Poverty, Pica, and Poisoning

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A RECENT study of 333 children living in an old, congested, low-income area of Baltimore, Md., showed that 44.4 percent of these children had abnormal blood lead values of 0.05 mg. percent and higher (*1*). The children were selected at random from those brought to the pediatric clinics of the University of Maryland Hospital for health supervision or for complaints other than those usually associated with lead poisoning.

Since the history of pica (eating of nonfood material) was found in 69.6 percent of the children, the source of lead apparently was paint chewed from surfaces of wood or plaster or particles of paint swallowed after it had flaked from the surfaces.

Samples of paint were collected from typical homes and the lead content determined. The lead content in each sample was in excess of the recommended 1 percent of the total weight of the contained solids (*2*).

The majority of the children apparently swallowed paint from indoor surfaces. However, others may have eaten paint which had peeled from exterior walls. The interior of the home of one child with lead poisoning did not have toxic amounts of lead on its painted surfaces, but it was learned that the child sat on the stoop outside and ate particles of paint fallen from the exterior walls. These contained toxic amounts of lead.

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Pica as the source of lead was also supported by the lack of abnormal blood lead values in infants under 10 months of age. However, the increased incidence of abnormal values beginning at 10 months of age continued through the third year of life and then declined. This age distribution corresponds to the period when the child is confined to the home, has greater need for oral gratification, and in crowded situations has fewer controlled interest opportunities. Further support for pica as the source of lead intoxication is found in current studies by Dr. J. E. Bradley and R. S. Mosser of blood lead values of children in different socioeconomic strata. The data obtained suggest that the mean values of the lower group will exceed many fold the mean values of the middle and upper groups.

Contributing factors to this high incidence of lead intoxication seems to be related directly to environment. First, these children live in houses where lead-containing paint, used many years ago, is now flaking and peeling from the surface. Second, there seems to be widespread ignorance or disregard of the hazards to the child through the ingestion of these particles. Despite vigorous education campaigns which have been conducted by the public health department in Baltimore, many parents continue to accept pica as a harmless manifestation of normal infantile development. Third, crowded conditions within the home, and in many instances the absence of supervision by adults who may be obliged to leave the children to earn a living, allow the infant and preschool child opportunity to eat toxic material without restraint.

The main hazard of lead poisoning in a child

is lead encephalopathy. The frequency of lead encephalopathy is unknown, since only a few communities require reports of these cases. The incidence is suggested from the report that 538 Baltimore children were admitted to Baltimore hospitals with lead encephalopathy from January 1, 1931, to January 1, 1956 (*3*). Lead encephalopathy, despite recent advances in treatment, continues to result in neurological sequela, mental retardation, or death (*4,5*).

The observations in the Baltimore study suggest that there is a high incidence of lead poisoning in other metropolitan areas where slums exist and where paint contains lead. The study also suggests that physicians need to be constantly aware and alert to the symptoms of lead poisoning in children.

Since this disease is essentially environmental, preventive measures are possible. This will require the cooperative effort of physicians, nurses, and social workers of municipal health and welfare departments who will warn parents constantly of the seriousness of pica in children.

REFERENCES

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(*2*) American Standards Association: American standard specifications to minimize hazards to children from residual surface coating materials. ASA Standard Z66.1. New York, 1955, p. 1.

(*3*) A new health department committee on the prevention of lead poisoning. Baltimore Health News 33: 80–81 (1956).

(*4*) Bessman, S. P., Rubin, M., and Leikin, S.: The treatment of lead encephalopathy; a method for the removal of lead during the acute stage. Pediatrics 3: 201–208 (1954).

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Legal Criteria for Evidence of Intoxication

A bill setting forth the legal significance of findings from alcoholic intoxication tests of drivers on trial in the District of Columbia became law March 4, 1958. The legislation eliminates a previous need for expert witnesses to explain the legal ramifications of test results.

If the defendant's blood has 0.15 percent or more alcohol by weight, or an equivalent proportion of alcohol in 2,000 cubic centimeters of his breath, he is presumed to be intoxicated. Alcohol equaling 0.20 percent by weight in the urine has the same legal significance.

Percentages of 0.05 or less in the blood or breath and 0.08 or less in the urine are proof of sobriety; and alcohol levels between 0.05 and 0.15 in the blood and breath and between 0.08 and 0.20 in the urine constitute relevant evidence but neither proof nor disproof of sobriety or intoxication.

Drivers are not obligated to submit to the tests, the results of which apply when they are tried for driving while intoxicated, for negligent homicide, or for manslaughter.

Only a physician acting at the request of a police officer may withdraw a blood sample for testing. The defendant may request that his own physician conduct additional chemical tests. Results are available to the tested person on request.