

Lead Poisoning in Small Companion Animals: An Update (1987-1992)

Rhea V Morgan DVM

Department of Urban Practice, College of Veterinary Medicine,
University of Tennessee, Knoxville TN 37901

ABSTRACT. Eighty-five cases of lead poisoning in small companion animals were retrospectively studied. Records from a lead toxicosis monitoring program at Angell Memorial Animal Hospital, Boston MA were reviewed from 1987 through 1992. The number of cases of lead poisoning substantially declined from the previous 6 y. Affected animals included 53 dogs, 20 birds, 8 cats, 3 rabbits and 1 iguana. Ages ranged from 0.3-48 y, with a median age of 1.5 y. Gastrointestinal and neurologic signs predominated. Blood lead concentrations ranged from 40 - 620 ug/dl. Seventy-three animals were successfully treated with chelation therapy; 7 animals required repeated treatments. Paint was identified as the most common source (32.4%) of lead exposure. Seventy of the 85 affected animals originated from the greater Boston area. Fifty-seven percent of the cases located within the Boston city limits could be traced to 3 inner city neighborhoods.

Lead poisoning in animals has been a widely-recognized problem in the New England area for many years. The disease in dogs was first characterized over 20 years ago in a series of papers from Angell Memorial Animal Hospital, Boston MA (1-6). During the 1970s, major steps were taken in the US to decrease the amount of lead in the environment by reducing lead content in gasoline, paints and other products. In 1972, the Hazardous Substance Act decreased lead content in paints to 0.5%, and this level was further reduced in 1977 to 0.06% (7). Following passage of the Clean Air Act in 1970, aggressive efforts were also

taken to remove lead from gasoline, resulting in a 99% reduction in the amount of alkyl lead consumed in gasoline additives between 1978 and 1988 (8).

During the 10 y following enactment of these more stringent regulations, the number of cases of lead poisoning in Boston area animals declined (9,10). Lead still remained an important toxicant, however, especially in areas with the highest percentage of people living in poverty (10). The purpose of this report is to examine data from an ongoing program that monitors the incidence, clinical and demographic trends

animals treated for lead toxicosis in the Boston area.

MATERIALS AND METHODS

Medical records of all animals diagnosed with lead poisoning at Angell Memorial Animal Hospital from 1987 through 1992 were reviewed. On the basis of blood lead values ≥ 40 $\mu\text{g/dl}$ (0.4 ppm), a positive diagnosis of lead poisoning was made in 79 animals (4,9). Six animals (5 birds, 1 iguana) without blood assays, yet with clinical signs compatible with lead toxicosis and having radiographic evidence of heavy metal in the gastrointestinal tract, were also included in the study.

The case records of all 85 animals were reviewed to obtain information on the month of diagnosis, yearly incidence, age, gender, species and breeds affected, and clinical signs and laboratory abnormalities noted. The types of treatment used, the sources of lead exposure and the geographic origins of affected animals were also analyzed.

RESULTS

Prevalence

Six to 27 cases of lead poisoning were diagnosed/y. A steady decline in the number of cases was noted during the study period. The number of positive diagnoses was 60% less than that of the preceding 6 years (Table 1). Between 1987 and 1992, lead poisoning accounted for 0.10% of all hospital admissions and 0.035% of the total hospital caseload. Sixty-two of the 85 cases of lead poisoning were diagnosed from April through September, with the highest number occurring in August and September (Fig 1).

Biographical Data

Five different types of animals were diagnosed with lead poisoning, including 53 dogs, 20 birds, 8 cats, 3 rabbits and 1 iguana. Of the dogs, 39 were purebred and represented 26 breeds. The most frequently affected breeds were the American cocker spaniel ($n=5$) and the toy poodle ($n=3$). The German shepherd-cross dog was the most commonly encountered ($n=4$) mixed-breed dog.

