others at risk for these disorders, and ignoring the NIH Consensus Statement with regard to this issue.[3] COL Pettett has accurately described the newborn screening "policy" of the US military and it is clear that COL Tiwary considers this inadequate, asserting, "State regulations for neonatal screening for metabolic diseases do not optimally serve the needs of the dependents of the US Armed Forces."[4]

As the uniformed services develop a comprehensive screening system to meet the needs of its dependents, it will be important to consider recommendations for the use of regionalized laboratories that have sufficient experience and sample throughput to assure adequate quality of their results.[5-7] The use of a US regionalized laboratory by European military facilities, beginning 2 years ago, is to be commended. It is not clear that other respondents to the survey cited by COL Pettett meet the appropriate criteria of experience and sample throughout.[1,6]

In my own clinical experience I have cared for three US military dependents with extremely late-treated phenylketonuria (PKU), all with developmental delay and behavioral problems attributable to inadequate screening and/or follow-up, and discussions with colleagues indicate that this experience is not unique. The first child was a 6-year-old boy with the diagnosis of autism and severe delay, whose sister was diagnosed with PKU after a positive newborn screening test. When taking the family history, I became concerned and these concerns were confirmed the next day when his serum phenylalanine was determined to be 35.4 mg/dL. He had been born in a Panamanian hospital because the military facility was full and Panama had no screening program. When he returned to the military facility for well child care, the only screening performed was a urine ferric chloride test, a test abandoned in the US in the 1960s because of its high false-negative rate and the development of the Guthrie bacterial inhibition assay. The second child was cared for by the same Medical Corps pediatric neurologist as the first boy, and this physician recognized their clinical similarities. This boy had been born in a military facility in Germany and his PKU screening test was performed by a military hospital laboratory. The third child was born in Korea and had a positive PKU screening test. When seen by the Medical Corps physician the parents were told that their baby's result must be incorrect because the mother was Korean and this physician stated that PKU did not occur among Koreans. (There is no data regarding the incidence of PKU in Korea; the incidence in China is similar to the US, and although the incidence in Japan is much lower, it does occur there). Her father recognized the similarity of her severe developmental delay and autistic symptoms to PKU when he was watching a television program describing PKU. These brief case histories show the need for programs developed for military dependents rather than reliance on local custom, the value of regionalized screening laboratories, and the importance of guidelines for follow-up when personnel encounter a positive screening test.

The Committee on Genetics has been working with the Uniformed Services Chapters of the American Academy of Pediatrics to develop forms for recording and prominent display of newborn screening results on the charts of dependents. This form will alert military physicians when results are outstanding to reduce the risk of missing a patient through inadequate follow-up. This simple and effective approach will be a model for the civilian community.

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Would Breast-Feeding Decrease Risks of Lead Intoxication?

To the Editor.—

As Shannon and Graef[1] point out, lead intoxication may be an important public health problem in young infants. And it is indeed likely that some babies receive too much lead from formula feeding. But this may be due to more than the fact that the lead content of the water is too high. Lead concentration of the formula itself, before dilution and even when packed in lead-free cans, may be several times that of human milk.[2] This has, as the authors point out, serious implications for infants fed on infant formula. Yet the authors never really seem to understand that the problem is one of formula feeding. Indeed, the words "breast milk" or "breastfeeding" are not used once in the whole article. By the way, how many of the 50 reported patients were breast-fed at all?

It is distressing that the authors suggest routine lead screening in children at 6 months of age, but ignore a more useful proposal. Promotion of breast-feeding, of more prolonged, and of exclusive or near exclusive breast-feeding up to 6 months of age would prevent excessive ingestion of lead by infants (as well as help prevent the iron deficiency mentioned as increasing the risk of plumbism) at a cost likely to be far less than the cost of routine testing. This, of course, requires hospitals and health professionals to be more aware of how to help mothers get started breast-feeding properly and how to help them continue if problems arise. Unfortunately, as is implied indirectly by the article, support of breast-feeding is rarely considered important.

It is a telling comment that 26% of the parents were "welfare-dependent." These parents would have the most to gain by their infants being breast-fed, not only because of cost, but also because of the prevention of illnesses, in addition to lead intoxication, which occur most commonly in the poor. If only a small percentage of the money used to buy formula for the Women, Infants, and Children program participants were used to promote breast-feeding, the benefits would be far reaching.

