

Lead Poisoning: From Screening to Primary Prevention

Committee on Environmental Health

Knowledge of the extent and seriousness of childhood lead poisoning has vastly expanded since the last statement regarding lead poisoning by the American Academy of Pediatrics in 1987.¹ Blood lead levels once thought to be safe have been shown to be associated with IQ deficits, behavior disorders, slowed growth, and impaired hearing.² In fact, lead poisoning is, according to the Department of Health and Human Services, "the most important environmental health problem for young children."³ The rapid development of the scientific database requires recognition by physicians of the significance of effects at lower levels and a change in clinical practice.

During the last 30 years the Centers for Disease Control and Prevention (CDC) has revised downward the definition of the blood level at which lead poisoning occurs from 60 µg/dL whole blood in the early 1960s, to 30 µg/dL in 1975, and 25 µg/dL in 1985. The 1991 CDC statement "Preventing Lead Poisoning in Young Children" recommended lowering the community intervention level to 10 µg/dL and setting several action levels (Table 1).² In 1987 the American Academy of Pediatrics stated that lead levels greater than 25 µg/dL were unacceptable for children.¹ The Academy now recognizes that impairment of cognitive function begins to occur at levels greater than 10 µg/dL, even though clinical symptoms are not seen. In the late 1970s, the average blood lead level for US children was 16 µg/dL.⁴ The mean blood lead level for US children has declined since 1976 due to the phaseout of lead in gasoline⁵ and the reduction of lead in food, and it is now between 4 and 6 µg/dL.⁶ However, severe lead poisoning still occurs, and there are still many children at high risk of exposure.

Childhood lead poisoning is preventable. In January 1991, the US Public Health Service issued a strategic plan to eradicate childhood lead poisoning which included a cost-benefit analysis.⁷ This represents a major change from primarily finding and treating exposed children toward an emphasis on preventing lead exposure. Identification and treatment of the child poisoned with lead continues to be essential, but of greater importance is *identification of the source and prevention of subsequent exposures* for that child and other children in the future.

Until children are in lead-free environments, blood lead screening is essential to prevent serious disease

and disability in the population. Pediatricians will continue to play a key role in the identification, treatment, and eradication of lead poisoning.

BACKGROUND

Lead poisoning has been recognized since antiquity. In the second century BC, Dioscorides, a Greek physician, said that "lead makes the mind give way."⁸ Childhood lead poisoning from lead-based paint was first described in Brisbane, Australia, in 1897.⁹ The cause of this endemic illness was identified as painted porch railings,¹⁰ and in 1920 the city of Brisbane passed the first act to prevent poisoning from lead-based paint. In the United States, plumbism from lead-based paint was described in the first decade of the 20th century.¹¹ It was believed initially that if a child recovered from the acute illness, there were no sequelae. Byers and Lord¹² refuted this in 1943 in their report of 20 children who had recovered from acute lead intoxication; 19 had obvious behavior disorders or mental retardation. During the early 1970s several studies were conducted to pursue this question further; some showed lead-related cognitive deficits but these studies were controversial.¹³ Better designed and more sophisticated studies have been carried out since that time, and there is a general consensus of opinion about the relationship between lead and cognitive function.¹³

EPIDEMIOLOGY

Lead poisoning is not a disease of poor or minority children alone. In 1984, the last year for which national estimates are available, 17% of American children had blood lead levels greater than 15 µg/dL.¹⁴ There were 12 million children who lived in lead-painted homes, and 6 million children living in homes built before 1940 when paint with the highest concentrations of lead was used. For white children, 7% in higher socioeconomic status areas and 25% in poorer communities had blood lead levels greater than 15 µg/dL. For black children in poor communities, this prevalence was 55%. Studies are underway to obtain national estimates of lead levels in children. Although children at highest risk certainly deserve the most attention, exposure throughout all strata of society presents a problem for all pediatric practices. Certainly any child living in a house containing lead-based paint may be at risk. Such housing and other sources of lead are found throughout the United States and not, as commonly believed, just in cities in the Northeast. Age of housing, not geographic location, is the best predictor for presence of lead-based paint.¹⁵

The recommendations in this statement do not indicate an exclusive course of treatment or procedure to be followed. Variations, taking into account individual circumstances, may be appropriate.

