

LEARNING OBJECTIVES

- Describe the epidemiology of lead poisoning, especially in children
- Explain the neurologic, cognitive, and other manifestations associated with lead exposure
- Outline the clinical evaluation of and screening guidelines for children exposed to lead
- Review the management and treatment of lead poisoning

Working to prevent lead poisoning in children: Getting the lead out

Despite clean environment policies and safe housing initiatives, lead poisoning is still a serious health threat. PAs need to know how to recognize lead toxicity, especially in children.

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Lead is a naturally occurring metal that has no known biological role in the body. It is a common environmental contaminant and a neurotoxin associated with impaired cognitive, motor, developmental, and behavior skills in children. Lead poisoning is often insidious and asymptomatic, and it frequently goes unrecognized. The *Healthy People 2010* objectives for the nation recommend eliminating blood lead levels (BLLs) of 10 µg/dL or higher by 2010.¹ However, no safe threshold has been determined for the potentially harmful effects on children.

EPIDEMIOLOGY

Environmental lead exposure and poisoning are preventable. Unfortunately, they remain a serious public health risk, and children are the most vulnerable. Although mortality is low because of screening and the aggressive use of chelating agents, morbidity is still significant.²

Most exposures to lead are caused by inorganic lead entering the body through inhalation of dust or fumes or swallowing when eating or drinking. Lead can also be absorbed transdermally, although this is not a common route of exposure for children in a household setting. Industrial exposure, where lead dust accumulates on clothing, hair, and skin; pica; use of folk remedies and imported cosmetics, such as kohl; or use of imported ceramic serving dishes can increase the risks. Lead poisoning in women can be transferred to the fetus in utero because lead crosses the placenta; it can also be transferred through breast-feeding. Lead that is not excreted initially is distributed into the blood, soft tissues, and bones.

Children aged 9 months to 6 years are at greatest risk of lead exposure. Children have more hand-to-mouth behaviors,



A swab test shows this painted surface is positive for lead.

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and rapid growth and absorption make them more susceptible than adults to the effects of lead. Children who live in older substandard housing in urban areas—particularly houses built before 1978, when lead-based paint was banned—are at greatest risk. In addition, older homes are more likely to have leaded pipes, brass fittings and solder, and vinyl mini-blinds, all of which contain lead. Chipping and peeling paint can contaminate dust and soil. Living and playing near industrial sites also expose children to lead contaminants. Despite measures to reduce housing-related lead exposure, lead-paint hazards are present in an estimated 24 million homes, 1.2 million of which are occupied by low-income families with children younger than 6 years.³ Children living in poverty are also more likely to have poor diets, further placing them at risk. One randomized community-based trial found no significant difference in children's BLLs at the end of the year, even with intensive case management that includes home visits and education. These findings suggest that safe housing, rather than environmental exposure reduction, is necessary once the BLLs are elevated.⁴

Adults with jobs or hobbies that use lead can put the children who live with them at risk. Nationally, 95% of adult lead exposures are occupational,⁵ and exposed adults are predominantly men. Occupations such as painting, housing renovation, automotive repair, ceramic production, bridge construction and repair, jewelry making, building demolition, scrap metal recovery, and foundry operations may expose persons to lead products. Certain avocations, such as furniture stripping, stained-glass making, welding, making bullets, use of firearms in an indoor firing range, and making or using fishing sinkers, also pose a risk.

The CDC tracks BLLs in children through the National Health and Nutrition Examination Survey and surveillance data from state and local health departments. BLLs in children have declined considerably, largely as a result of education and policies that curtail lead contamination in the environment, such as the elimination of leaded gasoline in the United States and safe housing initiatives.^{5,7} BLLs of 40 $\mu\text{g}/\text{dL}$ were not unusual in the 1970s. Currently, average BLLs range from 2 to 4 $\mu\text{g}/\text{dL}$.⁸

Overall, the prevalence of elevated BLL in the US population aged 1 year and older was 0.7% for 1999-2002, 68% lower than the prevalence reported in the 1991-1994 survey.⁶ During the 1999-2002 survey, prevalence of elevated BLL was highest (1.6%) among children aged 1 to 5 years. This indicates approximately 310,000 US children aged 1 to 5 years had a BLL higher than 10 $\mu\text{g}/\text{dL}$, the level at which the CDC recommends intervention and the American Academy of Pediatrics (AAP) defines lead poisoning.² African-American children may be disproportionately affected because of socioeconomic conditions. Although surveys have found a lower prevalence of elevated BLL among black non-Hispanic children associated with more recently built homes,

