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Lead Toxicosis in Mallard Ducks[1]

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ABSTRACT

Eight #6 lead pellets placed in the ventriculus of mallard drakes induced maturation arrest of promegaloblastic-like erythroid cells in the bone marrow, a hypochromic microcytic anemia with poikilocytosis in the peripheral blood, and acid-fast intranuclear inclusions in the renal proximal tubule cells.

INTRODUCTION

Lead toxicosis is a well recognized and serious disease of wild waterfowl populations. Literature reviews on the progress of research relating to lead poisoning in waterfowl are incorporated in publications by Bellrose,[1] Trainer and Hunt[10], and Locke, et al.[6] The blood dyscrasia in lead-poisoned ducks is described by Coburn et al.[3] This is a report of studies on the pathogenesis of the anemia in ducks with induced lead toxicosis.

METHODS

Healthy adult mallard *(Anas platyrhynchos)* males were radiographed to ascertain the presence or absence of lead shot in their digestive tract lumens. They were weighed and bled to establish baseline hemogram values. Twenty-one birds had digestive tracts free of lead. With a flexible plastic tube, eight #6 lead shot were placed in the ventriculus of each of 10 ducks and 11 others were left as controls. The birds were fed a ration of yellow corn, a grain found by Jordan and Bellrose[5] to be helpful in evincing lead toxicosis and were examined daily. Bleeding from the jugular vein was repeated in mid-syndrome and when the moribund birds were euthanatized.

Standard hematologic techniques[12] were used throughout this study. Erythrocytes were counted in duplicate on a Coulter counter. Three hundred cell differentials were done on all blood smears. Peripheral blood and bone marrow smears were stained with modified Wright-Giemsa stain.

After euthanasia, all birds were necropsied, weighed, and their alimentary canals were checked for retained lead shot.

Livers, spleens, and colon contents were cultured on appropriate mediums.

Selected tissues from 5 toxic and 3 control birds were fixed in 10% formalin, embedded in paraffin, sectioned at 6 microns, and stained with hematoxylin and eosin. Duplicate kidney sections were stained with Ziehl-Neelsen acid-fast technique; duplicate brain sections were stained with azure eosinate and also with Luxol fast blue-periodic acid Schiff technique; and duplicate liver sections were stained with the Prussian blue technique. In addition, frozen liver sections were sectioned at 20 microns and stained with flaming red.

Brain and liver specimens were evaluated for lead with dithizone procedure, a colorimetric method, the limitations of which are indicated by the authors[4].

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[1] These results have been presented in part at the 1967 Annual Wildlife Disease Conference, Urbana, Illinois, June 15-17, 1967.

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RESULTS

A. *Clinical*

Within 1 to 2 weeks after receiving the lead pellets, the test birds developed marked weakness, inappetence, and weight loss. Their mucous membranes were pale and their exercise tolerances were diminished. The "roof-shaped position" of wings over the dorsum described by Jordan and Bellrose[5] was noted terminally in most birds. Although they became listless, coma and hypersensitivity were not observed. Signs other than those that conceivably might be attributed to the profound asthenia of severe anemia were not noted.

The only natural death occurred 13 days post-dosage. Eight other birds were euthanatized in a feeble condition by post-dosage day 25. One interesting duck (#2380) expelled its lead shot dosage spontaneously and appeared to have made a complete clinical recovery when euthanatized 41 days post-dosing.

B. *Gross Lesions*

Although control birds came to necropsy lesionless and plump, the lead-poisoned individuals (excepting duck #2380) were found to be profoundly emaciated. These toxic fowl had conspicuous paleness of all tissues, a loss of fat in the regional depots, and atrophy of musculature and internal organs. Proventricular impaction was not encountered. The spleens were very small, not resembling the swollen spleens usually seen with hemolytic anemia. The number of pellets remaining in the ventriculus of the nine moribund cases at necropsy averaged six per bird and ranged from four to eight. The pellets recovered at necropsy averaged 35% less than the original weights.

