Lead Poisoning in Childhood:

Epidemiology, Manifestations, and Prevention

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Lead intoxication in childhood is largely a disease of the big cities, and especially of the slum areas where old deteriorated housing prevails. Here is an account of observations and experiences in the metropolis of New York City.

LEAD poisoning in child ntinues to be a major public health probl w York City and other urban areas. It lea eath in about 15 to 20 per cent of cases, and to neurologic and mental disturbances from encephalopathy in over 25 per cent of the survivors. Of 61,167 poisonings reported to the New York City Poison Control Center between 1955 and 1963, 3 per cent or 1,704 cases were due to lead. Figure 1 summarizes the distribution of poisonings according to etiology during this period.

For many years, lead poisoning was recognized in children only when associated with lead encephalopathy—when clinical signs and symptoms of central nervous system irritation aroused the examining physician's suspicion of the possibility of lead poisoning. Little, if anything, was known by the average physician of the sub-clinical or asymptomatic phase. Systematic, careful epidemiologic investigations of the natural history of the disease were not widely available and many victims went unrecognized and untreated.

Lead poisoning in children may be compared to an iceberg, with the small visible portion being cases of lead encephalopathy and the major portion being the invisible and as yet asymptomatic patients.

Epidemiology of Lead Poisoning

Significant gains will be made in controlling and preventing lead poisoning in children, only when we have more knowledge of its natural history. The essential tools of epidemiology must be utilized to obtain needed facts about the agent, host, environment, mode of occurrence, place of occurrence, and the interplay and interactions between these components. An intensive study must be made of how lead poisoning occurs, where, when, to whom, and why. The population at risk, the injurious agent and the unsafe environment must be identified, investigated, and defined.

The agent is usually lead-containing paint. The host is a child between one and five years of age, particularly, one with pica, the etiology of which has been extensively outlined elsewhere.[1] The usual environment is an old, poorly repaired house in a slum area with many coats of lead-containing paint, one on top of another, covering the walls, ceilings, woodwork and other indoor surfaces. Fallen paint flakes from walls and window sills and painted plaster from walls are easily accessible to the young child who is a "mouther."[2] While inhalation of lead fumes and absorption through the skin may also cause lead poisoning, all cases in children reported in New York City in the past decade have occurred as a result of ingestion.

The Search for Cases

Pica is a particularly valuable clue in the epidemiology of lead poisoning.[3] Its incidence is now an integral part of the medical records in child health stations of the New York City Department of Health. These agencies follow over 200,000 infants and children from birth to school entrance. Since 1955, a history of pica has been a routine part of each medical history. Inquiry is also made about pica in other members of the family.

Where pica history is positive the examining physician, alerted to the possibility of lead poisoning, conducts examination with special reference to possible associated signs and symptoms. A specimen of blood is obtained in a lead-free tube from every child with pica, as well as from all siblings under six years of age. The blood is examined in Health Department Laboratories for lead content.

If the concentration of lead is 0.06 mg./ml. whole blood or higher, the child is referred for

* Deceased February 8, 1966.
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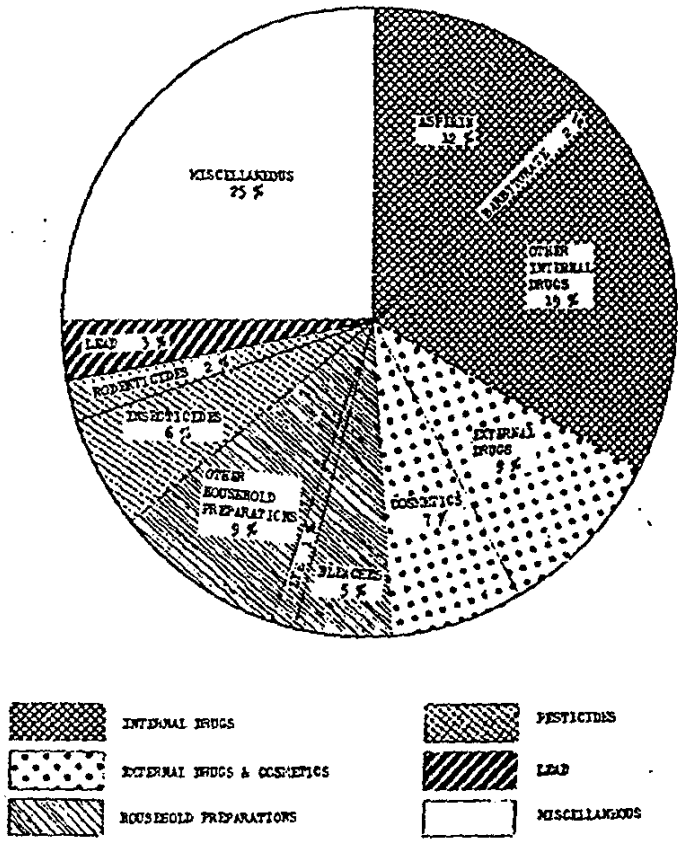


FIG. 1. Poison Control Center, New York City Department of Health, percentage distribution of 61,167 cases of poisoning reported in children under 20 years of age, by type of poison. New York City: 1955–1963.

treatment to his family physician or to a hospital clinic. Over 80 per cent of children with pica in our study had lead poisoning. Siblings of confirmed lead poisoning cases, though usually asymptomatic, produce a high case yield as determined by blood lead investigation.

