Lead Poisoning : Successes and 21st Century Challenges

Danielle Laraque and Leonardo Trasande

Pediatrics in Review 2005;26;435
DOI: 10.1542/pir.26-12-435

Lead Poisoning: Successes and 21st Century Challenges

Danielle Laraque, MD,*
Leonardo Trasande, MD, MPP†

Author Disclosure
Drs Laraque and Trasande did not disclose any financial relationships relevant to this article.

Objectives

After completing this article, readers should be able to:

1. Describe the sources of lead exposure and their changes over time.
2. Delineate the cognitive difficulties associated with lead exposure and uptake.
3. Describe the screening recommendations and the tests available for determining blood lead levels.
4. Discuss how to manage an increased blood lead level.

Introduction

An 8-month-old Latino male is seen for an initial visit to a pediatric practice. Blood is obtained via fingerstick for routine complete blood count and blood lead level (BLL). The hemoglobin concentration is 12.9 g/dL (129 g/L), and the BLL is 17 mcg/dL (0.82 mcmol/L). The family is called back for confirmatory testing. Detailed environmental history reveals that the child's father works with leaded glass and reports coming home in his work clothes in the past several months. BLL obtained on the father is 37 mcg/dL (1.8 mcmol/L), and he is referred to the occupational medicine service. The child's repeat venous blood lead result is 9 mcg/dL (0.43 mcmol/L). Specific risk reduction is reviewed with the family, and the child is monitored carefully over time for any re-exposure and progress of growth and development.

This illustrative case is not atypical in the 21st century. Over the past generation, epidemiologic studies have provided the foundation for efforts to combat children's exposure to lead through primary prevention, early identification via detailed environmental history and blood lead screening, and various treatment modalities. Although a BLL of 40 mcg/dL (1.9 mcmol/L) was considered commonplace and healthy in the 1940s, and the absence of obvious symptoms reassured pediatricians of that era, this no longer is the case. With improved understanding of the subclinical toxicity of lead and other environmental hazards, the standard of care has become proactive screening and environmental intervention to prevent any elevation in BLL. Landmark work by Herbert L. Needleman demonstrated an inverse relationship between dentine lead and intelligence quotient (IQ), and studies by others further defined populations that remain at risk, documenting the toxic effects of lead at levels less than 10 mcg/dL (0.48 mcmol/L) and establishing that these effects have no apparent threshold. Improved techniques for establishing the prevalence of lead hazards (eg, lead-based paint) also have contributed to progress. Advocacy efforts have been critical in bringing about legislation to continue to reduce exposure and to force compliance with removal of lead from the environment. The change in the clinical presentation of children who are lead poisoned has demanded vigilance on the part of clinicians and consideration of the long-term consequences of lead exposure. Nonetheless, many challenges remain.

Historical Perspective

The toxic effects of lead have been recognized for more than 2,000 years. The Romans were so fond of its diverse uses and sweet taste that they minimized the hazards it posed. During the Industrial Revolution, lead was used widely because of its durability, malleability, low melting point, and ability to form compounds. Its use in gasoline, interior and exterior paints, plumbing, and food and beverage containers led to broad and sweeping contamination of the environment. Numerous case reports of poisoning in occupational

*Editorial Board.
†Assistant Professor, Division of General Pediatrics, Center for Children's Health and the Environment, Mt. Sinai School of Medicine, New York, NY.

settings were followed by the President of the National Lead Company acknowledging that lead is toxic. These events culminated in the temporary suspension of leaded gasoline in 1925, but the Surgeon General subsequently found no evidence to ban lead from gasoline. Lead remained in gasoline and in common use; over the ensuing 40 years, clinical cases of lead poisoning were common.