C. *Microscopic Lesions*

The brain sections with the described histologic routine and special stains lacked unequivocal lesions, but it is possible that special techniques, such as the capillary permeability test employed by Pentschew et al.[8] on lead-poisoned suckling rats, might have demonstrated definite damage. Vacuolization of most liver cord cells was shown not to be due to hepatic lipidosis. Both hepatocytes and Kupffer cells contained amorphous agglomerates of iron-containing pigment considered to be hemosiderin. Kidney lesions were largely in the proximal convoluted tubules, the cells of which had both nuclear and cytoplasmic damage. With hematoxylin and eosin, these nuclei in the toxic ducks had chromatin clumped against the nuclear membrane; and with the acid-fast stains many nuclei contained one or more irregularly contoured acid-fast inclusions, which were not present in the control ducks. Cytoplasmic alterations were not as marked as the nuclear changes, but the renal proximal tubule cells in the poisoned ducks often had cloudy swelling and hyaline droplet formation, individualization and hypereosinophilia.

D. *Hemogram*

The averages for control and poisoned ducks are plotted in Graph 1, 2 and 3, and the calculated indices are presented in Table 1. Smears of bone marrow and peripheral blood of poisoned ducks revealed obvious developmental defects in the erythroid line. Conspicuous in the marrow of the toxic birds were promegaloblastic-like cells with deep blue cytoplasm and fine nuclear chromatin network. These large cells appeared to be close to the reticulum. Few of them appeared to be progressing toward maturity and release into peripheral blood. This maturation arrest allowed accumulations of these erythrocyte precursors in the bone marrow with a corresponding diminution in tissue fat spaces (Fig. 1, Fig. 3).

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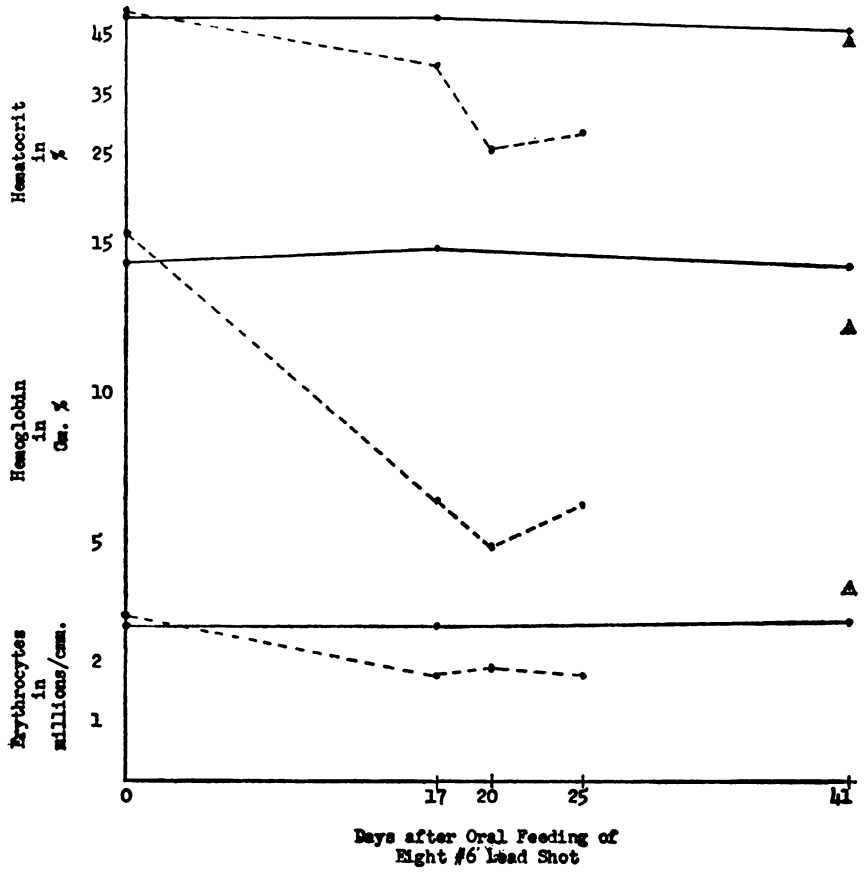

Graph I. Hematocrit, hemoglobin and erythrocyte levels for control and toxic ducks.

——— Control Ducks
- - - - Poisoned Ducks
△ Solitary Convalescent Duck 2380

Imperfections were pronounced in many of the red cells reaching circulation: unipolar and bipolar spindle cells, Maltese crosses, anuclear cells, and oat-shaped cells. The peripheral red blood cells also showed marked hypochromasia, polychromatophilia, and a distinct increase in mitotic activity (Fig. 2, Fig. 4, Fig. 5).

