tive of humility—that we as practitioners can be better, that our current modes of work and care are not as good as they might. It is hard to say we need to improve when we are on the defensive, with many questioning our performance and even our good will. Nonetheless, the commitment to improvement is the only perspective that over time can sustain us.

Actions by individual practitioners and practices can likely do more to accelerate improvement than structural changes at a national organization. If practitioners take the following steps, then pediatricians will be at the forefront of our profession in restoring public confidence in health care:

1. Make a commitment, yourself, to making the care you deliver tomorrow better than the care you gave today—and each day going forward. Choose an area you are passionate about—chronic illness, prevention, development, behavior, attention-deficit/hyperactivity disorder, medication safety.
2. Learn a little about improvement methods—read some of Don Berwick's or Paul Batalden's articles, go the Institute for Healthcare Improvement Web site, or go to a workshop at a national or chapter meeting.[12]
3. Examine your own practice—don't just curse the managed care organizations for auditing your charts—you decide what you want to look at, and look at it. Post the data for your partners and staff, and then measure again.
4. Involve your patients—find out what they really think about care or what ideas they have to make care better.
5. Involve your colleagues—they can give you ideas, and keep the momentum up.
6. Try, try, and try—improvement requires change.

Why should Academy practitioners care about this issue of quality? Think back, if you will, to the essay you wrote when you applied to medical school, or to your pediatric residency. For most of you, the essay emphasized your wish to make a difference in people's lives, to make the world a better place one person at a time. My residency essay starts off: "The outstanding experiences in my life to date [at all of 22] stem from my persistent activism in the realm of social welfare," and ends, "... my aspirations in medicine are a continuation of these earlier trends."

The way we influence children's lives as doctors is through the health care we provide. The federal Bureau of Primary Health Care places their focus on quality improvement administratively within their initiatives to reduce racial and economic disparities in health. It is through enhancing quality that we better fulfill our mission for being in medicine—and pediatrics.

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REFERENCES

1. Berman S. Are we ready to provide leadership to ensure all children's health? *AAP News*. 2000;17:192
2. Berwick DM. Taking the Lead—Keynote Address. Presented at: Annual Meeting of the AAP; October 12, 1999; Washington, DC
3. Kohn LT, Corrigan JM, Donaldson MS. *To Err Is Human: Building a Safer Health System*. Washington, DC: Institute of Medicine, National Academy Press; 1999
4. Appleby J. Jaundice-caused brain damage is on the rise. *USA Today*. October 11, 2000:81–86
5. ACGME General Competencies 1.3. Available at: http://www.acgme.org
6. Stockman JA III. Diplomate newsletter. Chapel Hill, NC: American Board of Pediatrics; 2000
7. Horbar JD, Rogowski J, Plsek PE, et al. Collaborative quality improvement for neonatal intensive care. *Pediatrics*. 2001;107:14–22
8. Robertson T. More doctors now offer same-day service. *Boston Globe*. October 15, 2000
9. Margolis PA, Lannon CM, Stuart J, Fried B, Moore D, Keyes-Elstein L. Improving delivery systems for prevention in primary care practices: a randomized trial. Paper presented at: APA Presidential Plenary; Pediatric Academic Society Meeting; April 30, 2001
10. American Academy of Pediatrics, Committee on Quality Improvement and Subcommittee on Attention-Deficit/Hyperactivity Disorder. Clinical practice guideline: diagnosis and evaluation of the child with attention-deficit/hyperactivity disorder. *Pediatrics*. 2000;105:1158–1170
11. American Academy of Pediatrics, Subcommittee on Attention-Deficit/Hyperactivity Disorder and Committee on Quality Improvement. Clinical practice guideline: treatment of the school-aged child with attention-deficit/hyperactivity disorder. *Pediatrics*. 2001;108:1033–1044
12. Berwick DM. Eleven worthy aims for clinical leadership of health system reform. *JAMA*. 1994;272:10

Targeted Screening for Elevated Blood Lead Levels: Populations at High Risk

ABBREVIATION. NHANES, National Health and Nutrition Examination Survey.

Lead poisoning is a preventable environmental disease without borders, affecting children worldwide. Currently, the Centers for Disease Control and Prevention defines an elevated blood level to be 10 μg/dL or greater.[1] The most recent National Health and Nutrition Examination Survey (NHANES) 1999 data demonstrated that the geometric mean blood lead level in the United States has decreased to 2 μg/dL.[2] That report did not present prevalence data because of small numbers. Despite the lowering of blood lead levels nationally, complacency about lead poisoning is not indicated. An analysis of childhood blood lead data collected by state surveillance programs found that prevalence of elevated blood lead levels varied from state to state and county to county, indicating that lead poisoning is still a problem at the local level.[2]

Additionally, data suggests that there may be effects of lead on cognitive ability at levels lower than previously reported.[3] Other data suggests that standard application of chelation therapy did not improve neuropsychological function in lead-poisoned children.[4] These data point out the need for im-

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proved prevention efforts, specifically, a shift to primary prevention through improved housing paired with continued, vigilant blood lead screening among populations at risk.

