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Emalee Gottbrath Flaherty

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Risk of Lead Poisoning in Abused and Neglected Children

Emalee Gottbrath Flaherty, M.D.

Summary: This study examined the prevalence of lead poisoning in 746 abused and neglected children taken into protective custody by Cook County, Illinois, Child Protective Services, Department of Children and Family Services (DCFS). Most of these children had a history of neglect (62%) or dependency (23%); they were predominantly African-American (88%). Venous blood lead concentrations (PbB) were obtained on 589 children, ages 6 months to 6 years, between January 1, 1992, and December 31, 1992. Three hundred eighty-one (64.7%) children tested had a PbB ≥ 10 $\mu\text{g}/\text{dL}$ (0.48 $\mu\text{mol}/\text{L}$); the mean PbB was 12.23 $\mu\text{g}/\text{dL}$ (0.587 $\mu\text{mol}/\text{L}$). These data support a recommendation for lead screening in any child with a history of abuse or neglect if environmental risk factors cannot be assessed.

Introduction

Lead exposure in children causes serious and permanent neurologic damage. Blood lead is inversely related to cognitive ability and is associated with an increased risk for behavioral problems.¹⁻³ Since the Centers for Disease Control and Prevention (CDC) determined these adverse effects could be associated with PbB as low as 10 $\mu\text{g}/\text{dL}$ (0.48 $\mu\text{mol}/\text{L}$), lead poisoning has been identified as the most common chronic illness of childhood.⁴

In October 1991, the CDC rec-

ommended universal lead screening for all children from age 6 months to 6 years.⁴ Several studies have questioned the need for universal testing after demonstrating that the prevalence of elevated lead levels is quite low in some areas.⁵⁻⁸ These authors urge that the recommendations be revised and suggest that careful questioning about the home environment will identify those children at risk for lead poisoning. This recommendation poses a problem for physicians caring for abused and neglected children in protective custody and foster care because, typically, little information is available about the victims' previous medical history, and even less information can be obtained about lead paint hazards that may have existed in previous homes.

Bithoney et al⁹ reported that children suspected of having been physically abused had significantly higher PbB than a comparison group. They also demonstrated

that children with nonorganic failure to thrive had elevated PbB.¹⁰ However, these data were collected before 1990. Blood lead concentrations in this country have declined significantly since efforts have been made to reduce the environmental exposure to lead.¹¹⁻¹³ The National Health and Nutrition Examination Surveys (NHANES) found that the mean blood lead dropped 77% (13.7 - 3.2 $\mu\text{g}/\text{dL}$ [0.658 - 0.154 $\mu\text{mol}/\text{L}$]) between 1976 and 1991 in children aged 1 to 5 years.

We hypothesized that (1) despite the reduction of lead in the environment, abused and neglected children continue to be a high-risk group for lead poisoning; (2) compared with other categories of child-protective service intervention, neglected children have the highest risk for elevated lead concentrations; and (3) young age and summer season would increase their risk for lead poisoning.

Department of Pediatrics
Columbus-Cabrini Medical Center and
Department of Pediatrics
Northwestern University Medical School
Children's Memorial Hospital
Chicago, Illinois

Address correspondence to: Emalee
Gottbrath Flaherty, M.D., Columbus-Cabrini
Medical Center, Department of Pediatrics,
2520 North Lakeview, Chicago, IL 60614

Table 1

LEAD LEVEL COMPARISONS

	Abused and neglected children study	National study	Cook County	Chicago	Other urban	Urban/suburban	Rural	
Site	Chicago	NHANES III ¹¹	Chicago, 1991	Chicago, 1992	Washington DC ¹⁶	Syracuse, NY ²⁰	Minnesota/St. Paul ⁶	North Carolina ¹⁴
Year of study	1991	1988-1991	1991	1992	1991	1991	10/91-8/92	11/92-4/93
N	589	2,234	223	410	2,057	233	4,678	20,720
µg/dL (µmol/L)								
	%	%	%	%	%	%	%	%
≥10 (0.48)	64.7	18.8	17.0	NR	32.6	2.5	20.2	
≥15 (0.72)	25.3	5.8	4.9	NR	NR	0.9	3.2	
≥20 (0.97)	8.7	2.6	0.8	NR	1.7	0.5	1.1	
≥25 (1.21)	4	1.1	1	NR	1.5	NR	NR	
Mean µg/dL (µmol/L)	12.2 (0.58)	3.7 (0.18)	NR	NR	NR	NR	7.4 (0.355)	
NR = Not Reported								

NR = Not Reported

Methods

In 1992, Cook County Department of Children and Family Services (DCFS) took protective custody of 746 children who were between the ages of 6 months and 6 years; 79% of these children were taken to the Columbus-Maryville Reception Center for a medical evaluation prior to their placement in a foster home or other residential facility. DCFS bypassed this center if it had already located a placement for the child, usually in the home of a relative.