Table 1. Total cases of lead poisoning diagnosed per year at Angell Memorial Animal Hospital

Year	Cases	Year	Cases
1981	28	1987	27
1982	36	1988	17
1983	47	1989	16
1984	38	1990	10
1985	36	1991	6
1986	23	1992	9
Total	208		85

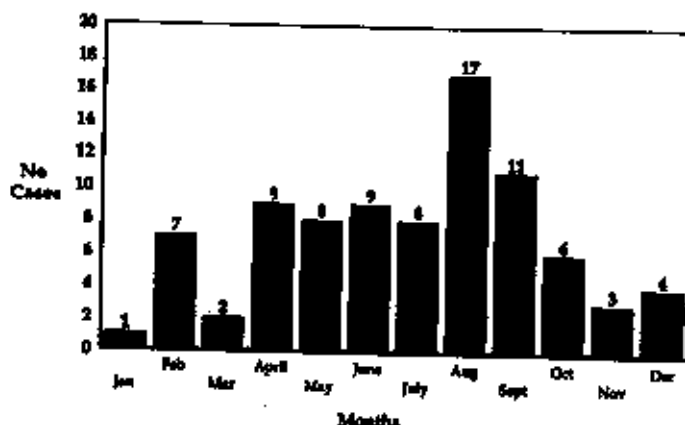


Figure 1. Distribution of diagnosed lead poisoning cases by month.

Of the 8 cats, 6 were domestic shorthair cats, 1 was a domestic longhaired cat, and 1 was a Himalayan. Affected birds were 8 parrots, 7 parakeets, 2 conures, 1 lovebird, 1 macaw and 1 cockatiel.

Males were the most frequently affected gender; castrated males were the least affected (Table 2). Ages of affected animals ranged from 0.3 to 48 y, with mean age of 3.8 y and median age of 1.5 y (Table 2).

Clinical Signs

Vomiting, anorexia, lethargy, seizures and diarrhea were the most common clinical signs (Table 3). Dogs had the greatest variety of clinical signs, with vomiting, seizures, lethargy, anorexia, diarrhea and hysteria being most frequent. Cats often exhibited anorexia, vomiting, seizures, lethargy and weight loss. Increased frequency of very liquid droppings, regurgitation, lethargy and anorexia were common in birds. Hemoglobinuria was noted in 6 parrots and 1 conure. Paresis and inability to perch occurred in 4 parakeets, 1 conure and 1 lovebird. Anorexia, weight loss, hindlimb weakness and twitching were noted in the rabbits. The iguana had anorexia, lethargy and limb rigidity.

Laboratory Findings

Nucleated red blood cells (nRBC) were found in routine blood smears in 57.6% of the animals, including dogs, cats and rabbits. Basophilic stippling of RBC was found in 35.8% of the dogs, 3 cats and 1 rabbit. Leukocytosis ($n=18$) occurred in dogs, cats and rabbits. Increased liver enzymes ($n=20$) were noted in dogs, cats, birds and rabbits. Other abnormalities included anemia ($n=17$), increased serum cholesterol ($n=9$) and hyposthenuria ($n=8$).

Radiographic studies revealed radiopaque material in the gastrointestinal tract of 22 animals, including 12 dogs, 1 cat, 8 birds and 1 iguana. Blood lead concentrations ranged from 40 to 620 $\mu\text{g/dl}$ (Table 4). Mean blood lead value for the entire group was 120 $\mu\text{g/dl}$.

Table 2. Gender and Age Distribution of Animals with Lead Poisoning

Species	Gender					Age (years)		
	Female	Surveyed Female	Male	Castrated Male	Unknown	Range	Mean	Median
Dog	12	14	30	7	0	0.3-16.0	3.2	1.5
Cat	2	4	0	1	0	0.8-7.0	3.8	4.8
Bird	9	0	7	0	8	0.8-48.0	5.7	1.3
Rabbit	1	0	2	0	0	0.5-3.5	2.0	2.0
Iguana	0	0	0	0	1	NA		
TOTAL	20	18	39	8	8	0.3-48.0	3.8	1.5

NA = Not available; age unknown

Treatment and Outcome

Chelation therapy was successful in 73 (85.9%) animals. Calcium disodium edetate (CaEDTA) was administered to 66 animals from all 5 groups. Prior to administration, the CaEDTA was diluted to a 1% solution, using 5% dextrose/water. Birds and the iguana were given 30-35 mg/kg of body weight im 3 times daily for 5 d. All other animals received 25 mg/kg sc every 6 h for 5 d.

