Effect of a Screening Program on \linespread{1.3} Changing Patterns of Lead Poisoning

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A biphase program of screening and treating high-risk children for lead poisoning resulted in a 30% fall in mean lead values in the target areas over a 5-year period. The mean and median for subjects under 6 years was 4–10 \( \mu \text{g}/100 \text{ ml} \) higher than for those over 6. Median for a high incidence area was 42 \( \mu \text{g}/100 \text{ ml} \) in 1967 and 30.0 in 1971; for a low incidence area, 33 and 20 \( \mu \text{g}/100 \text{ ml} \) in the equivalent years.

Ingestion of lead paint was observed or demonstrated by x-ray in 90% of 2200 patients treated in the Lead Clinic. Gross neurologic sequelae were limited to two cases of mild, persistent ataxia. Impaired intellectual performance was observed subsequently in several asymptomatic patients with initial blood lead levels (PbB) \( \geq \) 100 \( \mu \text{g}/100 \text{ ml} \).

A concerted effort in Chicago to screen children at risk for lead poisoning and to treat those with elevated blood lead resulted in marked diminution of incidence over a 5-year period in which over 200,000 were tested. All subjects with elevated PbB (blood lead) were referred to the Lead Clinic for evaluation, where treatment was given on an ambulatory basis unless hospitalization was indicated (1).

The extent of elevated PbB, as revealed by the screening program, was far greater than anticipated (Table 1). In 1967, the initial year, PbB of 50 \( \mu \text{g}/100 \text{ ml} \) or more was present in 8.5% of children tested; in 1968 the number was reduced to 3.8%, and remained at 2% in each of the subsequent 3 years. In all there were 6800 PbB results over 49 \( \mu \text{g}/100 \text{ ml} \) and three to four times as many between 40 and 49 \( \mu \text{g}/100 \text{ ml} \).

Of the 6800 subjects with PbB of at least 50 \( \mu \text{g}/100 \text{ ml} \), there were 77.5% in the 50–
of the patients. In another 10%, the source of lead was established by disclosure of paint particles in the abdomen on x-ray. Occasionally mothers denied pica for paint and plaster because of embarrassment or guilt. Several mothers confessed to giving the child an enema before coming to the clinic in hope the x-ray would prove negative. An x-ray of the abdomen was taken only once unless elevated blood lead recurred. If it had been possible to repeat x-rays of the abdomen at each clinic visit, evidence of paint ingestion might have been observed in all patients.

Although the screening was directed at a well-child population, 465 of the patients had PbB levels between 80 and 275 μg/100 ml.

This incidence, if the disease had been polio, would have been designated an epidemic and a national emergency would have been declared; however, since lead poisoning is a disease of the poor with deep roots in the politically delicate area of real estate, no alarm was sounded. Housing has never been the subject of a conference such as this.

Dwellings in the target areas date from the late 1800's to 1930. Interior walls are generally of plaster, now crumbling, to which lead base paint had been applied. In other instances, wallpaper was used to cover the paint, or paint was applied over the wallpaper as well as on woodwork. Chicago homes have back porches and back stairs painted, traditionally, battleship gray or dark green. The lead concentration of paint flakes from these surfaces may be over 30%.

This review of the effectiveness of the combined program is limited to the peak months of the summer, when lead poisoning is at its height in number of reported cases, of dangerously high PbB levels, and of patients with symptomatic disease. Three areas in diverse sections of the city were selected for comparison. The first two, with a largely black population, were known as high incidence areas in both mortality and morbidity for some years. The third is a lower incidence area primarily Spanish in composition. Children who had reached their sixth birthday were studied separately because pica is uncommon in the school child and PbB tends to be lower.

Since mean values were so high in the summer of 1967, the results of the following summer were also studied to determine what impact the previous year's effort would have on the prevalence of the disease. Both were then compared to 1971, the fifth year of the combined program.