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TABLE 1. The Centers for Disease Control and Prevention (CDC) Recommends Follow-up Using the Following Schedule*

	Class and Blood Lead Level ($\mu\text{g}/\text{dL}$)					
	I <10	IA 10-14	II 15-19	III 20-44†	IV 45-69††	V 70††
Risk: low						
Age 6-36 mo	If initial screen at 12 mo, then retest at 24 mo, if possible	Retest every 3-4 mo until 2 consecutive tests <10 or 3 are <15; then retest in 1 y. History, education, and test for iron deficiency§	Refer for case management	Refer for medical evaluation and follow-up, identify and eliminate environmental lead sources	Refer for urgent medical and environmental follow-up (within 48 h)	Admit for immediate chelation therapy
Age 37-72 mo	None	None	Same as above	Same as above	Same as above	Same as above
Risk: high						
Age 6-36 mo	Retest every 6 mo until 2 consecutive tests <10 or 3 are <15; then screen annually if possible	Same as for low-risk group 6-36 mo	Same as above	Same as above	Same as above	Same as above
Age 37-72 mo	Screen annually if possible	Screen annually if possible	Same as above	Same as above	Same as above	Same as above

* Adapted from CDC.²

† Based on confirmed venous blood lead level.

‡ Children in this range with symptoms of blood lead poisoning should be considered medical emergencies and admitted immediately for chelation therapy.

§ The CDC recommends these interventions begin at 15 $\mu\text{g}/\text{dL}$ for lead poisoning prevention programs.

TOXICITY

Lead is absorbed by ingestion or inhalation. The relationship between exposure and blood lead levels is a dynamic process in which blood lead represents a product of recent exposures, excretion, and equilibration with other tissues. Children deficient in iron, protein, calcium, and/or zinc absorb lead more readily.¹⁶ Most retained lead is stored in the bones.

At high blood levels (more than 70 $\mu\text{g}/\text{dL}$), lead may cause encephalopathy and death. Survivors of encephalopathy may have lifelong severe disabilities, such as seizures and mental retardation.¹⁷ Lead toxicity affects almost every organ system, most importantly, the central and peripheral nervous systems, kidneys, and blood.¹⁸ Levels at which the toxic effects of lead are reported to occur are shown in Figure. Lead interferes with enzymes that catalyze the formation of heme. It also inhibits both prenatal and postnatal growth.¹⁹⁻²³ Lead impairs hearing acuity.^{24,25} Lead is a carcinogen in laboratory animals, and there is some evidence for carcinogenicity in workers exposed to lead but not in children.²⁶

Although the impairment of cognition in young children at levels above 10 $\mu\text{g}/\text{dL}$ has been reported, no threshold has been identified. At lower blood lead values, the impact on an individual child may be undetectable. In contrast, there may be a significant impact on a population of children with such blood levels. A number of studies have found an association between lead levels and intellectual functioning of children.^{13,27-43} In one study this resulted in an increase in the number of children with severe defi-

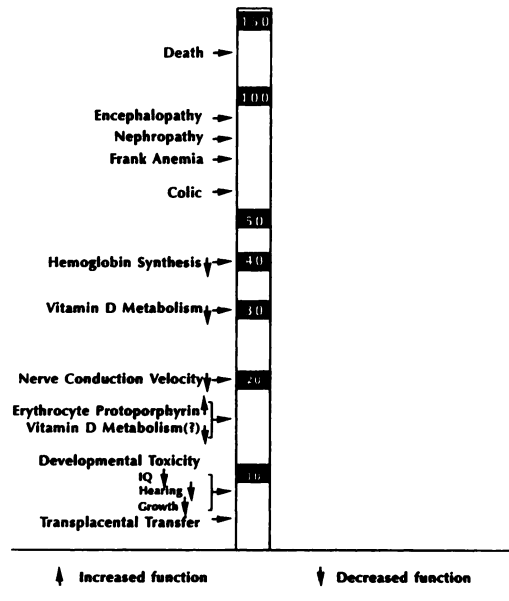


Figure. Lowest observed effect levels of inorganic lead in children. The levels in this diagram do not necessarily indicate the lowest levels at which lead exerts an effect. These are the levels at which studies have adequately demonstrated an effect. Source: Agency for Toxic Substances and Disease Registry, 1990.

