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EDITORIAL

Lead Poisoning

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Lead poisoning is a common medical problem and has been for centuries [1]. It is known to cause death and mental retardation after severe exposure. But, the sequelae of prolonged low level exposure to lead remain unknown. Its basic treatment has been removal of the patient from the source of exposure—a treatment better suited to an individual than a community. Lead exposure can be divided into three categories: occupational, community and individual.

It is the impression of industrial physicians that lead intoxication is now an infrequent mild disease. But as recently as 1913, Hoffman [2] recorded 164 cases of chronic lead poisoning among 915 workers in five large storage battery factories. And, Aub et al. [3] noted a 13.5 per cent incidence of plumbism among the men and women who glazed and decorated pottery. During the eleven years 1914–1924, 841 painters in the United States registration area died of lead poisoning [2]. They accounted for one half of the total reported mortality from lead poisoning for that time period.

At least a painter or miner was likely to be aware of his exposure to lead. The consumer of lead products may not be as fortunate. In colonial America, for example, the entire population was exposed daily to lead in pewter plates and vessels and inadequately glazed pottery [4,5]. This chronic diet of lead was the cause of the "dry gripes," an ailment which was widespread in the colonies until the 1780's. It appeared in Europe under several names including Colica Pictonum and Poitou Colic.

Distillers of alcohol have apparently escaped occupational exposure to lead but are likely to have succumbed as con-

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sumers. The use of lead to sweeten wine was common until the 18th century. The use of lead in stills became recognized as a hazard to good business if not health. The Massachusetts Bay Colony on September 3, 1723, promulgated a law "For preventing abuses in distilling of rum or other strong liquors with leaden heads or pipes" [6]. It provided for inspectors and fines. The action was prompted by their customers in North Carolina who attributed a high incidence of "dry bellyache" to use of Massachusetts Bay rum. It is of note that the law was passed before the classic studies naming lead as the cause of the Devonshire colic by Sir George Baker and James Hardy (1767–1778). The former investigator demonstrated that lead used to line cider presses and vats appeared in the product [7]. The latter supported his colleague, but also pointed out that the cider was shipped, stored and drunk from cheap lead-glazed earthenware jugs which, he demonstrated, quickly gave up their lead to acid cider [8]. Hardy [9] defended his hypothesis convincingly. "If the 30th part of a grain of iron taken daily for a few weeks can produce the most salutary effects, is it difficult to believe one half of that same quantity of a noxious mineral dissolved in wine taken occasionally for a number of years, should ultimately prove injurious?" The gradual disappearance of lead colic was largely due to cultural, economic and technologic changes in society without significant intervention by the medical profession.

Exposure of the individual to lead has often been caused by an unusual set of circumstances, such as a child swallowing a foreign body [10,11]. It has been lead gone astray from its usual path, such as the lead poisoning that occurred in families which burned old battery cases to heat their homes [12–14]. Often it has been a flash-back to bygone days—the use of inadequately glazed pottery [15–17] or decorative pewter for food containers; the making of home-made wine [18–20] or "moonshine whiskey" [21–23], and the use of products, such as cosmetics [24,25], from countries with less stringent product regulation.

The child, at the turn of the century, lived in a world of lead, as Ruddock remarked. There was lead paint on the walls, woodwork and window sills, porch railings, furniture, play-pens and cribs. Lead chromate was used as a coloring for food [26]. Toys were made of lead and painted with lead paint. Even young infants were unprotected; lead was used for nipple shields. Holt [27] described an eight month old girl with no teeth whose source of lead was found to be a lead acetate ointment, "diachylon compound," used by the mother on her breast for eczema. In Japan, lead salts were used in the manufacture of toilet powders causing lead meningitis in hundreds of infants [28]. Many of these sources could be effectively removed from the market place and the home, but house paint once applied became a source of lead for future decades.

The widespread prevalence of lead paint poisoning in childhood was brought to medical attention by the Australian workers Gibson [29] and Turner [30] in the 1890's. Gibson [31] traced the principal source of lead exposure to house paint which he said turned a home into "a simple lead trap." He postulated that the paint, especially on porches, powdered in the hot Queensland sun and that the children ingested what stuck on their hands or accumulated under their fingernails.

