

## 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.<sup>1</sup>

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.<sup>2</sup> 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.<sup>3</sup> 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.<sup>4</sup> 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 Stein<sup>1</sup> 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 regulation<sup>1</sup> 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.