A diagnosis of lead poisoning is made if the blood lead concentration is 0.06 mg./100 ml. or higher and if two or more of the following signs and symptoms are present: gastro-intestinal signs of anorexia, vomiting, abdominal pains or constipation; hematologic findings of pallor or anemia; neurologic signs of irritability, stupor, lethargy or convulsions; and roentgenologic signs of increased density of the long bones or opacities representing lead flakes in the abdomen.

If there is a history of pica plus a blood concentration of lead of 0.06 mg. per 100 ml., but no signs or symptoms of disease, the case is classified as "possible" lead poisoning. The family is told of the harmful effects of pica and of the need for periodic repeat blood lead determinations.

In addition, a public health sanitarian visits the home of all confirmed and possible lead poisoning cases to make a general sanitary inspection, with particular reference to evidence of availability and ingestion of lead paints. He also examines plumbing for lead pipes, takes samples of water for chemical examination where indicated, and samples of painted plaster and paint peelings for chemical analysis of lead content.

In the lead belt, the high incidence areas, public health sanitarians visit every house in designated blocks. They take scrapings for lead determination, inquire about pica, take samples of urine from all children under six years for a coproporphyrin test, and advise the parent about the harmful effects of pica and its association with lead poisoning. In the case of a hospitalized child with lead poisoning, a public health nurse visits the home prior to discharge. She obtains additional epidemiological data, urges parents to bring siblings in for examination, and educates the family in the prevention of lead poisoning.

Incidence of Lead Poisoning in New York City

Accurate information is not available on the true incidence of lead poisoning in childhood, since many cases go unidentified and undiscovered. Lead poisoning has been reportable in New York City for many years, but only 143 cases were reported from 1950 to 1954, an average of 29 cases per annum. Last year, 1964, over 500 cases were reported. The case fatality rate from 1950 to 1954 was 27 per cent, compared with less than 2 per cent in 1964.

There is no reason to believe that the incidence is higher in 1964 than it was in 1954. In fact, there is more new housing now with low lead-containing paint, plus the comprehensive educational program carried on against lead poisoning. The apparent increased incidence is undoubtedly due to the vigorous case-finding program carried on since 1955, which has discovered cases in the asymptomatic stage, prior to the onset of encephalopathy.

Prior to 1958, nearly 60 per cent of all cases were reported from only three hospitals, chiefly because one or more staff members were particularly interested in the condition. Since 1958, however, public health officers have carried on an intensive professional training program. They speak before hospital staffs, display exhibits, and distribute published articles to acquaint physicians in New York with the problem of lead poisoning in childhood. Last year, over 30 hospitals reported cases and requested blood lead examinations.

Table 1 summarizes the incidence of lead poisoning in children, plus case fatalities in New York City from 1954 to 1963. The years through 1950 and 1954 produced only 143 reported cases, but 30 of these terminated fatally—a case fatality rate of 27 per cent. The apparent increased incidence, but decreased fatality rate in subsequent years as summarized in Figure 2 is, we feel sure, due primarily to the additional efforts in case findings currently employed and to the physicians' increased awareness of this problem.

Table 1 also demonstrates a 264 per cent increase occurring in the number of cases reported in 1964 compared to 1960, and a decrease of 87 per cent occurring in the case fatality rate. In addition to the confirmed cases, there were over 100 possible cases in 1964 with blood lead levels of 0.05 to 0.06 mg./100 ml. blood and with a history of pica, but with no symptoms indicative of lead poisoning. These cases are being followed.

Case distribution by age, sex and race is shown in Table 2. The younger age groups, the one to three year olds, contributed 82.5 per cent of the total cases. Distribution varied from 0.5 per cent in the group under one year to 40.7 per cent at age two. After the third year of life the incidence decreased with age. While more cases were reported in males, sex distribution variation was not statistically significant.