The calculated indices presented in Table 1 indicate that the erythrocytes in the peripheral blood of the poisoned ducks were reduced in size, hemoglobin content, and hemoglobin concentration.

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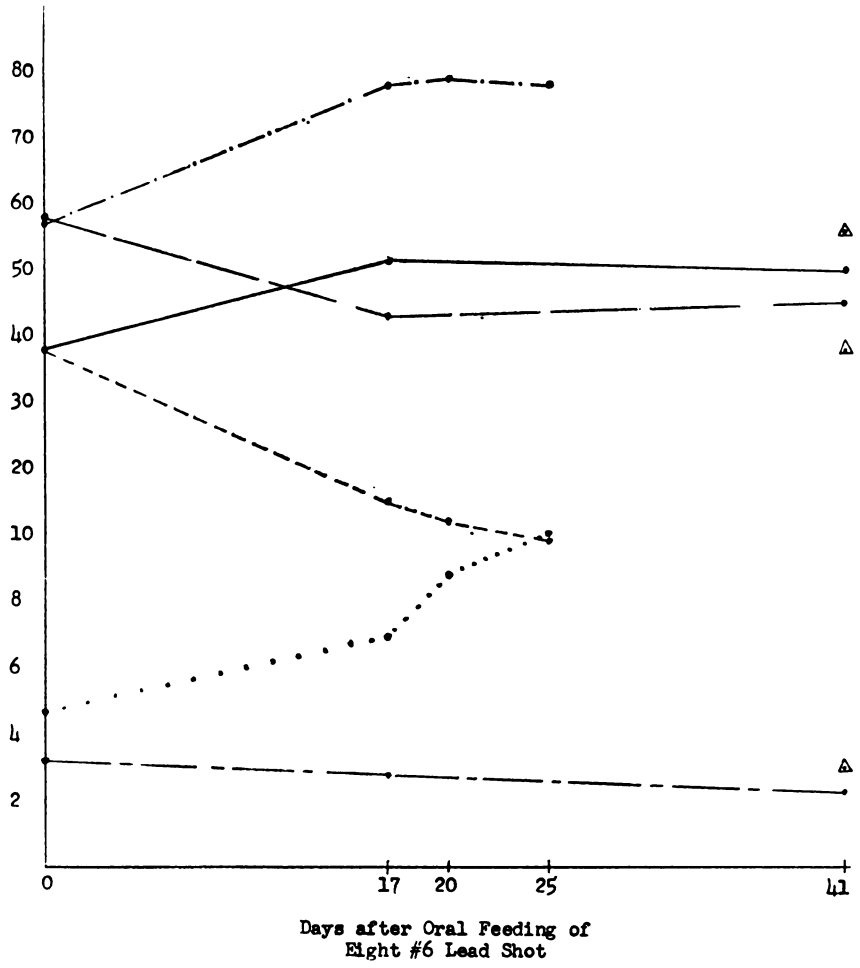

Graph II. Differential leukocyte levels of control and toxic ducks.

—·—% Monocytes, Peripheral Blood, Control
·····% Monocytes, Peripheral Blood, Poisoned
———% Lymphocytes, Peripheral Blood, Control
- - - -% Lymphocytes, Peripheral Blood, Poisoned
— —% Neutrophils, Peripheral Blood, Control
—·—·% Neutrophils, Peripheral Blood, Poisoned
△ Solitary Convalescent Duck 2380

