Epidemiology of Lead Poisoning in Cattle

by

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The case histories of lead poisoning diagnosed in 63 herds of cattle over the past five years were studied. The toxicosis was found to be most prominent during the spring months and resulted in a 61 percent case fatality rate. The clinical signs observed involved mainly the central nervous and gastrointestinal systems, as did the postmortem findings. The mean tissue lead levels were determined and correlated with the source of the toxicant.

INTRODUCTION

Lead poisoning has been a part of man's history since probably 4200 B.C. It has been incriminated as a part of the cause of the fall of the Roman Empire.[10] Even with the increased awareness of lead toxicity today, it still continues to be a common toxicant of both livestock, pets, and man. Buck reported lead as one of the most common causes of livestock poisoning in the United States.[3] Zook stated it was the most common cause of poisoning in dogs.[20] Perlstein and Attale reported in 1966 that lead accounted for 80 percent of all deaths in children due to accidental poisoning.[16]

This report contains the findings of 63 episodes of lead poisoning in cattle that were compiled from the records of the Iowa Veterinary Diagnostic Laboratory. The episodes span a five-year period from April of 1965 through May of 1970.

METHODS

Lead determination in liver and kidney tissues, rumen contents, and environmental specimens such as paint, oil, and grease employed the method reported by Berman using a spectrophotometer.[2]*

TABLE 1—Seasonal Occurrence of Episodes of Clinical Lead Toxicoses in Cattle*

Month	No. of Cases	Percent of Total	Seasonal Total Percent
January	7	12	
February	3	5	29
March	7	12	
April	5	8	
May	16	26	41
June	4	7	
July	9	15	
August	2	3	21
September	2	3	
October	0	0	
November	1	2	9
December	4	7	
Totals	60	100	100

* This covers a four-year period (June 1, 1966, to May 30, 1970) instead of the five-year period that the rest of the data represents.

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* Model 303 Spectrophotometer, Perkin-Elmer Corp., Norwalk, Connecticut.

Blood lead determinations were done using the procedure described by Hessel.[13]

RESULTS

Season Occurrence of Episodes

A definite seasonal trend was noted with 91 percent of the episodes occurring in the first nine months of the year. Also, three months (April, May, and June) accounted for 41 percent of the total for the year (Table 1). One month (May) alone accounted for 26 percent of the total episodes (Fig. 1).

Morbidity and Fatality Rates

In cases where sufficient information was known, the statistics, shown in Table 2, were determined. Used oil (Category 2) as a source of clinical lead poisoning produced a lower morbidity rate than did Category 1 (lead batteries, paint, grease); but a higher percentage of those affected from oil sources died than those exposed to other sources.

Clinical Signs

Ninety percent of the affected cattle exhibited some sign(s) of central nervous system involvement, and 60 percent exhibited signs of gastrointestinal involvement. This information is summarized in Table 3. Body temperatures were recorded in only 15 cases and were normal in eight but increased (up to 111° F.) in the other seven. Acute death (less than 24 hours after onset of clinical signs) was reported in about one-third of the episodes. Many animals, however, survived for a longer period of time ranging from a few hours to about ten days.

Necropsy Findings

Necropsies were done in 37 of the 63 episodes. Of these, no gross lesions were observed in ten. In the remaining cases the following lesions were observed: oil in the gastrointestinal tract (30%); gastritis and/or enteritis (24%); petechiation of epicardium, and/or myocardium (21%); pulmonary congestion (16%); kidney degeneration (16%).

Others mentioned (in $<10\%$ of the episodes) were: fatty liver; pale and watery muscle; petechiation of subcutaneous tissues, thymus, and trachea; cystitis; cloudy cornea; hemorrhage in eyeballs; brain edema and hyperemia; metal or paint materials in rumen and reticulum; swollen mesenteric lymph nodes.

Sources of Lead

The source of lead in each episode, where it was known, is tabulated in Table 4.