TABLE 1. *Cases of Childhood Lead Poisoning Plus Fatalities in New York City From 1954 to 1958*

Year	Cases	Deaths	Case Fatality*
1954	80	12	15.0
1955	115	18	15.7
1956	99	9	9.1
1957	85	9	10.6
1958	116	21	18.1
1959	171	12	7.0
1960	146	18	12.3
1961	181	6	3.3
1962	198	9	4.5
1963	338	7	2.1
1964	509	7	1.4
Total	2,038	128	6.3

* Deaths per 100 cases.

A marked ethnic variation was observed with a disproportionately high incidence in the Puerto Rican and nonwhite groups. Of the 1,460 known cases, nonwhites contributed 46.7 per cent; Puerto Ricans, 39.5 per cent; and Caucasians, 13.8 per cent. This is in marked contrast to the general population distribution in New York City.

According to the 1960 census, the nonwhite population under five years of age was 19.9 per cent, and the Puerto Rican 13.6 per cent of the total. Thus, these two high risk groups contribute only 33.5 per cent of the total population under five years, but they contributed 86.2 per cent of reported cases of lead intoxication. Specific case rates by race are shown in Table 3. It can be seen that the nonwhite rate is about five times higher than the white.

This disparity is a reflection of environmental factors, particularly, dilapidated sub-standard housing. Such homes provide ready access to broken, painted plaster from walls and paint peelings from window sills and ceilings. In nearly 90 per cent of the reported cases, a careful history revealed that affected children ingested flakes of plaster or paint peelings for several months prior to diagnosis.

Distribution of Deaths Due to Lead Poisoning

Nearly 54 per cent of all deaths from lead poisoning occurred in two year olds. No significant difference was noted in the mortality incidence based on sex, except among whites. Here, females outnumbered males two to one, but the total number of deaths in this group, however, are too few to warrant any valid conclusions as

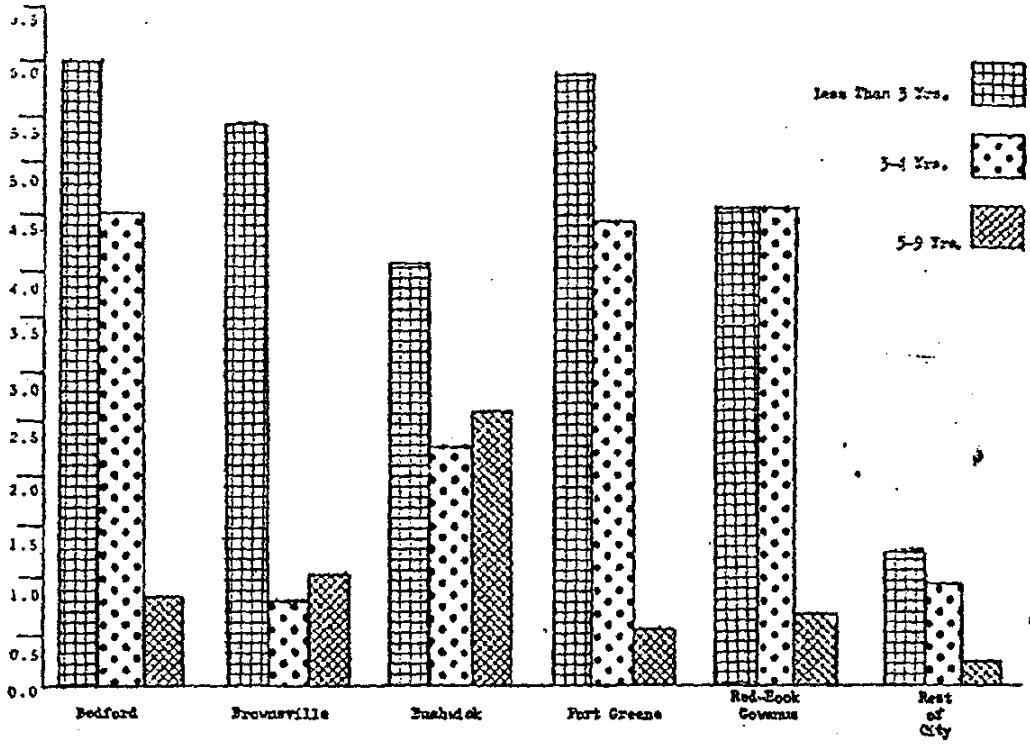


FIG. 2. Age specific rates of reported cases of lead poisoning in January, February and March, 1963, for health districts nos. 2, 3, 4, 6, 8 in Brooklyn: rest of city (per 10,000 population).

to whether the female is more vulnerable. Death statistics are summarized in Table 4.

The Lead Belt

A majority of the cases of lead poisoning in New York City occur in several slum areas outlined in Table 5. A random study conducted from January through March, 1963, showed 151 cases of confirmed and possible lead poisonings. Nearly 46 per cent came from five health districts in Brooklyn and 54 per cent from the remaining 25 health districts. New York City is divided administratively into 30 Health Districts each with approximately 300,000 population.

