in those organizations also has proved to have merit, both for improving outcomes and reducing costs, especially those of hospitalization.) As Perrin et al point out, even relatively simple and familiar home-visitor programs have tremendous potential for improving services and reducing morbidity and cost. Such services can often be provided by specially trained community workers working alongside traditional professionals in a very cost-effective manner.

There are many other existing examples and demonstrations of the possible changes in service delivery systems which can improve the quality of services to children with chronic illnesses; these will need to be pursued even in a better funded environment. Clearly, different solutions will have to be sought in different regions of the United States, among differing populations, and in the wide variety of new financing schemes. We should not come away from the discussion of Newacheck et al with the impression that if we do all they recommend, the problems of medical services to children with special needs will be solved. Without accompanying changes in service delivery coordination, the major barriers to many of these children's optimal health care will remain unaddressed, and their plight little improved. And, although short-term costs may be moderated, long-term costs of continuing morbidity and lost opportunities for these children as productive citizens will continue to grow. Many, if not most, of the services which children with special health care needs require do not fall into the realm of direct medical services; some are in the loosely defined "public health" sector, whereas others have been put together from bits and pieces of public and private maternal and child health and social service programs. Even with universal insurance, free of "prior existing condition" clauses, these children and their families will still need to work tirelessly to find and keep the necessary services unless attention is paid to the system of care delivery on which they depend. Those of us who care for and about this large, growing group of children must remain committed to them in the political as well as in the medical arena. The need for our advocacy on their behalf will not go away with health care reform; indeed, the reform process demands heightened vigilance and creativity.

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REFERENCES

1. Newacheck PW, Hughes DC, Stoddard JJ, Halfon N. Children with chronic illness and Medicaid managed care. *Pediatrics*. 1994;93:497–500
2. Perrin JM, Kahn RS, Bloom SR, et al. Health care reform and the special needs of children. *Pediatrics*. 1994;93:504–506

Childhood Lead Poisoning in the '90s

Lead is an extremely toxic metal: even a single atom of lead, once in the human body, binds to a protein and induces some damage; the greater the exposure, the more serious the effects. Lead has no physiological function; any amount of body lead reflects environmental pollution.[1]

In the 1990s, in the United States, is lead poisoning a devastating environmental threat to our children or is childhood lead poisoning a threat of the past? Probably neither of these statements is correct. It may be worthwhile to look at the current situation in detail.

In the 1970s, before efforts were made to reduce environmental lead, common effects in children of the widespread environmental contamination with lead were encephalopathy and even death. In 1976 the National Health and Nutrition Examination Survey (NHANES II) 1976–1980 study estimated the average blood lead level for the entire US population at 16 μg/dL.[2] Preliminary data from the ongoing NHANES III study indicate that the average blood lead level of the US population is now <4 μg/dL. For comparison, an average blood lead level of 3 μg/dL was observed by us in children living in the pristine air of the highlands of the Himalayas, far away from industrialization and civilization.[3] Even for children of low socioeconomic class living in upper Manhattan, the average blood lead level, that was 19 μg/dL just a few years ago, has now declined to 5 μg/dL.[4] Clinically overt lead poisoning has essentially disappeared, and it is now so rare that it can probably be called a threat of the past. We have gone a long way toward elimination of environmental lead and of severe childhood lead poisoning.

What is the cause of this decline? It would be gratifying to this author to think that it resulted from the widespread screening of US children, utilizing the erythrocyte porphyrins test developed in his laboratory. The real reason for the nationwide decline of blood lead, instead, is the reduction of lead from many sources, and, most of all, the near-complete elimination of lead from gasoline. In the heated arguments at the Environmental Protection Agency for the revision of the air quality standard for lead in the 1980s, none of us who argued for a lower standard anticipated that the effect of removing lead in gasoline was going to be so extensive. It is ironic that the removal of lead from gasoline was decreed to protect not the children, but the automobile's catalytic converter. On the other hand, the reduction of blood lead levels with the reduced environmental contamination with lead confirms the ancient saying, . . . *an ounce of prevention* . . . . The removal of lead has reduced background exposure and thus reduced the blood lead level of all US children; it has made them much less likely to attain lead levels associated with overt toxicity. Certainly,

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the tragic situation of the 1970s has been largely remedied. The reduction of childhood lead poisoning represents a rare triumph against manmade ills. All those who contributed to this goal should be justly proud.

