

On the basis of these findings, it seems reasonable to introduce chelation protocol for newborns with elevated lead levels to prevent both free radical damage and the late onset of neurological illness caused by subclinical toxicity of lead. In the neonatal period it is relatively easy to implement lead screening because blood from the umbilical cord is available at delivery.

D-Penicillamine (DPA) "would be an appropriate or the preferred chelating agent" for the treatment of lead poisoning in neonates too, presuming that this protocol "begins and ends with removing lead from the child's environment."

Our DPA research embraces a period of more than 20 years. In our pediatric departments we have had experiences in the DPA therapy of neonatal jaundice since 1973. In several papers we have reported that the use of this drug in premature infants is associated with a marked decrease in the incidence of severe retrolental fibroplasia. Infants were given three daily doses of 100 mg of DPA/kg body weight intravenously for 3 days in treating neonatal hyperbilirubinemia. Subsequently, the very low birth weight infants continued to receive a dose of 50 mg/kg of DPA once daily intravenously until the end of the second week of life to prevent retinopathy.⁴ In the last 19 years, we have treated approximately 20 000 term and preterm infants with DPA in Hungary, observing neither acute nor long-term adverse effects nor any late complications during several years follow-up. The drug has no bilirubin-displacing effect on the albumin-bilirubin complex, and none of the side effects described in adults have ever been encountered in our patients.⁵⁻⁷

I believe that these clinical observations provide sufficient evidence for a controlled trial, which we intend to perform in the immediate future, to determine the effectiveness of DPA in reducing elevated blood lead level in newborns and to analyze results relating to the long-term outcome of these babies.

I look forward to reading about the comments of pediatricians familiar with lead-related issues.

LAJOS LAKATOS, MD, DSc
Department of Pediatrics
Kenézy County Hospital
4043 Debrecen, Bartók u.4.
Hungary

REFERENCES

1. Glotzer DE, Bauchner H. Management of childhood lead poisoning: a survey. *Pediatrics*. 1992;89:614-618
2. Needleman HL, Jackson RJ. Lead toxicity in the 21st century: Will we still be treating it? *Pediatrics*. 1992;89:678-680
3. Goyer RA. In: Seiler HG, Sigel H, eds. *Handbook on Toxicity of Inorganic Compounds*. New York: Marcel Dekker Inc; 1988:360-382
4. Lakatos L, Oroszlá Gy, Lakatos Zs. D-Penicillamine in the neonatal period. In: Stern L, Orzalesi M, Friis-Hansen B, eds. *Physiologic Foundations of Perinatal Care*. New York: Elsevier; 1989;3:188-198
5. Lakatos L, Szabó I, Csáthy L. The effects of D-penicillamine on the renal and liver functions in neonates and the in vitro influence on granulocytes. *Acta Paediatr Scand Suppl*. 1989;360:135-139
6. Vekerdy Zs, Lakatos L, Oroszlán Gy, Itzés B. One year longitudinal follow-up of premature infants treated with D-penicillamine in the neonatal period. *Acta Paediatr Hung*. 1987;28:9-16
7. Vekerdy-Lakatos Zs, Lakatos L, Itzés-Nagy B. Infants weighing 1000 g or less at birth outcome at 8-11 years of age. *Acta Paediatr Scand Suppl*. 1989;360:62-67

To the Editor.—

The commentary offered by Needleman and Jackson¹ has raised some interesting points for discussion regarding the mythology surrounding the issue of lead poisoning and its treatment. Whereas the opening statement of the authors suggests that standards of practice are falling behind the scientific understanding of lead, the article under discussion by Glotzer and Bauchner makes it clear that both the science and standards of practice are very much lacking in the area of treatment for low level lead exposure. For a number of years it appears that our research efforts have focused on the impact of lead on children, and studies regarding therapeutic intervention have lagged well behind. It is time to place an emphasis not only on preventive aspects, but also to gain

prospectively information regarding the relative risks and benefits of treatment for low level lead exposure, particularly with newer chelating agents such as succimer.

