publication of our 1985 article addressing the prevention of urinary tract infection with circumcision,[4] I believed we had opened Pandora's box and felt somewhat obligated to further examine the many concerns surrounding the practice.

I resent the implication that parents were "coerced" into having their sons circumcised. Virtually all of them sought to have the procedure performed. Informed consent counseling was achieved, with an explicit explanation of the potential risks and benefits. Furthermore, Doctors Anderson and Schwarz state that the neonatal circumcision complication rate of 0.2% that we reported is the lowest published in the literature. They are wrong. Speert[5] found a 0.06% complication rate among almost 11 000 circumcised boys, while Gee and Ansell[6] found an identical complication rate (0.2%) among >5000 circumcised boys. We are unable to say the procedure is less safe among older children. However, other than our report, there are no other investigations that have addressed the issue. Furthermore, these physicians have incorrectly included meatal stenosis among complications of neonatal circumcision. As the American Academy of Pediatrics Task Force on Circumcision pointed out, meatal stenosis does not result from the procedure.[7] Additionally, contrary to their statement, there are no reports of nerve damage from local anesthesia.

There is a dearth of information regarding this, the most common surgery performed in the United States. Rather than following the circumcision debate "with interest," why don't these two physicians make a contribution by pursuing some aspect of the issue? Before commenting on features of the controversy, I would suggest they do their homework.

I was fascinated by Dr Katz's remarks. The traditional briss is performed at the end of the first week of life, not "routinely" at 3 to 15 months. Why the delay among these older boys? He apparently likes the speed with which he can perform the procedure using the Mogen clamp. However, with speed as the major objective, I have seen many unaesthetic results by others using this device. To date, no investigations have compared the Mogen clamp with either of the two other commonly used circumcision devices, the Gomco clamp and the PlastiBell.

Children are acutely aware of the pain associated with the procedure. In contrast to Dr Katz, I believe all boys deserve some form of analgesia. He does not consider the dorsal penile nerve block to be safe. However, there are only a few anecdotal reports of complications other than local hematoma formation.

I am astounded he does not perform circumcisions with sterile technique. Well-known, albeit rare, complications include cellulitis, staphylococcal scalded skin syndrome, necrotizing fasciitis, and systemic bacteremia. Furthermore, there are anecdotal reports of infections such as syphilis being passed from one boy to many others by using the same, nonsterile instruments. How does he know there is "almost a zero rate of infection"? Is Dr Katz a pediatrician who routinely performs follow-ups with these boys or would they be likely to go elsewhere for complications?

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Lead Questionnaire Not Always Efficient

To the Editor.—

Tejeda et al found the lead risk assessment questionnaire to be an effective screening method for identifying increased lead levels in their population.[1] However, as Drs Gellert and Wagner pointed out in the same issue of *Pediatrics*,[2] depending on the prevalence of elevated lead levels, screening may not even be necessary. Therefore, using this questionnaire may be inappropriate in certain populations.

In Arizona, Medicaid requires that physicians screen with these questions at 6, 9 to 12, 15 to 18, 24 to 36, and 48 to 60 months of age. Therefore, we decided to determine if this practice was warranted in our population of Native Americans. Of 143 infants/children tested thus far, one (0.7%) had an elevated lead level (11 μg/dL), and the remainder had levels <10 μg/dL. However, 29/143 (20%) and 20/143 (14%) of the care-givers answered yes to one of the screening questions and home questions, respectively. The infant with elevated lead had a positive response to question number four. Therefore, if the questionnaire was used for screening in this population, 28/143 (19.6%) normal patients would have been tested to identify one with a lead level of 11 μg/dL.

This highlights the importance of knowing the local prevalence of elevated lead levels to avoid unnecessary questioning and subsequent testing. In our population the screening questions may not prove useful and could lead to inappropriate testing. Readers of this study must know the local prevalence of elevated lead levels before using this questionnaire in their practice.

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Lead Debate Goes On

To the Editor.—

In his recent commentary (*Pediatrics*, February 1994), Harvey argued that universal screening for lead exposure, as recommended in the 1991 Centers for Disease Control statement, is wasteful of resources, and that screening in low prevalence areas should be curtailed. The value of universal screening for any disease deserves careful evaluation. This is unarguable; we live and work, after all, in a period of finite resources. It is also self-evident that a sensible judgment on this matter requires solid facts and up-to-date information. But Dr Harvey's statements about the neurotoxicity of lead, the accuracy of blood lead measurement, and the therapeutic and preventive responses available correspond neither to the latest nor the best scientific information. Indeed, they reflect pediatric thinking circa 1980.

As investigators of lead toxicity, we present what we believe is a more current and accurate review of recent progress in the epidemiology and toxicology of lead. This should permit a more informed judgment as to who should be screened.

