CDC

CENTERS FOR DISEASE CONTROL
AND PREVENTION

Preventing Lead Poisoning in Young Children

A STATEMENT BY THE CENTERS FOR DISEASE CONTROL — OCTOBER 1991

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES / Public Health Service

CONVERSION TO SYSTEME INTERNATIONAL (SI) UNITS

Blood Lead

1.0 μg/dL = 0.04826 μmol/L 1.0 μmol/L = 20.72 μg/dL

0 μg/dL	=	0. μmol/L
5 μg/dL	=	0.241 μmol/L
10 μg/dL	=	0.483 μmol/L
15 μg/dL	=	0.724 μmol/L
20 μg/dL	=	0.965 μmol/L
25 μg/dL	=	1.206 μmol/L
30 μg/dL	=	1.448 μmol/L
35 μg/dL	=	1.689 μmol/L
40 μg/dL	=	1.930 μmol/L
45 μg/dL	=	2.172 μmol/L
50 μg/dL	=	2.413 μmol/L
55 μg/dL	=	2.654 μmol/L
60 μg/dL	=	2.896 μmol/L
65 μg/dL	=	3.137 μmol/L
70 μg/dL	=	3.378 μmol/L

Erythrocyte Protoporphyrin

1.0 μg/dL = 0.01778 μmol/L 1.0 μmol/L = 56.25 μg/dL

28 μg/dL	=	0.498 μmol/L
35 μg/dL	=	0.622 μmol/L
70 μg/dL	=	1.245 μmol/L

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Preface

This is the fourth revision of the statement on *Preventing Lead Poisoning in Young Children* by the Centers for Disease Control (CDC). The recommendations continued herein are based mainly on the scientific data showing adverse effects of lead in young children at increasingly lower blood lead levels. They are tempered, however, by practical considerations, for example, of the numbers of children who would require followup and the resources required to prevent this disease. It is possible that further scientific data and development of infrastructure and technology will result in a lowering of the blood lead level at which interventions are recommended at a future time.

This statement is a departure from previous ones in several ways. Perhaps most important is the emphasis on primary prevention and the need for coordination between pediatric health-care providers and public agencies. This statement reflects the vision expressed in the Department of Health and Human Services' *Strategic Plan for the Elimination of Childhood Lead Poisoning*, which calls for a concerted, coordinated societywide effort to eliminate this disease.

In writing this statement, we identified several areas where better data are needed in order to provide scientifically sound guidance. These range from evaluating the efficacy of chelation therapy at lower blood lead levels in terms of preventing the adverse effects of lead to developing science-based criteria for determining when an abated unit is cleaned up enough for rehabitation. We hope that the appropriate research to answer such questions will be conducted in a timely manner, and we will continue to update the statement to reflect current understanding.

We are aware of concerns about the impact the changes in the statement will have on childhood lead poisoning prevention programs, laboratories, and pediatric health-care providers. In this new statement, we recognize the need for a transition period until we are able to implement fully the new recommendations; it will take time and a concerted effort to implement this new guidance.

CDC is conducting several activities which bear directly on the implementation of the statement. First, as noted above, the *Strategic Plan for the Elimination of Childhood Lead Poisoning* was released by Dr. Louis W. Sullivan, Secretary of the Department of Health and Human Services, on February 21, 1991. In addition to laying out the actions needed to eliminate childhood lead poisoning, this plan describes the need for infrastructure and technology development, including for the evaluation of blood and environmental lead levels. Second, CDC is aggressively pursuing research and development efforts in collaboration with several instrument manufacturers to develop a field-rugged, relatively inexpensive, and simple-to-operate blood lead instrument, which would markedly enhance blood lead screening efforts. Initial results are encouraging, but the effort is still in the developmental stage. If all goes well, new instrumentation could be ready in 2 to 3 years. Third, we are continuing our efforts to help laboratories improve the quality of their blood lead measurements through our proficiency testing program and through our Blood Lead Laboratory Reference System. Finally, CDC also has a grant program in childhood lead poisoning prevention, through which state and local health agencies receive Federal money to screen children for lead poisoning, ensure environmental and medical followup for poisoned children, and provide education about lead poisoning. By the end of FY 1991, we will be funding 13 state and 2 city childhood lead poisoning prevention programs, and the President's budget for 1992 includes almost a doubling of the FY 1991 budget. We continue to encourage CDC-funded programs to address infrastructure issues.

Other Federal agencies, like the Environmental Protection Agency and the Department of Housing and Urban Development, have also released plans that deal with aspects of the childhood lead poisoning problem. These agencies are also working to build the needed infrastructure for and expand the scientific knowledge on reducing exposure to lead in the environment.

I wish to thank the members of the Committee and consultants, as well as the numerous other people who assisted in the development and review of this document. I believe this document will be a major landmark in the effort to eliminate childhood lead poisoning from the United States.

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Chapter 1. Introduction

Summary

New data indicate significant adverse effects of lead exposure in children at blood lead levels previously believed to be safe. Some adverse health effects have been documented at blood lead levels at least as low as 10 micrograms per deciliter of whole blood (μg/dL).

The 1985 intervention level of 25 μg/dL is, therefore, being revised downwards to 10 μg/dL.

A multitier approach to followup has been adopted.

Primary prevention efforts (that is, elimination of lead hazards before children are poisoned) must receive more emphasis as the blood lead levels of concern are lowered.

The goal of all lead poisoning prevention activities should be to reduce children's blood lead levels below 10 μg/dL. If many children in the community have blood lead levels ≥10 ug/dL, communitywide interventions (primary prevention activities) should be considered by appropriate agencies. Interventions for individual children should begin at blood lead levels of 15 μg/dL.

Childhood lead poisoning is one of the most common pediatric health problems in the United States today, and it is entirely preventable. Enough is now known about the sources and pathways of lead exposure and about ways of preventing this exposure to begin the efforts to eradicate permanently this disease. The persistence of lead poisoning in the United States, in light of all that is known, presents a singular and direct challenge to public health authorities, clinicians, regulatory agencies, and society.

> Lead poisoning is one of the most common and preventable pediatric health problems today.

Lead is ubiquitous in the human environment as a result of industrialization. It has no known physiologic value. Children are particularly susceptible to lead's toxic effects. Lead poisoning, for the most part, is silent: most poisoned children have no symptoms. The vast majority of cases, therefore, go undiagnosed and untreated. Lead poisoning is widespread. It is not solely a problem of inner city or minority children. No socioeconomic group, geographic area, or racial or ethnic population is spared.

Previous lead statements issued by the Centers for Disease Control (CDC) have acknowledged the adverse effects of lead at lower and lower levels. In the most recent previous CDC lead statement, published in 1985, the threshold for action was set at a blood lead level of 25 μg/dL, although it was acknowledged that adverse effects occur below that level. In the past several years, however, the scientific evidence showing that some adverse effects occur at blood lead levels at least as low as 10 μg/dL in children has become so overwhelming and compelling that it must be a major force in determining how we approach childhood lead exposure.

This document provides guidelines on childhood lead poisoning prevention for diverse groups. Public health programs that screen children for lead poisoning look to this document for guidance on screening regimens and public health actions. Pediatricians and other health-care practitioners look to this document for information on screening and guidance on the medical treatment of poisoned children. Government agencies, elected officials, and private citizens seek guidance about what constitutes a harmful level of lead in blood—what the current definition of lead poisoning is and what blood lead levels should trigger environmental and other interventions.

It is not possible to select a single number to define lead poisoning for the various purposes of all of these groups. Epidemiologic studies have identified harmful effects of lead in children at blood lead levels at least as low as 10 μg/dL. Some studies have suggested harmful effects at even lower levels, but the body of information accumulated so far is not adequate for effects below about 10 μg/dL to be evaluated definitively. As yet, no threshold has been identified for the harmful effects of lead.

Because 10 μg/dL is the lower level of the range at which effects are now identified, primary prevention activities—communitywide environmental interventions and nutritional and educational campaigns—should be directed at reducing children's blood lead levels at least to below 10 μg/dL. Blood lead levels between 10 and 14 μg/dL are in a border zone. While the overall goal is to reduce children's blood lead levels below 10 μg/dL, there are several reasons for not attempting to do interventions directed at individual children to lower blood lead levels of 10-14 μg/dL. First, particularly at low blood lead levels, laboratory measurements may have some inaccuracy and imprecision, so a blood lead level in this range may, in fact, be below 10 μg/dL. Secondly, effective environmental and medical interventions for children with blood lead levels in this range have not yet been identified and evaluated. Finally, the sheer numbers of children in this range would preclude effective case management and would detract from the individualized followup required by children who have higher blood lead levels.

The single, all-purpose definition of childhood lead poisoning has been replaced with a multitier approach, described in Table 1-1. Community prevention activities should be triggered by blood lead levels ≥10 μg/dL. Medical evaluation and environmental investigation and remediation should be done for all children with blood lead levels ≥20 μg/dL. All children with blood lead levels ≥15 μg/dL should receive individual case management, including nutritional and educational interventions and more frequent screening. Furthermore, depending on the availability of resources, environmental investigation (including a home inspection) and remediation should be done for children with blood lead levels of 15-19 μg/dL, if such levels persist. The highest priority should continue to be the children with the highest blood lead levels.

Other differences between the 1985 and 1991 statements are as follows:

Screening test of choice. Because the erythrocyte protoporphyrin level is not sensitive enough to identify children with elevated blood lead levels below about 25 μg/dL, the screening test of choice is now blood lead measurement.

Universal screening. Since virtually all children are at risk for lead poisoning, a phase in of universal screening is recommended, except in communities where large numbers or percentages of children have been screened and found not to have lead poisoning. The full implementation of this will require the ability to measure blood lead levels on capillary samples and the availability of cheaper and easier-to-use methods of blood lead measurement.

Table 1-1. Interpretation of blood lead test results and follow-up activities: class of child based on blood lead concentration

Class	Blood lead concentration (μg/dL)	Comment
I	≤9	A child in Class I is not considered to be lead-poisoned.
IIA	10-14	Many children (or a large proportion of children) with blood lead levels in this range should trigger communitywide childhood lead poisoning prevention activities (Chapter 9). Children in this range may need to be rescreened more frequently.
IIB	15-19	A child in Class IIB should receive nutritional and educational interventions and more frequent screening. If the blood lead level persists in this range, environmental investigation and intervention should be done (Chapter 8).
III	20-44	A child in Class III should receive environmental evaluation and remediation (Chapter 8) and a medical evaluation (Chapter 7). Such a child may need pharmacologic treatment of lead poisoning (Chapter 7).
IV	45-69	A child in Class IV will need both medical and environmental interventions, including chelation therapy (Chapters 7 and 8).
V	≥70	A child with Class V lead poisoning is a medical emergency. Medical and environmental management must begin immediately (Chapters 7 and 8).

Primary prevention. Efforts need to be increasingly focused on preventing lead poisoning before it occurs. This will require communitywide environmental interventions, as well as educational and nutritional campaigns.

Succimer. In January, 1991, the U.S. Food and Drug Administration approved succimer, an oral chelating agent, for chelation of children with blood lead levels over 45 μg/dL.

Childhood lead poisoning prevention programs have had a tremendous impact on reducing the occurrence of lead poisoning in the United States. Because of these programs, deaths from lead poisoning and lead encephalopathy are now rare. These programs have targeted high-risk children for periodic screening; provided education to caretakers about the causes, effects, symptoms, and treatments for lead poisoning; and ensured medical treatment and environmental remediation for poisoned children. Screening and medical treatment of poisoned children will remain critically important until the environmental sources most likely to poison children are eliminated.

Federal regulatory and other actions have resulted in substantial progress in reducing blood lead levels in the entire U.S. population. In the last two decades, the virtual elimination of lead from gasoline has been reflected in reductions in blood lead levels in children and adults. Lead levels in food have also decreased since most manufacturers stopped using leaded solder in cans and since atmospheric deposition of lead on food crops declined as a result of reductions of lead in gasoline. In 1978, the Consumer Product Safety Commission banned the addition of lead to new residential paint.

Nevertheless, important environmental sources and pathways of lead remain. Lead-based paint and lead-contaminated dusts and soils remain the primary sources and pathways of lead exposure for children. In addition, children continue to be exposed to lead through air, water, and food, as well as occupations and hobbies of parents and caretakers. The focus of prevention efforts, therefore, must expand from merely identifying and treating individual children to include primary prevention—preventing exposure to lead before children become poisoned. This will require a shared responsibility among many public and private agencies. Public agencies will have to work with pediatric health-care providers to identify communities with childhood lead-poisoning prevention problems and unusual sources of lead and to ensure environmental followup of poisoned children. Public housing and economic development agencies will have to integrate lead paint abatement into housing rehabilitation policies and programs. Health-care providers will need to phase in virtually universal screening of children. Public and private organizations must continue to develop economical and widely-available blood lead tests to make such screening possible. Public and private housing owners must bear a portion of the financial burden for abatement.

The changes in this statement are not meant to create an enormous burden on primary pediatric health-care providers. These changes will only be useful if public health and other agencies effectively complement health-care providers' activities. Several efforts have begun to increase federal support of childhood lead poisoning prevention programs and of followup activities. Ongoing efforts to develop infrastructure and technology by the public and private sectors include 1) the development of inexpensive, easy-to-use portable methods for measuring blood lead levels; 2) the development of training and certification programs for lead paint inspectors and abatement contractors; and 3) the development and testing of new abatement methods, including encapsulants. The changes in this statement are also not meant to increase the emphasis on screening of children; the long-term goal of this statement is **prevention**. Until primary prevention of childhood lead poisoning can be achieved, however, increased screening and followup of poisoned children is essential.

Elimination of Childhood Lead Poisoning

Will require efforts from both the private and public sectors.

Will require a shift in emphasis to primary prevention.

Will take time and resources.

Should proceed in a rational manner, with the highest risk children being made the highest priority.

Can be achieved.

In February 1991, the U.S. Department of Health and Human Services released a *Strategic Plan for the Elimination of Childhood Lead Poisoning* (HHS, 1991). This plan describes the first 5 years of a 20-year societywide effort to eliminate this disease. It places highest priority on first addressing the children at greatest risk for lead poisoning. The U.S. Department of Housing and Urban Development (HUD, 1990) and the Environmental Protection Agency (EPA, 1991) have both released plans dealing with the elimination of lead hazards. To eliminate this disease will require a tremendous effort from all levels of government as well as the private sector, but we believe that the benefits to society will be well worth it. We look forward to the day when childhood lead poisoning is no longer a public health problem.

References

CDC (Centers for Disease Control). Preventing lead poisoning in young children: a statement by the Centers for Disease Control. Atlanta: CDC, 1985; CDC report no. 99-2230.

EPA (Environmental Protection Agency). Strategy for reducing lead exposures: report to Congress. Washington (DC): EPA, 1991.

HHS (U.S. Department of Health and Human Services). Strategic plan for the elimination of childhood lead poisoning. Atlanta: CDC, 1991.

HUD (U.S. Department of Housing and Urban Development). Comprehensive and workable plan for the abatement of lead-based paint in privately owned housing: report to Congress. Washington (DC): HUD, 1990.

Chapter 2. Background

Summary

The blood lead level considered to indicate lead toxicity has progressively shifted downwards.

In general, children are more at risk for lead exposure than adults.

Large numbers of children in the United States continue to have blood lead levels in the toxic range.

This chapter describes the health effects of lead on children and fetuses, the metabolism of lead, and the demographics of lead exposure in the United States. It explains why the definition of childhood lead poisoning is being revised.

EFFECTS OF LEAD ON CHILDREN AND FETUSES

Lead affects virtually every system in the body.
The blood lead level considered to indicate lead poisoning has fallen steadily since the 1970s.
Blood lead levels at least as low as 10 μg/dL are associated with adverse effects.
Although the effects of low-level lead exposure may not seem severe in the individual child, on a population basis they are extremely important.

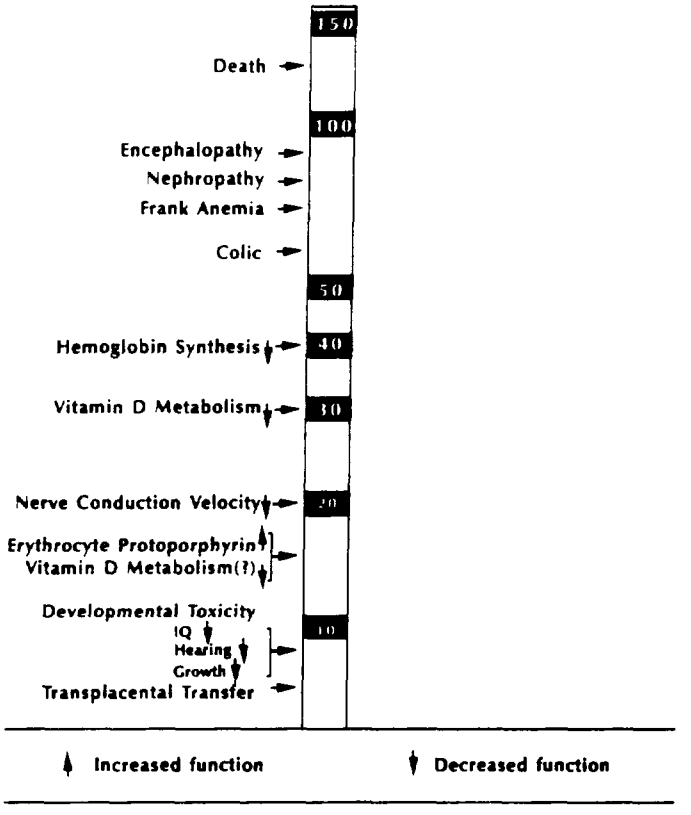
Lead is a poison that affects virtually every system in the body. It is particularly harmful to the developing brain and nervous system of fetuses and young children. The adverse effects of lead on children and adults are summarized in Figure 2-1.

The risks of lead exposure are not based on theoretical calculations. They are well known from studies of children themselves and are not extrapolated from data on laboratory animals or high-dose occupational exposures.

Levels of Concern

Since 1970, our understanding of childhood lead poisoning has changed substantially. As investigators have used more sensitive measures and better study designs, the generally recognized level for lead toxicity has progressively shifted downward. Before the mid-1960s, a level above 60 μg/dL was considered toxic (Chisolm and Harrison, 1956). By 1978, the defined level of toxicity had declined 50% to 30 μg/dL. Figure 2-2 shows how the federal definition of an elevated blood lead level has changed over the years.

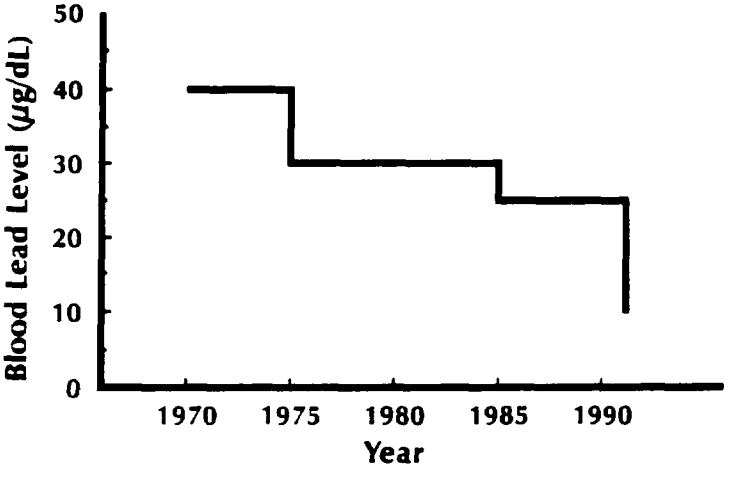
Figure 2-1. Lowest observed effect levels of inorganic lead in children*



*Note: 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: ATSDR, 1990.

Figure 2-2. Blood lead levels considered elevated by the Centers for Disease Control and the Public Health Service



Range of Effects of Lead

Very severe lead exposure in children (blood lead levels ≥80 μg/dL) can cause coma, convulsions, and even death. Lower levels cause adverse effects on the central nervous system, kidney, and hematopoietic system. Blood lead levels as low as 10 μg/dL, which do not cause distinctive symptoms, are associated with decreased intelligence and impaired neurobehavioral development (Davis and Svendsgaard, 1987; Mushak et al., 1989). Many other effects begin at these low blood lead levels, including decreased stature or growth (Schwartz et al., 1986; Bornschein et al., 1986; Shulka et al., 1989), decreased hearing acuity (Schwartz and Otto, 1987), and decreased ability to maintain a steady posture (Bhattacharya et al., 1988). Lead's impairment of the synthesis of the active metabolite 1,25-$(OH)_2$ vitamin D is detectable at blood lead levels of 10-15 μg/dL. Maternal and cord blood lead levels of 10-15 μg/dL appear to be associated with reduced gestational age and reduced weight at birth (ATSDR, 1988). Although researchers have not yet completely defined the impact of blood lead levels <10 μg/dL on central nervous system function, it may be that even these levels are associated with adverse effects that will be clearer with more refined research.

Studies of Low-Level Lead Effects on the Central Nervous System

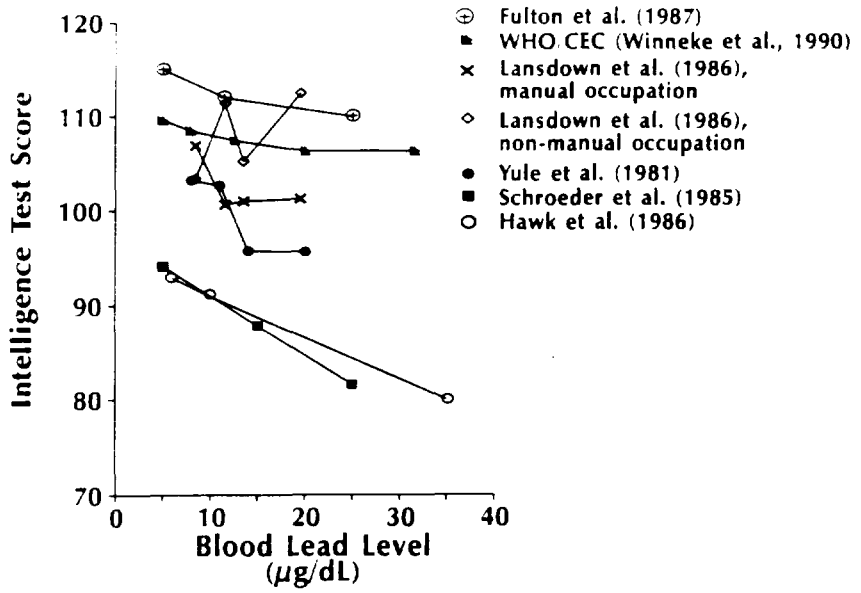
The concern about adverse effects on central nervous system functioning at blood lead levels as low as 10 μg/dL is based on a large number of rigorous epidemiologic and experimental studies. In particular, recent cross-sectional and prospective studies have provided new evidence about the association between low-level lead exposure and child development.

Several well-designed and carefully conducted cross-sectional and retrospective cohort studies in many different countries have been conducted (Lansdown et al., 1986; Fulton et al., 1987; Fergusson et al., 1988; Silva et al., 1988; Bergomi et al., 1989; Hansen et al., 1989; Hatzakis et al., 1989; Winneke et al., 1990; Lyngbye et al., 1990; Needleman et al., 1990; Yule et al., 1981; Lansdown et al., 1986; Hawk et al., 1986; Schroeder et al., 1985). Figure 2-3 shows the mean intelligence quotient (IQ) scores (in most cases adjusted for potential confounding factors) achieved by children with different blood lead levels from several of these studies. Some inconsistencies can be found in the results of these studies, but the weight of the evidence clearly supports the hypothesis that decrements in children's cognition are evident at blood lead levels well below 25 μg/dL. No threshold for the lead-IQ relationship is discernable from these data.

Most investigators report lower IQ scores among the more highly exposed children but these differences have not uniformly reached statistical significance (that is, $p<.05$). One way to synthesize the data from different studies is meta-analysis. Recent evaluation of 24 major cross-sectional studies provides strong support for the hypothesis that children's IQ scores are inversely related to lead burden (Needleman and Gatsonis, 1990).

Although available evidence is not sufficient to conclude that lead-associated deficits are irreversible, a recent follow-up study reported that the educational success of a cohort of young adults was significantly inversely associated with the amount of lead in teeth they shed as first and second graders (Needleman et al., 1990). In this study, dentine lead levels above 20 ppm were associated with a seven-fold risk of not graduating from high school, a six-fold risk of having a reading disability, deficits in vocabulary, problems with attention and fine motor coordination, greater absenteeism, and lower class ranking. Although dentine lead levels did not correspond in any simple way to blood lead levels, the available preschool blood lead levels

Figure 2-3. Blood lead levels and IQ scores of children, from cross-sectional and retrospective cohort studies*



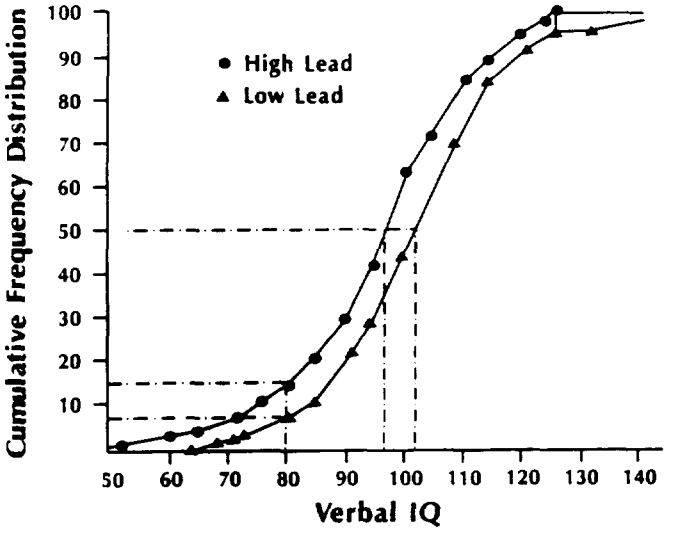
*Data from prospective studies are not included.

of the more highly exposed children averaged 35 μg/dL (Needleman et al., 1979). Increased circumpulpal dentine lead levels (>16 ppm) have been linked to higher rates of learning disabilities in a recent Danish study as well (Lyngbye et al., 1990).

To address methodological limitations of cross-sectional studies of lead and child development, a number of prospective studies were begun during the 1980s. Blood lead measurements were begun during the prenatal period and continued for several years, along with assessment of development. In several but not all cohorts, prenatal exposures have been associated with slower sensory-motor and delayed early cognitive development (Bellinger et al., 1987; Bellinger et al., 1991; Dietrich et al., 1987; Ernhart et al., 1986; Dietrich et al., 1991). With low postnatal exposures and favorable socioeconomic conditions, some of these early associations may attenuate as children grow older (Bellinger et al., 1991). In addition, several studies have noted that children's cognitive performance in the preschool period may be associated with early postnatal lead exposures (McMichael et al., 1988; Bellinger et al., 1991). It will be necessary for these prospective studies to follow their respective cohorts into the school-age years in order for the full implications of these early patterns to become clear.

