CHAPTER
31

Implications of Toxic Substances in Clinical Disorders

JILL A. RICHARDSON, DVM

Greg J. Harrison

Fig 31.1 | A lutino cockatiel with excessive yellow feather pigmentation commonly seen in nutritional disorders. Helga Gerlach first proposed that consumption of foreign bodies is mediated by nutrient imbalances. Since that time, the use of formulated diets has become much more prevalent. Practitioners have empirically noted a concomitant and precipitous decline in foreign body ingestion.

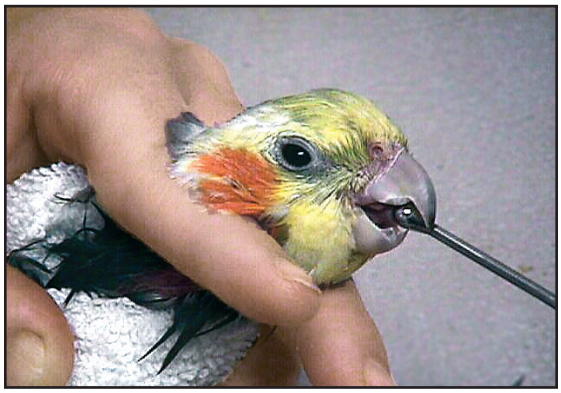
Birds are curious in nature and certain dangerous objects may be attractive to them (Fig 31.1). As most pet birds have clipped wings, remain caged or have limited activity outside their cages, toxicosis are not common. However, birds with free household access or free-ranging birds are most at risk of becoming exposed to toxicants. With any potential toxicosis, proper and prompt treatment including stabilization and decontamination is essential. Supportive care is also a critical factor in the full recovery of the bird.

Assessing the condition of the bird is the initial step in managing potential toxicosis. This assessment should be performed quickly and in a manner that will limit stress to the bird. Sedation with isoflurane or sevoflurane gas anesthesia may be required to limit stress or when dealing with fractious birds. Toxic birds are prone to both active and passive regurgitation, and care must be taken to prevent aspiration. To help decrease stress, birds should be examined and maintained in a quiet, warm environment.[1] The assessment should include an evaluation of the respiratory rate and effort, capillary refill time and general attitude (see Chapter 6, Maximizing Information from the Physical Examination). The examination of a bird that is unconscious, in shock, seizuring or in cardiovascular or respiratory distress must be conducted simultaneously with stabilization measures (see Chapter 7, Emergency and Critical Care).

Stabilizing the bird is a life-saving priority.[1] Once the animal is stabilized, a comprehensive history of the bird including exposure history can be obtained. Common presentations of birds that require stabilization include dyspnea, cyanosis, severe depression, emaciation, severe diarrhea, seizures and hemorrhage.[1] Minimal handling is imperative with dyspneic birds in order to decrease

Gwen Flinchum

Fig 31.2 | Fluids being given subcutaneously to a toxic duck.



Greg J. Harrison

Fig 31.3 | Lavaging the digestive system with warmed saline via a crop tube. If lavage is contraindicated, the liquids can be used to dilute toxins and/or aspirate fluids in the crop that may contain toxins

oxygen requirements. Dyspneic birds should be placed in a cage supplemented with oxygen before and during examination.[1] A diuretic may be indicated with the presence of pulmonary edema.[1]

Anti-convulsant therapy such as diazepam at 0.6 mg/kg IM should be given if the bird is seizuring.[7] Intravenous, intraosseous or subcutaneous fluids may be needed if the bird is severely dehydrated (Fig 31.2).

Decontamination

Preventing absorption of the substance is an important step in treating a toxicosis.

DERMAL EXPOSURES

With light dermal exposures, the bird can be gently spritzed with a solution of mild liquid dishwashing detergent and warm water, softly rubbed and then spritzed with plain warm water to remove soap. This process can be repeated as needed, making sure all soap is removed.

