



LEVEL OF TOXICITY
Animals can present with acute or chronic lead toxicity depending upon the amount and form ingested.

MANAGEMENT STRATEGIES

Lead Toxicity:

A Threat to Wildlife

**MEET THE AUTHOR**

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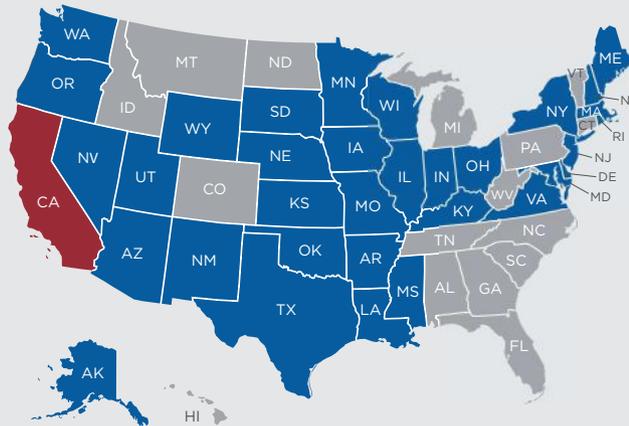
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S *enario: A fisherman finds a juvenile trumpeter swan (Cygnus buccinator) sitting quietly next to his favorite fishing hole. As he approaches the swan, he notices that she does not try to run or fly away. In fact, she appears to be too weak to stand or hold her head up. He contacts the local Department of Natural Resources officer, who immediately transports her to the nearest animal hospital. The veterinarian has seen similar clinical signs displayed by previous swan patients and immediately asks the veterinary nurse to collect samples to check blood lead levels and to prepare to take full-body radiographs.*

An estimated 20 million animals, including more than 130 differing species throughout the food chain, die each year from lead poisoning, according to the Humane Society of the United States.¹ National attention to the impact of lead poisoning on waterfowl increased considerably in 1991 when conservationists estimated that 2 million ducks died annually from lead poisoning caused by ingestion of spent lead ammunition.¹ To resolve the high mortality rate in waterfowl, the US Fish and Wildlife Service used their jurisdictional authority under the Migratory Bird Treaty Act to outlaw the use of lead ammunition to hunt migratory waterfowl and American coots (*Fulica americana*).^{1,2} Six years after the ban on lead ammunition was enforced, researchers found a 64% reduction in annual lead poisoning deaths in Mallard ducks (*Anas platyrhynchos*).² However, lead ammunition remains widely used by hunters for other animals, despite the evidence that lead ammunition is detrimental to all wildlife.¹ Twenty-six

Nonlead Ammunition Requirements

In addition to federal regulation requiring nonlead shot for waterfowl hunting



- Full nonlead requirement with a phase-in for the take of all wildlife.
- Partial nonlead requirements that go beyond the federal waterfowl ban.
- No nonlead requirements that go beyond the federal waterfowl ban.



Source: State hunting regulations & statutes 2014.

years after the 1991 ban on lead ammunition to hunt waterfowl, the lasting effects of lead in the environment continues to affect wild animals worldwide.

WHAT IS LEAD?

Lead (Pb) is a naturally occurring, highly toxic metal that has no known biological purpose.² The low cost and physical properties of lead (high density, low melting point, malleability, corrosion resistance) have made it a favored metal to be used in the production of batteries, caulks, pigments, dyes, paints, gasoline, galvanized wire, ammunition, and fishing sinkers.^{2,3} Lead is also found in many household items, such as toys, jewelry, ceramics, linoleum, electronics, drapery weights, wine bottle foil, and plumbing materials.⁴

HOW ARE WILDLIFE ANIMALS EXPOSED TO LEAD?