Penicillamine was used as the primary therapeutic agent in 12 dogs. They were given 8 mg/kg po every 6 h for 7 d. Treatment was suspended for 1 w and then repeated.

The owners of 2 dogs declined treatment with either chelating agent, and these cases were lost to follow-up. A large gastrointestinal lead foreign body was surgically removed from 1 dog. Two animals were euthanized without treatment. Eight animals died, including 5 birds.

Changes in Blood Lead Concentrations

Following chelation therapy, blood lead concentration was remeasured in 26 animals. The interval from termination of treatment to submission of the follow-up sample was usually 10-14 d. Blood lead levels following CaEDTA therapy (n=20) fell 21-242 ug/dl, with a mean decrease of 75 ug/dl. After penicillamine therapy (n=4) blood lead levels declined by 21-412 ug/dl, with a mean decrease of 138 ug/dl.

Table 3. Clinical Signs in Animals with Lead Poisoning

Clinical Signs	Total Cases (%)	Dogs (%)	Cats (%)	Birds (%)	Rabbits (%)
Vomiting	41	58	50	10	0
Anorexia	39	50	75	33	100
Lethargy	28	20	38	33	0
Seizures	27	33	50	10	0
Diarrhea	-	27	25	NA	0
Abnormal Droppings	-	NA	NA	40	NA
Polypuria	-	9	13	NA	0
Regurgitation	11	4	0	33	0
Urine Weakness, Paralysis	9	2	13	33	20
Myositis	8	13	0	0	0
Weight Loss	7	8	25	5	0
Comatose	6	2	0	15	0
Generalized Weakness	6	4	0	10	0
Abdominal Pain	6	8	0	0	0
Others	8	8	0	30	30

NA = Not applicable

Chelation therapy was repeated in 7 animals either because the initial blood lead concentration was > 100 ug/dl or blood lead levels remained persistently elevated on follow-up assays. CaEDTA was repeated in 1 cat; 6 dogs received follow-up therapy with penicillamine.

Sources of Lead

The actual source of exposure of lead was known in only 41.1% of the cases. Paint was the most common source identified and was incriminated in 34.2% of the cases. Lead-containing foreign bodies were found in the gastrointestinal tract of 4 animals. Two animals were believed to contract lead poisoning from exposure to lead in soil and from a wine bottle covering.

Geographic Origins

Eighty-four cases originated in Massachusetts; 1 animal was from Rhode Island. Of the Massachusetts cases, 14 came from outside the greater Boston area. Thirty-five affected animals lived within the city limits of Boston and 35 lived in communities immediately adjacent to the city.

Boston is divided into 14 distinct neighborhoods. Angell Memorial Animal Hospital is located in Jamaica Plain and is approximately 1.5 miles from the geographic center of the city. Fifty-seven percent of the city's lead poisoning caseload derived from 3 specific neighborhoods: Jamaica Plain, Dorchester and South Boston (Fig 2). High numbers of lead toxicosis were also found in the adjacent city of Cambridge.

DISCUSSION

Prevalence and Seasonality

Lead poisoning has been commonly recognized at Angell Memorial Animal Hospital for over 25 years (6). From 1969-1971 lead poisoning was diagnosed in 965 animals, representing approximately 0.85% of all hospital admissions and 0.21% of the total caseload. Between 1977-1986 (347 cases) the rate decreased to 0.25% of all hospital admissions and 0.07% of the total caseload (9). The 85 cases reported here represented 0.10% of all hospital admissions and 0.035% of the total caseload from 1987-1992.

