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S. Bret Synder

Michael J. Richard

John P. Thilsted

*New Mexico Veterinary Diagnostic Services*

Roderick C. Drewien

*University of Idaho*

James C. Lewis

*U.S. Fish and Wildlife Service*

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## LEAD POISONING IN A WHOOPING CRANE

S. BRET SNYDER, DVM, *Rio Grande Zoo, 903 Tenth Street, S.W.,  
Albuquerque, NM 87102*

MICHAEL J. RICHARD, DVM, *Rio Grande Zoo, 903 Tenth Street, S.W., Albuquerque,  
NM 87102*

JOHN P. THILSTED, DVM, PH.D, *New Mexico Veterinary Diagnostic Services, 700  
Camino de Salud NE, Albuquerque, NM 87106*

RODERICK C. DREWIEN, *Wildlife Research Institute, P.O. Box 3246, University of  
Idaho, Moscow, ID 83843*

JAMES C. LEWIS, *U.S. Fish and Wildlife Service, P.O. Box 1306,  
Albuquerque, NM 87103*

**Abstract:** The first known incident of lead poisoning in a whooping crane (*Grus americana*) occurred as the affected bird completed its second migration from Grays Lake National Wildlife Refuge (NWR), Idaho to Bosque del Apache NWR, New Mexico. It fell ill about 4 1/2 weeks after reaching the winter site and was captured. The bird exhibited signs of anorexia and starvation, weakness with drooped wings, greenish watery diarrhea and an unusual gaping of the mouth with abnormal swallowing motions. Blood showed marked polychromasia (>50%) and reticulocytosis (>80%) of RBC's. Serum chemistry values were not diagnostic. A radiograph showed the gizzard contained material more dense than normal grit. Blood lead levels were 566 mg/dl (5.66 ppm). The bird died after 48 hours in captivity, failing to respond to supportive care and chelation (1 injection of calcium versenate @35 mg/kg IM). At necropsy the gizzard contained approximately 890 tiny lead particles (8.35 g) as well as many particles of plastic and aluminum. Liver lead levels were 24 ppm (wet basis), and kidney 10.4 ppm (wet basis). The source of the ingested lead was thought to be a small plastic encased battery or fishing sinker or similar object. The crane had also been shot with a shotgun sometime previously, but this was not the cause of death.

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Lead poisoning is uncommon in cranes (Wallace et al.1983; Windingstad et al.1984), and to our knowledge has not previously been noted in whooping cranes. Yearling whooping crane 82-13 was a member of the experimental Rocky Mountain flock and had spent summer 1983 at Grays Lake NWR, Idaho, migrating south on 11 October in the company of many sandhill cranes (*Grus canadensis*). The next several weeks were spent in the San Luis Valley, Colorado, near the Monte Vista NWR, Colorado. It was first observed on the winter grounds along the Rio Grande in central New Mexico on 25 November, at the Belen State Refuge. It was seen several times in the area from Los Lunas to Bosque del Apache NWR during the next 4 1/2 weeks. It appeared in good health and behaved normally throughout this time. On the morning of 28 December, it was seen leaving a roost site at Bosque del Apache along with many sandhill cranes and at least 1 other whooping crane. The birds left the refuge that morning in response to disturbance of goose hunting. Whooper 82-13 was next seen on 5 January 1984,

obviously sick and lethargic. It exhibited unusual mouth gaping and retching behavior, and feathers of the left wing, left side and breast, and on the head, were stained reddish. The gaping behavior continued unabated, but it otherwise appeared to gain in strength and became more alert. Cold weather set in on 16-17 January and the bird's condition noticeably deteriorated. It then remained isolated from other cranes, appeared unwilling to fly, and made little or no effort to forage. It was captured by night lighting (Drewien et al.1967) on 21 January and was transported directly to the Rio Grande Zoo, Albuquerque, New Mexico, the nearest facility for medical care.

### DIAGNOSIS AND TREATMENT

When the bird was examined it stood with both wings drooped, head erect and with alert mentation. It made gaping movements with its bill, exaggerated swallowing efforts, and shook its head as if to dislodge a foreign body in it's mouth. The head, eyes, ears and oral cavity were normal.

The keel bone was prominent with apparent atrophy of pectoral muscles. There was a general state of starvation/inanition, weight 4 kg. (normal weight would be 5.6 kg). Feces were copious, watery greenish bile with urates. Cloacal temperature was 99.6 F (37.5 C), heart rate >280/minute and respiratory rate 20/minute. The skin and contour feathers were normal, there being no visible blood stains or wounds. It was moderately infested with lice. No palpable bone fractures, dislocations or other evidence of trauma or injury were seen.

Hematology results were PCV 52%, WBC 2-3/oil immersion field, RBC polychromasia with approximately 50% immature appearing RBC's, reticulocytosis >80% by new methylene blue stain. Bile feces examined by direct wet mount showed numerous flagellate protozoa (TNTC/HPF). No coccidial oocysts or helminth ova were seen. A radiograph showed three shotgun pellets in the left thigh region. The ventriculus contained a large mass of radiodense material, its significance being uncertain.