It was about 1920 when clinicians in the United States began linking paint, pica and the possibility of unsuspected lead poisoning. Blackfan [32] in 1917 described four children with lead poisoning. In one instance, a father stated that the patient, age two and a half years, and his brother had recently ruined a set of parlor furniture by eating the paint off it—a striking example of what a patient may have to do to bring our attention to a problem. Blackfan concluded, "I would urge that energetic prophylactic measures be taken with children who habitually eat painted articles, in order to guard against the development of lead poisoning. Since my attention has been directed to lead poisoning, I have found a number of children who nibble the white paint from enameled cribs." Strong [33] confirmed paint as a cause of lead poisoning in a nineteen month old boy.

It was Ruddock [34] in 1924 who first presented a strong case for the association of pica and lead poisoning in children. He also recognized that there were many unsuspected mild cases of lead poisoning in children. By 1926, McKhann [35] stated that lead poisoning was a relatively frequent occurrence in children: "In our cases it has usually been associated with pica . . . which causes ingestion of lead paint."

Reports from hospitals and health departments in the United States have documented the continuing occurrence of lead poisoning due to paint ingestion. More accurately documented than the cases themselves has been the variability of medical interest in this disease. In a Chicago hospital, Tanis [36] attributed the finding of thirteen children with plumbism in 1953 to in-

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creased physician interest. In fact, this represented such an increase in cases that it prompted a request by the city for aid from the Communicable Disease Center because they thought it might be an epidemic of viral meningitis [37]. From 1931 to 1955, 462 children were admitted to Baltimore hospitals with lead ingestion [38]. In New York City, McLaughlin [39] reported 143 cases and thirty-nine deaths from 1950 to 1955. Ingalls et al. [40] reported approximately fifty cases yearly in Philadelphia in the 1955–1960 period. The 338 cases in New York City in 1963 were contrasted by Jacobziner [41] with the single case reported in 1950.

In 1966, Chicago began the first mass screening program using a blood lead test to detect lead poisoning in children living in low income neighborhoods [42]. Over a two year period, 68,800 children, one to six years old were tested; 5.7 per cent had blood lead levels of 50 μg/100 ml or higher, a total of 3,935 cases. In smaller studies elevated blood lead levels had been reported in up to 40 per cent of those tested, but it was noted that in this large scale survey there had been no prior screening of the children by another test or history of pica.

Old paint is a formidable source of lead exposure for children. It is estimated that 450,000 apartment units in New York City are in such a state of disrepair that children living in them will be exposed to the hazard of lead paint poisoning. Approximately 120,000 children are living in these dwellings. The New York City Health Code banned the use of high content lead paint on interior surfaces in 1959. However, most deteriorating housing stock was built before World War II. In January 1970, the New York City Health Department created the Bureau of Lead Poisoning Control [43]. The Bureau's approach has been to seek out and attempt to protect the child who is most likely to suffer from lead poisoning. It is recognized that the long-term solution is to replace the deteriorating housing of the inner city.

When a child is found to have a blood lead level of 60 μg/100 ml, the Health Department notifies the physician submitting a specimen. A nurse and sanitarian visit the home to plan for medical supervision and to determine the source of lead available to the child. If Health Department laboratory analysis finds paint samples with more than 1 per cent lead content, the owner of the building is ordered to correct the condition within five days. If he fails to comply, the work is done by the city's Emergency Repair Program.

When we speak of a case of lead poisoning, "case" has a connotation of symptoms and "poisoning" has a connotation of damage; neither is necessarily true. However, we have been unable to find any other verbal designation which describes the situation more succinctly. New York City's current definition of a "case" is a child who, on a single occasion, has a blood lead level of 60 μg/100 ml or greater. These children, as a rule, do not have symptoms which can be attributed to lead intoxication. We believe this level does indicate that the child has access to lead in his environment and is taking the lead into his system. That this situation is not unusual can be seen in the accompanying tables.