In view of this incredible success, should we now ignore the residual environmental lead? Although the environmental situation has improved, several studies indicate that subtle, but measurable, effects of lead are detectable even at extremely low levels. This was predictable based on our understanding of the toxicity of lead. It has been known for years that there is no threshold for the effects of lead, when searched by refined biochemical techniques. The pioneer work of Hernberg, in Finland, demonstrated that the enzyme δ-aminolevulinic acid dehydratase is damaged at any level of lead exposure.[5] The accumulation of δ-aminolevulinic acid underlies some of the neurological toxicity of lead. Excess δ-aminolevulinic acid is excreted in the urine, and excess erythrocyte protoporphyrin accumulates in the red blood cells with an exponential increase in relation to blood lead levels.[6]

Several epidemiological studies have shown that some adverse effects of lead on the neuropsychological function are also detectable at low levels of exposure. The initial study of H. Needleman in this area[7] became the center of controversy at the hearings for lead air quality standards at the Environmental Protection Agency; those acrimonious arguments continued in various forums. Needleman's work, however, has been vindicated by the fact that his observations have essentially been confirmed by other independent investigators. These issues have been critically reviewed recently by the National Research Council.[8]

Although there is no doubt that detectable adverse effects of lead are demonstrable at very low levels, two questions must now be addressed: (1) at what level of exposure do the adverse effects of lead become trivial, and (2) what measures, if any, should be taken to reduce exposure to lead, in view of the current situation?

There is substantial epidemiological evidence of adverse effects of lead at blood levels <20 μg/dL.[8,9] Harvey has argued, however, that "evidence that BPb levels <20 μg/dL at age one year cause a clinically important decrease in intelligence and increase in neuro-behavioral problems by the time the child enters school is lacking."[10] We know that the neurobehavioral effects of lead tend to be permanent and prudence advises that, when in doubt, to consider the adverse effects as potentially permanent. On the other hand, one cannot ignore that the adverse effects of very low lead exposure are certainly minor.

Can we tell our education-conscious parents that a loss of 2 to 3 IQ points is trivial? Our society undoubtedly has the responsibility to protect all our children from every avoidable threat. But, *what is the best approach to optimally protect our children from lead poisoning*? This is the crucial question. As in any other case, some consideration should be given to cost/benefit ratio. This should not be limited to its pecuniary aspect; parental anxiety, validity of intervention, and disruption of life should be also weighed against potential benefits of screening.

The Centers for Disease Control (CDC) published an ambitious "Strategic Plan for the Elimination of Childhood Lead Poisoning"[1] in 1991. This document made it clear that the most urgent priority in the United States is the correction of the thousands of dwellings in which lead paint represents a constant hazard for children. However, the same document already contained the self-fulfilling prophecy that the blood lead level for intervention was going to be decreased further by a forthcoming CDC experts committee. There is no doubt that the residual sources of lead should be eliminated, if the residual risk of lead is to be eradicated. Yet, given the opportunity, the CDC chose not to focus on the cause of lead poisoning, but on its potential victims. A committee of experts (including the author) was assembled by CDC and was charged to make a recommendation, without regard to practical considerations such as cost and or effectiveness. As a result, a well-meaning, but abstract recommendation was made to lower the threshold blood lead level for intervention to 10 μg/dL.[9] An ambitious, expensive program thus has been launched for the universal screening of all children, whether living in a lead-laden environment or not.

