ORIGINAL ARTICLES

Newark has the most serious pediatric lead poisoning problem in the USA.

Lead Poisoning in Newark

The Situation Prior To a Case Finding and Intervention Program

Ann A. Browder, M.D./Newark*

Newark recognized an emergency in lead poisoning with three deaths in rapid succession during the spring of 1969. On a population basis, these deaths made the Newark mortality greater than any other city in the USA. The problem relates, at least in part, to the inordinate number of deteriorated and dilapidated buildings. According to the 1960 census,[1] 85 per cent of units with nonwhite heads of household were so characterized. It is augmented by the great prevalence of pica† among the children. Some 30 to 70 per cent of all children attending municipal clinic in deprived areas may be expected to have a history of this oral behavior.[2, 3, 4] Newark is not likely to have a lower prevalence since pica† often reflects the cultural patterns of the rural south and half of Newark's young mothers[5] were born in southern states.

Alarmed by the apparent severity of the problem, the College of Medicine and Dentistry of New Jersey at Newark established an Environmental Toxicology unit in the Department of Public Health and Preventive Medicine, July 1, 1970. A survey was undertaken to assess what has been done and what was known.

Situation Prior to July 1970

Screening in Newark from January 1, 1969 through March, 1970 was carried out by urine analysis using the Davis method** for detection of δ-aminolevulinic (ALA). During this period, 1973 ALA tests were done with 113 positives, a yield of under 6 per cent. Additionally, 833 coproporphyrin (COPR) tests were made of which 21 were positive, a yield of 2.6 per cent. Subsequent blood lead levels on the children with positive urine tests are shown in Table I. Forty-six per cent of the bloods showed lead concentrations of 40 mg per cent or higher. This

Table I

Blood Lead Levels in Children with Positive Urine Screening Tests

Urine Test	Number Positive	Number with Blood Lead Levels 60mg	59-40 mg	40 mg	No Blood Lead Determination
Aminolevulinic acid	113	23	10	45	35
Coproporphyrin	21	2	6	4	9
Total	134	25	16	49	44

means that fifty-four per cent of the bloods taken because the ALA test was positive were considered non-diagnostic. In Newark 94 children were hospitalized for lead poisoning between January 1, 1969 and March 1, 1970, 28 of these as a result of the urine screening program and the other 66 by direct hospital admission. An additional 43 children had blood lead levels 60 mg per cent or higher detected during the screening program, but were not admitted. Many authorities believe the diagnosis of lead poisoning should be made if blood levels exceed 40 mg per cent. If this is used as the upper limit of normal, the number of children with dangerously high levels who were not hospitalized increases from 45 to 65. Thus, the maximum number of children recognized as lead poisoned was 159. In most cases among the 66 hospitalized with symptoms of lead poison-

*The author is Assistant Professor of Public Health at the College of Medicine and Dentistry of New Jersey at Newark.

†Many of today's medical students here never heard of "pica"—a craving for substances not fit for food.

**Bio-Rad, New York City.

ing, the diagnosis of lead intoxication was made promptly by the private physician or hospital staff. But in an important minority the correct diagnosis was not made early in the course of the disability.

The various presenting manifestations in these children, summarized in Table II, account for some of the difficulties in establishing the diagnosis. Delay in recognition and

Table II
Mode of Presentation of 94 Cases Subsequently Treated for Lead Poisoning, Newark 1969

Asymptomatic		
Screening with ALA[1] in urine	26	
Screening with COPR[2] in urine	2	
Case of lead poisoning in relative	7	
Pica	9	
Neurologic symptoms	18	
Convulsions		12
Drowsiness		6
Gastrointestinal symptoms	18	
Vomiting		14
Anorexia		3
Abdominal pain		1
Bone abnormality on x-ray	2	
Anemia	2	
Other, including combinations of symptoms	10	
Total	94	

[1]aminolevulinic acid
[2]coproporphyrin

treatment of this chameleon disease, which masquerades as a common illness of the nervous system, the gastrointestinal system, and/or the bone marrow, can be most serious as is illustrated in the following cases.

A 3 year-old girl had been followed for tuberculous adenitis regularly during the year before her death. During that year she had shown no weight gain. One week before her terminal admission she was treated symptomatically in the emergency ward for persistent vomiting. Four days later an infiltrate was found on a chest x-ray and she was hospitalized for presumed tuberculosis. The vomiting suggested lead intoxication and blood for lead levels was obtained. Treatment was not initiated pending return of the laboratory studies. Three days later she convulsed, and, despite reduction of blood lead concentration from 100 mg per cent to 20 per cent, she died.