cits (IQs less than 80) from 4% to 16%. In this sample 5% of children were expected to have IQs more than 125; of the children with high levels of lead, none had an IQ more than 125.^{27,28} This body of literature has been examined by meta-analysis. The relationship

between lead levels and IQ deficits was found to be remarkably consistent.²⁹⁻³¹ A number of studies have found that for every 10 µg/dL increase in blood lead levels, there was a lowering of mean IQ in children by four to seven points.^{32,36,39,43-45}

Effects of lead on cognition are found after adjustment for factors such as parental intelligence, socioeconomic status, education, and iron deficiency.^{13,46,47} Although many of the earlier studies of lead exposure at lower dose did not deal adequately with such factors, studies published since 1979 have taken these into account. Some demonstrated the effect of lead nonetheless; this was true in the study by Hansen et al conducted with a group of Danish children among whom there was little variability in factors such as ethnicity, culture, and medical care.^{37-39,41}

The effects of lead exposure in infants from birth onward have also been studied. Bellinger and colleagues followed a cohort of children born at Boston Hospital for Women. Most of these subjects came from middle-class white families. Covariate-adjusted Bayley developmental quotient scores to age 3 were significantly lower in children having cord blood lead levels greater than 10 µg/dL (mean, 14 µg/dL).⁴⁶ The blood lead level at age 2 years exerted an effect when the children reached the age of 57 months. An increase in the 24-month lead level of 10 µg/dL in the 0 to 25 µg/dL range was associated with a 5.8-point decrease in the Weschler Intelligence Scale For Children-Revised. At 57 months, the effect of prenatal exposure on development was no longer apparent.⁴⁷ Several studies in other populations have confirmed the association between prenatal lead levels and scores on the Bayley infant development scale.⁴⁸⁻⁵⁰ Two other studies confirmed the possible importance of the 24-month lead level on cognition in school age children.^{51,52} Exposures during the first two years of life incur a risk of sustained developmental delays and impaired cognition.

Recent evidence suggests that the effects of early lead exposure can persist.⁵³ Subjects in a group were classified by dentine lead levels in the first and second grade and followed up into adulthood. This study showed that those with high tooth lead levels as children were seven times more likely not to graduate from high school and six times more likely to have reading scores at least two grades below expected, after adjustment for a number of factors including socioeconomic status and parental IQ.⁵³ The children also had higher absenteeism in the final year of school, lower class rank, poorer vocabulary and grammatical reasoning scores, longer reaction times, and poorer hand-eye coordination.

SOURCES

Lead paint is the major source of lead poisoning for children. As lead paint deteriorates or is removed, house dust and soil become contaminated, and lead enters the body through normal hand-to-mouth activity.⁵⁴ Children may also ingest paint chips. Before 1955 much white house paint was 50% lead and 50% linseed oil. In 1955, manufacturers adopted a voluntary house paint lead-content standard of 1%, but

house paint with higher levels of lead continued to be manufactured.⁵⁵ The amount of lead allowable in paint was lowered by law in two steps, to 1% in 1971 and then to 0.06% in 1977. Occasionally lead paint manufactured for nonresidential purposes continues to be used to paint houses. It is estimated that 5 million tons of lead have been applied to houses in the United States. Of the homes built before 1960, 70% are estimated to have lead paint. Most dangerous are the 3.8 million homes with decaying or deteriorating lead paint in which 2 million children younger than 6 years of age live.¹⁸

Uncontaminated soil contains lead concentrations less than 50 ppm, but soil lead levels in many urban areas exceed 200 ppm. Areas near lead mines, lead-using industries, and smelters may have high levels of contamination in soil (up to 60 000 ppm).⁵⁶ In the United States, the use of leaded gasoline has released an estimated 30 million tons of lead into the air. Lead in house dust is an important source of exposure and comes from paint, soil, and other sources.