A thorough bathing may be indicated with heavy exposures. Following the bath, the bird should be lightly patted dry, kept warm and monitored for signs of hypothermia. Removal of the toxicants from the feathers is contraindicated if the bird is seriously ill; always stabilize the patient first. With corrosive or irritating substances, the bird's skin should be monitored for redness, swelling or pain.

OCULAR EXPOSURES

With ocular exposures, the bird's eyes should be gently flushed with tepid tap water or with physiologic saline. The use of an eyedropper to gently administer the flush is recommended in small birds. Fluorescein staining and follow-up examinations are warranted with exposures to corrosive agents or if clinical signs of redness, pain or ocular discharge occur.

ORAL EXPOSURES

Dilution with milk or water in combination with demulcents is recommended in cases of corrosive ingestion. Close monitoring is recommended following ingestion of corrosive agents, which can lead to tissue necrosis and inflammation of the mouth, esophagus and crop. Severity of injury depends on the concentration and duration of contact.

Never induce emesis in a bird. Emesis is considered unsafe in birds, due to the potential of aspiration and ineffectiveness of emetic medications.[10] Crop lavage may be considered with recent ingestion of toxicants (Fig 31.3). Contraindications to performing a crop lavage include ingestion of corrosive substances or petroleum distillates. With ingestion of corrosive agents, gastric lavage is not recommended.[3,4] Instead, oral dilution with milk or water is preferred.[3,4] Dilution is most effective if it is performed early. Sedation is recommended for frightened or fractious birds. Isoflurane or sevoflurane gases are the optimal anesthetic agents, and an endotracheal tube should be inserted during the process to prevent aspiration. To lavage the crop, body-temperature saline is gently flushed into the crop and aspirated repeatedly (3-4 times).[10]

Activated charcoal is considered a non-specific adsorbent that binds to many substances through weak forces, and prevents their systemic absorption. It is not an effective adsorbent for corrosive substances, petroleum distillates or heavy metals.[3,4,6,15] Activated charcoal can be given to birds with a dosing syringe, an eyedropper or lavage tube, although extreme caution must be used to avoid aspiration. Dosage of activated charcoal in most species

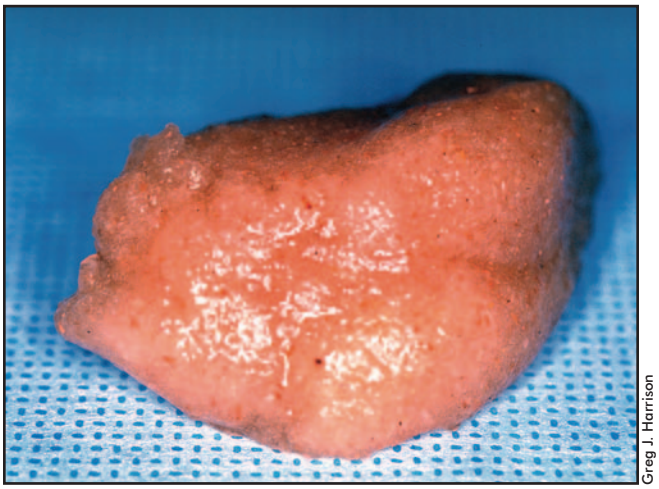

Greg J. Harrison

Fig 31.4 | Psyllium is high in mucopolysaccharides and forms a slick mass that can sweep the digestive tract free of a multitude of toxins. Using more than 2% concentration can lead to gastrointestinal blockage by this hydroscopic mass. Given with balanced electrolytes and dextrose it will simultaneously act to rehydrate the patient.


