Although immersion burns in hospitals are rare, staff should be informed of the hazards of exposure to institutional tap water, because other hospitals may also have to elevate their water temperature for infection control purposes. We believe that in evaluating tapwater temperature before the bathing of infants or older individuals lacking appropriate responses to pain, brief immersion of the elbow or hand should not be used as the sole measure of appropriate water temperature. Instead, an ungloved hand should be immersed for >10 seconds to assess water temperature. In addition one of two other options should be considered. A thermometer can be employed to determine if the temperature is between 36°C and 39°C.[9,10] A commercially available thermometer for this specific purpose is available (Frog Prince, Clinitemp Corp) with a light indicator on the heart of a frog at the correct bathing temperature of 101°F. Installation of a mixing valve with set temperature of no more than 50°C at the distal hot water supply can also be used. Periodic disinfection of such a valve appears to be sufficient to prevent *Legionella* overgrowth.[11]. These precautions should reduce the risk of potentially catastrophic immersion burns in hospitalized patients.

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Asymptomatic Children With Venous Lead Levels >100 μg/dL

ABBREVIATIONS. CDC, Centers for Disease Control and Prevention; RBC, red blood cell; FEP, free erythrocyte protoporphyrin; IM, intramuscular; BAL, British anti-Lewisite; IV, intravenous; $CaNa_2EDTA$, calcium disodium edetate.

Childhood lead poisoning is a preventable condition that may result in significant developmental disabilities and multi-organ involvement. Past research has shown that long-term neurocognitive deficits can result from lead exposure during the first few years of life.[1] In 1991, the Centers for Disease Control and Prevention (CDC) recommended that blood lead screening be instituted for all children between the ages of 6 months and 6 years, because subtle morbidity has been documented in children with levels as low as 10 μg/dL.[2] Critics of universal screening cite limited health care resources, parental anxiety, and the invasive nature of screening as reasons to modify the current CDC recommendations.[3] Although recent data show that the prevalence of lead poisoning has decreased in the United States, the risk to children has not been eradicated.[4] Because children with lead poisoning are almost always asymptomatic, screening is the only way to accurately determine the blood lead level. We report on three asymptomatic children in whom routine screening revealed blood lead levels >100 μg/dL.

CASE REPORTS

Patient 1

In May 1992, a 34-month-old African-American boy was found to have a capillary lead level of 278 μg/dL on routine screening. Due to a reporting delay, confirmatory venous lead level was not obtained until 1 month later at which time it was 177 μg/dL. The value was reverified at 172 μg/dL, and he was admitted to the hospital for chelation therapy. The child was asymptomatic with no history of anorexia, constipation, abdominal pain, irritability, ataxia, or mental status changes. The mother did report that he had been "hyper" during the week before presentation. His birth history, past medical history, and developmental history were unremarkable. The child had a history of pica, and had been observed to put his hands, paper, toys, clothing, plaster, and paint chips into his mouth. The child's physical and neurological examinations were within normal limits for age, and an abdominal radiograph was negative for radiopaque material. His admission laboratory evaluation included a venous lead level of 112 μg/dL, red blood cell (RBC) free erythrocyte protoporphyrin (FEP) 1597, and hematocrit 34%. He was admitted to the hospital and received chelation therapy. Because of his high lead level, his 5-1/2-year-old and 14-month-old siblings were screened and were found to have venous lead levels of <10 μg/dL and 23 μg/dL, respectively.

Patient 2

In March 1993, routine screening by the primary care provider revealed a capillary lead level of 101 μg/dL in a 23-month-old, asymptomatic, African-American girl. Several days later, a confirmatory venous sample showed a lead level of 102 μg/dL, RBC

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FEP 1570, and hematocrit 33%. Her birth history, past medical history, and developmental history were unremarkable. She had not been screened for lead before this time. The family had lived in their house for several generations, and it had peeling and flaking paint on the window sills, window frames, and exterior front porch. The child's crib was situated next to a window sill which was found to have tooth marks on it. She had also been observed to eat flowers and dirt. Other hand-to-mouth activity included sucking her fingers, biting her nails, and mouthing toys. There were no other children in the household; the adults in the home were screened and had blood lead levels <10 μg/dL. Her physical and neurological examinations were within normal limits for age, and an abdominal radiograph was negative for radiopaque material. After her venous lead level was confirmed, she was admitted to the hospital for chelation therapy. Subsequent health department inspection revealed that the areas of peeling paint in her home were heavily leaded.