Acidic water of low mineral content can leach large amounts of lead from lead pipes or solder.¹⁴ This is particularly apt to occur when water has been standing in pipes for extended periods, and hot water may be of particular concern. An estimated 16% of household water supplies have lead concentrations greater than the proposed standard of 20 µg/dL.⁵⁷ Lead solder and fittings can also be found in older drinking water coolers and coffee urns. Brass fixtures may also be contaminated with lead.¹⁸ Lead-contaminated water has been linked to lead poisoning in children given reconstituted infant formula.⁵⁸

Lead may also contaminate food. Soil lead is taken up by root vegetables and atmospheric lead may fall onto leafy vegetables.¹⁸ Lead may also be added to food during processing. Cans with soldered seams can add lead to foods. In the United States, soldered cans have largely been replaced by seamless aluminum containers but some foreign and large commercial-sized cans still have lead-soldered seams.¹⁸ Other modes of food contamination include some ceramic tableware (especially imported), certain "natural" calcium supplements, and bright red and yellow paints on bread bags.¹⁸

Other lead sources are ethnic folk remedies (azarcon and greta used by Hispanics and pay-loo-ah used by Southeast Asians),¹⁸ eye cosmetics (kohl used by Moslems and surma by Hindus), hobbies (eg, stained glass, artist paints, shooting ranges), and accidental ingestion of small lead objects (eg, fishing weights, curtain weights). Parents who are employed in a lead-using industry may bring lead dust home on clothing or expose children by allowing them to visit work sites.¹⁸ Identification of the source of lead requires a careful history and usually investigation of the household.

PREVENTING CHILDHOOD LEAD POISONING

There are two major ways to prevent lead poisoning in children: removal of environmental lead and lead screening.

Remove Lead From Children's Environment

Removal of environmental lead is the most effective preventive measure. For past contamination problems, source reduction involves removal of lead or modification of the environment to prevent children's contact with lead. Screening of children for lead poisoning is useful in identifying areas in most need of environmental cleanup and in preventing other cases of lead exposure.

Screen Children for Lead

Lead poisoning can be prevented with routine screening followed by appropriate educational and case management programs. In 1988, the Agency for Toxic Substances and Disease Registry estimated that 250 000 children had blood lead levels of 25 µg/dL or more; of those, only 12 000 were identified by lead screening programs.¹⁸ Past screening efforts have been inadequate. For example, in California, from 1987 through 1990, less than 100 children with blood lead ≥ 25 were identified annually. In late 1991 lead screening of low-income children was implemented; and in the first 8 months of 1992, 707 cases were identified.⁵⁹ Even though average levels have declined nationwide, lead exposures of a magnitude to cause blood lead levels of concern are common, even among children of middle-income families, far more common than rates of phenylketonuria, hypothyroidism, or galactosemia.

Most lead poisoning is clinically inapparent. Even a carefully taken history can miss many of the commonly encountered sources of lead. A clinical history cannot achieve the sensitivity of a blood lead test. In a recent survey in a suburban area generally believed to have no lead poisoning problem, 20% of children were found to have blood lead levels of 10 µg/dL or above.⁶⁰

The toxicity of lead is a function of both dose and duration of exposure. It is the role of the pediatrician to give realistic reassurance that early detection and source control can minimize intellectual and behavioral consequences for the individual child. Parents can also become alarmed if pediatricians dismiss their concerns. For the emotional well-being of the family appropriate measures to identify and reduce exposure should be instituted promptly.

Since the 1970s the blood erythrocyte protoporphyrin (EP) has been used for screening. Due to low sensitivity at lower blood lead concentrations, the CDC in 1991 recommended a change from EP screening to a venous blood lead sample.^{2,61} A finger-stick sample can be tested and is preferable from the standpoint of ease and practicality. However, a finger-stick sample is contaminated easily by environmental lead, thereby increasing the false-positive rate. Hence, a finger-stick value exceeding 15 µg/dL should be confirmed with a venous blood lead sample. Reliability of the finger-stick sample depends on the blood collection technique, and problems should be obvious from the false positive rate. Where venous blood lead samples are readily available, they can be used for initial screening. The laboratory test for determining lead levels requires a high degree of

proficiency, best acquired by participation in proficiency testing programs, such as that run by the CDC.