Greg J. Harrison

Fig 31.5 | Inexpensive pet carriers are often made from hardware cloth. This metal wire is always toxic to chewers as it is high in lead and zinc. This 27-year-old Amazon traveled safely in such a carrier its entire life. The owner was lucky this did not kill the bird.

is 1 g/kg (or 1-3 mg/g body weight).[3,4]

Cathartics are substances that enhance the elimination of activated charcoal, but should be used cautiously in birds. Cathartics can be added to solutions of activated charcoal, or premixed combinations are available. Never use cathartics when the bird is dehydrated. Bulking agents can be useful in removing small solid objects from the bird's gastrointestinal tract, such as lead paint chips. One half teaspoon of psyllium (Fig 31.4) mixed with 60 cc baby food gruel has been suggested as a bulking agent for birds, and can be administered with a dosing syringe or eyedropper.[10] Use extreme caution to avoid aspiration. The mixture may be repeated to ensure complete removal of the objects. Peanut butter also has been recommended as a bulking agent.[10]

The bird should be monitored closely during treatment. Routinely evaluate vital signs and the parameters most likely to be affected. Preventive measures such as gastric protection or antibiotics may be needed. Additional measures such as nutritional and hydration support are key components for full recovery. Daily maintenance fluid requirements in most birds are 50 ml/kg per day.[1] It is extremely important to maintain the bird's nutritional requirements. Hospitalized birds eating voluntarily can be fed their normal diet. Tube-feed ill birds that are not eating, unless vomiting or delayed emptying of the crop is present.[1] Good nursing care should be given until the bird completely recovers. The propensity for hypothermia in a bird that is ill for any reason should be considered and external heat and humidity provided as required.

Common Hazards

HEAVY METALS

Zinc

Sources of zinc include hardware such as wire (Fig 31.5), screws, bolts, nuts, and USA pennies. Pennies minted since 1983 contain 99.2% zinc and 0.8% copper and one penny contains approximately 2440 mg of elemental zinc.[15] The process of galvanization involves the coating of wire or other material with a zinc-based compound to prevent rust. Owners often are not aware of galvanization on the wire used for making cages (Fig 31.6). Food and water dishes also may be galvanized and sufficient zinc may leak into the water or food to create toxicity.

(Ed. Note: In tracking cases of zinc toxicity over 7 years, computer records indicate that in 82 cases of zinc toxicity, approximately half of the clients denied any potential exposure of their birds to zinc or other heavy metal. The idea that a bird out of its cage is truly "supervised" is overrated. Also, powder-coated cages made outside of the USA, especially in China, have been known to use zinc to expedite setting of the powder coating [F. VanSant, personal communication, 2000]).

Although the exact toxicologic mechanisms of zinc in birds or other animals is not known, zinc toxicosis can affect the renal, hepatic and the hematopoietic tissues. Clinical signs of zinc toxicosis in birds may include polyuria, polydipsia, diarrhea, weight loss, weakness, anemia, cyanosis, seizures and death.[2,16] An underreported clinical sign of zinc toxicity is polydipsia with

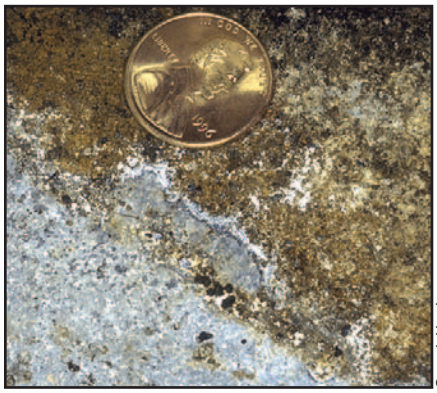

Greg J. Harrison

Fig 31.6 | Two sources of zinc: a USA 1-cent coin and a galvanized metal cage tray. The zinc in the tray has oxidized to its most toxic form — a pure white powder — evident at the margin of the rust and the galvanization.


Greg J. Harrison

Fig 31.7 | Placing a bird unanesthetized in a paper bag is often a safe and accurate way to diagnose metal toxicosis.


Greg J. Harrison

Fig 31.8 | Lead used to balance automobile wheels and fishing weights are modern sources of lead toxicosis.

passive regurgitation of water when the bird is handled. Mild anemia also is frequently encountered. Concurrent marked elevations in the total WBC are noted in the majority of cases of heavy metal toxicosis. Whether this is a true infection or an inflammatory response is not documented, so treatment with antibiotics will depend on the practitioner's evaluation. However, the presence of a high WBC should not convince the practitioner that the discovery of metal in the ventriculus is not the primary etiology of illness.

