Impact of Community Screening on Diagnosis, Treatment, and Medical Findings of Lead Poisoning in Children

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THE IMPACT OF A LEAD POISONING PREVENTION program can be measured in part by analyzing children's records in hospitals and clinics subsequent to their being diagnosed as lead poisoned. In 1969, soon after a move to Newark, N.J., members of the Department of Preventive Medicine and Community Health, New Jersey Medical School (NJMS), began to look seriously at the lead poisoning of children in that city. The condition's frequent occurrence and a variety of informal data suggested that Newark had as serious a problem as any community in the nation (*1*). After three lead-related deaths of Newark children in 1969, the limited screening of the city's division of health was increased, and the services of the New Jersey Medical School were added to mount a lead poisoning control program. Federal funding made it possible for the program to expand citywide in 1972.

Using a mobile screening van, which moved throughout high-risk areas of Newark, and local pediatric clinics as the primary sources of blood samples, the Newark Childhood Lead Poisoning Prevention and Control Program began to respond to the problem. Developed as a collaborative effort, the program was jointly administered by the Newark Division of Health and the medical school's departments of preventive medicine and of pediatrics. Its objectives included testing all at-risk children, ages 1 to 6 years, first with a micromethod (fingerstick) and then, if the PbB exceeded 40, with a confirmatory venous blood examination. (In this paper we use the term PbB, meaning whole blood lead concentration, expressed in micrograms of lead per deciliter of whole blood.) If both studies showed levels of 40 or greater (*2*), the child was referred for medical observation and treatment as needed. In 1975, the addition of an erythrocyte protoporphyrin (EP) test reduced the need for obtaining a repeat PbB determination on tests with initially high results. A confirmed PbB of 60 or more in asymptomatic children mandated prompt in-hospital or outpatient treatment.

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Antonia Ty, MD, of the school's Department of Pediatrics; Steven Marcus, MD, Department of Pediatrics, Beth Israel Hospital; and Anna Haratounian, MD, Department of Pediatrics, United Hospitals; provided information and access to the appointment files and medical records of their institutions' lead poisoning clinics in Newark.

Screening clinics included baby keep-well stations, hospital outpatient departments, and neighborhood health centers in addition to special 1-day programs called Lead Days. Pediatricians in private practice also screened their patients through the program so that virtually all PbB determinations on Newark children were carried out under program auspices. Information on each child tested was sent to the Central Lead Registry at the NJMS for collection, compilation, and followup information.

This study was designed to review and analyze data retrieved from medical records of lead-poisoned children who had been hospitalized for chelation or chelated on an outpatient basis. Our purpose was to ascertain the impact of the lead program on the incidence of the condition, on whole blood lead concentration levels, repeat episodes, and medical findings about the children.

Methodology

Charts from January 1, 1972, through December 1976 with a discharge diagnosis of lead poisoning were reviewed in the five Newark hospitals where children had undergone chelation for lead poisoning. In addition, outpatients' charts were searched for information on outpatient (OPD) chelations. During this designated period the Newark Lead Poisoning Prevention and Control Program was in full operation. Certain of its major components were administered within the medical school; these included the laboratory for testing PbB, the registry of all tests performed, and a special lead clinic for followup of patients with elevated levels. The program also involved the cooperative efforts of the five Newark hospitals, including the primary teaching hospital and four general community hospitals; three of these were teaching affiliates. A community education program sought to enlighten parents and guardians about childhood lead poisoning by providing information on its cause, prevention, screening sites, and treatment sources.

The lead testing procedures included the fingerstick micromethod—spotting the specimen of whole blood on filter paper (*3*)—and the macromethod that used peripheral vein blood. With both techniques, the lead level was determined by atomic absorption spectrophotometry.

The Central Lead Registry recorded the PbB and hematocrit along with demographic data (mother's name, child's name, address, birth date, race, and sex), source of blood sample, and whether the test was for screening, confirmation, or diagnosis of a clinically suspect case or of a hospitalized patient.

Only children residing in Newark at the time of initial diagnosis were included in the study. Since retrieval of medical charts differed in the record rooms of the five hospitals, supplementary data were obtained from the computerized lead registry to search for additional hospitalized children who might have been missed. This search focused on children with PbBs greater than 50 because chelation was infrequently performed at lower PbB concentrations (*4*).