The first notable decrease in the incidence

Table 4. Blood lead concentrations of affected animals (ug/dl)

Species	Range	Mean
Dogs	40-460	106
Cats	40-500	175
Birds	45-620	147
Rabbits	50-72	61

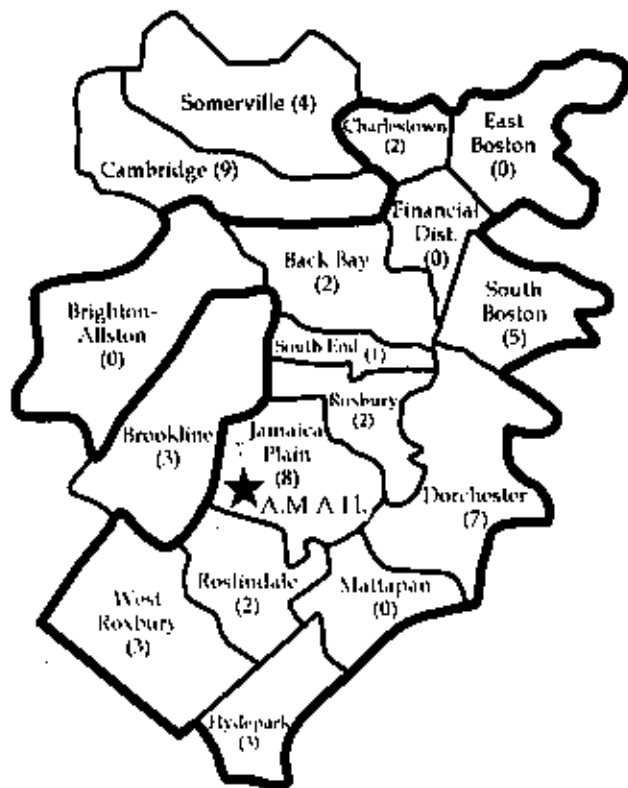


Figure 2. Map of Boston with its respective neighborhoods and adjacent communities. The city limits of Boston are demarcated by the darkest lines. Numbers in parentheses represent the cases of lead poisoning from each sector. AMAH = Angell Memorial Animal Hospital.

of lead poisoning at Angell Memorial Animal Hospital began within 1 y of the passage of the Hazardous Substance Act. Beginning in 1973, the number of cases diagnosed steadily declined, and this trend continued throughout the current study period. Although specific reasons for the most recent decline are not readily apparent, decreased exposure rates from lowered levels of lead in the environment or greater owner awareness of the potential sources of lead may play a role.

A seasonal trend similar to previous reports was found in this study, with the highest number of cases occurring in the spring/summer and the lowest numbers in late fall/early winter (11,12). No epidemiologic studies exist to explain the seasonal nature of lead poisoning, but changes in household activity, increased hours spent in contact with dust and soil, and alterations in lead absorption from exposure to sunlight have all been postulated as contributing factors (12). The seasonal trend noted in this report occurred not only in dogs, but also in cats and birds. Since several of the cats and all of the birds were confined indoors, the latter 2 postulates may have had no clinical impact.

Clinical Trends

Although dogs remained the most common species diagnosed with lead toxicosis,

the distribution of plumbism among animal groups changed during the study period. From 1987-1992, 62.4% of the cases were dogs, 23.5% were birds, 9.4% were cats and 3.5% were rabbits. Between 1977 and 1986, dogs represented 80.9%, birds 15%, cats 2.9% and rabbits 0.6% of the cases (9). Since the species distribution in the hospital caseload has remained constant for the last 23 y (dogs = 70%, cats = 28% of caseload), lead poisoning has actually decreased in dogs and increased in the other groups. These results support the heightened awareness of lead as an important toxicant in caged birds and other types of animals (13,14).

No major changes were noted in the breeds, gender or ages of the animals affected in this study compared to those of prior reports (1,3,6,9,11,12). The predominate clinical signs and laboratory abnormalities encountered were also similar to other studies (9,12).

Treatment Outcome

Chelation therapy with CaEDTA remained the most common therapy administered to animals with plumbism. Treatment with either CaEDTA or penicillamine was successful in most animals; overall mortality in the study was 9.4%. Compared with the previous 10 years, mortality in the dogs declined (1.8% vs 5.6%), but mortality in the birds increased (25% vs 15%) (10). One rabbit and the iguana also died.