Diagnosis

Radiography of the abdomen may reveal the presence of metallic objects in the gastrointestinal tract. This need not be a properly positioned radiograph, but rather can be done as a stress-free scan for the presence of metal in the ventriculus (see Chapter 1, Clinical Practice for "bag rad" scan) (Fig 31.7). Serum zinc levels may be obtained using blood collected from plastic syringes (no rubber grommets) and stored in royal blue-top vacutainers or directly into vacutainers with appropriate needle to minimize contamination with exogenous zinc.[15] In general, blood zinc levels of >200 μg/dl (2 ppm) are considered to be diagnostic.[16] The pancreas is considered to be the best tissue for postmortem zinc analysis.[2,16] Pancreatic tissue zinc levels greater than 1000 μg/g are suggestive of a zinc toxicosis.[16]

Treatment

It is imperative to remove the sources of zinc from the gastrointestinal tract. Removal of zinc-containing foreign bodies via endoscopy or proventriculotomy/enterotomy may be required. The success of the removal process can be assessed with radiographs. Since most zinc items swallowed by pet birds are galvanized iron, the use of magnets attached to an enteral tube is an effective means of removing ferrous items that are zinc coated[12] (see also Chapter 14, Evaluating and Treating the Gastrointestinal System). Activated charcoal is not indicated, as it is of little benefit in binding zinc.[15] Bulk cathartics, psyllium (sodium sulfate 125-250 mg/kg), peanut butter, mineral oil and corn oil may aid in the removal of zinc objects from the GI tract. The use of chelators may not be necessary in cases where prompt removal of the zinc source is accomplished. If chelation therapy is instituted, careful monitoring of renal parameters is important for the duration of therapy. Elevated uric acids in heavy metal poisoning and a decrease with therapy have been reported (E. Odberg, personal communication, 2001). The following chelating agents have been suggested for zinc poisoning in birds: Ca EDTA 35 mg/kg BID, IM for 5 days.[7] If needed, the second course of therapy is given after a 5- to 7-day waiting period. If/when the bird is able to tolerate oral medication, D-penicillamine (Cuprimine) can be administered orally at 55 mg/kg BID PO for 1 week.[7] A second course of 1-week therapy can be given, if needed, after a 1-week rest. Succimer, (2, 3 dimercaptosuccinic acid) at 25 to 35 mg/kg for 5 days per week for 3 to 5 weeks also has been used to treat zinc toxicosis in birds.[7] In addition, treatment for symptomatic animals should include blood replacement therapy as needed, parenteral fluids and good nursing care such as force-feeding or hand-feeding.

Lead

Sources of lead include paint, toys, drapery weights, linoleum, batteries, plumbing materials, galvanized wire, solder, stained glass, fishing sinkers (Fig 31.8), lead shot, foil from champagne bottles and improperly glazed bowls.[4,16] Lead is considered to be the most commonly reported of avian toxicosis with acute toxicities more common in captive birds and chronic in free-ranging birds (Fig 31.9).


Greg J. Harrison

Fig 31.9 | Pelicans are presented with fishing lines and hooks swallowed or tangled in their extremities. Radiographs for lead are always indicated to ensure a bird with a lead weight in its digestive system is not released.


Greg J. Harrison

Fig 31.10 | Peregrine falcons are carnivorous and can consume prey that were shot with lead-containing pellets. Radiographs are indicated.


Greg J. Harrison

Fig 31.11 | Blue-winged teal and other water birds can dabble in tidal marshes or ponds that may contain decades-old hunting remnants from lead shot.