All chelations of outpatients had to be traced in this way since the hospitals did not keep a registry of such patients; the information was available, however, from the charts of individual patients of the hospitals' outpatient clinics. To find the OPD chelations, a computer list was compiled for each hospital source of all PbBs of children who had at least one test level of more than 50. Those who had tests over several successive weeks with an accompanying drop in the PbB were selected as possible cases. Cards in the lead registry files were pulled to check accuracy of data. Once a hospital's list was in final form, charts were requested from the hospital and information collected. Using these methods, we attempted to identify every child who, as a hospital inpatient, had a diagnosis of lead poisoning or had received chelation treatment as an outpatient.

Children with a confirmatory PbB of 60 or higher were referred for immediate medical treatment. Other children were tested for lead poisoning after being admitted for various somatic complaints. In either circumstance, chelation was not initiated until the PbB was determined, and then it consisted of a 5-day course of EDTA–BAL or Ca EDTA. Continued communication among the appropriate clinicians at the five hospitals concerning diagnosis and treatment led to a similar approach at each institution.

Data collected included date of hospitalization or initiation of OPD chelation treatment; length of hospital stay; blood lead level; child's age, sex, and ethnic group; symptoms or signs exhibited leading to treatment; and medical findings. This information was then punched onto cards, tabulated by computer, and analyzed.

Results

During the 5-year study, 525 children under 7 years were diagnosed for the first time as lead poisoned. An additional 27 were treated during the study period after having been diagnosed as lead poisoned during the period 1970–71, but these were not included in the basic study group. The ethnic distribution of the study group was 88 percent black, 10 percent Hispanic, and 2 percent white; males constituted 55 percent of the group.

Table 1. Lead poisoning diagnoses by year, Newark, N.J.

Year	Number of diagnoses	Inpatients		Outpatients	
		Number	Percent	Number	Percent
1972	184	150	83	34	17
1973	280	195	70	85	30
1974	177	101	56	76	44
1975	106	73	68	33	32
1976	122	71	58	51	42
Total	869	590	69	279	31

During the same 5 years, 41,972 children received their first blood lead level test. The yearly distribution of these initial tests follows:

Year	Number
1972	7,656
1973	9,692
1974	12,110
1975	6,900
1976	5,614

Of these children 95 percent, or 39,873, were under 7 years and slightly more than 50 percent were boys.

Medical management of the 525 children, after they were diagnosed as having lead poisoning, varied as follows:

Course of action	Number
Inpatient observation only	11
Inpatient treatment of conditions other than lead poisoning	6
Inpatient chelation treatment	307
Outpatient chelation treatment	117
Both inpatient and outpatient chelation treatment	84
Total	525

Thus, of the 525 children diagnosed as lead poisoned, 508 (97 percent) received chelation treatment. The remaining 3 percent included cases in which the parents refused treatment, the child was admitted for another problem, tested for lead poisoning but not treated, or the child was admitted for symptomatic indications of lead poisoning but confirmatory PbBs were not high enough to mandate chelation. In these cases, the patient was hospitalized long enough to verify the PbB or the child was treated for other illnesses. The number of total treatments per child (hospital or OPD chelation or both) varied according to the following distribution:

Number of treatments	Children	
	Number	Percent
0	17	3
1	326	62
2	109	21
3	37	7
4	19	4
5	5	1
6	6	1
7	3	0.5
8	2	0.3
9	1	0.2

A hospitalization did not involve more than one course of chelation treatment, and a single span of medication on an OPD basis was counted as one treatment. OPD treatment as a followup to a hospital treatment did not constitute a new treatment. Although 62 percent of the group had only one treatment and 3 percent had none, 35 percent had multiple treatments. Because of these multiple episodes of lead poisoning, there were a total of 869 diagnoses of which 676 (78 percent) involved asymptomatic presentations and 193 (22 percent), symptomatic presentations. Chelation was administered in 838 episodes (97 percent), including 66 instances in which the PbB determinations were less than 50.

Treatments were most frequently administered during July and August (29 percent or 254). The number of

Table 2. Treatment of children by year of initial diagnosis of case and span of treatment, Newark, N.J.

Year and frequency of treatment	Case initially diagnosed in 1972	Ratio of single to multiple treatments	Case initially diagnosed in 1973	Ratio of single to multiple treatments	Total
Children with single treatment	74	....	110	....	184
Children with 2 or more treatments: [1]					
1972	22	3:1	...	....	22
1973	24	3:1	27	4:1	51
1974	8	....	17	6:1	25
1975	2	....	4	....	6
1976	3	....	6	....	9
Total initial diagnoses	133	....	164	....	297

[1] Case counted in study period year when child received final treatment.