Urgency and extent of follow-up depend on the risk classification and confirmed venous blood lead level. The first step is to perform a confirmatory venous blood lead level. This should be done immediately if the screening result is more than 70 µg/dL; within 48 hours, if between 45 and 69 µg/dL; within 1 week, if 20 to 44 µg/dL; and within 1 month, if 15 to 19 µg/dL (Table 2). Individual follow-up of children begins with lead levels at 15 µg/dL and medical evaluation at 20 µg/dL.

MANAGEMENT AND TREATMENT OF BLOOD LEAD LEVELS OF 10 MG/DL AND ABOVE

General Principles

Table 1 shows the treatment and follow-up as recommended by the CDC.² The most important aspect of treatment is removal of the source of exposure.

Patient Education

Education of parents about nutritional sources of calcium, iron, zinc, and ascorbate is important for all children, but especially for children with blood lead levels of 10 µg/dL and above. Parents' attention should be directed to the following steps to avoid lead exposure in their children: (1) removing lead-based paint, renovating, and remodeling in the home should be done by trained and experienced individuals with the family out of the home and proper cleanup before the family returns; (2) controlling dust and paint chip debris; (3) preventing the children from eating dirt or other foreign substances; (4) changing work clothes and cleaning up before going home from a lead-related job; (5) avoiding the use of lead around the home for hobbies and other purposes; (6) hand washing; and (7) using cold tap water for drinking and especially for mixing infant formula. Detailed information is available from local public health agencies and in the CDC Statement "Preventing Lead Poisoning in Children."²

Nutritional Treatment

Children deficient in iron, calcium, zinc, and ascorbate more readily absorb and/or retain lead from their diets.⁶² Dietary fat may promote lead absorption.⁶³ Treatment of iron deficiency is important for all children but especially for children with blood lead levels of 10 µg/dL and above.

TABLE 2. Suggested Timetable for Confirming Capillary Blood Lead Results With a Venous Blood Lead Measurement*

Blood Lead Level (µg/dL)	Time Within Which Blood Lead Level Should be Obtained
<10	Not applicable
10-14	Not applicable
15-19	Within 1 mo
20-44	Within 1 wk
45-69	Within 48 h
≥ 70	Immediately

* From the Centers for Disease Control and Prevention.²

Environmental Intervention

For persistent blood lead levels of 15 µg/dL or above or a confirmed venous lead level of 20 µg/dL, referrals should be made so that the child's environment is investigated and cleaned up. The local public health agency should be contacted and care should be coordinated. Public health agencies should keep the physician informed of results of the investigation.

MEDICAL EVALUATION AND MANAGEMENT OF CONFIRMED BLOOD LEAD LEVELS 20 MG/DL AND ABOVE

History

Clinicians should inquire further about the nature of housing, condition of paint, pica behavior, use of folk remedies or imported ceramics, hobbies, and parental occupation.

Iron Status

Children with elevated blood lead levels should be evaluated for iron deficiency. Iron deficiency can occur in the absence of anemia. A serum ferritin level less than 12 µg/dL or an abnormally low ratio of serum iron to iron binding capacity is the most specific indicator of iron deficiency.⁶⁴

Other Diagnostic Procedures

Many traditional tests for lead poisoning are unnecessary. Abdominal radiographs are helpful only in cases of acute ingestion or unusual persistence of high blood lead values. Lead lines on bone radiographs and basophilic stippling of red blood cells may be associated with chronic high level exposure, but can be negative even in the presence of serious lead exposures. Because testing of hair and fingernails is subject to external environmental contamination, it is an uncertain estimate of body burden and is not recommended.

Follow-up Testing

Serial lead level measurements provide the best information about lead exposure. The CDC guidelines (Table 1) provide recommendations for periodicity of follow-up testing in children with elevated blood lead levels.

Testing Other Children

Children in the same household of a child with a blood lead level exceeding 20 µg/dL should also be tested, if exposure is believed to have occurred in the home. If other locations such as child-care centers, schools, playgrounds, or baby-sitters' homes are identified as being lead contaminated, children in those environments should be tested as well.

