Chapter 31

Lead

Childhood lead toxicity has been recognized for at least 100 years. As recently as the 1940s, many believed that children with lead poisoning who did not die during the acute toxic episode had no residual effects. After it was recognized that learning and behavior disorders occurred in children who recovered from acute toxicity, many believed that only children with frank symptoms suffered neurobehavioral deficits. In the 1970s and 1980s, however, studies worldwide demonstrated that asymptomatic children with higher levels of lead had lower IQ scores,[1,2] more language difficulties,[3] attention problems, and behavior disorders.[4,5] With better epidemiologic studies, the definition of a harmful level of lead has changed markedly. As recently as 1968, children were discharged from the hospital when the blood lead level decreased to 60 μg/dL,[6] and through the 1970s,[7] children with blood lead levels up to 29 μg/dL were thought to have inconsequential lead exposure. Over subsequent years, however, effects were seen at lower and lower levels. There is, as yet, no reliable threshold for these long-lasting effects of lead exposure on cognitive test scores. In 2 independent meta-analyses conducted in the 1990s of prospective studies from several countries,[1,2] damage was documented beginning at a blood lead level of 10 μg/dL. More recent studies have demonstrated a relationship between blood lead level at the time of testing and decreased scores on reading and arithmetic tests that is apparent even in children 6 to 16 years of age, including those whose blood lead levels by then are less than 5 μg/dL.[8] Canfield et al[9] reported that among 172 children followed prospectively with measurements of blood lead level, 101 had never had a blood lead level greater than 10 μg/dL, and there was still a strong negative relationship between blood lead level and IQ when the children were 3 to 5 years of age, a result subsequently confirmed by Bellinger and Needleman.[10]

Since lead was removed from gasoline and paint more than 3 decades ago, fatal lead encephalopathy has all but disappeared and symptomatic lead poisoning is now rare. The continued exposure, however, of thousands of children to lead-laden dust in deteriorating housing mars what would otherwise be a public health triumph. Such low-level lead exposure produces cognitive impairment without identifiable clinical symptoms, and it is this asymptomatic cognitive impairment that constitutes most lead poisoning in the United States. The focus has, thus, shifted from the care of symptomatic children to primary prevention of lead exposures and reduction of exposures for children with elevated levels and subclinical effects. Although much of the management of children at risk of lead poisoning is nonclinical, pediatricians commonly find themselves participating in or even directing these activities.[11]

ROUTES AND SOURCES OF EXPOSURE

Children most often are exposed to lead through the unintentional ingestion of lead-containing particles, such as dust from paint or soil, or from water or foreign bodies. Lead can be absorbed from the pulmonary tract if inhaled as fumes or respirable particles.

Lead (Pb) is an element and occurs naturally, but blood lead levels are low in the absence of industrial activities.[12] In the United States, there have been 2 major sources of industrially derived lead for children: airborne lead, mostly from the combustion of gasoline containing tetraethyl lead, and leaded chips and dust, mostly from deteriorating lead paint.

The years since 1980 have witnessed a substantial decrease in childhood exposure to airborne lead in the United States. Federal legislation in the 1970s removed lead from gasoline and reduced smokestack emissions from smelters and other sources, causing blood lead levels in children to decrease. From 1976 to 1980, before the regulations had their full impact, US children 1 to 5 years of age had a median blood lead level of 15 μg/dL; 88% of them had levels at or above 10 μg/dL.[13] From 1999 to 2004, the most recent data available, only 1.4% of children 1 to 5 years of age had blood lead levels at or above 10 μg/dL.[14] Although levels have decreased in all children, black children and poor children continue to have relatively high blood lead levels. Airborne lead should no longer be a source of exposure in most US communities. However, residual lead in the soil in areas heavily affected by airborne lead, such as around smelters, continues to be a problem even decades after closure of the worst sites.[15]

The source for most lead-poisoned children now is the dust and chips from deteriorating lead paint on interior surfaces. Children living in homes with deteriorating lead paint can achieve blood lead levels of at least 20 μg/dL without frank pica.[16] This exposure commonly arises from normal, developmentally-appropriate hand-to-mouth behavior in an environment that is

contaminated with lead dust. Children can ingest lead-laden dust with their cereal, for example, by dropping dry cereal on the floor at mealtime and then hunting it down and eating it later, or with their banana by squishing the fruit through their dust-laden hands in preparation for consumption.[17] Children can ingest lead from mouthing contaminated toys.

