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Lead Poisoning: Basics and New Developments

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Author Disclosure
Drs Chandran and Cataldo have disclosed no financial relationships relevant to this article. This commentary does contain a discussion of an unapproved/investigative use of a commercial product/device.

Objectives After completing this article, readers should be able to:

1. Delineate the disparities of pediatric lead poisoning among socioeconomic and racial groups.
2. List three common sources of lead.
3. Describe the emerging data on immediate and long-term neurotoxic effects of lead on the developing brain.
4. Discuss two chelating agents and their common adverse effects.
5. Employ a multipronged, multidisciplinary approach in the overall management of pediatric lead poisoning.

History

For many centuries, starting as early as 4000 BC, lead has been used for a variety of purposes. Ancient Romans used lead for glazing pottery, piping, cooking utensils, and sweetening of wine. Lead toxicities were well documented in Egyptian papyrus rolls, describing its use for homicidal purposes. Over the centuries, lead poisoning was noted by different terms such as “the miner’s disease,” “lead blindness,” “lead colic,” “lead gout,” and “plumbism.” It was a common cause of morbidity and mortality among shipbuilders, wine drinkers, and potters. Lead encephalopathy and lead psychosis also were recognized early in human history, when potters were described as “paralytic, splenetic, lethargic, cachectic, and toothless.” The use of lead became widespread during the industrial revolution. Use of lead-based paints, gasoline, and food containers resulted in profound environmental contamination. The toxic clinical effects of lead poisoning in children were linked to lead-based paint used in the early 20th century. More than half of the homes built in the United States before 1950 contained lead paint. Lead-based paints were banned in the United States in 1977 (the maximum allowable amount is now 0.07 mg/cm²), and the United States Environmental Protection Agency phased out lead from gasoline between 1975 and 1986.

Epidemiology

Due to measures such as the banning of lead in gasoline and paints, as well as increased awareness and screening, the average blood lead levels (BLLs) in children have continued to decrease over the past several decades. Based on updated information regarding the toxicity of lead, the Centers for Disease Control and Prevention (CDC) have continued to lower the acceptable threshold for “normal” BLLs. In 1970, an acceptable BLL was less than 40 mcg/dL (1.9 mmol/L), and in 1990, this level was reduced further to less than 10 mcg/dL (0.48 mmol/L). The percentage of children whose BLLs were greater than 10 mcg/dL (0.48 mmol/L) has dropped from 88% to 1.21% in the past 3 decades. As more data have accumulated on the subclinical toxicity of low-level lead exposure, no measurable amount of lead in blood currently is considered safe. One of the Healthy People 2010 objectives, which will not be achieved, is the elimination of BLLs of 10 mcg/dL (0.48 mmol/L) or greater in the United States.

The annual costs of lead-related health care in the United States are estimated to be \$43.5 billion. As of 2000, 38 million United States homes were considered significant lead hazards. The age of the house is more predictive of lead hazard than its location. Children living in high-risk housing (built before 1950) are almost six times more likely to have

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abnormal BLLs than are those who live in lower-risk housing. Significant disparities exist in pediatric lead poisoning in the United States, with African American children, those receiving Medicaid, those living in lower socioeconomic neighborhoods or urban areas, recent immigrants, and international adoptees affected disproportionately. The incidence of lead poisoning peaks at around 18 to 30 months of age for most children. However, among children who have developmental delays such as autism or pervasive developmental delay, prolonged and repetitive oral exploratory behaviors may cause an increased risk of ongoing and later peaking of BLLs.

Sources of Lead

The major sources of lead in the United States are deteriorated lead-based paints from house decay or renovation, lead-contaminated soil (from prior exposure to leaded gasoline or paints), and lead-laden dust (Table 1). Water that is stagnant in old lead pipes for long periods and lead-soldered food cans (imported to the United States) are additional sources. House dust is a frequent source among young children, who have increased hand-to-mouth activity. Among new immigrant communities, several unique sources of lead have appeared. Lead has been found in candy ingredients, such as tamarind and chili powder, imported from Mexico and in traditional folk medicines used in East Indian, Middle Eastern, and Hispanic cultures.

