Pediatric Lead Screening in a Suburban Family Practice Setting

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Background. The adverse effects of lead on behavioral and intellectual development have been recognized for many years. During the past 10 years several studies have shown that lead is toxic to children at levels previously thought to be harmless. Black children living in urban neighborhoods have been identified as being at greatest risk.

Methods. To determine whether children seen in a suburban family practice center were being exposed to lead, voluntary screening of 1-year-old patients was performed.

Results. Over a 7-month period venous specimens for blood lead level were obtained from 40 children. Seventeen (43%) of the children had levels of 0.0 to 0.2 μmol/L (0 to 4 μg/dL). Fifteen (38%) had levels of 0.24 to 0.43 μmol/L (5 to 9 μg/dL). Eight (20%) children had levels of 0.48 μmol/L (10 μg/dL) or greater. The highest value obtained was 0.82 μmol/L (17 μg/dL). Seventy-five percent of the children with significantly elevated lead levels resided in suburban communities.

Conclusions. Children seen in a suburban family practice setting are at risk for lead exposure, and screening should be considered by primary care physicians who practice in nonurban settings.

Key words. Lead; pediatrics; mass screening.
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Despite the apparent reduction in cases of lead poisoning in American children over the past decade, lead remains a significant health hazard in this country. Recent data indicate that millions of children are being exposed to lead through a variety of sources.[1,2] These sources include leaded paint, drinking water, ambient air, food, and soil. This exposure is particularly true among black children who reside in areas of urban poverty.[3]

While it was once believed that blood lead levels less than 1.9 μmol/L (40 μg/dL) were safe, this is no longer the case. The Centers for Disease Control (CDC) considers a level greater than 1.2 μmol/L (25 μg/dL) to be abnormal; however, studies done since 1980 continue to show that levels of 0.7 μmol/L (15 μg/dL) are associated with adverse cognitive, behavioral, and neurodevelopmental effects.[4-6] In addition, levels of 0.2 to 0.7 μmol/L (5 to 15 μg/dL) have been shown to interfere with hemesynthesis and vitamin D-dependent processes throughout the body.[5] More concretely, a study by Bellinger et al[6] published in 1987 showed lead levels greater than 0.5 μmol/L (10 μg/dL) to be associated with a five-point decrease in intelligence quotient scores. In response to some of the above studies and others, the Agency for Toxic Substances decreased the threshold for neurobehavioral lead toxicity to 0.5 to 0.7 μmol/L (10 to 15 μg/dL).[7]

Lead exposure has also been shown to affect cellular growth, impair hearing, and suppress both the humoral and cell-mediated components of the immune system.[3] As further scientific evidence becomes available, it is likely that no degree of lead exposure will be considered acceptable or safe.

Childhood lead poisoning is currently defined by the American Academy of Pediatrics (AAP) and the CDC as whole blood concentrations of 1.2 μmol/L (25 μg/dL) or greater with an erythrocyte protoporphyrin level of 0.6 μmol/L (35 μg/dL).[1,8] This definition does not require the presence of symptoms. Recommendations of the Agency for Toxic Substances and Disease Registry (ATSDR), which released its report to the US Congress in 1988, included (1) establishing and maintaining effective lead-screening programs, (2) conducting further intensive re-

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search on childhood lead poisoning, and (3) developing better environmental measurement techniques.[7]

Routine lead screening is still done in many urban clinics; however, it has not been part of the usual well-child care in suburban settings such as Abington (Montgomery County), Pennsylvania, because this patient population was not felt to be at risk for lead exposure. As a result of more recent findings related to lead toxicity, the AAP recommended in 1987 that all children in the United States at risk for lead exposure be screened at approximately 1 year of age.[1] Similar screening guidelines were also issued in 1989 by the US Preventive Services Task Force.[8]

It was through the consideration of the above recommendations that a lead-screening study was initiated involving children seen at the suburban Abington Memorial Hospital Family Practice Center. Although the majority of patients seen in this practice lived in a nonurban setting, some did have potential risk factors for lead toxicity including being of Afro-American heritage and of lower socioeconomic class. It was attempted to determine whether these children were being exposed to lead, and if so, to what degree.