TABLE 2. *Distribution of Lead Poisoning in New York City According to Age, Sex, and Race From 1954 to 1963**

Age (in Yrs.)	Male					Female						
	White	Non-White	Unk. Race	Total	%	White	Non-White	Unk. Race	Total	%	Total	%
Under 1	4	3	0	7	0.9	1	0	0	1	0.1	8	0.5
1	101	127	7	235	28.8	101	97	8	206	29.0	441	28.9
2	156	153	13	322	39.5	152	132	14	298	42.0	620	40.7
3	79	54	4	137	16.8	74	56	7	137	19.3	274	17.9
4	29	22	5	56	6.9	26	13	1	40	5.6	96	6.3
5	17	6	3	26	3.2	11	7	0	18	2.5	44	2.9
6 & over	18	11	1	30	3.7	7	1	0	8	1.1	38	2.5
Unk. age	0	0	2	2	0.2	2	0	0	2	0.3	4	0.3
Total	404	376	35	815	100.0	374	306	30	710	100.0	1525	100.0
Per Cent	26.5	24.6	2.3	53.4	24.5		20.1	2.0	46.6	100.0		

* Four cases not included in the above figures, are of unknown age, sex and race. Puerto Ricans constitute 74.2% of "White" race classification.

TABLE 3. *Number of Cases of Lead Poisoning in Persons under 21 Years of Age per 100,000 Population with Distribution According to Race*[*]

Race	1954	1955	1956	1957	1958	1959	1960	1961	1962	1963
White[**]	1.5	3.3	2.2	2.3	3.3	5.1	4.0	4.3	4.5	8.1
Non-white	6.4	8.0	11.7	8.5	10.8	15.4	14.7	21.3	23.4	36.3
Total	2.4	4.2	3.9	3.4	4.7	7.0	5.9	7.3	7.0	13.1

* 1960 Population used as standard.
** White race includes Puerto Ricans.

Housing facilities, socio-economic standards and educational attainment are all poor in the districts where the incidence of lead poisoning is highest. The ages of affected children in these areas are outlined in Figure 5. Physicians in these areas of high incidence have developed great skill in diagnosing cases, particularly in the asymptomatic stage.

We previously indicated that identification of lead poisoning is to a large degree dependent on how hard one looks for it. Our motto is "look and ye shall see." Because of our intensive professional educational program, we are now finding cases of lead poisoning in areas of the City which were formerly considered to be lead free.[4]

Lead paint poisoning is inextricably linked with old, dilapidated housing. No cases are being reported among children living in newly constructed housing projects even in the districts of highest incidence. A house of old vintage in a slum area is an excellent clue, and blood lead determination in the children under five confirms many asymptomatic and previously unrecognized cases of lead poisoning.

Seasonal Incidence

Lead poisoning and lead encephalopathy is encountered more frequently in the summer months, but it is not strictly a summer time disease. Distribution of cases by month are outlined in Table 6. The higher the physician's awareness, the more cases he will identify even during the winter. In 1963, 42 cases of confirmed lead poisoning were reported during December; this figure increased to 68 in December, 1964. Again, the more intensive the search, the greater the yield.

There certainly is a marked seasonal variation in reported cases of lead poisoning with its peak during the summer. Forty-five per cent of the total cases reported between 1954 and 1963 occurred between June and September. The exact cause for this seasonal variation is unknown. Reasons postulated include: (1) a metabolic disturbance whereby there is increased lead absorption because of the actinic rays of the summer sun; and (2) a greater opportunity to ingest lead containing paint used for exterior surfaces.

The seasonal incidence of deaths due to lead poisoning is striking as seen in Table 7. There has not been a single death in November, December, or January since 1951. Of the 121 deaths from lead poisoning since 1954, over 61 per cent occurred between June and September. This may indicate a greater risk and severity of lead in-

TABLE 4. *Deaths in Children under Twenty Years Due to Lead Poisoning in New York City From 1954 to 1963*

Race and Sex	Age						Total
	Under 2	2	3	4	5	6 and Over	
White							
Male	—	3	2	—	—	—	5
Female	3	6	1	—	—	—	10
Non-white							
Male	6	17	4	—	—	—	27
Female	8	15	6	—	—	—	29
Puerto Rican							
Male	5	14	4	—	2	—	25
Female	1	14	7	2	1	—	25
Total	23	69	24	2	3	—	121

TABLE 5. *Number of Cases of Lead Poisoning Reported by Health Districts during January, February, and March, 1963, in New York City*