“BLL increases quickly after acute exposure, gradually reaching equilibrium. Once elevated, it takes months to years to decline.”

these children remain at greater risk of developing lead poisoning. During 1999-2002, a higher percentage of non-Hispanic blacks and Mexican Americans had an elevated BLL (1.4% and 1.5%, respectively) than did non-Hispanic whites (0.5%).⁶ Among children aged 1 to 5 years, non-Hispanic blacks had the highest prevalence of elevated BLL (3.1%). Intensified efforts are needed to meet the 2010 goal of lowering BLLs to less than 10 $\mu\text{g}/\text{dL}$.

NEUROLOGIC AND COGNITIVE EFFECTS

The CDC Advisory Committee on Childhood Lead Poisoning Prevention reviewed the scientific literature regarding the adverse health effects associated with a BLL below 10 $\mu\text{g}/\text{dL}$.⁷ This review included 23 published reports on intelligence quotient (IQ) and cognitive index outcomes and 12 publications related to other outcomes. The Committee found an inverse association between BLL and cognitive

KEY POINTS

- Most exposures to lead are caused by inorganic lead entering the body through inhalation of dust or fumes or swallowing when eating or drinking. Lead can also be absorbed transdermally, although this is not a common route of exposure for children in a household setting.
- Despite measures to reduce housing-related lead exposure, lead-paint hazards are present in an estimated 24 million homes, 1.2 million of which are occupied by low-income families with children younger than 6 years. Children living in poverty are also more likely to have poor diets, further placing them at risk.
- The CDC tracks blood lead levels (BLLs) in children through the National Health and Nutrition Examination Survey and surveillance data from state and local health departments. BLLs in children have declined considerably, largely as a result of education and policies that curtail lead contamination in the environment, such as the elimination of leaded gasoline in the United States and safe housing initiatives. BLLs of 40 $\mu\text{g}/\text{dL}$ were not unusual in the 1970s. Currently, average BLLs range from 2 to 4 $\mu\text{g}/\text{dL}$.
- Screening guidelines increasingly focus on targeted rather than universal screening. Both the CDC and the American Academy of Pediatrics recommend that health care providers administer BLL tests to children enrolled in or eligible for Medicaid and to those identified to be at risk according to state and local screening or risk-assessment measures.

function, with no evidence of a weaker association in populations with lower levels of elevated BLL. Thus, the Committee concluded a causal relationship between lead exposure and impaired cognitive function was likely.

In one prospective study published in 2008, children were followed from age 6 months to 6 years, and IQ was assessed based on the Wechsler Preschool and Primary Scale of Intelligence-Revised.⁹ Compared with children with a BLL below 5 µg/dL, those with a BLL of 5.5 to 9.9 µg/dL scored 4.9 points lower on the full-scale IQ test. Thus, findings from this cohort study suggest intellectual function can be impaired at concentrations below the current defined level for elevated BLL. This is consistent with an earlier study published in 2003 that found a 4.6-point reduction in IQ at ages 3 and 5 years for every 10 µg/dL increase in average BLL concentration.¹⁰ The effects were even greater for a given change in lead concentration in children with a BLL below 10 µg/dL than in children with a higher BLL. A 2005 international pooled analysis study also found an association between a change in lead concentration and intellectual deficits among children with a BLL below 7.5 µg/dL.¹¹ In addition, abnormalities in verbal comprehension and expression and auditory abilities can occur.⁵ A study that examined the relationship between exposure to tobacco smoke and exposure

to environmental lead concluded that both are risk factors for attention-deficit/hyperactivity disorder in children.¹² Other studies have suggested long-term negative health outcomes, such as teen pregnancy and tobacco use, may be associated with childhood lead poisoning.¹³

OTHER MANIFESTATIONS OF LEAD TOXICITY AND POISONING

Lead can affect any organ in the body. The effects on the nervous system, kidneys, and bones are the most well-known; however, the cardiac, hemopoietic, GI, and reproductive systems can also be affected. BLL increases quickly after acute exposure, gradually reaching equilibrium. Once elevated, BLL takes months to years to decline.

