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Primary Prevention of Childhood Lead Poisoning — The Only Solution

LEAD poisoning in children was first recognized in its severe acute form, known as lead encephalopathy.[1] This condition is characterized by seizures, coma, and — not infrequently — death, and it is associated with severe neurologic sequelae in survivors. Although lead encephalopathy has become rare in the past 15 to 20 years, the dangers of clinically asymptomatic lead poisoning in children have become increasingly clear. Longitudinal studies of development from birth to adolescence show that irreversible cognitive damage can occur with blood lead levels considerably lower than those typically associated with overt symptoms.[2,3] Recognition of this problem has led to routine screening programs and clear guidelines for the management of severe lead poisoning (defined by whole-blood lead levels of 45 μg per deciliter or more). In contrast, the appropriate management of moderate lead poisoning (defined by whole-blood lead levels of 20 to 44 μg per deciliter) remains uncertain.

In this issue of the *Journal,* Rogan et al.[4] report the results of a multicenter, randomized trial of an oral lead-chelating agent, succimer, in children whose blood lead levels were 20 to 44 μg per deciliter. The performance of these children on several cognitive assessments was below average — an observation consistent with previous reports of deficits in academic achievement, abstract thinking, attention span, conceptual reasoning, and visuospatial perception in children with moderately high blood lead levels.[5] Although chelation therapy lowered blood lead levels, it had no effect on any of the several neurobehavioral and cognitive measures used in the study. The study suggests that even with succimer therapy, the neurocognitive effects of chronically elevated blood lead levels and total-body lead burden are irreversible. Such irreversibility of developmental deficits due to neurotoxicity has been documented previously in children who did not receive chelation therapy.[6,7]

Chelation therapy was initially introduced to manage severe, and frequently acute, lead poisoning that would most likely result in death or severe, overt neurologic sequelae in survivors.[1] In affected children, whose blood lead levels were often greater than 100 μg per deciliter, chelating agents rapidly lowered blood lead levels by brisk diuresis and stopped the progression of lead poisoning to the point of frank encephalopathy.[8,9] This treatment saved lives but did not eliminate the neurologic consequences, which were permanent. Very rarely, if ever, does chelation therapy for such severe lead poisoning reverse or prevent the signs and symptoms of lead-induced neurotoxicity. Rather, the benefit of chelation therapy for children with severe acute or chronic lead poisoning is defined in relation to the terrible prognosis for children who are not so treated.

Chelation therapy is now used routinely in children who have blood lead levels of 45 μg per deciliter or more, with the goal of preventing neurologic deterioration, lead encephalopathy, and death. However, there have been no clear guidelines from the Centers for Disease Control and Prevention or other advisory bodies regarding therapy for children with blood lead levels of 20 to 44 μg per deciliter,[6,7] who were represented by the participants in the present study.[4]

The study by Rogan et al. used the most promising chelating agent, succimer, and what was considered to be a highly effective dosing regimen. We believe it unlikely that any other study design, study population, chelating agent, or dosing regimen would produce a materially different result. Neurocognitive tests assessed measures characteristically affected by lead toxicity. The study population accurately reflected, both demographically and socioeconomically, the children at highest risk in urban communities in the United States.

The lack of efficacy of succimer for the prevention of neurotoxic harm due to moderate blood lead levels in the children in the study by Rogan et al. and the limited efficacy of chelation therapy in cases of severe lead poisoning clearly cast doubt on the value of public health programs that rely primarily on treatment after lead poisoning has occurred. Rogan et al. emphasize the importance of the primary prevention of lead poisoning, which is the only satisfactory solution to this devastating problem. The predominant source of toxic exposure to lead for children in urban areas is lead paint,[10] although some incremental

but far less substantial toxicity may be due to other sources, such as tap water contaminated by lead pipes.

For the primary prevention of lead poisoning from paint, we recommend permanent abatement — that is, the complete removal or replacement of lead paint before a child lives in a home.[11,12] In contrast, "interim" measures, which were introduced for the short-term reduction of hazards associated with lead paint[11,13] and which involve scraping and painting over deteriorated surfaces and controlling household dust, have been claimed by some to save substantial cost; however, there is no evidence of savings in terms of net benefit over cost in the long-term prevention of childhood lead poisoning. Lead-painted surfaces in good condition rarely remain so. What was once intact lead-based paint is the source of all lead-bearing dust and paint chips. Therefore, it is the presence of lead paint on surfaces that defines the hazard, not the condition of surfaces containing lead paint.

Although succimer therapy resulted in lower blood lead levels, its failure to reverse neurocognitive deficits in the study by Rogan et al. confirms the need for collective and concerted efforts to prevent lead poisoning in children.

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RACIAL DISPARITIES IN MEDICAL CARE

MANY studies have shown that black Americans are less likely than whites to receive a wide range of medical services, including potentially life-saving surgical procedures.[1,2] Despite these data, which span two decades, there is little evidence that racial disparities in medical care or in measures of health have substantially diminished. Black Americans have an average life expectancy that is 6 years shorter than that of white Americans, and there has been little change in this disparity during the past 30 years.[3]

Why has the evidence on racial disparities failed to galvanize a response within the medical community? Four factors have contributed to the slow pace of change. First, much of the evidence has been based on administrative data, leading some clinicians to believe that racial disparities reflect unmeasured differences in clinical or socioeconomic factors. Second, clinicians may believe that whites are more likely than blacks to prefer intensive medical treatment or surgical therapy. Indeed, some research has suggested that there are such differences in preferences according to race, although the differences are small and probably cannot explain large racial differences in treatment.[4] Third, few studies have evaluated differences in clinical outcomes that may be directly related to disparities in the use of medical services or surgical procedures or have examined whether black Americans are less likely than whites to receive clinically important, needed services.[5-8] Fourth, even if the will to reduce racial disparities is strong, the pathways from intervention to improved quality of care have not been well charted.[9]

Despite these uncertainties, racial disparities in medical care are particularly troubling for at least two reasons: the possibility that they reflect discrimination or racial bias on the part of physicians and their potentially deleterious effects on health outcomes. The article by Chen et al.[10] in this issue of the *Journal* provides information relevant to both of these points and represents an advance in our understanding of racial disparities in medical care. Approximately 40,000 Medicare beneficiaries who were hospitalized in 1994 or 1995 with acute myocardial infarction were followed to assess whether they had undergone cardiac catheterization within 60 days after hospital admission and to determine their rates of death during the ensuing 3 years. White patients were significantly more likely than black patients to undergo cardiac catheterization. However, this racial difference in care did