Wildlife exposure to lead has been linked to lead distribution in the environment from mining, coal combustion, battery processing, fuel, and waste incineration, but the primary exposure pathway for

birds is through direct ingestion of spent ammunition or lost fishing tackle.⁵ Scientists refer to lead ammunition as the “greatest, largely unregulated source of lead knowingly discharged into the environment in the United States.”⁶ Shotgun shells are available in many sizes for different shooting situations. Depending on the size, one shotgun shell could contain pellet counts in the hundreds. The percentage of lead in lead pellets is more than 95%. Once a single shotgun pellet enters the gastrointestinal (GI) system of an animal, lead is gradually released into the circulatory system.⁷ One single shotgun pellet contains enough lead to cause organ failure and death.^{1,4,5,6,8-10}

Wildlife is at risk for lead poisoning through direct or indirect lead exposure.^{1,2,5,6,8-10}

Direct Lead Exposure

Direct lead exposure occurs when an animal ingests spent lead ammunition, fragments of ammunition, lost fishing tackle, or other lead source found in the environment.^{1,4,8-10} This commonly occurs when birds mistake lead objects for seed or grit while foraging for food on the ground.^{1,4} Depending on environmental conditions, lead pellets can remain available for accidental ingestion by waterfowl in wetland environments for 25 years or longer.² Ingestion of lead fishing gear accounts for up to 50% of adult common loon (*Gavia immer*) mortality where recreational fishing occurs in locations highly populated by common loons.⁹

Indirect Lead Exposure

Indirect lead exposure occurs when an animal preys on another animal that has ingested lead or has been shot with lead ammunition.^{1,4,8-10} Indirect lead exposure affects carnivorous mammals and predatory or scavenging birds, such as eagles, vultures, and condors.^{2,4,10} Lead poisoning acquired from scavenging behavior is the greatest known killer of the highly endangered California condor (*Gymnogyps californianus*).² Indirect exposure can also occur from ingestion of soil, water, or lower organisms, such as earthworms, that are contaminated with lead.^{2,8} Lead ammunition and fishing sinkers in the environment degrade slowly and leach into the surrounding soil and water.¹⁰ The American robin (*Turdus migratorius*) and American woodcock (*Scolopax minor*) are at risk for lead poisoning via indirect exposure when they feed on earthworms in areas of high environmental lead levels.²

WHAT ARE THE CLINICAL SIGNS OF LEAD TOXICITY?

Lead causes similar toxic effects in all vertebrates, although clinical signs may vary slightly among species groups and will not always correlate with blood lead concentrations.^{2,8-10} This article focuses on clinical signs in the avian species because they have been significantly studied and are predominately affected.¹⁰

After lead is ingested, it moves through the digestive tract to the ventriculus, where the grinding action of the stomach's muscular walls and the stomach's acid begin to dissolve the lead into toxic lead salts.⁹ The toxic lead salts enter the intestinal tract, where they are absorbed into the bloodstream through the same transport mechanism used for calcium absorption.^{2,8,10} The first physiologic effect of lead in the circulatory system is the inhibition of delta-aminolevulinic acid dehydratase (ALAD) and ferrochelatase, enzymes necessary for hemoglobin synthesis.^{9,10} Moderate inhibition of ALAD results in a delay in erythrocyte maturation and anemia, characterized by decreased hemoglobin and a lowered hematocrit.^{8,9} Lead also competes with calcium and zinc for binding sites throughout the body, resulting in neurologic effects.⁸⁻¹⁰ The ability of lead to substitute for several metal ions throughout the body can cause cell damage in all body systems, resulting in a wide range of clinical signs.⁸ Approximately 90%



FIGURE 1. Adult bald eagle at Iowa State University Lloyd Veterinary Medical Center displaying clinical signs of lead poisoning, including lethargy, weakness, wing droop, and ataxia.