Raptors can ingest lead shot from preying on animals that have been shot with or have ingested lead shot (Fig 31.10).[20] Lead toxicosis also has been documented in an Amazon parrot that had been fed portions of game birds that contained lead shot.[18] Between 1983 and 1986, the National Wildlife Health Center examined 1041 moribund or dead waterfowl and diagnosed lead poisoning in approximately 40% (Fig 31.11).[5] Although lead shot has since been banned for hunting waterfowl, spent shot is still present in waterways.[20] Ingestion of 1 to 3 lead shotgun pellets has been reported to be lethal to waterfowl.[4]

Lead affects multiple tissues, especially the gastrointestinal tract, renal and nervous systems. Lead combines with erythrocytes in circulating blood, increasing RBC fragility, anemia and capillary damage. It also can cause segmental demyelination of neurons and necrosis of renal tubular epithelium, GI tract mucosa and liver parenchyma. Clinical signs seen in psittacine birds are often vague and may include lethargy, weakness, anorexia, regurgitation, polyuria, ataxia, circling and convulsions.[4] In some species such as Amazons, hemoglobinuria also may be noted.[11]

Diagnosis

Radiography of the abdomen may reveal evidence of metal in the ventriculus. Blood levels of lead are helpful in confirming lead toxicosis in birds with suspicious radiographic changes.[19] Whole blood levels greater than 0.6 ppm are viewed as diagnostic for lead toxicosis when accompanied by appropriate clinical signs.[19] The basophilic stippling and cytoplasmic vacuolization of red blood cells are not always seen with lead poisoning in avian species.[19]

Treatment

Removal of lead particles via bulk diet therapy, endoscopy or surgery is recommended. Succimer and Ca EDTA are both considered to be effective chelating agents in avian species.[4] Succimer has been reported to decrease blood lead concentration by 87% when given at a dose of 30 mg/kg PO BID for 7 days minimum, with no apparent adverse secondary effects, however, a dose of 80 mg/kg resulted in death.[9] The therapeutic dose of succimer in pet birds is 25 to 35 mg/kg PO BID 5 days a week for 3 to 5 weeks.[7] Calcium EDTA is considered the preferred initial chelator for lead toxicity in birds and is given at a dose of 35 mg/kg BID, IM for 5 days, off 3 to 4 days, and repeated if needed.[7] Fluid therapy is recommended to prevent renal effects from Ca EDTA during treatment.[11] Penicillamine and diethylene triamine pentaacetic acid (DTPA) have also been used to treat avian lead toxicosis.[7]

Since lead can be immunosuppressive, broad-spectrum antibiotics may be indicated.[4] In addition, good supportive care including seizure control is recommended until full recovery.

Nicotine Products

Tobacco products contain varying amounts of nicotine (Table 31.1), with cigarettes containing 3 to 30 mg and cigars containing 15 to 40 mg.[15] Butts contain about 25%

Table 31.1 | Nicotine Content of Common Sources of Nicotine

Nicotine Product	Nicotine Content
Cigarettes	3-30 mg per 1 whole cigarette
Cigarette butts	.75-7.5 mg
Cigars	15-40 mg
Moist snuff	4.6-32 mg/g
Dry snuff	12.4-15.6 mg/g
Chewing tobacco	2.5-8.0 mg/g
Nicotine gum	2-4 mg per piece
Transdermal patches	15-114 mg per patch
Nicotine nasal sprays	10 mg per ml
Nicotine inhaler rods	10 mg per cartridge

of the total nicotine content. Nicotine also is found as a natural form of insecticide. Signs develop quickly in most species, usually within 15 to 45 minutes, and include excitation, tachypnea, salivation and emesis. Muscle weakness, twitching, depression, tachycardia, dyspnea, collapse, coma and cardiac arrest may follow. Death from nicotine toxicosis occurs secondary to respiratory paralysis.[15] A less serious but common response to cigarette smoke deposition on the feathers is feather-destructive behavior.

(Ed. Note: One timneh grey that expired at 21 years of age reportedly had lived its entire life with a heavy smoker. The histopathologic diagnosis of multiple masses in the lungs was carcinoma, but was not definitely labeled as bronchiogenic).