The other patient was first admitted for lead poisoning in September 1968. He was treated successfully and discharged. In June 1969 he was seen again with a two-week history of lethargy and convulsions. Medical attention had not been sought during this 14 day period. On admission, hemoglobin was 5.6 gm per cent and the blood lead 190 mg per cent. Despite vigorous therapy, the child died on the seventh hospital day.

The children admitted for therapy generally responded favorably. But, on discharge, they usually face the environment in which they had originally been poisoned. One third of the children hospitalized in this 15 month period were suffering second or third episodes of lead intoxication.

The importance of follow-up of poisoned children is underscored by our cases. Children with lead poisoning clearly cannot be discharged to be seen only if symptomatic. The next episode, if not fatal, is too likely to be one from which the child does not fully recover. Without a specific focus on the dangers of repoisoning, this possibility was usually ignored during the asymptomatic and more reversible stages. When mothers brought a previously poisoned child for care of acute and unrelated conditions, only infrequently was the opportunity used to obtain a blood for lead level determination. The child's fracture, lacerations, abscess, hernia, or common cold was the sole focus of the emergency room physician's attention.

To provide adequate follow-up, the Department of Pediatrics of the New Jersey College of Medicine and Dentistry at Newark established in March 1970 a weekly "Lead Clinic" for children discharged from Martland Medical Unit after admissions for lead poisoning. (Three-fourths of the cases in Newark are cared for in this insitution.) This clinic arranges for these children (together with all members of their household under age 5) to be seen every three months. This is likely to be an interval within which asymptomatic rises in blood lead levels could be appreciated before more serious reactions developed. The follow-up includes review of growth and development, analysis of current symptoms, and determination of hematocrit, hemoglobin, serum iron, and total iron-binding capacity, urinary ALA, and blood lead levels. Pica, past or present, is sought for in the mother and father as well as all children. We made a pre-

liminary study in Newark of the association of pica in parents and children, of 25 cases and 25 control families. Forty-two per cent of the case mothers had pica whereas only 24 per cent of the controls gave this history. Three of the case fathers had pica; none of the control families described this symptom. Although this trend appears interesting, the numbers were small and the differences are not statistically significant.

Siblings of lead poisoned children were usually ignored. Of the children hospitalized for lead poisonings or with blood lead levels 60 mg per cent and over, 60 per cent of the potentially involved siblings (born 1963 through 1968) had neither blood lead nor ALA urine screening done (Table III).

Table III

Results in Known Siblings Born 1963 through 1968 of Children with Blood Leads over 60 mg per cent or Hospital Admissions for Poisoning, January 1, 1969 through March 31, 1970.

	Number of Children	
No blood or urine test performed	54	
Children with evidence of increased body lead	28	
admitted for chelation		14
blood lead 60 mg per cent and over, no admission		4
blood lead 40-59 mg per cent		7
ALA urine positive, no blood lead determinations made		3
Children without evidence of increased body lead (blood lead under 40 mg per cent)	9	
Total	91	

Preliminary Study of Groups of Potentially Lead-Poisoned Children

Under the Division of Epidemiology a summer project to screen children by blood lead levels used two approaches. Initially, a target area was defined by its poor housing conditions and randomly sampled blocks were reviewed systematically by door-to-door search for children age 1 through 5. Subsequently, four "Lead Days" were held in community storefronts, open as a screening service to all who came. The distribution of blood lead levels in children tested by these two approaches is shown in Table IV. More than a third of the children had blood leads above 40 mg per cent; over 7 per cent exceeded 59 mg per cent.

Although a substantial number of lead poisoned children have been identified, the figure for those actually poisoned is presumably much higher. It is estimated[2, 6, 7, 8] that 3 to 8 per cent of children under age 5 residing in city slums have blood leads 50 mg per cent or higher. Newark has a major problem with housing, with 85 per cent of families with nonwhite heads of household living in deteriorated or dilapidated housing in 1960. Since 1960, the ratio of blacks in the population has almost doubled. Currently over 8500 infants are born each year to Newark residents creating a population of children between age 1 and 5 of over 40,000. According to preliminary screening, the number with excessive body burdens of lead is likely to be in the 13,000 range, constituting a public health problem of the first magnitude.

Spanish-speaking families with lead poisoning have been infrequently noted in Newark. Few children with Spanish family names appear among the 94 hospitalized despite the fact that in 1967 Newark was estimated to have a Spanish-speaking population of 12 per cent.[5] This probably does not reflect differences in housing. It *may* reflect a lesser amount of pica in this group. The most likely explanation, however, is that few Spanish-speaking children have been hospitalized because this group receives even less medical attention than the rest of Newark.