Harvey states that "Because of a variety of confounding factors, and because of the inability to correlate blood lead (BPb) at age 1 year with cumulative body burden, evidence that BPb levels <20 μg/dL cause a clinically important decrease in intelligence and an increase in neurobehavioral problems by the time a child enters school is lacking." This is wrong. There are many studies of quality that show behavioral effects down to 10 μg/dL and perhaps lower. There is convincing evidence that blood lead levels above 10 μg/dL at age 2 are associated with lower IQ at age 10. Over 24 studies of asymptomatic children have shown lead-related IQ deficits. Many of these show lead effects down to 10 μg/dL. There is no database of such size for any other neurotoxin.

Not one of these studies is perfect, and not all have found an effect. But four meta-analyses have concluded that the joint prob-

ability that the findings are attributable to chance is vanishingly small.[1-4] It is well-accepted by knowledgeable epidemiologists (and common sense tells us) that when many studies, each imperfect in a different area, agree that there is an effect, something is going on.

These data provide convincing evidence that effects of lead extend down to 10 μg/dL and perhaps below. The slope of regression lines shows no evidence of leveling off at 20 μg/dL. The most recent study from New Zealand, where exposures are not high, show that early small IQ and behavioral differences due to lead are associated with later significant decreases in reading, scholastic ability, and increases in measures of inattention/restlessness.[5] The longest follow-up of asymptomatic lead-exposed children showed a sevenfold increase in failure to graduate from high school and a sixfold increase in the rate of reading disabilities.[6] Harvey speaks of confounding as a barrier to concluding that lead is neurotoxic at these levels. The naming of potential confounders is not the same as demonstrating confounding. Indeed, the best forward studies of lead and IQ controlled for the logical and empirically demonstrated variates that could confound.

It is on the basis of this body of epidemiologic evidence from around the world, and supporting data from animal studies, that the Centers for Disease Control,[7] the American Academy of Pediatrics,[8] the Environmental Protection Agency[9] and, most recently, the National Academy of Sciences[10] have agreed that health effects at blood lead levels below 20 μg/dL are established fact. This is an unprecedented coherence of opinion in neurotoxicology. That does not mean that it is exempt from revision. But to do this requires a more thorough documentation of the claim that effects below 20 μg/dL lack evidence to support their existence.

Epidemiology has never pretended to provide perfect control of all variates. Multivariate space is infinite and grant support; therefore, number of subjects is not. Where epidemiology is lacking, toxicology fills the gap. Toxicologists can administer lead in systematically controlled doses, to animals randomly assigned to group, and eliminate the problem of confounding. Where this has been done, lead has been demonstrated to impair neurochemical function[11] and animal behavior.[12,13] If this massive database is not persuasive for lead, then no other chemical can be considered to have been demonstrated to be toxic.

Dr Harvey asks: "Is there an accurate, reliable test to determine BPb?" The answer is a resounding "Yes." At the recent meeting of the Centers for Disease Control Lead Task Force, it was clearly stated that precise measurements of lead down to below 3 μg/dL were obtainable. The National Academy of Sciences recent report supports this assertion.

Dr Harvey then asks: "Is there an effective intervention?" He seems to argue that because there are no published studies of the effects of environmental control on IQ, and because the costs of dust control would be "staggering," they need to be further evaluated before they are accepted as good practice. There are a number of nonpharmacologic, low technical responses to a child with a blood lead level of 15 μg/dL. The first is source identification. Second is education about dust control, handwashing, and nail biting. Third is nutritional evaluation and counseling. Fourth is surveillance, so that a child with a blood lead level of 10 is not next seen with a blood lead of 50. All of these types of intervention are a great part of pediatric practice, and are what pediatricians excel in.

Pediatrics has in general been slow to recognize the scientific progress in lead toxicology. Until the 1940s, the prevailing pediatric belief was that if a child did not die of lead exposure, he or she was left untouched by the illness. After Byers' landmark paper showed that 19 of 20 recovered patients had central nervous system impairment, that belief was replaced by the conviction that a child had to display symptoms to have experienced harm. We now know that this is wrong.

The gap between the amount that has been known about lead and what has been done about it has always been vast. As scientific knowledge of lead has moved forward, this gap has grown. We no longer believe that death or complete recovery are the only outcomes. It is clear, however, that there are pockets of skepticism about the reality of health effects of lead at low dose, and about the need to prevent this exposure.

Benjamin Franklin, writing to a friend about the same gap, said:

> "You will see by it, that the opinions of this mischievous effect from lead is at least 60 years old, and you will observe with Concern how long a useful Truth may be known, and exist, before it is generally receiv'd and practis'd on."

That was 230 years ago.

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