Early asymptomatic lead exposure and development at school age

Sixty-seven 7-year-old children, who had asymptomatic lead exposure between 1 and 3 years of age, were compared in their performance on a series of psychologic tests to 70 children of the same age and socioeconomic background who presumably did not have significant exposure to lead. Exposed children had deficits in global IQ and associative abilities, in visual and fine motor coordination, and in behavior. School failure due to learning and behavior problems was more frequent in the lead exposed than in the control group.

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The institution of large scale lead screening programs has resulted in the identification of many children with subclinical lead exposure. Despite numerous studies1-3 the crucial question of whether damage to the growing central nervous system of children occurs with subclinical lead exposure has not been definitely answered.

Several investigators have shown a relationship between asymptomatic lead exposure and deficits in subsequent development.4-6 The authors found significant differences on psychologic test results at 4 years of age when lead-exposed children were compared to control children with a low probability of exposure.7

In the present study we have attempted to provide further evidence on the nature and incidence of long-range effects of subclinical lead exposure by retesting the same groups of children at 7 years of age and by evaluating their school performance through the third grade.

MATERIALS AND METHODS

This research was conducted within the framework of the Child Development Study at the Medical College of Virginia, a participant in the Collaborative Study on Cerebral Palsy, Mental Retardation, and Other Neurologic Disorders of Infancy and Childhood, directed by the National Institute of Neurological Diseases and Stroke.

The test group consisted of 67 children who had had a history of eating plaster and paint between 1 and 3 years of age. At that time their qualitative urinary coproporphyrin tests were positive and they had elevated blood lead levels (0.04 mg/dl and above) and/or radiologic findings on long bones or abdominal flat plate, but no clinical symptoms.

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<th>Abbreviations used</th>
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<td>WISC: Wechsler intelligence scale for children</td>
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<td>ITPA: Illinois test of psycholinguistics</td>
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<td>IQ: intelligence quotient</td>
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The 70 children in the control group, also drawn from the collaborative project population, had no history of plaster or paint intake and their urinary coproporphyrin tests were negative. Since blood lead levels or radiographic studies could not be done, control children were carefully selected by their living conditions to exclude paint or plaster intake.

Three lead-exposed youngsters and two control subjects were excluded from the original test groups because of central nervous system disease or injury occurring between 4 and 7 years of age. Otherwise each group represented the same samples of children whose psychologic test performance had been compared at 4 years.8
The two groups were comparable in age, sex, race, and several socioeconomic variables (Table I). At 7 years a thorough pediatric neurologic evaluation was done and recorded on standardized forms. The same examination was repeated on 58 lead-exposed and 58 control children at approximately 8 years of age. The examiners had no information on the outcome of the prior evaluation.

All children had a series of psychologic tests at 7 years (average age: 7-0 years for both groups). These tests were routinely administered by experienced collaborative project examiners who were unaware of the child’s prior history at the time of testing. The test battery included: WISC (1949); Bender-Gestalt (Koppitz system); wide range achievement test (1965); Goodenough-Harris draw-a-person; auditory-vocal association subtest of the 1961 Illinois test of psycholinguistics; tactile finger recognition from the Reitan Indiana battery; and a behavior profile of fifteen five point scales. Normal, suspect, and abnormal ratings were made by the examiner according to criteria established in the manuals for the entire collaborative project.

Fifty-four lead-exposed children and 49 control children were attending Richmond public schools. School records on these 103 students were reviewed, summarizing academic performance, behavior, and health as reflected in grades and comments by teachers and school nurses. To preserve uniformity, children outside the Richmond public school system were not included for school follow-up. The reduction in the number of children did not change the compatibility of both groups by their original matching variables.

Since analysis of teeth for lead content has been used as an index of previous lead exposure, we have collected as many deciduous teeth as possible from the original patient sample, obtaining teeth from 29 lead-exposed and 32 control children (43.3% and 45.7%, respectively, of each 7-year group). The teeth were analyzed for lead at the School of Dental Medicine, University of Pennsylvania, Philadelphia, Pennsylvania, using the technique reported by Shapiro and associates. The mean tooth lead content for lead-exposed children was 202.1 μgm/gm (SEM ± 34.6) as compared to 111.6 μgm/gm (SEM ± 13.3) for the control group. Mean tooth lead levels in the 200-500 μgm range are considered to be intermediate between those for inner city children and those with recognized lead poisoning. In our study 44.8% of the lead-exposed children had tooth lead values above 200 μgm/gm compared to only 9.3% of the control children (SEDp = 11.2%, z = 3.17, P < 0.001), thus supporting our contention that the two groups differed in lead exposure.

RESULTS

The outcome of the neurologic examinations at 7 and at 8 years can be seen in Table II. The diagnosis was based on the examiners’ judgment of various combinations of neurologic signs (clumsiness on gross motor tests, difficulties with fine motor performance, hyperactivity, or other behavioral abnormalities during examination), in accordance with prescribed procedures, which were regularly monitored by quality control trials for the entire collaborative project. Compared to control children, more than twice as many lead-exposed youngsters had deficits on neurologic examination at 7 years of age when suspect and abnormal ratings were combined. Approximately one year later additional neurologic abnormalities were evident in the lead-exposed group, resulting in a shift from the normal to the suspect category, whereas the distribution within the control group remained the same.