	City & District															
	New York City		Bedford		Brownsville		Bushwick		Ft. Greene		Red-Hook Gowanus		Rest of city	
(In Yrs.)	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Less than 3	90	59.6	11	61.1	11	73.3	6	46.1	9	64.3	5	55.6	48	58.5
3–4	40	26.5	5	27.8	1	6.7	2	15.4	4	28.6	3	33.3	25	30.5
5–9	21	13.9	2	11.1	3	20.0	5	38.5	1	7.1	1	11.1	9	11.0
Total	151	100.0	18	100.0	15	100.0	13	100.0	14	100.0	9	100.00	82	100.0
Per Cent	100.0		11.9		9.9		8.6		9.3		6.0		54.3	

toxication during the summer. If stored and ingested lead is, indeed, more readily absorbed and distributed to the tissues during the summer because of greater ultraviolet ray exposure, the nerve cell, being highly vulnerable, is more susceptible to the ravages of lead, and, hence, the higher frequency of encephalopathy during the summer.

Our epidemiologic studies show that lead encephalopathy is much more frequent during the summer, but asymptomatic lead poisoning is a year-round disease. The same etiologic ingredients which cause lead poisoning prevail throughout the year, so it is exceedingly important to think of lead poisoning year-round and to detect cases in their incipiency, prior to the onset of the summer. This will minimize the possibility of lead encephalopathy with its resultant brain damage and possible death.

Sources of Lead

Lead enters the body by ingestion or inhalation. Sources include food, water, contaminated air, glazed dishware, painted toys and cribs, and discarded lead battery casings. I have reviewed the lead source in each case reported in this study, however, and greatest sources by far are painted wall plaster and paint peelings from ceilings and window sills.

Sources of lead in this study were: painted indoor surfaces—63 per cent; painted surfaces in the halls of multiple unit dwellings—4 per cent; undetermined and nonspecific—30 per cent. In 91 per cent of the cases, a definite history of pica was obtained, and evidence of broken painted surfaces and of paint peelings were evident in 85 per cent of the homes surveyed. The bedroom was the area of greatest potential exposure, fol-

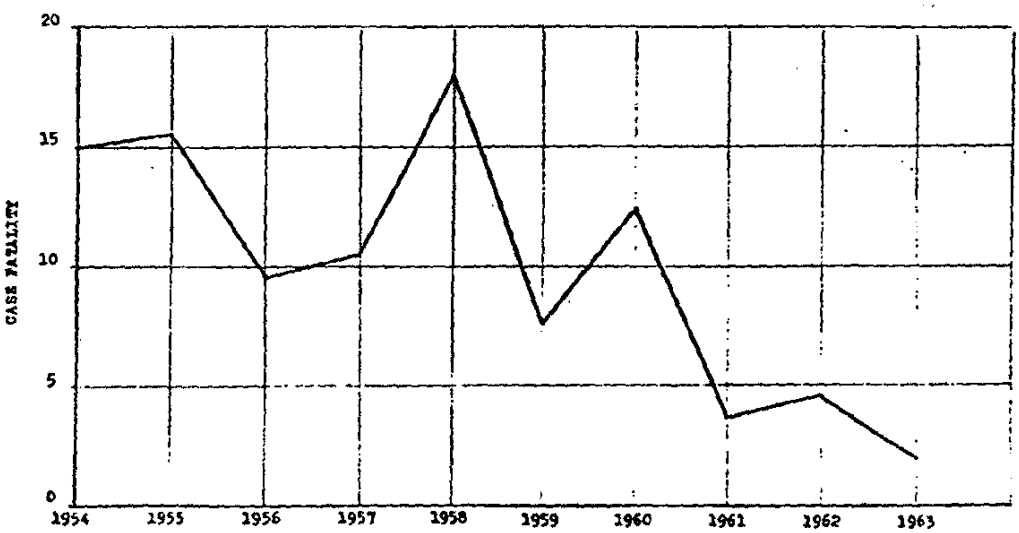


FIG. 3. Lead poisoning—case fatality—deaths per 100 cases—New York City: 1954–1963.

TABLE 6. *Distribution by Month of Cases of Lead Poisoning in New York City, 1954–1963*

Month	Total 1954–1963	1954	1955	1956	1957	1958	1959	1960	1961	1962	1963
January	71	—	—	7	7	4	9	10	10	13	11
February	50	—	6	6	3	—	3	9	12	2	9
March	95	1	12	5	3	2	6	15	13	19	19
April	87	4	5	7	5	2	12	11	6	18	17
May	106	4	5	5	8	3	14	15	11	17	24
June	132	1	12	4	5	10	19	17	18	17	29
July	179	3	10	15	12	15	21	14	28	19	42
August	163	11	4	13	11	9	16	15	18	24	42
September	216	6	32	14	10	30	29	13	19	29	34
October	172	13	15	13	11	20	16	5	18	11	50
November	112	14	7	4	6	11	20	10	11	10	19
December	146	23	7	6	4	10	6	12	17	19	42
Total	1,529	80	115	99	85	116	171	146	181	198	338

lowed by the kitchen, then the bathroom, and finally the living room.