The use of heavily leaded paint on interior surfaces ceased in the United States by 1978. However, in 1998, of the 16.4 million homes with 1 child or more younger than 6 years, 27% still had significant lead paint hazards (lead-based paint in such a deteriorated condition that exposure is likely).[18] Dust also is a final resting place for old airborne lead from gasoline, and lead in urban soils can recontaminate cleaned houses.[19]

Individual children may be exposed to lead fumes or respirable dust resulting from sanding or heating old paint, burning or melting automobile batteries, or melting lead for use in a hobby or craft. Some toy jewelry is made of lead, and a child who ingested a lead charm died of lead poisoning in 2006.[20] Some old toys made in the United States and some imported toys were painted with lead-based paint, and some plastic toys and vinyl have lead added as a softener. The US Consumer Product Safety Commission (CPSC) has required recalls of some of these toys and is working with importers and manufacturers to prevent further importation of products containing unsafe amounts of lead. Although individual children could chew on or ingest these products and, thus, absorb lead from them, it is still not clear how much toys and plastics are contributing to the exposure of most children.

Lead plumbing (Latin "plumbus" means lead) has contaminated drinking water for centuries. In 2003-2004, some tap water in Washington, DC, was found to exceed US Environmental Protection Agency (EPA) regulations. This was thought to be caused by a change in water disinfection procedures, which increased the water's ability to leach lead from connector pipes between the water mains and interior plumbing in old houses. The extent of this problem in Washington and other cities is not yet known. It was recommended that affected families drink filtered or bottled water until the pipes can be replaced. Uncommon sources of exposure include cosmetics, folk remedies, pottery glaze, old or imported cans with soldered seams, and contaminated vitamin supplements.

Table 31.1 lists risk factors for lead poisoning and prevention strategies.

SYSTEMS AFFECTED

For lead exposure now seen in the United States, subclinical effects on the central nervous system (CNS) are the most common effects. The best-studied effect is cognitive impairment, measured by IQ tests. The strength of this association and its time course are characteristic and have been similar in

Table 31.1: Risk Factors for Lead Exposure and Prevention Strategies

RISK FACTOR	PREVENTION STRATEGY
Environmental	
Paint	Identify, evaluate, and remediate
Dust	Control sources
Soil	Restrict play in area, plant groundcover
Drinking water	Check with local authorities about morning flush of water from faucet; use cold water for cooking and drinking, especially if tap water used for preparing formula
Folk remedies	Avoid use
Some imported cosmetics (eg, kohl or surma)	Avoid use
Old ceramic or pewter cookware, old urns/kettles, decorative pottery from Mexico and ceramics from China	Avoid use
Some imported toys, crayons	Avoid use
Parental occupations (painter, lead-paint abatement, etc)	Shower and remove work clothing and shoes before leaving work
Hobbies	Proper use, storage, and ventilation
Home renovation	Proper containment, ventilation; pregnant women and young children should vacate premises while work is done and not reenter until premises certified as lead-safe
Buying or renting a new home	Inquire about lead hazards, look for deteriorated paint before occupancy, hire certified lead risk assessor to evaluate hazard and recommend control options
Host	
Hand-to-mouth activity (or pica)	Control sources; frequent hand washing
Inadequate nutrition	Adequate iron and calcium
Developmental disabilities	Enrichment programs as available

multiple studies in several countries.[21] In most countries, including the United States, blood lead levels peak at approximately 2 years of age and then decrease without intervention. Although there is some relationship between peak blood lead level and IQ tested later, it is now clear that contemporaneous blood lead, even though it is lower, is more strongly associated with school-aged IQ.[21,22] The Centers for Disease Control and Prevention (CDC)[23] and American Academy of Pediatrics (AAP)[11] currently use 10 μg/dL as the level that should prompt public health action.[24] A blood lead level of 10 μg/dL should not be interpreted as a threshold; no threshold for effects has been identified.[24,25] Although lead is a risk factor for developmental and behavioral problems, its impact has significant individual variability, which may be modulated by the psychosocial environment and educational experiences of the developing child.[24] Many factors affect cognition and behavior.

