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The
INSTITUTE FOR MEDICAL EDUCATION
BULLETIN

Screening for Elevated Blood Lead Levels in High-Risk Children

Testing Recommended at both 1 and 2 Years of Age

By
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With
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Developed: March 2007 **Approved:** May 2007 **Expiration Date for CME Credit:** May 31, 2008
Estimated Time to Complete: 30 Minutes **Target Audience:** Family Practice, Internal Medicine and Other Primary Care Physicians; Nurse Practitioners and Physician Assistants; Pharmacists; Nurses; Other Health Care Professionals

Description

This learning activity reviews the rationale for lead screening in Medicaid-eligible and other high-risk children and the importance of testing high-risk children for elevated blood lead levels at both 1 and 2 years of age.

Objectives

Following this learning activity, the learner will be able to:
- Name the two principal sources of lead exposure for children in the United States.
- Discuss why young children are at greatest risk for environmental lead exposure.
- Discuss why all Medicaid-eligible children should be screened for elevated blood lead levels.
- State the rationale for testing high-risk children for elevated blood lead levels at both 1 and 2 years of age.

Accreditation

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Disclosure of Faculty and Sponsor Relationships

Cynthia J. Lord, MN, PHN, RN; David W. Griffin, MD; and Gary Freeman, MD have indicated no financial interests, affiliations, or intent to discuss unapproved or investigative use of commercial products or devices.

Evaluation

When you have finished studying these materials, please complete the post-test and evaluation and return them to:

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Lead is a well-known neurotoxin that is particularly harmful to the developing nervous systems of fetuses and young children. Lead exposure is believed to contribute to cognitive [(1)] and behavioral [(2)] deficits that may be long-lasting or permanent [(3)]. Eliminating blood lead levels >10 μg/dL [(4)] among young children is a goal of the Healthy People 2010 initiative.

The principal sources of lead exposure for children in the United States are 1) house dust contaminated by dust or chips from deteriorated lead paint [(5)] and 2) soil contaminated by lead paint dust or chips and/or residue from the combustion of gasoline containing tetraethyl lead [(6)]. **Most lead poisoning in children is due to chips or dust from deteriorating lead paint on interior surfaces** [(7)]. Young children are at greatest risk for environmental lead exposure due to normal mouthing behaviors that cause them to place their hands and objects contaminated with lead-laden dust or soil in their mouths (a speck of lead dust no larger than a grain of salt is sufficient to cause an elevated blood lead level).

Although the number of U.S. homes with lead-based paint has declined from 64 million in 1990 to 38 million in 2000, an estimated 24 million housing units still contain substantial lead paint hazards [8]. **These homes are more likely to be occupied by low income/Medicaid-eligible and minority populations with young children** [9] who have the highest prevalence of elevated blood lead levels [10]. Medicaid-eligible children represent 60% of children with blood lead levels >10 μg/dL and 83% of those with blood lead levels >20 μg/dL.

The Centers for Disease Control and the American Academy of Pediatrics currently recommend that children at increased risk for lead exposure be screened for elevated blood lead levels (i.e., those living in older housing; those who had a sibling or playmate with an elevated blood lead level; or those who had lived in or visited a structure that might contain deteriorated, damaged, or recently remodeled lead-painted surfaces) [11].

The Centers for Medicare and Medicaid Services also *requires* screening of all Medicaid-eligible children for elevated blood lead levels as part of prevention services provided through the Early and Periodic Screening, Diagnosis, and Treatment program (in Minnesota, this includes children with Medical Assistance and MinnesotaCare).

Blood lead levels usually peak at about 2 years of age, but the discovery of an elevated blood lead level at that age may be too late to prevent significant exposure and permanent injury [12]. In addition, an at-risk child with a blood lead level <10 μg/dL at age 1 year might have an elevated level by age 2 years. ***It is therefore recommended that children at increased risk for lead exposure be screened for elevated blood lead levels at both 1 and 2 years of age*** (note that Medicaid reimburses for two screenings, one at 1 year of age and the other at 2 years of age).