Examples of such remedies include Greta and Azarcon (Mexican remedy for upset stomach, teething), Ghasard (Indian health tonic), Day Tway (Thailand and Myanmar digestive aid), and Ba-baw-san (Chinese remedy for colic). Cosmetics such as Sindoor (red powder

used by married Indian women) and surma (eye makeup in India) as well as traditional Ayurvedic medicines from India can be heavily laden with lead. Imported toys from China have been found to be inadvertent sources of lead exposure among children in the United States. Pica and dietary supplements can result in lead ingestion, and occupational exposure in pregnant women can cause significant lead poisoning of the fetus. Oral ingestion and gastrointestinal absorption remains the major route of lead exposure for children.

Toxic Effects

Compared with adults, children are at much greater risk of lead toxicity for several reasons. Increased hand-to-mouth behavior, concomitant iron deficiency anemia, increased lead absorption, lead exposure from pica, increased deposition of lead in soft tissues as opposed to bones, the immature blood-brain barrier leading to greater neurotoxicity, and the potential for injury at the cellular level while their body systems are still developing all contribute to a higher toxic potential among children.

Lead exerts its toxic effects in multiple organ systems. Most patients who have elevated BLLs are asymptomatic. Abdominal colic, constipation, growth failure, hearing loss, microcytic anemia, dental caries, spontaneous abortions, renal disease, seizures, encephalopathy, and death are potential outcomes from lead toxicity. Inhibition of the rate-limiting enzymes in heme synthesis such as ferrochelatase and delta amino levulinic acid dehydrogenase results in accumulation of heme intermediates and a microcytic hypochromic anemia. Lead also inhibits T-cell function and affects cartilage mineralization. It inhibits conversion of vitamin D to its active form in the kidneys, thus causing osteopenia and decreased bone

Table 1. Sources of Lead

Source	Comments
Paint chips/dust	Houses built before 1950, renovations causing lead-laden dust
Contaminated soil	Leaded gasoline use deposited 4 to 5 million tons of lead in soil; frequent oral mouthing behaviors in children, urban living with higher traffic areas
Air	Leaded gasoline was the dominant source; industrial emissions now account for most airborne lead
Water	Lead-soldered pipes and hot water permits more leaching of lead into the water
Folk remedies	Litargio, Greta, Azarcon, Alkohl, Bali Bali, Coral, Ghasard, Liga, Pay Loo Ah, Reuda, Ayurvedic medicines
Parental occupational exposure	Transportation workers, soldering, stained glass work, battery reclamation, automobile repair
Other imported sources	Imported toys and foods, ceramics, pottery, cosmetics such as surma (eye makeup in South Asia), soldered pots, kettles

growth. Elevated BLLs among pregnant women are associated with miscarriage, preterm births, and subsequent developmental delays. New data suggest that lead poisoning is a cause of cardiovascular disease, hypertension, and chronic renal dysfunction in adulthood.

The greatest concern for lead toxicity, however, lies in its neurotoxic potential. Before chelation therapy was available, up to 45% of lead-poisoned children who presented with signs and symptoms of encephalopathy (BLL typically >60 mcg/dL [2.9 mmol/L]) died, and a significant percentage had permanent brain damage manifesting as intellectual disability and seizures. However, significant evidence now has accumulated regarding the toxic effects of even very low BLLs on the brain. The neurotoxic effects of lead are believed to be related to two predominant mechanisms: interference with neurotransmission and disruption of cell migration during critical periods of brain development due to interference with cell adhesion molecules. School failure, cognitive loss, hyperactivity, aggression, inattention, distractibility, and delinquent behaviors have been reported with lead poisoning. However, no single neurodevelopmental finding or constellation of symptoms is considered the hallmark for lead neurotoxicity.