Table 3. Number and average age of children, by year of initial diagnosis, Newark, N.J.

Year	Children with symptoms			Children without symptoms		
	Number	Percent	Average age (months)	Number	Percent	Average age (months)
1972	24	18	26	109	82	31
1973	39	24	35	125	76	32
1974	23	22	42	80	78	35
1975	18	29	31	45	71	32
1976	27	44	43	35	56	36
Total	131	..	..	394	..	..

hospitalizations per year peaked in 1973 at 195 and then dropped to 71 in 1976 (table 1). This trend also applied to the overall number of treatments that included both inpatient and outpatient chelations. Hospitalizations per child ranged from 1 to 9. Multiple treatments involved a large number of children. Available data for cases originally diagnosed in 1972 and 1973 are shown in table 2 as an indication of the span of chelations required by children in the study. For example, in 1976, three children were still being treated after having been initially diagnosed in 1972.

Table 3 shows that the average age of the children with asymptomatic lead poisoning at the time of initial diagnosis did not change much over the 5 years; it ranged from 2½ to 3 years. However, the average age of symptomatic children at first diagnosis varied more widely, rising from slightly over 2 years to 3½ years, dipping to 2½ years in 1975, and rising again to 3½ years in 1976.

Symptoms recorded on admission and medical findings at the time of diagnosis provided a basis for assessing the severity of illness. Major signs and symptoms included convulsions, abdominal pain, vomiting, irritability, anorexia, diarrhea, or constipation. All such information was accepted as charted by the physician. Children with pica or anemia alone were considered asymptomatic. Anemia is endemic in Newark and is difficult to ascribe specifically to lead in most cases. An abnormal EEG finding was considered indicative of central nervous system (CNS) involvement.

Although the number of lead poisoning diagnoses per year peaked in 1973, the proportion of children presenting with symptoms decreased from 50 percent in 1967–68, to a low of 17 percent in 1972 and then climbed back to 41 percent in 1976 (table 4). This fluctuation reflected not so much a change in the number of symptomatic cases but rather the detection of larger numbers of asymptomatic cases from 1971 to 1974, resulting from the emphasis on screening activities. In 1975, the program began to focus more heavily on followup services.

Over the 10-year period encompassing this and an earlier study (*5*, *6*) there were no consistent changes in

Table 4. Lead poisoning diagnoses of children with and without symptoms, Newark, N.J.

Time interval	With symptoms		Without symptoms		Total
	Number per year	Percent	Number per year	Percent	
	Data of Browder and co-workers (*5*)				
1967–68	25	50	26	50	102
1969–70	34	41	48	59	164
1971	26	18	120	82	146
	Present study				
1972	32	17	152	83	184
1973	56	20	224	80	280
1974	29	16	148	84	177
1975	26	24	80	76	106
1976	50	41	72	59	122

Table 5. Occurrence of symptoms in lead-poisoned children, Newark, N.J.

Year	Seizures		CNS involvement short of seizures [1]		Gastrointestinal symptoms [2]		Total occurrences per year
	Episodes per year	Percent	Episodes per year	Percent	Episodes per year	Percent	
	Data of Browder and co-workers (5)						
1967–68	6	35	6	35	5	30	17
1969	10	40	9	36	6	24	25
1970–71	8.5	20	18	40	18	40	44.5
	Present study						
1972	2	6	5	16	25	78	32
1973	4	7	6	11	46	82	56
1974	1	3	4	14	24	83	29
1975	0	0	4	15	22	85	26
1976	3	6	5	10	42	84	50

[1] Irritability or abnormal EEG or both. [2] 1 or more of abdominal pain, vomiting, anorexia, diarrhea, constipation.

the number of symptomatic episodes seen yearly, but changes in the proportion of symptomatic children with seizures or other CNS manifestations were striking. CNS abnormalities short of seizures included an abnormal EEG or irritability, or both. Gastrointestinal symptoms included one or more of the following: abdominal pain, vomiting, anorexia, diarrhea, or constipation. For comparison, each category was mutually exclusive with episodes assigned according to the most serious symptom exhibited. Before the screening program was in full swing, 20 to 40 percent of symptomatic episodes involved seizures, and an additional 35 to 40 percent included CNS abnormalities short of seizures. In contrast, between 1972 and 1976, seizures ranged from 3 to 7 percent and CNS manifestations involved 10 to 16 percent of the symptomatic episodes per year (table 5).