Methods

The study population comprised children seen by resident physicians at a hospital-based family practice center from November 1988 until May 1989. Basic criteria for enrollment included age of approximately 12 months (range of 11 to 13 months). Privately insured, self-paying, and Medicaid patients were all given the option to participate. The cost of the laboratory tests, however, was not paid for by private insurers; thus children covered by Medicaid ultimately made up 92% of the total population studied.

One year was chosen as the age studied because it was consistent with the AAP recommendation for screening. In addition, 1 year is the age at which these patients are routinely screened for anemia. Lead screening was not offered to other siblings or the parents at the time of the visit, as the focus of the study was strictly 1-year-old children.

During the time of the 1-year well-child visit, a form explaining the lead-screening project was given to the primary caretaker. The caretaker was asked to read the form, then complete a brief questionnaire that included the child's date and place of birth (hospital), residence during the past 12 months, and the approximate age of their house or apartment. It was the responsibility of the resident physician to address any questions the caretaker had concerning the study. Completion of the form, including a signature, was interpreted as consent to participate in the study.

After the office visit, the child was sent to the outpatient laboratory where venous sampling was performed to obtain a complete blood count and a blood lead level. The complete blood count was done in the hospital laboratory and the blood sample for lead was sent to the Smith-Kline laboratory in Norristown, Pennsylvania. Results were reported directly to the physicians at the family practice center.

Although the AAP and the US Preventive Services Task Force recommend using erythrocyte protoporphyrin as the initial screening test for lead exposure, it was decided to use blood lead, as the cost for this latter test was significantly less than the erythrocyte protoporphyrin. In addition, the sensitivity of the erythrocyte protoporphyrin test is only 50% in detecting levels less than 2.4 μmol/L (50 μg/dL), which limits its usefulness in early detection of lead exposure.[8]

Results

As of May 1989, a total of 40 patients were entered into the study. During the previous 7 months, approximately 105 children were seen for 1-year well-child visits. Of this total, 60% had Medicaid as their health insurance. It is therefore estimated that 63% of financially eligible children participated in the study.

In terms of sex distribution, 21 of the 40 children were female. Racial distribution broke down to 20 white children, 18 black children, 1 child of Indian (Asian) heritage, and 1 child of mixed parentage.

Geographically, the children lived primarily in Montgomery and Philadelphia counties, with three children residing in Bucks County. This variation is consistent with the overall makeup of the practice population, of which 60% is from Montgomery County, 30% from Philadelphia County, and 10% from Bucks County.

Thirty-six of the 40 children were born at the Abington Memorial Hospital.

In 17 of the children there was no lead detected in the specimens tested, indicating a level of 0.0 to 0.2 μmol/L (0 to 4 μg/dL). None of these children lived in Philadelphia County. Lead levels ranging from 0.24 to 0.43 μmol/L (5 to 9 μg/dL) were found in 15 of the children. Eight patients had lead levels of 0.48 μmol/L (10 μg/dL) or greater.

Of the eight patients with the highest lead levels, none was significantly anemic. Six lived in Montgomery County and two lived in Philadelphia County. Two of the eight children lived in housing built before 1960, when the use of interior lead-based paint was common.

Table 1. Data on Patients Having a Blood Lead Level of 0.48 μmol/L (10 μg/dL) or Greater

Patient	Sex	Race	Lead Level (μmol/ L)	Hematocrit	Age of Housing (years)*	County
1	F	W	0.67	0.363	10	Montgomery
2	M	B	0.53	0.350	20	Philadelphia
3	F	B	0.53	0.382	15	Montgomery
4	F	B	0.50	0.340	5	Montgomery
5	F	B	0.63	0.342	10	Montgomery
6	M	M†	0.53	0.340	50	Montgomery
7	M	B	0.50	0.372	10	Montgomery
8	M	I‡	0.82	0.368	30	Philadelphia

F—Female, M—male, B—black, W—white.
**Age of housing is estimated by parents.*
†*Child of mixed (black/white) heritage.*
‡*Child of Indian (Asian) heritage.*