Patient 3

In November 1994, a 26-month-old African-American boy was screened by his primary care provider and was noted to have a venous lead level of 78 μg/dL. Because of difficulty convincing the mother of the urgency of this matter, a repeat venous level was not obtained until 7 days later. This was found to be 138 μg/dL, and the child was admitted to the hospital immediately for chelation therapy. The child's past medical history was unremarkable, and his mother stated that he occasionally put things into his mouth. His capillary lead level at age 12 months had been <10 μg/dL. Two siblings also had previously documented lead levels <10 μg/dL. The child's physical and neurological examinations were within normal limits for age, and an abdominal radiograph was negative for radiopaque material. During the child's admission, his home was inspected by the Baltimore City Health Department and was found to be in poor repair with multiple areas of peeling paint and holes in the walls and roof. X-ray fluorescence of surfaces in the home revealed more than 50 areas of high lead content. Review of Baltimore City Health Department records showed that this dwelling had been documented to have lead hazard violations since 1983. The family had moved into this home 2 months before the child's admission. Repeat screening of his two younger siblings revealed that the 7-month-old had a lead level of 21 μg/dL, and the 19-month-old had a lead level of 41 μg/dL. The 19-month-old was also admitted to the hospital for chelation.

DISCUSSION

The three children in this case series had blood lead levels in excess of 100 μg/dL and were asymptomatic. Although lead encephalopathy has been seen in children with blood lead levels >100 μg/dL, it is impossible to predict in which children or at what point this might occur. The onset and clinical course of encephalopathy are unpredictable, and there may or may not be prodromal manifestations.[5] This is well-illustrated by a series of 293 children with lead poisoning who were treated between 1931 and 1970, most of whom had a blood level ≥100 μg/dL.[6] These children displayed a wide spectrum of presentations, ranging from no symptoms to en-