The children brought to this reception center were primarily African-American (88%). Only 6% of these children were Caucasian and 5% Hispanic. African-American children were overrepresented:

only 33% of young children in Cook County are African-American. This same disproportionate representation is noted in the reporting of abuse and neglect in Illinois: African-American children account for 41% of abuse reports although they make up only 19% of the population.

On admission to the reception center, the children receive a complete medical examination and screening tests appropriate for their age. A blood lead concentration is ordered on all children between the ages of 6 months and 6 years. All lead measurements are performed on venous blood by the Chicago Board of Health laboratory.

A retrospective chart review was conducted on all children who had

blood lead levels done between January 1, 1992, and December 31, 1992, and whose age was between 6 months and 6 years at the time of the exam. The child's age, reason for coming into protective custody, date of the examination, and blood lead level were recorded.

Results

Children were brought to the center for the following reasons: neglect (62%), dependency (23%), abuse (5%), and risk of harm (1%). No reason for removal was documented for the remaining 9% of children. Children are taken into custody for dependency when no family member can care for the child, e.g., the mother is hospital-

Table 2

LEAD LEVEL BY REASON FOR REFERRAL (N = 527)

Reason for referral	<10 µg/dL (0.48 µmol/L)		≥10 µg/dL (0.48 µmol/L)		Mean µg/dL (µmol/L)
	n	%	n	%	
Abuse	12	44.4	15	55.6	12
Neglect	117	33.3	234	66.7	12
Risk of harm	5	71.4	2	28.6	12
Dependency	54	38.0	87	62.0	12

$\chi^2 = 6.29$, $df = 4$, $P = 0.18$ (not statistically significant)

ized or incarcerated. "Risk of harm" refers to those children residing in homes where another child has been found to be significantly abused or neglected and the welfare agency determines that this child may be at risk for harm if the child remains in that home.

A substantial number of these abused and neglected children, 381, or 64.7%, had PbB concentrations of ≥ 10 µg/dL (0.48 µmol/L) or higher (Table 1). The mean PbB for these children was 12.23 µg/dL (0.587 µmol/L), with concentrations ranging from 1-61 µg/dL (0.048-2.928 µmol/L). Most children had a mild elevation of their PbB, with values between 10 and 25 µg/dL (0.48 and 1.21 µmol/L). Only three children had a PbB of 40 µg/dL (1.92 µmol/L).

Blood lead concentrations were compared with the reason for custody. Children removed because of neglect and dependency had a higher prevalence of PbB concentrations ≥ 10 µg/dL (0.48 µmol/L) than children removed because of abuse and risk of harm (Table 2), although the differences between categories were not statistically significant.

The age of the child at the time

of the evaluation was also analyzed. The percentage of children with PbB levels ≥ 10 µg/dL (0.48 µmol/L) rose rapidly after age 1 and peaked between 24 and 36 months, when 68.7% of the children had elevated levels. More than 64% of the children age 3 to 6 years continued to have PbB ≥ 10 µg/dL (0.48 µmol/L) [$\chi^2 = 5.47$, $P = \text{N.S.}$]

The season of the year did not appear to affect lead levels. Elevated blood lead concentrations were seen most often in December, when 84.6% of the children had PbB concentrations ≥ 10 µg/dL (0.48 µmol/L). The lowest percentage of elevated levels was seen in March. No particular seasonal pattern was identified because more than 70% of the study population was found to have elevated lead levels in February, June, August, November, and December. Approximately equal numbers of children were screened each month ($\chi^2 = 10.49$, $P = \text{N.S.}$).