Children are affected at lower levels than adults, and effects may be more significant at higher levels. Acute high-level exposure may lead to lethargy, seizures, and death or significant damage to vital organs, including the brain, kidneys, and heart. Lead can affect any part of the CNS and peripheral nervous system, and manifestations vary based on the duration and level of exposure.⁵ Whereas adults may be more likely to develop a peripheral motor neuropathy, children may be more likely to experience encephalopathy caused by lead intoxication, which can be irreversible or fatal. A 4-year-

TABLE 1. Lead risk assessment questionnaire

Instructions: Administer to parents/caregivers of children 6 years or younger who are not already targeted for screening. A “yes” or “don’t know” response to any question indicates the child is at risk for lead exposure and should receive a blood lead test and follow-up evaluation.

1. Does your child live in or regularly visit (once a week or more) a house or building built before 1978?
2. Does your child live in or regularly visit a house or building that has recently undergone renovation?
3. Does your child frequently come into contact with an adult whose job or hobby involves exposure to lead or have a playmate or sibling with an elevated blood lead level?
4. Does your child have contact with cosmetics, kohl, candies, spices, jewelry, ceramic dishes, or folk remedies manufactured outside the United States?
5. Does your child play in soil near a busy road or an industrial site, such as a battery recycling plant, junkyard, or lead smelter?
6. Have you seen your child eat dirt or place his or her mouth on painted surfaces, paint chips, toys, jewelry, or vinyl mini-blinds?
7. Has your child visited or lived in another country for an extended period of time?

Adapted with permission from the Florida Department of Health, Childhood Lead Poisoning Prevention Program. *Childhood Lead Poisoning: Screening & Case Management Guide*. Adopted January 2008. Tallahassee, FL: Office of the State Surgeon General; 2008.

TABLE 2. Management guidelines for elevated BLL

BLL <10 µg/dL: No action is required.

BLL 10-14 µg/dL: Obtain confirmatory test within 1 mo. If still in this range, provide lead education and repeat test in 3 mo.

BLL 15-19 µg/dL: Same as for BLL 10-14 µg/dL, but repeat within 2 mo. If the level increases or is still in this range after 3 mo, proceed according to protocol for BLL 20-44 µg/dL.

BLL 20-44 µg/dL: Obtain confirmatory BLL within 1 wk to 1 mo (sooner with higher levels). If still in this range, undertake comprehensive medical (history, physical examination for neurodevelopment monitoring, and laboratory evaluation [hemoglobin or hematocrit and iron level]), nutritional, and environmental assessment. Lead hazard reduction is implemented with the assistance of the local health department.

BLL 45-69 µg/dL: Obtain a confirmatory BLL within 2 d. If still in this range, perform assessment and take actions as indicated for BLL 20-44 µg/dL; also obtain EP level. Chelation therapy is recommended at this level. If unable to do this in a lead-free environment, hospitalization is indicated. Monitor CBC, electrolytes, and liver function.

BLL >70 µg/dL: Hospitalize and obtain confirmatory BLL, initiate chelation therapy, and implement actions as for BLL 45-69 µg/dL.

Key: BLL, blood lead level; EP, erythrocyte protoporphyrin.
Data from Badawy MK and Connors GP.² American Academy of Pediatrics Committee on Environmental Health,¹⁷ and Centers for Disease Control and Prevention.²¹

old boy in Minnesota died from encephalopathy caused by acute lead poisoning. He had swallowed a heart-shaped metallic charm that contained lead; his BLL was 180 µg/dL.¹⁴

Acute substantial lead exposure is associated with anemia, neuropathy, and Fanconi-like syndrome. Chronic lead nephropathy can lead to the progressive development of interstitial nephritis, resulting in hypertension and gout. Lead and calcium can be used interchangeably by bone; therefore, lead can replace calcium depositions in growing bone, especially in the metaphysis of the rapidly growing bones, such as the femur, tibia, and distal radius. On radiographs, this appears as opaque lead lines.

CLINICAL EVALUATION

Screening guidelines As the prevalence of elevated BLL has decreased, screening guidelines have increasingly focused on targeted rather than universal screening. Both the CDC and AAP recommend that health care providers test BLLs of children enrolled in or eligible for Medicaid and of those identified to be at risk according to state and local screening or risk assessment measures.¹⁵⁻¹⁷ Available state and local screening plans can be found at the CDC Web site (www.cdc.gov/nceh/lead/grants/contacts/CLPPP%20Map.htm).