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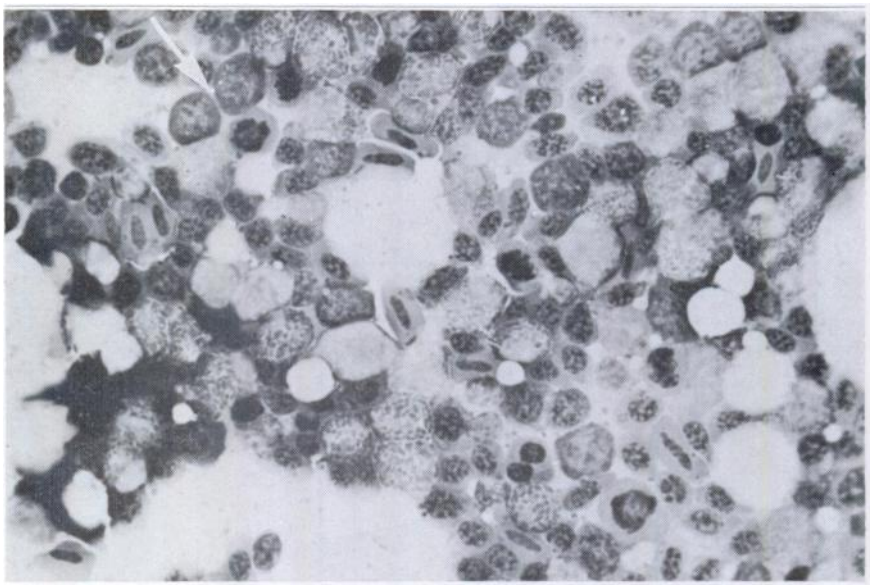

FIGURE 1. *Bone marrow of control duck #2396. There are conspicuous fat spaces and many developing myeloid cells. Arrow points to two normal pronormoblasts.*

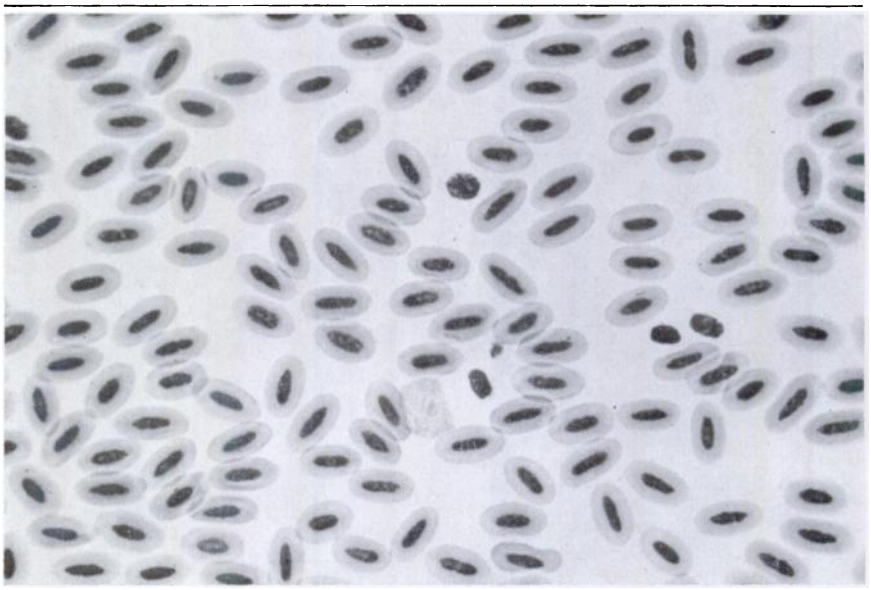

FIGURE 2. *Peripheral blood pattern of control duck #2396.*

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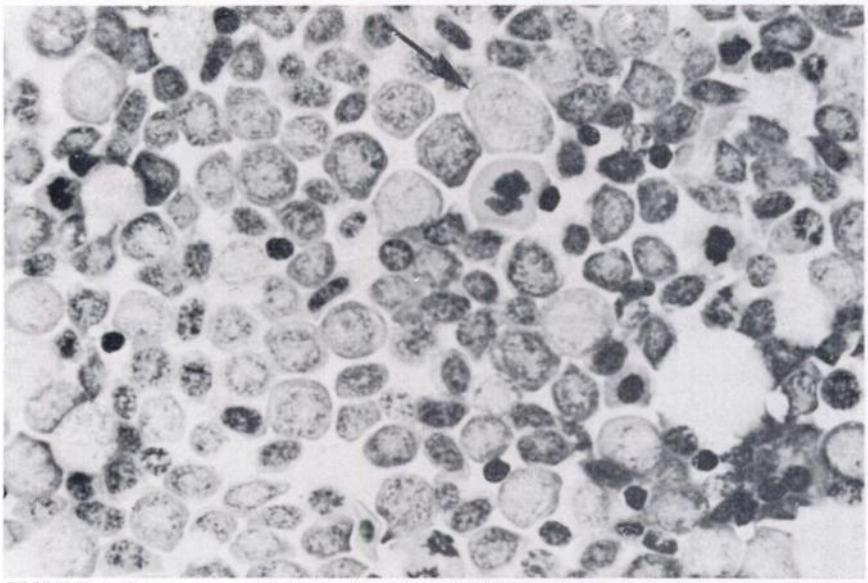