Questions are frequently raised about the practical significance of the difference frequently observed between the IQ scores of more exposed and less exposed children. For the previously described population of children studied by Needleman et al. (Needleman et al., 1979), a shift in mean IQ score of 4-6 points as a result of lead exposure was associated with a substantial increase in the prevalence of children with severe deficits (that is, IQ scores less than 80) (Figure 2-4). Similarly, in this population the shift was associated with an absence of children who achieved superior function (that is, IQ scores greater than 125).

Figure 2-4. Cummulative frequency distribution of verbal IQ scores in children with high and low tooth lead levels



Source: Needleman et al., 1979.

ABSORPTION OF LEAD

Children are at higher risk for lead exposure because

They have more hand-to-mouth activity than adults.

They absorb more lead than adults.

Many factors can affect the absorption, distribution, and toxicity of lead. Children are more exposed to lead than older groups because their normal hand-to-mouth activities may introduce many nonfood items into their gastrointestinal tract (Lin-Fu, 1973). The efficiency of gastrointestinal absorption of lead in food and beverages in children has been estimated to be around 40% (Ziegler et al., 1978). From experimental studies, gastrointestinal absorption of lead from nonfood sources is decreased in the presence of food (Rabinowitz, 1980). Efficiency of absorption is probably also affected by the particle size and form of lead (Barltrop and Meek, 1979). Deficiencies in iron, calcium, protein, and zinc are related to increased blood lead levels and perhaps increased vulnerability to the adverse effects of lead (Mahaffey, 1981; Mahaffey and Michaelson, 1980).

BLOOD LEAD LEVELS IN THE UNITED STATES

Large numbers of children continue to have blood lead levels high enough to cause adverse effects.

Substantial progress has been made, however, in reducing blood lead levels in the United States.

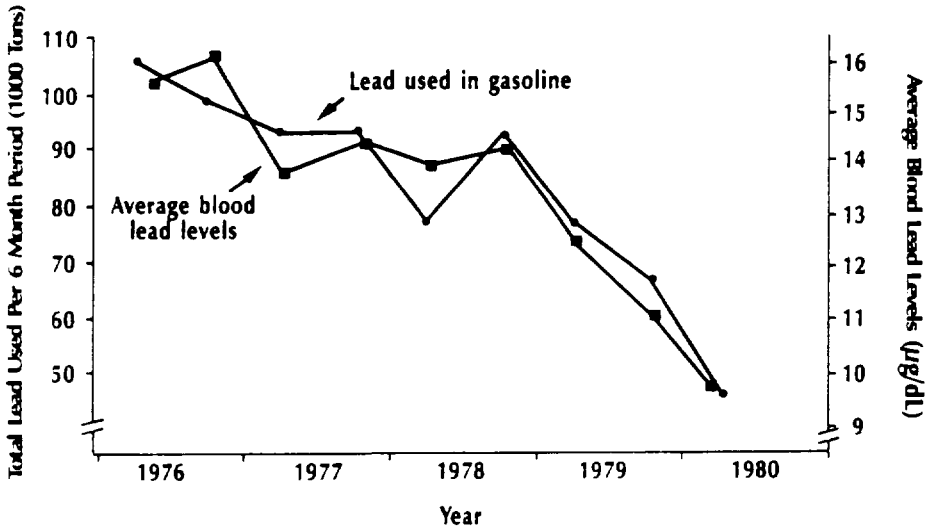
Lead-based paint remains the major source of high-dose lead poisoning in the United States.

The Agency for Toxic Substances and Disease Registry estimated that in 1984, 17% of all American preschool children had blood lead levels that exceed 15 μg/dL (ATSDR, 1988). Although all children are at risk for lead toxicity, poor and minority children are disproportionately affected. Lead exposure is at once a by-product of poverty and a contributor to the cycle that perpetuates and deepens the state of being poor.

Substantial progress has been made in reducing blood lead levels in U.S. children. Perhaps the most important advance has been the virtual elimination of lead from gasoline. Close correlations have been demonstrated between the decline in the use of leaded gasoline and declines in the blood lead levels of children and adults between 1976 and 1980 (Annest, 1983) (Figure 2-5). Levels of lead in food have also declined significantly, as a result both of the decreased use of lead solder in cans and the decreasing air lead levels.

Lead-based paint remains the major source of high-dose lead poisoning in the United States. Although the Consumer Products Safety Commission (CPSC) limited the lead content of new residential paint starting in 1978, millions of houses still contain old leaded paint. The Department of Housing and Urban Development estimates that about 3.8 million homes with young children living in them have either nonintact lead-based paint or high levels of lead in dust (HUD, 1990).

Figure 2-5. Change in blood lead levels in relation to a decline in use of leaded gasoline, 1976–1980



Source: Annest JL, 1983.

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Chapter 3. Sources and Pathways of Lead Exposure

Sources and Pathways of Lead Exposure in Children Include:

Lead-based paint.

Soil and dust.

Drinking water.

Parental occupations and hobbies.

Air.

Food.

For some children, other sources and pathways, such as "traditional" medicines, may be critical.

INTRODUCTION

A child's environment is full of lead. Children are exposed to lead from different sources (such as paint, gasoline, and solder) and through different pathways (such as air, food, water, dust, and soil). Although all U.S. children are exposed to some lead from food, air, dust, and soil, some children are exposed to high dose sources of lead. Lead-based paint is the most widespread and dangerous high-dose source of lead exposure for preschool children.

Lead entering the body from different sources and through different pathways presents a combined toxicological threat (ATSDR, 1988). Multiple, low-level inputs of lead can result in significant aggregate exposure. Indeed, for children with lower (but still elevated) blood lead levels (for example, in the range of 10-20 μg/dL) identifying a single, predominant environmental source or pathway is not always possible.

This chapter describes the most important sources and pathways for childhood lead exposure. Information about the levels or concentrations of concern in different pathways is based on information assembled by regulatory agencies and other published data. Nothing in this chapter should be interpreted as suggesting standards for acceptable or unacceptable levels or concentrations of lead in different environmental media.

LEAD-BASED PAINT

Lead-based paint is the most common high-dose source of lead exposure for children.

About 74% of privately owned, occupied housing units in the United States built before 1980 contain lead-based paint.

Children are exposed to lead when they ingest chips of lead-based paint or ingest paint-contaminated dust and soil.

Many cases of lead poisoning result when homes containing lead-based paint are remodelled or renovated without precautions being taken.

Removing lead from housing is important both for the treatment of poisoned children and for the primary prevention of childhood lead poisoning.

Lead-based paint remains the most common high-dose source of lead exposure for preschool children. Lead-based paint (containing up to 50% lead) was in widespread use through the 1940s. Although the use and manufacture of interior lead-based paint declined during the 1950s and thereafter, exterior lead-based paint and lesser amounts of interior lead-based paint continued to be available until the mid-1970s (CEH/CAPP, 1987). (Lead-based paint produced after the 1940s tended to have much lower lead concentrations than lead-based paint produced earlier.) In 1978, the Consumer Product Safety Commission banned the manufacture of paint containing more than 0.06% lead by weight on interior and exterior residential surfaces, toys, and furniture. Unfortunately, lead-based paint that is still available for industrial, military, and marine usage occasionally ends up being used in homes.

Nationwide, about 3 million tons of lead remain in an estimated 57 million occupied private housing units built before 1980 (representing 74% of all such housing). Of particular concern are the 14 million housing units believed to contain lead paint in unsound condition and the 3.8 million deteriorated units occupied by young children (HUD, 1990).

Pica, the repeated ingestion of nonfood substances, has been implicated in cases of lead poisoning; however, a child does not have to eat paint chips to become poisoned. More commonly, children ingest dust and soil contaminated with lead from paint which flaked or chalked as it aged or which has been disturbed during home maintenance or renovation. This lead-contaminated house dust, ingested via normal repetitive hand-to-mouth activity, is now recognized as a major contributor to the total body burden of lead in children (Bornschein et al., 1986). Because of the critical role of dust as an exposure pathway, children living in sub-standard housing and in homes undergoing renovation are at particular risk for lead poisoning.

Numerous studies have established that the risk of lead poisoning is related to the presence of lead-based paint and to the condition of such paint (ATSDR, 1988; EPA, 1986). Children who live in rehabilitated lead-free housing or who return to lead-reduced housing after undergoing medical treatment have significantly lower blood levels than children living in similar, nonrehabilitated housing (Bornschein et al., 1986; Chisolm et al., 1985). Data from several urban lead poisoning prevention programs indicate that deleading the home of a poisoned child can reduce blood lead levels substantially (Rosen et al., in press; Amitai et al., in press; G. Copley, unpublished data). Deleading or lead paint abatement can be an effective method of

reducing children's exposure to dangerous levels of lead in paint and house dust if properly done (Farfel and Chisolm, in press), but may actually increase dust lead levels if not done properly (Farfel and Chisolm, 1990).

Lead paint is typically found on kitchen and bathroom walls and throughout pre-1950 homes on doors, windows, and wooden trim. The risks of lead poisoning are greater when lead paint or the underlying surface are in deteriorated condition and when lead paint (even intact paint) is located on surfaces accessible to children (EPA, 1986). Lead paint on interior and exterior window components is particularly of concern because it is abraded into dust by the repeated opening and closing of these windows (Farfel and Chisolm, 1990).

Many cases of childhood lead poisoning that result from renovation or remodelling of homes have been reported (Marino, 1990). Before older homes undergo any renovation that may generate dust, they should be tested for the presence of lead-based paint. If such paint is found, contractors experienced in working with lead-based paint should do the renovations.

There is no uniform standard for safe or allowable amounts of lead in existing painted surfaces. States and the federal government use values ranging from 0.7-1.2 mg/cm^2 of wall when lead is measured using a portable x-ray fluorescence analyzer (XRF) or a standard of 0.5% lead by weight when tests are performed using laboratory analysis. These regulatory limits are based mostly on practical, not health, considerations.

Lead paint also continues to be used on the exterior of painted steel structures, such as bridges and expressways. In addition to the obvious risk to workers, increased lead absorption has been reported in children exposed to chips or dust during the deleading or maintenance of such structures (Landrigan et al., 1982).

Deleading, even when performed in the homes of children who have already been poisoned, is an important method of primary lead poisoning prevention because it reduces or removes the lead hazard from that housing unit for all future occupants. Methods for the safe abatement of residential lead paint are detailed in Chapter 8. The Department of Housing and Urban Development has primary responsibility for issues related to lead-based paint in housing.

SOIL AND DUST

Soil and dust act as pathways to children for lead deposited from paint, gasoline, and industrial sources.

The long-term efficacy and cost-effectiveness of different measures to reduce lead levels in soil need to be evaluated.

Reduction of dust lead is important both as part of deleading and as a means of interim risk reduction.

Soil and dust act as pathways to children for lead deposited by primary lead sources such as lead paint, leaded gasoline, and industrial or occupational sources of lead. Since lead does not dissipate, biodegrade, or decay, the lead deposited into dust and soil becomes a long-term source of lead exposure for children. For example, although lead emissions from gasoline have largely been eliminated, an estimated 4-5 million metric tons of lead used in gasoline remain in dust and soil, and children continue to be exposed to it (ATSDR, 1988).

Because lead is immobilized by the organic component of soil, lead deposited from the air is generally retained in the upper 2-5 centimeters of undisturbed soil (EPA, 1986). Urban soils and other soils that are disturbed or turned under may be contaminated down to far greater depths. Soil lead levels within 25 meters of roadways are typically 30-2,000 parts per million (ppm) higher than natural levels, with some roadside soils having concentrations as high as 10,000 ppm. Soils adjacent to houses painted with exterior lead paints may also have lead levels above 10,000 ppm. Measured lead levels in soil adjacent to smelters range as high as 60,000 ppm (EPA, 1986).

As part of normal play and hand-to-mouth exploratory activities, young children may inhale or ingest lead from soil or dust. Ingestion of dust and soil during meals and playtime activity appears to be a more significant pathway than inhalation for young children (EPA, 1986).

Different investigators have found widely varying relationships between levels of lead in soil and dust and children's blood lead levels. Blood lead levels generally rise 3-7 μg/dL for every 1,000-ppm increase in soil or dust lead concentrations (EPA, 1986; Bornschein et al., 1986; ATSDR, 1988). Particle size and the chemical form of lead may affect the bioavailability of lead in soil and dust; access to soil, behavior patterns, presence of ground cover, and a variety of other factors also influence this relationship (Barltop and Meek, 1979).

Even if ongoing deposition of lead into soil and dust is eventually halted, measures will have to be taken to reduce exposures from lead-contaminated soils and dusts. Until data demonstrating the efficacy and cost-effectiveness of permanent soil and dust abatement measures are available, interim risk reduction steps will be needed in some places. Dust control via wet mopping and frequent hand washing has been shown to reduce the blood lead levels of children with high blood lead levels (Charney et al., 1983), but this is not a permanent solution so long as the source of the lead in the dust remains. For urban and smelter communities, where outdoor soil can be a major source of lead in house dust (Diemel et al., 1981; Yankel et al., 1977), indoor dust abatement may not be effective unless abatement of soil lead is also conducted. Soil abatement may consist of either establishing an effective barrier between children and the soil or the removal and replacement of at least the top few centimeters of soil. Grass cover, if properly maintained, may be an effective means of limiting exposure to dusts originating from lead-contaminated soil (Jenkins et al., 1988).

DRINKING WATER

Contamination of drinking water with lead usually occurs in the distribution system.
Several properties of water and its pattern of use affect how much lead contamination results from a particular water distribution system.
Some practical measures can lower the lead content of drinking water.

Lead levels are typically low in ground and surface water, but may increase once the water enters the water distribution system. Contamination of drinking water can occur at five points in or near the residential, school, public, or office plumbing, including 1) lead connectors (that is, goosenecks or pigtails), 2) lead service lines or pipes, 3) lead-soldered joints in copper

plumbing throughout the building, 4) lead-containing water fountains and coolers, and 5) lead-containing brass faucets and other fixtures. The 1986 Safe Drinking Water Act Amendments banned the use of lead in public drinking water distribution systems and limited the lead content of brass used for plumbing to 8%.

Several properties of water and its patterns of use affect the extent of lead contamination that results from a particular water delivery system. These factors include 1) the corrosiveness of water (that is, pH, alkalinity, and mineral content), 2) age of the lead-soldered joints and other lead components (the newer ones often pose a higher risk), 3) quantity and surface area of lead materials, and 4) standing time and temperature of water in contact with leaded surfaces.

Typically, lead pipes are found in residences built before the 1920s, with the oldest cities having the most frequent use of lead pipes. Pipes made of copper and soldered with lead came into general use in the 1950s. Overall, lead leaching from copper pipes with lead-soldered joints represents the major source of water contamination in homes and public facilities such as schools.

In some areas of the United States (for example, Pennsylvania), cisterns are used to store water, especially rain water that may be acidic. Cisterns also can be roof-collection systems, which are common in some island areas (for example, Hawaii, the Florida Keys). When lead solder is used either in the construction of these cisterns or to repair leaks, or the cistern has a lead liner, the potential for lead contamination of the water is substantial. If the water has a relatively low pH, has low concentrations of cations such as Ca^{++} or Mg^{++} (that is, "soft" water), or has an elevated organic content, the water is probably aggressive in dissolving lead from the cistern. Corrosion control may be effective in reducing water lead levels in the case of corrosive water.

Lead in drinking water is probably absorbed more completely than lead in food. Adults absorb 35%-50% of the lead they drink, and the absorption rate for children may be greater than 50% (ATSDR, 1988).

In general, lead in drinking water is not the predominant source for poisoned children. In some circumstances, however, lead exposures from water are unusually high. Some water cooler-fountains have been found to have lead-soldered or lead-lined tanks. Patterns of intermittent water use from these fountains results in the water standing in the tanks longer than in typical residential situations, which can increase the amount of lead that is leached from the tanks. Several babies have been poisoned when hot tap water, which was then boiled (resulting in concentrating the lead), was used to make baby formula (J. Graef, personal communication).

Practical measures to reduce exposure to lead in drinking water include using fully-flushed water for drinking and cooking and always drawing water for ingestion from the cold water tap. The effectiveness of many point-of-use devices (treatment devices that are installed at the tap) in reducing lead in water varies and may be affected by the location of the device in relation to the lead source and by compliance with manufacturer's use and maintenance instructions. Some, like reverse osmosis and distillation units, may be effective. Carbon, sand, and cartridge filters do not remove lead.

The Environmental Protection Agency regulates the permissible lead content of water.

OCCUPATIONS AND HOBBIES

Children may be exposed to high lead levels when workers take home lead on their clothing or when they bring scrap or waste material home from work.

Hobbyists may also inadvertently expose their families to lead.

The current Occupational Safety and Health Administration standards may not adequately protect the health of workers.

A variety of work and hobby environments expose people to lead and may result in lead exposures for their families. Occupations frequently reported to have resulted in adult lead poisoning are shown in Table 3-1. Many potential hazardous activities, like furniture refinishing and making stained glass, may be either hobbies or occupations. Other activities that may be associated with lead exposure include using indoor firing ranges, doing home repairs and remodeling, and making pottery. "Take-home" exposures may result when workers wear their work clothes home or launder them with the family laundry or when they bring scrap or waste material home from work (Grandjean and Bach, 1986).

Strict compliance by industrial operations with the Occupational Safety and Health Administration (OSHA) General Industry Lead Standard governing lead exposures (29 CFR 1910.1025) would greatly reduce both occupational lead exposure and the associated indirect exposures in the homes of these workers. Unfortunately, not all occupational settings are covered by this regulation. Workers in construction—including lead abatement workers–are excluded from coverage under the General Industry Lead Standard; they are covered under a much weaker construction standard. Numerous workers in these work environments have been

Table 3-1. Industries identified by surveillance for elevated blood lead levels, California and New York, 1991

Industry Description	Standard Industrial Classification Number
Secondary smelting and refining of nonferrous metals	3341
Storage batteries (lead batteries)	3691
Valve and pipe fittings (except plumber's brass goods)	3494
Plumbing fixture fittings and trim (brass goods)	3432
Brass/copper foundry	3362
Glass products, made of purchased glass	3231
Motor vehicle parts and accessories	3714
Firing range workers	7997,9221
Pottery, nec	3269
Chemical and chemical preparations	2899
Bridge, tunnel, and elevated highway construction	1622
Automotive repair shops	7539
Industrial machinery and equipment	5084
Inorganic pigments	2816
Primary batteries, dry and wet	3692

Sources: Baser and Marion, 1990; Maizlish et al., 1990.

excessively exposed to lead, with construction workers particularly having a high risk of very high blood lead levels (Maizlish et al., 1990). Compliance with the OSHA comprehensive lead standard is inadequate (Landrigan, 1990; Maizlish, et al., 1990) even by those industries required to be in compliance. Furthermore, the current OSHA standard may not adequately protect the health of workers (Landrigan, 1990). OSHA plans to revise its standard within the next several years.

AIRBORNE LEAD

> Although lead use in gasoline has been markedly reduced, previous use has resulted in widespread contamination of soil and dust.
>
> Except around point sources, airborne lead is only a minor exposure pathway.

Until recently, the combustion of leaded gasoline by motor vehicles was the predominant source of airborne lead in the United States. However, the Environmental Protection Agency (EPA) ordered the reduction of almost all lead in gasoline during the 1970s and 1980s, and 1990 amendments to the Clean Air Act will completely prohibit the use of lead as a gasoline additive beginning as early as January, 1992 and concluding no later than December 31, 1995. As discussed in the previous section, however, soil and dust contaminated by deposition of lead-containing particles can contain high concentrations of lead.

Except around point sources, like smelters and battery manufacturing plants, inhalation of airborne lead is now a minor exposure pathway for individual children. Other industrial activities may also result in localized exposures to lead, including burning solid waste in incinerators and sandblasting or demolishing bridges and other lead-painted metal structures. These localized activities, however, can be important sources of high-dose exposure.

FOOD

> The quantity of lead in the U.S. diet has decreased markedly in recent years.
>
> Improperly fired ceramicware, leaded crystal, and lead-soldered cans result in lead leaching into foods.
>
> Some food-handling practices can increase the lead content of foods.

During the 1980s, the quantity of lead in the U.S. diet decreased markedly. "Market basket" data from the U.S. Food and Drug Administration (FDA), used to estimate typical lead intake, show that the average dietary lead intake for a 2-year-old child was about 30 μg/day in 1982, about 13 μg/day by 1985, and about 5 μg/day in the period 1986-1988. This reduction was achieved through substantially restricted use of lead-soldered side-seam cans and the phasing out of lead as an additive in gasoline. In 1980, 47% of domestically produced food and soft drink cans were lead-soldered. By 1989 use of lead-soldered cans declined to 1.4% of domestically produced cans. Counter to this trend is the continued use of lead solder in cans of imported

foods, because cans manufactured outside the United States typically continue to contain lead solder.

Lead in foods comes from several sources in addition to lead solder: soil in which the plant is grown; air and rain; food processing (including lead leaching from some types of metal cans described above); contact with lead solder or ceramic vessels used to store the food; and contact with lead dusts in the home. If lead contamination is unusually severe, the quantity of lead in the diet will be much higher than the "Market Basket" estimates. Examples include imported food from countries that do not restrict the use of lead solder in cans; storage of foods packaged in lead-soldered cans for over a year or so, even if the can is unopened; storage of acidic foods in ceramic containers made with improperly applied leaded glazes; and food processed with lead-contaminated water.

Under some circumstances, food grown in "urban gardens" may have an elevated lead content if the garden soil is high in lead or if there are high lead concentrations in the air or water used for irrigation. Soil conditions (for example, pH, phosphorus content, buffering capacity, and the amount of organic matter) and the type of plant have a great effect on how much lead is transferred to the plant. The amount transferred is difficult to predict because many factors affect lead uptake. It is recommended that the crops grown on contaminated soil be tested to determine their lead uptake. Such tests may be arranged through the Agriculture Extension Service, state or federal departments of agriculture, or private laboratories.

Occasionally, food supplements can be seriously contaminated with lead. Examples have included various dietary supplements from "natural" sources, such as calcium supplements derived from animal bone sources.

In addition, some food-handling practices in the home can increase the lead content of foods and should be avoided. Foods should not be stored in unopened, lead-soldered cans for over a year or so. Foods should not be stored, even under refrigeration, in opened cans even if the can is subsequently covered. Food should be stored only in containers that do not release lead (for example, glass, stainless steel, or plastic containers). If ceramic food containers are ever used to store food, they should be made with lead-free glazes. Leaded crystal should not be used to store food for prolonged periods of time and should not be used to hold baby formula or juices.

Lead solders should never be used to repair food containers or to construct or repair cooking utensils. High lead levels may be present in hot water prepared in lead-soldered tea pots.

OTHER SOURCES

Other Sources and Pathways of Lead Exposure
"Traditional" medicines
Cosmetics
Casting ammunition, fishing weights, or toy soldiers
Making stained glass
Making pottery
Refinishing furniture
Burning lead-painted wood

Published data, as well as anecdotal evidence from clinicians and others who work with lead-poisoned children, have identified a variety of other sources of concern.

Many "non-Western" medicines (for example, greta and azarcon used to treat diarrhea or gastrointestinal upset) and cosmetics (for example, surma or kohl used around the eye for decorative or medicinal purposes) contain substantial quantities of lead and other metals. Rather than occurring as trace ingredients or trace contaminants, various lead compounds are used as major ingredients of traditional medicines in numerous parts of the world. "Traditional healers," using non-Western pharmacopeias, manufacture these products, which are often brought to recent immigrant groups by friends and relatives. Examples of such exposures have been reported from the Arab cultures, from the Indo-Pakistan subcontinent, from China, and from Latin America.

Folk remedies containing lead include:	
Alarcon	Ghasard
Alkohl	Greta
Azarcon	Liga
Bali Goli	Pay-loo-ah
Coral	Rueda

Many hobbies can result in substantial exposures to lead. For example, molten lead can be used in casting ammunition and making fishing weights or toy soldiers; leaded solder is used in making stained glass; leaded glazes and frits are used in making pottery; and artists' paints may contain lead. Furniture refinishing may also result in lead exposure.

In some areas, the burning of lead-painted wood in home stoves and fireplaces is a source of lead exposure. Lead fumes are generated, ashes contaminate the home, and ashes are often disposed of in the back yard, resulting in contamination of the environment.

SOURCES OF LEAD OUTSIDE THE UNITED STATES

Childhood lead poisoning is a problem worldwide.

Childhood lead poisoning is a problem worldwide. In other parts of the world, however, predominant sources of lead are very different than in the United States. For example, leaded gasoline is still widely used in many countries and contributes to elevated blood lead levels, especially in urban children. Poorly glazed pottery leading to high food lead levels can be the most prominent source of lead in some areas, for example, in parts of Latin America. Point industrial sources may dramatically increase air and soil lead levels in parts of the world where environmental controls have not been effectively implemented, for example, in Eastern Europe. Lead contamination from cottage industries that recycle lead, often in backyards, is a problem in Central America and elsewhere. For children moving to or from the United States, an assessment of potential lead hazards requires specific knowledge of the country involved.

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Chapter 4. The Role of The Pediatric Health-Care Provider

The Pediatric Health-Care Provider Should

Provide anticipatory guidance about childhood lead poisoning and its prevention.

Provide screening for lead poisoning following established screening schedules.

Conduct appropriate diagnostic blood lead testing in children with symptoms or signs consistent with lead poisoning or pica.

Interpret blood lead results.

Educate parents about reducing blood lead levels.

Coordinate with local public health officials.

Ensure that poisoned children receive appropriate medical, environmental, and social service followup.

Pediatric health-care providers, working as part of the public health team, must play a critical role in the prevention and management of childhood lead poisoning. Their roles include 1) educating parents about key causes of childhood lead poisoning; 2) screening children and interpreting blood lead test results; 3) working with appropriate groups in the public and private sectors to make sure that poisoned children receive appropriate medical, environmental, and social service followup; and 4) coordinating with public health officials and others involved in lead-poisoning prevention activities.

ANTICIPATORY GUIDANCE

Anticipatory guidance means

Teaching parents about major sources of lead and how to prevent poisoning.

Tailoring guidance to likely hazards in the community.

Pediatric health-care providers consider education to be an integral part of well-child care. Along with educating parents about nutrition and developmental stages, providers should discuss the potential hazards of lead. They should focus on the major preventable sources of high-dose lead poisoning—lead-based paint and take-home exposures from parents' occupations and hobbies. Parents should be told of the potential dangers of peeling lead-based paint, the potential hazards of renovating older homes, and the need for good work practices if their occupations or hobbies expose them to lead. (These sources and pathways of exposure are

discussed in Chapter 3.) Other education should be tailored to likely exposures in the community. For example, in some communities parents should be warned about the potential for lead exposure from improperly fired ceramicware and imported pottery. Where water lead levels are a concern, parents could be advised to use only fully-flushed water (that is, water that has not been standing in pipes for a prolonged time) from the cold-water tap for drinking, cooking, or preparing infant formula.

SCREENING FOR CHILDHOOD LEAD POISONING

Screening for lead poisoning requires

Determining the child's risk for high-dose lead exposure by asking a few questions.