Source of Lead

The actual source of exposure to lead for most cases (58.9%) was unknown. Recently emphasis has been placed on the importance of dust, soil, water and airborne lead as sources of exposure, but like similar studies in children, paint chips remained the most common source identified for affected animals (8,15-18).

Geographic Origins

Most cases of lead poisoning originated within the greater Boston area. This may be explained by the physical location of Angell Memorial Animal Hospital and by the general trend of plumbism to be an urban disease (8,17,19,20). Between 1977-1986, the number of lead poisoning cases originating within the city limits of Boston was twice that of the adjacent communities (10). In this study, 35 cases came from Boston and 35 came from adjacent cities. Whether these figures represent an actual decrease in lead poisoning from within the city is uncertain. Heightened awareness that lead toxicosis is no longer confined to the inner city may have led to increased screening of animals in the neighboring areas (8).

Cases from within the city of Boston were scattered throughout the city, but over half could be localized to the 3 neighborhoods of Jamaica Plain, Dorchester and South Boston. Together with the Roxbury section of Boston these neighborhoods were also

possible for the majority of cases of lead poisoning the previous 10 years. Demographically, these neighborhoods have the highest percentage of people living in poverty, which is a proven positive correlate to lead poisoning in animals (10,21).

New Developments

During the 6 y studied, 7 animals were admitted with clinical signs compatible with lead poisoning, but with blood lead concentrations < 40 ug/dl. The animals included 4 dogs, 2 birds and 1 rabbit. In 4 of these animals nRBCs were found in the peripheral blood, and serum liver enzymes were elevated. Anemia was noted in 3 animals and 2 had radiopaque material in their gastrointestinal tract. Blood lead levels ranged from 12-36 ug/dl. Clinical signs resolved in all 7 animals following chelation therapy with CaEDTA.

It is interesting to note that these 7 animals had clinical evidence of lead intoxication, yet their blood lead values were < 40 ug/dl. Over the years, the threshold value to define lead poisoning in animals has decreased from 60 ug/dl to 40 ug/dl (3,9,22,23). A similar change has occurred in people. The 1978 blood lead level criteria for lead intoxication in people was 30 ug/dl (24). This value was lowered in 1985 to 25 ug/dl, and recently toxic effects of lead have been documented at blood lead levels previously considered noninjurious (24,25). Several studies have shown that neurotoxic developmental changes may occur in children with blood lead levels of 10-15 ug/dl (8,17,24,25). The discovery of 7 animals in this study with clinically apparent plumbism and low blood lead concentrations suggests that the criteria for lead poisoning in animals could also be reevaluated.

CONCLUSIONS

Based on the data presented here, the overall prevalence of lead poisoning in small companion animals appears to be declining. This trend reflects a decrease in the number of affected dogs, yet lead remains an important toxicant in birds and other species. While inner city neighborhoods are over-represented in the geographical distribution of lead toxicosis in Boston, a proportionate increase in the number of lead poisoning cases also occurred in communities immediately adjacent to the city. Evidence of clinical lead poisoning in animals with low blood lead levels requires that further studies be performed to redefine the diagnostic value of blood lead

concentrations and to review the criteria for institution of chelation therapy.