Chelation Therapy

Chelation is not recommended at less than 25 µg/dL. Blood lead reduction has been demonstrated in response to chelation, but there are no data on improvement or prevention of cognitive delay. It is not a substitute for removing a child from exposure. If the physician is not experienced in the treatment of lead poisoning, it is wise to seek consultation or re-

ferred when chelation therapy is being considered and in areas where there are dedicated "lead programs."

Four chelating agents are available: CaNaEDTA, British Anti-Lewisite (BAL), D-penicillamine, and succimer.² Chelation therapy speeds urinary excretion of lead.⁶⁵ Most of the attendant risks of chelation are associated with excretion of essential metals (particularly calcium, magnesium, and zinc) along with the lead.

D-Penicillamine has side effects similar to other penicillins. BAL and succimer are mercaptans; BAL and CaNaEDTA are nephrotoxic.

The current practice has been to chelate children with a blood lead level of 45 µg/dL or above and to monitor and consider chelation for blood lead levels of 25 µg/dL and above.⁶⁵ At blood lead values less than 70 µg/dL, when chelation is warranted, three drugs, CaNaEDTA, D-penicillamine, and succimer, are used for children. D-Penicillamine is not labeled for use in lead poisoning, and the label for succimer specifies use for levels greater than 45 µg/dL.² For blood lead levels of 70 µg/dL and above, treatment with two drugs, CaNaEDTA and BAL in combination, is generally recommended.⁶⁵ When the child has signs of encephalopathy, treatment should be provided in an intensive care setting. Chelating agents can greatly enhance the absorption of lead from the gastrointestinal tract.⁶¹ Drug therapy may be dangerous unless the child is removed from lead exposure.² In the initial stages of treatment, this may require hospitalization when measures to discontinue lead exposure and to assure full compliance and follow-up have not been implemented. For this reason, the oral drugs succimer and D-penicillamine are given most appropriately in inpatient settings, until efforts have been made to reduce the child's exposure to lead.

CONCLUSIONS

Childhood lead exposure continues to be a public health problem. Lead exposure is not a disease of the poor or minorities only or a result of poor child-rearing practices. The "lead problem" has been reduced but has not been solved. Despite past legislation to remove lead from gasoline and paint, lead exposure persists. The problem is not well enough understood to permit easy solutions. A coordinated effort by public health, environmental protection, housing, and pediatric care providers will be expensive. Only with such an effort can this preventable disease be eradicated. Until lead poisoning has been eliminated, screening programs will be necessary. The following recommendations address the need for more acceptable screening methods and more aggressive follow-up and cleanup programs.

AAP RECOMMENDATIONS TO PEDIATRICIANS

1. *Anticipatory Guidance.* Pediatric providers need to provide anticipatory guidance and education to parents. This includes information on:
 - Promotion of safe environmental and occupational practices so that parents can prevent the exposure of their children to lead; this would

include contact via hobbies and contaminated work clothing.

- The risk of normal childhood hand-to-mouth activity and other likely sources of lead exposure (paint, house dust, soil, drinking water, etc).
 - Provision of instruction in general measures (hygiene, nutrition) for preventing exposure.
 - The high risk of children with developmental disabilities, who often engage in pica behavior and can least afford to lose whatever cognitive or behavioral strengths they have.
2. **Blood Lead Screening.** Pediatric care providers should increase their efforts to screen children for lead exposure. Blood lead screening should be a part of routine health supervision for children and can be addressed best by increasing children's access to health care. Because lead is ubiquitous in the US environment, this screening should occur at about 9 through 12 months of age and, if possible, again at about 24 months of age. The CDC has raised the possibility that there may be low-risk communities that don't require screening, but no explicit guidance has been developed for determining a community's risk. As more data are collected it may become evident that there are locales where selective screening of children is more appropriate than routine screening. Currently there is not adequate laboratory capacity nationwide to screen each child, but the requirement to phase-in screening should generate those resources.
 3. **Clinical Indications for Lead Testing.** A history of possible lead exposure should be assessed at health supervision visits between the ages of 6 months and 6 years using a number of specific questions (Table 3). The risk questions identify children at high risk who should be screened more frequently for blood lead levels (Table 1). Lead poisoning should be considered in the evaluation of the following disorders, either because the lead may cause these disorders or because the conditions may be associated with increased lead ingestion: developmental delay, learning disabilities, behavior disorder, autism, convulsions, iron deficiency anemia, intestinal parasitic infections,

speech and hearing deficits, encephalopathy, recurrent vomiting, and recurrent abdominal pain.