Inhalants

The avian respiratory tract is extremely sensitive to inhalants. Any strong odor or smoke could potentially be toxic (Table 31.2).[17] Polytetrafluoroethylene (PTFE)-coated cookware or cooking utensils can emit toxic fumes when overheated (>280° F).[17] Clinical signs may include acute death, rales, dyspnea, ataxia, depression and restless behavior.[2,10] Hemorrhage and edema in pulmonary tissues leads to respiratory failure and death. Prognosis is usually guarded to poor. Treatment for inhalation toxicosis includes the administration of oxygen, rapidly acting corticosteroids, diuretics, analgesics, parenteral antibiotics and topical ophthalmic antibiotic ointment.[1] A bronchodilator may be needed for bronchospasms[1] (see Chapter 7, Emergency and Critical Care for an updated therapy). In most cases, prognosis is guarded to poor.*

**Ed. Note: The first-time heating of several new non-stick pans is a frequent finding with PTFE toxicosis. One empirical report (Beckett, personal communication, 2001) had a bird indirectly exposed days later when a wooden perch had been "sterilized" on a PTFE-coated metal cooking sheet and the perch was then later placed in the bird's cage.*

Table 31.2 | Examples of Noxious Inhalants

- Some non-stick surfaces (pots, pans, cookware, irons, ironing boards)
- Heating elements on reverse cycle air conditioners
- Gasoline or other volatile gas fumes
- Any source of smoke
- Automobile exhaust
- Carbon monoxide
- Self-cleaning ovens and drip pans for ranges
- Aerosol sprays
- Cleaning products such as ammonia or bleach
- Paint fumes
- Fumigants
- Candles with lead wicks, scented plug-in items

AVOCADO (*Persea americana*)

The toxic principle in avocado is persin, and leaves, fruit, bark and seeds of the avocado have been reported to be toxic to birds and various other species.[10,15,17] Several varieties of avocado are available, but not all varieties appear to be equally toxic. In birds, clinical effects seen with avocado toxicosis include respiratory distress, generalized congestion, hydropericardium, anasarca and death.[10,17] Onset of clinical signs usually occurs after 12 hours of the ingestion, with death occurring within 1 to 2 days of the time of exposure.[10] Small birds such as canaries and budgies are considered to be more susceptible, however, clinical signs have been observed in other species. Treatment for recent avocado ingestion includes decontamination via crop lavage and activated charcoal; bulking diets may help prevent absorption. Close monitoring for cardiovascular and pulmonary signs should follow. With symptomatic animals, treatment with humidified oxygen and minimal handling may be required. Diuretics may be helpful in cases with pulmonary edema.[4]

POISONOUS PLANTS

The following is a partial list of plants that have been shown to cause toxicity in small animals. The severity of signs or toxicity of these plants in birds has not been thoroughly studied.

Potentially Cardiotoxic Plants

- Lily of the valley (*Convallaria majalis*)
- Oleander (*Nerium oleander*)
- Rhododendron species
- Japanese, American, English and Western yew (*Taxus* spp.)
- Foxglove (*Digitalis purpurea*)
- Kalanchoe species
- Kalmia species

Plants That Could Cause Kidney Failure

- Rhubarb (*Rheum* spp.) - leaves only

Plants That Could Cause Liver Failure

- Cycad, Sago, Zamia palms (*Cycad* spp.)
- *Amanita* mushrooms

Plants That Can Cause Multisystem Effects

- Autumn crocus (*Colchicum* sp.)
- Castor bean (*Ricinus* sp.)

Plants Containing Calcium Oxalate Crystals

Peace lilies, Calla lilies, philodendrons, dumb cane,


Greg J. Harrison

Fig 31.12 | Houseplants need to be considered as potential toxicants, especially in birds with livers stressed by nutritional disorders.


Greg J. Harrison

Fig 31.13 | A poinsettia plant growing wild in the rare species breeding aviary in Tenerife, Spain.


Greg J. Harrison

Fig 31.14 | A lantana plant in the same aviary as in Fig 31.13.


Greg J. Harrison

Fig 31.15 | A budgie has been oiled by an ill-informed owner.

mother-in-law, and Pothos plants contain insoluble calcium oxalate crystals. These crystals can cause mechanical irritation of the oral cavity and tongue of birds when plant material is ingested. Clinical signs usually include regurgitation, oral pain, dysphagia and anorexia. The signs are rarely severe and usually respond to supportive care.