REFERENCES

1. Zook BC, Carpenter JL, Leads EB: Lead poisoning in dogs. *J Am Vet Med Assoc* 155: 1329-1341, 1969.
2. Zook BC, McConnell C, Gilmore CE: Basophilic stippling of erythrocytes in dogs with special reference to lead poisoning. *J Am Vet Med Assoc* 157: 2092-2099, 1970.
3. Zook BC, Carpenter JL, Roberts RM: Lead poisoning in dogs: Occurrence, source, clinical pathology, and electroencephalography. *Am J Vet Res* 33: 891-902, 1972.
4. Zook BC, Kopito L, Carpenter JL et al: Lead poisoning in dogs: analysis of blood, urine, hair and liver for lead. *Am J Vet Res* 33: 903-909, 1972.
5. Zook BC: The pathologic anatomy of lead poisoning in dogs. *Vet Pathol* 9: 310-327, 1972.
6. Zook BC: Lead intoxication in urban dogs. *Clin Toxicol* 6: 377-388, 1973.
7. Consumer Product Safety Commission: Ban of lead-containing paint and certain consumer products bearing lead-containing paint. Consumer Product Safety Commission, Washington DC, Title 16, Code of Federal Regulations, Sections 1303.1-1303.5, 1977.
8. Needleman HL: The persistent threat of lead: medical and sociological issues. *Curr Probl Pediatr* 18: 703-744, 1988.
9. Morgan RV, Moore FM, Pearce LK et al: Clinical and laboratory findings in small companion animals with lead poisoning: 347 cases (1977-1986). *J Am Vet Med Assoc* 199: 93-97, 1991.
10. Morgan RV, Pearce LK, Moore FM et al: Demographic data and treatment of small companion animals with lead poisoning: 347 cases (1977-1986). *J Am Vet Med Assoc* 199: 98-102, 1991.
11. Hamir AN: Lead poisoning of dogs in Australia. *Vet Rec* 108: 438-439, 1981.
12. Berry PJ, Cote LN, Buck WB: Case reports of lead poisoning in dogs from the National Animal Poison Control Center and the Centre National d'Informations Toxicologiques Veterinaires: Anecdotes or reality? *Vet Hum Toxicol* 34: 26-31, 1992.
13. Tully TN Jr, Morris JM: Lead toxicosis: a danger lurking in many forms. *Proc Assoc Avian Vet* 369-371, 1990.
14. Mautino M: Avian lead intoxication. *Proc Assoc Avian Vet* 245-247, 1990.
15. Khamis C, Boermans HJ, Woods P et al: Lead toxicosis and changes in the blood lead concentration of dogs exposed to dust containing high levels of lead. *Can Vet J* 33: 815-817, 1992.
16. McElvaine MD, DeLungria EG, Matte TD et al: Prevalence of radiographic evidence of paint chip ingestion among children with moderate to severe lead poisoning. St. Louis, Missouri, 1989 through 1990. *Pediatrics* 89: 740-742, 1992.
17. Simon P, Zimmerman A: Childhood lead poisoning: A Rhode Island perspective. *RI Med J* 74: 287-292, 1991.
18. Berde N, Wietlisbach V, Rickenbach M et al: Lifestyle and environmental factors as determinants of blood lead levels in a Swiss population. *Environ Res* 55: 1-17, 1991.
19. Mielke HW, Anderson JC, Berry KJ et al: Lead concentrations in inner-city soils as a factor in the child lead problem. *Am J Public Health* 73: 1366-1369, 1983.
20. Thomas CM, Rising JL, Moore JK: Blood lead concentrations in three groups of dogs from a suburban Illinois community. *J Am Vet Med Assoc* 187: 995-999, 1975.
21. Government Documents: Boston: neighborhood profiles, commissioned by Mayor Raymond L. Flynn, 1988.
22. Hoffmeyer MS: Lead poisoning in a cat. *Compend Contin Educ Pract Vet* 10: 724-728, 1988.
23. Stratton GR, Kowalczyk DR: Lead poisoning. In Kirk RW: Current Veterinary Therapy 10, Small Animal Practice. WB Saunders, Philadelphia: 152-159, 1989.
24. Current trends: Childhood lead poisoning. *MMWR* 37: 481-485, 1988.
25. Davis JM: Risk assessment of the developmental neurotoxicity of lead. *Neurotoxicol* 11: 285-292, 1990.

Bosses should be wary of overvaluing their own knowledge and judgement. Effective: Insist that subordinates question your assumptions and opinions—without fear of retribution. If your position is right, you can explain it to them and they'll get a better understanding of the business. And if you're making a mistake, you'll want it to be pointed out before it becomes costly.

Aging Comedian Milton Berle relates this story. "I went to a Rest Home for Elderly Citizens where most folks greeted me with 'Yea, Uncle Miltie!' However, I spoke to one little old lady, who was 93, and dozing in a wheelchair. I put my arm around her and asked, 'Do you know who I am?' She looked up at me and said, 'No, but if you ask at the front desk, they will tell you'."