

CHILDHOOD LEAD POISONING IN CHICAGO-AN OVERVIEW

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Until the 1960's, lead poisoning was recognized primarily as an occupational health problem, associated with the recovery of old lead from batteries. The inhalation of lead fumes from burning batteries used for heat in crowded living conditions was recognized as the major cause of lead poisoning in children until the sixties. In the early sixties, many clinicians and public health officials were beginning to recognize the consequences of ingested lead, but the significance of the problem was not appreciated because no data ^{were} ~~was~~ available.

In 1963 in Chicago, lead poisoning was made a reportable disease, just as any communicable disease. By 1967, the first community screening program was under way. Twenty-eight thousand children were screened that year. At that time a blood lead concentration of 50 ug.% was considered the upper limits of normal for children. Approximately 650 of the 28,000 children who were screened had elevated blood leads identified that year, with evidence of symptomatic disease in a great number of these.

Since our lead poisoning program began in 1963, we have screened over 250,000 children. In recent years we have combined our efforts with other official agencies, such as the Chicago Building Departments, as well as unofficial groups such as the Chicago Committee on Urban Opportunity. Coordinated efforts have been aimed at identifying groups of children that should be brought under surveillance, educating community residents about the nature of childhood lead poisoning through the use of neighborhood health

aids, and at deleading residences where children with elevated blood leads live.

Our latest effort has been the adoption of a city ordinance making it unlawful to use paint with a lead content of more than .06 per cent of the dried product on interior surfaces. This level coincides with the recommendations of the Bureau of Community Environmental Management and of the American Academy of Pediatrics. So legally we are making progress at the same time that we are making progress in the control of disease.

Our other area of intense interest in Chicago deals with research aspects of lead ingestion. In several of our clinical units child psychiatrists are attempting to understand the phenomenon of pica. What is the difference between children in the same family, living under similar conditions, who are known to exhibit pica behavior and those who don't? Can some approach be found that will identify factors that lead to pica in some children? Another area of similar interest involves the possible subtle neurological changes that occur following lead ingestion, but before "elevated" blood lead levels are attained. Recent evidence indicates that there are children who do poorly in school but who do not have lead poisoning by our present standards, but rather do poorly because they have ingested lead in sufficient quantity over a period of time.

With some data accumulated after six years of experience, we are beginning to understand the enemy; we are hopeful of overcoming him. We are far from satisfied with the results that we have at the moment, but don't let that discourage anyone. We have cut the death rate dramatically in children. What we have now, we must use and use vigorously.