A second and perhaps more troublesome aspect of this article is the call-to-arms for funding to begin the deleading process for housing in the United States. Certainly tremendous strides have been made by the reduction of lead in gasoline and improvements in a number of factors such as nutrition. The interaction of lead with environmental factors such as nutrition and child rearing practices has been a subject of over-debate, perhaps. One consistent thread in all the studies relating intelligence to lead has been the finding that maternal and socioeconomic factors are strong predictors of intellectual outcome. A recent study published in abstract³ suggests that Head Start Programs can obviate the impact of lead on intellectual development. This study will require scrutiny after peer review publication but it does support an idea that seems generally believed in the medical community: preventive health programs such as Head Start and The Special Supplemental Food Program for Women, Infants, and Children (WIC) remain our best lines of defense against the multifactorial effects of poverty on intellectual development. Historically, our government has taken the approach of reacting to public pressure to fund interventional programs by shifting dollars from existing programs. This may be by subtly changing eligibility requirements or allowing the effects of inflation to gradually erode the numbers of individuals eligible for these programs. As we consider the needs of this nation to undertake lead abatement, it is absolutely critical that we guard against the "band wagon" approach that may undermine programs likely to have a greater impact on improving children's health than lead abatement.

A further issue not addressed by this commentary is that of regional priorities. Although we must all accept responsibility for being citizens of the world and nation, it is also important to retain some advocacy for the children of our region. In response to the Centers for Disease Control (CDC) guidelines,⁴ we were concerned that a large amount of current resources might be diverted toward lead screening in a state where virtually no lead screening currently is undertaken. To assess our needs we undertook a survey of children in high risk areas of our inner city. Children from lower socioeconomic status living in parts of the city with older construction were screened after informed consent was obtained. In 261 children screened to date, we have failed to identify any children with a blood lead in excess of 15 µg/dL and only 4.2% in excess of 10 µg/dL. Coincidentally, during the time of the screening study, public health officials were restricting school recess activities to indoors because of concerns regarding the air quality in our region. The recent CDC guidelines state that all US children should be screened for lead "...unless it can be shown that the community in which these children live does not have a childhood lead poisoning problem."⁴ It is unclear to us, according to this statement, whether we can abandon routine screening in our area based on our findings. The decision is not trivial as budgetary constraints will likely require shifting of the costs of screening from existing programs.

In summary, the challenge for the individual child is to develop effective and safe strategies for dealing with heavy body burdens of lead. Our public health challenge is to develop an overall program to improve the health of children in poverty rather than allowing a single issue to take precedence over others. A third challenge is to allow public policy to reflect regional priorities to avoid divisive arguments within the community of pediatricians.

WILLIAM BANNER JR., MD, PhD
Divisions of Critical Care and Clinical
Pharmacology
Department of Pediatrics
University of Utah

BARBARA I. VUIGNIER, PHARM D
College of Pharmacy
University of Utah

JANNETTE B. PAPPAS, MT(ASCP)
Dept of Pharmacology
University of Utah
Salt Lake City, UT 84113-1103

REFERENCES

1. Needleman HL, Jackson RJ. Lead toxicity in the 21st century: will we still be treating it? *Pediatrics*. 1992;89:678-680
2. Glotzer DE, Bauchner H. Management of childhood lead poisoning: a survey. *Pediatrics*. 1992;89:614-618
3. Howard B, Charney E. Effects of Head Start on children with lead poisoning. *AJDC*. 1991;145:384
4. Centers for Disease Control. *Preventing Lead Poisoning in Young Children: A Statement by the Centers for Disease Control*. Atlanta, GA: US Dept of Health and Human Services; 1991

In Reply.—

I thank Dr Lakatos for his generous comments. His finding that DPA reduces bilirubin levels in neonates is intriguing; whether this is related to neonatal lead levels is unknown. Considerable work needs to be done before chelation can be considered as a therapy for newborn lead burdens.

Banner et al are concerned about the "mythology surrounding the issue of lead poisoning and its treatment," but they manage to propagate a few new myths of their own. They argue that calling for deleading of houses is troublesome; that because maternal and socioeconomic factors are predictors of development, the neurotoxic properties of lead assume lesser importance; that stimulation of lead-exposed children can replace removal of the lead; and finally, that an incidence of 4% elevated blood lead levels is a reassuring finding.

Science differs from mythology in at least two ways: it weights empirical sense data more heavily than fantasy or wish, and it employs the rigors of hypothetico-deductive logic to generate trustworthy inferences. There are many myths about lead; it was the replacement of some of them with tested facts that permitted the Public Health Service to announce its Strategic Plan to Eliminate Childhood Lead poisoning.¹

Byers disproved the widely accepted myth in the 1940s that if a lead-poisoned child did not die of the disease, he or she was left with no residua. He simply observed recovered children and found that a high proportion had school failure and behavior disorder.² The resulting void then was occupied by a new myth: that only symptomatic children had psychological deficits due to lead. This has been cancelled effectively by a host of careful studies from around the world that show IQ deficits and behavioral aberrations in asymptomatic children with blood lead levels greater than 10 µg/dL. Many of these studies controlled for maternal and socioeconomic factors.³ The human data are strongly buttressed by animal studies of rodents and primates that show learning impairment and attentional disturbances at corresponding lead levels. Confounding by maternal rearing and socioeconomic factors was obviously not an issue.⁴ None of these are noted by Banner et al in their letter.