Other aspects of CNS function also may be affected by lead, but they are less well documented. Subclinical effects on hearing[26] and balance[27] may occur at commonly encountered blood lead levels. Some studies have measured tooth or bone lead levels, which are thought to represent integrated, possibly lifetime, exposure. Teachers reported that students with elevated tooth lead levels were more inattentive, hyperactive, disorganized, and less able to follow directions.[3,28] Further follow-up in 1 of the studies[3] showed higher rates of failure to graduate from high school, reading disabilities, and greater absenteeism in the final year of high school.[29] Elevated bone lead levels were associated with increased attentional dysfunction, aggression, and delinquency.[30]

Although there are reasonable animal models of low-dose lead exposure and cognition and behavior,[31] the mechanisms by which lead affects CNS function are not known. Lead alters very basic nervous system functions, like calcium-modulated signaling, at very low concentrations in vitro.[32] The age of 2 years, when lead levels peak, is the same age at which a major reduction in dendrite connections occurs, among other events crucial to development. It is, thus, plausible that lead exposure at that time interferes with a critical development process in the CNS, but what that process is has not been identified. Brain imaging studies in adults with elevated blood lead levels in childhood have demonstrated region-specific reductions in gray matter volume,[33] alterations of white matter microstructure,[34] and a significant impact of lead on brain reorganization associated with language function.[35]

Lead also has important nonneurodevelopmental effects. The kidneys are a primary target organ; children exposed to lead are at significantly greater risk of becoming hypertensive adults. Another renal effect of lead in children is impaired 1-*d*-hydroxylation of vitamin D, a necessary step towards activating this vitamin. Lead interferes with heme synthesis beginning at blood lead levels of approximately 25 μg/dL.[36] Both d-aminolevulinate dehydratase, an

early-step enzyme, and ferrochelatase, which closes the heme ring, are inhibited. Ferrochelatase inhibition is the basis of an erstwhile screening test for lead poisoning that measured erythrocyte protoporphyrin, the immediate heme precursor. Because it is insensitive to the lower levels of blood lead that are of concern now, the test is now obsolete for that use. A recent cross-sectional study suggests that environmental exposure to lead may delay growth and pubertal development in black and Mexican-American girls.[37] Finally, episodes of severe lead poisoning can cause growth arrest of the long bones, producing "lead lines."

CLINICAL EFFECTS

Some children with blood lead levels greater than 60 μg/dL may complain of headaches, abdominal pain, loss of appetite, or constipation or may be asymptomatic. Children displaying clumsiness, agitation, or decreased activity and somnolence are presenting with premonitory symptoms of CNS involvement that may rapidly proceed to vomiting, stupor, and convulsions.[6] Symptomatic lead toxicity should be treated as an emergency. Although lead can cause abdominal colic, peripheral neuropathy, and renal disease in adults with occupational exposures, these are rare in children.

DIAGNOSTIC MEASURES

The diagnosis of lead poisoning or increased lead absorption depends on the measurement of a blood lead level. This is best performed on a venous sample, but finger-stick samples can be used if care is taken to avoid contamination. Most initial blood lead measurements are now performed as screening tests, because children meet some general eligibility criteria or because of parental concern rather than because children have symptoms suggestive of lead poisoning.