TABLE 4—Sources of Clinical Lead Toxicoses in Cattle

Source	No. of Episodes	Percent of Total
Paint	18	29
Oil	16	25
Unknown	15	24
Junk piles	7	11
Grease	4	6
Batteries	3	5
Totals	63	100

Paint proved to be the most common source of poisoning. This included old board fences and farm buildings painted with lead-containing paints thinned with boiled linseed oil (probably containing litharge, a lead product),[2] and old paint buckets left in or near pastures and feedlots. Used crankcase oil was the next most common source. This included old oil buckets and cans left in lots and pastures, and even one case of draining a tractor crankcase near a fence and letting the oil run into a feedlot where cattle were kept.

Chemical Analysis

Tissue lead levels were determined in most cases. The results (in ppm lead on a wet weight basis) are seen in Table 5. In Category 1, rumen content mean includes one value of 11,875 parts per million lead that was considerably greater than the other values found; this is reflected in that parameter. Note that the Category 1 mean levels were higher than those for oil (Category 2).

TABLE 2—Morbidity and Fatality Rates in Relation to Source of Clinical Lead Toxicoses in Cattle

	Category 1*	Category 2†	Category 3‡	All Sources
No. of episodes	15	9	12	37
Total no. animals	864	374	1,030	2,268
Total no. animals clinically affected	130	46	110	286
Total no. animals died	73	38	62	173
Morbidity $\frac{\text{No. affected}}{\text{Total no. inherd}} \times 100$	15%	12%	11%	13%
Cause specific death rate $\frac{\text{No. died}}{\text{Total no. in herd}} \times 100$	9%	10%	6%	8%
Case fatality rate $\frac{\text{No. died}}{\text{No. affected in herd}} \times 100$	56%	83%	56%	61%

(Grease, Lead Batteries, and paint sources)
† Used crankcase oil.
‡ Junk piles and other unknown sources.

TABLE 3—Clinical Signs Noted in Lead-Poisoned Cattle

System	Clinical Sign	No. of Episodes	Percent of Total Episodes
CNS	Blindness	32	51
	Muscle twitching	25	40
	Hyperirritability	21	33
	Depression	20	32
	Convulsions	20	32
	Grinding teeth	15	24
	Ataxia	11	18
	Circling	10	16
	Pushing against objects	7	11
	(One or more of these signs were reported in 90% of the episodes)		
Gastro-intestinal	Excessive salivation	26	45
	Anorexia	16	21
	Tucked abdomen	6	10
	Diarrhea	6	10
	(One or more of these signs were reported in 60% of the episodes)		
Other*	Acute death	22	35
	Bellowing	8	13

* Other signs reported (in <8% of the cases) were: Abnormal posture, decreased milk production, down and unable to rise, rhythmic head jerking, paddling, emaciation, abortion, nasal and eye discharge, gray gum line, stiffness, dehydration, pharyngeal paralysis, rumen atony, aimless walking, constant dribbling of urine, dyspnea, vomition, blistered teats, hyperkeratinized skin areas, protruding tongue, pica, opisthotonus, nystagmus, coma.
In 2 episodes clinical signs were not observed.

TABLE 5—Levels of Lead in Tissues and Rumen Contents Associated with Clinical Lead Toxicosis in Cattle

Category 1
(Grease, Batteries, and Paint Sources)

Tissue	No. of Episodes	PPM Lead on Wet Weight Basis	
		Mean	Range
Liver	40	33.7	5.0 –50.0
Kidney	44	60.7	14.0 –110
Rumen contents	32	507.8	3.0 –11,875
Blood	8	0.96	0.57–3.80

Category 2
(Used Crankcase Oil Sources)

Tissue	No. of Episodes	PPM Lead on Wet Weight Basis	
		Mean	Range
Liver	7	22.4	4.0 –70.0
Kidney	8	52.3	6.0 –125
Rumen contents	7	237.1	10.0 –921
Blood	3	0.51	0.20–1.00