Discussion

Looking collectively at all 40 patients, several aspects of the study are noteworthy. None of the children tested had a level of greater than 1.2 μmol/L (25 μg/dL), the current toxic level as defined by the CDC and the AAP. Seventeen (43%) of the children had no detectable lead in their blood, and an additional 15 (37%) had levels less than 0.48 μmol/L (10 μg/dL). This 80% total is considerably less than the National Health and Nutrition Examination Survey (NHANES II)[9] reported, in which only 16% of their 6-month to 2-year-old population had levels less than 0.48 μmol/L (10 μg/dL). It is also much less than the estimated 55% of urban black children found to have lead levels of 0.70 μmol/L (15 μg/dL) or greater.[8]

The data in Table 1 include the remaining 20% of the patients with potentially harmful lead levels. The number of black children make up 75% of the total, even though fewer than one half of all the children tested were black. This finding is in keeping with the data of Needleman[3] and Mahaffey and Annest,[9] in whose studies black children were found to have lead levels an average of 0.3 μmol/L (6 μg/dL) greater than white children. This difference occurred irrespective of social class or place of residence and has yet to be fully explained.

Sex differences in lead levels have not been previously significant. This study showed an equal male-to-female distribution.

In terms of geographic location, urban populations are still considered to be at greatest risk for lead toxicity.[10,11] This risk is primarily due to lead-based paint found in older inner-city homes along with increased exposure from automobile emissions. Applying these facts to this study, some correlations can be made. The three children from rural Bucks County all had negligible lead levels. In contrast, all the children who lived in urban Philadelphia County had lead levels of at least 0.24 μmol/L (5 μg/dL) or greater. Of the children listed in Table 1, 75% lived in Montgomery County, a suburban county but one with significant industry and highways.

As mentioned previously, lead is known to interfere with hemesynthesis.[5] This inhibition usually occurs at levels of 0.7 μmol/L (15 μg/dL) or greater, but can be variable in its effect on the hematocrit.[3] The children in this study, including the child with a lead level of 0.82 μmol/L (17 μg/dL), were all found to have hematocrit levels within the range of normal for age.

The adverse neurodevelopmental and cognitive effects of lead exposure are well documented. The major studies, however, have followed the children exposed for periods of 3 to 14 years.[12,13] None of the children in Table 1 were noted to be neurodevelopmentally delayed based on the routine medical examination. More formal testing was not performed. Nevertheless, close follow-up, including repeat lead levels and careful periodic developmental assessment, should be done on these children.

Seven of the eight children in Table 1 were insured primarily through Medicaid. Assuming they are from households with low incomes, their having elevated blood lead levels is again in keeping with the findings of the NHANES II study.[9] Conversely, a potential bias exists here because the majority of the children were of lower socioeconomic status. This aspect of the study does not allow the findings to be generalized to a private practice population.

Although some authorities believe treatment of children with lead concentrations of 0.7 μmol/L (15 μg/dL) may be warranted,[14] treatment of children with levels below 1.2 μmol/L (25 μg/dL) is not currently recommended.[10] None of the children in this study had levels elevated to this degree. Thus, no efforts at treatment or provocative testing for lead mobilization were made.

Two of the eight children (Table 1) lived in what is

considered "older housing," which may have been a factor in their lead exposure. Environmental investigation of the home and parental workplace is warranted for the child who is found to have an elevated blood lead level. Subsequent interventions, particularly in the examples of de-leading gasoline and paint, have shown to be effective in reducing childhood lead levels.[10,15] At lower levels of lead exposure, however, finding a definite environmental source becomes more difficult. The limits of this study did not allow for a more intensive evaluation of the home environment beyond determining the age of the patient's housing.

Conclusions

From the collective data, the studied patient population is definitely at risk for lead exposure and possible toxicity. Approximately 60% of all children screened had some degree of detectable lead in their blood. More important, 20% had levels at or above the threshold for neurobehavioral lead toxicity.[7,8] Based on this information, it appears that routine lead screening in the majority of patients should be performed, particularly in children who are black, live in Philadelphia and Montgomery counties, and are from lower socioeconomic backgrounds. Primary care physicians practicing in nonurban settings may want to address their pediatric population more closely and consider implementing a lead-screening program for these children. Moreover, continued removal of lead from housing, water, air, and other sources should be a goal of governmental agencies.

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Suggested Readings

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