Screening

Until 1997, the AAP and CDC recommended that virtually all children have at least one measurement of blood lead beginning at 12 months of age, with a retest at 24 months of age, if possible. Because the prevalence of elevated blood lead levels has decreased substantially, the CDC in 1997 recommended that health departments determine a lead screening strategy for their jurisdictions on the basis of prevalence of housing risks and children with blood lead levels ≥10 mg/dL. However, regardless of the local recommendation, federal policy requires that all children enrolled in Medicaid receive screening blood lead tests at ages 12 and 24 months and that blood lead screening be performed for children 36 to 72 months of age who have not been screened previously.[38] Most children with elevated blood lead levels are Medicaid eligible. The CDC Advisory Committee on Childhood Lead Poisoning Prevention has proposed criteria by which a state might become exempt from this requirement,[39] but they have not yet been implemented.

Blood lead screening and assessments of risks for exposures to lead vary considerably by locale from universal blood lead screening to application of targeted blood lead testing determined by risk assessment tools. Clinicians should consult city, county, or state health departments to determine the appropriate recommendations for their jurisdiction. This information is available for most states on the CDC Web site (http://www.cdc.gov/nceh/lead/programs.htm). Children not on Medicaid and residing in states with no screening policy should have blood lead testing in accordance with Medicaid guidelines. The sensitivities of personal risk questionnaires and other substitutes for measuring blood lead levels vary by population assessed and often are unacceptably low.

Children of all ages who are recent immigrants, refugees, or adoptees have an increased prevalence of elevated—sometimes very elevated—blood lead levels and, thus, should be screened at the earliest opportunity. Those 6 months to 6 years of age and older children, as warranted, should be tested again 3 to 6 months after moving into permanent residences.[25] These children may have had lead exposure in their native country, but it is also possible that their exposure occurred in unsuitable housing once they arrived in the United States. The CDC Web site has a toolkit that discusses risks for these children.

Because of lead's effects on the developing fetus, some states have developed lead screening guidelines for pregnant women. The CDC recently published guidelines on the screening of pregnant women for lead, medical and environmental management, and follow-up of mothers and infants when maternal lead levels are ≥5 μg/dL.[40] Care of the infant includes measuring cord or neonatal blood lead to establish a baseline; further management guidelines depend on baseline blood lead level. Lead is transmitted in human milk. However, because breastfeeding is an optimal source of infant nutrition and is associated with many beneficial aspects of growth and development, the guideline calls for an interruption of breastfeeding only if the maternal blood lead level is ≥40 μg/dL; above this level, women should pump and discard their milk until after their blood lead level decreases below 40 μg/dL. These breastfeeding infants require repeated blood lead evaluations and potentially other medical and environmental evaluations to ensure that their blood lead levels are not increasing excessively.

Diagnostic Testing

Some experienced clinicians measure the blood lead level in children with growth retardation, speech or language dysfunction, anemia, and attentional or behavioral disorders, especially if the parents have a specific interest in lead or in health effects from environmental chemicals. However, persistent elevation of blood lead levels into school age is unusual, even if peak blood lead level at 2 years of age was high and the child's housing has not been abated. Thus, a

relatively low blood lead level in a school-aged child does not rule out earlier lead poisoning. If the question of current lead poisoning arises, however, the only reliable way to make a diagnosis is with blood lead measurement. Hair[41] or urine lead levels give no useful information and should not be performed.

MANAGEMENT OF CLINICAL AND LOW-LEVEL LEAD TOXICITY

Management should be provided to all children with a blood lead level of 10 μg/dL or greater[24] (see Table 31.2). Proper management includes finding and eliminating the source of the lead, instruction in proper hygienic measures (personal and household), optimizing the child's diet and nutritional status, and close follow-up (see Tables 31.3 and 31.4). Because most children with higher blood lead levels live in or visit regularly a home with deteriorating lead paint, successful therapy depends on eliminating the child's exposure. Any treatment regimen that does not control environmental exposure to lead is considered inadequate. Pediatricians should refer poisoned children to local public health offices for environmental assessment of the child's residence(s). Public health staff should conduct a thorough investigation of the child's environment and family lifestyle for sources of lead.

