

upper respiratory tract infections, is unlikely to show otitis media, although in this study the number of "other" symptoms was too small to support a statistical association.

A recent survey of parent's worries about children compared to actual risks by Stickler et al<sup>5</sup> showed that ear infection was the most common health concern, outranking other illnesses including acquired immunodeficiency syndrome, sudden infant death syndrome, and pediatric cancers. It is possible that parents also use ear pulling as a "ticket of admission" when actually concerned about sleep problems or other disorders. It may be useful to reassure parents that simple ear pulling is not likely to be a sign of otitis media. Telephone advice protocols should reflect this knowledge.

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#### Lack of Money—Barrier to Immunization

To the Editor.—

Schulte et al<sup>1</sup> reported in 1991 that 73% of participating Dallas County, Texas, primary care physicians referred some children to immunization clinics in 1988. As a pilot project, we interviewed 31 of 37 (84%) eligible board-certified primary care physicians in urban/suburban areas across Minnesota. Participants rated the likelihood of referring children with various types of insurance coverage to public clinics for immunization. More physicians (52%) were likely to refer children from economically disadvantaged homes without insurance than either children who had Medicaid coverage (10%,  $P < .001$ ) or private insurance (0%,  $P < .001$ ). Furthermore, more participants referred children with Medicaid coverage than children with private insurance ( $P = .05$ ). We conclude that lack of reimbursement is an important barrier to immunization and that some physicians consider Medicaid reimbursement inadequate.

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In Reply.—

I reviewed the letter from Drs Zimmerman, Giebink, and Street concerning our article authored by Shulte et al (*Pediatrics*. 1991;87:204-207) which studied patterns of referral for immunization in Dallas County, Texas. We believe their comments are insightful and agree that financial barriers are a major reason that these children are referred. Their experience documents a financial gradient from the uninsured to the Medicaid to the fully insured population as concerns treatment or referral decisions from primary care physicians. I believe their study compliments our earlier study.

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#### Lead Ingestion and X-rays

To the Editor.—

In a recent article entitled "Prevalence of Radiographic Evidence of Paint Chip Ingestion Among Children with Moderate to Severe Lead Poisoning, St. Louis, Missouri, 1989 through 1990," McElvaine et al<sup>1</sup> correlated the presence of radiopaque substances on abdominal radiographs with the elevation of blood lead levels in children who were referred for chelation. They assumed in their article that all radiopaque densities seen on radiographs were lead paint chips. This may be inaccurate because other substances which children ingest may also show up as radiopaque substances.

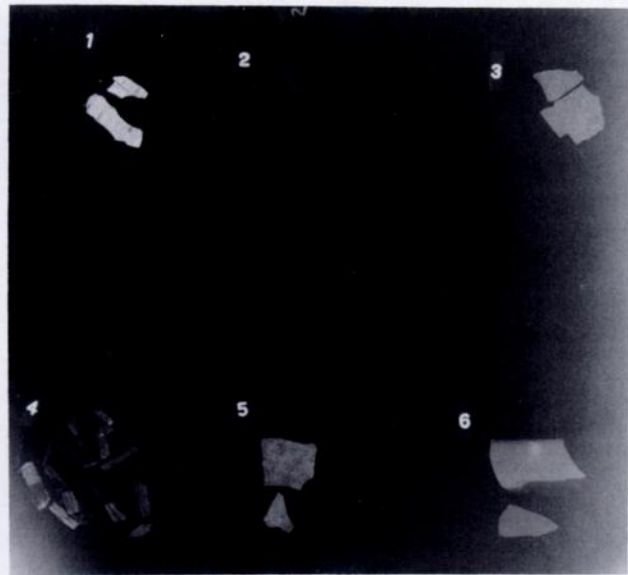
While caring for a 9-year-old autistic child who had recurrent elevations of blood lead levels, we consistently found radiopaque densities on his abdominal radiographs. Repeated house inspections found no source of lead paint in their present home, although he had been exposed in a prior living situation. His mother did point out an area of the closet where he had been picking at the plaster. He was known to have significant pica. His lead levels and ZnP were consistent with a child who had been exposed and treated in the past and was having rebound elevation of his blood lead levels.

Samples of lead-based paint, non-lead-based paint, plaster, and wall paper were obtained from the Cleveland Lead Poisoning Prevention Program. These samples were collected on home visits performed because children with elevated blood lead levels lived in these homes. The person collecting the samples coded them by the level of lead that was found using an x-ray fluorescence analyzer. These substances were then x-rayed (Fig 1). The high lead paint was significantly more radiopaque than the low lead paint; however, there was no difference between low lead paint or unpainted plaster. Paint is considered to have lead in it if it has a lead concentration above 0.7 to 1.2 mg/cm<sup>2</sup> of lead (cut-off point varies within this range by local ordinance).<sup>2</sup> In our autistic child, the radiopaque densities on his abdominal radiographs were felt to be plaster chips. Radiopaque densities in the gastrointestinal tract are consistent with pica, and all radiopaque substances are not necessarily lead chips. The x-ray findings need to be correlated with examination of the environment as well as with evaluation of the child's blood lead levels.