Holes in walls were noted in 32 per cent of houses with highest frequency again in the bedroom, followed by the kitchen, living room and bathroom. Broken walls were observed in 20 per cent of the households with the bedroom still heading the list, and the kitchen and bathroom close behind. Chewed window ledges were observed in 11 per cent of houses surveyed with most again noted in the bedroom. No potential sources of lead were found in 17 per cent of the cases.

The New York City Health Code prohibits the use of lead paint in excess of 1 per cent. When the Department finds paint in dwellings which contain more than 1 per cent metabolic lead, based on the non-volatile content of the paint, it may order removal of the paint and the refinishing of the apartment, room, or part of a room with a suitable finish which is not in violation of the Health Code regulations.

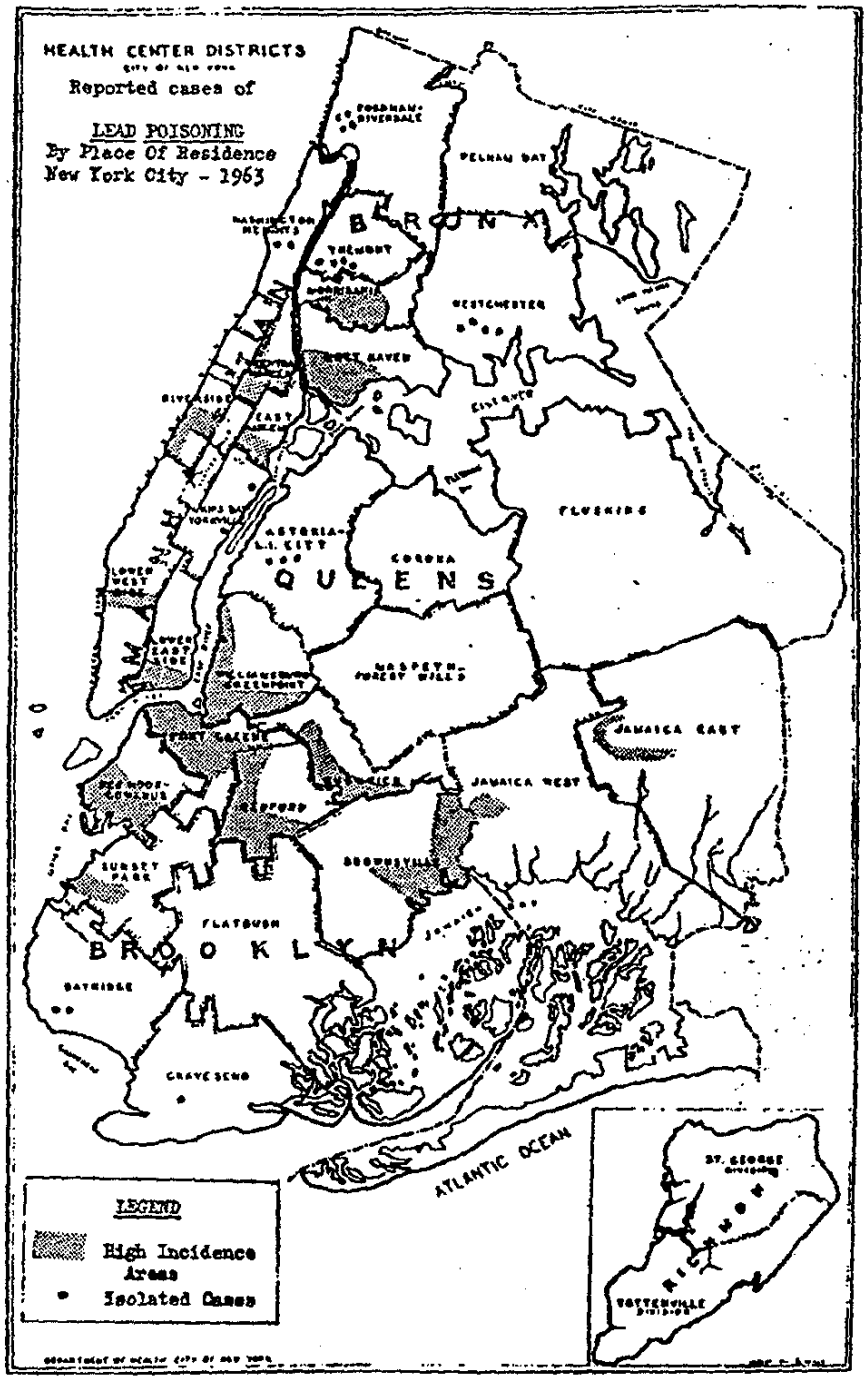
As soon as a real or possible case of lead poisoning is reported to the Department, a Public Health Sanitarian inspects the patient's dwelling looking for peeling paint, broken plaster, or lead water pipes and utensils. He notifies the landlord of any violations and also informs him of any other problems, such as plumbing defects or rodent conditions. He questions the mother about pica in any of her children under seven. He takes paint peelings and scrapings from any lead water pipes to the laboratory for analysis.