FIGURE 3. *Bone marrow of lead poisoned duck #2384. There is marked erythroid hyperplasia with almost complete lack of developing myeloid cells. Arrow points to a large early erythroid cell having characteristic promegaloblastic fine reticular chromatin pattern.*

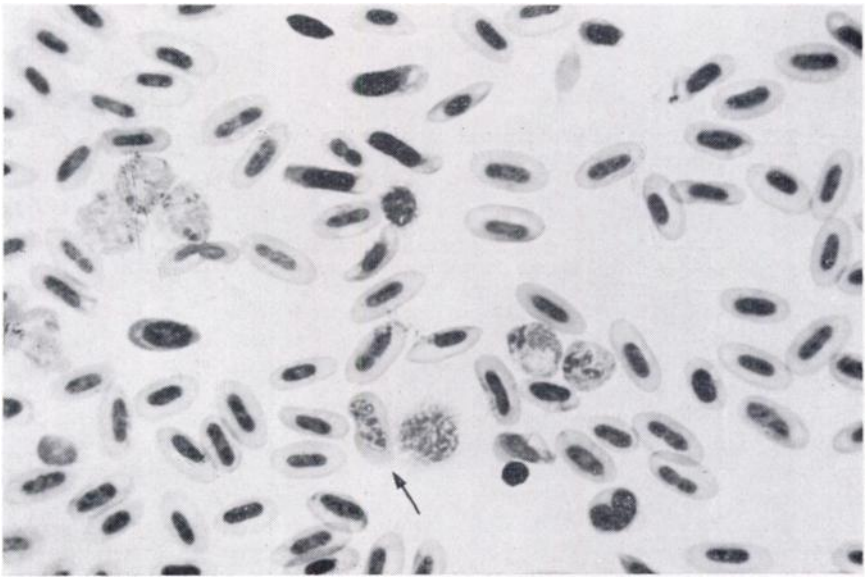

FIGURE 4. *Peripheral blood of lead poisoned duck #2384. There are marked changes in erythrocytes. Arrow points to late polychromatic erythrocyte having retention of fine nuclear chromatin pattern.*

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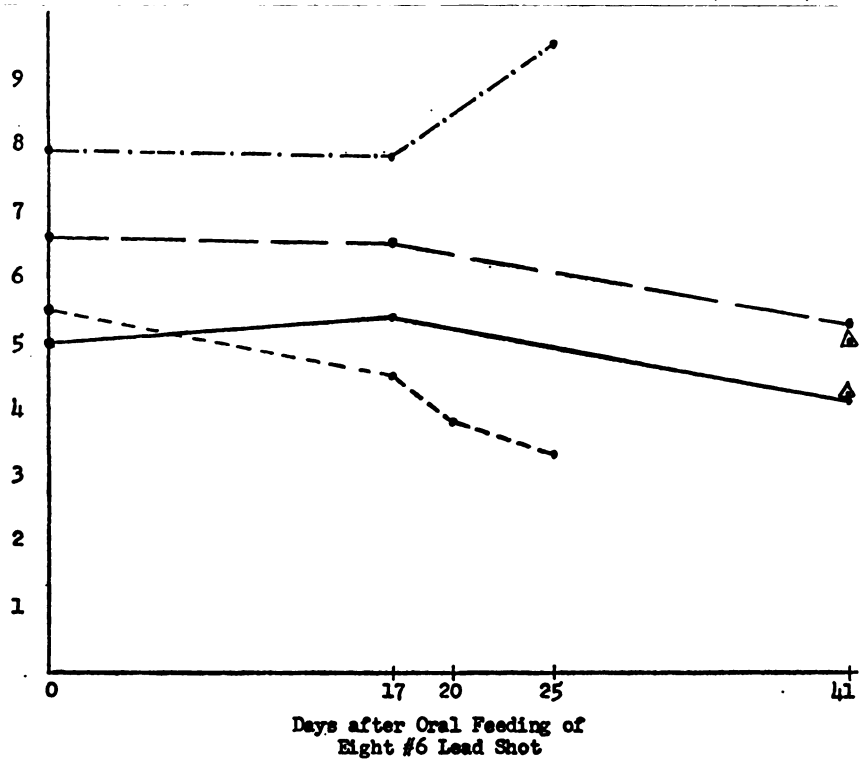