Paralleling the widespread use of lead was documentation by pediatricians of its dangers to children. In 1904, J. Lockhart Gibson, an Australian, was among the first English-language authors to identify lead poisoning among children who chewed on the painted walls and verandas of houses. In 1914, Americans Henry Thomas and Kenneth Blackfan described the case of a Baltimore boy who died of lead poisoning after ingesting white lead paint from the railing of his crib. Fortunately, persistent advocacy finally led the United States Environmental Protection Agency to promulgate the phase-out of lead from gasoline, which occurred in the United States from 1975 to 1986 and in Europe in the 1990s. In addition, the United States Consumer Product Safety Commission placed a final ban on lead-based paint in 1977. In 1992, the United States Congress passed the Residential Lead-Based Paint Hazard Reduction Act of Title X of the Housing and Community Development Act, expanding on previous efforts.

Exposure

Although other sources for lead poisoning can be important, lead-based paint in older housing currently is the most important source of lead exposure. Children are exposed by eating paint chips or ingesting lead-contaminated house dust or soil through normal hand-to-mouth contact. Recent studies indicate that lead in household dust is the strongest predictor of childhood BLL. Children's mouthing behaviors and pica for chipping paint are recognized widely as causes of elevated blood lead concentrations. Lead-based paint also is responsible for the contamination of house dust and residential soil when housing units decline or are demolished or renovated. A recent study by Jacobs and associates (1) indicated that 38 million housing units had lead-based paint (and 24 million posed significant lead-based paint hazards), a decrease from the 1990 estimate of 64 million.

Leaded gasoline was responsible for most airborne lead before 1970. Less common sources of lead also have included glazed ceramics, storage battery casings, bullets, cosmetics, leaded glass, jewelry, and farm equipment. Avoiding the use of imported pottery for cooking, serving, or storing food reduces the risk of lead exposure. Sniffing of leaded gasoline also causes lead poisoning,

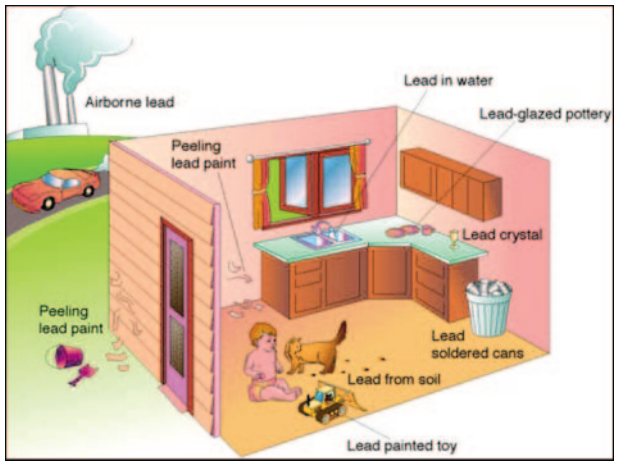

Figure. Sources of lead in the home and environment.

especially in developing countries where leaded gasoline still is used. Clinicians also should be aware of imported products from the Middle East, India, Pakistan, Bangladesh, China, Mexico, and Dominican Republic, among others, that contain lead. An example of such a product is *litargirio*, a powder that is sold as a home remedy and used as an antiperspirant/deodorant, which may contain up to 80% lead. Local Departments of Health Web sites are good sources of information that provide guidance to clinicians as they seek potential exposures that may occur among children from specific communities or cultural backgrounds.

Adults whose occupations use lead can inadvertently expose children to lead from contaminated clothes and shoes. Studies have shown that focused dust-control programs consisting of wet-mopping floors and windowsills and washing toys, pacifiers, bottles, and children's hands frequently in addition to home lead reduction can decrease BLLs significantly.

Pathogenesis, Definition, and Clinical Presentation

Young children normally explore their environment via hand-to-mouth activity and play close to the ground, behaviors that are likely to increase the lead intake of a child who lives in an environment that has hazards such as leaded paint in poor repair or elevated levels of lead in house dust or yard soils (Figure). Numerous studies also show that children have an uncanny ability to absorb lead from the gastrointestinal tract (40% compared with 10% in adults). Retention of lead also is increased in children, making more lead biologically available in children. In children, 60% to 70% of lead is in the bone compared with 90% in adults. Additionally, the role of nutritional

status (eg, deficiency of iron, calcium, zinc, various vitamins) in modifying susceptibility to lead toxicity is an important factor in the comprehensive evaluation and treatment of children.