Discussion

Our results demonstrate that the majority of abused and neglected children in Cook County

are at high risk for lead poisoning; 64.7% of the children in our study had elevated blood lead concentrations. This high prevalence alone establishes this group as a high-risk group. The 21% of Cook County neglected-abused who bypassed this facility may somehow differ from this study population and may have a different prevalence. Our results indicate that these children also should be screened unless it is subsequently determined that they are not at risk for plumbism.

These results may not be generalizable to abused and neglected children from other urban, suburban, or rural areas. But because the prevalence of the problem is so high in our study group, this potential problem should be further investigated in other populations.

Despite lack of a control group, these results are important because the prevalence of lead poisoning in our study group is substantially higher than the prevalence reported in other studies conducted during the same time period (Table 1). Our group was two to three times as likely to have elevated PbB as children residing in urban and rural areas and 30 times as likely to have elevated

PbB as children residing in suburban areas.^{5-8,11,14-17} Blood lead concentrations $>25 \mu\text{g/dL}$ ($1.21 \mu\text{mol/L}$) were three to four times more prevalent in our study group.

Because the children in our study population were primarily African-American, we compared our results to the NHANES III data for African-American children ages 1 to 5 years living in a central city with a population ≥ 1 million. Only 36.7% of that population had a PbB $\geq 10 \mu\text{g/dL}$ ($0.48 \mu\text{mol/L}$).¹¹ The prevalence of elevated PbB in our group was two to three times the prevalence in the two groups in Table 1 with substantial African-American representation — North Carolina (51% African-American) and Rochester (64% African-American).^{14,17} Previous studies also have shown that children who are African-American, with family incomes less than \$6,000, and living inside central cities with >1 million population have the highest risk for lead poisoning. Etiologic reasons for these findings are lacking.

Children removed because of neglect had the highest prevalence of plumbism; the majority of children removed because of abuse and dependency had elevated PbB. Although only 28.6% of those removed because of risk of harm had PbB concentrations $\geq 10 \mu\text{g/dL}$ ($0.48 \mu\text{mol/L}$), even this group had a higher prevalence rate than reported in other Chicago-based studies.

This study did not investigate the reason these children were at such high risk. Although we could not determine the socioeconomic status of the families, we can speculate that they had fewer financial resources and were more likely to live in deteriorating old housing. More information about their previous housing would have contributed to our understanding, but, unfortunately, addresses for these

families were frequently not available and often inaccurate. Because the neglected children had the highest risk, we can speculate that the parents' failure to recognize or address environmental risks and their failure to adequately supervise their children may have increased the risk for their children.

These parents may not have fed their children a proper diet; deficiencies in iron, calcium, protein, and zinc have been shown to be related to increased blood lead levels.¹⁹ Although we did not evaluate the nutritional status of our study group, we do know that iron deficiency anemia is more common in children brought to this facility than in the general population.²⁰

The prevalence of lead poisoning peaked in December. Contrary to our hypothesis, we did not identify a seasonal pattern, an outcome different from the summer peak noted in other studies.^{6,11,12,21} Blatt and Weinberger²¹ found that the prevalence of elevated blood lead concentrations during the summer months was twice the prevalence during the winter months. They speculated that the seasonal elevation was caused by increased exposure to lead-contaminated dirt as children played outside in the summer. Others have speculated that summer sun exposure may cause an escalation in bone turnover; the lead stored in the bone is recirculated into the soft tissue pool, increasing the measurable blood lead level.

We do not know why our sample did not demonstrate this summer peak. We can speculate that its source of lead exposure may have been different or that its play patterns may have been dissimilar. Children living in public housing in large urban areas where violence is prevalent may stay inside their homes during the warmer months. If the home contains lead-

based paint, spending extended hours in this environment may increase their exposure to lead at all times of the year. This speculation deserves further investigation.

The peak elevation of lead at age 2 has been noted in previous studies.^{7,11,12,21} This finding gives further credence to the American Academy of Pediatrics' recommendation that children at risk for lead poisoning be tested initially before 18 months and again between 18 and 24 months. Because a large percentage of children continued to have elevated PbB through age 5, children at risk or with previously elevated PbB should continue to be tested until at least this age.

Children who have been abused and neglected are already at risk for learning problems, school failure, and maladaptive behavior. Elevated PbB places these children at further risk for cognitive and behavioral problems. Many abused and neglected children have not had adequate medical care, particularly health supervision care. If they are removed from their homes and placed in foster care, their medical care generally becomes even more discontinuous and sporadic. Information about medical problems is frequently not passed on to new health-care providers. The results of our study indicate that physicians should make screening of this population a high priority. They should monitor any elevations closely and institute appropriate treatment and careful follow-up of this high-risk group.