Category 3
(Junk Piles and Other Unknown Sources)

Tissue	No. of Episodes	PPM Lead on Wet Weight Basis	
		Mean	Range
Liver	16	21.3	1.0 –83.0
Kidney	16	65.8	12.0 –137
Rumen contents	8	171.9	3.0 –650
Blood	6	0.66	0.30–1.14

Category 4
(All Sources)

Tissue	No. of Episodes	PPM Lead on Wet Weight Basis	
		Mean	Range
Liver	52	29.7	1.0 –83.0
Kidney	57	57.7	6.0 –137
Rumen contents	41	442.5	3.0 –11,875
Blood	17	0.78	0.20–3.80

DISCUSSION

Since lead poisoning in cattle usually involves several animals in a herd, the term "episode" is used in this paper for each instance in which lead poisoning was diagnosed in one or more animals within the same herd.

The criteria used in diagnosing lead poisoning in our laboratory includes four types of evidence; that is, history or circumstantial, clinical signs, postmortem findings, and chemical analyses. We consider it essential to find at least 10 parts per million lead on a wet weight basis in the liver or kidney tissues and/or at least 0.35 part per million lead in the whole blood to justify a definitive diagnosis of lead poisoning in cattle. It is also imperative that clinical signs and history or circumstantial evidence be compatible with that of lead poisoning. It is important to employ microbiological as well as gross and histopathologic examination of the tissues as a means of differentiating lead poisoning from other diseases. Polioencephalomalacia, infectious thromboembolic meningoencephalomyelitis, coccidiosis, rabies, organic insecticide poisoning, brain abscesses, and other diseases which affect the central nervous and gastrointestinal systems of cattle should be included in the differential diagnosis.

The range of tissue lead levels within the same animal may frequently be quite broad. Some tissues may have insignificant lead levels while other tissues in the same animal have levels great enough to warrant a diagnosis of lead poisoning. Thus, it is possible to have liver lead levels of 1 part per million with kidney and blood lead levels in the same animal of 20 and 1.0 parts per million, respectively. If the history and clinical signs are compatible, a diagnosis of lead toxicosis is then justified. For this reason it is imperative that analyses for lead be conducted on as many different specimens as are available from a single animal. This also explains the broad range of lead levels in the various specimens for all animals (Table 5). These data agree in general with those reported by Hatch and Funnell[12] of 175 cases of lead poisoning in cattle in Canada over a 15-year period.

It is apparent from these data and other reports that the bovine animal is more susceptible to lead poisoning than is perhaps the human, dog, and laboratory animals such as rats and mice.[4,6,7,8,9,10,11,20] Lead poisoning has been confirmed by our laboratory in cattle having blood lead levels as low as 0.20 parts per million in both experimental and naturally occurring cases. On the other hand, the maximum

allowable concentration for lead in human blood is reported to be 0.8 parts per million[9] while others report somewhat lower values.[9,15,18] Dogs are apparently asymptomatic with levels of up to 0.6 part per million in the blood.[20] Hammond and Aronson have reported an average of 0.4 parts per million lead in the blood of 13 horses grazing forages contaminated by a smelter in Minnesota.[11] About half of these horses had severe lead poisoning and died. They also reported blood lead levels of 0.04 parts per million in 16 horses hospitalized for minor ailments but not known to have been exposed to excessive levels of lead. These data would indicate that horses are equal to cattle in their susceptibility to lead poisoning.

The high spring seasonal incidence of lead poisoning in Iowa is probably due to several factors. The months of April and May are usually when cattle are turned out into the pastures where junk piles are located. At this time melting snow may make lead sources more available. Also, the increased sunlight during the spring and summer months aids in Vitamin D production by the skin. Vitamin D is thought to aid in the intestinal absorption of lead.[6] The lower winter incidence could relate to these same factors.