Lead is toxic to most body systems, including the peripheral and central nervous system, liver (it diminishes cytochrome P-450 activity), and hematopoietic and renal systems (affecting the metabolism of vitamin D and calcium). The toxicity of lead derives from its avidity for the sulfhydryl group of proteins, irreversibly binding and impairing function of enzymes in the pathway to heme synthesis. This inhibitory action includes lead's effect on delta-aminolevulinic acid dehydratase (ALA) in cytoplasm and ferrochelatase in mitochondria. ALA requires an intact sulfhydryl group and a zinc atom per subunit to be fully active, and lead is believed to displace zinc from the enzyme.

Since the early 1990s, accumulated data have provided evidence that toxic effects occur at levels below 10 mcg/dL (0.5 mcmol/L).

Effects on red blood cells can result in microcytic, hypochromic anemia and, rarely, in hemolysis and basophilic stippling. Elevations of ALA concentrations are likely to play a role in the neurotoxic effects of lead. Elevation of erythrocyte protoporphyrin (EP) results when the ferrochelatase enzyme is blocked; measurement of this compound was used for years in screening for lead poisoning. However, with the reduction of the value at which interventions are recommended to 10 mcg/dL (0.5 mcmol/L), EP no longer is a sensitive screening measure, and direct measurement of BLL is indicated.

The most severe sequelae of lead poisoning occur at levels of 70 mcg/dL (3.4 mcmol/L) and greater and can consist of death, coma, papilledema, ataxia, afebrile seizures, apathy, incoordination, and various changes in mental status. Other symptoms that occur at lower levels include anorexia, abdominal pain, vomiting, lethargy, and constipation. Fortunately, such presentations are rare in the United States because of the removal of lead from the manufacturing of gasoline and paint.

The blood level at which lead poisoning is defined has changed several times over the past decades. In 1991, in response to mounting evidence of the toxic effects of lead at levels as low as 10 mcg/dL (0.5 mcmol/L), especially those related to intelligence and neurodevelopment, the Centers for Disease Control and Prevention (CDC) redefined the "acceptable" threshold BLL at 10 mcg/dL (0.5 mcmol/L). Similarly, in 2000, the Secretary of Health and Human Services defined a new Healthy People 2010 objective: the elimination of BLLs of 10 mcg/dL (0.5 mcmol/L) and greater among children ages 1 to 6 years. However, since the early 1990s, accumulating data have provided evidence that toxic effects occur at levels below 10 mcg/dL (0.5 mcmol/L). One notable study (2) documented an inverse and significant relationship between BLL and IQ. In the investigators' linear model, an increase of 10 mcg/dL (0.5 mcmol/L) in the lifetime average BLL was associated with a 4.6-point decrease in IQ (P=.004); interestingly, however, for the subsample of children who had maximal BLLs less than 10 mcg/dL (0.5 mcmol/L), the associated decrease in IQ was greater. Others have documented cognitive and academic deficits associated with BLLs of less than 5 mcg/dL (0.24 mcmol/L) and documented effects on GABAergic and dopaminergic neurotransmission at these low levels. (3)(4)

Screening for Lead Exposure

Lead screening has evolved with the evolution of the epidemiology of lead exposure to ensure that screening meets the goal of identifying asymptomatic exposure for which interventions can prevent adverse effects. Understanding the epidemiology of lead poisoning in the United States and locally within communities is key to determining appropriate screening procedures. The CDC has used the National Health and Nutrition Examination Surveys (NHANES–1976 to 1980, 1988 to 1991, 1991 to 1994, 1999 to 2000) to track children's BLLs. State and local child blood lead surveillance data also have been helpful to these efforts. According to the 1999 to 2000 NHANES data, an estimated 434,000 children (95% confidence interval [CI] of 189,000 to 846,000), representing 2.2% of children 1 to 5 years of age, had BLLs greater than or equal to 10 mcg/dL (0.5 mcmolL). This point prevalence is a reduction from the numbers in 1976 to 1980, 1988 to 1991, and 1991 to 1994 of 88.2% (95% CI, 83.8 to 92.6), 8.6% (95% CI, 4.8 to 12.4), and 4.4% (95% CI, 2.9 to 6.6), respectively.