The screening program conducted by the Newark Division of Health during 1969 and into 1970 was based on aminolevulinic acid determination. The low yield of true positives, and the high rate of false positives among these urine tests (compared to blood lead levels as standards) reflect both the limits of the procedure and the difficulty of carrying out suitable collections. The specimens should be from the first voiding of the day corrected to proper pH and either stored fro-

zen or studied promptly. None of these requirements is met easily either by the average household or the usual screening programs. Large-scale programs in Chicago and New York City have not used urine ALA as the screening procedure, but have instead focused on blood concentrations.

The small proportion of those at risk who did receive screening (less than 7 per cent) emphasizes the need for wider educational efforts. Clearly, the delay in recognition is not only due to a lack of awareness on the part of physicians but also on the part of parents as illustrated by our second case and by another child not yet two years of age who vomited daily for three weeks before he was brought to medical attention. At that time, the blood lead concentration was found to be 290 mg per cent. Parental ignorance can also create the problem. Some mothers in Newark have used lead-containing plaster as pacifiers. One well-intentioned mother collected paint flakes for the child to eat "like potato chips" while watching television.

Public education can help bring the child with pica, or suspicious symptoms, to medical attention. An article on lead poisoning made one father recognize his child's pica for the danger it was. The child's blood lead was found to be 260 mg per cent. In six other families, lead intoxication was found because the parents brought their children in for review after learning that pica could lead to lead poisoning. An additional seven families had first been concerned for their own children because of a case of lead poisoning known to them.

The necessity of following children once identified as having excessive body burdens of lead is obvious. The children who were identified at any stage as requiring further investigation but who failed to obtain it are summarized in Table V. Most disturbing were the 43 cases with blood leads 60 mg per cent or higher in which there was neither a subsequent hospital admission nor another blood lead level obtained. Sometimes the failure of further investigation related to difficulties inherent in locating families. In other cases the family could not be persuaded to have additional tests made. In still others, lack of follow-up was due to inadequate communication among various interested groups. For example, the public health nurses and sanitarians, on whom the identification of the source of poisoning currently depends, did not have all names of positive ALA determinations or of all children with bloods over 60 mg per cent. Failure to follow siblings is all the more distressing in the light of the data showing that one-third of siblings[9] of children with lead poisoning may be expected to show increased body burdens of lead. These studies are supported by our own data. In siblings who were tested, almost half required treatment.

Inadequate housing has been a major obstacle to protection of families with lead-poisoned children. During or shortly after hospitalization, the home is usually visited by the Newark public health nurse and the sanitarian. The latter reports on both the condition of the paint-bearing surfaces and the per cent lead by weight in a sample paint chip. Chips of paint were tested from 64 houses in which a lead poisoned child lived. Only one-third were reported to contain over 1 per cent lead. From studies in other slum areas,[10] the rate of positives should be 50 to 70 per cent or higher. The low yield in the Newark experience may be explained by the paucity of samples submitted from each dwelling unit, in many instances only one chip.

Table V

Failure to Continue Indicated Investigation for Lead Poisoning Among Children in Newark

		%
COPR urines positive with no subsequent blood lead	99/21	43
ALA urine positive with no subsequent blood lead	33/113	31
Total blood leads with values 40 to 59 mg per cent without subsequent blood	22/169	13
Total blood leads with values 60 mg per cent or higher without treatment	43/172	25
Total		109

Some 59 notices were served on the dwellings involved in these cases for violation of the city code. Not more than three landlords have actually been fined, although code compliance was rare. In few instances was any definitive action taken during the child's hospitalization either to relocate the family or redecorate the dwelling. With half the families known to be on welfare, few could afford to repair the apartments with their own resources. Children had to be discharged, on the average, after six or seven days, the need for acute care pediatric beds making longer hospital stays impossible. Then the children returned to the environments in which they had originally been poisoned.

Newark may have the most serious lead problem in the nation: over 7 per cent of the blood concentrations were found to be 60 mg per cent and higher. Another 32 per cent of the sample had blood lead levels of 40 to 59 mg per cent. This is considerably higher than the most recent New York figures showing three-fourths of the values under 40 mg per cent, 19 per cent between 40 and 59 mg per cent, and 5 per cent 68 mg per cent and higher (Table IV). The samples may not be similar in their relevant characteristics but the gravity of Newark's problem is emphasized by the comparison.