Measuring blood lead levels in children who are at the greatest risk for high-dose lead poisoning when they are 6 months old.

Measuring blood lead levels in children who are at lower risk for high-dose lead exposure at 12-15 months of age.

Conducting necessary followup blood lead testing of children.

The recommended screening schedule is discussed in detail in Chapter 6. Since virtually all children are at risk for lead poisoning, universal screening is recommended, except in communities where large numbers or percentages of children have been screened and found not to have lead poisoning. (A more inexpensive and widely available blood lead test is under development.) Just as pediatric health-care providers ask screening questions about a child's development and eating habits, providers should also ask questions about a child's risk for high-dose lead exposure at every visit. (It is important to ask at every visit, since children's exposures may change over time.) On the basis of the parents' answers to these questions, the pediatric provider will be able to classify most children as being at either high or low risk for high-dose exposure to lead. The highest risk children should be screened starting when they are 6 months old, since that is when blood lead levels begin to rise. Lower risk children should be screened for the first time when they are 12-15 months old. Followup screening schedules should be based on the pediatric health-care provider's assessment of the child's risk for high-dose lead exposure and previous blood lead levels.

DOING APPROPRIATE DIAGNOSTIC BLOOD LEAD TESTING

Pediatric health-care providers should include lead poisoning in the differential diagnosis of a number of conditions. These include growth failure, developmental delays, hyperactivity, behavior disorders, hearing loss, and anemia. Children with parasites may be exhibiting pica, and the pediatric health-care provider should also consider measuring blood lead levels in such children.

INTERPRETATION OF BLOOD LEAD LEVELS

In interpreting blood lead levels, the provider should
Understand the scientific basis for concern.
Understand the degree of imprecision and inaccuracy in blood lead measurements.
Explain carefully why followup is or is not needed.

The studies which form the basis of our concern about childhood lead poisoning are described in Chapter 2. These studies suggest that adverse effects of lead occur at blood lead levels at least as low as 10 μg/dL. The following paragraphs provide guidance on what might be told to a parent, depending on the blood lead levels of the child.

Blood lead level <10 μg/dL. A blood lead level <10 μg/dL is not considered to be indicative of lead poisoning.

Blood lead level 10-14 μg/dL. Children with blood lead levels in this range are in a border zone. Since the laboratory tests for measuring blood lead levels are not as accurate and precise as we would like them to be at these levels, many of these children's blood lead levels may, in fact, be <10 μg/dL. Although a detailed environmental history should be taken since an obvious remediable source of lead may be found, it is unlikely that there is a single predominant source of lead exposure for most of these children. Thus, a full home inspection is not recommended. It is, however, prudent to try to decrease exposure to lead with some simple interventions (Page 30). (The required education can be done face-to-face or by distributing brochures or other written materials.) In addition, these children should receive followup blood lead testing in about 3 months. The adverse effects of blood lead levels of 10-14 μg/dL are subtle and are not likely to be recognizable or measurable in the individual child. It is important to make sure that these children's blood lead levels do not go up.

Example: Johnny was a 12 month old child without any risk factors for high-dose exposure. A capillary blood lead test was performed, and his blood lead level was 14 μg/dL. His pediatrician told his mother that Johnny's blood lead test was in a kind of "border" zone, but that it was high enough to require careful followup. The pediatrician explained that laboratory test results have some inaccuracy and imprecision, but, nevertheless, suggested some housekeeping and nutritional interventions to reduce Johnny's exposure. Johnny had a venous blood lead measurement three months later, which was 7 μg/dL. Three months after that, when Johnny was 18 months old, his blood lead level was 5 μg/dL. His blood lead level was measured one year later and was 5 μg/dL, and he received no further followup.

Blood lead level 15-19 μg/dL. Children with venous blood lead levels 15-19 μg/dL need more careful followup. The pediatric health-care provider should take a careful history, asking about sources of lead exposure (Chapter 3). Parents should receive guidance about interventions to reduce blood lead levels (Page 30). Children with blood lead levels in this range are at

risk for decreases in IQ of up to several IQ points and other subtle effects. The effects of lead at these levels are significant enough that the health-care provider should emphasize to parents the importance of followup screening to make sure the levels do not increase. The provider should also discuss interventions to reduce the blood lead levels. In addition, these children should receive followup testing (Chapter 6). If their blood lead levels persist at ≥15 μg/dL, environmental investigation and remediation should be completed, if resources permit. In some communities, childhood lead poisoning prevention programs may be able to manage the environmental investigation and remediation.

Blood lead level 20-69 μg/dL. Children with venous blood lead levels in this range should have a full medical evaluation (Chapter 7). This includes a detailed environmental and behavioral history (asking about reading or other learning disabilities, language development, pica, etc.), a physical examination, and tests for iron deficiency. Particularly for children needing urgent medical followup (that is, blood lead level ≥45 μg/dL), pediatric health-care providers with limited experience in treating lead poisoning should consider referring such children to a clinic with experience in managing childhood lead poisoning. These children should also have complete environmental investigations so that lead hazards can be reduced. The local public childhood lead poisoning prevention programs will often work as a team with the pediatric health-care provider and the child's family to ensure appropriate environmental followup.

Blood lead level ≥70 μg/dL. Children with blood lead levels this high constitute a medical emergency that preferably should be managed by someone with experience in treating children who are critically ill with lead poisoning. Medical and environmental management must begin immediately (Chapters 7 and 8).

EDUCATING PARENTS ABOUT REDUCING BLOOD LEAD LEVELS

What can parents do to reduce blood lead levels?

Housekeeping interventions to reduce exposure to dust.
Interventions to reduce exposure to other sources of lead.
Attention to nutrition.

There are many interventions parents can use to help reduce blood lead levels. These interventions are not a substitute for lead hazard abatement.

Housekeeping Interventions

Particularly in older homes, which may have been painted with lead-based paint, interventions to reduce exposure to dust may help reduce blood lead levels. These include:

- Make sure your child does not have access to peeling paint or chewable surfaces painted with lead-based paint. Pay special attention to windows and window sills and wells.

- If the house was built before about 1960 and has hard surface floors, wet mop them at least once a week with a high phosphate solution (for example, 5-8% phosphates). (The phosphate content of automatic dishwashing detergents and other cleaning substances is often listed on the label and may be high enough for this purpose. Otherwise, trisodium phosphate can be purchased in hardware stores.) Other hard surfaces (such as window sills and baseboards) should also be wiped with a similar solution. Do not vacuum hard surface floors or window sills or wells, since this will disperse dust. Vacuum cleaners with agitators remove dust from rugs more effectively than vacuum cleaners with suction only.
- Wash your child's hands and face before he/she eats.
- Wash toys and pacifiers frequently.

Other Interventions To Reduce Exposure To Lead

- If soil around the home is or is likely to be contaminated with lead (for example, if the home was built before 1960 or the house is near a major highway), plant grass or other ground cover. Since the highest concentrations of lead in a yard tend to be near surfaces that were once painted with lead paint, like exterior walls, if exterior lead paint was likely to be used, plant bushes around the outside of your house so your child cannot play there.
- In areas where the lead content of water exceeds the drinking water standard, use only fully-flushed water from the cold-water tap for drinking, cooking, and making formula. In communities where water conservation is a concern, use the first-flush water for other purposes.
- Do not store food in open cans, particularly if the cans are imported.
- Do not use pottery or ceramicware that was inadequately fired or is meant for decorative use for food storage or service.
- Make sure that take-home exposures are not occurring from parental occupations or hobbies (Chapter 3).

Nutrition

- Make sure your child eats regular meals, since more lead is absorbed on an empty stomach.
- Make sure your child's diet contains plenty of iron and calcium.

Examples of Sources of Iron and Calcium

Iron	Calcium
Liver	Milk
Fortified cereal	Yourt
Cooked legumes	Cheese
Spinach	Cooked greens

COORDINATING WITH PUBLIC SECTOR OFFICIALS

Public health officials should tell the pediatric health-care provider about

- The magnitude of the childhood lead poisoning problem in the provider's community.
- Unusual sources of lead exposure in the provider's community.
- Public sector services that can be used to ensure appropriate followup for poisoned children.
- Interventions being conducted through public sector actions for children with lead poisoning.

Pediatric health-care providers should notify public sector officials about

- Poisoned children they identify.
- Unusual sources or pathways of exposure they identify.

The responsibilities of public sector officials are described in Chapter 5. These officials are an important source of information for the pediatric health-care provider. They can alert the provider to the extent of the lead poisoning problem in the provider's catchment area. They can provide information about particular lead sources that may be of concern in a given neighborhood. Often these officials can assist in the management of the lead-poisoned child, doing followup screening, conducting environmental investigations, and ensuring lead hazards are abated. They should keep the provider informed of actions they take on the child's behalf. The pediatric health-care provider is responsible for informing public health officials about lead-poisoned children, reporting any unusual sources or pathways of exposure, and reporting elevated blood lead levels, if this is required.

APPROPRIATE FOLLOWUP

Appropriate followup includes

- Education.
- Followup blood lead testing.
- Medical evaluation, if appropriate.
- Pharmacologic treatment, if appropriate.
- Environmental investigation, if appropriate.
- Referral to infant stimulation or child development programs, if appropriate.
- Referral for social services.

Not all aspects of a poisoned child's followup will be managed by the pediatric health-care provider, although the provider is an important part of the team. Through his or her interactions with the child and family and the responsible public health agency, the provider

should make sure that any appropriate interventions are occurring. The provider should make sure that the family receives education about childhood lead poisoning and ways of preventing it, and he or she should make sure that the child receives the appropriate followup blood lead testing. If the child needs a medical evaluation (for a blood lead level ≥20 μg/dL) or pharmacologic treatment (Chapter 7), either the provider should do it or should refer the child to a place that treats large numbers of poisoned children. The provider should make sure that the child receives an appropriate environmental investigation and remediation with the help of the public health agencies. Particularly if the child is developmentally delayed, the provider should refer the child to an appropriate infant stimulation or child development program. In many cases, lead-poisoned children and their families will also benefit from social services followup.

Chapter 5. The Role of State and Local Public Agencies

A variety of local, state and federal agencies play a role in preventing childhood lead poisoning. Pediatric health-care providers and parents should know about what these agencies do so that they can use these resources effectively. In turn, these agencies must coordinate their activities to ensure that all aspects of childhood lead poisoning prevention—health, housing, and environment—are being addressed, and to provide the most comprehensive and cost-effective services to at-risk children, their parents, and their health-care providers.

Government operations vary widely from state to state. In some states, city or local government takes the leading role in providing public health services such as lead poisoning prevention. In other states, county or state agencies take the lead role. Similarly, housing and environmental agencies with responsibility for addressing lead hazards may exist at the local, county, or state level.

PUBLIC HEALTH AGENCIES

The public health agency should

- Ensure that necessary screening services are provided.
- Analyze surveillance and other data to identify exposure patterns and high-risk populations.
- Develop and implement a primary prevention plan that focuses on the highest risk sources and populations.
- Coordinate prevention activities with other pertinent health, housing, and environmental agencies.
- Ensure that medical and environmental followup services for poisoned children are provided.

In most cities and some towns, counties, and states, lead poisoning prevention programs are included within the public health department or agency. Traditionally, these programs have focused on screening for lead poisoning and ensuring medical or environmental followup for children identified as being poisoned. Many also undertake public education activities, but, historically, lack of resources has limited these agencies' ability to focus on primary prevention.

A comprehensive, multifaceted approach to preventing childhood lead poisoning would include screening and surveillance, risk identification, primary prevention activities, interagency coordination, and services for poisoned children.

Screening

As explained in Chapter 6, screening children for lead poisoning is important both to identify poisoned children and to provide data that can be used to target communitywide interventions.

Public health agencies should have the primary responsibility for ensuring that children receive necessary blood lead screening. While it is not realistic for public agencies to actually perform most of the required screening, these agencies must work with individual and institutional pediatric health care providers to ensure that the private sector provides as much screening as possible. Screening by public agencies should focus on children who would not otherwise receive it in the private sector.

Surveillance and Risk Assessment

Before a public health agency can design and implement a primary prevention plan, the agency must assess the sources of lead in the community, exposure patterns, and high-risk populations. The lead public health agency should generally take responsibility for the types of risk assessment activities described in Chapter 9, soliciting cooperation and assistance from housing and environmental agencies when appropriate. As explained in Chapter 9, blood lead screening provides data for assessing the extent and nature of a given community's lead problem. Public health agencies should also take the lead in conducting or coordinating the collection of the environmental, housing, and demographic data needed to undertake a community-based risk assessment.

Primary Prevention

One of the most important themes of this document is the need to identify and remove sources of exposure to lead before children are harmed, that is, the need for primary prevention. Public health agencies must take a leading role in designing and implementing primary prevention programs. One important activity for public health agencies is to use the data collected from screening and surveillance to develop a primary prevention plan designed to target resources to the most pervasive sources and the highest risk populations.

Interagency Coordination

Public health agencies cannot be expected to implement primary prevention activities by themselves. Many steps that must be taken lie within the expertise or jurisdiction of other government agencies, especially those dealing with housing or the environment. To prevent lead poisoning, all public agencies with a connection to this problem need to be enlisted in the effort. The activities of different types of agencies at different levels of government must be coordinated, preferably through formal arrangements under which the different agencies meet and consult. Public health agencies should take the lead in organizing interagency task forces or committees and in ensuring that all involved agencies communicate regularly.

Providing Services to Poisoned Children

Public health agencies have traditionally been responsible for ensuring that lead-poisoned children receive appropriate medical and environmental followup, often through a formal case-management system. Until lead poisoning has successfully been eradicated, public health agencies will have to devote much of their lead-poisoning prevention resources to case management services for poisoned children.

HOUSING AGENCIES

Housing agencies should

Work with public health agencies to provide needed housing and environmental services to poisoned children.

Enforce code requirements regarding lead hazards.

Assist public health agencies in educating property owners, tenants, real estate professionals, and building contractors about lead hazards in housing.

Use regulations and policies to increase the amount of safe and effective abatement performed.

Most states, and some cities and counties, have agencies with the responsibility for regulating housing quality or developing policies to ensure that people are provided with safe and affordable housing. As the focus shifts from severely poisoned children to lead hazards in children's environments, the role of housing agencies will expand. A comprehensive, multifaceted role for housing agencies would include providing services to poisoned children; enforcing housing codes; educating the public; and making regulations and policies to increase the number of safe and effective abatements.

ENVIRONMENTAL AGENCIES

Environmental agencies should

Participate in interagency efforts to prevent lead poisoning.

Adopt a multimedia approach to addressing environmental lead hazards.

Undertake monitoring, regulatory, licensing, and enforcement activities to reduce environmental exposure to lead.

Most states, and some cities and counties, have agencies responsible for regulating exposures to toxic substances in the environment. Traditionally, such agencies have dealt with exposure to lead in the air, in drinking water, and in hazardous and solid waste, but have had little or no role in addressing the hazards associated with lead in paint. Some environmental agencies have begun to address the problems of toxic substances in housing, such as asbestos and radon, and they may also be willing to join an interagency effort to reduce exposure to lead hazards in housing. A comprehensive, multifaceted role for environmental agencies would include participation in interagency efforts, multimedia lead hazard reduction programs, monitoring, and regulation.

Chapter 6. Screening

Summary

Screening is important both to ensure that poisoned children are identified and to generate data to target primary prevention activities.

Virtually all children should be screened for lead poisoning. Screening children with a high probability of exposure to high-dose sources is the highest priority.

Screening should be done using a blood lead test.

Children at greatest risk for high-dose lead exposure should be screened more frequently.

Analytical considerations affect interpretation of blood lead test results, particularly at low levels.

Traditionally, the main purpose of a childhood lead poisoning screening program has been to identify asymptomatic lead-poisoned children and to intervene as quickly as possible to reduce their blood lead levels. An additional benefit of screening programs is that abatement of lead sources for poisoned children results in prevention of lead poisoning for children who would have been exposed to those sources in the future. As the focus in lead poisoning prevention turns more to primary prevention, an additional benefit of screening is that data generated can be used in targeting interventions to places with children at high risk for lead poisoning.

SUGGESTED PRIORITIES FOR SCREENING

Virtually all U.S. children are at risk for lead poisoning.
Children at highest risk should be given the highest priority for screening.

In 1984, the last year for which estimates are available, it is believed that between 3 and 4 million children younger than age 6 years (17% of all U.S. children in this age group) had blood lead levels above 15 μg/dL (ATSDR, 1988). Furthermore, about 74% of occupied, privately owned housing built before 1980 contains lead-based paint (defined as ≥1 milligram per square centimeter (mg/cm^2)) (HUD, 1990). **Because almost all U.S. children are at risk for lead poisoning (although some children are at higher risk than others), our goal is that all children should be screened, unless it can be shown that the community in which these children live does not have a childhood lead poisoning problem.** (Deciding that no problem exists requires that a large number or percentage of children be tested.[1]) The full implementation of this will require the ability to measure blood lead levels on capillary samples and the availability of cheaper and easier-to-use methods of blood lead measurement. Children

[1]The health departments need to take lead role in assessing whether or not a community has a childhood lead poisoning problem.

at highest risk for lead poisoning are the highest priority for screening. Table 6-1 provides guidance on the groups for which repeated screening is most strongly indicated.

Children ages 6 to 72 months who live in or are frequent visitors to deteriorated old buildings, including day care centers, make up the highest priority group. Because the highest concentrations of lead in paint were used in the early 1900s, homes built before about 1960 are of greatest concern. Children whose homes are being renovated are also at extremely high risk. Since siblings, housemates, visitors, and playmates of children with confirmed lead poisoning may have similar exposures to lead, they also should be promptly screened. In communities with a high prevalence of lead poisoning, health departments should consider door-to-door screening, since many children with lead poisoning may be missed by fixed-site screening.

Children with parents whose work or hobbies involve lead may also risk lead exposure (Chapter 3). Also, children living near lead smelters or other industries where lead is processed may be at increased risk for lead poisoning.

In general, screening and assessment for lead poisoning should focus on children younger than 72 months of age, particularly on children younger than 36 months of age. Young children engage in the most hand-to-mouth activity (and therefore are at highest risk for lead exposure) and have the most rapidly developing nervous systems, making them more vulnerable to the effects of lead. Children with developmental delays, who may exhibit pica or have more extensive hand-to-mouth activity than other children, would be expected to be at increased risk for lead poisoning even if they are 72 months of age and older. These children may have to be screened more often during early infancy, and may require screening into their school years.

Children who have unexplained seizures, neurological symptoms, abdominal pain, or other symptoms that are consistent with lead poisoning should also have their blood lead levels measured. In addition, the possibility of lead poisoning should be considered in any child with growth failure, developmental delay, hyperactivity, behavior disorders, hearing loss, anemia, etc.

Table 6-1. Priority groups for screening

- Children, ages 6 to 72 months, who live in or are frequent visitors to deteriorated housing built before 1960.
- Children, ages 6 to 72 months, who live in housing built before 1960 with recent, ongoing, or planned renovation or remodelling.
- Children, ages 6 to 72 months, who are siblings, housemates, or playmates of children with known lead poisoning.
- Children, ages 6 to 72 months, whose parents or other household members participate in a lead-related occupation or hobby.
- Children, ages 6 to 72 months, who live near active lead smelters, battery recycling plants, or other industries likely to result in atmospheric lead release.

SCREENING METHOD

Screening should be done using a blood lead test.

Since erythrocyte protoporphyrin (EP) is not sensitive enough to identify more than a small percentage of children with blood lead levels between 10 and 25 μg/dL and misses many children with blood lead levels ≥25 μg/dL (McElvaine et al., 1991), measurement of blood lead levels should replace the EP test as the primary screening method. Unless contamination of capillary blood samples can be prevented, lead levels should be measured on venous samples. Obtaining capillary specimens is more feasible at many screening sites. Contamination of capillary specimens obtained by finger prick can be minimized if trained personnel follow proper technique (see Appendix I for a capillary sampling protocol). Elevated blood lead results obtained on capillary specimens should be considered presumptive and must be confirmed using venous blood. At the present time, not all laboratories will measure lead levels on capillary specimens.

Programs will need to increase their capacity to perform blood lead testing. During the transition to the use of the blood lead test as the primary screening method, some programs will temporarily continue to use EP as a screening test. In addition, some nutrition programs (for example, the Supplemental Food Program for Women, Infants, and Children (WIC)) use the EP test to identify children with iron deficiency.

For a discussion of the units used to report EP results (Page 48). All EP test results of ≥35 μg/dL if standardized using 241 L cm-1 mmol-1, ≥28 μg/dL if standardized using 297 L cm-1 mmol-1, or ≥70 μmol ZnPP/mol heme, if the hematofluorometer reports in these units, must be followed by a blood lead test (preferably venous) and an evaluation for iron deficiency (Page 53). Work on developing easy-to-use, cheap, portable instruments for blood lead testing is ongoing.

ANTICIPATORY GUIDANCE AND ASSESSING RISK

Anticipatory guidance helps prevent lead poisoning by educating parents on ways to reduce lead exposure.
Questions about housing and other factors are used to identify which children are at greatest risk for high-dose lead exposure.
Anticipatory guidance and assessment of risk should be tailored to important sources and pathways of lead exposure in the child's community.

Guidance on childhood lead poisoning prevention and assessment of the risk of lead poisoning should be part of routine pediatric care. Anticipatory guidance is discussed in more detail in Chapter 4. The guidance and risk assessment should emphasize the sources and exposures that are of greatest concern in the child's community (Chapter 3). Because lead-based paint has been used in housing throughout the United States, in most communities it will be necessary to focus on this source.

SCREENING SCHEDULE

The Screening Schedule

The screening schedule is based on the fact that children's blood lead levels increase most rapidly at 6-12 months and peak at 18-24 months.

Anticipatory guidance on preventing lead poisoning and assessing the risk for high-dose lead exposure should be part of routine pediatric care.

The urgency and type of follow-up depends on the screening blood lead test result.

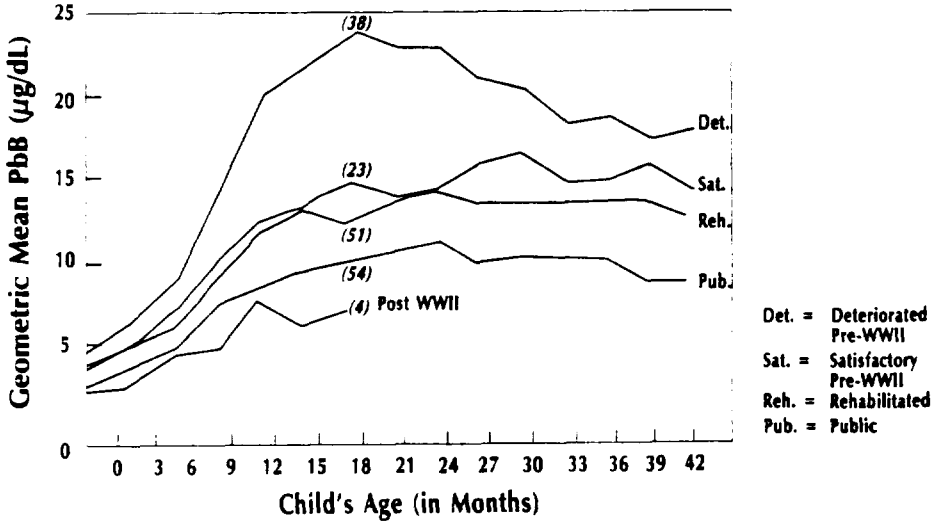
Background

The rationale for the screening schedule is based on data such as those shown in Figure 6-1. Those data were collected in a prospective study in Cincinnati (Clark et al., 1985). Blood lead levels were measured every 3 months from birth onward, and illustrate the trends in blood lead concentration in relation to the child's age and housing age and condition. Blood lead concentrations increase steadily up to at least 18 months of age. The most rapid rate of increase occurs between 6 and 12 months of age. The highest blood lead levels occur in children living in deteriorated older housing.

Assessment of Risk

Table 6-2 has sample questions. Starting at 6 months of age and at each regular office visit thereafter, pediatric health-care providers should discuss childhood lead poisoning and assess the child's risk for high-dose exposure. The questions asked should be tailored to the likely sources of exposure in the community. **The questions are not a substitute for a blood lead test.**

Figure 6-1. Relationship between children's blood lead levels and housing age and condition, Cincinnati



Note: Number of children at 18 months of age indicated in parentheses.

Source: Clark et al., 1985.

Table 6-2. 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 remodelling?
3. Have a brother or sister, housemate, or playmate being followed or treated for lead poisoning (that is, blood lead ≥15 μg/dL)?
4. Live with an adult whose job or hobby involves exposure to lead (see Chapter 3)?
5. Live near an active lead smelter, battery recycling plant, or other industry likely to release lead?

Using Questionnaire Results

If answers to the questionnaire indicate that the child is not at high risk for high-dose lead exposure, the child should be screened at 12 months of age, and, if resources allow, at 24 months of age.

If answers to the questionnaire indicate that the child is at risk for high-dose lead exposure, the child should be screened starting at 6 months of age.

For children previously at low risk, any history suggesting that exposure to lead has increased should be followed up with a blood lead test.

On the basis of responses to questions such as those in Table 6-2, children can be categorized as low or high risk for high-dose lead exposure. If the answers to all questions are negative, the child is at low risk for high-dose lead exposure and should be screened by a blood lead test at 12 months and again, if possible, at 24 months (since blood lead levels often peak at ages greater than 12 months). If the answer to any question is positive, the child is potentially at high risk for high-dose lead exposure, and a blood lead test should be obtained. **For children previously at low risk, any history suggesting that exposure to lead has increased should be followed up with a blood lead test.**

Example: A pediatrician in southern California almost exclusively serves communities built after 1988. She is aware, however, that some of her patients' families store juice and punch in pottery imported from Mexico. In her guidance to parents, she warns them that lead can leach from improperly fired pottery. At every routine visit, she asks parents about the use of this pottery and screens any children whose parents use this pottery. In her anticipatory guidance and assessment of her patients' risks of lead poisoning, she emphasizes sources of exposure that are common in the community she serves.

Screening Schedule

The following sections provide a minimum screening schedule for children aged 6 up to 36 and 36 to 72 months. The schedule is not rigid. Rather, it is a guide for pediatric health-care providers and screening programs to use in conjunction with other pertinent information in determining when an individual child should be tested.

Children 6 up to 36 months of age:

A questionnaire should be used at each routine office visit to assess the potential for high-dose lead exposure and, therefore, the appropriate frequency of screening.

- ***Schedule if the child is at low risk for high dose lead exposure by questionnaire:***

  A child at *low risk* for exposure to high-dose lead sources by questionnaire should have an initial blood lead test at 12 months of age.

  If the 12-month blood lead result is **<10 μg/dL**, the child should be retested at 24 months if possible, since that is when blood lead levels peak.

  If a blood lead test result is **10-14 μg/dL**, the child should be retested every 3 to 4 months. After 2 consecutive measurements are <10 μg/dL or three are <15 μg/dL, the child should be retested in a year.