In 1991, the CDC lowered the intervention level for blood lead from 25 mcg/dL (1.21 mcmol/L) to 10 mcg/dL (0.5 mcmol/L), recommended the use of a five-question lead risk questionnaire, and advocated universal screening for children 9 to 72 months of age, except in communities that have had sufficient data to determine that their children were not at risk. Given that most communities did not have these data, this statement essentially recommended universal screening. The 1993 (and later 2000) American Academy of Pediatrics (AAP) Policy Statement mirrored these recommendations and called for "blood lead screening as part of routine health supervision for children at 9 through 12 months of age and, if possible, again at about 24 months of age."

The Early and Periodic Screening, Diagnosis and Treatment Program, which is designed to improve primary health care benefits for children through emphasizing preventive services as part of the federal Medicaid program, mandates lead screening according to the periodicity schedule set by the CDC (and consistent with AAP policy). Some studies also have shown that children may have elevated BLLs as early as 6 months of age, so annual risk assessment from 6 months to 6 years of age for risk of lead exposure is recommended. Screening before the age of 6 months may be indicated by the clinical history, including prenatal history of maternal lead exposure and occupational histories.

In 1997, the CDC updated its screening guidelines and incorporated, for the first time, screening policies that use local BLL data or housing data collected by the United States Bureau of Census. No longer was universal screening recommended in those areas where, because of limited exposure to lead, the prevalence of elevated BLLs is so low that targeted (selective) screening is more appropriate than universal screening. Instead, current guidelines state that health care professionals should use blood lead tests to screen children at ages 1 and 2 years and children 36 to 72 months of age who previously had not been screened if they met any of the following criteria: 1) child resides in a zip code that has 27% or more of houses built before 1950; 2) child receives benefits from public assistance programs such as Medicaid and Women, Infant, and Children; and 3) child's parent/guardian answers "yes" or "don't know" to any of these three risk assessment screening questions:

- Does your child live in or regularly visit a house or child-care facility built before 1950?
- Does your child live in or regularly visit a house or child care facility built before 1978 that is being or has recently been renovated or remodeled (within the last 6 months)?
- Does your child have a sibling or playmate who has or did have lead poisoning?

Targeted screening by these guidelines was recommended in communities where 1) fewer than 12% of children had BLLs of 10 mcg/d (0.5 mcmol/L) or greater or 2) less than 27% of houses were built before 1950. The revised guidelines also stated that in the absence of a statewide plan or guidance from officials, universal screening was indicated. The AAP policy statement published in 1998 was consistent with the CDC guidelines and also called for pediatricians to work with local health agencies to help develop risk assessment questionnaires to supplement the screening questions noted previously. They also called for managed care health organizations and third-party payers to cover the costs of screening and follow-up. The AAP statement recommended that screening also be considered for other high-risk children, including those who are abused and neglected.

Sensitivity and Specificity of Screening Questions

Following the revised 1991 CDC guidelines, the utility of the screening questions was raised. Although not intended to replace blood lead testing as a primary screening tool, many practitioners used the questions to target screening rather than to apply universal screening. Studies, therefore, were needed to examine how sensitive and specific the questions were in identifying at risk children. One study, (5) using the original five CDC questions from 1991 (Table 1), showed that using a positive answer to any one question to define a high-risk child, as suggested by the CDC, had a sensitivity for detecting BLLs of 10 mcg/dL (0.5 mcmol/L) or greater of nearly 90% and a specificity of 75%, with a negative predictive value of 99%. The individual questions had sensitivities in the 70% range and negative predictive values exceeding 90%, but low positive predictive values of less than 30%. A "yes" answer to the question regarding the presence of chipping paint or remodeling was significantly associated with elevated BLLs in those children. Other investigators have shown that in preschoolers, pica in general and ingestion of paint chips specifically are significantly associated with elevated BLLs.