A decline of 2 to 3 points in children's intelligence quotient (IQ) scores for each rise above 10 mcg/dL (0.48 mmol/L) of BLL has been established by several meta-analyses. Recent studies strongly suggest that the relationship between BLL and decline in IQ scores may not be linear; the rate of decline may be higher at BLLs below 10.0 mcg/dL (0.48 mmol/L) than those above 10.0 mcg/dL (0.48 mmol/L). One international pooled analysis of seven population-based longitudinal cohort studies involving 1,333 children concluded that the reduction in IQ was nonlinear. Between BLLs of 2.4 and 10 mcg/dL (0.12 and 0.48 mmol/L), IQ was reduced by 3.9, between BLLs of 10 and 20 mcg/dL (0.48 and 0.97 mmol/L) by 1.9, and between BLLs of 20 and 30 mcg/dL (0.97 and 1.5 mmol/L) by 1.1. (1)

A biologic mechanism postulated for such a nonlinear relationship involves different lead saturation pathways: a lead-sensitive rapidly saturating pathway below BLLs of 10 mcg/dL (0.48 mmol/L) and other slowly saturating pathways involved in higher BLLs. Elevated BLLs at 2 years of age have been associated with long-term consequences such as deficits in intellectual and academic performance at age 10 years. Rearing in a nurturing and stimulating environment may decrease the severity of the effects of lead neurotoxicity. This beneficial effect of the social environment has been proven repeatedly in

animal studies. The CDC recommends encouraging parents to provide such a nurturing, stimulating environment and experiences to their children to ameliorate the toxic effects of lead on the brain.

Screening and Initial Assessment

The risk of lead poisoning is not distributed uniformly among children. In recognition of this reality, the universal screening guidelines developed by the CDC in 1991 were replaced by targeted screening guidelines in 1997 to 1998. Five screening questions were developed to assess the child's individual risk:

1. Does your child live in or regularly visit a house with peeling or chipping paint built before 1960?
2. Does your child live in or regularly visit a house built before 1960 with recent or ongoing renovation or remodeling?
3. Does your child have a sibling or playmate being treated for lead poisoning?
4. Does your child live with an adult whose job or hobby involves exposure to lead?
5. Does your child live near an active lead smelter, battery recycling plant, or other industry likely to release lead?

The American Academy of Pediatrics and the CDC developed new recommendations for the detection and management of lead poisoning in 2005. Under these guidelines, all Medicaid-eligible children and those whose families receive any governmental assistance must be screened at age 1 and 2 years. Children living in high-risk environments, such as those in which more than 12% of children have elevated BLLs, also should be screened. Other children should be screened based on their state/city health departments' targeted screening guidelines. Children who have siblings with elevated BLLs above 10 mcg/dL (0.48 mmol/L), recent immigrants, and any child whose parents are concerned should be considered for screening. The website www.aoc.org/pehsu.htm lists specifically designated regional pediatric environmental health specialty units that are excellent resources for clinicians. Immigrant children, refugees, or international adoptees should be screened upon entering the United States. BLLs should be measured for children ages 36 to 72 months if they have not been screened previously. Approximately 43% of children who had elevated BLLs did not have a previous screening test, and 46% of those found to have abnormal BLLs do not receive adequate follow-up care.

Accurate measurement of the BLL is critical. Federal proficiency standards for laboratories that measure BLLs establish a total allowable error of ± 4 mcg/dL

(0.19 $\mu\text{mol/L}$) or $\pm 10\%$, whichever is greater. Venous lead levels are more accurate than fingerstick measurements due to higher contamination from skin surfaces. An elevated capillary BLL should be confirmed with a venous sample.

Management

Pharmacologic therapy with chelating agents does not reverse neurocognitive defects in children who have lead neurotoxicity. Accordingly, case identification is not an effective measure in reducing the ill effects of lead on a community. Primary prevention involving environmental management, family education, and nutritional supplementation are critical. Lead poisoning should be managed with a longitudinal and multidisciplinary approach. The CDC has stratified lead levels to five classes and recommended specific action items for each class of lead poisoning (Table 2).

