Neurological and Psychomotor Functions in Children with an Increased Lead Burden *

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Fifty-eight children with an increased lead burden underwent comprehensive investigations and were reevaluated 1½ to 3 years later. Of these children 23–27% were noted to have minor neurological dysfunction and various forms of motor impairment during each evaluation. While the initial psychological assessment revealed low average mental abilities in the majority of children, during follow-up examination a significant increase in certain areas of intellectual functioning was observed.

Introduction

Many reports in the literature describe the increased morbidity observed in severe lead poisoning when associated with central nervous system insult (*1–6*). However, limited information is available concerning long-term follow-up of neuromuscular function, psychomotor development, academic achievement, and behavioral issues of children who experienced insiduous lead poisoning in early childhood, and there is also a paucity of data on the effect of long-sustained minor elevations of blood and tissue lead levels in young children (*6*).

Methods

Initially in a house-to-house survey of a run-down neighborhood of Boston, parents of 705 children were interviewed and data were recorded relating to the children's habits of eating nonfood materials, to the likelihood of lead exposure in their immediate environment, and the possible existence of symptoms compatible with acute or chronic lead poisoning. A hair sample was obtained from all children and subsequently examined for its lead content by atomic absorption spectroscopy. When the hair lead concentration was >100 μg/g, when a marked difference was found between the proximal and distal segments of the given hair sample, or when there was a definite history of pica, 3 ml of venous blood was obtained and its lead content was determined. Thus, 98 children were found with an increased lead burden, a condition defined by a blood lead level exceeding 0.5 μg/g and/or when chelation yielded a urinary lead output of more than 500 μg/24 hr. A random sample of 58 children underwent an in-depth study of various neurological and psychomotor functions soon after the diagnosis was made and reevaluated 1½ to 3 years later. The results of the laboratory investigations and part of the clinical studies have been reported previously (*7*).

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Results

Analysis of historical information concentrated on neurological and behavioral symptoms: one third of the study population presented with irritability and clumsiness. Although the nonspecific nature of these complaints casts doubt on the validity of attributing them to an increased lead burden, a comparison of a matched control group of healthy children revealed a significant increase of those symptoms in the study group ($P < 0.01$).

During subsequent neurological examinations 23% of the children in the study exhibited unsteadiness, clumsiness, and limitations in fine motor function. An independent motor evaluation also showed 27% of children with an increased lead burden to display various forms of fine and gross motor impairment which included abnormal gait, poor balance, and muscle weakness. An additional nine children displayed borderline normal motor function. When those children with neurological deficits were compared with a control group of apparent healthy children with normal blood lead levels, matched for age, sex, ethnic background, and residence, a significant increase in neurological findings was noted in the study population ($P < 0.001$).

Psychological assessment of the majority of children in the study found their mental functioning to be in the low average range of normal intelligence, the mean IQ was 86 with a standard deviation of 15. Equivalent results were obtained during further delineation of psychological functioning employing the Seguin Formboard, Visual-Motor Integration Test, and Behavior Ratings. In contrast to the significant difference found between the study population and the control group during neuromuscular examinations, children of a control group living in similar socioeconomic conditions functioned intellectually in the same range as those in the study group and no significant difference was noted in various test situations between these two groups of children.

Children with an increased lead burden underwent chelation therapy. Edetate disodium calcium was administered intramuscularly 50 mg/kg for a 5–8 day period.

Simultaneously, an intensive educational program was initiated whereby television coverage, radio announcements, newspaper articles, posters, and pamphlets focused on the dangers inherent in lead poisoning. Since environmental rehabilitation and removal of the existing health hazards has been recognized to be of paramount importance in the elimination of lead poisoning (*8*), parents of affected children were informed of what they could do to avoid undue exposure of their children to lead-containing materials in the home, and pertinent aspects of plumbism were discussed in group meetings as well as in individual sessions. In addition, public health nurses and housing inspectors visited the homes of the children and advised parents of preventive measures, surveyed the environment for offending lead-containing materials, obtained paint samples for lead determination, and initiated necessary improvements and repairs.

These efforts did not yield the expected results. Only 60% of the parents engaged initially in minor repairs. Although landlords had been notified of the environmental hazards and had been asked to secure living conditions appropriate for human habitation, they often did not comply with the housing authority's regulations. Only in nine instances did owners carry out major repairs, while in another 13 apartments minor improvements were done; in 14 cases no action was taken by the landlord. Parents of 38 children preferred relocation, either immediately after the diagnosis had been made, or later, when the landlord was unwilling to engage in the needed repairs.