The distribution of blood lead levels (analyzed by Atomic Absorption Spectrophotometry) of children from the lower socioeconomic areas of New York City is presented in Table I. Comparing 1969 and 1970, we see the effect of a change in the population screened from a restrictive program based on a child's history of pica toward a community screening program stressing the character of housing in the neighborhood. In 1970, a greater per-

TABLE I Lead Content of Blood Specimens Drawn from New York City Children—1969–1971

Blood Level (μg/100 ml)	1969*			1970			Total Blood Samples 1/1–9/24/1971			First Blood Samples 1/1–9/24/1971		
	No.	%		No.	%		No.	%		No.	%	
10	157	5.9		5,050	6.0		5,428	6.2		5,310	6.5	
20	552	20.8		26,650	31.6		33,577	38.3		32,910	40.3	
30	738	27.8		28,422	33.7		27,882	31.8		26,941	33.0	
40	556	21.0		13,582	16.1		11,730	13.4		10,623	13.0	
50	319	12.0		5,619	6.6		4,979	5.7		3,953	4.8	
60	156	5.9		2,757	3.3		2,282	2.7		1,137	1.4	
70–100	135	5.2	12.5	2,134	2.5	6.0	1,569	1.8	4.6	702	0.9	2.4
110+	35	1.4		154	0.2		112	0.1		50	0.1	
	2,648	100.0		84,368	100.0		87,559	100.0		81,626	100.0	

* Data available on 2,648 of approximately 10,000 samples.

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TABLE II **Per Cent of Screened New York City Children with Lead Levels in First Blood Sample of 60 μg/100 ml or More, by Age and Race—January 1–September 30, 1971**

Age (yr)	Rate per 100 Children Screened				
	Black	Puerto Rican	Other	Unknown	Total
<1	2	1	1	1	2
1	5	1	2	2	3
2	6	2	2	1	3
3	4	1	2	1	2
4	3	1	0.4	1	1
5	2	1	1	0.4	1
6	1	1	0.4	1	1
7	1	1	0.4	0.4	1
>7	1	1	0	0.4	1
Unknown	0.5	0.1	0	0.1	2

centage of children appeared in the group with blood lead levels in the 20 and 30 μg range, whereas the percentage of children with blood lead levels in the range of 60 μg and above dropped from 12.5 to 6 per cent. In 1970 and 1971, the children screened were comparable according to age and race. There again was some shift in the distribution to lower blood lead levels, but it was not as marked as in 1969–1970.

Comparing the total number of blood specimens obtained in the first nine months of 1971 with the number of first blood specimens obtained from individuals for the same period, the distribution of blood lead levels was similar. As expected, the higher the blood lead level of the first specimen, the more likely was a repeat blood analysis performed. Roughly, tests were repeated in one of ten children with levels of 40 μg and in one of four with levels of 50 μg. In those with levels of 60 μg and above, the total number of tests was double the number of first blood specimens. Over-all, 6.8 per cent of the blood specimens were "repeat samples." In this table a first blood specimen represents the first sample obtained from an individual in 1971. It is the closest we have to true point prevalence of blood lead levels in this age group. The twelve month totals for 1971 may show a slight increase in the percentage of blood specimens with lower levels of lead, as the lead levels of blood specimens drawn during the last three months of the year might be expected to be somewhat lower than the levels in blood drawn in the summer months.

TABLE III **Lead Poisoning Cases (Blood Lead ≥60 μg/100 ml) by Month in New York City—1969–1971**

Month	Year		
	1969	1970	1971
January	71	29	103
February	58	44	119
March	50	85	177
April	59	112	124
May	85	177	146
June	84	325	232
July	77	376	251
August	72	471	234
September	55	383	188*
October	43	251	...
November	33	217	...
December	40	179	...
Total	727	2,649	...

* Provisional.

It has been thought that the predominance of cases among black children reflected a greater chance of a black child being tested. In Table II we see that in each year of age up to and including five years, the black child was indeed more likely to have a blood lead level of 60 μg or greater. This is surprising, since both black and Puerto Rican populations in New York City apparently live in housing of similar disrepair.

Although previous case reports pointed to children in the one to four year age group as being the most affected, it was also true that children at these ages were most likely to be tested. The risk of lead poisoning for each year of age can be compared in Table II. For categories other than black, the variability from year to year is not striking. However, in black children there is a sharp decrease in the incidence with increasing age.

The data in Table II are based on the first blood sample ever taken from an individual. Equal numbers of black and Puerto Rican children were tested. The data on white children are included in the "Other" column. The percentages shown are based only on cases uncovered by a first blood sample. Thus, the data for a child who had been found to have a lead level of 60 μg in his second or third blood sample would not be included in this calculation. Thus this eliminates a potential error which might arise if one group was more likely to be retested than another.