The Sanitarian leaves specimen bottles so that the mother can collect urine samples of children from one to seven years of age and makes an appointment to pick these up and take them to the laboratory. He also undertakes the job of health education. He warns the mother about the dangers of eating paint and leaves literature explaining this hazard.

Landlords are required to comply with the Health Code. The Department insists that loose paint be scraped off and the walls and ceilings

TABLE 7. *Monthly Distribution of Deaths Due to Lead Poisoning in New York City, 1954–1963*

Month	Total 1954–1963	1954	1955	1956	1957	1958	1959	1960	1961	1962	1963
January	2	0	0	0	0	0	1	1	0	0	0
February	2	0	1	0	0	0	0	0	0	1	0
March	5	0	1	0	1	0	0	0	1	1	1
April	3	0	1	0	0	0	0	1	0	1	0
May	9	0	1	1	2	0	1	2	0	1	1
June	14	4	1	1	2	2	1	0	0	2	1
July	22	0	2	1	3	6	2	3	3	1	1
August	16	3	1	3	0	1	1	4	1	2	0
September	22	1	6	1	1	3	4	3	1	0	2
October	8	0	2	1	0	2	2	0	0	0	1
November	4	0	0	0	0	4	0	0	0	0	0
December	14	4	2	1	0	3	0	4	0	0	0
Total	121	12	18	9	9	21	12	18	6	9	7



be painted smoothly with lead free paint. Mothers are advised to cover window sills with plastic. Premises are re-inspected to ensure that appropriate repairs are made.

Routine surveys are also carried out in the high lead areas. Sanitarians go from dwelling to dwelling filling out questionnaires about the sizes of families, history of pica, and other pertinent data.

Considerable health education on prevention and control of lead poisoning is also provided during these visits.

We are pleased to find that in over 95 per cent of cases of Code violations, the landlord complies within two weeks to three months from the time of notification. We feel that this action has greatly reduced the number of repeat lead intoxications in children in the past two years.

Family Characteristics

Lead poisoning occurs to a high degree in families of low socio-economic and low educational level, and in those with a high degree of unemployment. We also found a high incidence of home disorganization. There was no father present in over 50 per cent of these families and 26 per cent of the parents were separated. These percentages are higher than those which prevail in the same districts among the general population. In 17 per cent of the households, other environmental problems were also found, and 22 per cent of the families had additional medical problems. Over 80 per cent of the children were judged to be active and curious and 15 per cent mischievous—not dissimilar to the general child population distribution in this age group.

Supervision in the home of the affected child was judged to be inadequate by the visiting public health nurse in about 50 per cent of the cases. Many children were under no adult supervision and a considerable number were under the supervision of grandparents, uncles, aunts or older siblings. Over 90 per cent of the families were cooperative and provided information freely to the visiting public health nurse or sanitarian. Most important to us, however, was the fact that in also 90 per cent of the cases of lead poisoning, the family knew that the child was ingesting paint, but did not know that this practice was hazardous.

In many families, multiple cases were found as a result of our routine search for siblings and performing on them blood lead determinations. This has proven a valuable case-finding device with a very high yield.

Signs and Symptoms of Lead Poisoning

A recently reported random sample of 300 confirmed cases showed that 76 per cent had no presenting complaints. On inquiry, however, 48 per cent had anorexia and 9 per cent had vomiting. Twenty-four per cent had roentgenologic changes in the long bones, but only 2 per cent had x-ray findings in the abdomen. Twelve per cent had encephalopathy. The blood lead content ranged from 0.06 mg. per 100 ml. to 0.34 mg. per 100 ml. The average was 0.08 mg. per 100 ml. of blood.

The child with the highest blood level in this group was a two-year-old Negro female with a history of pica of undetermined duration. The only presenting complaint was anorexia; she was otherwise entirely asymptomatic. Her hemoglobin on admission to the hospital was 7.5 grams and the urinary coproporphyrin was 1+. X-rays of the long bones showed condensations at the epiphyses. The child was subsequently treated with Calcium Versenate and made a complete recovery.