There is general agreement by public health authorities that primary prevention, ie, preventing exposure, is more effective and less expensive than secondary or tertiary prevention, ie, preventing disease or preventing disability. But Banner et al argue on the basis of a single abstract that the benefits of a Head Start experience can remediate exposure to lead. The logic employed here has mythic properties; it is analogous to stating that physical therapy could superannuate polio vaccination. The myth that lead abatement takes away money from other antipoverty programs is popular with lead industry spokespersons; it does not seem to need much factual nourishment to survive. No lead screening money has been taken from Head Start Funds. This is not a zero sum game.

It violates common sense to say that employing people to reduce the amount of lead in the environment of children would hurt the antipoverty effort. Such a program would improve the quality of the homes people live in while reducing unemployment, certainly the most serious affliction of poverty. In the Public Health Service Strategic Plan, a careful econometric analysis states that the net (after paying for deleading) monetized benefit for deleading the housing stock in this country would be \$28 billion. I know of no data supporting the opposing case.

Banner et al seem to argue that because only 4.2% of children in their exploratory screening program had blood lead levels >10 µg/dL, the need for screening in their area is dubious. I have no data on screening practices in Utah, but doubt that they have

stopped screening for tuberculosis, phenylketonuria, or neonatal hypothyroidism, all diseases with prevalences less than 0.01%.

HERBERT L. NEEDLEMAN, MD
University of Pittsburgh School of Medicine
Pittsburgh, PA 15213

REFERENCES

1. Centers for Disease Control. *Strategic Plan for the Elimination of Childhood Lead Poisoning*. US Department of Health and Human Services; Atlanta, GA: 1991
2. Byers RK, Lord EE. Late effects of lead poisoning on mental development. *AJDC*. 1943;66:471-483
3. Needleman HL, Gatsonis C. Low level lead exposure and the IQ of children. *JAMA*. 1990;263(5):673-678
4. Rice DC. Behavioral impairment produced by developmental lead exposure: Evidence from primate research. In: Needleman HL, ed. *Human Lead Exposure*. Boca Raton, FL: CDC Press; 1992

SSI Benefits and Functional Assessment

To the Editor.—

Perrin and Stein¹ have done a commendable job of informing the pediatric community about the recent changes in the adjudication of Supplemental Security Income (SSI) claims for children. The process of assessing disability in children has been streamlined further since the publication of this article. It now involves sequential analysis of medical impairments followed by analysis of functional limitations in five domains of development: cognitive, communicative, motor, personal-social, and behavioral. The later is called individual functional assessment (IFA), and involves comparing a child's function in various domains with an average child of that age. Information for the IFA is obtained from parents, school teachers, and pediatricians. Attention, concentration, and persistence in tasks are also taken into consideration while evaluating a school age child. Thus, a child's function is not compared to that of an adult, as was the practice in the past, but to a child of similar age. A child can be considered disabled if child cannot function appropriately, effectively, and independently in an age-appropriate manner in two or more than two domains of development, even if the medical impairments do not meet the criteria of the federal guidelines called the listings.

This scheme of functional assessment should bring about a major change in the thinking of pediatricians from a strictly medical model to a functional model. Pediatricians should think not only in terms of impairments, but also consider the impact of medical impairments on a child's age-appropriate function.

V. BHUSHAN, MD, MPH
Metropolitan Hospital Center
New York Medical College
1901 First Avenue
New York, NY 10029

REFERENCES

1. Perrin JM, Stein REK. Reinterpreting disability: changes in supplemental security income for children. *Pediatrics*. 1991;88:1047-1051

In Reply.—

Dr Bhushan helpfully points out one of the important changes in the determination of childhood disability by the Social Security Administration. To meet the Supreme Court requirement that children receive a functional assessment similar to that available to adults, when they do not meet specific diagnostic criteria, the Administration has developed guidelines for measuring childhood functioning in age-appropriate ways. Furthermore, the regulation¹ formulated by the Administration in response to the Supreme Court decision lists several criteria for presumptive disability; that is, considering the child eligible for disability benefits with only limited further evaluation needed. These criteria include premature infants with birth weights less than 1200 g (at least until age 1 year), tracheostomy in a child less than age 3 years, and Down Syndrome.