Environmental Management

Environmental management includes assessment for potential lead hazard by laypeople; inspection and testing of the house by a certified professional; temporary abatement using weekly clean-up and frequent hand washing of children; and permanent abatement using certified lead abatement contractors who use replacement, encapsulation, and paint removal techniques. Recent evidence brings into question the value of household interventions such as weekly clean-up. The authors of a 2008 meta-analysis examined 12 studies and concluded that there is no evidence that household interventions for education or dust control measures are effective in reducing BLLs in children. (2)

Dietary Management

Children deficient in iron, zinc, protein, calcium, and vitamin C are at heightened risk for enhanced absorption of ingested lead. Calcium inhibits absorption, and the bioavailability of lead and zinc competes with lead for binding sites on the delta amino levulinic acid dehydratase enzyme.

Medical Management

The following practices are not recommended at any BLL: searching for gingival lines; evaluating renal function (except for treating with ethylenediaminetetraacetic acid [EDTA]); testing of hair, teeth, or fingernails for lead; radiographs of long bones; and radiographic fluorescence of long bones.

Pharmacologic Therapy

No randomized clinical trials indicate that chelation improves clinical outcomes, particularly neurocognitive outcomes. Treatment protocols have been based on clinical judgment and experiences. Table 3 lists the available chelating agents, their doses, and common adverse effects. One of the oldest agents used in the treatment of severe lead poisoning is dimercaprol, also known as British Anti Lewisite (BAL). The high degree of adverse effects (eg, prolonged partial thromboplastin time, hemolysis, hepatotoxicity), contraindications for patients who have peanut allergy, and uncomfortable route of administration (deep intramuscular) make this agent not useful from a practical perspective. Currently, dimercaprol is only used only when BLLs are greater than 70 mcg/dL (3.4 $\mu\text{mol/L}$).

Calcium disodiumethylenediaminetetraacetic acid (CaNa₂ EDTA, edetate calcium disodium) increases urinary excretion of lead and removes lead efficiently from the extracellular compartment. Its nephrotoxicity can be reduced significantly by ensuring adequate hydration during therapy. For BLLs lower than 70 mcg/dL (3.4 $\mu\text{mol/L}$), it can be used as a single agent administered as a once-daily dose intravenously or intramuscularly for 5 days. It is considered a second-line drug for this purpose.

Treatment with CaNa₂ EDTA can increase redistribution of lead to the central nervous system. Therefore, for those whose BLLs are greater than 70 mcg/dL (3.4 $\mu\text{mol/L}$) or who have encephalopathy, a combination of dimercaprol and CaNa₂ EDTA is preferred; the latter is administered 4 hours after the intramuscular administration of dimercaprol. Some evidence suggests that a combination of CaNa₂ EDTA with dimercaprol may result in faster decline of BLL.

Dimercaptosuccinic acid (DMSA, succimer) is a water-soluble analog of dimercaprol that is administered orally to children who have BLLs of 45 mcg/dL (2.2 $\mu\text{mol/L}$) or greater. This agent, approved by the United States Food and Drug Administration in 1991, is widely used as the primary and preferred treatment for mild and asymptomatic cases of lead poisoning. DMSA enhances urinary excretion of lead, is slightly more effective and less toxic than Ca Na₂ EDTA, and results in less urinary loss of essential minerals. No data support the use of succimer for BLLs between 20 and 44 mcg/dL (1.0 and 2.1 $\mu\text{mol/L}$). Adverse effects include transient rash, elevation of liver enzymes, neutropenia, and abdominal cramping. The strong sulfur odor is difficult for children to tolerate, and the medication usually is sprinkled into chocolate pudding or applesauce to mask the odor. A course of DMSA consists of 19 days, with

Table 2. Centers for Disease Control and Prevention Recommendations for Management of Elevated Blood Lead Levels (BLLs)