RECOMMENDATIONS TO GOVERNMENT

1. **Follow-up by Public Health and Housing Agencies.** Lead testing of children is futile in the absence of public health programs to ensure environmental investigation and follow-up for individual cases. The government should establish and fund such programs. Lead screening programs in high-risk areas should be integrated with other public health activities.
2. **Environmental Cleanup.** The Academy supports efforts of environmental and housing agencies to remove lead from housing and other areas where children may be exposed. Training and certification of inspectors and abatement workers and approved training programs are needed to avoid creating lead hazards. Some state health departments can provide lists of certified contractors and other experts. In addition, there is a need for the development of less expensive, safe technologies for abatement, in order to make primary prevention efforts more cost-effective.
3. **Reduction of New Sources of Lead into the Environment.** The Academy supports legislation seeking to reduce the entry of lead into the overall environment.
4. **Identification of Areas Where Lead Risks Exist.** More data about the rates of elevated levels of blood lead in specific communities are needed. The Academy encourages government to conduct focused surveys in small areas to determine where lead screening is and is not needed or where eradication efforts have been successful. A better understanding of the distribution of lead poisoning would allow more efficient screening efforts.
5. **Research to Develop a Better Lead Test.** There is a pressing need for a more efficient and less invasive test for lead levels or lead toxicity. The ideal measure could be used routinely on outpatients, be inexpensive, rapid, sensitive, resistant to contamination, and reliable.
6. **Lead Poisoning as a Reportable Disease to the Centers for Disease Control and Prevention.** The Academy supports making lead poisoning reportable by laboratories on a nationwide basis.
7. **Sponsorship of Clinical Studies.** The Academy recommends more research in the area of determining effectiveness of various strategies to prevent and treat lead poisoning, comparison of methods for abating lead in households, and controlled clinical trials of chelating agents with long-term follow-up.
8. **Periodic Update of the Screening Recommendations.** The Centers for Disease Control and Prevention needs to conduct studies of the efficacy of lead screening and monitor the scientific literature to assure that lead screening is carried out in the most public health-protective, least intrusive, and most cost-effective manner possible. In particular, the risk questions and frequency of follow-up recommendations need to be studied and evaluated. Recommendations about screening also need to be

TABLE 3. Assessing the Risk of High-Dose Exposure to Lead: Sample Questionnaire*

Does your child—

1. Live in or regularly visit a house with peeling or chipping paint built before 1960? This could include a day-care center, preschool, the home of a babysitter or a relative, etc.
2. Live in or regularly visit a house built before 1960 with recent, ongoing, or planned renovation or remodeling?
3. Have a brother or sister, housemate, or playmate being followed up or treated for lead poisoning (that is, blood level ≥ 15 mg/dL)?
4. Live with an adult whose job or hobby involves exposure to lead?
5. Live near an active lead smelter, battery recycling plant, or other industry likely to release lead?

* From the Centers for Disease Control and Prevention.²

reevaluated in the light of research published since the time of the 1991 guidelines.

9. *Adequate Funding of Screening Tests.* The US Department of Health and Human Services needs to reconsider its decision to continue using the outdated erythrocyte protoporphyrin test for Medicaid-funded children and instead pay for blood lead tests. The Early Periodic Screening, Diagnosis and Treatment (EPSDT) Program needs to cover the blood lead test as a separate billable item on a nationwide basis.

COMMITTEE ON ENVIRONMENTAL HEALTH, 1992 TO 1993

J. Routt Reigart, MD, Chairperson

Ruth A. Etzel, MD

Lynn R. Goldman, MD, MPH

Jim G. Hendrick, MD

Howard C. Mofenson, MD

Peter R. Simon, MD

LIAISON REPRESENTATIVES

Henry Falk, MD, Centers for Disease Control and Prevention

Robert W. Miller, MD, National Cancer Institute

Walter Rogan, MD, National Institute of Environmental Health Sciences

CONSULTANTS

Richard J. Jackson, MD

Herbert L. Needleman, MD

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