In areas where universal blood lead screening is not indicated,[5] identifying populations at high risk for lead poisoning permits effective use of targeted screening for elevated blood lead levels. Additionally, identification of risk permits communities to focus education and preventive efforts, such as housing remediation. Recent reports highlight the risks of children in low-income families and children who have immigrated to the United States.

An important risk factor for lead poisoning is low socioeconomic status, a criterion for Medicaid eligibility. Based on data from NHANES III, Phase 2 (1991–1994), among an estimated 890 000 children with elevated blood lead levels, 535 000 (60%) were on Medicaid.[6] Furthermore, Medicaid children accounted for 83% of children ages 1 to 5 with blood lead levels ≥20 μg/dL. Although the Centers for Medicare and Medicaid Services (formerly known as the Health Care Financing Administration) mandates that children enrolled in Medicaid receive blood lead screening, an estimated 81% of Medicaid children had not been screened for lead poisoning.[6]

In the July 2001 issue of *Pediatrics*, Geltman et al demonstrated that refugee children entering the United States from abroad constitute an additional population at risk for lead poisoning. Geltman et al reported elevated blood lead levels in 693 refugee children who resettled in Massachusetts from 1995 to 1999 from multiple countries.[7] Most striking was that 37% of children from Asia and 40% from Central America and the Caribbean had blood levels ≥10 μg/dL. Among children without elevated levels when they were resettled in the United States, 6% had elevated levels when tested 6 months or more later. The authors also cite several other reports of lead poisoning in refugee populations that have resettled in the United States.

In part, elevated blood lead levels in these children may be attributed to low socioeconomic status: refugee children in the United States are often among the most financially disadvantaged, and therefore may be more likely to live in older, substandard housing that may contain deteriorated, lead-based paint. Also, other nations may have less stringent regulation of environmental lead sources (eg, gasoline, paint); children may enter the United States with blood lead levels already elevated as a result of environmental exposures in their countries of origin. Similarly, environmental and occupational lead exposure of the parents may result in exposure to children, through take-home or intrauterine exposure routes. Use of lead-glazed cooking vessels or folk remedies and herbal and mineral preparations, which have been documented as a source of lead exposure, may be continued in immigrant communities within the United States. Lastly, immigrant children may receive continuing lead exposure during visits back to their native countries.

Like refugees, children adopted from abroad may be at particular risk for lead poisoning. Chinese and Russian children adopted by US citizens have been reported to have elevated blood lead levels.[8,9] The American Academy of Pediatrics recommends that children who have been adopted or emigrated from countries where lead poisoning is prevalent should receive blood lead tests.[10] When indicated, testing should be performed as a component of medical screening of refugees, which is mandated by federal regulation to occur within 90 days of arrival into the United States.[11] As nearly all refugee children have Medicaid coverage for a minimum of 8 months after arrival in the United States, such screening meets Medicaid Early and Periodic Screening, Diagnosis, and Treatment program testing requirements, which should be applied regardless of local risk once in the United States.[12] Content of refugee health screening varies between states; therefore, clinicians evaluating newly arrived refugees and immigrants in localities with low environmental lead exposure risk must be both cognizant of the elevated prevalence of lead poisoning among refugees and immigrants and mindful of Medicaid screening requirements.

The article by Geltman et al suggests the need for heightened concern and additional study about the risk for elevated lead levels in immigrant and refugee populations. To eliminate childhood lead poisoning, health programs and providers should be particularly vigilant about lead screening in high-risk groups. There are >6 000 000 Medicaid enrollees between ages 1 and 6 who are 3 times more likely than non-Medicaid children to have elevated blood lead levels; better enforcement of existing screening guidelines for these children is of paramount importance.[12] Similarly, immigrant and refugee children deserve close attention. We would agree with the Academy recommendation for lead screening of children who have emigrated (or been adopted) from countries where lead poisoning is prevalent. Whether the data will support screening of *all* adopted or emigrated children or only targeted children from specific regions remains to be determined. At this time it would be prudent to consider lead screening of these children.

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REFERENCES

1. Centers for Disease Control and Prevention. *Preventing Lead Poisoning in Young Children.* Atlanta, GA: US Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention; 1991
2. Centers for Disease Control and Prevention. Blood lead levels in young children—United States and selected states, 1996–1999. *MMWR Morb Mortal Wkly Rep.* 2000;49:1133–1137
3. Lanphear BP, Dietrich K, Auinger P, Cox C. Cognitive deficits associated with blood lead concentrations <10 μg/dL in US children and adolescents. *Public Health Rep.* 2000;115:521–529
4. Rogan WJ, Dietrich KM, Ware JH, et al. The effect of chelation therapy with succimer on neuropsychological development in children exposed to lead. *N Engl J Med.* 2001;344:1421–1426