All children aged 6 months to 6 years should be assessed periodically for possible lead exposure. Because many children are asymptomatic, a thorough home, dietary, environmental, and developmental history is necessary to identify those children at risk. Environmental exposures include living in or regularly visiting a house built before 1950 or, if undergoing renovation, before 1978; living in a community with a high or unknown prevalence of elevated BLL; near a lead industry or heavy traffic; or in a household with an adult with occupation- or hobby-related exposure to lead. Behaviors that may increase lead exposure include hand-to-mouth activity; pica; and use of imported folk remedies, candies, and ceramics. Careful assessment of developmental history can document any delays or deficits in achieving milestones. Assessing diet is important because lead absorption is enhanced by a high-fat diet, particularly when calcium and iron are deficient. Foster, immigrant, refugee, and foreign-born children also should be screened. In addition, nonspecific symptoms such as constipation, fatigue, nausea, headache, and abdominal pain or discomfort should elicit further assessment² (see Table 1).

BLL screening is recommended for children at risk at age 12 months, with retesting at age 24 months. Screening is mandated for children enrolled in Medicaid at ages 12 and 24 months, and at age 36 to 72 months if the child was not previously screened.^{15,16,18} Typically in children exposed to lead, BLL peaks at age 2 years.^{8,18} However, one retrospective case control study found that children at risk who have a BLL of 7 to 9 µg/dL before age 2 years were more likely to have lead poisoning at repeat testing after age 3 years.¹⁹

Although a woman of childbearing age who has a high BLL can transfer lead to her unborn child, no national organizations currently recommend screening pregnant women.¹⁵ Local data and conditions, along with individual

“Abdominal radiographs can be used to identify foreign objects, providing an opportunity to prevent further lead absorption.”

risk assessment, should be used to determine if lead screening is appropriate for a pregnant woman.

Physical examination Physical signs may be absent, subtle, or nonspecific. Skin pallor may be caused by anemia, and in some cases, pyorrhea may be evident. Growth failure; hearing loss; or speech, language, and attention deficits (inattentiveness, impulsiveness) may be evident. The child's activity level can be hyperactive or lethargic. Chronic exposure can result in distal motor neuropathies with weakened extensor muscles and diminished reflexes. Sensory function usually remains intact. Any child presenting with signs of increased intracranial pressure, such as impaired consciousness, bradycardia, hypertension, respiratory depression, papilledema or coma, and unexplained and prolonged gastric symptoms, such as vomiting, should be evaluated for lead poisoning.¹⁴

Diagnostic tests Whether to order laboratory tests for lead poisoning depends on the presenting circumstances. In most children, lead poisoning is found only on screening. Parental barriers to BLL testing can be reduced with appropriate education on the causes and effects of lead poisoning.²⁰

BLL screening can be done initially via capillary blood draw. However, since contamination can occur during collection, any capillary specimens that test positive must be confirmed by testing a standard venous whole blood specimen, usually within 1 week to 3 months depending on the BLL. Although federal standards permit a degree of error of 4 µg/dL +/-10%, clinicians should use laboratories that achieve a +/-2 µg/dL performance at levels below 10 µg/dL.⁷ BLL can vary by season, with levels being higher in the summer because of increased exposure. Erythrocyte protoporphyrin levels may be indicated in select patients (BLL 55 µg/dL or higher) to confirm and manage an elevated lead level.² Hair sample analysis for lead levels is less sensitive than blood specimens and is not recommended.

In acute intoxications, laboratory analysis should include a glucose level, CBC, electrolytes, urinalysis, and a chemistry panel. ECG may demonstrate cardiac conduction disturbances. Chronic low-dose lead exposure can cause increased hemolysis resulting in iron deficiency and microcytic anemia, with a compensatory increase in the number of RBCs.

Neuropsychologic testing of children with elevated BLL is the best indicator of the level of cognitive dysfunction. These tests can document therapy-related improvements in attention, visual-spatial abnormalities, and memory.⁵

Plain long-bone radiographs may be useful in identifying opaque lead lines that occur with chronic exposure and a

CASE STUDY: AN ENVIRONMENTAL EXPOSURE

A 12-month-old Hispanic girl was evaluated in May 2005 at a County Health Department (CHD) clinic in a routine well-child checkup. The child was found to have an elevated blood lead level (BLL). Initial capillary BLL was 39 $\mu\text{g}/\text{dL}$, and confirmatory venous BLL was 26 $\mu\text{g}/\text{dL}$. The child was referred to the CHD Childhood Lead Poisoning Prevention Program for evaluation. A home visit and environmental investigation for lead exposure followed.

Medical history The child had no significant medical history. Her parents denied any symptoms or signs associated with lead poisoning. The girl had a good appetite and ate a well-balanced diet, with no use of supplemental vitamins or routine medication.