To make matters more complex, current technology can not adequately measure with accuracy blood lead levels in the 10 to 15 μg/dL range. Even in the best laboratories the error of the measurement is at least 2 to 3 μg/dL. This high degree of inaccuracy requires that a blood lead level in this range be reverified soon, and often the classification must be revised downward.

On the other hand, even if the blood lead level were 10 to 15 μg/dL, its detection would accomplish no useful purpose for the individual child. In that range, the demonstration of minor effects of lead requires large numbers of children and sophisticated techniques of detection and analysis.[8] Yet, with the current recommendation, pediatricians must explain to petrified parents that their child has been found by screening to have lead poisoning, as defined by CDC, the custodian of US health. The same wording, lead poisoning, is used indiscriminately for a child with a blood lead level of 11 μg/dL as for a child with a blood lead level of 110 μg/dL. In most human illnesses in which there is a continuous degree of severity, this is acknowledged to the patient by the physician (moderate hypertension, severe hypertension, etc). In the CDC statement, instead, lead poisoning becomes an all or none phenomenon; but the recommendations for management are quite different, depending on its severity. Similarly, adverse effects of lead are grouped together indiscriminately, regardless of their impact. These broad definitions result in catastrophic statistics about lead poisoning, without distinguishing how many of the children counted are indeed significantly affected.

It seems that we have not learned from our experience in this area. The reduction in blood lead levels in the United States has resulted not from extensive screening, but from the removal of the most

important sources of environmental lead. Screening is critical only in the high-risk areas, but is very inefficient in communities without lead exposure. Instead of testing all the children and declaring many of them poisoned, it would be more logical today to test all the houses in which children live that were built before lead-based paint was eliminated. Several communities have laws mandating window guards in all apartments where young children live or are moving in. It would be more effective if state legislatures, instead of decreeing mandatory screening of all children <6 years, voted instead to establish mandatory inspection of all dwellings built before 1950 in which those children may live. State and federal funds would be better spent to help home owners and landlords remedy the residual lead risk, rather than undertaking the expensive and useless screening of children who are not at risk.

The situation has been well synthesized recently by Oski, "It would be prudent to spend the money on prevention rather than detection."[12]

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REFERENCES

1. Patterson CC. Contaminated and natural lead environments of man. *Arch Environ Health*. 1965;11:344–348
2. Annest JL, Pirkle JL, Makug D, Neese JW, Bayse DD, Kowar MG. Chronological trend in blood lead levels between 1976 and 1980. *N Engl J Med*. 1983;308:1373–1377
3. Piomelli S, Corash L, Corash MB, et al. Blood lead concentrations in a remote Himalayan population. *Science*. 1980;210:1135–1137
4. Piomelli S, Flaster E, Seaman C, Carriero D. Decrease in the average blood lead level in a population of middle class urban children between 1985 and 1991. *Pediatr Res*. 1992;31:99A
5. Hernberg S, Nikkanen J. Enzyme inhibition by lead under normal urban conditions. *Lancet*. 1970;1:63–64
6. Piomelli S. Lead poisoning. In: Nathan D, Oski F, eds. *Hematology of Infancy and Childhood*. 4th ed. Philadelphia, PA: WB Saunders and Co; 1992:472–494
7. Needleman HL, Gunnoe C, Leviton A, et al. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *N Engl J Med*. 1979;300:689–695
8. National Research Council, Board on Environmental Studies and Toxicology. *Measuring Lead Exposure in Infants, Children and Other Sensitive Populations*. Washington DC: National Academy Press; 1993
9. Centers for Disease Control. *Preventing Lead Poisoning in Young Children*. US Dept of Health and Human Service, Public Health Service, Centers for Disease Control; 1991
10. Harvey B. Should blood lead screening recommendations be revised? *Pediatrics*. 1994;93:201–204
11. Centers for Disease Control, Risk Management Subcommittee. *Strategic Plan for Elimination of Childhood Lead Poisoning*. US Dept of Health and Human Service, Public Health Service; 1991
12. Oski FA. Lead poisoning—what are the facts? *Contemp Pediatr*. 1993;10:145–156

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