In 1999, researchers in Illinois studied the use of a shorter questionnaire in the context of targeted screening. Screening questions similar to those recommended by the CDC had a 98% negative predictive value for BLLs

Table 1. 1991, Original Centers for Disease Control and Prevention Five-Question Lead Risk Questionnaire

Does your child–

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

greater than 10 mcg/dL (0.5 mcmol/L) among children living in so-called "low-risk" zip codes. Interestingly, the sensitivity and specificity for that approached 62% and 62%, respectively, compared with 75% and 88% associated with an Illinois approach that includes questions about the occupations and hobbies of families. Municipalities and researchers have used various iterations of questionnaires in assessing lead risk exposure. Care should be taken to review the reliability and validity of questionnaires that deviate greatly from those published.

Venous Lead Measurement

The 1998 AAP policy statement recommends the use of venous samples for initial screening when possible. As per these guidelines, a capillary micro-lead measurement could be substituted, but levels above 10 mcg/dL (0.5 mcmol/L) must be confirmed by a venous sample because micro-lead sampling is more likely to yield false-positive results due to contamination from environmental lead. The policy urged use of laboratories that participate in a proficiency program; a list of such programs can be obtained from the CDC. The use of a portable instrument for analysis of blood lead has been reported.

Treatment

Table 2 details the medical management of children based on BLLs. Management of lead-poisoned children should focus on nutritional and environmental intervention; any treatment that does not include environmental intervention is considered inadequate. Pediatricians should refer affected children to local public health offices for environmental assessment of their residences. Treatment of lead poisoning traditionally has been guided by the BLL and the risk of the acute neurotoxic effects of extreme elevations of blood lead. Chelation therapy is the mainstay of treatment for BLLs greater than 45 mcg/dL (2.2 mcmol/L) and should be performed in consultation with a pediatrician experienced in managing children who have lead poisoning. Table 3 highlights the major chelating agents, dosages, indications for treatment, and adverse effects. The toxicity of the chelating agents and the safety concern of redistribution of lead from bone to soft tissue, especially the brain, has guided therapeutic interventions. Although treatment has been shown to decrease the BLL acutely, rebound levels have been documented with all treatments, and the impact of treatment on neurodevelopmental outcomes for all but the most symptomatic children remains unclear. Prevention of lead poisoning, therefore, remains the most effective method of averting the negative effects of toxicity.

Advocacy Perspectives

Despite the gains of the past decades, recent reports have documented the significant and persistent prevalence of lead-based paint hazards in American homes. According to the latest report from the United States Department of Housing and Urban Development (HUD), 24 million homes in America have hazards that could place children at risk for lead poisoning. Lead-based paint hazards were especially prevalent in housing older than 1978, and more than 30% of housing units that had lead-based paint contained hazards even when the paint was in good condition.

As noted previously, leaders in federal and state government have recognized the importance of eradicating lead poisoning through an approach that emphasizes primary prevention with continued screening of high-risk populations. In 2000, the Secretary of Health and Human Services serving on a Presidential Task Force on Environmental Health Risks and Safety Risks to Children recommended a $230 million annual investment over 10 years in HUD's Lead Hazard Control Grant Program to eliminate lead poisoning. Despite this recommendation, it now appears unlikely that the federal government will eliminate lead poisoning by the year 2010. Federal funding of the Lead Hazard Control Grant Program has only increased from $60 million in fiscal year 2000 to $140 million in fiscal year 2003.