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In Reply.—

We thank Dr Newman for his comments and agree that, for many reasons, more can and should be done to encourage breast-feeding. Unfortunately, it is unclear that this practice protects infants from lead in any way and, inevitably, there will be some women who choose formula. While time did not permit us to re-review these cases, we estimate that, based on current data in our area, 60% to 70% of the infants in our study were breast-fed. Both the work of Rabinowitz et al[1] and a recent, as yet unpublished study (Hu H. Personal communication) conducted in the Boston area revealed unacceptably high concentrations of lead in breast milk samples obtained from some lactating women who delivered their infants at the Brigham and Women's Hospital. Indeed, lacta-

tion and pregnancy have been shown to mobilize lead from bony stores even in women whose current exposure may not be high.[2] With respect to the overall lead hazard, Dr Newman is fortunate to live in Canada, a country whose health authorities had the foresight to recognize the dangers of lead paint in the 1920s and took steps at that time to restrict the lead content of paint to less than 5% by body weight. Unfortunately, the lead industry was able to prevent this step from occurring in the United States until 1977, long after it had replaced lead in paint with titanium (a step taken for *economic* not public health reasons). Thus, lead remains an ubiquitous hazard in our country and its presence in high concentrations in the milk of lactating women is only another sad testimony to that fact.

Although it is true that breast milk iron is more bioavailable than that from formula, it is also true that infants exclusively breast-fed may begin to outstrip the iron supply by the age of 6 to 12 months, the period when most of these infants began their lead exposure. It is also true, at least in the US that current breast-feeding practices are to begin weaning at 4 to 6 months of age, a time when many women return to the work force. This is yet another unfortunate reflection of the skewed priorities in our society. So, while we agree with Dr Newman that breast-feeding should be encouraged, it did not prevent these infants from becoming lead-poisoned. Until we have data showing that breast-feeding can indeed help to reduce potential lead exposure in an area where lead is endemic, we can only shake our heads and wonder why there is still resistance in the pediatric community to recognizing the true extent of the lead catastrophe in our society.

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The Bilirubin Debate

To the Editor.—

We would like to respond to Dr Lois Johnson's commentary[1] on our paper "Evaluation and Treatment of Jaundice in the Term Newborn: A Kinder, Gentler Approach."[2] One of Dr Johnson's concerns was that summarizing the effect of bilirubin on IQ in the Collaborative Perinatal Project (CPP) with linear regression could be misleading if the relationship between bilirubin and IQ were nonlinear. We are able to reassure her on this point. Since the submission of our paper, Newman and Klebanoff have re-analyzed the CPP data and found no association between bilirubin and IQ (adjusted for confounding variables) in either linear or nonlinear models.[3]

We believe Dr Johnson makes too much of the study by Gerver and Day,[4] who studied untreated babies with Rh disease born in the 1940s. As we emphasized in our paper, extrapolations from such infants to term babies without hemolysis in the 1990s are almost certainly not valid. Furthermore, the results of Gerver and Day are completely confounded by birth order. First-born children generally score higher on IQ tests, and all the control children (and none of the Rh cases) in Gerver and Day's study were first-born.

Seidman et al[5] found high bilirubin levels associated with an increase in the proportion of males with IQ <85, but no association between IQ and bilirubin in females, and no effect on mean IQ in males. In discussing this result, Dr Johnson draws an analogy with lead poisoning. But the analogy does not fit: lead is associated with a shift in the entire distribution of IQ values. In the study of lead poisoning that she cites,[6] this shift *was* associated with a four-point difference in mean IQ, which *was* statistically significant, even with a much smaller sample size. In the paper by Seidman et al, adjusted mean IQ values for those with mild, moderate, and severe jaundice were 102.8, 103.0, and 102.0 in males and 102.0, 103.1, and 103.6 in females. If the males had had a clinically significant increase in the number with low IQs, and a *decrease* in the number with high IQs, there should have been more of a difference between the means, as was seen with lead poisoning.[6]

Even if there were a relationship between bilirubin and IQ (which the CPP and other studies suggest there is not), and even if it were shown to be causal, *there is no evidence that treating babies to lower their bilirubin levels has any effect on their IQ.* In the collaborative phototherapy trial,[7] phototherapy lowered bilirubin levels, but had no effect on IQ. Similarly, although the study by Seidman et al[5] spanned time periods in which phototherapy was and was not used, phototherapy was not associated with an effect on IQ.

Aggressive treatment of jaundiced infants is expensive and intrusive, and it cannot be regarded as totally benign. As we have discussed previously, we do not have data to prove (nor do we believe) that high levels of bilirubin pose no risk whatsoever to term infants without hemolysis. We do believe, however, that in some infants the risk of bilirubin toxicity is low enough that it might be exceeded by adverse effects of therapy. A recent report that premature babies fed breast milk had an eight-point IQ advantage at age 7 years compared with those fed formula (controlling for numerous potential confounders, including mother's intention to nurse)[8] should remind us that interventions such as phototherapy (which may lead to less breast-feeding[9]) can be double-edged swords. In the absence of convincing evidence that high bilirubin levels are hazardous in term infants without hemolysis and that the benefits of treatment exceed the risks, a kinder, gentler approach to many jaundiced infants is warranted.

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