treatments are described in the literature.

Avitrol (4-aminopyridine; 4AP): This is used to kill blackbirds, sparrows, pigeons, crows, and other passerines. It's reregistration as a Restricted Use Pesticide with the EPA is pending. Birds demonstrate convulsions and involuntary vocalizations 10–30 minutes post-ingestion, death following up to 4 hours later.[18] 4AP is rapidly excreted or metabolized and is, therefore, non-cumulative in birds and mammals. In Schafer, 1984, the risk to raptors was identified but considered very low.[18]

Alpha-chloralose: This is licensed in the US as a sedative for capture of wild birds. It is also sometimes used as an avicide for sparrows, pigeons, and crows.[24] Mental depression, dehydration, and hypothermia are seen with intoxication of raptors. Treatment should include thermal and fluid support until the poison is metabolized and excreted.

Others: Fenthion is an organophosphate insecticide and avicide has largely been removed from use in the United States. In 2001 it was defined as a "Restricted Use Pesticide" by the EPA and was only approved for use as a mosquiticide in Florida.[25] In 2003, Bayer voluntarily canceled its registrations for fenthion and related compounds.[26] Rid-A-Bird (endrin) is no longer available in North America.

Pesticides

Organochlorines: Organochlorines (endrin, DDT, chlordane) have largely been banned in North America but their use continues in other parts of the world. Organochlorines, such as DDT, are extremely persistent in the environment and can still be detected in wildlife. Acute intoxication with organochlorines produces tonic-clonic convulsions, mental depression, ataxia, and the appearance of blindness.[27] Birds may also be emaciated and anemic. Anti-seizure medications (eg, diazepam) or barbiturates may be of benefit. Organochlorine intoxication is identified by residues in brain tissue although residues can also be found in body fat and blood. Interpretation can be difficult if intoxication studies have not been completed in the species in question, although Porter, 1993, describes a method for estimating "DDT equivalents," which could be helpful in assessing whether levels were acutely toxic. Low-level organochlorine intoxication adversely affects reproductive success. DDT is particularly well-recognized for its thinning effect on eggshells which led to poor hatchability.

Organophosphates and carbamates: These are neurotoxins which inactivate the enzyme acetylcholinesterase (AChE) leading to continuous stimulation and fatigue of muscles and cholinergic end-organs.[27] Respiratory arrest follows. Environmental and biologic persistence is not as great as organochlorines. Symptoms include ataxia, opisthotonus, muscle twitches, salivation, tachypnea, mental dullness, clenched feet, and fluttering of the nictitating membrane. Atropine sulfate is recommended at 0.2–0.5 mg/kg IM/IV q3–4h. If the dose is sufficient, results should be seen within 15 minutes. Other supportive care should also be provided as indicated.

Neoplasms of Raptors (Ford)

Neoplasia is uncommonly diagnosed in free-ranging wild raptors.[28] This could be because the birds typically die and are not discovered or they succumb to injuries, degenerative changes, and harsh environmental conditions before they reach ages where the incidence of neoplasia becomes significant. Reviews of admissions to wildlife treatment facilities have also revealed a low incidence of cancer.[29,30] Raptors kept in captivity can probably be expected to demonstrate a higher incidence of neoplasia diagnosed because of the increased vigilance of their health and because of their increased longevity. Still, there is a dearth of published reports of neoplasia in raptors. Forbes et al (2000) and Tristan (2010) (summarized in Table 1) provide the most comprehensive reviews of neoplasms reported in raptors.[31.32] Relatively few treatments of

neoplasia in raptors are found in the literature. Heatley, 2007, described successful resolution of a squamous cell carcinoma in a golden eagle utilizing excision and Strontium-90 radiation.[33]

[Table 1 around here]

Teratomas

A teratoma is a neoplasia that includes tissues derived from at least 2 of the 3 most primitive cell-lines of a vertebrate organism: mesoderm, endoderm, or ectoderm. The tumors can arise in a variety of locations, though in mammals they most typically arise from gonadal tissue. Teratomas have been reported in a bald eagle and in a peregrine falcon (S. F., written communication, November 2010).[34] The bald eagle was approximately 1 year old and the peregrine was approximately 3 months old. In both cases, a firm, distended abdomen, tachypnea, and weakness were common components. Radiographs revealed large soft tissue-density masses and displaced viscera. The peregrine demonstrated polycystic tissue of mixed echogenicity in the abdomen. The bald eagle was moribund upon admission and died within 2 hours, despite aggressive supportive care measures. The most significant bloodwork abnormalities of the bald eagle were severe anemia and severe hypoproteinemia, which were probably due to chronic malnourishment. The peregrine's bloodwork results were unremarkable except for elevated creatinine phosphokinase (CK: 1678 U/L) and hyperuricemia (30.5 mg/dl). Abdominocentesis of the peregrine was helpful for alleviating respiratory distress but was not very helpful diagnostically (low cellularity, modified transudate). The bald eagle died spontaneously whereas the peregrine was euthanized based upon recognition of a large, inoperable mass on ultrasonic and radiographic images. In both cases, teratoma was suspected based upon gross appearance—a singular, large, polycystic, irregular mass composed of whorls of tissue of varying density and color, centered in the cranial abdomen. Both were confirmed by histopathology. In other species, complete excision has been reported as a successful treatment. This could have been true of these 2 cases in raptors provided the lesions were discovered while still of feasible dimensions for surgical removal.