Deteriorated lead paint is the most common source of exposure. However, other sources that should be considered include tableware, cosmetics such as surma and kohl, home remedies, dietary supplements of calcium, tap water, and parental occupation. Some children will have persistently elevated blood lead levels without access to lead paint. Their exposure may come from any of the sources listed in Table 31.1. Blood lead levels should decrease as the child passes the age of 2 years or so, and a stable or increasing blood lead level past that age is likely to be attributable to ongoing exposure. In children who have spent prolonged periods in a leaded environment, blood lead levels will decrease more slowly after exposure ceases,[42] probably because bone stores are greater.

The CDC Advisory Committee on Childhood Lead Poisoning Prevention issued case management guidelines for children with lead poisoning in March 2002.[43] These guidelines should be consulted as needed.

Although there are no studies that have identified effective strategies to reduce blood lead levels <10 μg/dL, guidelines for potential strategies for managing blood lead levels <10 μg/dL have been published by the CDC Advisory Committee on Childhood Lead Poisoning Prevention.[24] Because nutritional deficiencies can influence lead absorption and may have their own associations with health outcomes independent of lead exposures, specific attention should be paid to identifying and treating iron deficiency and ensuring adequate calcium and zinc intake.

Chelation therapy for children with blood lead levels of 20 to 44 μg/dL can be expected to lower blood lead levels but has not been shown to reverse or

Table 31.2: Recommended Follow-up Actions, According to Blood Lead Level (BLL)*

BLL (µg/dL)	ACTIONS
<10	Continued surveillance For a child whose BLL is approaching 10 µg/dL, more frequent blood lead screening (ie, more than annually) might be appropriate, particularly if the child is aged <2 years old, was tested at the start of warm weather when BLLs tend to increase, or is at high risk of lead exposures[24]
10–14	Obtain a confirmatory venous BLL within 1 month; if still within this range: ■ Provide education to decrease lead exposure ■ Repeat BLL test within 3 months
15–19	Obtain a confirmatory venous BLL within 1 month; if still within this range: ■ Take a careful environmental history ■ Provide education to decrease lead exposure and to decrease lead absorption ■ Repeat BLL test within 2 months
20–44	Obtain a confirmatory venous BLL within 1 week; if still within this range: ■ Conduct a complete medical history (including an environmental evaluation and nutritional assessment) and physical examination ■ Provide education to decrease lead exposure and lead absorption ■ Refer the patient to the local health department or provide case management that should include a detailed environmental investigation with lead hazard reduction and appropriate referrals for support services ■ Chelation not currently recommended for BLLs <45 µg/dL
45–69	Obtain a confirmatory venous BLL within 2 days; if still within this range: ■ Conduct a complete medical history (including an environmental evaluation and nutritional assessment) and a physical examination ■ Provide education to decrease lead exposure and lead absorption ■ Refer the patient to the local health department or provide case management that should include a detailed environmental investigation with lead hazard reduction and appropriate referrals for support services ■ Begin chelation therapy in consultation with clinicians experienced in lead toxicity therapy
≥70	Hospitalize the patient and begin medical treatment, including parenteral chelation therapy, immediately in consultation with clinicians experienced in lead toxicity therapy ■ Obtain a confirmatory BLL immediately ■ The rest of the management should be as noted for management of children with BLLs between 45 and 69 µg/dL

*Adapted from Centers for Disease Control and Prevention.[43]

Table 31.3: Clinical Evaluation*

Medical History

Ask about

- Symptoms
- Developmental history
- Mouthing activities
- Pica
- Previous blood lead level measurements
- Family/maternal history of exposures to lead

Environmental History

Paint and soil exposure

- What is the age and general condition of the residence?
- Is there evidence of chewed or peeling paint on woodwork, furniture, or toys?
- How long has the family lived at that residence?
- Have there been recent renovations or repairs in the house?
- Are there other sites where the child spends significant amounts of time?
- What is the character of indoor play areas?
- Do outdoor play areas contain bare soil that may be contaminated?
- How does the family attempt to control dust/dirt?