The absolute number of episodes with CNS disease also declined—from 96 to 34—between the 1967–71 and the 1972–76 periods. These clinical findings are mirrored by the mean PbBs of symptomatic children. In the study by Browder and co-workers, PbBs of symptomatic children averaged 130 in 1967–68 and 86 in 1971 (5). During the subsequent 5 years, concentrations in symptomatic children continued to fall, although very slowly (table 6). In the last 3 years of our study, concentrations ranged from 67 to 71. Mean concentrations in asymptomatic children fell consistently during the study period from 72 to 59, suggesting progressively earlier intervention in these lead-intoxicated but asymptomatic children. For the purpose of comparing the data in the two studies, arithmetic means are shown in table 6. However, since it has been observed that PbBs in children are log normally distributed (7), geometric means ± 2 standard deviations are also shown in table 6.

Discussion

Symptomatic and asymptomatic cases detected through intensive program efforts can provide a reasonably accurate picture of the extent of childhood lead poisoning in an area or population group. In this study, the total number of detected children increased yearly with screening until 1973 and then decreased considerably.

During the period under study, 16.4 percent (6,335)

Table 6. Average blood lead concentration for symptomatic and asymptomatic lead-poisoned children at first diagnosis, Newark, N.J.

Year	Symptomatic children		Asymptomatic children	
	Lead levels	SD	Lead levels	SD
	Data of Browder and co-workers (5)			
1967–68	130	...	76	...
1969	140	...	100	...
1970–71	86	...	78	...
	Arithmetic means, present study			
1972	73	16.8	72	23.3
1973	83	48.0	71	17.2
1974	67	15.9	68	18.9
1975	71	32.8	65	18.1
1976	67	29.0	59	10.8
	Geometric means, $\pm$ 2 SD, present study [1]			
1972	70.4	45.1–109.9	69.8	41.8–116.7
1973	75.8	34.2–168.2	69.3	44.2–108.6
1974	65.2	40.6–104.5	66.1	41.9–104.2
1975	66.6	32.3–137.3	63.4	39.2–102.4
1976	62.5	30.2–129.3	57.8	40.0– 83.4

[1] Calculated from logarithmic transformations of original lead values.

of the children screened had PbBs of 40 or greater, but only 3,130 of these had confirmatory tests, and only 1,566 (4 percent) had a PbB of 40 or greater on the repeat test (*8*). An additional 2,500 children were initially tested, with the macromethod—by physicians, hospitals, and clinics—because they exhibited symptoms suggestive of lead poisoning. Although both groups constituted the total of initially tested children, data are not available on confirmatory tests for the group with suggestive symptoms.

In a Milwaukee study of 11,675 children screened during 1972–74, 22 percent had confirmed PbBs of 40 or higher (*9*). Data from childhood lead poisoning control projects for the period July 1, 1974, to June 30, 1975, showed that 440,650 children were screened, and 28,597 (6 percent) were found to have a lead problem (*10*). High proportions of lead-poisoned children (confirmed undue lead absorption) were found among the total tested in St. Louis, with 19 percent, in Boston and Milwaukee with 12 percent, Baltimore with 11 percent, Philadelphia with 10 percent, and New York City with 9 percent. In contrast, the proportion in Newark was only 7 percent of those tested.

The lower percentage in Newark may well reflect the fact that extensive screening was confined to a defined high-risk lead belt area in the central city, an area that also included public housing units which were considered lead free. A study by Griggs and co-workers (*11*) confirmed that public housing constructed since 1950 was not likely to induce lead poisoning. Another factor which must be considered is the failure to follow initially high PbBs with confirmatory tests in 50 percent of the children screened.

The large proportion of children who required multiple treatments (190 in the total 5-year period) suggests a high level of repoisoning because of failure to eradicate sources of lead. This component of the control program proved to be difficult to implement and may well account for the need for a majority of the successive treatments although, in some cases, reoccurrence of elevated blood levels may represent endogenous mobilization of lead from tissue deposits. A followup study by Foster and co-workers (*12*) on efforts in Newark to eradicate lead at its source revealed an extremely low rate of success; only 25 percent of the houses investigated had been properly abated.