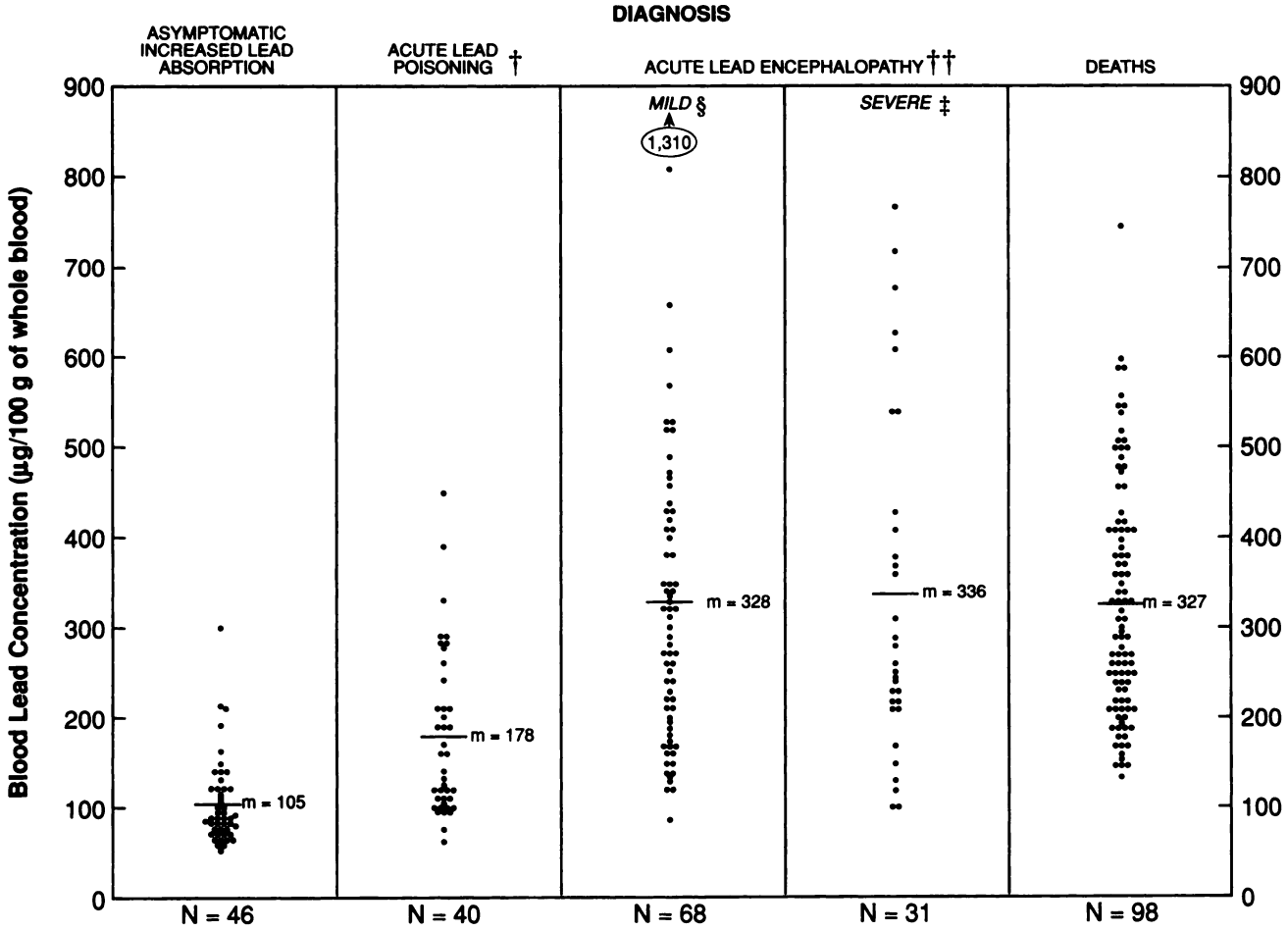

Figure. Spectrum of presentations in children with extremely high blood lead levels. †, Patients with clinical symptoms such as vomiting, anorexia, and/or constipation; ††, patients with neurologic signs such as ataxia, seizures, and/or altered state of consciousness; §, duration of encephalopathy ≤24 hours; ‡, duration of encephalopathy >24 hours. Adapted with permission from *Lead: Airborne Lead in Perspective*. Courtesy of the National Academy of Sciences, Washington, DC.

cephalopathy, and death (see Figure). In an experimental model of lead encephalopathy in rat pups, retention of lead in the brain persisted long after blood lead levels began to decrease, resulting in poor correlation between blood and brain lead levels.[7] The authors postulated that chronic lead ingestion in children results in progressive accumulation of lead in the brain. Therefore, the lead level at the critical organ site, ie, the cells in the brain, is most likely responsible for encephalopathy. There is no way to measure the brain lead level directly, nor to use the blood lead level to predict precisely when the brain's tolerance threshold will be surpassed.[8]

The three children described were asymptomatic and presented to the primary care provider for well-child visits; screening for lead was performed as part of routine health maintenance as delineated in the CDC guidelines. Lead poisoning is the number one environmental threat to children in the United States at the present time.[9] This condition can affect any child, regardless of gender, ethnic group, or socioeconomic status. Because the majority of children with lead poisoning are asymptomatic, screening of well children is the only accurate way to identify affected children. Lead screening is usually initiated between the ages of 9 and 12 months and repeated thereafter at a schedule dependent on the child's age, blood lead level, and risk of lead poisoning.[2] Blood lead levels are highest between the ages of 12 and 36 months, with the peak occurring at approximately 24 months.[10] Children in this age range are at highest risk due to their increasing mobility, tendency to crawl and play on the floor, age-appropriate hand-to-mouth activity (thumb sucking, mouthing of toys), and poor hygiene habits (eating food that has fallen on the floor). When screening reveals a high blood lead level in an index case, it is imperative that siblings and other young housemates also be evaluated. These three cases illustrate the importance of early screening, consistent follow-up, and screening of household members.

If a documented venous sample is obtained initially and is excessively elevated, it must be believed despite a lack of neurologic symptoms in the child. In each of these cases, there was delay while blood samples were repeated to confirm an elevated venous sample. The CDC recommends that a child with a blood lead level $\geq$70 μg/dL be chelated immediately, while awaiting confirmatory test results.[2] Venous samples should be used to confirm an elevated capillary sample; this must be performed immediately if the capillary level is $\geq$70 μg/dL.

The current CDC recommendation for chelation of a blood lead level $\geq$70 μg/dL is to use a combination of intramuscular (IM) British anti-Lewisite (BAL) and intravenous (IV) calcium disodium edetate ($CaNa_2EDTA$).[2] An initial dose of BAL 75 mg/m^2 is given alone by deep IM injection. At least 4 hours after the first dose of BAL is given, $CaNa_2EDTA$ 1500 mg/m^2/day is begun via continuous IV infusion. Adequate urine flow must be established before administering $CaNa_2EDTA$, because it is not metabolized and its elimination depends on renal excretion. BAL is then continued simultaneously at a dose of 450 mg/m^2/day in divided doses of 75 mg/m^2 IM every 4 hours. $CaNa_2EDTA$ can be mixed in dextrose and water or normal saline in a concentration that does not exceed .5%. In children with lead encephalopathy who have the potential for cerebral edema, the IV dose of $CaNa_2EDTA$ can be given IM to minimize the child's fluid intake. To assist with pain control, $CaNa_2EDTA$ can be mixed with .5% procaine, and EMLA cream can be used topically for IM injections.

In the past, $CaNa_2EDTA$ was used alone; this was associated with initial deterioration in some patients with extremely high blood lead levels, with a significant amount of associated morbidity and mortality.[2,11-13] Lead encephalopathy in children is manifested by a rapid onset of mental status changes, seizures, and cerebral edema. A child's risk of encephalopathy cannot be predicted from the blood lead level. This is the reason that children were placed in an intensive care unit setting for close observation during chelation. With the advent of combination BAL/$CaNa_2EDTA$ therapy and the use of the initial priming dose of BAL, clinical deterioration and precipitation of lead encephalopathy are minimized.[11] The theoretical risk of encephalopathy remains attributable to the high lead level itself; therefore, monitoring in the intensive care unit continues to be the recommended standard of care for these children.[2,5,8] Once a child has received 24 to 48 hours of chelation without developing neurologic abnormalities, he/she can be safely transferred to a nonacute hospital ward to complete treatment.