Depending on environmental conditions, lead pellets can remain available for accidental ingestion by waterfowl in wetland environments for 25 years or longer.²

of lead is found in the circulatory system, while the other 10% is widely distributed in soft tissue and can be stored in bone for years.⁸ Lead is eliminated from the body by sloughing of the renal tubular epithelial cells, bile, and/or pancreatic secretions.⁸

Animals can present with acute or chronic lead toxicity, depending on the amount and form of lead ingested.^{8,10} Acute lead toxicity occurs when the animal is exposed to high levels of lead and dies rapidly without showing other clinical signs.^{8,10} Chronic lead toxicity is extended lead exposure at a level that does not cause immediate organ failure and death but may eventually result in death.¹⁰ Lead toxicity primarily effects the hematopoietic, GI, renal, and central nervous systems.^{4,10} Clinical signs associated with chronic lead toxicity in birds include lethargy, weakness, muscle wasting, emaciation, wing droop, ataxia, blindness, head tremors, head tilt, green diarrhea, staining of feathers around vent, regurgitation, decreased GI tract motility, paralysis, and seizures (**FIGURE 1**).^{2,8,10} Lead toxicity in waterfowl and poultry causes clinical signs similar to those seen with botulism.¹¹

HOW IS LEAD TOXICITY DIAGNOSED?

Lead toxicity can be diagnosed by testing whole blood lead levels.^{4,8} Blood lead level testing is widely available through veterinary diagnostic laboratories.⁸ Most laboratories accept whole blood samples as small as 20 mcL (0.2 mL) in any anticoagulant. Serum and plasma are not appropriate samples for measurement of lead levels because 90% of circulating lead is in red

blood cells.^{8,11} Whole blood levels >0.2 ppm suggest lead toxicity.¹¹ Whole blood levels >0.4 to 0.6 ppm are considered diagnostic for lead toxicity.¹¹ In addition, a chemistry panel is likely to reveal elevations of lactate dehydrogenase, aspartate aminotransferase, and creatine phosphokinase as a result of liver and neuronal damage caused by circulating lead. Some affected birds will have hypochromic, regenerative anemia.¹¹

Whole-body radiography should be performed to check for evidence of metal in the GI tract, especially in the ventriculus (**FIGURE 2**).⁴ However, the absence of metallic densities within the GI tract does not necessarily rule out lead toxicity when clinical signs are present.⁴

Lead poisoning can be diagnosed postmortem by detection of lead particles within the GI tract and by measuring lead concentration in liver and kidney tissue.⁸

WHAT ARE THE TREATMENT AND PROGNOSIS OF LEAD TOXICITY IN WILDLIFE?

The fundamental treatment for lead toxicity is chelation therapy.⁸ Calcium (Ca) EDTA and succimer are both effective chelating agents, although Ca EDTA is the preferred chelator for birds.^{4,8} Ca EDTA is administered via the IM route to most avian species at a dose of 35 mg/kg q12h for 5 days, alternating with 3

days off, and repeated if necessary.⁴ A drug formulary should be consulted when determining the dose and frequency of Ca EDTA to be administered because the dosage recommendations may vary with the species and the patient's symptoms. Fluid therapy and assessment of renal function are recommended during Ca EDTA treatment because of the potential nephrotoxicity of Ca EDTA.^{4,8} The recommended dose for succimer is 25 to 35 mg/kg orally twice daily for 5 days out of the week for 3 to 5 weeks.⁴ Succimer is more effective than Ca EDTA in removing lead from soft tissues.⁸ Succimer also decreases lead concentrations in the central nervous system faster than Ca EDTA and is not nephrotoxic.⁸ After each course of chelation therapy, whole blood lead levels should be assessed.⁸ If whole blood lead levels continue to be elevated, another course of chelation therapy is indicated.⁸



FIGURE 2. A ventrodorsal radiographic projection of a juvenile trumpeter swan revealing metallic objects in the ventriculus.