Blood Lead Levels	Recommendations
<10 mcg/dL (0.48 mcmol/L) Risk Level I	<ol style="list-style-type: none"> 1. Obtain careful environmental history. 2. Provide risk reduction and nutrition education. 3. If assessment indicates exposure is likely, retest within 3 months of education.
10 to 14 mcg/dL (0.48 to 0.67 mcmol/L) Risk Level IIA	<ol style="list-style-type: none"> 1. Recommendations 1 and 2 from Risk Level I. 2. Report BLL to local department of health. 3. Repeat capillary sample, confirming with a venous sample within 1 month for new cases and 1 to 3 months for known cases.
15 to 19 mcg/dL (0.72 to 0.91 mcmol/L) Risk Level IIB	<ol style="list-style-type: none"> 1. Follow steps from above Risk level IIA. 2. If BLL remains within this range for 3 months, proceed with recommendations for BLLs of 20 to 44 mcg/dL (0.96 to 2.1 mcmol/L) 3. Follow with Lead Poisoning Prevention Programs (LPPPs), which provide home inspections and necessary services. 4. If initial sample was capillary, repeat with venous sample in 1 week to 1 month.
20 to 44 mcg/dL (0.96 to 2.1 mcmol/L) Risk Level III	<ol style="list-style-type: none"> 1. Follow steps for BLLs of 10 to 14 mcg/dL (0.48 to 0.67 mcmol/L) 2. Undertake a complete medical evaluation, environmental history, developmental assessment; in particular, neurodevelopmental history, physical examination, and evaluation for iron deficiency anemia. If ingestion is suspected, obtain abdominal radiographs and proceed with bowel decontamination using cathartics, if necessary. 3. Consider chelation therapy (although not currently indicated for BLLs <45 mcg/dL [2.2 mcmol/L]) in conjunction with a clinician experienced with lead toxicity. 4. Contact LPPPs, which should provide home inspections and necessary services.
45 to 69 mcg/dL (2.2 to 3.3 mcmol/L) Risk Level IV	<ol style="list-style-type: none"> 1. Confirm BLL with venous sample within 24 to 48 hours before beginning chelation therapy. 2. Provide chelation within 48 hours. Child must be in lead-safe environment during therapy. 3. Follow steps for Risk Level III. 4. Administer oral succimer (DMSA) at 10 mg/kg orally every 8 hours for 5 days followed by 10 mg/kg every 12 hours for 14 days (maximum dose, 1,500 mg/day). 5. Undertake a complete neurologic examination and consider free erythrocyte or zinc protoporphyrin testing to assess response to medical management.
>70 mcg/dL (3.4 mcmol/L) Risk Level V	<ol style="list-style-type: none"> 1. Hospitalize immediately and begin chelation with those who have experience with treating lead poisoning (may consult with LPPP medical directors or poison control center), while confirming BLL with venous sample as emergency test. 2. Administer dimercaprol 25 mg/kg per day intramuscularly divided in six doses for a minimum of 72 hours (first dose of dimercaprol administered 4 hours before administration of CaNa₂ EDTA) plus CaNa₂ EDTA for a total of 5 days. 3. Follow steps for Risk Level III. 4. Undertake a complete neurologic examination and consider free erythrocyte or zinc protoporphyrin testing to assess response to medical management.