  If any blood lead test result is **≥15 μg/dL**, the child needs individual case management, which includes retesting the child at least every 3 to 4 months (Page 45).

- ***Schedule if the child is at high risk for high dose lead exposure by questionnaire:***

  A child at *high risk* for exposure to high-dose lead sources by questionnaire should have an initial blood lead test at 6 months of age.

  If the initial blood lead result is **<10 μg/dL**, the child should be rescreened every 6 months. After 2 subsequent consecutive measurements are <10 μg/dL or three are <15 μg/dL, testing frequency can be decreased to once a year.

  If a blood lead test result is **10-14 μg/dL**, the child should be screened every 3 to 4 months. Once 2 subsequent consecutive measurements are <10 μg/dL or three are <15 μg/dL, testing frequency can be decreased to once a year.

  If any blood lead test result is **≥15 μg/dL**, the child needs individual case management, which includes retesting the child at least every 3 to 4 months (Page 45).

Children ≥36 months and <72 months of age:

As for younger children, a questionnaire should be used at each routine office visit of children from 36 to 72 months of age. Any child at high risk by questionnaire who has not previously had a blood lead test should be tested. All children who have had venous blood lead tests ≥15 μg/dL or who are at high risk by questionnaire should be screened at least once a year until their sixth birthday (age 72 months) or later, if indicated (for example, a developmentally delayed child with pica). Children should also be rescreened any time history suggests exposure has increased. Children with blood lead levels ≥15 μg/dL should receive followup as described below.

Followup of children with blood lead levels ≥15 μg/dL

Followup of children with blood lead levels ≥15 μg/dL is discussed in more detail in later chapters and is briefly summarized below. In general, such children should receive blood lead tests at least every 3 to 4 months.

If the blood lead level is 15-19 μg/dL, the child should be screened every 3-4 months, the family should be given education and nutritional counselling as described in Chapter 4, and a detailed environmental history should be taken to identify any obvious sources or pathways of lead exposure. When the venous blood lead level is in this range in two consecutive tests 3-4 months apart, environmental investigation and abatement should be conducted, if resources permit.

If the blood lead level is ≥20 μg/dL, the child should be given a repeat test for confirmation. If the venous blood lead level is confirmed to be ≥20 μg/dL, the child should be referred for medical evaluation and followup as described in Chapter 7. Such children should continue to receive blood lead tests every 3-4 months or more often if indicated. Children with blood lead levels ≥45 μg/dL must receive urgent medical and environmental followup, preferably at a clinic with a staff experienced in dealing with this disease. Symptomatic lead poisoning or a venous blood lead concentration ≥70 μg/dL is a medical emergency, requiring immediate inpatient chelation therapy, as described in Chapter 7.

CLASSIFICATION ON THE BASIS OF SCREENING TEST RESULTS

On the basis of screening test results, children can be classified into categories according to their risk for adverse effects of lead. The urgency and type of followup are based on these risk classes. These classes are shown in Table 6-3.

MEASUREMENT OF BLOOD LEAD LEVELS

Venous blood is preferred for blood lead measurement.
Analytical variation in the laboratory can affect blood lead results.
Laboratories measuring blood lead levels should participate successfully in a blood lead proficiency testing program.

Several factors can influence the quality of blood lead measurements. The ubiquity of lead in the environment makes contamination of specimens during collection a major source of error. Analytical variation in the laboratory can affect results. Accuracy and precision of blood lead measurements, particularly at low concentrations, can be assured by the use of appropriate analytical standards, maintenance of equipment, training of personnel, and participation in external proficiency testing programs.

Since blood collected by venipuncture has a low likelihood of contamination compared to blood collected by fingerstick, venous blood is the preferred specimen for analysis and should be used for lead measurement whenever practicable. In addition, venous specimens provide a larger volume for analysis and are less prone to clotting and other problems that can be encountered with capillary specimens (DeSilva and Donnan, 1977; Mitchell et al., 1974).

Table 6-3. Class of child and recommended action according to blood lead measurement

Class	Blood Lead Concentration (μg/dL)	Action
I	≤9	Low risk for high-dose exposure: rescreen as described in text. High risk for high-dose exposure: rescreen as described in text.
IIA	10-14	Rescreen as described in text. If many children in the community have blood lead levels ≥10, community interventions (primary prevention activities) should be considered by appropriate agencies (see Chapter 9).
IIB	15-19	Rescreen as described in text. Take a history to assess possible high-dose sources of lead. Educate parents about diet, cleaning, etc. Test for iron deficiency. Consider environmental investigation and lead hazard abatement if levels persist.
III	20-44*	Conduct a complete medical evaluation. Identify and eliminate environmental lead sources.
IV	45-69*	Begin medical treatment and environmental assessment and remediation within 48 hours.
V	≥70*	Begin medical treatment and environmental assessment and remediation IMMEDIATELY.

*Based on confirmatory blood lead level.

Fingerstick specimens are acceptable for blood lead screening, provided that special collection procedures are followed to minimize the risk of contamination. Personnel must be thoroughly trained in collection procedures. A procedure for collecting fingerstick specimens is described in Appendix I. At the present time, not all laboratories will accept capillary samples for blood lead analysis.

Elevated blood lead results obtained on capillary specimens are presumptive and must be confirmed using venous blood. In general, children who have blood lead levels ≥15 μg/dL on capillary samples should have these levels confirmed on venous samples, according to the timetable in Table 6-4. A child with a blood lead level ≥70 μg/dL or with symptoms of lead poisoning should be treated immediately while the results of an immediate confirmatory test are awaited.

Additional Analytical Considerations

Blood lead levels can be determined by several analytic methods. The method used can affect the specimen volume required, the choice of anticoagulant (usually heparin or ethylenediaminetetraacetic acid (EDTA)), and other aspects related to specimen suitability. Specimen collection procedures and equipment must be checked for compatibility with laboratory

Table 6-4. 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 month
20-44	Within 1 week
45-69	Within 48 hours
≥70	Immediately

requirements. Special lead-free evacuated tubes are available for blood collection, but standard tubes containing EDTA or heparin (lavender or green caps) can be acceptable after screening each lot to determine the lead content of the containers, needles, etc. Though reports of unsuitable levels of background lead in other collection materials are infrequent, all materials used should be determined to be lead-free before use.

Several analytical techniques available can be used to make accurate blood lead measurements at levels <25 μg/dL. These techniques are electrochemical techniques, usually anodic stripping voltammetry (ASV), and atomic absorption spectroscopy (AAS). Either of these techniques is capable of achieving detection limits <2 to 5 μg/dL. Success by these methods, however, requires careful and meticulous attention to the details of the analysis.

The reliability of a set of blood lead measurements is greatly enhanced by the use of high quality lead standard solution for instrument calibration. In the United States, the National Institute of Science and Technology has made such a material (SRM 3121) available. In addition, a set of whole blood reference materials (SRM 955A, Lead in Blood) provides a useful set of control materials over a wide range of concentrations—about 6 to 70 μg/dL.

Laboratories where blood is tested for lead levels should be successful participants in a blood lead proficiency testing program, such as the program conducted jointly by CDC, the Health Resources and Services Administration, and the University of Wisconsin. In interpreting laboratory results, it should be recognized that a proficient laboratory should measure blood lead levels to within several μg/dL of the true value (for example, within 4 or 6 μg/dL of a target value). The blood lead level reported by a laboratory, therefore, may be several μg/dL higher or lower than the actual blood lead level.

Analytical variability must be considered when interpreting blood lead results. Changes in successive blood lead measurements on an individual can be considered significant only if the net difference of results exceeds the limit of analytic variance that the laboratory allows. As a general rule, trends should not be considered significant unless the magnitude of the change is ≥5 μg/dL.

The degree of analytical variability between laboratories that employ different analytic methods usually exceeds that within a single laboratory. Therefore, a single laboratory using one analytical method should be used to best compare multiple blood lead results from an individual or a population.

ERYTHROCYTE PROTOPORPHYRIN (EP)

EP is not a sensitive test for identifying children with blood lead levels below about 25 μg/dL.

An EP level is elevated if it is ≥35 μg/dL when standardized using 241 L cm-1 mmol-1, ≥28 μg/dL when standardized using 297 L cm-1 mmol-1, or ≥70 μmol/mol when measured in μmol/mol units. All elevated EP results should be followed by a venous blood lead test.

Laboratories measuring EP levels should be successful participants in an EP proficiency testing program.

Interpreting EP Results and Following Up on Children with High EP Levels

EP is not a sensitive test to identify children with blood lead levels below about 25 μg/dL, and therefore it is no longer the screening test of choice. Generally, EP is measured using a two-step extraction process followed by direct fluorometric measurement or by front-surface fluorometry (hematofluorometry). Most protoporphyrin in erythrocytes (about 90%) exists as zinc protoporphyrin (ZnPP) (Smith et al., 1980; Gotelli et al., 1980). This fraction is preferentially measured by hematofluorometers. Extraction methods measure all the protoporphyrin present, but strip the zinc from the ZnPP during the extraction process. For this reason, extraction results are sometimes referred to as [zinc] free erythrocyte protoporphyrin (FEP). Although the chemical forms measured by the two methods differ slightly, on a weight basis they are roughly equivalent, so results reported as EP, ZnPP, or FEP all reflect essentially the same analyte (Stanton et al., 1989).

In the past, an absorptivity of 241 L cm-1 mmol-1 has been used to determine EP levels. Recently, however, the correct absorptivity has been determined to be 297 L cm-1 mmol-1 (Gunter et al., 1989). Use of the correct absorptivity will result in EP values about 19% lower than those standardized using 241 L cm-1 mmol-1. Standardization of EP levels that are based on the correct absorptivity is expected to be widely adopted in 1992. Use of the correct standardization requires a change in calibration and is not simply a reduction of the screening cutoff value. Standardization criteria should also be considered when reviewing data in the literature.

An EP result of ≥35 μg/dL standardized using 241 L cm-1 mmol-1 or ≥28 μg/dL standardized using 297 L cm-1 mmol-1 is considered elevated. **All elevated EP results should be followed with a venous blood lead test to determine if lead poisoning is responsible for the elevation.** Elevated concentrations of EP also result from several health conditions other than lead intoxication, particularly iron deficiency (Reeves et al., 1984; Yip et al., 1983; Thomas et al., 1977). The iron status of children with elevated EP levels should always be determined, especially since iron deficiency and lead poisoning often coexist. In such cases, the EP may be disproportionately elevated in comparison to the blood lead level.

Some hematofluorometers report EP levels as μmol ZnPP/mol heme. For instruments that give results in these units, EP values ≥70 μmol/mol should be considered elevated and should be promptly investigated (Stanton et al., 1989).

Analytic Considerations

Only fresh blood is suitable for analysis by hematofluorometer (Blumberg et al., 1977). Complete oxygenation of sample hemoglobin is necessary to prevent low results in some instruments. The hemoglobin concentration in the sample can also affect hematofluorometer EP readings. Results obtained by extraction methods are not affected by these factors and can be used to confirm hematofluorometer EP results.

As with lead data, analytical variance must be considered when EP data are being interpreted. If trends in EP data are to be assessed correctly, analyses should preferably be performed by a single laboratory, and the variance of the method should be known when interpreting data. As with blood lead levels, interlaboratory variance usually exceeds intralaboratory variance. The observed variance for EP is wider than that for blood lead, underscoring the importance of analytical variance in the evaluation of EP data. In addition, because of substantial intermethod differences, extraction and hematofluorometer results should not be compared when assessing trends (Mitchell and Doran, 1985; Kaul et al., 1983; Peter et al., 1978). Laboratories that test patient specimens for EP levels should be participants in one or more external proficiency testing programs.

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Chapter 7. Diagnostic Evaluation and Medical Management of Children With Blood Lead Levels ≥20 μg/dL

Summary

Children with blood lead levels ≥20 μg/dL need complete medical evaluations.

Several pharmacologic agents can reduce blood lead levels; however, the most important factor is reducing the child's exposure to lead.

Research and new developments may change many aspects of the medical management of poisoned children.

Children with blood lead levels between 10 μg/dL and 19 μg/dL and their siblings need followup and repeat screening as described in previous chapters. They do not, however, need medical evaluation as described in this chapter.

The cornerstones of clinical management are careful clinical and laboratory surveillance of the child, medical treatment when indicated, and eradication of controllable sources of environmental lead. **The most important factor in case management is to reduce the child's exposure to lead.**

All children with confirmed venous blood lead levels ≥20 μg/dL require medical evaluation. The urgency of further medical evaluation depends on the blood lead level and whether symptoms are present.

The decision to institute medical management should virtually always be made on the basis of a venous blood lead measurement. No other screening test can be considered diagnostic. If the first evaluation was made on capillary blood, a confirmatory venous blood lead level must be done. Even if the first diagnostic measurement was on venous blood, it is preferable to retest before starting chelation therapy. For children with blood lead levels ≥70 μg/dL or clinical symptoms of lead poisoning, chelation should not be postponed while awaiting results of the repeat test.

SYMPTOMS OF LEAD POISONING

Symptomatic lead poisoning is a medical emergency.

Symptoms of lead poisoning in a child with an elevated blood lead level constitute a medical emergency, and the child should be hospitalized. Symptoms, which can mimic several other pediatric disorders, must be looked for so they are not missed (Piomelli et al., 1984).

Acute lead encephalopathy is characterized by some or all of these symptoms: coma, seizures, bizarre behavior, ataxia, apathy, incoordination, vomiting, alteration in the state of consciousness, and subtle loss of recently acquired skills. Any one or a mixture of these symptoms, associated with an elevated blood lead level, is an acute medical emergency. Lead encephalopathy is almost always associated with a blood lead level exceeding 100 μg/dL, although, occasionally, it has been reported at blood lead levels as low as 70 μg/dL. Even when identified and promptly treated, severe and permanent brain damage may result in 70%-80% of children with lead encephalopathy (Perlstein and Attala, 1966). Children with symptomatic lead poisoning with or without encephalopathy represent an acute medical emergency. **The possibility of lead encephalopathy should be considered in the differential diagnosis of children presenting with coma and convulsions of unknown etiology.**

Except for coma and seizures, symptomatic lead poisoning without encephalopathy is characterized by symptoms similar to those of lead encephalopathy. Symptomatic lead poisoning without encephalopathy is characterized by one or a combination of these symptoms: decrease in play activity, lethargy, anorexia, sporadic vomiting, intermittent abdominal pain, and constipation. These symptoms are usually associated with a blood lead levels of at least 70 μg/dL, although occasionally cases have been associated with levels as low as 50 μg/dL. If the blood lead level is below 50 μg/dL, other causes of the symptoms should be sought. **Since acute lead encephalopathy may develop in any symptomatic child, treatment and supportive measures must be started immediately on an emergency basis.**

EVALUATION OF THE CHILD WITH A BLOOD LEAD LEVEL ≥20 μg/dL

Take a careful history and do a physical examination. Include evaluation of the child's iron status and other special diagnostic tests.

History and Physical Examination

A child with a blood lead level ≥20 μg/dL should have a pediatric evaluation, whether or not symptoms are present.

Special attention should be given to:

1. A detailed history, including the presence or absence of clinical symptoms, child's mouthing activities, the existence of pica, nutritional status (especially iron and calcium intake), dietary habits, family history of lead poisoning, potential sources of lead exposure (including exposure due to home renovation), and previous blood lead measurements.
2. Detailed environmental and occupational histories of adults in the household or other places the child spends a lot of time.
3. The physical examination, with particular attention to the neurologic examination and psychosocial and language development. A neurobehavioral assessment may be useful in children receiving chelation therapy both at the time of diagnosis and as the child approaches school age. Findings of language delay or other problems can prompt referral to appropriate programs.

4. Evaluation of iron status using measurement of iron and total iron binding capacity or of ferritin.

Iron Status and Special Tests

1. Tests for Iron Deficiency

Because iron deficiency can enhance lead absorption and toxicity and often coexists with it, all children with blood lead levels ≥20 μg/dL should be tested for iron deficiency. Measurements of hemoglobin, hematocrit, and reticulocytes are not adequately sensitive, and erythrocyte protoporphyrin (EP) is not specific enough to diagnose iron deficiency (although EP can be used to screen for iron deficiency).

Serum iron and iron binding capacity (transferrin saturation) and ferritin are the most sensitive indicators of iron status. An abnormally low ratio of serum iron to iron binding capacity (transferrin saturation) of 0.2 is consistent with iron deficiency. The serum ferritin level, however, is the most definitive and accurate indication of overall iron status, although it is an acute phase reactant and may be falsely elevated in sick children; a value ≤12 μg/dL indicates iron deficiency. Although all iron deficient children should receive treatment for this condition, the treatment should not be started until after chelation is completed in children receiving dimercaprol (BAL).

2. EP Level

An elevated EP level indicates impairment of the heme biosynthetic pathway. EP levels are sensitive screening tests for iron deficiency, and iron status should be assessed in any child with an elevated EP level (that is, ≥35 μg/dL when standardized using 241 L cm-1 mmol-1, ≥28 μg/dL when standardized using 297 L cm-1 mmol-1, or ≥70 μmol/mol when measured in μmol/mol units).

Because EP levels take about 2 weeks to increase, EP levels may provide an indication of the duration of lead exposure (Chisolm, 1982; Chisolm, personal communication). Similarly, monitoring the EP level after medical and environmental interventions for poisoned children may be useful. If exposure to lead has ceased, EP values elevated because of lead poisoning decline slowly over several weeks or months (Piomelli et al., 1984). A progressive decline in EP concentrations indicates that combined medical and environmental case management is proceeding efficaciously.

3. Edetate Disodium Calcium ($CaNa_2EDTA$) Provocative Chelation Test

The mobilization test is used to determine whether a child with an initial confirmatory blood lead level of 25 to 44 μg/dL will respond to chelation therapy with a brisk lead diuresis (Piomelli et al., 1984; Markowitz and Rosen, 1991). Because of the cost and staff time needed for quantitative urine collection, this test is used only in selected medical centers where large numbers of lead-poisoned children are treated. Children whose blood lead levels are ≥45 μg/dL should not receive a provocative chelation test; they should be referred for appropriate chelation therapy immediately.

The outcome of the provocative chelation test is determined not by a decrease in the blood lead level but by the amount of lead excreted per dose of $CaNa_2EDTA$ given. This ratio correlates well with blood lead levels. In one study, almost all children with blood lead levels 45 μg/dL had positive provocative tests, 76% of the children with blood lead levels 35 to 44 μg/dL

had positive test results, and 35% of the children with blood lead levels 25 to 34 μg/dL had positive test results (Markowitz and Rosen, 1991). This test should not be done until the child is iron replete, since iron status may affect the outcome of the test (Markowitz et al., 1990).

Conducting a $CaNa_2EDTA$ Provocative Chelation Test. First, a repeated baseline blood lead level must be obtained. The patient is asked to empty the bladder, and then $CaNa_2EDTA$ is administered at a dose of 500 mg/m^2 in 5% dextrose infused **over 1 hour**. (A somewhat painful but practical alternative is to administer intramuscularly the same dose mixed with procaine so that the final concentration of procaine is 0.5%.) All urine must be collected with lead-free equipment over the next 8 hours. (An 8 hour mobilization test has been shown to be as reliable as a 24-hour mobilization test (Markowitz and Rosen, 1984).) An 8-hour test can be accomplished on an out-patient basis, but the patient should not leave the clinic during this test.) In the laboratory, the urine volume should be carefully measured and stored at 20°C until the lead concentration is measured. Extreme care must be taken to ensure that lead-free equipment is used.

The use of lead-free apparatus for urine collection is mandatory. Special lead-free collection apparatus must be used if valid test results are to be obtained. The laboratory that will perform the analysis should supply the proper collection apparatus. Preferably, urine should be voided directly into polyethylene or polypropylene bottles that have been cleaned by the usual procedures, then washed in nitric acid, and thoroughly rinsed with deionized, distilled water. For children who are not toilet trained, plastic pediatric urine collectors can be used. Urine collected in this manner should be transferred directly to the urine collection bottles.

Interpretation of a $CaNa_2EDTA$ Provocative Chelation Test. To obtain the total lead excretion in micrograms, the concentration of lead in the urine (in micrograms per milliliter) is multiplied by the total urinary volume (in milliliters). The total urinary excretion of lead (micrograms) is divided by the amount of $CaNa_2EDTA$ given (milligrams) to obtain the lead excretion ratio:

$$\frac{\text{Lead excreted } (\mu g)}{CaNa_2EDTA \text{ given (mg)}}$$

An 8-hour $CaNa_2EDTA$ chelation provocative test is considered positive if the lead excretion ratio is >0.6 (Markowitz and Rosen, 1991). Some clinicians use a cutoff of 0.5 for the lead excretion ratio (Weinberger et al., 1987). Children with blood lead levels 25 to 44 μg/dL and positive chelation test results should undergo a 5-day course of chelation.

Regardless of age, all children with elevated blood lead values and negative provocative chelation results should have blood lead levels measured monthly. If the elevation in blood lead values persists, the $CaNa_2EDTA$ provocative test can be repeated every 1 to 3 months and interpreted according to the above guidelines.

4. Radiologic Examination of the Abdomen

Radiologic examination of the abdomen (flat plate) may show radiopaque foreign material if the material has been ingested during the preceding 24 to 36 hours. Neither negative nor positive xray results are diagnostic or definitive. A flat plate of the abdomen may, however, provide information about the source of lead if paint chips or other lead objects are found.

5. Radiologic Examination of the Long Bones

Xrays of the long bones are unreliable for diagnosing acute lead poisoning, and they should not be obtained on a routine basis. They may provide some indication of whether lead poisoning

has occurred in the past or has been ongoing for a length of time, and this may occasionally be important. Lines of increased density in the metaphyseal plate of the distal femur, proximal tibia, and fibula may be caused by lead which has disrupted the metabolism of bone matrix. Although these lines are sometimes called lead lines, they are areas of increased mineralization or calcification and not xray shadows of deposited lead.

The following tests are **NOT** indicated for the diagnosis or clinical management of lead poisoning:

1. Microscopic Examination of Red Cells for Basophilic Stippling

Since basophilic stippling is not always found in severe lead poisoning and is insensitive to lesser degrees of lead poisoning, it is not useful in diagnosis.

2. Tests of Hair and Fingernails for Lead Levels

The levels of lead in hair or fingernails do not correlate well with blood lead levels, except in extreme cases of symptomatic lead poisoning; therefore, these tests are not useful in diagnosis. Children should never receive chelating agents on the basis of analyses of lead levels in hair or fingernails.

PHARMACOLOGY OF CHELATING AGENTS

Chelating Agents Used In Treating Children With Lead Poisoning

Product Name	Generic Name	Chemical Name	Abbreviation
Calcium Disodium Versenate	Edetate disodium calcium	Calcium disodium ethylenediamine tetraacetate	$CaNa_2$ EDTA
BAL in Oil	Dimercaprol	2,3-dimercapto-1-propanol	BAL
Cuprimine	D-penicillamine	3-mercapto-D-valine	D-penicillamine
Chemet	Succimer	Meso 2,3-dimercaptosuccinic acid	DMSA

Several drugs are used in the treatment of lead poisoning. These drugs, capable of binding or chelating lead, deplete the soft and hard (skeletal) tissues of lead and thus reduce its acute toxicity (Chisolm, 1968; Markowitz and Rosen, 1984; Piomelli et al., 1984; Rosen et al., in press). All drugs have potential side effects and must be used with caution (Piomelli et al., 1984). The basic pharmacologic characteristics of the various drugs are described below.

BAL

Mechanism of action. Two molecules of dimercaprol (BAL) combine with one atom of heavy metal to form a stable complex. BAL enhances fecal and urinary excretion of lead and diffuses well into erythrocytes. Because it is predominantly excreted in bile, BAL can be administered in the presence of renal impairment (Chisolm, 1968).

Route of administration and dosage. BAL is available only in peanut oil for intramuscular administration. It is usually given every 4 hours, although it may be given every 8 hours; dosages are discussed starting on page 59.

Precautions and Toxicity. For patients with glucose-6-phosphate dehydrogenase deficiency (G-6-PD), some clinicians recommend that BAL should be used only in life-threatening situations because it may induce hemolysis. Medicinal iron should never be administered during BAL therapy, because the combination of iron and BAL has been implicated in serious reactions. If iron deficiency coexists, it should not be treated until after BAL therapy has been completed. In cases of extreme anemia, blood transfusions are preferable.

Between 30% and 50% of patients who receive BAL will experience side effects. Mild febrile reactions and transient elevations of hepatic transaminases may be observed. Other minor adverse effects include, in order of frequency, nausea and occasional vomiting, headache, mild conjunctivitis, lacrimation, rhinorrhea, and salivation. Most side effects are transient and rapidly subside as the drug is metabolized and excreted. Intravenous hydration coupled with restricting oral intake can circumvent, in large part, gastrointestinal distress.

BAL should not be used for children who are allergic to peanuts or peanut products.

$CaNa_2EDTA$

Only $CaNa_2EDTA$ can be used for treating children with lead poisoning. Na_2EDTA (disodium edetate) should never be used for treating children with lead poisoning because it will induce tetany and possibly fatal hypocalcemia.

Mechanism of action. $CaNa_2EDTA$ increases urinary lead excretion twentyfold to fiftyfold. $CaNa_2$ EDTA removes lead from the extracellular compartment only, because it does not enter cells (Osterloh and Becker, 1986).

Route of administration and dosage. The preferred route for administration of $CaNa_2EDTA$ is intravenous. $CaNa_2EDTA$ must be diluted to a concentration of <0.5% either in dextrose and water or in 0.9% saline solution. It can be given as a continuous infusion or it can be given in two divided doses a day through a heparin lock over 30 to 60 minutes. $CaNa_2EDTA$ causes extreme pain when administered intramuscularly; therefore, when given by this route, it should be mixed with procaine so that the final concentration of procaine is 0.5%. $CaNa_2EDTA$ should never be given orally because it enhances absorption of lead from the gastrointestinal tract.

Dosages vary by situation and are detailed starting on page 59. Individual courses should be limited to 5 days and repeated courses should be given at a minimum of 2- to 5-day intervals. Particularly when $CaNa_2EDTA$ is given on an outpatient basis, some clinicians use sequential 3-day courses of treatment.

Precautions and Toxicity. During chelation therapy with $CaNa_2EDTA$, urine output, urine sediment, blood urea nitrogen (BUN), serum creatinine, and hepatocellular enzyme levels must be carefully monitored. The appearance of protein and formed elements in urinary sediment, and rising BUN and serum creatinine values reflect impending renal failure—the serious toxicity associated with inappropriately excessive or prolonged administration of $CaNa_2EDTA$. Liver transaminases may increase by the fifth day of therapy, but return to pretreatment levels within a week after treatment has ended.

When $CaNa_2EDTA$ is used alone without concomitant BAL therapy, it may aggravate symptoms in patients with very high blood lead levels. Therefore, it should be used in conjunction with BAL when the blood lead level is $\geq$70 μg/dL or overt clinical symptoms of lead poisoning are present. In such cases, the first dose of BAL should always precede the first dose of $CaNa_2EDTA$ by at least 4 hours.