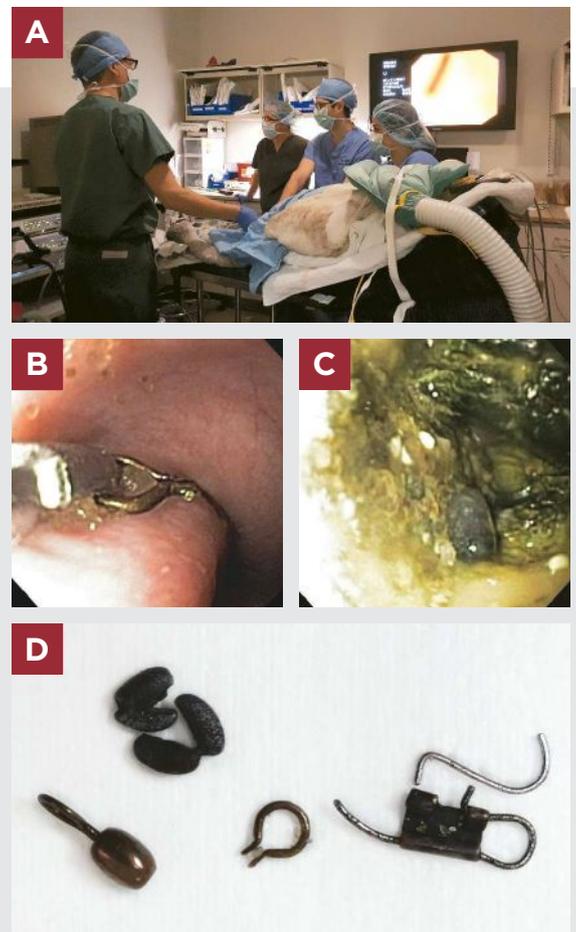


FIGURE 3. (A) An adolescent trumpeter swan undergoes an endoscopy procedure to remove metallic objects from the ventriculus. (B) An endoscopic image of a fishing hook within the esophagus. (C) An endoscopic image of a lead sinker in the ventriculus. (D) Fishing tackle after removal from the ventriculus using an endoscope.

Lead particles should be removed from the GI tract by emollient cathartics, saline gastric lavage, endoscopy, and/or surgery.^{4,8} Emollient cathartics, such as mineral oil and peanut butter, can be administered orally to aid in the passage of small metal particles through the GI system.¹¹ Administering 3 to 5 appropriately sized pieces of grit may also help remove lead particles from the ventriculus by reducing the size of the particles to facilitate passage through the GI tract.¹¹ Activated charcoal can be administered to bind to small lead particles, making them unavailable for further absorption.¹¹ Saline gastric lavage has been successful in removing lead particles from the GI tract of birds.⁸ However, endoscopy or surgical removal is often needed. Endoscopy can be used to remove lead objects that are entrapped in the crop, esophagus, proventriculus, or ventriculus (**FIGURE 3**).⁸ If endoscopy attempts fail, proventriculotomy may be indicated.⁸

Supportive care for lead toxicity may include IV fluid therapy, multicomplex B vitamins, and iron dextran.¹¹ Broad-spectrum antibiotics may also be indicated because of the immunosuppressive effect of lead.⁴ Waterfowl benefit from prophylactic treatment for aspergillosis.¹¹

CONCLUSION

Although there are numerous sources of lead in the environment, research reveals that spent lead ammunition and lost fishing tackle are the most frequent causes of lead exposure and poisoning in wildlife.⁹ The toxicity danger of lead has been known for thousands of years. However, it is only in recent decades that lead has been removed from paint, gasoline, and plumbing for the safety of humans.⁶ Lead is still readily available in the form of ammunition and fishing gear, resulting in thousands of tons of lead distributed into the environment, needlessly poisoning millions of wildlife animals each year.⁶ Alternatives, such as copper, steel, and bismuth ammunition, are readily available, are comparably priced, and do not pose the same toxic danger as lead.^{1,6}

Individual states are recognizing the negative impact of lead on the health of wild animals.⁶ Thirty-four states have continued to expand restrictions on the use of lead ammunition and fishing gear beyond the 1991 federal ban on lead ammunition use to hunt waterfowl.¹ Restrictions on lead fishing tackle are also becoming more common throughout the United States. To find



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out more about lead restrictions in your state, contact your local Department of Natural Resources or visit the U.S. Fish and Wildlife Service website (fws.gov). **TVN**

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