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**Fig 1.** Radiographs of substances found on inspection of homes of children with high lead levels. 1, paint chips with 10 mg/cm<sup>2</sup> of lead; 2, wallpaper painted with non-lead paint; 3, unpainted plaster; 4, wood with paint with 1.4 to 1.8 mg/cm<sup>2</sup> of lead; 5, plaster painted with non-lead paint; 6, paint chips with 1.5 mg/cm<sup>2</sup> of lead.

*In Reply.—*

We agree with Dr O'Connor's assertion that radiopaque foreign bodies seen on abdominal radiographs of children with pica are not necessarily leaded paint chips. However, we consider it reasonable to infer that essentially all of the densities noted in the lead-poisoned children whose records we reviewed were, in fact, leaded paint chips.<sup>1</sup> For the great majority of lead-poisoned children in St. Louis, deteriorated leaded paint is found in their primary residence or in other residences the children frequent. During the period covered by our study (1989 through 1990), leaded paint hazards were found in 80% (2892 of 3595) of all such residences inspected. Although separate environmental data are not tabulated for children with blood lead levels greater than 55 µg/dL (for whom 12 of the 13 radiographs showed positive findings), almost all such children are found to have been exposed to leaded paint. In addition, abdominal radiographs of lead-poisoned children in St. Louis generally show densities that are almost completely radiopaque. For reasons stated in our paper, our data do not show precisely the proportion of lead-poisoning cases caused by paint chip ingestion, but they do support our essential conclusion: leaded paint chip ingestion remains an important cause of childhood lead poisoning in the United States.

After comparing the radiographic appearance of samples of paint chips, bare and painted plaster, painted wood chips, and painted wall paper with determinations of their lead content by an x-ray fluorescence analyzer, Dr O'Connor concludes that "all radiopaque substances are not lead chips." We have no doubt that her conclusion is correct in general, but for two reasons we are less sure that the unpainted plaster and the samples painted with "non-lead paint" could not contain potentially hazardous amounts of lead. First, portable x-ray fluorescence analyzers of the type used by most lead poisoning prevention programs are not accurate or precise enough at measuring low levels of lead to ensure, without confirmatory laboratory analysis, that paint testing negative by this method is essentially lead-free.<sup>2</sup> Although paint with high levels of lead, easily detectable by x-ray fluorescence, is more hazardous,<sup>3</sup> visible chips of paint with lower levels of added lead could amount to a dangerous dose if ingested by a child with pica. Second, lead residues can be found in porous substrates like plaster after leaded paint has been removed from them.

Finally, it is important to emphasize that an abdominal radiograph that indicates recent ingestion of leaded paint chips is not sufficient or even necessary to diagnose lead paint poisoning. As Dr O'Connor suggests, blood lead measurements and an investigation of a child's environment are required. When characteristic

radiopaque densities are seen on an abdominal radiograph of a lead-poisoned child living in a residence with nonintact leaded paint, we believe it reasonable and prudent to assume that such densities are evidence of recent paint chip ingestion.

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**α-Fetoprotein and Hypothyroidism in Infants**

*To the Editor.—*

We recently have documented a relationship between serum α-fetoprotein (AFP) and congenital hypothyroidism (CH) in the newborn period. Previous studies in our laboratory and others had reported a relationship between elevated serum AFP and elevated thyroid-stimulating hormone (TSH)/low thyroxine T<sub>4</sub> in newborns and infants in the first few months of life.<sup>1-4</sup> The New York State Newborn Screening Program for CH routinely screens more than 300 000 specimens per year diagnosing 120 to 140 confirmed cases of CH annually. Recently, we assayed AFP in infants with thyroid dysfunction together with age-matched euthyroid controls from 1 to 3 months of age.<sup>5</sup> After the initial newborn dried blood screen and on physician's request, follow-up specimens from infants were assayed for serum AFP, TSH, and serum-bound T<sub>4</sub>. Twelve of 12 infants with confirmed CH displayed elevated serum AFP levels (>2.0 SD) above our age-related reference ranges<sup>6</sup> and were consistent with high TSH and low T<sub>4</sub> levels. Following treatment with thyroxine in six infants that were tracked, all serum AFP levels declined to normal for age. Three of three infants with transient hypothyroidism (high TSH, normal T<sub>4</sub>) also demonstrated elevated AFP levels. In 16 of 16 premature euthyroid infants (low T<sub>4</sub>, normal TSH) elevated serum AFP levels were also demonstrable. Thus, we find elevated neonatal serum AFP levels consistent with situations displaying either elevated TSH levels, lowered T<sub>4</sub> levels, or both.

The physiological mechanism by which serum AFP is elevated both prenatally and postnatally in CH remains to be elucidated. Larsson et al<sup>2</sup> suggested either that the rate of AFP liver synthesis is increased or that the repression of this synthesis is delayed in the hypothyroid fetus/infant. However, Mengreli et al<sup>4</sup> argue that the half-life of AFP is extended in these infants (12 vs 5 days) and postulate that low thyroxine levels may be responsible for a slower catabolism of serum AFP in the liver. In a report predating these proposals, Belanger et al<sup>7</sup> demonstrated that both glucocorticoid- and thyroxine-treated newborn rodents displayed depressed AFP serum levels through a selective blockage of its hepatic synthesis and did not alter serum clearance of the fetal protein. In addition, thyroidectomy of these rodents did not modify the effects of administered glucocorticoid or thyroxine. In light of these prior studies and the data we presently submit, it would appear that