The kidney is the principal site of potential toxicity. Renal toxicity is dose related, reversible, and rarely (if ever) occurs at doses <1500 mg/m^2 when the patient is adequately hydrated. $CaNa_2EDTA$ must never be given in the absence of an adequate urine flow (Piomelli et al., 1984).

D-penicillamine

The Food and Drug Administration (FDA) has approved D-penicillamine for the treatment of Wilson's disease, cystinuria, and severe, active rheumatoid arthritis. Although not approved for this use, it is used in some centers for treating lead poisoning. Until the recent approval of succimer, it was the only commercially available oral chelating agent. It can be given over a long period (weeks to months). D-penicillamine has been used mainly for children with blood lead levels <45 μg/dL.

Mechanism of action. D-penicillamine enhances urinary excretion of lead, although not as effectively as $CaNa_2EDTA$. Its specific mechanism and site of action are not well understood.

Route of administration and dosage. D-penicillamine is administered orally. It is available in capsules or tablets (125 mg and 250 mg). These capsules can be opened and suspended in liquid, if necessary. The usual dose is 25 to 35 mg/kg/day in divided doses. Side effects can be minimized, to an extent, by starting with a small dose and increasing it gradually, monitoring all the time for side effects. For example, 25% of the desired final dose could be given in week 1, 50% in week 2, and the full dose by week 3.

Precautions and Toxicity. Toxic side effects (albeit minor in most cases) occur in as many as 33% of patients given the drug (Shannon et al., 1988). The main side effects of D-penicillamine are reactions resembling those of penicillin sensitivity, including rashes, leukopenia, thrombocytopenia, hematuria, proteinuria, hepatocellular enzyme elevations, and eosinophilia. Anorexia, nausea, and vomiting are infrequent. Of most concern, however, are isolated reports of nephrotoxicity, possibly from hypersensitivity reactions. For these reasons, patients should be carefully and frequently monitored for clinically obvious side effects, and frequent blood counts, urinalyses, and renal function tests should be performed. In particular, blood counts and urinalyses should be done on day 1, day 14, day 28, and monthly thereafter. If the absolute neutrophil count falls to <1500/μL, the count should be rechecked immediately, and treatment should be stopped if it falls to <1200/μL. D-penicillamine should not be given on an outpatient basis if exposure to lead is continuing or the physician has doubts about compliance with the therapeutic regimen.

D-penicillamine should not be administered to patients with known penicillin allergy.

Succimer

The FDA approved succimer in January, 1991 for treating children with blood lead levels >45 μg/dL. Succimer appears to be an effective oral chelating agent. Its selectivity for lead is high, whereas its ability to chelate essential trace metals is low. Although its use to date has been limited, succimer appears to have promising potential, and a broader range of clinical research studies in children are being undertaken.

Succimer is chemically similar to BAL but is more water soluble, has a high therapeutic index, and is absorbed from the gastrointestinal tract (Aposhian and Aposhian, 1990). It is

effective when given orally and produces a lead diuresis comparable to that produced by $CaNa_2EDTA$ (Chisolm, 1990). This diuresis lowers blood lead levels and reverses the biochemical toxicity of lead, as indicated by normalization of circulating aminolevulinic acid dehydrase levels (Graziano et al., 1988). Succimer is not indicated for prophylaxis of lead poisoning in a lead-containing environment. **As with all chelating agents, succimer should only be given to children who reside in environments free of lead during and after treatment.**

Mechanism of Action. Succimer appears to be more specific for lead than the most commonly used chelating agent, $CaNa_2EDTA$; the urinary loss of essential trace elements (for example, zinc) appears to be considerably less with succimer than with $CaNa_2EDTA$ (Aposhian and Aposhian, 1990). The site of lead chelation by succimer is not known.

Route of Administration and Dosage. Succimer is administered orally. It is available in 100 mg capsules. The recommended initial dose is 350 mg/m^2 (10 mg/kg) every 8 hours for 5 days, followed by 350 mg/m^2 (10 mg/kg) every 12 hours for 14 days. A course of treatment, therefore, lasts 19 days. If more courses are needed, a minimum of 2 weeks between courses is preferred, unless blood lead levels indicate the need for immediate retreatment. These doses may be modified as more experience is gained in using succimer.

Patients who have received therapeutic courses of $CaNa_2EDTA$ with or without BAL may use succimer for subsequent treatment after an interval of 4 weeks. Data on the concomitant use of succimer and $CaNa_2EDTA$ with or without BAL are not available, and such use is not recommended.

If young children cannot swallow capsules, succimer can be administered by separating the capsule and sprinkling the medicated beads on a small amount of soft food or by putting them on a spoon and following with a fruit drink. Data are not available on how stable succimer is when it is suspended in soft foods for prolonged periods of time; succimer should be mixed with soft foods immediately before being given to the child.

Precautions and Toxicity. To date, toxicity due to succimer (transient elevations in hepatic enzyme activities) appears to be minimal (Graziano et al., 1988). The most common adverse effects reported in clinical trials in children and adults were primarily gastrointestinal and included nausea, vomiting, diarrhea, and appetite loss. Rashes, some necessitating discontinuation of therapy, have been reported for about 4% of patients. **Though succimer holds considerable promise for the outpatient management of lead poisoning, clinical experience with succimer is limited.** Consequently, the full spectrum and incidence of adverse reactions, including the possibility of hypersensitivity or idiosyncratic reactions, have not been determined.

If succimer is used, the following precautions must be taken:

1. Monitor for side effects (especially effects on liver transaminases), the rapidity of the initial decrease in blood lead levels, and the course of the rebound in blood lead levels once treatment has ended.
2. **Succimer, like other chelators, is not a substitute for effective and rapid environmental interventions.** Use succimer as part of an integrated environmental and medical approach to treating patients with lead poisoning.
3. Do not give succimer (or any other chelating agent) in situations where high dose lead sources are available to the child. In rats, gastrointestinal absorption of lead and whole body lead retention were reduced by a single oral dose of succimer (Kapoor et al., 1989). The potential for enhancing human lead absorption from the gastrointestinal tract during the use of succimer is under study.

4. Children with blood lead levels >45 μg/dL who are being treated with succimer, should, if possible, be hospitalized until their blood lead levels fall below 45 μg/dL and the lead hazards in their homes are abated or alternative lead hazard-free housing has been identified.
5. Children with blood lead levels ≥70 μg/dL should be immediately hospitalized. The decision to treat such children with succimer instead of $CaNa_2EDTA$ and BAL should be made with the understanding that experience with using succimer in children with these blood lead levels is limited.

TREATMENT GUIDELINES FOR CHILDREN WITH BLOOD LEAD LEVELS ≥20 μg/dL

The most important factor in managing childhood lead poisoning is reducing the child's exposure to lead.
Children with symptomatic lead poisoning, with and without encephalopathy, should be managed by a multidisciplinary team.
Asymptomatic children with blood lead levels ≥45 μg/dL should receive chelation therapy.
Different clinical centers and programs use different protocols to medically manage children with blood lead levels of 25 to 44 μg/dL.

The single most important factor in managing of childhood lead poisoning is reducing the child's exposure to lead; some children, however, will benefit from chelation therapy. One approach for pharmacologic treatment of children with lead poisoning follows. It is a general guide and is not the only pharmacologic regimen that can be used to treat poisoned children.

Medical Management of Symptomatic Lead Poisoning (with or without Encephalopathy)

General Management. Children with symptomatic lead poisoning (with or without encephalopathy) must be treated only at a pediatric center that has an intensive care unit. They should be managed by a multidisciplinary team that includes, as needed, critical care, toxicology, neurology, and neurosurgery. The child's neurological status and fluid balance must be carefully monitored.

The symptoms associated with lead poisoning (with or without lead encephalopathy) are described on page 51. One or more of those symptoms associated with an elevated blood lead level constitutes an acute medical emergency. Because chelation regimens are the same for cases of symptomatic lead poisoning (with and without encephalopathy), guidelines for clinical management have been included in a single section.

Chelation therapy. Although succimer has been approved for chelation of children with blood lead levels >45 μg/dL, experience in treating symptomatic children is limited. Therefore, the treatment regimen discussed here uses $CaNa_2EDTA$ and BAL. Chelation with succimer is discussed in more detail on page 57.

Start treatment with a dose of 75 mg/m^2 BAL only, given by deep intramuscular injection; administer BAL at a dose of 450 mg/m^2/day in divided doses of 75 mg/m^2 every 4 hours. Once this dose is given and an adequate urine flow is established, administer $CaNa_2EDTA$ at a dose of 1,500 mg/m^2/day. Give $CaNa_2EDTA$ as a continuous intravenous infusion in dextrose and water or in a 0.9% saline solution. The concentration of $CaNa_2EDTA$ should not exceed 0.5% in the parenteral fluid. (When treating a child with encephalopathy, the physician may choose to give $CaNa_2EDTA$ intramuscularly to reduce the amount of fluid administered.) Treat with combined BAL-$CaNa_2EDTA$ therapy for a total of 5 days. During treatment, monitor renal and hepatic function and serum electrolyte levels daily (Piomelli et al., 1984).

A second course of chelation therapy with $CaNa_2EDTA$ alone (at blood lead levels 45-69 μg/dL) or combined with BAL (at blood lead levels 70 μg/dL), may be required once there is a rebound in the blood lead level after chelation. Wait at least 2 days before giving a second course of chelation. A third course is required only if the blood lead concentration rebounds to a value >45 μg/dL within 48 hours after the second course of treatment. Unless there are unusual and compelling clinical reasons, wait at least 5 to 7 days before beginning a third course of $CaNa_2EDTA$ (Piomelli et al., 1984).

Medical Management of Asymptomatic Lead Poisoning

Clinical management of asymptomatic lead-poisoned children with blood lead levels high enough to require chelation is similar to that of symptomatic children. Focus on reducing the child's exposure to lead and decreasing the child's body burden of lead.

Although succimer has been approved for chelation of children with blood lead levels >45 μg/dL, experience with this drug is limited. Therefore, the treatment regimen discussed here uses $CaNa_2EDTA$ and BAL. Chelation with succimer is discussed in more detail on page 57.

Blood lead level ≥70 μg/dL. Children with blood lead levels ≥70 μg/dL (with or without symptoms) represent an acute medical emergency. If the blood lead level is ≥70 μg/dL, give both BAL and $CaNa_2EDTA$ in the same doses and using the guidelines as for treatment of symptomatic lead poisoning (Page 59). A second course of chelation therapy with $CaNa_2EDTA$ alone may be required if the blood lead concentration rebounds to a value ≥45 μg/dL within 5 to 7 days after treatment. In general allow at least 5 to 7 days before beginning a second course of $CaNa_2EDTA$. Some practitioners give a second course of chelation after a 3-day rest period if the immediate post-treatment blood lead level is >35μg/dL (J. Chisolm, personal communication).

Blood lead level 45 to 69 μg/dL. If the blood lead value is between 45 and 69 μg/dL, chelation treatment should be limited to $CaNa_2EDTA$ only. $CaNa_2EDTA$ is given for 5 days at a dose of 1,000 mg/m^2/day intravenously by continuous infusion or in divided doses, as described on page 56. During treatment, evaluate renal and hepatic function and serum electrolyte levels regularly. Do not continue $CaNa_2EDTA$ treatment for more than 5 days (Piomelli et al., 1984).

A second course of chelation therapy with $CaNa_2EDTA$ alone may be required if the blood lead level rebounds to 45 μg/dL within 7 to 14 days after treatment. Allow 5 to 7 days before beginning a second course of $CaNa_2EDTA$.

Blood lead level 25 to 44 μg/dL. For this blood lead range, the effectiveness of chelation therapy in decreasing the adverse effects of lead on children's intelligence has not been shown. Treatment regimens vary from clinic to clinic. Some practitioners treat children with lead levels

in this range pharmacologically. (Although it is not approved for this use, some use D-penicillamine for children in this blood lead range.) The minimum medical management for children with these blood lead levels is to decrease the children's exposure to all sources of lead, to correct any iron deficiency and maintain an adequate calcium intake, and to test frequently to ensure that the child's blood lead levels are decreasing. Many experienced practitioners decide whether to use chelation therapy on the basis of the results of carefully performed $CaNa_2EDTA$ mobilization tests (Page 53).

Blood lead level 20 to 24 μg/dL. **Only very minimal data exists about chelating children with blood lead levels below 25 μg/dL, and such children should not be chelated except in the context of approved clinical trials.** A child with a confirmed blood lead level of 20 to 24 μg/dL will require individual case management by a pediatric health-care provider. The child should have an evaluation with special attention to nutritional and iron status. The parents should be taught about 1) the causes and effects of lead poisoning, 2) the need for more routine blood lead testing, 3) possible sources of lead intake and how to reduce them, 4) the importance of adequate nutrition and of foods high in iron and calcium, and 5) resources for further information. (This is described in more detail in Chapter 4.) Sequential measurements of blood lead levels along with review of the child's clinical status should be done at least every 3 months. Iron deficiency should be treated promptly. Children with blood lead levels in this range should be referred for environmental investigation and management. **Identifying and eradicating all sources of excessive lead exposure is the most important intervention for decreasing blood lead levels (Chapter 8).**

POST-CHELATION FOLLOWUP

Recheck blood lead levels 7 to 21 days after treatment. Determine if retreatment is necessary.
Do not discharge a child from the hospital until a lead free environment can be assured.

At the end of each treatment cycle, the blood lead concentration usually declines to <25 μg/dL. Within a few days, however, reequilibration among body lead compartments takes place and may result in a rebound; thus, **the blood lead level must be rechecked 7 to 21 days after treatment to determine whether retreatment is necessary** (Piomelli et al., 1984; Chisolm et al., 1985).

Children who undergo chelation treatment require long-term followup preferably from pediatric health-care providers, nutritionists, environmental specialists, and community outreach workers. Community outreach workers provide a critical bridge between hospital-based or clinic-based (outpatient) medical care, health advocacy education, and environmental remediation outside the hospital. Children should **never** be discharged from the hospital **until they can go to a lead-free environment** (CDC, 1985; Piomelli et al., 1984). Lead-free safe housing (with friends, relatives, or in designated transitional housing), in which a treated child can live during the entire abatement process through the post-abatement clean-up, must be arranged. With appropriately carried-out public health measures, complete and safe abatement should be achieved during the treatment period (CDC, 1985).

Once a child is discharged to a safe environment, frequent followup is mandatory. In general, depending on the initial blood lead value, most children who require chelation therapy must be followed closely for at least one year or more. All children undergoing chelation treatment should be seen every other week for 6-8 weeks, then once a month for 4-6 months. A child treated with BAL and $CaNa_2EDTA$ should be followed more closely: weekly for 4 to 6 weeks, then monthly for 12 months.

At each clinic visit, housing information should be updated. If history suggests that exposure is increasing or if blood lead levels are rising, the dwelling must be reinspected to evaluate the possibility of new sources of environmental lead, inadequate abatement, or unsound structures in buildings (for example, poor plumbing with leaks) that cause further chipping or breakdown of a previously repaired dwelling (Piomelli et al., 1984).

RESEARCH AREAS AND FUTURE TRENDS IN THE MANAGEMENT OF CHILDHOOD LEAD POISONING

Further evaluation is needed on

Xray fluorescence (XRF) measurements of lead in bone.
Efficacy of chelating agents in reducing the adverse neurobehavioral effects of lead.
Uses of succimer.
Toxicity of $CaNa_2EDTA$ and other chelating agents.

Bone Lead Measurements Using Xray Fluorescence (XRF)

According to published data, L-line and the K-line XRF techniques permit non-invasive assessment of skeletal lead stores. These bone stores reflect the lead burden accumulated over an individual's life. In contrast, blood lead values reflect recent lead exposure and absorption during the past 1 to 3 months and provide limited information about lead toxicokinetics over time (Rabinowitz et al., 1977). Evaluations using the L-line methodology in children have shown that blood lead levels underestimate the body burden of lead in lead-poisoned children (Rosen et al., in press); and sequential measurements of lead in lead-poisoned children by the L-line technique have shown decreases in bone lead after $CaNa_2EDTA$ treatment or environmental intervention (Rosen et al., in press). K-line techniques have been used mainly to measure bone lead levels in workers. Quantitation of bone lead content of children takes about 16 minutes.

At present, XRF equipment is available only in a few centers in the United States and Europe.

Efficacy of Chelating Agents

The benefits of chelation therapy in symptomatic lead-poisoned children are well known (Chisolm, 1968). Prompt intervention with chelating agents prevents progression to symptomatic disease and normalizes biochemical indices of lead toxicity. However, the efficacy of chelating agents in reversing or modifying the adverse neurobehavioral effects at all blood lead

levels in apparently asymptomatic children needs to be carefully assessed. Better understanding of this issue is critical in deciding the end-point of medical treatment. It is also essential in defining when chelation should be used.

Succimer

Data are needed on the tissue sites of lead chelated by succimer, the adverse effects of succimer, the effect of succimer on absorption of lead from the gastrointestinal tract, and the effectiveness of different dose regimens of succimer. Assuming that no new significant adverse effects are noted after succimer is used more widely, the efficacy and appropriate use of succimer for treating lead- poisoned children with blood lead levels below 45 μg/dL needs to be established.

Toxicity of $CaNa_2EDTA$

Results of one animal study suggest that $CaNa_2EDTA$ may transiently increase brain lead levels (Cory-Slechta et al., 1987). The redistribution of lead during chelation needs further study.

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Chapter 8. Management of Lead Hazards In The Environment of The Individual Child

Summary

To eradicate childhood lead poisoning, lead hazards must be abated.

Environmental case management includes a number of actions prescribed for a child with lead poisoning.

Precautions must be taken to ensure that abatement is conducted in the safest and most effective manner possible.

Eradicating childhood lead poisoning requires a long-term active program of primary lead-poisoning prevention, including abatement of lead-based paint hazards in homes, day-care centers, and other places where young children play and live. For the child who is lead poisoned, however, efficient and effective interventions are needed as quickly as possible. Abatement means making the source of lead inaccessible to the child.

Lead-based paint is the most common source of high-dose lead poisoning. Complete abatement of lead-based paint means eliminating all lead-based paint in a housing unit as a source of lead for the child, either by removing the paint or by using permanent barriers. Complete abatement of the lead hazards in the child's environment is the most effective and only certain way to prevent further damage. Complete abatement is expensive, but once a dwelling is abated, many generations of children may live in that home and reap the benefits. Unfortunately, complete abatement may not always be possible, and shorter term, preventive maintenance procedures may have to be undertaken to minimize the potential for further damage.

Lead-based paint is rarely completely abated in many of the largest childhood lead poisoning prevention programs. Instead, various degrees of incomplete abatement—designed to eliminate the worst hazards and prevent near-term exposures—are conducted. Development of cost-effective, safe, simple, and widely applicable methods of complete paint abatement is a high priority.

Whether complete abatement or preventive maintenance is done, persons performing the work should be knowledgeable of the hazards of lead to themselves, to children, and to the environment. They should be trained in the proper procedures for abatement and preventive maintenance, since improperly performed work can actually increase the hazards to the child.

Each situation in which a child gets poisoned is unique and must be evaluated by a person or team of persons skilled and knowledgeable about lead poisoning, hazard identification, and interventions to reduce lead exposure, including abatement of lead-based paint in housing. Childhood lead poisoning prevention programs need to work closely with other relevant agencies (for example, housing and environmental agencies) to ensure that the quickest and most effective approach is taken to remediating the environments of poisoned children.

The 1985 CDC statement on *Preventing Lead Poisoning in Young Children* set the level for environmental intervention at 25 μg/dL. In this new statement CDC recommends environmental intervention for children with blood lead levels of ≥20 μg/dL, or of ≥15 μg/dL that persist. Where resources are limited, however, individual environmental intervention must first focus on those children with the highest blood lead levels. CDC also recommends that environmental interventions be directed at primary prevention of lead poisoning in communities with a large number or percentage of children with blood lead levels ≥10 μg/dL (Chapter 9).

> When resources are limited, environmental intervention must first focus on those children with the highest blood lead levels. When possible, abatement should be conducted for primary prevention of lead poisoning.

The Department of Housing and Urban Development has issued *Lead-Based Paint Interim Guidelines for Hazard Identification and Abatement in Public and Indian Housing*, hereafter called the HUD Guidelines (HUD, 1990, also published in the Federal Register 55FR14556). (The worker protection guidance was subsequently revised and published in the Federal Register, 55FR39873.) This document is referenced frequently in this chapter because it contains the most comprehensive information on identifying and abating lead-based paint hazards available. It is not expected that every childhood lead poisoning prevention program or every homeowner will follow the guidelines completely. These guidelines were written for lead hazards in public and Indian housing, particularly for use during comprehensive modernization programs. Such programs, carried out when the property is vacant and in multiple units at one time, offer opportunities for very thorough and complete abatements. Most abatement of lead-based paint in the private sector does not occur in such a context. In the private sector, abatement is generally done in occupied housing scattered throughout an area, often with limited resources. In the context of this chapter, the HUD guidelines are an information source on identifying and abating hazards.

ENVIRONMENTAL CASE MANAGEMENT

> **Environmental case management includes**
>
> Educating parents about the sources, effects, and prevention of lead poisoning.
>
> Investigating the environment to identify lead sources and effectively communicating the results of this investigation.
>
> Taking emergency measures to reduce lead exposure.
>
> Doing long-term interventions to reduce lead exposure.
>
> Evaluating the efficacy of the interventions.

Environmental case management includes a number of actions prescribed for a child with lead poisoning. Ideally, environmental case management should be conducted by a team of professionals in public health, environmental activities, medical management, and social

management. A team approach to intervention will help ensure that followup is timely and effective. The management team may need to solve many related problems, such as whether to investigate supplemental addresses, where to find temporary alternative housing, and how to use community resources to assist the family in dealing with the lead-poisoned child.

A team approach to case management is most effective when all team members:

1. Demonstrate professionalism.
2. Show genuine concern for the poisoned child and family.
3. Support other team members.
4. Use similar terms, descriptions, and reference points to communicate with the child's family.
5. Meet specific time frames for followup.
6. Reinforce education of the family at every encounter.

Time Frames for Investigations and Interventions

The following guidelines describe the maximum time within which environmental interventions should be implemented. All children with blood lead levels ≥20 μg/dL should have environmental interventions conducted as quickly as possible. Children with blood lead levels ≥45 μg/dL require prompt chelation therapy. **The homes of these children must be remediated before they are allowed to return.**

Blood lead levels ≥70 μg/dL. Children with blood lead levels above 69 μg/dL constitute a medical emergency and must be hospitalized immediately. They are at highest risk for severe, permanent neurologic damage due to lead exposure and must be given highest priority for followup. Environmental investigation and intervention should be started within 24-48 hours and should include the child's home and potential sites of exposure, such as a relative's home or a day-care center. The homes of these children must be remediated before they are allowed to return.

Blood lead levels between 45 and 69 μg/dL. These children can be given a slightly lower intervention priority than the children classified as medical emergencies. Environmental investigation and intervention should begin within 5 working days and should include the same components as for children with higher blood lead levels. The homes of these children must be remediated before they are allowed to return.

Blood lead levels between 20 and 44 μg/dL. Environmental investigation and intervention should begin within 10 working days. Since many of these children will not be hospitalized and since allowing exposures to continue might lead to further increases in blood lead levels, environmental interventions for these children should be conducted as quickly as possible.

Blood lead levels between 15 and 19 μg/dL. Environmental investigation and intervention for children at this level should be based upon program resources and the ability of program staff to respond. At a minimum, these children and their families should have education regarding lead poisoning. If blood lead levels ≥15 μg/dL persist, environmental intervention should be made where possible—including assisting the parents in locating potential sources of lead contamination in and around the home and instructing them about how to reduce the risk of lead contamination. If resources permit, a full environmental inspection for lead-based paint should be done for such children.

Although full environmental investigation and abatement is not recommended as part of the management of children with blood lead levels below 15 μg/dL, the identification and reduction of lead hazards in all high-risk housing is an important primary prevention measure (Chapter 9).

Educating Parents about Lead Poisoning

The parents of all lead-poisoned children should be educated about lead poisoning. In communities with a high incidence of lead poisoning, communitywide educational efforts should be considered. These efforts should provide information similar to that in the anticipatory guidance provided by pediatric health care providers. Information provided should include:

1. Causes and effects of lead poisoning.
2. Relationship of the child's blood lead level to the potential for adverse health effects.
3. Need for followup blood lead testing of the child.
4. The child's possible sources of lead intake and practical means for reducing and eliminating these sources.
5. Role of nutrition in decreasing lead absorption.
6. Resources where parents can get further information (addresses and telephone numbers of local health-care providers or public health agencies).

Ideally, this information should be provided during a face-to-face meeting with the parents. When local resources are limited, however, written material (in an appropriate language) may be mailed to the child's family. Educating parents about lead poisoning is further discussed in Chapter 4.

Investigating the Environment and Communicating the Results

The technical aspects of inspecting a home for lead-based paint are discussed below. In general, an investigation of the environment of a lead poisoned child should include the following steps:

1. Determine the most likely sources of high-dose exposure to lead.
2. Investigate the child's home to identify possible sources of lead. Include both the interior and exterior environment and give special attention to painted surfaces, dust, soil, and water. (Details on how to test for lead-based paint are in the next section.)
3. Advise parents and caretakers about identified and potential sources of lead and ways to reduce exposure.
4. In cases in which the parent does not own the home, notify the property owner immediately that a child residing on the property has lead poisoning. Discuss the results of the environmental investigation and the abatement interventions required with the property owner. Emphasize the importance of prompt abatement. When a child with a medical emergency from lead poisoning is identified, an immediate, face-to-face meeting with the property owner may best demonstrate the need for emergency intervention.
5. Advise parents and property owners that no residents or personal belongings should remain in the home during abatement.

6. Monitor the effectiveness and timeliness of abatement procedures closely.
7. Coordinate environmental activities with those of other professionals, including the health-care providers and persons responsible for public health and social management. A team approach to intervention will help provide a timely and effective followup.

Emergency Measures to Reduce Lead Exposure

The first phase of environmental intervention may be to use short-term emergency interventions to **temporarily** reduce lead hazards. As soon as a blood lead level ≥20 μg/dL (or, if resources permit, ≥15 μg/dL) is confirmed, parents should be advised of the hazards of lead-based paint and lead dust. They should be told not to attempt abatement themselves—improper abatement will most likely **increase** lead dust levels in the home and create additional, more severe exposure for the child. The temporary nature of interventions other than abatement should be emphasized.

When the source of lead is paint and paint-contaminated dust, parents can be instructed to stabilize the paint, wet-mop all floors, and wet-clean window sills and window wells at least twice per week. Cleaners high in phosphates appear to work particularly well. Sponges and rags used in this cleaning should be used for no other purpose. In particular, they should not be used to wash dishes or clean eating- or food-preparation surfaces, since dangerous contamination could result. Children's hands should be washed regularly, particularly before eating. Toys and pacifiers that are mouthed should be washed at least daily. Cribs and playpens should be moved away from chipping or peeling paint; furniture can be placed in front of areas that are not intact to make them less accessible. Dry sweeping of dust should be avoided, because it will stir up and spread the dust. Other measures to reduce lead exposure are discussed in Chapter 4.

