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Childhood Lead Poisoning Prevention

Too Little, Too Late

Bruce P. Lanphear, MD, MPH

One hundred years ago, Gibson described an epidemic of childhood lead poisoning from the ingestion of lead-based paint.[1] He showed that paint was the primary source of lead intake for these children by measuring lead on wipe samples collected from porch railings and houses that had recently been painted. Gibson speculated that educational efforts would prevent lead poisoning because many children with lead poisoning were reported to bite their nails or suck their fingers.[1] Four years later, after their educational efforts failed to prevent lead poisoning, Gibson's colleague, Turner, concluded, "Prevention is easy. Paint containing lead should never be employed . . . where children, especially young children, are accustomed to play."[2]

Despite these and other warnings, the United States continued to allow the use of lead-based paint until 1978.[3] In contrast, many European countries banned the use of lead-based paint as early as 1909.[4] The delay in banning lead-based paint in the United States was due largely to the marketing and lobbying efforts of the lead industry.[3,4] In 1984, Mayer, then president of the Lead Industries Association, boasted, "Our victories have been in the deferral of implementation of certain regulations."[5]

Prior to 1970, lead poisoning was defined by a blood lead concentration of 60 µg/dL or higher—a level often associated with overt signs or symptoms such as abdominal colic, anemia, encephalopathy, or death.[6] Since then, the blood lead concentration for defining lead toxicity gradually has been reduced from 60 µg/dL to 40 µg/dL in 1971, to 30 µg/dL in 1978, and to 25 µg/dL in 1985. In 1991, the Centers for Disease Control and Prevention (CDC) further reduced the definition of undue lead exposure to a blood lead concentration of 10 µg/dL or higher.[6]

See also p 2232.

Over that time, children's blood lead concentrations have declined dramatically. In the 1970s, 88% of US children younger than 6 years were estimated to have a blood lead concentration of 10 µg/dL or higher.[7] When lead was at long last banned from paint in 1978 and the reduction of lead in gasoline was started in the 1970s, children's blood lead levels began to decline almost immediately.[7] By the early 1990s, fewer than 5% of children younger than 6 years were estimated to have blood lead concentrations of 10 µg/dL or higher.[8]

Despite the dramatic decline in children's blood lead concentrations, lead toxicity remains a major public health problem. Environmental lead exposure in children—typically measured using lead in whole blood or teeth—has been associated with an increased risk for reading problems, school failure, delinquency, and criminal behavior.[9-14] Moreover, there is no evidence of a threshold for the adverse consequences of lead exposure.[15,16] Indeed, studies show that the decrements in intellectual function are, for a given increase in blood lead concentration, greater at blood lead levels lower than 10 µg/dL,[15,16] the level considered acceptable by the CDC.

The effects of lead exposure extend beyond childhood. In adults, lead exposure—measured in bone using an x-ray fluorescence analyzer or in whole blood—has been associated with some of the most prevalent diseases of industrialized society: cardiovascular disease,[17-19] tooth decay,[20] spontaneous abortion,[21] renal disease,[22] cognitive decline,[23,24] and cataracts.[25] Much of the lead found in adults was deposited decades ago. Thus, regulations enacted in the 1970s were too late to prevent lead-associated morbidity and mortality for many adults.

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Childhood lead toxicity is now concentrated in 2 groups: impoverished children who live in older, poorly maintained rental property and more affluent children whose families renovate older housing.[26-29] From 1999 to 2001, the CDC estimated that 430 000 (2.2%) preschool-aged children in the United States had a blood lead concentration of 10 µg/dL or higher.[30] In some cities, especially those in the Northeast and Midwest, the prevalence of children with blood lead levels exceeding 10 µg/dL is considerably higher.[26-28] African American children and, to a lesser extent, Hispanic children also have significantly higher blood lead levels than white children do, even after accounting for social, behavioral, nutritional, and environmental factors.[8,31]

In 1997, the CDC shifted away from universal screening and recommended targeted blood lead screening for children who were at high risk for lead exposure.[32] In 1998, the American Academy of Pediatrics issued similar recommendations.[33] The rationale for targeted screening of high-risk children was to focus resources on children who would especially benefit, such as children who received Medicaid.[34] Until now, there have been too few data to assess whether high-risk children who are identified as having elevated blood lead levels are being adequately tested.

In this issue of *JAMA*, Kemper and colleagues[35] report that 46% of children who had blood lead levels indicative of lead toxicity (≥10 µg/dL) did not receive adequate follow-up testing. Although follow-up testing was better for children who had blood lead levels of 45 µg/dL or higher, 20% of these children did not receive follow-up testing. Moreover, the authors reported that children who were at highest risk for lead toxicity—urban and minority children—were the least likely to receive follow-up testing, even though 58.6% of the children had at least 1 medical encounter in the subsequent 6 months.