Housing The child resided in a rental home with her parents; no other children or pets lived with them. The home was a detached apartment, built before 1930. It was not located near a major highway or any lead-producing industries; however, nearby buildings were undergoing renovation. The home contained older plumbing, but the family primarily used bottled water.

Parental occupational history The father's work involved renovating older homes, and the child frequently visited his work site. His work clothes were laundered separately from the household laundry, and he removed his shoes before entering the home.

Nontraditional sources The parents denied use of home remedies, imported spices, or foreign candies but acknowledged using imported candles. Samples of the candles were negative for significant lead content. The parents had observed the child sucking her thumb and putting nonfood items such as paint chips in her mouth.

Potential exposure sources Lead hazards were identified on windows with peeling lead paint and areas of bare soil adjacent to the windows. The parents were advised to refrain from stripping the paint, to mulch areas with bare soil, to keep the child away from the father's work site, and to implement good hand washing and housekeeping practices.

Medical intervention The child's venous BLL was 33 $\mu\text{g}/\text{dL}$ 1 month after the environmental inspection, and 6 weeks later, it was 46 $\mu\text{g}/\text{dL}$. The increasing BLL prompted hospitalization and administration of succimer chelation therapy. At discharge, a few days later, the BLL was 34 $\mu\text{g}/\text{dL}$. Subsequent BLL monitoring over the next 3 years showed a continuous decline, and the BLL drawn in February 2009 was 6 $\mu\text{g}/\text{dL}$.

Acknowledgement: This case was contributed by Dr. Aaron Hilliard and Dr Tiffany Turner, Duval County Health Department Childhood Lead Prevention Program, Jacksonville, Florida.

BLL of 70 to 80 $\mu\text{g}/\text{dL}$; however, they are not recommended by the AAP.¹⁷ Abdominal radiographs can be ordered selectively to identify foreign objects, such as paint chips, providing an opportunity for bowel decontamination therapy to prevent further lead absorption. Selective use of neuroimaging studies, such as CT and MRI, can demonstrate cerebral edema and microhemorrhages and exclude structural lesions in children with an altered mental status suspected to be caused by lead toxicity.

MANAGEMENT AND TREATMENT

Children with a confirmed BLL higher than 10 $\mu\text{g}/\text{dL}$ require follow-up testing.^{2,17,21} A home evaluation should be performed if a child has a BLL higher than 15 to 19 $\mu\text{g}/\text{dL}$ for 3 months or more or has an initial BLL higher than 20 $\mu\text{g}/\text{dL}$ to determine the source of the lead exposure. Other members of the household should also be tested. Chelating agents are the standard treatment for acute and high levels of lead toxicity. Children with a BLL higher than 45 $\mu\text{g}/\text{dL}$ should be treated with succimer. A lead level higher than 70 $\mu\text{g}/\text{dL}$ is a medical emergency. Children with lead encephalopathy are best treated in a children's hospital with pediatric intensivists and other specialized resources (see Table 2, page 42).

PREVENTION AND PATIENT EDUCATION STRATEGIES

The Lead Contamination Control Act of 1988 gave the CDC authority to establish programs to prevent and eliminate childhood lead poisoning.²² As a result of this legislation, nearly 60 childhood lead-poisoning prevention programs have been established, state and national screening surveillance for lead has been enhanced, and targeted screening and case management guidelines for children with elevated BLL have been developed.

PAs should familiarize themselves with the available resources in their community and state. Anticipatory guidance should be provided to the parents and caregivers of young children, including identifying sources of lead in the child's environment and lead-prevention counseling.⁶ Community-specific risk-assessment questionnaires and appropriate BLL screening will help identify children at risk. Timely referrals to developmental and early enrichment programs are critical in establishing a lead-safe environment. So are agencies and community resources, such as local health departments, that provide on-site home and environmental assessments, targeted risk-reduction strategies, and ongoing case management for children with elevated BLL (see "Case study: An environmental exposure").

Primary care clinicians are often the first line of defense in the efforts to prevent and eliminate childhood lead poisoning. A thorough risk assessment, targeted screening, appropriate caregiver education, and prompt referral can help eliminate the effects of lead exposure in children. **JAAPA**

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Postsplenectomy infection

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DRUGS MENTIONED

Amoxicillin/clavulanate (Augmentin)	Penicillin
Ceftriaxone (Rocephin, generics)	Penicillin V potassium
Cefuroxime axetil (Ceftin, Zinacef)	Vancomycin (Vancocin, generics)
Moxifloxacin (Avelox)	

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