Area E is one of the more notorious districts (Table 3). In the first summer, PbB values ranged from a low of 12 μg/100 ml, which is less than the mean obtained for rural residents, to a high of 150 μg/100 ml (2). Mean and median values were 43.9 and 42 μg/100 ml, respectively, with 55% of test results 40 μg/100 ml and over. By the following summer, the mean was lower by 12 μg but in 1971 it was still 80.7 μg/100 ml. However, the percentage of children showing PbB values over 40μg/100 ml continued to fall, eventually reaching 16%, indicating that even in peak summer the mode had shifted by about 10 μg/100 ml in Area E.

In the older children, the initial range is from 13 μg/100 ml, a low identified with children not exposed to lead, to a high of 63 μg/100 ml. The same rate of diminishing lead values appears here as in the younger children. Presumably the child who was 6 years old in 1971 was detected with incipient lead poisoning at age 3 and prevented from entering the morbid statistics of subsequent years.

Area L, another high incidence district (Table 4) which has suffered increasing economic distress in the past 25 years, reveals a similar trend. Over the 5-year period,
Table 3. Urban lead poisoning in peak summer months; high incidence area E; ethnic predominance black.

| Year | Number tested | Range, µg/100 ml | Mean, µg/100 ml | Median, µg/100 ml | % of total with PbB ≥40 µg/100 ml |
|------|---------------|------------------|-----------------|------------------|----------------------------------|
| <6 Years |     |               |                 |                  |                                  |
| 1967 |   95          | 12-150           | 43.9            | 42.0             | 55.0                             |
| 1968 | 210          | 10-78            | 31.3            | 32.0             | 20.0                             |
| 1971 | 250          | 10-160           | 30.7            | 30.0             | 16.0                             |
| >6 Years |     |               |                 |                  |                                  |
| 1967 | 30           | 13-59            | 33.3            | 34.5             | 20.0                             |
| 1968 | 103          | 12-59            | 25.9            | 26.0             | 9.6                              |
| 1971 | 58           | 10-47            | 24.0            | 24.0             | 8.6                              |

The mean in summer fell from 40.7 to 27.9 µg/100 ml. In 1971, only 10% of young children at risk have PbB levels above 40 µg/100 ml, in contrast to 42.0% 5 years previously. Those over 6 years show a 9 µg/100 ml drop in the mean, and in this group, none appeared in the 40's.

Lead poisoning is not as great a problem in Area D (Table 5), where family relationships are a little more stable and fewer mothers are employed. The mean initially is 33.7 µg/100 ml but after 5 years is down to 23.3 µg/100 ml, exhibiting roughly the same decrease of 30% as the other two districts. Similar improvement occurred in children over 6, whose final mean of 20.8

Table 4. Urban lead poisoning in peak summer months; high incidence area L; ethnic predominance black.

| Year | Number tested | Range, µg/100 ml | Mean, µg/100 ml | Median, µg/100 ml | % of total with PbB ≥40 µg/100 ml |
|------|---------------|------------------|-----------------|------------------|----------------------------------|
| <6 Years |     |               |                 |                  |                                  |
| 1967 | 100          | 19-200           | 40.7            | 36.0             | 42.0                             |
| 1968 | 100          | 10-67            | 35.8            | 34.0             | 29.0                             |
| 1971 | 100          | 14-80            | 27.9            | 28.0             | 10.0                             |
| >6 Years |     |               |                 |                  |                                  |
| 1967 | 30           | 22-53            | 34.2            | 31.5             | 13.3                             |
| 1968 | 30           | 18-41            | 28.1            | 28.5             | 10.0                             |
| 1971 | 30           | 14-38            | 24.8            | 24.0             | 0                                |

Table 5. Urban lead poisoning in peak summer months; moderately high incidence area D; ethnic predominance Spanish.