- Peace lilies (*Spathiphyllum* spp.)
- Calla lily (*Zantedeschia aethiopica*)
- Philodendron (*Philodendron* sp.)
- Dumb cane (*Dieffenbachia* sp.)
- Mother-in-law plant (*Monstera* sp.)
- Pothos (*Epipremnum* sp.)

(Ed. Note: A common presentation in cockatiels appears to be oral irritation from ingestion of small amounts of Pothos or philodendron species of plants (Fig 31.12)*. In this editor's practice, more than 15 cockatiels have presented over the course of 20 years with documentation of recent chewing on leaves of these plants and almost immediate production of clinical signs. The birds appear acutely depressed and anorectic, but still in good body weight. Examination of the tongue will reveal pronounced erythema, sometimes with obvious ulceration, and hypersalivation. Supportive care for 24 to 48 hours has resulted in 100% recovery. Lack of mortality also has lead to a lack of histopathology, so any additional toxic effects other than oral irritation have not been documented [TTL]).*

Aviculturists need to be sure potentially toxic plants are avoided in the plantings of the aviary (Figs 31.13, 31.14).

OIL-CONTAMINATED BIRDS

Oil spills are not an uncommon problem for aquatic species of birds. According to California's Oiled Wildlife Care Network, bird survival is dependent upon many factors, including the speed of recovery and the species' susceptibility to toxicity and captive stress.[13] The first step when treating oiled birds is to stabilize the animal and provide a warm (approximately 27° C) and stress-free environment.[13] Common presenting clinical signs include respiratory distress and seizures.[13] Following initial stabilization, a thorough exam should be performed. Most affected birds are hypothermic, hypoglycemic, hypoproteinemic and lethargic on presentation.[13] Anemia also has been reported.[13,14] Symptomatic care including nutritional support should be provided as needed.


Greg J. Harrison

Fig 31.16 | When presented to a rehabilitation center, an insectivorous raptor, the burrowing owl, should have pesticide toxicity investigated to document this common but seldom proven condition.


Greg J. Harrison

Fig 31.17 | The insectivorous passerine, such as this painted bunting, is seldom seen when ill. When presented for rehabilitation, pesticide toxicity should be investigated.

With oil contamination, feathers lose the ability to insulate, which can result in hypothermia[1] (Fig 31.15). Oil also can interfere with the animal's buoyancy.[13] Oil can be removed from the feathers once the animal is stable using dishwashing detergent in warm baths. The temperature of water used should be 106° F, and water should be softened to 2 to 3 grains of hardness to help completely remove oil and prevent mineral crystallization in the feathers.[13] Following thorough rinsing, the bird must be placed in a warm environment and allowed to dry. Multiple baths may be needed, however, repeat washings because of incomplete oil or soap removal are associated with increased mortality.[13] Other recommendations for care include the use of lactulose at 0.3 ml/kg PO q 12 h, papaya enzymes, 1 tablet PO q 12 h, aggressive fluid therapy for feather-eating species and warm-water exercise pools.[13]

Editor's Comments

While documentation of environmental toxins is hard to prove as the cause of death in wild birds, one must strongly suspect pesticides when seeing insectivorous birds like burrowing owls (Fig 31.16), the painted bunting (Fig 31.17) and related birds presented to rehabilitation centers with clinical signs consistent with toxicity. When the literature on pesticides is studied (see reference 9 in Chapter 11, Low-Risk Pest Management), it seems rather obvious the veterinary profession is commonly missing pesticide toxicosis.

In 1999, a massive die-off of white pelicans (*Pelecannus erythrorhynchos*), wood storks (*Mycteria americana*), great egrets (*Ardea albus*) and great herons (*Ardea herodias*) occurred on the shore of Lake Apopka in Central Florida. The University of Florida and USA Fish and Wildlife eventually detected the chemicals DDT, toxaphene and dieldrin in lethal levels in these fish-eating birds. These pesticides are believed to be carcinogenic and were banned in the 1970s and 1980s; however, the chemicals can persist for decades in soil and animal tissue.