The problems identified by Kemper et al are only the tip of the iceberg. A child identified through screening to have an elevated blood lead level already has an elevated risk for the persistent effects of lead toxicity.[9-16,36] Moreover, by 2 years of age—when children's blood lead levels typically peak and they are consequently identified as having an elevated blood lead level—children are already growing out of their mouthing behaviors and unlikely to benefit from any environmental interventions.[37] Thus, intervening only after children's blood lead levels exceed 10 µg/dL fails to protect them from the adverse consequences of lead toxicity.[38] Furthermore, as noted by Kemper et al, lead toxicity may underlie some of the prevalent health disparities found in socially disadvantaged children. Indeed, the social disparities in lead exposure may partly explain elevated rates of school failure, tooth decay, and criminal behaviors found among children in impoverished communities.[10-14,20]

The problem identified by Kemper et al is a symptom of a fragmented health care system, a system in which public health functions and medical care are largely divorced. Physicians are trained to provide clinic-based diagnosis and treatment. The prevention and management of common pediatric diseases with recognized environmental risk factors, such as lead poisoning, asthma, and injuries, require regulatory actions and close interactions with public health officials; the prevention of such diseases is not amenable to drug therapy or anticipatory guidance.[39,40]

Primary prevention of childhood lead poisoning from residential lead hazards is long overdue. Despite conclusive evidence that regulatory efforts were responsible for the dramatic decline in lead poisoning—and the early warnings by Gibson and Turner—educational efforts such as passing out brochures and mop buckets inexplicably continue to be emphasized, rather than the need for promulgation of regulations to protect children from residential lead hazards. Moreover, effective prevention interventions are typically withheld until after a child's blood lead concentration exceeds 15 µg/dL. The key to primary prevention is to require screening of high-risk, older housing units to identify lead hazards before a child is poisoned—before occupancy and after renovation or abatement. Voluntary recommendations will inevitably fail. Screening and follow-up testing of high-risk children will remain an important part of lead poisoning prevention programs, but they should serve as a safety net, not the focus. Unfortunately, public health and housing agencies lack the resources they need to protect children from lead poisoning, and even when they do act, the study by Kemper and colleagues is a cogent reminder that it is too little, too late.

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Table 1. Distribution of Patients Into 10 Most Common Cause-of-Death Categories by Method*

Cause-of-Death Categories	Method, %		
	Death Certificate	Last Diagnosis	Cost
Diseases of heart	29.9	22.3	20.7
Malignant neoplasms	26.9	24.4	24.1
Chronic obstructive pulmonary disease	7.3	7.5	7.1
Cerebrovascular diseases	5.5	5.0	4.9
Diabetes mellitus	3.2	5.1	5.4
Pneumonia and influenza	3.0	6.3	6.4
Chronic liver disease and cirrhosis	2.4	2.3	2.4
Alzheimer disease	1.1	1.3	1.1
Nephritis, nephrotic syndrome	0.9	4.0	3.9
Other diseases of arteries, arterioles, and capillaries	0.9	1.4	1.4
All other causes	18.8	20.5	22.5

*Because of rounding, percentages may not all total 100.

Table 2. Patient-Level Agreement Between Methods for the Top 2, 4, and 10 Cause-of-Death Categories

Cause-of-Death Categories	Comparison of Methods Agreement, %		
	Death Certificate vs Last Diagnosis	Death Certificate vs Cost	Last Diagnosis vs Cost
Top 2	68.8	67.8	84.3
Top 4	61.2	60.2	80.7
Top 10	51.8	50.4	76.5

cally have multiple diseases near the end of life,[4] and the underlying cause of death may have little relation to the condition that necessitates the majority of health care utilization near death.[1] The last-diagnosis method may be optimal in studies analyzing care very close to the end of life, because it best represents the reason for health care utilization closest to death. The cost method could result in more reliable classification than the last-diagnosis method, because it takes utilization directly into account. However, the cost method is sensitive to differences in practice patterns and insurance coverage and could be affected heavily by expensive procedures. When using the last-diagnosis and cost methods, there is the potential for errors if the validity of the diagnosis or cost data has not been established.

Although the methods classified similar percentages of patients into top cause-of-death categories, they did not place the same patients into each category. None of these methods is adequate in capturing the complete story of a decedent's cause of death, especially given the increasingly high proportion of deaths that have multifactorial causes.[1] However, each method provides potentially useful information on case mix at the end of life and researchers should consider the objectives of their study, the feasibility of applying each method, and the reasons they need to identify cause of death when deciding which method to use. When critically interpreting these studies, readers should also consider these issues.

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CORRECTIONS

Data Error: In the Editorial entitled "Childhood Lead Poisoning Prevention: Too Little, Too Late" published in the May 11, 2005, issue of *JAMA* (2005;293:2274-2276), there was a data error. On page 2275 in the third paragraph, the second sentence should read: "Although follow-up testing was better for children who had blood lead levels of 45 μg/dL or higher, 6% of these children did not receive follow-up testing."

Name Mispelled: In the Research Letter entitled "ABO Blood Group and Susceptibility to Severe Acute Respiratory Syndrome" published in the March 23/30, 2005, issue of *JAMA* (2005;293:1450-1451), an author's name was mispelled. The correct name is Yunfeng Cheng, MD, PhD.

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