Signs of toxicity appeared within a few hours or up to ten days after ingestion of lead. Central nervous system depression and anorexia were often the first signs noted. Overt nervous signs usually occurred later, such as muscle twitching, rhythmic head bobbing, blindness, ataxia, pushing against objects, circling, and grinding of teeth. Hyperexcitability and convulsions were signs of an unfavorable prognosis, and death usually occurred shortly thereafter. Since body temperatures were recorded in only 15 of the 63 cases, these results may be of questionable reliability. Opportunistic secondary infections or hypothalmic damage may be involved. The varied clinical signs are probably due partly to the lead poisoning synrome itself and also partly to the fact that many different people observed the affected animals. Hence, one sign may have been interpreted in more than one way. This same problem exists with the postmortem findings; i.e., the same lesion may have been interpreted in two or more ways. A higher percentage of necropsies would have improved the reliability of the results.

The tissue analyses revealed that paint, lead battery, and grease sources resulted in higher tissue lead levels than did oil sources (Table 5), yet the case fatality rate was greater in those episodes where oil was the source of lead (Table 2). This may reflect a difference in either absorption rate from the gastrointestinal tract or inate toxicity of the form of lead in question. Approximately 30 percent of lead from burned gasoline ends up in the crankcase oil; the remaining 70 percent goes into the atmosphere.[14] Used crankcase oil may contain many thousand parts per million of lead. A grease sample from one episode contained 45 percent (450,000 ppm) elemental lead by weight.

Note should be taken of a case which occurred in September, 1968, where a farmer spray-painted a full corn crib. The paint label did not indicate that lead was present, but the farmer became concerned and had a sample tested. It showed 370 parts per million lead. It was suggested that the contaminated corn not be fed to livestock. A little known fact that should be pointed out is that "lead-free" paint can contain up to 1 percent (10,000 ppm) lead and still retain an official "lead-free" designation.

Calves and younger cattle can be killed with single oral doses of 200–400 mg. lead/kg. body weight given as lead acetate.[3,7] Older cattle may require 600–800 mg./kg. Adult cattle have died of apparent lead poisoning after consuming as little as 1 pint (500 ml.) of used crankcase oil, licking the grease from motors or machinery from as small an area as 25 square inches (150 sq. cm.), or consuming as little as an estimated 1 cup (250 ml.) of grease containing lead. Chronic toxicity has been produced with as little as 6 mg./kg./day over a six-week period given as lead acetate.[5] Hammond and Aronson have reported that cattle may or may not exhibit toxicosis when fed hay containing 5–6

parts per million lead for extended periods of time.[11] Radeleff reported that herbage containing 25–46 parts per million lead was sufficient to poison cattle and sheep.[17] Grass near busy intersections has contained up to 500 parts per million lead,[19] undoubtedly increased by the lead expelled in automobile exhaust.

Poor farm management procedures are the main contributing factors to lead poisoning in cattle. This is supported by the occurrence and source data in this report. Allowing animals access to used oil, grease, lead batteries, chipped painted surfaces, paint buckets, and other materials in junk piles is responsible for most cases of lead poisoning in cattle.

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Student Chapter Approves New Emblem

The Iowa State Student Chapter of the American Veterinary Medical Association approved the design of a new official seal at the May meeting. The idea for the new emblem was conceived by the student public relations committee last fall and designed by William Liska of the sophomore class. The seal has the veterinary caduceus and reads "Iowa State University" and "College of Veterinary Medicine." Cardinal red and gold, Iowa State's official colors, are the colors of emphasis on the new emblem.

The seal was first introduced at the Veishea display last year. It was taken to the national convention in Las Vegas last summer and was displayed at the National Conference of Student Chapters. It is presently on display in the Dean's office and has been made into a decal which is on sale to faculty and staff from any of the Public Relations Committee members. It is the hope that this emblem can find wider use by the College of Veterinary Medicine into such items as stationary letterheads, catalogues and diplomas.