Long-Term Measures to Reduce Lead Exposure

The next phase of environmental intervention involves long-term hazard reduction. If the source of lead is paint and paint-contaminated dust, the lead hazards are permanently abated only when all lead-based paint is completely removed or otherwise made permanently inaccessible. Less extensive practices, which are commonly used by childhood lead poisoning prevention programs, may be called "long term abatement." Certain maintenance procedures (for example, frequent cleaning and keeping walls freshly painted) may be classified as "preventive maintenance," but in general these procedures offer no absolute assurance of safety. In cases other than "permanent abatement," how long the hazard will remain under control depends on such factors as the quality of the workmanship, the thoroughness of the procedure, the soundness of the underlying structure, and the condition of the plumbing and roof. Moisture from leaky pipes or roofs can quickly cause paint that was smooth and intact to blister and scale, generating hazardous levels of lead dust. Except in unusual situations (such as in the case of housing that is not likely to be viable for more than a couple of years or when no alternative housing is available), temporary measures to reduce exposure should not be a substitute for abatement or an excuse for delaying abatement.

Technical aspects of lead-based paint abatement are discussed below.

Evaluating Intervention Activities

The effectiveness of any intervention for a lead-poisoned child should be evaluated by its impact on the child's blood lead level. Measurement of environmental lead levels may also be helpful.

Assessing the Lead Problem in the Child's Community

If a number of children are identified as being lead-poisoned in a community, communitywide interventions as described in Chapter 9 should be considered.

TESTING FOR AND ABATING LEAD-BASED PAINT

> Tests for measuring the lead content of paint on walls have limitations; new tests for evaluating lead in paint are being developed.
>
> Proper abatement must be done by experts; untrained parents, property owners, workers or contractors should not attempt it.

NOTE: Remodeling or repainting homes with lead-based paint should be considered just as hazardous as abatement. Whenever lead-based paint must be disturbed by sanding, scraping, heating, or other forms of abrasion, the same precautions should be taken for remodeling or repainting as for abatement itself.

Inspection and Testing

Several methods are available for determining the lead content of paint. These include XRF, wet chemical methods, and chemical spot tests. Although XRF analyzers are convenient, instruments available at the present time have limitations. A study by the National Institute of Standards and Technology (NIST, 1989) indicated possible substrate errors in the direct-reading XRF's of as much as ± 2 mg/cm^2. These errors were caused by differences in base materials in walls and trim. (At very high readings, for example, above 3 mg/cm^2, this error has no practical significance). The spectrum analyzer, while considerably more expensive than the direct reader, provided much more accurate results. Only fully trained and experienced personnel should use XRF analyzers.

Wet chemical methods of analysis must be used if an XRF machine is not available or if it produces ambiguous results. Wet chemical methods require that a paint chip sample with all layers of paint on the surface be sent to a laboratory for analysis. Wet chemical analysis has two major disadvantages—results are not available immediately, and it is expensive.

Like XRF, chemical spot tests are performed on-site. A scratch is made through all layers of paint, and a chemical is placed on the scratch. If the scratch turns certain colors, further evaluation is needed. Chemical spot tests are qualitative, not quantitative, and the interpretation of the results is subjective. These tests are being refined and evaluated as to their safety, accuracy, and reliability.

Further information on proper testing procedures for lead-based paint is in the NIST study report and the HUD Guidelines.

The 1985 CDC statement on lead recommended an XRF value of 0.7 mg/cm^2 as the maximum level of lead in paint in a residence. The HUD standard, mandated by Congress, is 1.0 mg/cm^2. Several states have established their own XRF standards for lead in paint; these standards range from 0.7 mg/cm^2 to 1.2 mg/cm^2. The HUD document and some state regulations use a standard of 0.5% lead by weight for laboratory analysis.

Lead in paint should always be considered a "potential" hazard. An **immediate** lead hazard exists when lead-based paint is 1) chipping, peeling or flaking; 2) is chalking, thereby producing lead dust; 3) is on a part of a window which is abraded through the opening and closing of the window; 4) is on any surface which is walked on (like floors) or otherwise abraded; 5) can be mouthed by a child (for example, window sills); or 6) is disturbed by repainting or remodeling. A potential lead hazard can easily become an immediate hazard through natural aging, plumbing or roof leaks, or the paint being disturbed. All lead-based paint exceeding the action level should, therefore, be abated whenever possible. Otherwise, complicated records must be kept of unabated surfaces, and those surfaces must be inspected frequently to make certain that they have not become immediate hazards.

When inspecting for lead-based paint hazards, care must be taken to evaluate **all** types of surfaces, including walls, ceilings, doors and windows, trim and jambs, woodwork, stairway components, porch components, garages, sheds, fences, play equipment, and any other structures on the premises. Because of legal requirements in some areas, it may be necessary to test every surface that may be painted with lead paint (that is, every window, every door, every piece of trim, etc.). Often, however, abatement decisions can be made without this costly and time-consuming approach. Even with an XRF, a full inspection of all surfaces in an average home may take 4 hours or more. Sometimes, extrapolating XRF results to untested surfaces may make sense. Such extrapolation, however, should only be used for positive results. For example, if test results for one window are positive for lead, it is safe to assume that all similar windows are painted with lead-based paint; if test results for one window are negative, it is **not** safe to assume that no windows have lead-based paint.

Recent studies have indicated that many children are poisoned by lead-contaminated dust ingested through normal hand-to-mouth activity. This dust can come from lead contaminated soil that is tracked into the home on shoes or from the clothes of a parent who works with lead. However, the most common source of lead dust in the average old house is lead-based paint. Some believe that the level of lead dust in a house can be used as a measure of the severity of the immediate hazard.

Abatement

Proper abatement includes the following steps:

1. Proper training of all workers involved in the abatement.
2. Protecting those workers whenever they are in the abatement area.
3. Containing lead-bearing dust and debris.
4. Replacing, encapsulating, or removing lead-based paint.
5. Cleaning the abatement area thoroughly.
6. Disposing of abatement debris properly.
7. Inspecting to make certain the property is ready for reoccupancy.

Abatement should never be attempted by untrained parents, property owners, or contractors. The property owner's responsibility is not met until all the above steps have been completed.

Preparation: Just prior to abatement, all personal belongings, movable furniture, and drapes should be removed from the abatement area. In homes with deteriorated lead-based paint, furniture may be highly contaminated with lead dust. It is recommended that badly soiled carpets and drapes be discarded because of the difficulty of removing lead from them. Furniture should be cleaned before it is returned to the abated dwelling or it should be replaced. Wood, metal, glass and plastic surfaces should be washed with a high phosphate detergent. If possible, all upholstered furniture, carpets, drapes, and bare surfaces should be vacuumed with a High Efficiency Particle Accumulator (HEPA).

Precautions: Residents and their belongings should remain out of their homes during abatement. Under no circumstances should children and pregnant women be allowed to enter the dwelling unit during the abatement because abatement can generate large quantities of hazardous lead dust.

Training: All workers involved in a lead abatement project should be properly trained in the following: health effects of lead; proper procedures for worker protection, including procedures for personal hygiene and for wearing and caring for respirators; containment of an abatement project; various methods for abating lead-based paint and the safety and environmental hazards involved with each; and procedures for transporting and disposing of abatement debris properly.

Worker Protection: All workers on a lead abatement project and their families must be protected from the hazardous lead dust that will be generated. The minimum acceptable protection would be coveralls (preferably disposable); shoe coverings; hair covering; gloves; goggles; and a properly fitted, negative-pressure, half-mask respirator with a HEPA filter. Other, more protective respirators may be needed to protect from hazards such as organic vapors. If the abatement methods used would generate significant quantities of lead dust or organic vapors, workers must wear more protective respirators, such as supplied air-respirators.

The potential hazard to workers of lead dust **ingestion** is as significant, if not more significant, than inhalation. Workers must not eat, drink, or smoke on the job; and hands and face must be washed before breaks and at the end of the day. On-sight showers should, if possible, be provided. If on-site showers are not available, workers must shower and wash their hair immediately upon returning home. They must be careful not to carry hazardous levels of lead dust home on their bodies, shoes, or clothing. Therefore, work clothes should not be worn home; either workers should wear protective workclothes instead of street clothes at the worksite or they should wear protective garments over their street clothes. Work clothes should be disposed of or laundered by the employer to prevent the contamination of automobiles, homes, etc. with dust; lead-contaminated clothing should be handled with care and should not be laundered with other clothing of the worker or his family.

Note: The chapter in the HUD guidelines on worker protection was revised and published separately in the Federal Register on September 28, 1990 (55FR39873).

Containment: The work area should be contained with plastic (6 mil) to protect other living areas, yards, heating and ventilation systems, etc. from contamination. All nonmovable furnishings, such as counters, cabinets, and radiators should be covered with plastic. All floors should also be covered with plastic to prevent lead dust from being deposited in cracks and crevices and from being ground into the surface during the abatement.

Abatement: Abatement methods fall into three categories: 1) replacement, 2) encapsulation or enclosure, and 3) paint removal. These categories are discussed in more detail as follows:

Replacement: Removing the building component (such as a window, door, or baseboard) and replacing it with a new one.

Encapsulation: Covering a lead-painted surface with a material that will effectively prevent access to the lead-based paint and that will also prevent lead-bearing dust from that surface from entering the living environment.

Paint Removal: Stripping paint by heat, chemical, or mechanical means. This can be done either on-site or at the premises of a chemical stripping firm.

Certain methods of removing lead-based paint may be particularly hazardous to both the worker and the building occupants and may be banned in some areas. They are—

1. Removing paint with an open-flame torch or other heating device that operates at temperatures likely to volatilize lead (the melting point of lead is 621 °F).
2. Machine sanding surfaces with lead-based paint.
3. Sand blasting lead-based paint, except when the equipment is fitted with a vacuum device that prevents the dispersal of the debris.
4. Uncontained hydro-blasting.
5. Using chemical strippers containing methylene chloride. Methylene chloride is extremely toxic and protecting workers from exposure to this chemical is difficult.

If possible, all surfaces painted with lead-based paint should be abated by replacement, encapsulation, or paint removal. Ordinary paint is never an appropriate encapsulant; it is only part of a temporary maintenance procedure. Encapsulation materials should be durable and, where possible, affixed with both fasteners and adhesive. Paintlike coatings should be used with caution. Only coatings and adhesives that are proven to be safe and effective should be used. Any material that will eventually chip, peel, or flake upon aging or from water damage is not appropriate.

Paint removal is potentially the most hazardous abatement method because considerable amounts of lead dust and lead residue are generated. Paint removal from porous surfaces, such as wood or concrete, **always** leaves significant amounts of lead residue. This residue may not be visible and removing it requires extremely vigorous cleaning procedures (alternating washing with a high phosphate detergent and HEPA vacuuming (see below)). Painting over this residue can lead to lead dust problems when this paint begins to deteriorate or when it is abraded. Of particular concern are friction surfaces, such as window and door jambs.

Workers using any method that generates large volumes of dust or fumes should use caution. Such methods increase the difficulty of worker protection and the likelihood that hazardous levels of lead-bearing dust will remain in the dwelling unit or be deposited in the soil surrounding the home. Demolishing older structures with lead-based paint likewise can result in deposition of lead-bearing dust into the soil or on neighboring property, and dust suppression techniques should be used.

Clean-Up: All lead abatement activity is likely to generate quantities of hazardous lead dust. Unless this dust is properly cleaned, the dwelling unit will be more hazardous after abatement than it was before. This dust is difficult to remove. Daily clean-up, consisting of misting debris with water, carefully sweeping it, and placing it in double 4-mil or 6-mil plastic bags, is necessary to minimize the risk to workers of accumulated lead dust.

After abatement and before repainting, all surfaces in the dwelling must be thoroughly vacuumed with a HEPA vacuum; wet washed, preferably with a high phosphate detergent such as tri-sodium phosphate; and then vacuumed again. The property should be visually inspected before being repainted. The inspector should ascertain that all surfaces covered with lead-based paint have been abated and that no visible dust or debris remains on site.

Several states have adopted a post-abatement dust standard which has been included in the HUD Guidelines. This standard was set mainly on the basis of practicality rather than a health or risk assessment, and further research is needed on the adequacy and appropriateness of that standard. The standard allows the following maximum levels of lead in dust:

Floors	200 $\mu g/ft^2$
Window Sills	500 $\mu g/ft^2$
Window Wells	800 $\mu g/ft^2$

Inspectors and persons collecting dust samples and laboratories measuring dust lead levels should be thoroughly familiar with the recommended sampling and analysis protocols for dust in the HUD Guidelines.

After the inspection, abated surfaces should be repainted, if appropriate. Wooden floors should receive a coat of deck enamel or urethane, concrete floors should be sealed with deck enamel, and linoleum or tile floors should be waxed. Sealing the floors will bind any remaining dust particles and enable the occupants to clean those surfaces easily.

Disposal: Certain wastes from a lead-based paint abatement project, either liquid or solid, may be classified as hazardous. If so, they will have to be treated as such and handled by a licensed transporter or treatment firm. In any case, **all** debris from an abatement project, whether classified as hazardous or not, must be contained and transported in such a way as to prevent the dispersal of lead bearing dust, chips, or liquid into the environment. Lead debris should never be sent to a solid waste incinerator, a disposal method that disperses lead into the air.

References

HUD (Department of Housing and Urban Development). Comprehensive and workable plan for the abatement of lead-based paint in privately owned housing: report to Congress. Washington (DC): HUD, 1990.

NIST (National Institute for Standards and Technology). Methods for measuring lead concentrations in paint films. Washington (DC): NIST, 1989.

Chapter 9. Management of Lead Hazards In The Community

Community Level Intervention Includes

Screening and surveillance.

Risk assessment and integrated prevention planning.

Outreach and education.

Infrastructure development.

Hazard reduction.

In theory, primary prevention has always been the goal of childhood lead poisoning prevention programs. In practice, however, most programs focus exclusively on secondary prevention, dealing with children who have already been poisoned. As programs shift the emphasis to primary prevention, their efforts must be designed to systematically identify and remediate environmental sources of lead, including, most importantly, dwellings containing old lead paint.

The shift from case management to community-level intervention will require a fundamental shift in perspective. The focus must shift from the individual child to the population of children at risk and the environment in which they live. The purpose of community-level intervention is to identify and respond to sources, not cases, of lead poisoning. The responsibility for addressing lead poisoning will have to be expanded beyond health agencies to include a variety of housing, environmental, and social service agencies at the local, county, state, and national level.

To be successful, community-level intervention will involve at least five types of activities:

1. Screening and surveillance: Determining populations at risk and the locations of the worst exposures.
2. Risk assessment and integrated prevention planning: Analyzing all available data to assess sources of lead, exposure patterns, and high-risk populations and developing primary prevention plans.
3. Outreach and education: Informing health-care providers, parents, property owners, and other key people about lead poisoning prevention.
4. Infrastructure development: Finding the resources needed for a successful program of risk reduction.
5. Hazard reduction: Reducing the hazards of lead-based paint and lead in dust and soil, particularly in high-risk buildings and neighborhoods.

SURVEILLANCE

To identify the highest risks
Collect data on blood lead levels.
Conduct environmental surveys.
Collect demographic data.

For the most effective allocation of resources, data on the extent of the lead poisoning problems and the location of the worst lead hazards must be available for study. By combining data on blood lead levels, environmental sources of lead, and community demographics, public health agencies can identify and quantify the risk of lead poisoning in the community.

Data on Blood Lead Levels

Results of regular blood lead screening for pre-school children (as recommended in Chapter 6 of this report) will eventually provide an important source of information on the distribution of lead hazards in a community. Current data, which are based on limited public screening or the experience of practitioners or clinics, cannot provide the true prevalence of elevated blood lead levels in the children of a community. Communities may need to undertake additional, focused screening surveys to obtain data on the prevalence of elevated blood lead levels. Even after near-universal screening is in place, such targeted screening efforts will continue to be necessary in areas and populations in which substantial numbers of children do not have regular pediatric health-care providers. To be accurate, such surveys should use door-to-door (rather than fixed-site) sampling and blood lead (rather than EP) analysis.

Health officials can evaluate risks better if they have the results of all blood lead tests, not just the elevated blood lead levels. A convenient mechanism for gathering such information is for laboratories to report all blood lead testing results to an appropriate local or state health agency. Where mandatory reporting is not in place, health agencies should work with laboratories and pediatric health-care providers to obtain as much data as possible on blood lead test results.

Environmental Surveys

Environmental surveys that are designed to identify the common sources of childhood lead exposure can be undertaken in conjunction with or as a complement to community-based surveys of blood lead levels. Environmental surveys do not, however, replace measurement of children's blood lead levels. The environmental sources and pathways of lead that can be assessed in environmental surveys include lead-based paint, lead in dust and soil, lead in drinking water, lead from industrial sources and wastes, and lead from unusual sources such as folk medicines or ceramicware.

An environmental survey of the sources of lead around children's homes (paint, dust, and soil) can be undertaken in conjunction with a door-to-door blood lead screening program. A team would consist of a nurse or phlebotomist who would obtain the blood samples and an inspector who could use the most cost-effective combination of measurements of lead in dust,

soil, and paint (for example, XRF analyzers, chemical spot tests, or removal of paint chips for laboratory analysis). When screening for lead-based paint in housing, inspectors should obtain representative data on the prevalence of hazards and need not undertake the type of comprehensive inspections described in Chapter 8. Protocols for environmental sampling must be developed, and inspectors must be trained in sampling techniques before the survey program begins.

In addition to looking for lead hazards in housing, a comprehensive environmental lead testing program could look for other lead sources, including drinking water in schools and residential buildings, soil in playgrounds and schoolyards, street dusts, and lead-based paint in nonresidential buildings such as day-care centers and schools. In some cases, environmental data obtained for other purposes may be useful. For example, the federal Safe Drinking Water Act and Lead Contamination Control Act requires some testing for lead in drinking water, so health officials could, therefore, contact water suppliers and school officials to obtain test results. Agricultural extension services may have data on lead levels in soil.

Demographic Data

Health surveys, such as the National Health and Nutrition Examination Survey (NHANES), have correlated children's blood lead levels with demographic factors such as family income and place of residence (for example, center city vs. suburbs). Demographic data now becoming available from the 1990 census can be used to broadly identify high-risk areas. Variables to consider include the age of housing (pre-1960 housing has the most lead), income levels, socioeconomic status, ethnicity, and the number or density of preschool children in the area. For best results, communities would use this demographic information to predict where the greatest lead hazards might be located and then to conduct appropriate blood lead or environmental surveys to see if the predictions are true. Once the most predictive demographic variables have been identified, algorithms or survey instruments could be designed to accurately predict which areas pose the greatest risk on the basis of demographic data alone.

RISK ASSESSMENT AND INTEGRATED PREVENTION PLANNING

- Risk assessment involves using all available data to evaluate community lead hazards.
- Primary prevention planning should include representatives from the private and public sectors.
- A primary prevention plan should include outreach and education programs, infrastructure development, and hazard reduction.

Public health officials should use all of the information at their disposal—blood lead screening results, environmental survey data, and demographic information—to create the most accurate picture of community lead hazards, including sources of lead, exposure patterns, and high-risk populations. Whenever possible, officials should focus on specific sources and the smallest pertinent geographic area of concern. In some new suburban communities, for example, the risks may not justify a communitywide program to abate lead-based paint in

housing. Nevertheless, there may be a need to address specific sources (for example, drinking water in new houses with lead solder) or specific neighborhoods (for example, an old part of town where Victorian homes are being rehabilitated).

Because lead poisoning is completely preventable, public health officials should assess the success of current prevention efforts. Local communities should focus on how well the hazards of lead are being addressed in that community, rather than on whether the community has a bigger or smaller lead problem than other communities.

Once a decision is made to address at least some aspects of the lead problem in a community, public health officials should develop an integrated primary prevention plan. The plan should be assembled with input from other agencies (including housing and environmental agencies), pediatric health-care providers, parents, teachers, community groups, and other interested persons. The plan should identify which sources, geographic areas, or high-risk populations are to be addressed. Each element of the plan should include a description of who will have the primary responsibility for implementation, where financial and other resources will be obtained, and a time schedule for implementation. Plans should be as specific as possible in order to allow public officials and community groups to periodically assess whether and how the plan is being carried out.

The remaining sections of this chapter address in more detail three types of activities that should be addressed in any comprehensive primary prevention plan: outreach and education, infrastructure development, and hazard abatement.

OUTREACH AND EDUCATION

Must take place during every phase of the community activity.
Should involve many agencies and both the public and private sectors.
Should involve many people in various professions, including those related to real estate.

Outreach and education must take place during every phase of the community activity, beginning before health and environmental screening and ending when risk abatement is complete. Among the most important targets for outreach and educational programs are local officials, health-care providers, parents, property owners, day-care providers, and early childhood educators. The outreach programs can be carried out through pamphlets and other written materials, local news media, public meetings, school programs, and social service agencies.

Local health officials who have traditionally carried out all or most lead poisoning prevention activities in a community must begin by reaching out to other agencies that will have a role in communitywide primary prevention efforts. When possible, lead poisoning prevention should be part of an integrated program for creating safe and affordable housing or for providing poor people in the community with the full range of needed social services. Local, state, and federal agencies dealing with health, housing, environmental, and children's issues should be contacted.

Many health-care providers are unaware of the most recent developments in the field of lead poisoning prevention. Educational campaigns by local officials, licensing agencies, professional

associations, clinics, and hospitals are needed to ensure that pediatric health-care providers understand current thinking about the health and environmental aspects of lead poisoning. Outreach through pamphlets, grand rounds, and continuing education programs should be targeted to pediatricians, family practitioners, pediatric and community health nurses, obstetricians, and midwives.

For parents, including pregnant women, initial education should focus on the hazards of lead and the need for blood lead testing of children at regular intervals. Parents should know about risk factors that warrant frequent screening (Page 42). Educational materials should help parents understand the implications of the screening results. Finally, parents (and parents-to-be) should be informed about simple steps that can be taken to reduce risks, such as proper nutrition and housekeeping measures (Chapter 4). Such outreach efforts can be targeted to individual parents and to groups of parents and prospective parents.

Property owners and managers, realtors, and other real estate professionals need to learn how to maintain property in a safe and habitable condition. Banks, mortgage companies, and insurance companies could play an important role in conveying this information at critical junctures, such as when a property owner is buying a property or seeking financing for major renovations. In addition, property owners should be given written material that explains how to remove lead safely.

Day-care providers and early childhood educators should be given information about lead poisoning and its sequelae. Those taking care of young children should also be informed about the need to identify and abate lead hazards in day-care buildings and schools. Parents of lead-poisoned children can aid in this process by informing their child's teachers about the past lead poisoning, so that the teacher can make better informed decisions about the need for remedial measures.

INFRASTRUCTURE DEVELOPMENT

Infrastructure development includes

Regulations and rules on removing lead.

Trained inspection and abatement contractors.

Temporary housing for families whose homes are undergoing abatement.

Financial resources for lead poisoning prevention activities, including abatement.

Before a community can launch a broad-based program of preventive deleading and hazard reduction, many elements must be in place to support such activities.

First, regulations or other rules and standards are needed to define when and how inspections and deleading are to occur. One local agency (housing, environmental, or health) should be designated as the lead agency with respect to community intervention activities and a system should be put in place for coordinating regulatory and other activities among all involved agencies.

A second requirement is contractors who are trained 1) to identify lead hazards, including lead-based paint, and 2) to remove lead-based paint safely. Besides inspectors, abatement planners, contractors, supervisors, and workers are needed. Optimally, such persons should be licensed or certified by a federal or state agency to ensure that their work is of high quality.

A third infrastructure need is temporary housing for families during the deleading process. Because lead-based paint should not be removed while homes and apartments are occupied, communities must develop strategies to provide temporary alternative housing for families that need it. Communities should consider developing "safe houses" where families can live temporarily at little or no cost while their homes are being deleaded. If families are encouraged to "double up" with friends, measures should be in place to ensure that the home or apartment being used for temporary housing is free of lead hazards.

The final element of infrastructure involves financial resources for both the government agencies overseeing lead poisoning prevention programs and property owners or tenants seeking to delead. This may be the most difficult element, yet it is critical to a successful program. Existing federal and state housing funds (for example, Community Development Block Grants) can be used to finance lead removal if communities so choose. Starting in Fiscal Year 1992, a limited number of loans for abatement may be available from the Department of Housing and Urban Development through the HOME program.

HAZARD ABATEMENT

- Hazard abatement may involve a number of activities directed at multiple environmental sources and pathways.
- Abatement resources should be targeted to the highest risk neighborhoods and homes.
- The goal of hazard abatement is the systematic elimination of lead hazards in the community.

The final and most important step is actually abating the lead hazards. This may involve many activities, such as corrosion control to reduce the amount of lead in drinking water and covering or removing lead-contaminated soil in parks and playgrounds. In many cases, the primary risk will be lead-based paint and the primary form of risk reduction will be preventive deleading—abatement that occurs before children have been poisoned. Before the hazard abatement phase, the community must decide which lead hazards to target. Information gathered during risk assessment should be used to ensure that abatement resources are directed toward the highest risk neighborhoods and buildings.

Local officials have a variety of means at their disposal to promote preventive deleading—from education and outreach, programs designed to increase voluntary deleading, financial assistance to encourage deleading, and regulatory mechanisms to require deleading. If voluntary efforts are to be encouraged, outreach must go beyond general information to provide building owners with specific information about how to survey a building for lead hazards and how to abate those hazards.

If abatement is mandated by law, the law should require safe and effective abatements. Rental property owners should not be permitted to avoid abating their properties by evicting or refusing to rent to families with young children.

Whatever mechanisms are used, the goal of hazard abatement must be to systematically eradicate the lead hazards in the community. Such a program will protect not only lead-poisoned children but all children—and thus safeguard the community's future.

Appendix I.
Capillary Sampling Protocol

Microspecimens of blood collected by fingerstick are widely used to measure lead levels, yet there is no consensus on what constitutes the best collection procedure. Published data on collection methods are scant, and much of the data that do exist were published 10 or more years ago, when technology was not as advanced and blood lead levels of concern were significantly higher.

The high potential for lead contamination of capillary specimens during collection is well known (CDC, 1985; DeSilva and Donnan, 1980; Mitchell et al., 1974), and the special steps used to minimize the likelihood of contamination constitute the major differences among collection procedures. Special procedures used for minimizing contamination include thorough scrubbing of the hand and finger with soap and then alcohol (Sinclair and Dohnt, 1984; NECCLPP, 1985); using dilute nitric acid (Rosen, 1972; MHD, 1988); or using silicone or a similar barrier spray (Lyngbye et al., 1990; CDHS, 1990; NYSDH, 1989; Mitchell et al., 1974).

Several types of containers for collecting children's blood (maximum volume $\leq 500\ \mu L$) have been introduced in recent years and are widely used by screening programs. The new containers are better than glass tubes, since glass capillary tubes are very fragile. Whether these new containers are suitable for collecting blood for lead measurement has not been extensively studied.