The monthly incidence of lead poisoning reported over the past three years in New York City is presented in Table III. The increase in cases beginning in March 1970 reflects the initiation of a large scale community testing program. Whereas roughly 10,000 blood tests were performed in 1969, over 80,000 were performed in 1970. There was a steady testing program of approximately 10,000 blood samples a month in 1971. Although more cases become apparent in the summer

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months, a significant number of cases can be uncovered by screening throughout the year. Variations from month to month may be influenced by the pattern of screening, age group, neighborhood [43] and season [44].

After April 1971 there were fewer cases each month than in the same month of the preceding year. This suggests that some progress has been made through the program's educational and housing repair efforts. The decrease might best be explained by considering in theory the number of cases found in the first and second year of any screening program. ... example, the annual incidence of a disease condition is 2,500 and its duration three years, its prevalence would be 7,500 cases (Prevalence = Incidence × Duration). Given a screening program that is 100 per cent effective, the yield would be 7,500 cases during the first year and 2,500 new cases the second year. In effect, the first year would be a prevalence rate; the second year would be an incidence rate. A decline in reported cases in the first two years of a program would therefore not necessarily represent a change in the incidence of the disease condition.

Cases which occurred in the first nine months of 1971 are presented according to age and race in Table IV. More cases of lead poisoning occurred in black children than in all other children combined. The unknown column may indicate simply a clerical omission or may represent inability on the part of the observer to decide racial characteristics of the patient. The age pattern for 1970 in New York City was the same except that more cases occurred in the two year old group than in the one year old age group. This age distribution of cases is similar to that found over the past twenty years in major U.S. cities.

When apartments of children with lead poisoning (affected children) have been visited, lead paint and deteriorating surfaces have often been found [45]. It could be, however, that the home environments of these children and their "normal" young neighbors provide equal exposure to lead, but that the affected children have less parental supervision or more pronounced pica. To investigate this question, the Bureau of Lead Poisoning Control undertook a study in which apartments of the affected children were compared with apartments of a control group. The affected group consisted of one, two and three year old children with blood lead levels of 60 μg or greater. The control group was matched by age and had blood lead levels of 10 or 20 μg.

TABLE IV **Lead Poisoning Cases (Blood Lead Level ≥60 μg/100 ml) in New York City, by Age and Race—January 1–September 30, 1971**

Age (yr)	Black	Puerto Rican	Other	Unknown	Total No.	Total %
<1	17	10	2	3	32	2.0
1	280	78	22	50	430	27.3
2	229	91	16	42	378	24.0
3	150	59	11	33	253	16.1
4	111	37	3	21	172	10.9
5	79	40	6	24	149	9.5
6	43	28	1	17	89	5.7
7	22	12	1	4	39	2.5
>7	12	9	0	3	24	1.5
Unknown	5	1	0	2	8	0.5
	948	365	62	199	1,574	100.0

The results of apartment inspection are given in Table V. In only four of 135 cases did the sanitarian find an apartment with intact interior surfaces indicating "no cause for action." In contrast, sixty-four of 233 (27.5 per cent) apartments of the control group were in good condition. If peeling paint was found, the paint samples contained illegal amounts of lead about 80 per cent of the time in the apartments of the affected children and 50 per cent of the time in the apartments of the control group. Over-all, a potential source of lead within the apartment was uncovered in 76.3 per cent of the investigations of affected children, but in only 37.7 per cent of the investigations in the control group.

Until recently our knowledge of lead poisoning was based primarily on retrospective observations of symptomatic cases. This left us in much the same situation as if we knew diabetes only through observations of patients seen in hospitals. The increasing availability of mass screening technology which can measure the lead exposure of large segments of populations is creating a new opportunity to delineate the natural history of lead poisoning.

TABLE V **Apartment Inspection for Lead Exposure of Children with Lead Poisoning and a Control Group—New York City 1971**

Result of Inspection	Children with Lead Poisoning No.	Children with Lead Poisoning %	Control Group No.	Control Group %
No cause for action	4	3.0	64	27.5
Samples taken, negative	28	20.7	81	34.8
Samples taken, positive	103	76.3	88	37.7
Total	135	100.0	233	100.0

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SUMMARY

Lead poisoning is a common disease. Its reported incidence varies widely according to physician interest. A mass screening program in New York City has uncovered over 4,000 children with blood lead levels of 60 μg/100 ml or higher. Black children were at greatest risk. Analysis of homes of affected children and comparable controls showed significantly more lead paint available to the child who became a "case." New screening technology will enable us to better delineate the natural history of lead exposure and its toxicity.

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