Relevant Behavioral Characteristics of the Child

- To what degree does the child exhibit hand-to-mouth activity?
- Does the child exhibit pica?
- Are the child's hands washed before meals and snacks?

Exposures to and Behaviors of Household Members

- What are the occupations of adult household members?
- What are the hobbies of household members? (Fishing, working with ceramics or stained glass, and hunting are examples of hobbies that involve risk for lead exposure)
- Are painted materials or unusual materials burned in household fireplaces?

Miscellaneous Questions

- Does the home contain vinyl miniblinds made overseas and purchased before 1997?
- Does the child receive or have access to imported food, cosmetics, or folk remedies?
- Is food prepared or stored in imported pottery or metal vessels?

Nutritional History

- Take a dietary history
- Evaluate the child's iron status using appropriate laboratory tests
- Ask about history of food stamps or Special Supplemental Nutrition Program for Women, Infants, and Children program (WIC) participation

Physical Examination

- Pay particular attention to the neurologic examination and to the child's psychosocial and language development

*Adapted from Centers for Disease Control and Prevention.[43]

Table 31.4: Schedule for Follow-up Blood Lead Level (BLL) Testing*

VENOUS BLL (μg/dL)	EARLY FOLLOW-UP (FIRST 2–4 TESTS AFTER IDENTIFICATION)	LATE FOLLOW-UP (AFTER BLL BEGINS TO DECLINE)
10–14	3 months	6–9 months
15–19	1–3 months	3–6 months
20–24	1–3 months	1–3 months
25–44	2 weeks–1 month	1 month
≥45	As soon as possible	Chelation with subsequent follow-up

*Adapted from Centers for Disease Control and Prevention.[43]
Note: Seasonal variation of BLLs exists and may be more apparent in colder climate areas. Greater exposure in the summer months may necessitate more frequent follow-up. Some clinicians may choose to repeat blood lead tests on all new patients within a month to see whether their BLL is increasing more quickly than anticipated.

diminish cognitive impairment or other behavioral or neuropsychological effects of lead.[44]

If the blood lead level is greater than 45 μg/dL and the exposure has been controlled, treatment should begin. A pediatrician experienced in managing children with lead poisoning should be consulted—these can be found through the AAP Council on Environmental Health, at hospitals that participated in the clinical trial of succimer,[44] at Pediatric Environmental Health Specialty Units, or through lead programs at state health departments (http://www.cdc.gov/nceh/lead/grants/contacts/CLPPP%20Map.htm). Detailed treatment guidelines were published by the AAP in 1995.[45]

Frequently Asked Questions

Q *I'm worried that my child has any detectable lead in his blood. How can I eliminate exposure?*

A In children with low blood lead levels, recommended interventions have to be not only effective but also very safe. Generally, applicable recommendations include taking an environmental history to identify potential sources for lead exposure, testing the child for iron deficiency and correcting it if it is found, testing drinking water, inspecting any older building in which the child spends time for evidence of deteriorating paint, and then following US Department of Housing and Urban Development guidelines for necessary household renovation. Having a child with a blood lead level of 5 or 10 μg/dL may be a source of concern, but no specific drug therapies have been tested and shown to be safe and effective at these low levels.

Q *We have imported ceramic dishes. Is it safe to use them?*

A Some imported ceramics contain lead. Of particular concern have been pottery from Mexico and ceramic ware from China. At the dishes wear, become chipped or cracked, lead can leach from the dishes into foods. Some imported dishes labeled as "lead-free," have been found to contain unsafe amounts of lead. There are many safe alternatives, so using such dishes should be avoided. The US Food and Drug Administration began regulating lead in glazes used on dishes made in the United States in the 1980s and further strengthened regulations in the 1990s. Dishes made in the United States before these regulations took effect may contain lead.