Blood Lead Determinations

The blood lead determination is the most reliable procedure for identifying lead intoxication, particularly, in asymptomatic cases. We consider 0.06 mg./100 ml. whole blood as abnormal and requiring further investigation. A blood lead determination should be done when there is the slightest suspicion of lead poisoning.

Blood lead determinations on suspected cases in New York City during 1963 yielded a 37 per cent positive result—a concentration of 0.06 mg./100 ml. of blood or higher. A urinary coproporphyrin, however, gave a yield of only 5.5 per cent of coproporphyrin values of 2+ or higher. We, therefore, believe that the blood lead determination test is the only reliable indicator of lead intoxication.

Though our current accepted limit is 0.06, as the upper limit of normal, we have had a number of patients with lower concentrations, 0.05 mg./100 ml. or lower, who did have severe clinical plumbism. Recently, Moncrieff[4] re-investigated the value of blood lead estimation, and based on his studies, he suggested that 0.03 or 0.04 should be taken as the upper limit of normal.

The number of diagnosed lead poisonings in New York City has increased markedly with the routine use of blood lead determinations in cases of pica and their siblings and in all other suspected cases. In 1950 only one case of lead poisoning was reported in New York City. Obviously, many cases and fatalities were erroneously ascribed to other causes. Now, there is a marked correlation in the number of blood lead determinations done and the number of cases diagnosed.

For a program of case-finding to be successful, provision must be made by Health Departments for blood lead levels to be performed, and to make it freely available to all physicians and hospitals. In New York City, physicians and hospitals are encouraged to send blood specimens to Health Department Laboratories on all suspected cases. If the concentration is 0.06 mg./100 ml. or higher, the physician or hospital is notified by phone on the same day and a written

report is also promptly submitted. The Department of Health Laboratories also train hospital technicians to do blood lead levels within their own laboratories. The number of blood lead estimations done in our laboratories increased from 538 in 1956 to 3,113 in 1964.

Other Diagnostic Tests

The measurement of lead excretion after a standard intramuscular dose of sodium calcium-edetate was recently reported by Whitaker.[6] She reports a significant difference in excretion between normal children and those with definite or probable lead poisoning. Another test for the rapid diagnosis of lead poisoning described by the same author is the Fluorescence Erythrocyte Test. Red fluorescence of erythrocytes occurs in lead poisoning, and this observation of marked fluorescytosis may serve as a valuable aid, according to the author, in very early diagnosis of lead poisoning in children.

Another screening test recently described is a deposition of pigment in the retina.[7] It has been recommended as a reliable sign in the diagnosis of incipient lead poisoning. Stippling around the optic disc may be readily detected on an ophthalmoscopic examination. All these tests require further confirmation.

Increased Physician Awareness

There are no pathognomonic manifestations of lead poisoning in children. Thus, a prime prerequisite for diagnosis is an increased awareness of the diagnosis by physicians and a high index of suspicion—a lead poisoning "consciousness." Any slum area child between one and four years, particularly one who is nonwhite or Puerto Rican, and who is suffering from poor appetite, slight pallor, possible irritability, a sudden change of personality, or who may have headaches and other vague symptoms, should have a blood lead investigation.

Prevention—The Ideal Goal

Prevention of lead poisoning is, of course, the ideal goal. The disease is inextricably linked with poor housing. It is a community problem associated also with geography, culture, deprivation, education and many other influencing factors. Lead poisoning will be entirely wiped out only when old housing is eliminated and when lead free paint is universally employed.

The physician, however, plays a unique role in the control of morbidity and mortality, prevention of recurrent attacks and in the prevention of brain damage. This can and should be done by early diagnosis of the asymptomatic cases and early institution of appropriate treatment.

Prime prerequisites for prevention of morbidity and death are:

1. Increased physician awareness.
2. Increased index of suspicion.
3. Routine inquiry about pica.
4. Blood lead determination on all children with pica and also on their siblings.
5. Free and ample laboratory facilities for blood lead estimations.
6. Appropriate facilities for treatment.
7. Legislative action restricting the use of high lead-containing paints.
8. Repair of the physical environment prior to a child's return from the hospital to prevent re-exposure.
9. A view of lead poisoning as a common disease of childhood, endemic in slum areas, and of year-round prevalence.
10. Health education at all levels, professional and parental, on the harmful effects of pica.
11. Parents who will take exposed children immediately to a physician for examination and possible treatment.

Lead poisoning is preventable and must be eliminated. While the problem is formidable, it is capable of solution. Intensive and systematic research is needed on many phases of this complex and multifaceted problem. We need more information about its etiology, its seasonal distribution, about the treatment and prevention of pica, about productive health education approaches, and about many other areas associated with the prevention of lead poisoning.

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