| Year | Number tested | Range, µg/100 ml | Mean, µg/100 ml | Median, µg/100 ml | % of total with PbB ≥40 µg/100 ml |
|------|---------------|------------------|-----------------|------------------|----------------------------------|
| <6 Years |     |               |                 |                  |                                  |
| 1967 | 100          | 12-77            | 33.7            | 33.0             | 21.0                             |
| 1968 | 105          | 10-69            | 27.2            | 25.0             | 12.0                             |
| 1971 | 283          | 10-87            | 23.3            | 20.0             | 5.3                              |
| >6 Years |     |               |                 |                  |                                  |
| 1967 | 30           | 17-48            | 25.0            | 27.0             | 5.0                              |
| 1968 | 102          | 10-65            | 25.0            | 23.0             | 5.8                              |
| 1971 | 56           | 10-46            | 20.8            | 18.0             | 5.3                              |
The presence of other laboratory tests positive for lead poisoning, it may be impossible to differentiate these nonspecific complaints from those common to pediatric practice. In contrast to the few patients with symptoms at low lead levels are the many without evidence of clinical illness despite values as high as 250 \( \mu g/100 \) ml. Symptoms were absent or sufficiently mild in three-fourths of the 177 children with PbB over 100 \( \mu g/100 \) ml to permit them to be treated on an outpatient basis.

Maturational delay in speech and impaired intellectual performance were manifest in several with blood lead levels in excess of 100 \( \mu g/100 \) ml, even where symptoms were never apparent. Psychological testing of several school children who had subclinical lead poisoning at one to three years indicated impaired abstract reasoning and verbalization of concepts.

Mortality figures for Chicago in the preceding 5 years showed 74 recorded deaths. Between 1967 and 1971 there were 35, of which 25 were reported in the first 2 years and 10 in the next 3 years. Only one death occurred in the 200,000 children tested.

### Conclusion

A unified program for detection and treatment of children with elevated PbB resulted in a 30% decrease in mean PbB in the target areas over a 5-year period. Ingested lead-bearing paint was the sole etiologic agent in 90% of the patients as determined by history or by x-ray of the abdomen.

Screening covered only one-fourth of the children at risk yearly. The child who is not detected through screening, and whose pica does not cease spontaneously, is eventually brought to the emergency room with acute encephalopathy. Should pica stop before symptoms appear, PbB will in time recede to normal without lead poisoning having been detected. However, when he fails at school several years later, it is erroneously concluded that brain damage may result from low lead level toxicity.

Twenty years ago, Bradley found a mean

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**Table 6. Incidence of symptoms in first, second, and fifth year of lead poisoning detection program.**

| Year | No. of patients | No. with symptoms | % with symptoms |
|------|-----------------|--------------------|-----------------|
| 1967 | 582             | 71                 | 12.2            |
| 1968 | 573             | 32                 | 5.6             |
| 1971 | 333             | 14                 | 4.2             |

**Table 7. Symptoms of lead poisoning discovered in a well-child screening program (180 of 2200 patients).**

| Symptoms in order of frequency | PbB range, \( \mu g/100 \) ml |
|--------------------------------|-------------------------------|
| 1. Drowsiness                   | 63–232                        |
| 2. Vomiting                     | 60–208                        |
| 3. Irritability                 | 55–192                        |
| 4. Gastrointestinal             | 67–152                        |
| (stomach ache, constipation, diarrhea) |                     |
| 5. Behavior changes             | 88–208                        |
| (withdrawal, regression, retarded development) |                     |
| 6. Ataxia                       | 61–208                        |
| 7. Convulsions                  | 125–175                       |
| 8. Stupor                       | 119–208                       |
| 9. Peripheral neuropathy        | 140                            |
PbB of 43 μg/100 ml in 333 children at risk in Baltimore, with values ≥50 μg/100 ml in 44.4% (A). A recent study by Fine (5) of 6150 children in 14 smaller Illinois cities revealed a 19% incidence of PbB ≥40 μg/100 ml with 4% having PbB ≥60 μg/100 ml. At the Pediatric Academy sessions in 1972 on “Poison in the Walls” an HEW representative stated there are still 30 to 40 million houses in the country with hazardous lead paint.

Until the overwhelming problem of lead-paint in housing is solved, the contribution of lead to body stores from any other source cannot be correctly gauged. Until then, low level lead toxicity must be considered the endpoint of high level lead toxicity that was not recognized in time.

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