Although the HealthPartners Medical Group has been highly successful in screening Medicaid-eligible children for elevated blood lead levels [13], **only 25 % of children initially tested in 2005 were tested again in 2006**.

CONCLUSIONS

- **Lead is a well-known neurotoxin that is particularly harmful to the developing nervous systems of fetuses and young children.**
- **The principal sources of lead exposure in the United States are 1) house dust contaminated by dust or chips from deteriorated lead paint and 2) soil contaminated by lead paint dust or chips and/or residue from the combustion of gasoline containing tetraethyl lead.**
- **Houses containing lead paint hazards are more likely to be occupied by low income/Medicaid-eligible and minority populations with young children who have the highest prevalence of elevated blood lead levels.**
- **All Medicaid-eligible and other children at increased risk for lead exposure should be screened for elevated blood lead levels at both 1 *and* 2 years of age.**

1) Each 10 μg/dL increase in blood lead level has been associated with a 2-3 point decline in IQ. Other investigators have found a decline of 6-7 IQ points as the blood lead level increases from 1 μg/dL to 10 μg/dL. The economic cost associated with the loss of intellectual capacity due to lead exposure is significant. It has been estimated that the lifetime costs associated with each year's cohort of children exposed to lead is $43 billion.
2) In one study, students with elevated tooth lead concentrations were found by teachers to be more inattentive/distractible, hyperactive, disorganized, and less able to follow directions. On follow-up, some of these children showed higher rates of failure to graduate from high school, reading disabilities, and greater absenteeism from high school. Elevated bone lead has been associated with attentional dysfunction, aggression, and delinquency.
3) Blood lead is an estimate of recent lead exposure. Lead also accumulates in bone (clearance half-life 27 years) and is an estimate of an individual's lifetime cumulative retained dose of lead. Tibial lead can be measured using x-ray fluorescence and many individuals born before the 1970's have relatively high levels of bone lead. A recent study found that higher levels of bone lead in adults was associated with diminished cognitive function as measured by tests of non-verbal reasoning. The cognitive decline associated with lead exposure appeared to be progressive and was comparable to 5 years of aging. MRI-based volumetric studies of the brain have also identified an association between elevated tibial lead levels and an increased prevalence and severity of white matter lesions. Higher tibial lead levels were also associated with smaller volumes of the total brain, frontal and total gray matter, and parietal white matter.
4) From 1975 until 1991, the CDC's threshold for an elevated blood lead level was 30 μg/dL. This was lowered to 10 μg/dL based on studies done in the late 1980's linking blood lead levels with decreased intelligence and other adverse neurodevelopmental effects. However, there is no safe lead level and some authorities have advocated that the "blood lead action level" should be lowered to 2 μg/dL. Note that the median blood lead concentration of U.S. children age 1-5 years was 15 μg/dL from 1976-1980, 3.6 μg/dL from 1988-1991, and 1.9 μg/dL in 1999.
5) White lead (lead carbonate) was commonly used as a white pigment in interior and exterior paints (lead paint was most commonly used on exterior surfaces, interior woodwork, doors, and windows). Red lead (lead oxide) was used a primer on steel to prevent corrosion. Lead chromate pigments in colors of yellow, orange, or green were also common and are still used for safety paints such as the paint on traffic lanes or fire hydrants. Lead acetate was used in paint, varnish, and other coatings. The Lead Industries Association (formed in 1928 to promote the use of lead) continued to promote the usefulness of white lead in interior and exterior coatings as late as 1952, although paint manufacturers began to reduce the amount of lead in paints after about 1940 (painted surfaces in homes built prior to 1950 are more likely to have higher levels of lead than surfaces in homes built between 1950 and 1978). The National Lead Company used the Dutch Boy logo to market paints containing white lead. Paint containing more than 0.06% lead was banned for residential use in 1978 by the U.S. Consumer Product Safety Commission. After lead was banned from paint, pigments such as titanium dioxide and barium sulfate have replaced lead as the pigment in white paints.
6) Leaded gasoline was discovered on December 9, 1921 at the General Motors research labs in Dayton, Ohio. Leaded gasoline was typically a suspension of 3-4 mL of tetraethyl lead per gallon of gasoline. "Ethyl fluid" was used as a anti-knock compound, allowing the development of higher efficiency, higher compression engines. GM started marketing Ethyl fluid in 1923. In 1924, it joined with Standard Oil to form a partnership called Ethyl Corp. Leaded gasoline allowed an increase in engine power and efficiency by raising fuel anti-knock quality – what today is called the "octane rating". By the 1930s, tetraethyl lead could be found in nearly 90% of all American gasoline as alternatives such as ethanol and changes in refining techniques fell by the wayside. It eventually was phased out by government order, starting in 1975 and concluding in 1986. Tetraethyl lead continues to be used as a gasoline additive in many underdeveloped countries. As a youth, the editor of the IME Bulletin recalls being intrigued by signage at the local Standard Oil station in his hometown touting an additive in the gasoline called "m^2pg" ("more miles per gallon"), presumably a reference to tetraethyl lead.
7) Exposure to lead may also occur from items brought into the home such as candy, folk and traditional medications (including alarcon, alkohl, azarcon, bali goli, coral, ghasard, greta, liga, pay-loo-ah, and rueda), ceramic dinnerware, and metallic toys and trinkets. In 2004, the Consumer Product Safety Commission recalled 150 million pieces of imported metallic toy jewelry sold in vending machines. In February 2006, a 4 year old Minnesota child died of lead poisoning after swallowing a charm from a pair of athletic shoes that consisted of 99.1% lead. Lead-soldered cans were a common source of lead exposure until the 1980's (in 1980, 47% of domestically produced food and soft drink cans were lead-soldered).
8) Minnesota has about 600,000 pre-1950 housing units. Approximately 50% of U.S. housing units built before 1950 are located in seven states (California, Illinois, Massachusetts, Michigan, New York, Ohio, and Pennsylvania). Homes built before the 1920's may also contain lead water pipes. Copper pipes soldered with lead came into use in the 1950's