5. Centers for Disease Control and Prevention. *Screening Young Children for Lead Poisoning: Guidance for State and Local Public Health Officials.* Atlanta, GA: US Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention; 1997
6. US General Accounting Office. *Medicaid: Elevated Blood Lead Levels in Children.* Washington, DC: US General Accounting Office, 1998. GAO Publ. No. GAO/HEHS-98-78
7. Geltman PL, Brown MJ, Cochran J. Lead poisoning among refugee children resettled in Massachusetts, 1995 to 1999. *Pediatrics.* 2001;108: 158–162
8. Centers for Disease Control and Prevention. Elevated blood lead levels among internationally adopted children—United States, 1998. *MMWR Morb Mortal Wkly Rep.* 2000;49:97–100
9. Miller LC, Hendrie NW. Health of children adopted from China. *Pediatrics.* 2000;105(6). Available at: http://www.pediatrics.org/cgi/content/full/105/6/e76
10. American Academy of Pediatrics, Committee on Environmental Health. Screening for elevated blood lead levels. *Pediatrics.* 1998;101:1072–1078
11. 45 CFR §200.107
12. Centers for Disease Control and Prevention. Recommendations for blood lead screening of young children enrolled in Medicaid: targeting a group at high risk. Advisory Committee on Childhood Lead Poisoning (ACCLPP). *MMWR Morb Mortal Wkly Rep.* 2000;49(No. RR-14):1–13

Zinc Supplementation Saves the Lives of Children Living in Poverty

ABBREVIATIONS. SGA, small for gestational age; IUGR, intrauterine growth retardation.

The study by Sazawal et al[1] analyzes the effect of zinc supplementation, during the first 9 months of life, in preventing death in infants born small for gestational age (SGA) in slum areas of India. The study was randomized and controlled for most relevant confounding variables. The main finding is that supplemental zinc in conjunction with 1 or more of other micronutrients—riboflavin, folate, calcium, phosphorus, or iron—decreases the risk of death by two thirds during the supplementation period. The risk reduction was significant and of similar magnitude independent of whether the infant was breastfed or received artificial feeding. Unfortunately this analysis was not performed for the exclusively breastfed infant who had the lowest risk of dying (relative risk: 0.03; 95% confidence interval: 0.003–0.29). Zinc supplementation may in fact have benefited only those who were either artificially fed or partially breastfed.[2] Zinc prevented deaths mainly from diarrheal disease and sepsis, but differential mortality by cause of death could not be fully assessed because the number of deaths was 20 out of 1154 SGA infants enrolled. The study was performed in SGA infants consuming breast milk or artificial feeding in an environment where infection, especially diarrhea, may be highly prevalent and complementary foods do not contain meat or other zinc-rich foods. Children living in poverty, particularly SGA infants, are at risk of severe zinc deficiency, which leads to repeated infections and growth failure. Stunting, the most prevalent manifestation of malnutrition, has been demonstrated to increase the risk of death.[3] This study, and the existing body of literature of increased infant mortality in malnourished children, suggests that zinc deficiency may be an underlying cause in a large proportion of infant death in developing countries. The message is that malnutrition, whether in utero or after birth, is an important risk factor for dying early in life. Preventive strategies should include optimizing fetal as well as postnatal growth.

Countries where prevalence of low birth weight is high have a large proportion of low birth weight attributable to intrauterine growth retardation (IUGR). In communities where low birth weight rates are >30%, infant mortality is usually over 50 per 1000 live births. Global data analyzed by de Onis et al[4] suggests that nearly 75% of all IUGR infants are born in Asia, mainly in south-central Asia, and 20% in Africa. Most of them are at higher risk of early protein-energy malnutrition, infections, early weaning, and death. Few studies demonstrate a beneficial effect of maternal nutritional interventions to prevent IUGR. A review by de Onis et al[5] in 1998 found only 12 randomized, controlled trials, including protein-energy, vitamins (vitamin D, folate), minerals (calcium, magnesium, zinc, iron) and fish oil supplementation. A beneficial effect of marginal significance was found only with balanced protein-energy supplementation (odds ratio: 0.77; 95% confidence interval: 0.58–1.01). Studies on zinc supplementation during pregnancy failed to demonstrate an effect on prevention of IUGR.

This study demonstrates the importance of micronutrient malnutrition in determining high infant mortality in developing countries and the fact that this is further aggravated by fetal growth retardation. What this study fails to highlight is that exclusive breastfeeding provides greater protection from death than zinc supplementation.

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REFERENCES

1. Sazawal S, Black RE, Menon VP, et al. Zinc supplementation in infants born small for gestational age reduces mortality: a prospective randomized, controlled trial. *Pediatrics.* 2001;108:1280–1286
2. Castillo-Durán C, Rodríguez A, Venegas G, Alvarez P, Icaza G. Zinc supplementation and growth of infants born small for gestational age. *J Pediatr.* 1995;127:206–211
3. Pelletier DL. The relationship between child anthropometry and mortality in developing countries: implications for policies, programs and future research [review]. *J Nutr.* 1994;124(10 suppl):2047S–2081S
4. de Onis M, Blössner M, Villar J. Levels and patterns of intrauterine growth retardation in developing countries. *Eur J Clin Nutr.* 1998; 52(suppl 1):S5–S15
5. de Onis M, Villar J, Gülmezoglu M. Nutritional interventions to prevent intrauterine growth retardation: evidence from randomized controlled trials [review]. *Eur J Clin Nutr.* 1998;52(suppl 1):S83–S93

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