CaNa₂ EDTA=calcium disodium ethylene amine tetra acetate

Table 3. Chelating Agents for Lead Poisoning

Medication	Dose	Indication to Use	Adverse Effects	Comments
Dimercaprol (BAL)	25 mg/kg per day deep intramuscularly divided in six doses per day for at least 3 days	BLL \geq 70 mcg/dL (3.4 mcmol/L) or lead encephalopathy	<ul style="list-style-type: none"> • Hemolysis in G6PD deficiency • Kidney dysfunction • Zinc depletion • Nausea, vomiting, hypertension, headache, tachycardia, hyperpyrexia, leukopenia 	<ul style="list-style-type: none"> • If administered with CaNa₂ EDTA, first BAL dose usually given 4 hours prior • Dissolved in peanut oil; therefore, contraindicated in patients who have peanut allergy • Alkalinize urine during therapy
Calcium disodium-ethylenediaminetetraacetic acid (CaNa ₂ EDTA)	50 mg/kg per dose single-dose continuous infusion in normal saline or D5W, maximum 1 g/day for 5 days	BLL \geq 45 mcg/dL (2.2 mcmol/L) or lead encephalopathy	<ul style="list-style-type: none"> • Renal dysfunction • Hypokalemia 	<ul style="list-style-type: none"> • Hospitalize; monitor for adequate hydration, electrolyte status
Succimer (DMSA, 2–3 mesodimercaptosuccinic acid)	10 mg/kg per dose every 8 hours for 5 days, then every 12 hours for 14 days for a total of 19 days orally	BLL 45 to 69 mcg/dL (2.2 to 3.3 mcmol/L)	<ul style="list-style-type: none"> • Transient liver function test abnormalities • Reversible neutropenia 	<ul style="list-style-type: none"> • G6PD deficiency is not a contraindication • Availability of oral route makes this an appealing first-line pediatric option
D-penicillamine	10 to 15 mg/kg per day orally for 4 to 12 weeks	BLL 45 to 69 mcg/dL (2.2 to 3.3 mcmol/L)	<ul style="list-style-type: none"> • Renal dysfunction • Reversible leukopenia and thrombocytopenia 	<ul style="list-style-type: none"> • Oral chelator approved for Wilson disease; not specifically approved for chelation of lead • Monitor complete blood count, renal function • Third-line drug

BLL=blood lead level, CaNa₂ EDTA=calcium disodiummethylenediaminetetraacetic acid, D5W=dextrose 5% in water, G6PD=glucose-6-phosphate dehydrogenase deficiency

10 mg/kg per dose every 8 hours administered the first 5 days and the same dose twice daily the next 14 days. Hydration should be well maintained during chelation.

The chelating agent used for treating Wilson disease, D-penicillamine, also is available orally, but its safety and efficacy for chelation of lead has not been established. D-penicillamine is considered a third-line drug for the management of lead poisoning.

When chelation is performed (for BLLs of \geq 45 mcg/dL [2.2 mcmol/L]), patients should be in a lead-free environment because chelation enhances the gastrointestinal avidity for lead. Most often, children are hospitalized during the first few days of therapy while health department officials evaluate their homes for lead and find a lead-free environment for postdischarge stay. BLLs should be rechecked in 1 to 3 weeks after chelation because release of lead from storage sites and its redistribution results in a significant rebound of up to

70% of pretreatment values. Chelation is not indicated for patients whose BLLs are less than 45 mcg/dL (2.2 mcmol/L) due to lack of documented neurocognitive benefits of chelation and concerns about lead remobilization and adverse drug reactions.

Follow-up

Neurodevelopmental lags may not be evident immediately for a patient who has elevated BLLs. Delay may become apparent as the child faces the challenges of academic activities in school, particularly acquiring reading skills in first grade, using academic skills to learn new material in the third or fourth grade, and completing complex multistep tasks in middle school. (3) The CDC, therefore, recommends that neurodevelopmental surveillance not end at 6 years of age. Special educational

needs are addressed best on an individual basis, taking into account the child's specific presenting problem.

Summary

- Based on strong research evidence, no measurable BLL is considered safe.
- Because neurotoxicity associated with lower BLLs has been established by overwhelmingly consistent evidence from meta-analysis of several cohort studies, primary prevention of lead exposure is of paramount importance.
- Lead exerts its effects on multiple body systems (some evidence), with the developing brain being particularly vulnerable (strong evidence).
- No current evidence shows that pharmacotherapy in patients who have high BLLs reverses lead neurotoxicity. Targeted at-risk screening and early intervention for elevated BLLs, as currently recommended by the CDC and the American Academy of Pediatrics, should be conducted by every primary care practitioner to minimize the deleterious effects of lead in children.