and remain a source of lead contamination in drinking water in many homes (children may absorb more than 60% of lead dissolved in water; adults absorb 35-50%). Lead service lines connecting the city water supply to the house may also be a source of lead exposure in older housing.

9) About 1.2 million of the 24 million housing units still containing substantial lead paint hazards are occupied by families with young children.
10) The 1999-2002 National Health and Nutrition Survey found that the prevalence of elevated blood lead levels in children aged 1-5 years was 1.6% (approximately 310,000 children). The prevalence was highest (3.1%) in non-Hispanic black children aged 1-5 years. The mean blood level was 1.9 μg/dL for all children in this age group and was highest in non-Hispanic black children (2.8 μg/dL). In 2001, 1080 out of 47,096 tested Minnesota children (2.29%) were found to have elevated blood lead levels.
11) Recently resettled refugee children are also at increased risk for lead exposure due to living in older homes, acute and chronic malnutrition, and anemia (increases the absorption of lead). It has been suggested that all refugee children aged 6 months to 15 years be screened for elevated blood lead levels within 3 months of arrival. Follow-up testing of children <6 years of age should be performed 3-6 months after the initial screening. It has also been suggested that these children receive a pediatric multi-vitamin with iron daily to help reduce the absorption of lead.
12) Unfortunately, treatment of elevated blood levels has not been shown to reverse or diminish the cognitive impairment or other neuropsychological effects associated with lead. Children with a blood lead level greater than 20 μg/dL or have a level greater than 15 μg/dL for more than 3 months require an environmental investigation, possible lead hazard control, and follow-up blood lead testing. Blood lead levels greater than 45 μg/dL require exposure control and treatment with succimer. Children with symptoms of lead poisoning and/or blood lead levels greater than 70 μg/dL require hospitalization and treatment with parenteral EDTA.
13) In Minnesota as a whole, the percentage of children ages 9-30 months on Medicaid and MinnesotaCare who were tested for lead rose from 16% in 1999 to 29% in 2003.

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