Table 2. Medical Management of Children Based on Blood Lead Levels (BLLs)

Blood Lead Level mcg/dL (mcmol/L)	Recommended Action*
<10 (0.5) Risk Level: I	• Obtain a careful environmental history • Provide risk reduction and nutrition education • If risk assessment indicates exposure to lead is likely, consider retesting within 3 months
10 through 14 (0.5 through 0.68) Risk Level: II Moderate	• Report BLL to local Department of Health (refer to local laws) • Obtain a careful environmental history • Provide risk reduction and nutrition education • Repeat all capillary samples, confirming with a venous sample within 1 month for new cases and 1 to 3 months for known cases
15 through 19 (0.69 through 0.92) Risk Level: II Moderate	• Follow steps from BLL 10 through 14 mcg/dL (0.5 through 0.68 mcmol/L) • If BLL remains 15 through 19 mcg/dL (0.69 through 0.92 mcmol/L) for 3 months, proceed with actions for BLL 20 through 44 mcg/dL (0.93 through 2.1 mcmol/L) • Collaborate with lead poisoning prevention programs (LPPP), which will provide home inspection and other services • If initial sample was capillary, repeat with a venous sample in 1 week to 1 month. The higher the BLL, the more urgent.
20 through 44 (0.93 through 2.1) Risk Level: III High	• Follow steps for child who has BLL of 10 through 14 mcg/dL (0.5 through .68 mcmol/L) • Provide complete medical evaluation, including detailed environmental history, developmental assessment, physical examination, and evaluation for iron deficiency anemia. If particulate ingestion is suspected, obtain abdominal radiograph and order bowel decontamination if indicated • Consider chelation therapy (not currently recommended for BBL <45 mcg/dL [2.2 mcmol/L]) in consultation with a clinician experienced in lead toxicity treatment • Collaborate with LPPP, which will provide home inspection and other services
45 through 69 (2.2 through 3.3) Risk Level: IV Urgent	• Confirm BLL with venous sample within 24 to 48 hours before initiating chelation • Provide or refer for chelation therapy within 48 hours. Child must be in a lead-safe environment during chelation • Follow all steps for BLL of 20 through 44 mcg/dL (0.93 through 2.1 mcmol/L) • Perform complete neurologic examination and consider free erythrocyte (FEP) or zinc protoporphyrin (ZPP) testing to assist in evaluating child's response to management
≥70 (3.4 mcmol/L) Risk Level: V (Emergency)	• Arrange immediate hospitalization and chelation at a facility that has expertise in treating lead-poisoned children. Assistance can be obtained from some LPPP Medical Directors or Poison Control • Confirm BLL immediately with venous sample processed as an emergency laboratory test • Follow all steps for BLL 20 through 44 mcg/dL (0.93 through 2.1 mcmol/L) • Perform complete neurologic examination and consider FEP or ZPP testing to assist in evaluating child's response to management

*Adapted from the recommendations in Centers for Disease Control and Prevention. *Managing Elevated Blood Lead Levels Among Young Children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention*. Atlanta, Ga: Centers for Disease Control and Prevention; 2002, which is available at www.cdc.gov/nceh/lead/CaseManagement/CaseManage_main.htm, and the American Academy of Pediatrics. Screening for elevated blood lead levels. *Pediatrics*. 1998;101:1072–1078.

Often persuaded by economic arguments, policy makers should be informed that lead exposure in the American population exacts an estimated $40 billion annual toll from children in the form of loss in economic productivity. Children's environmental health is among parents' top concerns. To produce proactive responses and regulation to protect children, pediatricians must be aware of environmental risks such as those posed by lead poisoning and advocate strongly to create policies that support effective primary prevention.