Q *We have vinyl miniblinds. Should I get rid of them?*

A In the mid-1990s, some imported, nonglossy vinyl miniblinds were found to contain lead. Children who touch these miniblinds and put their fingers in their mouths may ingest small amounts of lead. Sunlight and heat can break down the blinds, causing release of lead-contaminated dust. If you purchase new miniblinds, look for products with labels that say "new formulation" or "nonleaded formula." Older ones must be discarded if they have begun to chalk or deteriorate.

Q *Is there still lead in canned food?*

A Cans with soldered seams can add lead to foods. In the United States, soldered cans have been replaced by seamless aluminum containers, but some imported canned products still have lead-soldered seams.

Q *What about testing for lead in water?*

A If you are using tap water to reconstitute infant formula or juice or there has been local concern, you may want to have your water tested. To help determine whether your water might contain lead, call the EPA Safe Drinking Water Hotline at 800-426-4791 or your local health department to find out about testing your water. Well water should be tested for lead when the well is new and tested again when a pregnant woman, infant or child less than 18 years of age moves into the home; for a discussion of well water for infants, see the AAP policy statement on drinking water from private wells.[46] Most water filters remove lead.

Q *How can I tell if a toy has lead paint or is made of lead?*

A Toys are not all routinely tested for lead. Many toys are imported from countries with poorly enforced safety rules by companies that do not test the toys before selling them. The AAP advises parents to monitor the Consumer Product Safety Commission Web site for notices of recalls and to avoid nonbrand toys and toys from discount shops and private vendors. Old and used toys should be examined for damage and clues to the origin of the toy. If the toy is damaged or worn or from a country with a history of poor monitoring of manufacturing practices, the safest action is to remove it from

use. Be particularly attentive to costume jewelry and other small metal pieces that can be swallowed.

Q What is the correct procedure to follow when a child is witnessed ingesting a piece of lead-containing paint?

A A diagnostic lead level is indicated when a parent expresses concern about ingestion of potential lead-containing substances. Such a test should be done right away, because it is likely that the child ingested similar substances even before someone identified it as a problem. With ingestions, blood lead levels rise rapidly (within hours to days) and can continue to rise during bowel transit of the object. Once the object has been excreted, the blood level falls to a new body equilibrium over the next month. An abdominal x-ray to assess presence of lead-containing substances is indicated if a child's lead level is 45 μg/dL or higher. X-rays of long bones to assess "lead lines" (ie, dense metaphyseal lines of growth arrest) are not indicated.

This might be a good occasion to also assess the child's iron status, because iron deficiency is associated with more efficient absorption of lead from the gut, and pica behavior has sometimes been associated with iron-deficient status. Low ferritin, even in the absence anemia, low mean corpuscular volume (MCV), or elevated red cell distribution width (RDW), should be treated with therapeutic doses of iron.

It would also be wise to check the paint to see whether lead is present. In homes built before 1978, paint chips should be assumed to contain lead unless tested and proven otherwise. Your local or state health department can answer your questions about obtaining a home inspection to check for lead.

Basophilic stippling may be seen, but generally at lead levels much higher than are common today. Also consider vitamin B_{12} and folate deficiencies as causes of basophilic stippling.

Q Should iron be prescribed for patients with lead levels between 10 and 20 μg/dL?

A Not unless they are iron deficient. Theoretically, iron could affect absorption of lead from the gut. Lead is taken up by the iron absorption machinery and secondarily blocks iron through competitive inhibition. There are no supporting research data to demonstrate the efficacy of prescribing therapeutic iron to all children with elevated lead. Iron therapy should not be prescribed unless iron status is deficient (low ferritin or another indicator).

Resources

National Lead Information Center

422 South Clinton Avenue
Rochester, NY 14620
Phone: 1-800-424-LEAD
Fax: (585) 232-3111

Office of Healthy Homes and Lead Hazard Control, Department of Housing and Urban Development

Web site: www.hud.gov/offices/lead

US Environmental Protection Agency Federal Plan for Eliminating Childhood Lead Poisoning

Web site: http://yosemite.epa.gov/ochp/ochpweb.nsf/content/whatwe_tf_proj.htm

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