More research on these and other issues is clearly needed before the best fingerstick collection procedures can be identified. Recognizing these constraints, a fingerstick procedure for collecting blood lead specimens follows.

A. NEEDED MATERIALS

1. Soap.
2. Alcohol swabs. If a surgical or other disinfectant soap is used, alcohol swabs can be eliminated.
3. Sterile cotton balls or gauze pads.
4. Silicone spray or swabs. The benefits of using a barrier spray, which forms a layer between the skin and blood droplets, have been debated. In addition to doubts about the spray's effectiveness in reducing specimen contamination, the spray makes the collection more expensive and complex. Some evidence exists, however, the spray reduces contamination (NYSDH, 1989; Mitchell et al., 1974), so it is included in this procedure.
5. Examination gloves.
6. Lancets. The type of lancet used is largely a matter of personal preference, so long as sterility is guaranteed.
7. Collection containers. If glass capillary tubes are used, sealing clay or tube caps will also be required. No additional supplied are needed for most other microcontainers. The laboratory should be consulted to ensure than an appropriate size capillary tube is used.
8. Adhesive bandages.

9. Trash bags suitable for medical waste and containers for sharps. Bags containing medical waste should be clearly identified as such.
10. Storage or mailing containers if needed. If specimens require shipment, follow the Postal Service or other appropriate regulations for shipping body fluids.

Materials used in the collection procedure that could contaminate the specimen (for example, blood containers, alcohol swabs, and barrier sprays) must be lead-free. **Before selecting equipment for use in blood collection, consult with the laboratory about its requirements.** In many cases, the laboratory will recommend or supply suitable collection equipment and may precheck the equipment for lead contamination. Some instrument manufacturers also supply collection materials that are pretested for lead content.

B. PREPARING FOR BLOOD COLLECTION

All personnel who collect specimens should be well-trained in and thoroughly familiar with the collection procedure. The skill of the collector will greatly influence the specimen quality. All equipment should be within easy reach. The environment should be clean, secure, and as nonthreatening to the child as possible. Any necessary consent should be obtained before specimen collection begins, and the procedure should be explained to the child and the parent or guardian. Used materials should be discarded into appropriate waste containers suitable for medical waste immediately following use.

C. PREPARING THE FINGER FOR PUNCTURE

NOTE: Puncturing of the fingers of infants less than 1 year of age is not recommended. Puncturing of the heel is more suitable for these children (NCCLS, 1986).

Collection personnel should wear examination gloves whenever the potential for contact with blood exists. If the gloves are coated with powder, it should be rinsed off with tap water.

The child's hands should be thoroughly washed with soap and then dried with a clean, low lint towel. If water is unavailable, foam soaps can be used without water (D. Griffin, Louisville/Jefferson County Department of Health, personal communication). Plain, unprinted, nonrecycled towels are best (WSLH, 1985). If desired, a brush can be used for cleaning the finger; brushing during washing can increase blood circulation in the finger (CDHS, 1990). Once washed, the finger must not be allowed to come into contact with any surface, including the child's other fingers.

The finger to be punctured (often the middle finger) must be free of any visible infection or wound; it should be massaged to increase circulation before being punctured with the lancet. This can be accomplished during or after washing (NYSDH, 1989; CDHS, 1990).

Steps for Preparing the Child's Finger

1. Select examination gloves. If necessary, rinse them to remove powder.
2. Wash the child's hands thoroughly with soap and water, and then dry them with an appropriate towel.
3. Grasp the finger that has been selected for puncture between your thumb and index finger with the palm of the child's hand facing up.

4. If not done during washing (see preceding notes), massage the fleshy portion of the finger gently.
5. Clean the ball or pad of the finger to be punctured with the alcohol swab. Dry the fingertip using the sterile gauze or cotton ball.
6. Apply the silicone barrier. If a spray is used, shake the can vigorously to mix the contents. Direct the spray away from child and collector. Silicone does not dry, and the finger can be punctured immediately.

D. PUNCTURING OF THE FINGER AND FORMING DROPS OF BLOOD

1. Grasp the finger and quickly puncture it with a sterile lancet in a position slightly lateral of the center of the fingertip.
2. Wipe off the first droplet of blood with the sterile gauze or cotton ball.
3. If blood flow is inadequate, gently message the proximal portion of the finger and then press firmly on the distal joint of the finger. A well-beaded drop of blood should form at the puncture site.
4. Do not let the blood run down the finger or onto the fingernail.

After the finger is ready, the puncture and subsequent steps of forming a drop of blood and filling the collection container should be performed quickly and efficiently, since any delay can make collection more difficult (for example, the blood may clot or the child may resist). Several types of lancets are suitable for puncturing children's fingers. The range from small manual lancet blades to spring-loaded assemblies. Regardless of the lancet used, the puncture should be made swiftly and cleanly and should be deep enough to allow adequate flow.

The site of the puncture should be slightly lateral to the ball of the finger. This region is generally less calloused, which makes puncturing easier and, possibly less painful (CDHS, 1990). The first drop of blood contains tissue fluids that will produce inaccurate results; it should be removed with a sterile gauze or cotton ball (NYSDH, 1989; CDHS, 1990).

A barrier material such as silicone will help a distinct "bead" of blood to form, which aids collection. Blood that runs down the finger or around the fingernail is no longer suitable. Blood flows better if the punctured finger is kept lower than the heart. Inadequate blood flow can be improved by gently massaging the proximal portion of the finger in a distal direction, then pressing firmly at the distal joint of the punctured finger (restricting blood flow out of the fingertip) and *gently* squeezing the sides of the fingertip. Excessive squeezing will cause tissue fluid to be expressed, and the fluid will compromise specimen integrity (NYSDH, 1989; CDHS, 1990). Do not let the blood run down the finger or fingernail.

E. FILLING THE COLLECTION CONTAINER

1. Continuing to grasp the finger, touch the tip of the collection container to the beaded drop of blood.
2. Draw the blood into the container maintaining continuous flow of blood.
3. When full, cap or seal the container as appropriate.
4. Agitate the specimen to mix the anticoagulant through the blood.
5. Check that the container is properly labeled, and place it in an appropriate storage area.
6. Stop the bleeding and cover the finger with an adhesive bandage. Bleeding should stop very quickly. If bleeding is slow to stop, apply pressure to the puncture site with a sterile gauze or a cotton ball. If bleeding continues after 3 to 5 minutes of applying pressure, consult a physician.

The proper procedure for filling and capping collection containers is somewhat specific to the container used. As a general rule, contact between the skin and the container is to be avoided. To prevent clotting of the specimen, blood must be mixed with the anticoagulant after filling the container. Depending on the container and anticoagulant used, the agitation needed can range from gentle rocking to vigorous shaking. Some procedures call for the collection container to be rotated during filling so that anticoagulant will be distributed quickly through the sample (MDPH, 1990).

To facilitate blood flow, many procedures call for the collection container be held nearly horizontal, with a slight downward angle. Blood flow into the container should be uninterrupted to avoid air bubbles in the specimen. Except for glass capillary tubes, containers come with appropriate caps, and these should be applied immediately following collection. Specimens in glass capillary tubes are often collected in duplicate and then sealed with rubber caps or plasticine sealing clay or both. Again, consulting with the laboratory and knowing the manufacturer's recommendations are important to ensure specimen integrity and suitability for analysis.

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Appendix II. Summary For The Pediatric Health-Care Provider

The following material summarizes those parts of the lead statement that are most important for the pediatric health-care provider. It does not include some of the critical information on such topics as primary prevention, sources of lead in the environment, and abatement. More information on all of the topics described herein is included in the complete statement.

CHAPTERS 1 AND 2. INTRODUCTION AND BACKGROUND

Childhood lead poisoning is one of the most common pediatric health problems in the United States today, and it is entirely preventable. Enough is now known about the sources and pathways of lead exposure and about ways of preventing this exposure to begin the efforts to permanently eradicate this disease. The persistence of lead poisoning in the United States, in light of all that is known, presents a singular and direct challenge to public health authorities, clinicians, regulatory agencies, and society.

Previous lead statements issued by the Centers for Disease Control (CDC) have acknowledged the adverse effects of lead at lower and lower levels. In the most recent previous CDC lead statement, published in 1985, the threshold for action was set at a blood lead level of 25 μg/dL, although it was acknowledged that adverse effects occur below that level. In the past several years, however, the scientific evidence showing that some adverse effects occur at blood lead levels at least as low as 10 μg/dL in children has become so overwhelming and compelling that it must be a major force in determining how we approach childhood lead exposure.

Because 10 μg/dL is the lower level of the range at which effects are now identified, primary prevention activities—communitywide environmental interventions and nutritional and educational campaigns—should be directed at reducing children's blood lead levels at least to below 10 μg/dL. Blood lead levels between 10 and 14 μg/dL are in a border zone. While the overall goal is to reduce children's blood lead levels below 10 μg/dL, there are several reasons for not attempting to do interventions directed at individual children to lower blood lead levels of 10-14 μg/dL. First, laboratory measurements of blood lead levels may be variable, so a blood lead level in this range may, in fact, be below 10 μg/dL. Secondly, effective environmental and medical interventions for children with blood lead levels in this range have not yet been identified and evaluated. Finally, the sheer numbers of children in this range would preclude effective case management and would detract from the individualized followup required by children who have higher blood lead levels.

The single, all-purpose definition of childhood lead poisoning has been replaced with a multitier approach. Community prevention activities should be triggered by blood lead levels ≥10 μg/dL. Medical evaluation and environmental investigation and remediation should be done for all children with blood lead levels ≥20 μg/dL. All children with blood lead levels ≥15 μg/dL require individual followup, including nutritional and educational interventions. Furthermore, depending on the availability of resources environmental investigation and remediation should be done for children with blood lead levels of 15-19 μg/dL, if such levels persist. The highest priority should continue to be the children with the highest blood lead levels.

Other differences between the 1985 and 1991 statements are as follows:

Screening test of choice. Because the erythrocyte protoporphyrin level is not sensitive enough to identify children with elevated blood lead levels below about 25 μg/dL, the screening test of choice is now blood lead measurement.

Universal screening. Since virtually all children are at risk for lead poisoning, a phase in of universal screening is recommended, except in communities where large numbers or percentages of children have been screened and found not to have lead poisoning. The full implementation of this will require the ability to measure blood lead levels on capillary samples and the availability of cheaper and easier-to-use methods of blood lead measurement.

Primary prevention. Efforts need to be increasingly focused on preventing lead poisoning before it occurs. This will require communitywide environmental interventions, as well as educational and nutritional campaigns.

Succimer. In January, 1991, the U.S. Food and Drug Administration approved succimer, an oral chelating agent, for chelation of children with blood lead levels over 45 μg/dL.

CHAPTER 3. SOURCES AND PATHWAYS OF LEAD EXPOSURE

A child's environment is full of lead. Children are exposed to lead from different sources (such as paint, gasoline, and solder) and through different pathways (such as air, food, water, dust, and soil). Although all U.S. children are exposed to some lead from food, air, dust, and soil, some children are exposed to high dose sources of lead. Lead-based paint is the most widespread and dangerous high-dose source of lead exposure for preschool children.

Lead-based paint (containing up to 50% lead) was in widespread use through the 1940s. Although the use and manufacture of interior lead-based paint declined during the 1950s and thereafter, exterior lead-based paint and lesser amounts of interior lead-based paint continued to be available until the mid-1970s. (Lead-based paint produced after the 1940s tended to have much lower lead concentrations than lead-based paint produced earlier.)

Pica, the repeated ingestion of nonfood substances, has been implicated in cases of lead poisoning; however, a child does not have to eat paint chips to become poisoned. More commonly, children ingest dust and soil contaminated with lead from paint which flaked or chalked as it aged or which has been disturbed during home maintenance and renovation. This lead-contaminated house dust, ingested via normal repetitive hand-to-mouth activity, is now recognized as a major contributor to the total body burden of lead in children. Because of the critical role of dust as an exposure pathway, children living in sub-standard housing and in homes undergoing renovation are at particular risk for lead poisoning.

Many cases of childhood lead poisoning that result from renovation or remodelling of homes have been reported. Before older homes undergo any renovation that may generate dust, they should be tested for the presence of lead-based paint. If such paint is found, contractors experienced in working with lead-based paint should do the renovations.

Other potentially important sources and pathways of lead exposure include soil and dust, water, "take home" exposures from parental occupations and hobbies, water, and food. Very high-dose exposure may occasionally result from sources other than lead-based paint in specific situations.

CHAPTER 4. THE ROLE OF THE PEDIATRIC HEALTH-CARE PROVIDER

Pediatric health-care providers, working as part of the public health team, must play a critical role in the prevention and management of childhood lead poisoning. Their roles include 1) educating parents about key causes of childhood lead poisoning; 2) screening children and interpreting blood lead test results; 3) working with appropriate groups in the public and private sectors to make sure that poisoned children receive appropriate medical, environmental, and social service followup; and 4) coordinating with public health officials and others involved in lead-poisoning prevention activities.

Along with educating parents about nutrition and developmental stages, providers should discuss the potential hazards of lead. They should focus on the major likely preventable sources of high-dose lead poisoning in their communities. Parents should be told of the potential dangers of peeling lead-based paint, the potential hazards of renovating older homes, and the need for good work practices if their occupations or hobbies expose them to lead. In some communities parents should be warned about the potential for lead exposure from improperly fired ceramicware and imported pottery. In others, where water lead levels are a concern, parents could be advised to use only fully-flushed water (that is, water that has not been standing in pipes for a prolonged time) from the cold-water tap for drinking, cooking, or preparing infant formula.

Pediatric health-care providers should provide information about simple ways parents can reduce exposure to lead. Some examples of these are discussed below.

Housekeeping Interventions. Particularly in older homes, which may have been painted with lead-based paint, interventions to reduce exposure to dust may help reduce blood lead levels. These include:

- Make sure your child does not have access to peeling paint. Pay special attention to windows and window sills and wells.
- If the house was built before about 1960 and has hard surface floors, wet mop them at least once a week with a high phosphate solution (for example, 5-8% phosphates). (The phosphate content of automatic dishwashing detergents and other cleaning substances is often listed on the label and may be high enough for this purpose. Otherwise, trisodium phosphate can be purchased in hardware stores.) Other hard surfaces (such as window sills and baseboards) should also be wiped with a similar solution. Do not vacuum hard surface floors or window sills or wells, since this will disperse dust. Vacuum cleaners with agitators remove dust from rugs more effectively than vacuum cleaners with suction only.
- Wash your child's hands and face before he/she eats.
- Wash toys and pacifiers frequently.

Other Interventions to Reduce Exposure to Lead.

- If soil around the home is or is likely to be contaminated with lead (for example, if the home was built before 1960 or the house is near a major highway), plant grass or other ground cover. Since the highest concentrations of lead in a yard tend to be near surfaces that were once painted with lead paint, like exterior walls, if exterior lead paint was likely to be used, plant bushes around the outside of your house so your child cannot play there.

- In areas where the lead content of water exceeds the drinking water standard, use only fully-flushed water from the cold-water tap for drinking, cooking, and making formula. In communities where water conservation is a concern, use first-flush water for other purposes.
- Do not store food in open cans, particularly if the cans are imported.
- Do not use pottery or ceramicware that was improperly fired or is meant for decorative use for food storage or service.
- Make sure that take-home exposures are not occurring from parental occupations or hobbies (Chapter 3).

Not all aspects of a poisoned child's followup will be managed by the pediatric health-care provider, although the provider is an important part of the team. Through his or her interactions with the child and family and the responsible public health agency, the provider should make sure that any appropriate interventions are occurring. If the child needs a medical evaluation (for a blood lead level ≥20 μg/dL) or pharmacologic treatment (Chapter 7), either the provider should do it or should refer the child to a place that treats large numbers of poisoned children. The provider should make sure that the child receives an appropriate environmental investigation and remediation with the help of the public health agencies. Particularly if the child is developmentally delayed, the provider should refer the child to an appropriate infant stimulation or child development program. In many cases, lead-poisoned children and their families will also benefit from social services followup.

CHAPTER 5. THE ROLE OF STATE AND LOCAL PUBLIC AGENCIES

A variety of local, state and federal agencies play a role in preventing childhood lead poisoning. Pediatric health care providers and parents should know about what these agencies do so that they can use these resources effectively. In turn, these agencies must coordinate their activities to ensure that all aspects of childhood lead poisoning prevention—health, housing, and environment—are being addressed, and to provide the most comprehensive and cost-effective services to at-risk children, their parents, and their health-care providers.

CHAPTER 6. SCREENING

Traditionally, the main purpose of a childhood lead poisoning screening program has been to identify asymptomatic lead-poisoned children and to intervene as quickly as possible to reduce their blood lead levels. An additional benefit of screening programs is that abatement of lead sources for poisoned children results in prevention of lead poisoning for children who would have been exposed to those sources in the future. As the focus in lead poisoning prevention turns more to primary prevention, an additional benefit of screening is that data generated can be used in targeting interventions to places with children at high risk for lead poisoning.

In 1984, the last year for which estimates are available, it is believed that between 3 and 4 million children younger than age 6 years (17% of all U.S. children in this age group) had blood lead levels above 15 μg/dL. Furthermore, about 74% of occupied, privately owned housing built before 1980 contains lead-based paint (defined as ≥1 mg/cm^2). **Because almost all U.S.**

children are at risk for lead poisoning (although some children are at higher risk than others), our goal is that all children should be screened, unless it can be shown that the community in which these children live does not have a childhood lead poisoning problem. (Deciding that no problem exists requires that a large number or percentage of children be tested.) The full implementation of this will require the ability to measure blood lead levels on capillary samples and the availability of cheaper and easier-to-use methods of blood lead measurement. Children at highest risk for lead poisoning are the highest priority for screening. Table 6-1 provides guidance on the groups for which repeated screening is most strongly indicated.

Children ages 6 to 72 months who live in or are frequent visitors to deteriorated old buildings, including day care centers, make up the highest priority group. Because the highest concentrations of lead in paint were used in the early 1900s, homes built before about 1960 are of greatest concern. Children whose homes are being renovated are also at extremely high risk. Since siblings, housemates, visitors, and playmates of children with confirmed lead poisoning may have similar exposures to lead, they also should be promptly screened. In communities with a high prevalence of lead poisoning, health departments should consider door-to-door screening, since many children with lead poisoning may be missed by fixed-site screening.

Children with parents whose work or hobbies involve lead may also risk lead exposure (Chapter 3). Also, children living near lead smelters or other industries where lead is processed may be at increased risk for lead poisoning.

In general, screening and assessment for lead poisoning should focus on children younger than 72 months of age, particularly on children younger than 36 months of age. Young children engage in the most hand-to-mouth activity (and therefore are at highest risk for lead exposure) and have the most rapidly developing nervous systems, making them more vulnerable to the effects of lead. Children with developmental delays, who may exhibit pica or have more extensive hand-to-mouth activity than other children, would be expected to be at increased risk for lead poisoning even if they are 72 months of age and older. These children may have to be screened more often during early infancy, and may require screening into their school years.

Children who have unexplained seizures, neurological symptoms, abdominal pain, or other symptoms that are consistent with lead poisoning should also have their blood lead levels measured. In addition, the possibility of lead poisoning should be considered in any child with growth failure, developmental delay, hyperactivity, behavior disorders, hearing loss, anemia, etc.

Table 6-1. Priority groups for screening

- Children, ages 6 to 72 months, who live in or are frequent visitors to deteriorated housing built before 1960.
- Children, ages 6 to 72 months, who live in housing built before 1960 with recent, ongoing, or planned renovation or remodelling.
- Children, ages 6 to 72 months, who are siblings, housemates, or playmates of children with known lead poisoning.
- Children, ages 6 to 72 months, whose parents or other household members participate in a lead-related occupation or hobby.
- Children, ages 6 to 72 months, who live near active lead smelters, battery recycling plants, or other industries likely to result in atmospheric lead release.

Screening Method

Since erythrocyte protoporphyrin (EP) is not sensitive enough to identify more than a small percentage of children with blood lead levels between 10 and 25 μg/dL and misses many children with blood lead levels ≥25 μg/dL, measurement of blood lead levels should replace the EP test as the primary screening method. Unless contamination of capillary blood samples can be prevented, lead levels should be measured on venous samples. Obtaining capillary specimens is more feasible at many screening sites. Contamination of capillary specimens obtained by finger prick can be minimized if trained personnel follow proper technique. Elevated blood lead results obtained on capillary specimens should be considered presumptive and must be confirmed using venous blood. At the present time, not all laboratories will measure lead levels on capillary specimens.

Anticipatory Guidance and Assessing Risk

Guidance on childhood lead poisoning prevention and assessment of the risk of lead poisoning should be part of routine pediatric care. Anticipatory guidance is discussed in detail in Chapter 4. The guidance and risk assessment should emphasize the sources and exposures that are of greatest concern in the child's community (Chapter 3). Because lead-based paint has been used in housing throughout the United States, in most communities it will be necessary to focus on this source.

Table 6-2 has sample questions for assessing a child's risk for high-dose lead exposure. Starting at 6 months of age and at each regular office visit thereafter, pediatric health-care providers should discuss childhood lead poisoning and assess the child's risk for high-dose exposure. The questions asked should be tailored to the likely sources of exposure in the community. **The questions are not a substitute for a blood lead test.**

On the basis of responses to questions such as those in Table 6-2, children can be categorized as low or high risk for high-dose lead exposure. If the answers to all questions are negative, the child is at low risk for high-dose lead exposure and should be screened by a blood lead test at 12 months and again, if possible, at 24 months (since blood lead levels often peak at ages greater than 12 months). If the answer to any question is positive, the child is potentially at high risk for high-dose lead exposure, and a blood lead test should be obtained. **For children previously at low risk, any history suggesting that exposure to lead has increased should be followed up with a blood lead test.**

Table 6-2. 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 remodelling?
3. Have a brother or sister, housemate, or playmate being followed or treated for lead poisoning (that is, blood lead ≥15 μg/dL)?
4. Live with an adult whose job or hobby involves exposure to lead (see Chapter 3)?
5. Live near an active lead smelter, battery recycling plant, or other industry likely to release lead?

Screening Schedule

The following sections provide a minimum screening schedule for children aged 6 up to 36 and 36 to 72 months. The schedule is not rigid. Rather, it is a guide for pediatric health-care providers and screening programs to use in conjunction with other pertinent information in determining when an individual child should be tested. Programs and pediatric health-care providers may choose to screen more frequently than described below.

Children 6 up to 36 months of age:

A questionnaire should be used at each routine office visit to assess the potential for high-dose lead exposure and, therfore, the appropriate frequency of screening.

Schedule if the child is at low risk for high dose lead exposure by questionnaire:

A child at *low risk* for exposure to high-dose lead sources by questionnaire should have an initial blood lead test at 12 months of age.

If the 12-month blood lead result is **<10 μg/dL**, the child should be retested at 24 months if possible, since that is when blood lead levels peak.

If a blood lead test result is **10-14 μg/dL**, the child should be retested every 3 to 4 months. After 2 consecutive measurements are <10 μg/dL or three are <15 μg/dL, the child should be retested in a year.

If any blood lead test result is **≥15 μg/dL**, the child needs individual case management and should be retested at least every 3 to 4 months (Page 96).

Schedule if the child is at high risk for high dose lead exposure by questionnaire:

A child at *high risk* for exposure to high-dose lead sources by questionnaire should have an initial blood lead test at 6 months of age.

If the initial blood lead result is **<10 μg/dL,** the child should be rescreened every 6 months. After 2 subsequent consecutive measurements are <10 μg/dL or three are <15 μg/dL, testing frequency can be decreased to once a year.

If a blood lead test result is **10-14 μg/dL**, the child should be screened every 3 to 4 months. Once 2 subsequent consecutive measurements are <10 μg/dL or three are <15 μg/dL, testing frequency can be decreased to once a year.

If any blood lead test result is **≥15 μg/dL**, the child needs individual case management and should be retested at least every 3 to 4 months (Page 94).

Children ≥36 months and <72 months of age:

As for younger children, a questionaire should be used at each routine office visit of children from 36 to 72 months of age. Any child at high risk by questionaire who has not previously had a blood lead test should be tested. All children who have had venous blood lead tests ≥15 μg/dL or who are at high risk by questionnaire should be screened at least once a year until their sixth birthday (age 72 months) or later, if indicated (for example, a retarded child with pica). Children should also be rescreened any time history suggests exposure has increased. Children with blood lead levels ≥15 μg/dL should receive followup as described below.

Followup of children with blood lead levels ≥15 μg/dL

Followup of children with blood lead levels ≥15 μg/dL is discussed in more detail in later chapters and is briefly summarized below. In general, such children should receive blood lead tests at least every 3 to 4 months.

If the blood lead level is 15-19 μg/dL, the child should be screened every 3-4 months, the family should be given education and nutritional counselling as described in Chapter 4, and a detailed environmental history should be taken to identify any obvious sources or pathways of lead exposure. When the venous blood lead level is in this range in two consecutive tests 3-4 months apart, environmental investigation and abatement should be conducted, if resources permit.

If the blood lead level is ≥20 μg/dL, the child should be given a repeat test for confirmation. If the venous blood lead level is confirmed to be ≥20 μg/dL, the child should be referred for medical evaluation and followup as described in Chapter 7. Such children should continue to receive blood lead tests every 3-4 months or more often if indicated. Children with blood lead levels ≥45 μg/dL must receive urgent medical and environmental followup, preferably at a clinic with a staff experienced in dealing with this disease. Symptomatic lead poisoning or a venous blood lead concentration ≥70 μg/dL is a medical emergency, requiring immediate inpatient chelation therapy, as described in Chapter 7.

Classification on the Basis of Screening Test Results

On the basis of screening test results, children can be classified into categories according to their risk for adverse effects of lead. The urgency and type of followup are based on these risk classes. These classes are shown in Table 6-3.

Measurement of Blood Lead Levels

Several factors can influence the quality of blood lead measurements. The ubiquity of lead in the environment makes contamination of specimens during collection a major source of error. Analytical variation in the laboratory can affect results. Accuracy and precision of blood lead measurements, particularly at low concentrations, can be assured by the use of appropriate analytical standards, maintenance of equipment, training of personnel, and participation in external proficiency testing programs.

Since blood collected by venipuncture has a low likelihood of contamination compared to blood collected by fingerstick, venous blood is the preferred specimen for analysis and should be used for lead measurement whenever practicable. In addition, venous specimens provide a larger volume for analysis and are less prone to clotting and other problems that can be encountered with capillary specimens. At the present time, not all laboratories will accept capillary samples for lead analysis.

Fingerstick specimens are acceptable for blood lead screening, provided that special collection procedures are followed to minimize the risk of contamination. Personnel must be thoroughly trained in collection procedures. A procedure for collecting fingerstick specimens is described in Appendix I.

Elevated blood lead results obtained on capillary specimens are presumptive and must be confirmed using venous blood. In general, children who have blood lead levels ≥15 μg/dL on capillary samples should have these levels confirmed on venous samples, according to the timetable in Table 6-4. A child with a blood lead level ≥70 μg/dL or with symptoms of lead poisoning should be treated immediately while the results of an immediate confirmatory test are awaited.