One and a half to three years after the initial evaluation, reexamination of the majority of children (84%) took place. Interim histories obtained from the parents of the 49 children returning for reevaluation evidenced that 40 of them were living in an environment where children did not have access to lead-containing paint and plaster crumbs. None of the children presented with symptoms usually observed in acute lead

poisoning. Eight of the 49 children still engaged occasionally in mouthing and eating of nonfood substances. Five children were described as hyperactive, and 13 others were said to have significant behavioral problems. Of 30 children who attended school, 11 were reported to have learning disabilities.

Upon neurological reevaluation, 24% of the study population again displayed dysfunction in various motor activities; balance and coordination appeared to be impaired most often. Hyperreflexia was noted in eight of the children. Interestingly, comparative examinations of distal and proximal upper extremity muscle groups yielded increased involvement of the distal intrinsic muscles in 25%. These results corresponded with those obtained on Visual-Motor Integration testing. However, the children's performance of other cognitive and mental functions differed, in particular when contrasted with the outcome of the children's previous psychological examinations: 35 children exhibited various changes of intellectual functioning and a significant increase of 8 IQ points was observed. Corresponding improvement was also noted on the Seguin Formboard, in verbal productivity, and behavior ratings.

Discussion

Previous studies of neurological and psychological sequelae subsequent to lead poisoning have dealt with children who often exhibited initially central nervous system symptoms (*1, 3, 5, 9*). Thus during reevaluation of their study population, Mellins and Jenkins found most of them to be mentally retarded (*9*). Perlstein reported that 39% of 425 children with lead poisoning in follow-up examinations showed neurological impairment. His patients with lead encephalopathy displayed later serious handicaps in 82%, and half of these children were classified as mentally retarded. Of Perlstein's study population, 22% of the total and 9% of initially asymptomatic children functioned intellectually in the retarded range (*1*). Byers reviewed the outcome of 45 children who previously had been admitted with lead poisoning to Children's Hospital Medical Center, Boston, and found that one third of them were mentally deficient (*5*).

In another paper, Byers and Lord (*10*) emphasized the findings of unsatisfactory school progress, limitations in mental functioning, sensory motor disabilities, and behavioral changes in a group of children who had lead poisoning without lead encephalopathy in the past. A more recent publication (*11*) reported similar results. Lead-exposed children displayed "inferior" performance when compared with those in the control group. The most significant difference between both groups was found in the fine motor and behavior areas. 65% of the control children, but only 35% of the lead exposed youngsters were noted to have normal intelligence (*11*).

Kotok (*12*) compared the development of children with elevated blood lead levels to that of a matched control group. He found deficiencies in fine motor function and language development in both groups, and there was no significant difference in their performances. The author interpreted his results as indicating that the observed developmental lag was not due to lead toxicity but the deficiencies were directly related to inadequacies in the children's environment (*12*). The outcome of Kotok's study corroborates with certain results of our investigations (initial psychological assessments and neuromuscular studies), yet differences are apparent which may be due to the small sample size in Kotok's study, the employment of a screening device (Denver Developmental Screening Test), rather than a comprehensive study of various developmental parameters by independent workers, and differences in the study populations. Smith and co-workers (*2*) could not demonstrate any abnormal findings in their children who previously were diagnosed as cases of "asymptomatic plumbism." Those authors concluded that significant sequelae will be observed only in children who suffered from lead encephalopathy (*2*). The results of this investigation and other studies (*10–12*) do not

agree with Smith's statement. While lead poisoning without encephalopathy in most instances will not cause severe mental retardation, there is evidence that an increased lead burden may result in various forms of neurological and psychomotor handicaps of a more subtle nature. In this investigation, neurological dysfunction and motor impairment were observed during a comprehensive evaluation in 23% and 27%, respectively, which appeared to be significant when compared with an appropriate control group. Also reevaluation of most of the children indicated that 25% of them again exhibited neuromuscular deficits which correspended with independent Visual-Motor Integration testing.

Long-term follow-up of these children is needed in order to delineate whether or not the reported findings in approximately one fourth of the children are lasting or transient in nature.

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