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There are 38 references for this section. Due to space restrictions, references are offered separately at www.alaskabirddoc.com or by e-mailed request to akeaglevet@yahoo.com.

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Table 1. Neoplasms of raptors reported in the literature, ranked by frequency.

Neoplasm	n (%)	Species	Age (yrs)	Ref
Adenocarcinoma[a]	18 (15%)	Multi, BAEA, GOEA	6–26	32, 35,38
Fibrosarcoma	17 (14%)	Multi	0.5-12	32
Squamous cell carcinoma[b]	14 (12%)	Multi, GOEA	4–15	32, 36
Papilloma	6 (5%)	Multi	0.5-9	32
Carcinoma	5 (4%)	Multi	1.5-2	32
Lipoma	4 (3%)	Multi	9–16	32
Lymphoid leukosis	4 (3%)	Multi	0.25-3	32
Malignant lymphoma	4 (3%)	Multi	>8–18	32
Osteoma/chondroma	4 (3%)	Multi	6–15	32
Bile duct carcinoma	3 (3%)	Multi	13–33	32
Lymphosarcoma	3 (3%)	Multi	22	32
Mast cell tumor	3 (3%)	Multi	U	32
Melanoma	3 (3%)	Multi	10–31	32
Teratoma	3 (3%)	EUBU, BAEA, PEFA	10	32,34
Xanthoma	3 (3%)	Multi	2->40	32
Adenoma	2 (2%)	Multi	7–20	30,32
Cholangiocarcinoma[a]	2 (2%)	RTHA, GOEA	U	32,38
Epidermoid carcinoma	2 (2%)	RTHA	U	32
Histiocytic carcinoma	2 (2%)	GHOW	<1y, Ad	32
Leiomyoma[c]	2 (2%)	GOEA, PEFA	17	37
Mesothelioma	2 (2%)	FEHA	4–5	32
Osteosarcoma	2 (2%)	Xfalc, EUBU	U, Ad	32
Adrenal cortical adenoma	1 (1%)	LCEA		32
Astrocytoma	1 (1%)	GHOW		32
Cystadenocarcinoma[c]	1 (1%)	PEFA	U	37
Erythoblastosis	1 (1%)	GYRF	U	32
Fibroma	1 (1%)	EUKE	U	32
Hemangioma	1 (1%)	PEFA	15	32
Malignant thymoma	1 (1%)	SAFA	12	32
Mixed cell tumor	1 (1%)	SEKE	30 days	32
Myxofibroma	1 (1%)	CGVU	17	32
Rhabdomyosarcoma	1 (1%)	LFVU	19	32
Thyroid cystic fibroadenoma	1 (1%)	BCBE	>27	32
Thyroid follicular cystadenoma	1 (1%)	CRCA	7	32
Total Cases	120			

[a] One GOEA: metastatic cholangiocellular carcinoma and renal adenocarcinoma
[b] Reportedly "common" in HAHA in UK. Also, 1 GOEA: unpub. data from author's records
[c] One PEFA: cystadenocarcinoma and leiomyoma
Key: Multi=Multiple; EUBU=European buzzard (*Buteo buteo*); RTHA=Red-tailed hawk (*Buteo jamaicensis*) BAEA=Bald eagle (*Haliaeetus leucocephalus*); PEFA=Peregrine falcon (*Falco peregrinus*); GOEA=Golden eagle (*Aquila chrysaetos*); HAHA=Harris' hawk (*Parabuteo unicinctus*);

GHOW=Great horned owl (*Bubo virginianus*); FEHA=Ferruginous hawk (*Buteo regalis*); LCEA=Long-crested eagle (*Lophaetus occipitalis*); GYRF=Gyrfalcon (*Falco rusticolus*); EUKE=European kestrel (*Falco tinnunculus*); SAFA=Saker falcon (*Falco cherrug*); SEKE=Seychelles kestrel (*Falco araea*); Cape griffon vulture (*Gyps coprotheres*); LFVU=Lappet-faced vulture (*Torgos tracheliotos*); BCBE=Black-chested buzzard eagle (*Geranoaetus melanoleucus*); CRCA=Crested caracara (*Caracara cheriway*); U=Unspecified; Ad=Adult