Some other precautions should be taken when treating children with these two agents.[2] First, physicians should be aware that BAL is suspended in peanut oil, which could cause anaphylaxis in children with peanut allergy. Second, BAL should be used with extreme caution and close monitoring in children with glucose-6-phosphate dehydrogenase deficiency, because severe hemolysis may result. Third, treatment of coexisting iron deficiency must be deferred until after chelation with BAL is completed, because iron combined with BAL can cause a severe toxic reaction.[11,14] This reaction consists of excessive emesis and cessation of the decrease in blood lead. Fourth, $CaNa_2EDTA$ must be used and not Na_2EDTA (disodium edetate), because the latter may induce tetany and fatal hypocalcemia. $CaNa_2EDTA$ should not cause any problems with calcium metabolism and no special monitoring of serum calcium levels is required.

A comprehensive environmental history should be taken in all cases of childhood lead poisoning, so that the source(s) of lead exposure can be identified and eliminated. Since lead-based residential paint continues to be the most common source of toxicant in the United States, questions should be asked about the condition of present and past dwellings. Families should be asked about recent moves, ongoing renovation/abatement in the home or neighborhood, and secondary addresses, such as babysitters or day care centers. The history should also cover other potential sources of lead such as water, ceramic glazes,

painted furniture or toys, folk remedies, and parental occupations/hobbies.

Once a child has been identified as having lead poisoning, a cascade of events is set into motion to alert the local health department, inspect the house, identify lead hazards, and notify the property owner of lead violations in the dwelling. Unfortunately, this system is backwards, because it requires that the child be poisoned and the risk of brain damage be present before homes are inspected and high-risk leaded environments identified. In addition, in many jurisdictions, there is no legislation which requires that lead violations be remediated. This is counter to the usual pediatric philosophy that places paramount importance on prevention of childhood morbidity and mortality. This is demonstrated by the third case, in which lead hazards had been documented in the home 10 years before the child's birth. The cost of proactive screening and abatement of houses would render moot the controversial topic of blood lead screening, and would be far less expensive than the societal costs incurred by the countless children who can be poisoned by a single dwelling.[15]

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The Natural History of a Treated Episode of Acute Otitis Media

The accurate diagnosis of acute otitis media is difficult, especially in its early hours. The vague symptoms and subtle physical signs of early otitis lead the practitioner to make too many diagnoses by default and to issue too much treatment out of caution. With the growth of resistant organisms limiting the effectiveness of safe, low-cost antibiotics, there is increasing pressure to prescribe antibiotics more sparingly.[1-3] Thus, the accurate diagnosis of otitis media is more important than ever.

To correctly diagnose acute otitis media the pediatrician must know what it looks like, not only in its early hours, but throughout the course of infection. Several authors have described the appearance of otitis media through its phases of hyperemia, exudation, suppuration, and resolution.[4] None of these

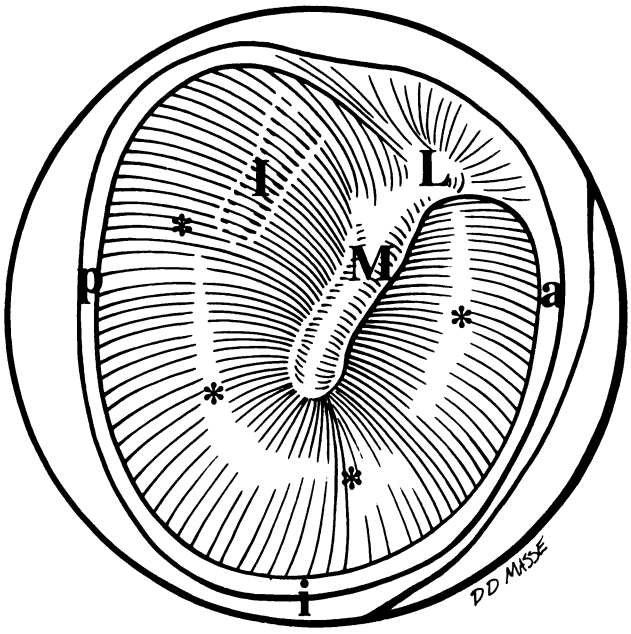

Fig 1. Drawing of a normal right tympanic membrane. Notice the outward curvature of the pars tensa (*) of the eardrum. The tympanic annulus is indicated anteriorly (a), inferiorly (i), and posteriorly (p). M = long process of the malleus; I = incus; L = lateral (short) process of the malleus.

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