Graph III. Total plasma protein and blood urea levels of control and toxic ducks

——— Total Plasma Protein of Control Ducks in Gm. %
– – – – Total Plasma Protein of Poisoned Ducks in Gm. %
— — B.U.N. of Control Ducks in mg %
—·—· B.U.N. of Poisoned Ducks in mg %
△ Solitary Convalescent Duck 2380

E. *Lead Levels*

Lead in the liver and brain of the control ducks was not detectable by the test employed. The terminal liver lead levels in the poisoned ducks averaged 33 micrograms/gram (wet weight) with a range of 20 to 64. The terminal brain levels averaged 5 micrograms/gram with a range of 3 to 6. The terminal level of each individual liver ranged from 5 to 11 times that for the lead level in the brain. This ratio also was observed in a later spontaneous case where both organs were assayed for lead content.

F. *Bacteriology*

Smears and cultures for bacterial pathogens were consistently negative.

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Table I. Comparision of red cell corpuscular values.

	Control Ducks, Initial	Control Ducks, Final	Test Ducks, Initial	Test Ducks, Final	Moribund Field Cases*
Mean Corpuscular Volume (cubic microns)	181.0	163.4	179.6	150.3	141.5
Mean Corpuscular Hemoglobin (micromicrograms)	52.6	51.0	56.0	30.0	21.1
Mean Corpuscular Hemoglobin Conc. (%)	29.1	31.2	31.2	19.9	14.8

* Average of 4 Mallard Males with Spontaneous Lead Toxicosis

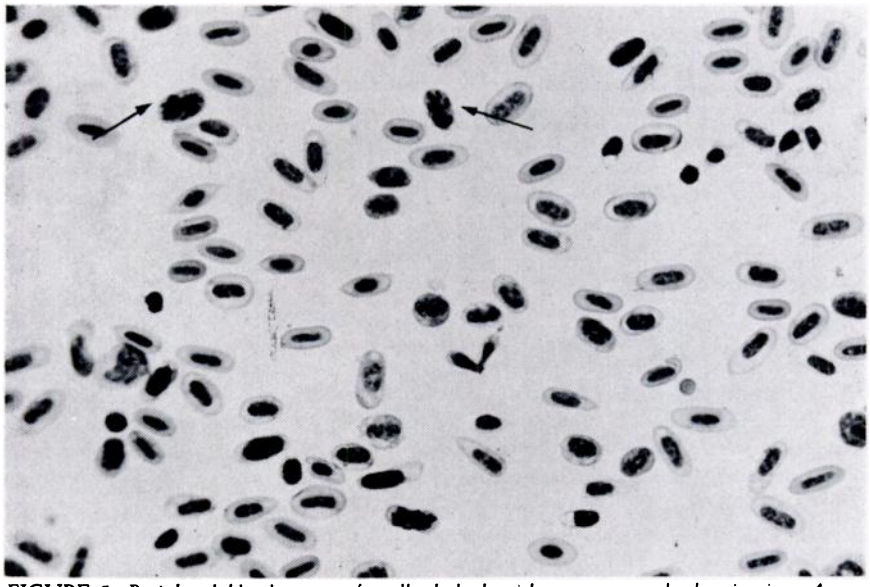

FIGURE 5. *Peripheral blood smear of mallard duck with spontaneous lead poisoning. Arrow points to two erythrocytes undergoing mitosis.*