The results of the psychologic test battery are presented in Table III. The majority of children in both groups had average intelligence; the mean full-scale IQ for lead-
exposed children was 86.6 (SD ± 10.4); for control children, 90.1 (SD ± 7.7). However, the number of youngsters performing in the borderline and mentally defective range was larger in the group of lead-exposed children. The difference between the two groups was significant at the 0.01 level for the WISC full-scale IQ but did not reach minimal significance on either the verbal or performance scales. Significantly more lead-exposed children performed in the suspect or abnormal range on the Bender and on the auditory-vocal association subtest of the ITPA as compared to controls. Some difference was found on the figure drawing and the tactile finger recognition test, but no significant difference was noted on the wide range achievement test.

The behavior of the children during the test situation was coded suspect or abnormal eight times more frequently in lead-exposed than in control children. Examination of the separate ratings showed that lead-exposed children tended to lack self-confidence, were more fearful in the test situation, and more often exhibited a constant need for attention or help. Almost one-third (32%) of the lead-exposed children had a very short attention span and displayed little effort toward reaching a goal; only 14% of control children had this type of behavior. No difference in the level of activity was found between the two groups. Four lead-exposed youngsters had impulsive or uncontrolled behavior during the test session; no control children reacted this way.

By comparing the overall results of the entire test battery for both groups we found that 65.7% of control children performed normally in all areas. A much smaller number of lead-exposed youngsters (43.3%) had no failures in the entire test series (P < 0.01).
The results of the school record review through the third grade on 54 lead-exposed and 49 control children were as follows: The mean full-scale IQ for these students at 7 years of age was 86.8 for lead-exposed youngsters and 88.4 for control children. There was no difference between the groups in the number of days absent from school and in the recorded home-school relationship. Fifteen (27.8%) lead-exposed children had made poor academic progress compared to two (4.1%) controls, and a considerably higher number of lead-exposed children repeated at least one grade (lead exposed 25.9%, controls 6.1%). In addition, the number of students who were not promoted but placed in the consecutive grade or attended special education was larger in the lead exposed than in the control group. Most of the children who failed in school had an average IQ at 7 years. School failure in many of these students was due to behavioral problems interfering with learning. Nineteen lead-exposed children were described as hyperactive, impulsive, and explosive, and as having frequent temper tantrums. They had trouble with peer relations by being aggressive and inconsiderate of others and their property. Five control children had these characteristics. In three lead-exposed children and one control child extreme shyness, withdrawal, and lack of self-confidence had lead to a rating of poor or fair on behavior.

Review of the school health records revealed no difference in general health between the two groups except for the three lead-exposed children receiving anticonvulsant therapy. Eleven lead-exposed children had speech impediments and were receiving speech therapy as compared to four control children.

DISCUSSION AND SUMMARY

The main purpose of this follow-up study of children who had been exposed to lead in early life and their controls was to determine whether differences detected on psychologic testing at 4 years would disappear, change in magnitude, or continue to exist at a later age. Our results on the 7-year evaluation, using different tests suitable for older children, were similar to those at 4 years. Differences in global IQ and associative abilities, visual motor and fine motor coordination, and in behavior were still present.

The most significant finding in 7-year-old children was the difference in the behavior ratings between the two groups. Though the same difference had been noted at 4 years, the functioning of the 4-year-old lead-exposed child in his family environment was adequate; these behavioral problems only became obvious and disturbing in the school setting. Without detailed study of these children at a younger age, the possible cause-and-effect relationship between lead exposure and behavior disturbances would have been less clear.

Differences in test performance of 4-year-old children may have been caused by continuously elevated tissue lead. In another study, utilizing the same two groups of school-aged children, we found no difference in blood lead levels, whereas tooth lead concentrations, an indicator of exposure in the past, were significantly at variance. These findings appear to exclude acute subclinical toxicity as a cause for the demonstrated deficits at 7 years of age and to increase the likelihood that permanent damage has been caused by the early exposure.

The pica habit itself has often been considered a symptom of an underlying psychologic disturbance. This raises the question whether our two groups differed in that respect prior to lead exposure. However, children who ate foreign materials other than paint or plaster were not excluded from the control group. We also found no significant difference between lead-exposed and control children when they were tested at 8 months of age with the Bayley developmental scale, which included ratings describing infant-maternal relationships. We feel reasonably certain, therefore, that the two groups had comparable behavior prior to lead exposure.

The results of the present study are similar to the findings of other investigators who suggested a relationship between asymptomatic lead exposure and deficits in cognitive, perceptual, and behavioral functioning; they further emphasize the necessity for early detection of subclinical lead exposure and for adequate preventive measures.

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