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Useful Websites and Phone Numbers

- Pediatric Environmental Health Specialty Units: www.aoec.org/pehsu.htm
- CDC Lead Poisoning and Prevention Program: <http://www.cdc.gov/nceh/lead/>
- CDC Childhood Lead Poisoning Publications: <http://www.cdc.gov/nceh/lead/publications/>
- CDC Information for parents/caregivers: <http://www.cdc.gov/lead/>
- CDC Policy and legislation resources: <http://www.cdc.gov/nceh/lead/policy.htm>
- CDC Resources for clinicians: <http://www.cdc.gov/nceh/lead/toolstraining.htm>
- Environmental Protection Agency information for clinicians and caregivers: <http://www.epa.gov/opptintr/lead/index.html>
- United States Department of Housing and Urban Development: <http://www.hud.gov/offices/lead/>
- Coalition to End Childhood Lead Poisoning (provides information to parents about treatment and prevention of childhood lead poisoning): 1-800-370-5323; <http://www.lead-safe.org/>
- Environmental Protection Agency Safe Drinking Water Hotline: 1-800-426-4791
- National Lead Information Center Hotline: 1-800-LEAD-FYI

HealthyChildren.org Parent Resources from AAP

Lead Poisoning

<http://www.healthychildren.org/English/safety-prevention/all-around/pages/Lead-Poisoning.aspx>

Lead Screening for Children

<http://www.healthychildren.org/English/safety-prevention/all-around/pages/Lead-Screening-for-Children.aspx>

Environmental Health

<http://www.healthychildren.org/English/safety-prevention/all-around/Pages/default.aspx>

PIR Quiz

Quiz also available online at pedsinreview.aappublication.org.

1. Which of the following currently available commercial products may be the source of significant lead toxicity in children in 2010?
 - A. Bottled water.
 - B. Gasoline.
 - C. Household paints.
 - D. Imported cosmetics from India.
 - E. Smoked salmon.
2. What is the *most* common symptom found in children who have elevated blood lead levels?
 - A. Failure to thrive.
 - B. Learning disabilities.
 - C. Lethargy.
 - D. Most children are asymptomatic.
 - E. Vomiting.
3. Which of the following statements regarding the treatment of lead toxicity in children is true?
 - A. Chelation therapy is indicated for all children whose blood lead levels are greater than 20 mcg/dL (1.0 mmol/L).
 - B. Combination therapy with dimercaprol and edetate calcium disodium is indicated for blood lead levels greater than 70 mcg/dL (3.4 mmol/L).
 - C. D-penicillamine is considered first-line therapy.
 - D. Dimercaprol is recommended for asymptomatic children whose blood lead levels are between 10 and 29 mcg/dL (0.5 and 1.4 mmol/L).
 - E. Reversal of lead-induced neurotoxicity is a good marker of successful chelation therapy.
4. You are counseling the parents of a 5-year-old child who has just been found to have an elevated capillary lead level of 19 mcg/dL (0.9 mmol/L). The child has symptoms of a mild learning disability. The family moved into a newly constructed apartment building 6 years ago. Of the following, the *most* accurate information that you can give these parents is that:
 - A. Following successful chelation therapy, the child's learning problems are likely to improve significantly if they were due to lead toxicity.
 - B. It is premature to state that this child has an abnormal blood lead level.
 - C. Therapy with lead-chelating agents must begin immediately to prevent additional neurologic damage.
 - D. The most likely cause of this child's abnormal capillary lead level is ingestion of paint chips from the window ledges in the apartment.
 - E. Treatment should be considered if the capillary lead level remains above 15 mcg/dL (0.7 mmol/L) over the next 3 months.
5. Which of the following statements regarding the epidemiology of lead toxicity in children in the United States is true?
 - A. According to the Centers for Disease Control and Prevention (CDC), blood lead levels less than 20 mcg/dL (1.0 mmol/L) are considered the acceptable threshold for "normal."
 - B. Blood lead levels less than 5 mcg/dL (0.2 mmol/L) are considered to be "safe."
 - C. Children who receive Medicaid have a lower rate of abnormal blood lead levels than those who have private insurance.
 - D. The incidence of lead toxicity peaks around age 12 years as children enter middle school.
 - E. The percentage of children whose blood lead levels are greater than 10 mcg/dL (0.48 mmol/L) has decreased from near 90% 3 decades ago to less than 2% today.

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