Table 3. **Lead Chelating Agents**

Agent	Recommended Dose	Indication	Adverse Effects	Contraindications and Recommended Monitoring
Dimercaprol (also known as dimercaptopropanol, British Anti-lewisite, BAL, in oil)	25 mg/kg per day IM divided in six doses for a minimum of 72 hours. (First dose of BAL is given 4 hours prior to administration of $CaNa_2EDTA$)	BLL ≥70 mcg/dL (3.4 mcmol/L) or with clinical symptoms of encephalopathy	Pain at the injection site; hemolysis in glucose-6-phosphatase (G6PD)-deficient patients; toxic complex formation if given with iron; nausea, vomiting, hypertension, fever, lacrimation, and paresthesias	Should be withheld in the presence of hepatic disease. Peanut allergy. Close monitoring of cardiovascular and mental status required.
Calcium Disodium Ethylenediaminetetra-acetic acid (EDTA)	50 mg/kg per day as a single dose infused over several hours or as a continuous infusion is safe, diluted to a concentration not greater than 0.5% in dextrose and water or 0.9% saline solution (maximum dose, 1,000 mg). IM $CaNa_2EDTA$ should be given with 0.5% procaine to decrease the pain	BLL ≥45 mcg/dL (2.2 mcmol/L)	Proteinuria, hematuria, casts, hypercalcemia, fever, chills, malaise, thirst, nausea, vomiting, and hypokalemia	Should not be used in anuric patients. Hydration, careful monitoring of electrolytes, blood urea nitrogen/creatinine, calcium, phosphorus, urinalysis, and for cardiac arrhythmias are indicated
Succimer (2,3-mesodimercaptosuccinic acid [DMSA])	10 mg/kg PO every 8 hours for 5 days, followed by 10 mg/kg every 12 hours for 14 days (maximum dose, 1,500 mg/d)	BLL ≥45 mcg/dL (2.2 mcmol/L)	Mild gastrointestinal symptoms, general malaise, transient elevation of liver enzymes, decreased hemoglobin levels, and reversible neutropenia. Hypersensitivity reactions	Consider following liver function tests and complete blood count. Unlike dimercaprol, G6PD deficiency is not a contraindication.
D-Penicillamine	100 mg/kg per day to 20 mg/kg per day PO for 4 to 12 weeks (empirically derived)	Elevated BLL ≥45 mcg/dL (2.2 mcmol/L), but not labeled specifically for treatment of lead	Reversible leukopenia, mild thrombocytopenia (occurs in about 10% of treated children), eosinophilia, angioedema, urticaria, maculopapular eruptions, nausea, vomiting, proteinuria, microscopic hematuria, and urinary incontinence	Hypersensitivity reactions

*References for further details of the mechanism of action and treatment regimen:

A Summary of Pharmacological and Clinical Data: Chemet® Succimer Capsule 100 mg. Fort Washington, Pa: McNeil Consumer Productions Company; 1991

American Academy of Pediatrics Committee on Drugs. Treatment guidelines for lead exposure in children. *Pediatrics.* 1995;96:155–160

Centers for Disease Control and Prevention. *Managing Elevated Blood Level Levels Among Young Children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention.* Atlanta, Ga: Centers for Disease Control and Prevention; 2002

Friedheim E, Graziano JH, Popovac D, Dragovic D, Kaul B. Treatment of lead poisoning by 2,3-dimercaptosuccinic acid. *Lancet.* 1978;2:1234–1236

Graziano JH, Lolacono NH, Meyer P. Dose-response study of oral 2,3-dimercaptosuccinic acid in children with elevated blood lead concentrations. *J Pediatr.* 1988;113:751–757

Piomelli S, Rosen JF, Chisolm JJ Jr, Graef JW. Management of childhood lead poisoning. *J Pediatr.* 1984;105:523–532

References

1. Jacobs DE, Clickner RP, Zhou JY, et al. The prevalence of lead-based paint hazards in U.S. housing. *Environ Health Perspect.* 2002;110:A599–A606

2. Canfield RL, Henderson CR Jr, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual impairment in children with blood lead concentrations below 10 micrograms per deciliter. *N Engl J Med.* 2003;348:1517–1526

3. Lanphear BP, Dietrich K, Auinger P, Cox C. Cognitive deficits associated with blood lead concentrations <10 microg/dL in US children and adolescents. *Public Health Rep.* 2000;115:521–529

4. Schwartz J. Low-level lead exposure and children's IQ: a meta-analysis and search for a threshold. *Environ Res.* 1994;65:42–55

5. Tejeda DM, Wyatt DD, Rostek BR, Solomon WB. Do questions about lead exposure predict elevated lead levels? *Pediatrics.* 1994; 93:192–194