Problems recognized from this exam were 1) starvation/ inanition, 2) dehydration, 3) weakness, 4) abnormal head and mouth movements, 5) gunshot wound, 6) enteritis, 7) polychromasia and reticulocytosis of RBC's, and 8) radiodensities in the gizzard. The preliminary diagnosis was gunshot wound with sequelae from internal injury and debilitation. Therapy consisted of fluid, electrolyte and nutrient replacement. Liquid nutrition in elemental form was given by stomach tube at 50 ml every 6 hours (Vivonex, Norwich Eaton). Intravenous fluids (LR5D + aminoplex + glucose) and a corticosteroid (solu-delta-cortef, Squibb, 90 mg) were administered twice in the first day (28cc as a bolus, and 100cc over 45 minutes). In both instances there was an immediate improvement in condition. An antibiotic (ampicillin @ 250 mg tid, IM) was administered in the event that infection or sepsis was ongoing. The bird was placed under heat lamps in a darkened room for recovery. Prognosis was guarded to poor.

Lead poisoning was suspected on the second day; the clinical presentation was very similar to that of an earlier report of lead poisoning in captive sandhill cranes (Kennedy et al.1977). The radiodense materials in the gizzard seen on the radiograph were then interpreted as probable lead. The polychromasia and reticulocytosis of RBC's was also supportive evidence. Anemia may have been masked by dehydration. Blood was submitted for lead determination (New Mexico Veterinary Diagnostic Laboratory, Albuquerque) and chela-

tion therapy was initiated (calcium versonate @ 35 mg/kg tid IM). The crane died on 23 January after 1 chelation injection, approximately 48 hours after capture.

Blood lead levels were later reported as 566 mg/dl (5.66 ppm). Serum chemistry values from the initial blood sample were also reported later as glucose 300 mg/dl, total protein 3.2 g/dl, albumin 1.6 g/dl, alk phos 20 IU/L, SGOT 200 IU/L, SGPT 24 IU/L, LDH 456 IU/L, CPK 168 IU/L, Na QNS, K 4.4 mEq/L, Ca 7.2 mg/dl, inorganic phosphorus 4.8 mg/dl, total bilirubin 0.8 mg/dl direct bilirubin 0.4 mg/dl, triglycerides 83 mg/dl, cholesterol 224 mg/dl; lipase 3.2, uric acid QNS, urea nitrogen 8 mg/dl, and creatinine 0.8 mg/dl. Bacterial cultures recovered no pathogens.

## PATHOLOGY

**Necropsy** - A complete necropsy was performed by J. Thilsted at the zoo facility on 24 January. External exam was similar to that noted in the clinical work-up. Internal exam revealed no subcutaneous fat stores, and a very small amount of intraabdominal fat, and fat still present in the coronary groove of the heart. Mild atrophy of pectoral muscles was evident. The 3 lead pellets were dissected from muscles of the left leg. Adjacent muscle was also collected. No hemorrhage or tracts were evident. The recovered shot was dark grey, soft metallic, and appeared to be lead. The bird was a male, with small, black elongated testes (2 cm long x 0.2 cm diameter). No gross lesions were evident in the airsacs, liver, spleen, trachea, lungs, oral cavity, brain, pituitary or kidneys. The spleen measured 1.5 cm in diameter. The adrenals were yellow and appeared enlarged.

One measured 1.8 cm x 1.5 cm x 1.0 cm, the other 1.8 cm x 1.0 cm x 1.0 cm. Poorly defined pale streaking was evident on the surface of the right ventricle and apical region of the left ventricle of the heart. The lumen of the ventriculus (gizzard) was filled with dark green plant fiber, and a mass of metal fragments, plastic fragments and small stones. The content was washed, sifted and analyzed.

**Ventriculus (Gizzard) Content** - The non-plant materials recovered from the gizzard lumen were:

- 1) Small Metal Fragments - Approximately 890 tiny greyish-silver, soft metallic fragments which appeared as lead were found. They were uniformly elongated and flattened, each

0.5-5.0 mm long x 0.5-3.0 mm wide x 0.2-1.0 mm thick, and weighed 8 . 35 g.

- 2) Large Metal Fragments - Approximately 45 greyish-silver flattened metal fragments were distinguishable from the small fragments (they settled more slowly in water) and measured 1.0 - 10.0 mm long x 1.0 - 6.0 mm wide x 0.2 - 1.0 mm thick and weighed 0 . 2 g.
- 3) Small Stones - Approximately 235 rounded stones were found, 2-4 mm in diameter and weighing 16.15 g.
- 4) Plastic Fragments - Approximately 45 plastic fragments, most of which were 3 mm x 10 mm x 0 . 5 mm, were found, most of which were red on 1 side and pale pink on the other. Two large yellow-grey plastic fragments (2.0 cm x 0.7 cm x 1.3 cm) were also present.