It was the nation's worst pesticide poisoning in decades. Over 800 documented great white pelicans were killed by this toxic exposure, and the extrapolated number of dead created great concern for the survival of this species in North America. However, political and legal concerns kept the problem from being widely publicized.

Pesticides have tremendous residual potential. Their prolonged half-lives, combined with the general lack of both infrastructure and funding for testing and detection of pollutants, make it likely that a great deal more exposures to toxic pesticides will occur in many species than is suspected or reported.

As veterinarians, we have an opportunity to be cognizant of this dangerous potential, to appropriately diagnose and treat these toxicities in individual birds, and also to report suspected toxicities to responsive authorities, requesting and expecting an appropriate investigative response.

References and Suggested Reading

1. Agnes AE: Critical Care of Pet Birds: Procedures, Therapeutics, and Patient Support. *In* Veterinary Clinics of North America, Exotic Animal Practice. Philadelphia, WB Saunders Co, 1:1 1998, pp 11-42.
2. Bauck L, LaBonde J: Toxic diseases. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 604-613.
3. Beasley VR., Dorman D: Management of toxicosis. Vet Clin North Amer 20:2, 1990, pp 307-338.
4. Beasley VR, et al: A Systems Affected Approach to Veterinary Toxicology. Urbana, IL, University of Illinois Press, 1999, pp 27-69.
5. Beyer WN, et al: Retrospective study of the diagnostic criteria in a lead-poising survey of waterfowl. Arch Environ Contam Toxicol, 35(3):506-512, 1998.
6. Buck WB, Bratich PM: Activated charcoal: Preventing unnecessary death by poisoning. Food Animal Practice. Veterinary Medicine January 1986:73-77.
7. Carpenter JW, Mashima TY, Rupiper DJ: Exotic Animal Formulary 2nd ed. Philadelphia, WB Saunders Co, 2001.
8. Dumonceaux G, Harrison G: Toxins. *In* Avian Medicine: Principles and Application. Brentwood, TN, HBD International Inc, 1999, pp 1030-1052.
9. Hoogesteijn AL, et al: Oral treatment of avian lead intoxication with meso-2,3-dimercaptosuccinic acid. J Zoo Wildl Med 34(1):82-87, 2003.
10. LaBonde J: Toxicity in Pet Avian Patients. Semin Avian Exotic Pet Med 4(1):23-31, 1995.
11. Lightfoot TL: Common Avian Medicine Presentations II. Proceeding Notes. Western Veterinary Conference 2002.
12. Lumeij JT: Gastroenterology. *In* Avian Medicine: Principles and Application. Brentwood, TN, HBD International Inc, 1999, p 503.
13. Mazet JAK, et al: Advances in Oiled Bird Emergency Medicine and Management. J Avian Med Surg 16(2):146-149, 2002.
14. Newman SH, et al. Haematological changes and anaemia associated with captivity and petroleum exposure in seabirds. Compar Haematol Int 9:60-67, 1999.
15. POISINDEX editorial staff: (Toxicologic Management: Nicotine, Zinc, Avocado). *In* Rumack BH, et al (eds): POISINDEX System vol 100. Englewood, CO, Micromedex, 2003.
16. Pushner B, St. Leger J, Galey FD: Normal and toxic zinc concentration in serum/plasma and liver of psittacines with respect to genus differences. J Vet Diagn Invest 11(6):522-527, 1999.
17. Richardson JA, et al: Managing Pet Bird Toxicosis. Exotic DVM 3:1 2001, pp 23-27.
18. Riggs SM, Puschner B, Tell LA: Management of ingested lead foreign body in an Amazon parrot. Vet Human Toxicol 44(6):345-348, 2002.
19. Ritchie BW: Diagnosis, Management, and Prevention of Common Gastrointestinal Disease. Atlantic Coast Veterinary Conference 2002.
20. Shimmel L, Snell K: Case Studies in Poisoning- Two Eagles. Semin Avian Exotic Pet Med 8(1):12-20, 1999.