Thus, in Newark until late 1970, screening involved too few children, the ALA test was found not to be reliable, evaluation of siblings was inadequate, and children once suspected of lead poisoning often had inadequate investigation of themselves, their siblings, and their environment. Screening by blood levels in the summer of 1970 sharpened the sense of urgency about lead poisoning in Newark.

The inadequacy of present resources as well as the inefficiency in their deployment became a community concern. It then became clear to parents, community leaders, professional workers in health and health administration, the city administration, and the medical college that the results of phases 1 (the evaluation of the 15 month preblood screening period) and 2 (the preliminary blood screening) mandated that the entire estimated at-risk population in Newark have lead determinations made in 1971. The outcome of the planned phase 3 (total screening) and 4 (remedial and preventive efforts) will be the subject of a second report in 1972.

Summary

Assessment of lead poisoning in Newark during the 15 months January 1, 1969 through March 31, 1970 showed that the number of children screened, treated, and followed were far below the number projected at risk of lead poisoning. The estimates of the severity of the problem were based on two types of screening with blood lead level determinations which showed that 7.4 per cent of the children had blood lead levels 60 mg per cent or higher, while another 32 per cent had blood leads 40 to 59 mg per cent. Community interest and resources have been mobilized by the definition of the gap between the needs of the city's children and the amount of control achieved.

Table IV.

Blood Lead Levels by Type of Screening, Summer 1970.

mg %	Newark						New York City	
	Block study		Lead Days		Total			
	N	%	N	%	N	%	N	%
less than 40	81	58.7	282	61.8	363	61.1	27,184	75.4
40-59	44	31.9	144	31.6	188	31.5	6,991	19.1
60 and over	13	9.4	30	6.6	43	7.4	1,896	5.2
Total	138	100	456	100	594	100	36,071	100

N = number

Bibliography

1. U.S. Census of Housing 1960, Newark, N.J., U.S. Department of Commerce

2. Bradley, J. Edmund, *et al.: J of Ped,* 49:1 (July 1956)

3. Lanzkowsky, Philip: *Arch Dis of Child,* 34:140 (1959)

4. Baritrop, D.: *Amer J Dis Child,* 112:116 (August 1966)

5. Newark, New Jersey, Population and Labor Force Spring 1967, Institute of Management and Labor Relations, Rutgers-The State University

6. Oberle, Mark W.: *Science,* 165:991 (September 5, 1969)

7. Griggs, Robert C., *et al.: JAMA,* 187:703 (March 7, 1964)

8. Rothschild, Edmund: *NEJM,* 283:704 (September 24, 1970)

9. Chisolm, J. J. and Harrison, H. E.: *Pediatrics,* 18:943 (1956)

10. Schucker, George W., *et al.: Public Health Repts,* 80:969 (November 1965)

11. Spector, Michael J. and Guinee, Vincent F.: "Epidemiology of lead poisoning in NYC-1970," APHA 98th Annual Meeting, Houston, Texas, October 26, 1970)

100 Bergen Street

Radiation Safety Standards

The Food and Drug Administration has proposed that equipment manufactured for x-ray diagnosis in the healing arts incorporate new radiation control features to reduce exposure during x-ray examination. The new standard was developed by the FDA Bureau of Radiological Health. X-ray examinations, the FDA noted, constitute the largest source of man-made radiation reaching American people.

"While the beneficial applications of radiation in the healing arts are well recognized, exposures from diagnostic x-ray procedures have far more public health significance than exposures from all other man-made radiation sources," said Charles C. Edwards, M.D., Food and Drug Commissioner. "More than 90 per cent of all human exposure to man-made radiation comes from the diagnostic use of x-ray, in contrast with about one per cent from radioactive discharges from nuclear power plants about which there has been so much public concern."

One new proposal would require stationary general purpose x-ray machines to be designed to insure limitation of the beam to approximately the size of the body area to be examined. The other would require radiographic equipment to be able consistently to reproduce exposure and, therefore, image quality for given settings for voltage, current, and time.

Failure to keep the x-ray beam within the area of the body undergoing diagnostic examination is one of the commonest causes of unnecessary x-ray exposure. Reduction of as much as 50 per cent or more in the genetically significant dose from diagnostic x-ray can result from limiting the beam to approximately the size of the film or other x-ray receptor. The exposure reproducibility requirement of the standard would enable the technologist to know with confidence what results to expect at various equipment settings and thus to avoid picture retakes, a common cause of unnecessary diagnostic x-ray exposure.

X-ray equipment manufacturers would be given a year following the effective date of the standard to produce equipment complying with its provisions. The proposed standard would become effective upon republication in the *Federal Register* after 60 days for public comment.