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DISCUSSION

Under the conditions of this experiment, toxicosis occurred only in the acute form. The signs were characterized by a lack of diagnostic specificity. The blood dyscrasia, if found in a field disease outbreak, would probably eliminate many other common wild duck diseases from serious consideration. The gross necropsies were likewise not particularly distinctive except for emaciation, anemia, and the presence of lead shot in the ventriculi. A few lead shot may be present in the ventriculi of ducks dying from other causes; therefore, death cannot be attributed to lead toxicosis merely on the presence of shot in the lumen. Histologically, intranuclear inclusion bodies in the proximal renal tubules dominated the findings. These inclusions, recently described in lead-poisoned mallard ducks,[6] were striking in acid-fast stained kidney sections, but could have easily been overlooked on hematoxylin and eosin stained sections. The first bird to die (#2392) 13 days post-dosing with retention of all 8 pellets was the only poisoned bird histologically examined that lacked these renal inclusion bodies. At the other extreme, the convalescing bird (#2380) that expelled its entire load of 8 pellets and was euthanatized 41 days post-dosing had unequivocal inclusions. It appears that these renal inclusions may persist for some time after a poisoned bird has lost its pellets and recovered.

Yellow corn as the sole ration was adequate for the maintenance of reasonable weight and hemograms for all control ducks for the duration of this experiment, although it is a nutritionally inadequate diet.

The basis of the anemia of lead poisoning is not completely resolved. Anemia can be caused both by a decrease in erythrocyte production and an increase in erythrocyte destruction. In a recent general review on the pathogenesis of the anemia of lead toxicosis,[11] the equivocalness of the hemolytic anemia (destruction of erythrocytes) and the relative certainty of seriously impaired erythropoiesis (production of erythrocytes) are emphasized. The maturation arrest of erythroid elements in the bone marrow and the aberrant morphology of the erythrocytes in the peripheral blood of the ducks of this experiment indicates defective erythrocyte production and impaired release of these cells from the marrow as a prime source of the anemia.

McConnell et al.,[7] using radioselenium-tagged red cells in White Peking ducks, found the intravascular life of transfused cells to be only 11.7 days. Brace and Altland[2] using glycine-2-C^{14} and Rodan et al.[9] using $Na_2Cr^{51}0_4$ found the life span of duck erythrocytes to be 42 days. Therefore, the precipitous drop in erythrocytes, hematocrit, and hemoglobin values (Graph 1) may be due to the cessation of replacement production of red cells.

Deviation in the composition of the leukocyte population also resulted from lead toxicosis. In the poisoned birds, the per cent of monocytes and neutrophils rose while the per cent of lymphocytes fell sharply (Graph 2). The blood urea nitrogen levels of the terminal poisoned ducks rose slightly, while their plasma protein levels fell during the same period (Graph 3). The blood urea rise and plasma protein fall may reflect dysfunction of kidney and liver, respectively, two organs in which morphologic damage could be observed microscopically.

One duck (#2380) developed signs of toxicity but never became moribund; it expelled its lead pellets at some undetermined time and when euthanatized at 41 days, it had not only regained its weight and strength but each of its blood values (indicated on Graphs 1, 2, and 3 by Delta) had been restored to the proximity of the baseline.

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ABSTRACT

WHITTEN, C. F. 1967.

Innocuous Nature of the Sickling (Pseudosickling) Phenomenon in Deer.

British Jour. Haematology *13* (5): 650-655.

Sickle-shaped deer cells have a normal life span because they are non-fragile, and they do not obstruct blood vessels because they are small and pliable. Thus, the presence of sickle-shaped cells in deer is innocuous. These findings have relevance to the sickling phenomenon in humans in that they emphasize the importance of the physical properties of sickled cells, exclusive of shape, in the pathogenesis of sickle cell anaemia.

The data in this study in conjunction with previous observations in the literature indicate that there are five basic differences between the sickling phenomenon in deer and humans. (a) The haemoglobin of deer differs from human sickle haemoglobin in composition and in electrophoretic behaviour. (b) The formation of sickle-shaped deer cells can be induced by oxygenation (which blows off carbon dioxide and induces a pH change), whereas human cells sickle when de-oxygenated. (c) The internal structure of deer sickle-shaped cells is probably that of a gel instead of the tactoid formation found in human cells. (d) Sickle-shaped cells of deer are non-fragile and pliable whereas human sickle cells are fragile and rigid. (e) The presence of sickle-shaped cells in humans produces a disease state, whereas their presence in deer is innocuous. Since the shape of the cells is the only property that is similar in deer and human sickling, it is more appropriate to designate the phenomenon in deer as 'pseudo-sickling'.

Author's summary.

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