Suggested Reading

Binns H. Evaluation of risk assessment questions used to target blood lead screening in Illinois. *Pediatrics.* 1999;103:100–106

Centers for Disease Control and Prevention. *Managing Elevated Blood Lead Levels Among Young Children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention*. Atlanta, Ga: Centers for Disease Control and Prevention; 2002

Gibson J. A plea for painted railings and painted walls of rooms as the source of lead poisoning amongst Queensland children. *Australas Med Gaz.* 1904;23:149–153

Kemper AR, Cohn LM, Fant KE, Dombrowski KJ, Hudson SR. Follow-up testing among children with elevated screening blood lead levels. *JAMA.* 2005;293:2232–2237

Lanphear BP. Childhood lead poisoning prevention: too little too late [editorial]. *JAMA.* 2005;293:2274–2276

Lanphear BP, Wright RO, Dietrich KN. Environmental neurotoxins. *Pediat Rev.* 2005;28:191–198

Needleman HL. Childhood lead poisoning: a disease for the history texts. *Am J Public Health.* 1991;81:685–687

Neuspiel DR. Lead levels under 10 micrograms per deciliter are hazardous. *AAP Grand Rounds.* 2003;10(1):1–2. Commentary on Canfield RL, Henderson CR Jr, Cory-Slechta DA, et al. Intellectual impairment in children with blood lead concentrations below 10 mcg per deciliter. *N Engl J Med.* 2003;348: 1517–1526

Screening for elevated blood lead levels. American Academy of Pediatrics Committee on Environmental Health. *Pediatrics.* 1998;101:1072–1078

Treatment guidelines for lead exposure in children. American Academy of Pediatrics Committee on Drugs. *Pediatrics.* 1995;96: 155–160

PIR Quiz

Quiz also available online at www.pedsinreview.org.

1. Lead has been used in a variety of products for many years because of its durability and ability to form stable compounds. As a result, a large amount of lead has accumulated in the environment, exposing many children to this toxic compound. Which source is the *most* likely to cause lead toxicity in a child?

   A. Burning of batteries.
   B. Lead-based paint.
   C. Leaded gasoline.
   D. Lead pipes.
   E. Teething powders.

2. A number of tests have been used to detect children exposed to lead. The test that remains the *most* reliable for detecting lead poisoning is:

   A. Blood smear revealing basophilic stippling.
   B. Capillary blood lead level.
   C. Erythrocyte protoporphyrin level.
   D. Long-bone films for lead lines.
   E. Venous blood lead level.

3. In communities that have a reduced risk of lead poisoning, public health officials have recommended the use of screening questionnaires to determine whether a blood lead screening is necessary for an individual child. The *most* useful screening item in detecting exposed children is:

   A. How close they live to a freeway.
   B. Whether the child has pica.
   C. Whether the family uses pottery for serving meals.
   D. Whether the home has chipping paint.
   E. Whether the home was built before 1980.

4. A 2-year-old child visits his grandparents' antique shop while his mother is at work. The boy has become tired, pale, and constipated. Screening blood tests reveal a hemoglobin level of 8.5 g/dL (85 g/L). A blood smear demonstrates a hypochromic microcytic smear. The grandparents report that these symptoms began during the past 6 months since a shooting gallery moved in next door to their antique shop. You suspect that this child is suffering from lead poisoning. What blood lead level requires immediate intervention with hospitalization and chelation therapy?

   A. 30 mcg/dL (1.5 mcmol/L).
   B. 40 mcg/dL (1.9 mcmol/L).
   C. 50 mcg/dL (2.4 mcmol/L).
   D. 60 mcg/dL (2.9 mcmol/L).
   E. 70 mcg/dL (3.4 mcmol.L).

Lead Poisoning : Successes and 21st Century Challenges

Danielle Laraque and Leonardo Trasande

Pediatrics in Review 2005;26;435
DOI: 10.1542/pir.26-12-435