**Histopathology** - In the heart, multiple small foci of hyalin degeneration were evident in various sections of myocardium. There was fibrinoid degeneration in a few arterioles of the left ventricular wall. In liver moderate amounts of brown granular pigment resembling hemosiderin were present in Kupffer cells. Smaller amounts of finely granular pigment were present in hepatocytes. There was a sparse mononuclear cell infiltrate in portal areas. Abundant iron positive granular material was present in Kupffer cells with lesser amounts in hepatocytes. The kidney had droplets of golden brown pigment in tubular epithelial cells. No acid-fast inclusions were evident in this organ, but granular iron positive material was present in tubular epithelial cells.

Aggregates of degenerating RBC's and leukocytes were in the cornified region of the mucosa of the ventriculus. Tissue surrounding the pellets removed from the left leg had a fibrous connective tissue capsule (approximately 200 microns thick). No inflammatory cell infiltrate or hemorrhage were present within the adjacent muscle. There was diffuse congestion of the lungs and moderate lymphoid depletion of the spleen. No significant microscopic lesions were evident in colon, cecum, proventriculus, small intestine, pancreas, thyroid, parathyroid or adrenals. There were 2 small foci of gliosis in the ventro-medial cerebrum, and no significant lesions in sections of midbrain, optic lobe of cerebrum, cerebellum or medulla. The testes were inactive with no spermatozoa present.

**Toxicology** - Lead analysis of the antemortem

EDTA blood sample yielded a blood lead concentration of 566 mg/dl (5.66 ppm). A homogenized liver sample revealed a lead level of 24.0 ppm (wet basis). Homogenized kidney had a lead level of 10.4 ppm (wet basis). The small metal fragments recovered from the gizzard were lead, with analysis revealing a composition of lead 96.2%, antimony 3.4%, arsenic trace, copper trace and tin trace. The larger metal fragments from the ventriculus were aluminum 90%, lead 1.8%. Analysis of the pellets recovered from the left leg revealed lead 98.7% and antimony 1.3%. The metal analyses were performed by Inductively Coupled Plasma Emission Spectroscopy and confirmed by Atomic Absorption Spectroscopy (New Mexico Veterinary Diagnostic Laboratory, Albuquerque).

Samples of the metallic and plastic particles recovered from the gizzard were also submitted to a forensic laboratory for analysis to determine the source of lead (Federal Bureau of Investigation, Washington, DC, report on file). Composition was not the same as that of standard gun shot. It further appeared that the plastic particles were from 8 casing surrounding the lead, because 1 surface of the plastic had a greater concentration of lead than the other. The exact source could not be determined, but the best guess of FBI technicians included 1) a small battery such as a penlight, or 2) a plastic covered fishing sinker or similar object.

**Bacteriology** - No bacteria were isolated from necropsy specimens of heart blood, left lung, right lung, left airsac or right airsac. No enteric pathogens were isolated from cultures of cecum and colon. A few colonies of *E. coli* were isolated from the liver. Standard laboratory methods for microbiology were utilized (New Mexico Veterinary Diagnostic Laboratory, Albuquerque).

**Parasitology** - Many flagellate protozoa were observed in direct smears of feces. These were not identified except to rule they were not *Giardia* sp. No helminth ova were found in either direct smears or concentrated specimens.

## DISCUSSION

The clinical signs of illness in this crane were similar to those previously reported for lead poisoning in sandhill cranes (Kennedy et al.1977), most notably anorexia, drooped wings, green watery diarrhea and the peculiar gaping of the mouth and abnormal swallowing motions (probably a neurological sign). Also similarly, a marked polychromalia (>50%) and reticulocytosis (>80%) of RBC's was present.

The gross and microscopic pathology in this case were compatible with descriptions of other cases of lead toxicosis in birds. These changes are not marked, nor are they pathognomonic. Myocardial necrosis, fibrinoid degeneration of blood vessels, hepatic hemosiderosis and emaciation were found in this whooping crane and have previously been reported in swans, geese and sandhill cranes dying of lead toxicity (Kennedy et al. 1977; Cook & Trainer 1966). Acid-fast intranuclear inclusions in kidney tubular epithelial cells have been described in some cases of lead poisoning (Locke et al. 1966), but these were not seen in this bird.

The blood lead level of 566 mg/dl (5.66 ppm) was higher than that of 146 mg/dl to 378 mg/dl reported in lead-poisoned sandhill cranes (Kennedy et al. 1977). The liver lead level was 24.0 ppm, which is comparable to liver concentrations found by Locke et al. (1966) and Windingstad et al. (1984). Liver lead concentrations have similarly been reported for other avian species dying of lead toxicosis to include geese (Bagley et al. 1967; Cook & Trainer 1966), ducks (Del Bono & Bracha 1973) and loons (Locke et al. 1982).

There is a little information on the incidence of poisoning from ingested lead in cranes. Fatal cases in sandhill cranes have been reported from ingestion of lead based paint (Kennedy et al. 1977) and from ingestion of fishing sinkers and .22 caliber rifle cartridges (Windingstad et al. 1984). Sandhill cranes were found to have a low incidence of lead shot ingestion (0.8%) by Wallace et al. (1983). This was thought to be due to their feeding behavior and low hunting pressure.

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