There is also evidence that sources other than lead paint may be involved. A study by Cohen and co-workers (*13*) suggested that urban areas may contain high levels of airborne lead, thus compounding the problem. Caprio and co-workers (*14*) found the concentration of lead in soil to rise in relation to its proximity to heavily traveled arteries, suggesting that children who live close to heavy traffic areas are at greater risk. Newark has a number of such thoroughfares, and this possible source may excerbate the problem.

In evaluating this program over time, the decreases in numbers of children suffering seizures and CNS involvement and the decrease in the average blood lead level of symptomatic children from the higher levels in preprogram years strengthen the contention that constant surveillance through screening, health education, and followup treatment can prevent the serious consequences of lead poisoning. Nevertheless, these do not reflect efforts to address the etiology of this condition.

Evaluation of the NJMS Lead Registry data suggests that the program has had only a limited impact on the incidence of elevated blood lead levels among Newark children, indicating that primary prevention was not successful (*15*).

Even if encephalopathy and mental retardation are prevented, there remains the serious possibility of minimal brain dysfunction in both symptomatic and asymptomatic children. Pueschel (*16*) reported minor neurological dysfunction and fine and gross motor impairment in 23 to 27 percent of a group of 58 asymptomatic lead-poisoned children. In a related study, de la Burdé and Choate (*17*) followed a group of children and found that lead-exposed children at age 7 fell into the suspect or abnormal range of neurological functioning more than twice as often as nonexposed children and were eight times as likely to have behavioral problems. Mellins and Jenkins (*18*) investigated 21 symptomatic cases of lead poisoning in 1953 and found marked impairment in the children's fine motor coordination and language ability as well as an increase in behavior problems. Neurological sequelae were detected in about 30 percent of a group of poisoned children presenting with gastrointestinal symptoms in a study by Perlstein and Attala (*19*). Data obtained during a 1973 study in El Paso, Tex., (*20*) indicated a significant difference in performance IQ among children with PbB determinations of 40 to 80 but no difference in verbal IQ scores. In a more recent study, Needleman and co-workers (*21*) found that children with high dentine lead levels were less competent in areas of verbal performance and auditory processing, had impaired ability to sustain attention, and presented a higher level of undesirable classroom behavior. Such studies, although not conclusive, strongly suggest that developmental problems are a consequence of asymptomatic poisoning or poisoning with mild to moderate abdominal, but no CNS, manifestations.

Obviously, these are critical questions demanding additional research. During the study period, only 50 per-

cent of those children with a PbB of 40 or greater at the time of initial screening had confirmatory tests (*8*). Of these, half had PbBs of 40 or greater on the repeat test (made within 2 months of the initial test). An additional 2,500 were tested as suspect, using venous blood, after they had symptoms of possible lead poisoning. Of the total, only 525 were judged to require treatment. The small number of these treated was due to failure to follow all initially screened children with positive findings, failure of the high levels to persist on repeated testing (the level was often caused by exogenous contamination of the fingerstick specimen), and to the fact that a large number of the children with PbBs over 40 appeared healthy and were not treated.

Conclusion

Review of the medical records of 525 children diagnosed as lead poisoned during the 5 years indicates that a citywide lead poisoning prevention program has resulted in a decrease in the number of children with lead-induced seizures and with CNS involvement, as well as a decrease in the mean PbBs in symptomatic children. However, the large number of multiple treatment episodes suggests a serious problem of re-poisoning because lead was not eradicated from the children's environment. The high proportion of children with PbBs of 40 or greater also indicates that the program has had a limited impact on the incidence of elevated blood lead levels.

Findings of this study, then, indicate that, although extensive screening and clinical followup are effective in preventing the more serious aspects of lead poisoning, they do not address the issue of prevention, either of the initial lead poisoning episode or of subsequent ones. If abatement efforts are not thorough and effective and if they are not coordinated with medical treatment, the consequences of lead exposure continue, even at a lower level.

One then has the responsibility to ask: are re-poisonings and repeated chelations caused by continuing exposure to lead, even at lower levels, consistent with the goal of lead poisoning prevention, and is this a satisfactory deployment of considerable resources in an extensive community health program? However, considering the possibility of subsequent neurological impairment caused by high PbBs, must not one overlook the frustrations of inadequate control of the environmental sources and continue mass screening of children as perhaps the major avenue of prevention currently available?

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