Conclusions

Abused and neglected children have a high prevalence of lead poisoning. If a physician cannot assess environmental risk factors, abused and neglected chil-

dren should be screened for lead poisoning. Careful tracking and appropriate treatment should be instituted for all children identified to have elevated PbB.

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REFERENCES

1. Dietrich KN, Berger OG, Succop PA. Lead exposure and the motor developmental status of urban six-year-old children in the Cincinnati Prospective Study. *Pediatrics*. 1993;91:301-307.
2. Needleman HL, Gatsonis CA. Low-level lead exposure and the IQ of children. *JAMA*. 1990;263:673-678.
3. Bellinger DC, Stiles KM, Needleman HL. Low-level lead exposure, intelligence and academic achievement: a long-term follow-up study. *Pediatrics*. 1992;90:855-861.
4. Centers for Disease Control. *Preventing Lead Poisoning in Young Children: A Statement by the Centers for Disease Control*. Atlanta, GA: U.S. Department of Health and Human Services; October 1991.
5. Binns HJ, LeBailly SA, Poncher J, et al. Is there lead in the suburbs? Risk assessment in Chicago suburban pediatric practices. *Pediatrics*. 1994;93:164-171.
6. Nordin JD, Rolnick SJ, Griffin JM. Prevalence of excess lead absorption and associated risk factors in children enrolled in a midwestern health maintenance organization. *Pediatrics*. 1994;93:172-177.
7. Tejeda DM, Wyatt DD, Rostek BR, et al. Do questions about lead exposure predict elevated lead levels? *Pediatrics*. 1994;93:192-194.
8. Gellert GA, Wagner GA, Maxwell RM, et al. Lead poisoning among low-income children in Orange County, California. *JAMA*. 1993;270:69-71.
9. Bithoney WG, Vandeven AM, Ryan A. Elevated lead levels in reportedly abused children. *J Pediatr*. 1993;122:719-720.
10. Bithoney WG. Elevated lead levels in children with nonorganic failure to thrive. *Pediatrics*. 1986;78:891-895.
11. Brody DJ, Pirkle JL, Kramer RA. Blood lead levels in the US population: Phase 1 of the Third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1991). *JAMA*. 1994;272:277-283.
12. Hayes EB, McElavine MD, Orbach HG, et al. Long-term trends in blood lead levels among children in Chicago: relationship to air lead levels. *Pediatrics*. 1994;93:195-200.
13. Pirkle JL, Brody DJ, Gunter EW, et al. The decline in blood lead levels in the United States: The National Health and Nutrition Examination Surveys (NHANES). *JAMA*. 1994;272:284-291.
14. Norman EH, Bordley WC, Hertz-Picciotto I, et al. Rural-urban blood lead differences in North Carolina children. *Pediatrics*. 1994;94:59-64.
15. Selvarajan RM, Nunez A. Lead toxicity and iron deficiency anemia in children. *Chicago Medicine*. 1994;97:27-29.
16. Rifai N, Faser C, Cohen G, et al. Lead poisoning in young children in Washington, DC: a crisis that remains to be addressed. *Am J Dis Child*. 1992;146:1259-1260.
17. Schaffer SJ, Szilagyi PG, Weitzman M. Lead poisoning risk determination in an urban population through the use of a standardized questionnaire. *Pediatrics*. 1994;93:159-163.
18. Crocetti AF, Mushak P, Schwartz J. Determination of numbers of lead-exposed U.S. children by areas of the United States: and integrated summary of a report to the U.S. Congress on childhood lead poisoning. *Environ Health Perspect*. 1990;89:109-120.
19. Mahaffey KR. Nutritional factors in lead poisoning. *Nutr Rev*. 1981;39:353-362.
20. Flaherty EG, Weiss H. Medical evaluation of abused and neglected children. *Am J Dis Child*. 1990;144:330-334.
21. Blatt SD, Weinberger HL. Prevalence of lead exposure in a clinic using 1991 Centers for Disease Control and Prevention recommendations. *Am J Dis Child*. 1993;147:761-763.