Table 6-3. Class of child and recommended action according to blood lead measurement

Class	Blood Lead Concentration (μg/dL)	Action
I	≤9	Low risk for high-dose exposure: rescreen as described in text. High risk for high-dose exposure: rescreen as described in text.
IIA	10-14	Rescreen as described in text. If many children in the community have blood lead levels ≥10, community interventions (primary prevention activities) should be considered by appropriate agencies (see Chapter 9).
IIB	15-19	Rescreen as described in text. Take a history to assess possible high-dose sources of lead. Educate parents about diet, cleaning, etc. Test for iron deficiency. Consider environmental investigation and lead hazard abatement if levels persist.
III	20-44*	Conduct a complete medical evaluation. Identify and eliminate environmental lead sources.
IV	45-69*	Begin medical treatment and environmental assessment and remediation within 48 hours.
V	≥70*	Begin medical treatment and environmental assessment and remediation IMMEDIATELY.

*Based on confirmatory blood lead level.

Table 6-4. 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 month
20-44	Within 1 week
45-69	Within 48 hours
≥70	Immediately

Blood Lead Levels—Additional Analytical Considerations

Blood lead levels can be determined by several analytic methods. The method used can affect the specimen volume required, the choice of anticoagulant (usually heparin or ethylenediaminetetraacetic acid (EDTA)), and other aspects related to specimen suitability. Specimen collection procedures and equipment must be checked for compatibility with laboratory

requirements. Special lead-free evacuated tubes are available for blood collection, but standard tubes containing EDTA or heparin (lavender or green caps) can be acceptable after screening each lot to determine the lead content of the containers, needles, etc. Though reports of unsuitable levels of background lead in other collection materials are infrequent, all materials used should be determined to be lead-free before use.

Laboratories where blood is tested for lead levels should be successful participants in a blood lead proficiency testing program, such as the program conducted jointly by CDC, the Health Resources and Services Administration, and the University of Wisconsin. In interpreting laboratory results, it should be recognized that a "proficient" laboratory need only measure blood lead levels to within several μg/dL of the true value (for example, within 4 or 6 μg/dL of a target value). The blood lead level reported by a laboratory, therefore, may be several μg/dL higher or lower than the actual blood lead level.

Erythrocyte Protoporphyrin (EP)

EP is not a sensitive test to identify children with blood lead levels below about 25 μg/dL, and therefore it is no longer the screening test of choice. In some programs, however, it will continue to be used until the transition to blood lead measurements is complete.

Only fresh blood is suitable for analysis by hematofluorometer. Complete oxygenation of sample hemoglobin is necessary to prevent low results in some instruments. The hemoglobin concentration in the sample can also affect hematofluorometer EP readings. Results obtained by extraction methods are not affected by these factors and can be used to confirm hematofluorometer EP results.

In the past, an absorptivity of 241 L cm-1 mmol-1 has been used to determine EP levels. Recently, however, the correct absorptivity has been determined to be 297 L cm-1 mmol-1. Use of the correct absorptivity will result in EP values about 19% lower than those standardized using 241 L cm-1 mmol-1. Standardization of EP levels that are based on the correct absorptivity is expected to be widely adopted in 1992. Use of the correct standardization requires a change in calibration and is not simply a reduction of the screening cutoff value. Standardization criteria should also be considered when reviewing data in the literature.

An EP result of ≥35 μg/dL standardized using 241 L cm-1 mmol-1 or ≥28 μg/dL standardized using 297 L cm-1 mmol-1 is considered elevated. **All elevated EP results should be followed with a venous blood lead test to determine if lead poisoning is responsible for the elevation**. Elevated concentrations of EP also result from several health conditions other than lead intoxication, particularly iron deficiency. The iron status of children with elevated EP levels should always be determined, especially since iron deficiency and lead poisoning often coexist. In such cases, the EP may be disproportionately elevated in comparison to the blood lead level.

Some hematofluorometers report EP levels as μmol ZnPP/mol heme. For instruments that give results in these units, EP values ≥70 μmol/mol should be considered elevated and should be promptly investigated.

CHAPTER 7. DIAGNOSTIC EVALUATION AND MEDICAL MANAGEMENT OF CHILDREN WITH BLOOD LEAD LEVELS ≥20 μg/dL

Children with blood lead levels between 10 μg/dL and 19 μg/dL and their siblings need followup and repeat screening as described in previous chapters. They do not, however, need medical evaluation as described in this chapter.

The cornerstones of clinical management are careful clinical and laboratory surveillance of the child, medical treatment when indicated, and eradication of controllable sources of environmental lead. **The most important factor in case management is to drastically reduce the child's exposure to lead.**

All children with confirmed venous blood lead levels ≥20 μg/dL require medical evaluation. The urgency of further medical evaluation depends on the blood lead level and whether symptoms are present.

The decision to institute medical management should virtually always be made on the basis of a venous blood lead measurement. No other screening test can be considered diagnostic. If the first evaluation was made on capillary blood, a confirmatory venous blood lead level must be done. Even if the first diagnostic measurement was on venous blood, it is preferable to retest before starting chelation therapy. For children with blood lead levels ≥70 μg/dL or clinical symptoms of lead poisoning, chelation should not be postponed while awaiting results of the repeat test.

Symptoms of Lead Poisoning

Symptoms of lead poisoning in a child with an elevated blood lead level constitute a medical emergency, and the child should be hospitalized. Symptoms, which can mimic several other pediatric disorders, must be looked for so they are not missed.

Acute lead encephalopathy is characterized by some or all of these symptoms: coma, seizures, bizarre behavior, ataxia, apathy, incoordination, vomiting, alteration in the state of consciousness, and subtle loss of recently acquired skills. Any one or a mixture of these symptoms, associated with an elevated blood lead level, is an acute medical emergency. Lead encephalopathy is almost always associated with a blood lead level exceeding 100 μg/dL, although, occasionally, it has been reported at blood lead levels as low as 70 μg/dL. Even when identified and promptly treated, severe and permanent brain damage may result in 70%-80% of children with lead encephalopathy. Children with symptomatic lead poisoning with or without encephalopathy represent an acute medical emergency. **The possibility of lead encephalopathy should be considered in the differential diagnosis of children presenting with coma and convulsions of unknown etiology.**

Except for coma and seizures, symptomatic lead poisoning without encephalopathy is characterized by symptoms similar to those of lead encephalopathy. Symptomatic lead poisoning without encephalopathy is characterized by one or a combination of these symptoms: decrease in play activity, lethargy, anorexia, sporadic vomiting, intermittent abdominal pain, and constipation. These symptoms are usually associated with a blood lead levels of at least 70 μg/dL, although occasionally cases have been associated with levels as low as 50 μg/dL. If the blood lead level is below 50 μg/dL, other causes of the symptoms should be sought. **Since acute lead encephalopathy may develop in any symptomatic child, treatment and supportive measures must be started immediately on an emergency basis.**

Evaluation of the Child with a Blood Lead Level ≥20 μg/dL

A child with a blood lead level ≥20 μg/dL should have a pediatric evaluation, whether or not symptoms are present.

Special attention should be given to:

1. A detailed history, including the presence or absence of clinical symptoms, child's mouthing activities, the existence of pica, nutritional status (especially iron and calcium intake), dietary habits, family history of lead poisoning, potential sources of lead exposure (including exposure due to home renovation), and previous blood lead measurements.
2. Detailed environmental and occupational histories of adults in the household or other places the child spends a lot of time.
3. The physical examination, with particular attention to the neurologic examination and psychosocial and language development. A neurobehavioral assessment may be useful in children receiving chelation therapy both at the time of diagnosis and as the child approaches school age. Findings of language delay or other problems can prompt referral to appropriate programs.
4. Evaluation of iron status using measurment of iron and total iron binding capacity or of ferritin.

Tests

1. Tests for Iron Deficiency. ***Because iron deficiency can enhance lead absorption and toxicity and often coexists with it, all children with blood lead levels ≥20 μg/dL should be tested for iron deficiency.*** Measurements of hemoglobin, hematocrit, and reticulocytes are not adequately sensitive, and erythrocyte protoporphyrin (EP) is not specific enough to diagnose iron deficiency (although EP can be used to screen for iron deficiency).

Serum iron and iron binding capacity (transferrin saturation) and ferritin are the most sensitive indicators of iron status. An abnormally low ratio of serum iron to iron binding capacity (transferrin saturation) of 0.2 is consistent with iron deficiency. The serum ferritin level, however, is the most definitive and accurate indication of overall iron status, although it is an acute phase reactant and may be falsely elevated in sick children; a value ≤12 μg/dL indicates iron deficiency. Although all iron deficient children should receive treatment for this condition, the treatment should not be started until after chelation is completed in children receiving dimercaprol (BAL).

2. EP Level. An elevated EP level indicates impairment of the heme biosynthetic pathway. EP levels are sensitive screening tests for iron deficiency, and iron status should be assessed in any child with an elevated EP level (that is, ≥35 μg/dL when standardized using 241 L cm-1 mmol-1, ≥28 μg/dL when standardized using 297 L cm-1 mmol-1, or ≥70 μmol/mol when measured in μmol/mol units).

Because EP levels take about 2 weeks to increase, EP levels may provide an indication of the duration of lead exposure. Similarly, monitoring the EP level after medical and environmental interventions for poisoned children may be useful. If exposure to lead has ceased, EP values elevated because of lead poisoning decline slowly over several weeks or months. A progressive decline in EP concentrations indicates that combined medical and environmental case management is proceeding efficaciously.

3. Edetate Disodium Calcium ($CaNa_2EDTA$) Provocative Chelation Test. The mobilization test is used to determine whether a child with an initial confirmatory blood lead level of 25 to 44 μg/dL will respond to chelation therapy with a brisk lead diuresis. Because of the cost and staff time needed for quantitative urine collection, this test is used only in selected medical centers where large numbers of lead-poisoned children are treated. Children whose blood lead levels are ≥45 μg/dL should not receive a provocative chelation test; they should be referred for appropriate chelation therapy immediately.

The outcome of the provocative chelation test is determined not by a decrease in the blood lead level but by the amount of lead excreted per dose of $CaNa_2EDTA$ given. This ratio correlates well with blood lead levels. In one study, almost all children with blood lead levels 45 μg/dL had positive provocative tests, 76% of the children with blood lead levels 35 to 44 μg/dL had positive test results, and 35% of the children with blood lead levels 25 to 34 μg/dL had positive test results. This test should not be done until the child is iron replete, since iron status may affect the outcome of the test. Details on how to conduct and interpret a provocative chelation test are in Chapter 7.

4. Radiologic Examination of the Abdomen. Radiologic examination of the abdomen (flat plate) may show radiopaque foreign material if the material has been ingested during the preceding 24 to 36 hours. Neither negative nor positive xray results are diagnostic or definitive. A flat plate of the abdomen may, however, provide information about the source of lead if paint chips or other lead objects are found.

5. Radiologic Examination of the Long Bones. Xrays of the long bones are unreliable for diagnosing acute lead poisoning, and they should not be obtained on a routine basis. They may provide some indication of whether lead poisoning has occurred in the past or has been ongoing for a length of time, and this may occasionally be important. Lines of increased density in the metaphyseal plate of the distal femur, proximal tibia, and fibula may be caused by lead which has disrupted the metabolism of bone matrix. Although these lines are sometimes called lead lines, they are areas of increased mineralization or calcification and not xray shadows of deposited lead.

The following tests are **NOT** indicated for the diagnosis or clinical management of lead poisoning:

1. Microscopic examination of red cells for basophilic stippling. Since basophilic stippling is not always found in severe lead poisoning and is insensitive to lesser degrees of lead poisoning, it is not useful in diagnosis.

2. Tests of hair and fingernails for lead levels. The levels of lead in hair or fingernails do not correlate well with blood lead levels levels, except in extreme cases of symptomatic lead poisoning; therefore, these tests are not useful in diagnosis. Children should never receive chelating agents on the basis of analyses of lead levels in hair or fingernails.

Pharmacology of Chelating Agents

Several drugs are used in the treatment of lead poisoning. These drugs, capable of binding or chelating lead, deplete the soft and hard (skeletal) tissues of lead and thus reduce its acute toxicity. All drugs have potential side effects and must be used with caution. The basic pharmacologic characteristics of the various drugs are described below.

Chelating Agents Used In Treating Children With Lead Poisoning			
Product Name	**Generic Name**	**Chemical Name**	**Abbreviation**
Calcium Disodium Versenate	Edetate disodium calcium	Calcium disodium ethylenediamine tetraacetate	$CaNa_2$ EDTA
BAL in Oil	Dimercaprol	2,3-dimercapto-1-propanol	BAL
Cuprimine	D-penicillamine	3-mercapto-D-valine	D-penicillamine
Chemet	Succimer	Meso 2,3-dimercaptosuccinic acid	DMSA

BAL

Mechanism of action. Two molecules of dimercaprol (BAL) combine with one atom of heavy metal to form a stable complex. BAL enhances fecal and urinary excretion of lead and diffuses well into erythrocytes. Because it is predominantly excreted in bile, BAL can be administered in the presence of renal impairment.

Route of administration and dosage. BAL is available only in peanut oil for intramuscular administration. It is usually given every 4 hours, although it may be given every 8 hours; dosages are discussed below.

Precautions and Toxicity. For patients with glucose-6-phosphate dehydrogenase deficiency (G-6-PD), some clinicians recommend that BAL should be used only in life-threatening situations because it may induce hemolysis. Medicinal iron should never be administered during BAL therapy, because the combination of iron and BAL has been implicated in serious reactions. If iron deficiency coexists, it should not be treated until after BAL therapy has been completed. In cases of extreme anemia, blood transfusions are preferable.

Between 30% and 50% of patients who receive BAL will experience side effects. Mild febrile reactions and transient elevations of hepatic transaminases may be observed. Other minor adverse effects include, in order of frequency, nausea and occasional vomiting, headache, mild conjunctivitis, lacrimation, rhinorrhea, and salivation. Most side effects are transient and rapidly subside as the drug is metabolized and excreted. Intravenous hydration coupled with restricting oral intake can circumvent, in large part, gastrointestinal distress. **BAL should not be used for children who are allergic to peanuts or peanut products.**

$CaNa_2EDTA$

Only $CaNa_2EDTA$ can be used for treating children with lead poisoning. Na_2EDTA (disodium edetate) should never be used for treating children with lead poisoning because it will induce tetany and possibly fatal hypocalcemia.

Mechanism of action. $CaNa_2EDTA$ increases urinary lead excretion twentyfold to fiftyfold. $CaNa_2EDTA$ removes lead from the extracellular compartment only, because it does not enter cells.

Route of administration and dosage. The preferred route for administration of $CaNa_2EDTA$ is intravenous. $CaNa_2EDTA$ must be diluted to a concentration <0.5% in dextrose and water or in 0.9% saline solution. It can be given as a continuous infusion or it can be given in two divided doses a day through a heparin lock over 30 to 60 minutes. $CaNa_2EDTA$ causes

extreme pain when administered intramuscularly; therefore, when given by this route, it should be mixed with procaine so that the final concentration of procaine is 0.5%. $CaNa_2EDTA$ should never be given orally because it enhances absorption of lead from the gastrointestinal tract.

Dosages vary by situation and are detailed in Chapter 7. Individual courses should be limited to 5 days and repeated courses should be given at a minimum of 2- to 5-day intervals. Particularly when $CaNa_2EDTA$ is given on an outpatient basis, some clinicians use sequential 3-day courses of treatment.

Precautions and Toxicity. During chelation therapy with $CaNa_2EDTA$, urine output, urine sediment, blood urea nitrogen (BUN), serum creatinine, and hepatocellular enzyme levels must be carefully monitored. The appearance of protein and formed elements in urinary sediment, and rising BUN and serum creatinine values reflect impending renal failure—the serious toxicity associated with inappropriately excessive or prolonged administration of $CaNa_2EDTA$. Liver transanimases may increase by the fifth day of therapy, but return to pretreatment levels within a week after treatment has ended.

When $CaNa_2EDTA$ is used alone without concomitant BAL therapy, it may aggravate symptoms in patients with very high blood lead levels. Therefore, it should be used in conjunction with BAL when the blood lead level is ≥ 70 μg/dL or overt clinical symptoms of lead poisoning are present. In such cases, the first dose of BAL should always precede the first dose of $CaNa_2EDTA$ by at least 4 hours.

The kidney is the principal site of potential toxicity. Renal toxicity is dose related, reversible, and rarely (if ever) occurs at doses <1500 mg/m^2 when the patient is adequately hydrated. $CaNa_2EDTA$ must never be given in the absence of an adequate urine flow.

D-penicillamine

The Food and Drug Administration (FDA) has approved D-penicillamine for the treatment of Wilson's disease, cystinuria, and severe, active rheumatoid arthritis. Although not approved for this use, it is used in some centers for treating lead poisoning. Until the recent approval of succimer, it was the only commercially available oral chelating agent. It can be given over a long period (weeks to months). D-penicillamine has been used mainly for children with blood lead levels <45 μg/dL.

Mechanism of action. D-penicillamine enhances urinary excretion of lead, although not as effectively as $CaNa_2EDTA$. Its specific mechanism and site of action are not well understood.

Route of administration and dosage. D-penicillamine is administered orally. It is available in capsules or tablets (125 mg and 250 mg). These capsules can be opened and suspended in liquid, if necessary. The usual dose is 25 to 35 mg/kg/day in divided doses. Side effects can be minimized, to an extent, by starting with a small dose and increasing it gradually, monitoring all the time for side effects. For example, 25% of the desired final dose could be given in week 1, 50% in week 2, and the full dose by week 3.

Precautions and Toxicity. Toxic side effects (albeit minor in most cases) occur in as many as 33% of patients given the drug. The main side effects of D-penicillamine are reactions resembling those of penicillin sensitivity, including rashes, leukopenia, thrombocytopenia, hematuria, proteinuria and hepatocellular enzyme elevations, and eosinophilia. Anorexia, nausea, and vomiting are infrequent. Of most concern, however, are isolated reports of nephrotoxicity, possibly from hypersensitivity reactions. For these reasons, patients should be carefully and frequently monitored for clinically obvious side effects, and frequent blood counts,

urinalyses, and renal function tests should be performed. In particular, blood counts and urinalyses should be done on day 1, day 14, day 28, and monthly thereafter. If the absolute neutrophil count falls to <1500/μL, the count should be rechecked immediately, and treatment should be stopped if it falls to <1200/μL. D-penicillamine should not be given on an outpatient basis if exposure to lead is continuing or the physician has doubts about compliance with the therapeutic regimen. **D-penicillamine should not be administered to patients with known penicillin allergy.**

Succimer

The FDA approved succimer in January, 1991 for treating children with blood lead levels >45 μg/dL. Succimer appears to be an effective oral chelating agent. Its selectivity for lead is high, whereas its ability to chelate essential trace metals is low. Although its use to date has been limited, succimer appears to have promising potential, and a broader range of clinical research studies in children are being undertaken.

Succimer is chemically similar to BAL but is more water soluble, has a high therapeutic index, and is absorbed from the gastrointestinal tract. It is effective when given orally and produces a lead diuresis comparable to that produced by $CaNa_2EDTA$. This diuresis lowers blood lead levels and reverses the biochemical toxicity of lead, as indicated by normalization of circulating aminolevulinic acid dehydrase levels. Succimer is not indicated for prophylaxis of lead poisoning in a lead-containing environment. **As with all chelating agents, succimer should only be given to children who reside in environments free of lead during and after treatment.**

Mechanism of Action. Succimer appears to be more specific for lead than the most commonly used chelating agent, $CaNa_2EDTA$; the urinary loss of essential trace elements (for example, zinc) appears to be considerably less with succimer than with $CaNa_2EDTA$. The site of lead chelation by succimer is not known.

Route of Administration and Dosage. Succimer is administered orally. It is available in 100 mg capsules. The recommended initial dose is 350 mg/m^2 (10 mg/kg) every 8 hours for 5 days, followed by 350 mg/m^2 (10 mg/kg) every 12 hours for 14 days. A course of treatment, therefore, lasts 19 days. If more courses are needed, a minimum of 2 weeks between courses is preferred, unless blood lead levels indicate the need for immediate retreatment. These doses may be modified as more experience is gained in using succimer.

Patients who have received therapeutic courses of $CaNa_2EDTA$ with or without BAL may use succimer for subsequent treatment after an interval of 4 weeks. Data on the concomitant use of succimer and $CaNa_2EDTA$ with or without BAL are not available, and such use is not recommended.

If young children cannot swallow capsules, succimer can be administered by separating the capsule and sprinkling the medicated beads on a small amount of soft food or by putting them on a spoon and following with a fruit drink. Data are not available on how stable succimer is when it is suspended in soft foods for prolonged periods of time; succimer should be mixed with soft foods immediately before being given to the child.

Precautions and Toxicity. To date, toxicity due to succimer (transient elevations in hepatic enzyme activities) appears to be minimal. The most common adverse effects reported in clinical trials in children and adults were primarily gastrointestinal and included nausea, vomiting, diarrhea, and appetite loss. Rashes, some necessitating discontinuation of therapy, have been reported for about 4% of patients. **Though succimer holds considerable promise for the**

outpatient management of lead poisoning, clinical experience with succimer is limited. Consequently, the full spectrum and incidence of adverse reactions, including the possibility of hypersensitivity or idiosyncratic reactions, have not been determined. Other precautions that need to be taken with succimer are discussed in the full statement.

Treatment Guidelines For Children With Blood Lead Levels ≥20 μg/dL

The single most important factor in managing of childhood lead poisoning is the reducing the child's exposure to lead; some children, however, will benefit from chelation therapy. Sample regimens for treating children with lead poisoning are described in Chapter 7.

Medical Management of Symptomatic Lead Poisoning (with or without Encephalopathy)

Children with symptomatic lead poisoning (with or without encephalopathy) must be treated only at a pediatric center that has an intensive care unit. They should be managed by a multidisciplinary team that includes, as needed, critical care, toxicology, neurology, and neurosurgery. The child's neurological status and fluid balance must be carefully monitored.

Medical Management of Asymptomatic Lead Poisoning

Blood lead level ≥45 μg/dL. Children with blood lead levels ≥45 μg/dL (with or without symptoms) should undergo chelation therapy. A blood lead level ≥70 μg/dL is a medical emergency.

Blood lead level 25 to 44 μg/dL. For this blood lead range, the effectiveness of chelation therapy in decreasing the adverse effects of lead on children's intelligence has not been shown. Treatment regimens vary from clinic to clinic. Some practioners treat children with lead levels in this range pharmacologically, some use D-penicillamine. The minimum medical management for children with these blood lead levels is to decrease exposure to all sources of lead, to correct any iron deficiency and maintain an adequate calcium intake, and to test frequently to ensure that the child's blood lead levels are decreasing. Many experienced practioners decide whether to use chelation therapy on the basis of the results of carefully performed $CaNa_2EDTA$ mobilization tests.

Blood lead level 20 to 24 μg/dL. **Only very minimal data exists about chelating children with blood lead levels below 25 μg/dL, and such children should not be chelated except in the context of approved clinical trials.** A child with a confirmed blood lead level of 20 to 24 μg/dL will require individual case management by a pediatric health-care provider. The child should have an evaluation with special attention to nutritional and iron status. The parents should be taught about 1) the causes and effects of lead poisoning, 2) the need for more routine blood lead testing, 3) possible sources of lead intake and how to reduce them, 4) the importance of adequate nutrition and of foods high in iron and calcium, and 5) resources for further information. (This is described in more detail in Chapter 4.) Sequential measurements of blood lead levels along with review of the child's clinical status should be done at least every 3 months. Iron deficiency should be treated promptly. Children with blood lead levels in this range should be referred for environmental investigation and management. **Identifying and eradicating all sources of excessive lead exposure is the most important intervention for decreasing blood lead levels (Chapter 8).**

Post-Chelation Followup

At the end of each treatment cycle, the blood lead concentration usually declines to <25 μg/dL. Within a few days, however, reequilibration among body lead compartments takes place and may result in a rebound; thus, **the blood lead level must be rechecked 7 to 21 days after treatment to determine whether retreatment is necessary**.

Children who undergo chelation treatment require long-term followup preferably from pediatric health-care providers, nutritionists, environmental specialists, and community outreach workers. Community outreach workers provide a critical bridge between hospital-based or clinic-based (outpatient) medical care, health advocacy education, and environmental remediation outside the hospital. Children should **never** be discharged from the hospital **until they can go to a lead-free environment**. Lead-free safe housing (with friends, relatives, or in designated transitional housing), in which a treated child can live during the entire abatement process through the post-abatement clean-up, must be arranged. With appropriately carried-out public health measures, complete and safe abatement should be achieved during the treatment period.

Once a child is discharged to a safe environment, frequent followup is mandatory. In general, depending on the initial blood lead value, most children who require chelation therapy must be followed closely for at least one year or more. All children undergoing chelation treatment should be seen every other week for 6-8 weeks, then once a month for 4-6 months. A child treated with BAL and $CaNa_2EDTA$ should be followed more closely: weekly for 4 to 6 weeks, then monthly for 12 months.

CHAPTER 8. MANAGEMENT OF LEAD HAZARDS IN THE ENVIRONMENT OF THE INDIVIDUAL CHILD

Eradicating childhood lead poisoning requires a long-term active program of primary lead-poisoning prevention, including abatement of lead-based paint hazards in homes, day-care centers, and other places where young children play and live. For the child who is lead poisoned, however, efficient and effective interventions are needed as quickly as possible. Abatement means making the source of lead inaccessible to the child.

Each situation in which a child gets poisoned is unique and must be evaluated by a person or team of persons skilled and knowledgeable about lead poisoning, hazard identification, and interventions to reduce lead exposure, including abatement of lead-based paint in housing. Childhood lead poisoning prevention programs need to work closely with other relevant agencies (for example, housing and environmental agencies) to ensure that the quickest and most effective approach is taken to remediating the environments of poisoned children.

Environmental case management includes a number of actions prescribed for a child with lead poisoning. Ideally, environmental case management should be conducted by a team of professionals in public health, environmental activities, medical management, and social management. A team approach to intervention will help ensure that followup is timely and effective. The management team may need to solve many related problems, such as whether to investigate supplemental addresses, where to find temporary alternative housing, and how to use community resources to assist the family in dealing with the lead-poisoned child.

CHAPTER 9. MANAGEMENT OF LEAD HAZARDS IN THE COMMUNITY

In theory, primary prevention has always been the goal of childhood lead poisoning prevention programs. In practice, however, most programs focus exclusively on secondary prevention, dealing with children who have already been poisoned. As programs shift the emphasis to primary prevention, their efforts must be designed to systematically identify and remediate environmental sources of lead, including, most importantly, dwellings containing old lead paint.

The shift from case management to community-level intervention will require a fundamental shift in perspective. The focus must shift from the individual child to the population of children at risk and the environment in which they live. The purpose of community-level intervention is to identify and respond to sources, not cases, of lead poisoning. The responsibility for addressing lead poisoning will have to be expanded beyond health agencies to include a variety of housing, environmental, and social service agencies at the local, county, state, and national level.

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