

CHILDHOOD LEAD POISONING . . .

an eradicable disease

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victims, and those between 1 and 3 years of age comprise 85 to 90 percent of the cases. Boys and girls are affected equally. A high incidence is reported among Negroes and Puerto Ricans, probably because such a large proportion of these ethnic groups live in "lead belts." Lead poisoning occurs the year round, but lead encephalopathy (brain injury caused by lead), a very serious complication, is much more frequent during the summer. Some cases occur in winter when leaded battery casings are burned for fuel and the fumes are inhaled or there is prolonged contact with the ashes.

Nobody knows how many children in the United States are exposed to this health hazard and how many are actually poisoned, for many cases of lead poisoning are never diagnosed. But since the problem is closely related to poor housing conditions, an educated guess may be made on the basis of the number of old deteriorating houses in the United States and the known prevalence rate of lead poisoning among children living in such houses. According to the 1960 Housing Census, 30.6 million of the occupied housing units in the United States were built in or before 1939, when lead paint was still commonly used for interiors. Of these units, 5.6 million were classified as deteriorating and 1.8 million as dilapidated.

Although since the 1940's lead pigment has been replaced by titanium in interior paints, recent surveys in Baltimore, Philadelphia, and Minneapolis revealed that from 40 to over 80 percent of houses in selected slum areas still contain dangerous quantities of flaking lead paint that was applied many years ago. Surveys have indicated that among children living in such dwellings from 10 to 25 percent of those between 1 and 6 years of age have absorbed potentially dangerous quantities of lead, although clinical symptoms of lead poisoning have been present in only 2 to 5 percent of the children.¹ It is thus apparent that childhood lead poisoning is disturbingly prevalent in well delineated areas in many old cities.

Equally disturbing are the mortality and morbidity associated with this disease. Until the advent of chelating agents—therapeutic agents that bind the lead ions and remove them from the body—about two-thirds of the children with lead encephalopathy died. Even with the use of chelating agents, first BAL (British anti-lewisite) and later EDTA (ethylenediaminetetracetate), the fatality rate remained a shocking 30 percent for many years. More recently, with the use of BAL and EDTA in combination and other supportive therapy, the fatality rate has been reduced to less than 5 percent.

But the reduction of the fatality rate in treated

● In the history of modern medicine, few childhood diseases occupy a position as unique as lead poisoning. It is a preventable disease. The etiology, pathogenesis, epidemiology, and symptomatology have all been well defined. Methods for screening, diagnosis, and treatment have long been available. In the past three decades, concerted efforts to conquer infectious diseases have resulted in the development of vaccines for such viral diseases as polio and measles, the discovery of many antibiotics for bacterial and other infections, and the systematic application of these therapeutic agents, but little has been done to eradicate lead poisoning. Yet this man-made disease exists in epidemic proportions in many cities.

While mortality and morbidity associated with such diseases as polio, tuberculosis, meningitis, and pneumonia have declined sharply, lead poisoning has continued to take a high toll among children. Silently, almost unnoticed, it causes the needless death of many children and leaves many more with mental retardation, cerebral palsy, convulsive seizures, blindness, learning defects, behavior disorders, kidney diseases, and other handicaps.

Lead poisoning, or plumbism, is largely an occupational disease in adults. But in children it is almost invariably caused by repeated ingestion of chips and flakes of lead-containing paint and plaster from the walls, windowsills, and woodwork of dilapidated pre-World War II houses. Because of its roots in dilapidated housing in old urban neighborhoods, it has a high incidence only among children living in city slums. In these areas, accessibility to flaking and peeling lead paint and broken plaster, lack of knowledge among parents that ingestion of lead paint is dangerous and even lethal, frequent inadequate parental supervision of young children, and a high incidence of pica (a perverted appetite for nonfood items such as dirt, paper, paint, and plaster) all set the stage for lead poisoning.

Children between 1 and 6 years old are the main



All the ingredients for an incipient case of lead poisoning are present in this picture—old peeling plaster containing lead-based paint within reach of a toddler, who, like most children of her age, is likely to pick up and eat anything.

cases is neither evidence of adequate control of the disease nor a cause for complacency, because many of the survivors are left severely handicapped. A follow-up study of 425 children in Chicago who were treated for lead poisoning revealed that 39 percent had some kind of neurological sequelae. Among the 59 children in this group who before treatment presented symptoms of lead encephalopathy, 82 percent were left with handicaps: 54 percent had recurrent seizures, 38 percent were mentally retarded, 13 percent had cerebral palsy, and 6 percent had optic atrophy. Some had multiple handicaps.²

In Queensland, Australia, extensive epidemiological studies have demonstrated a high incidence of chronic kidney disease among patients who had lead poisoning in childhood 10 to 40 years ago. Half of these patients with kidney damage also suffered from gouty arthritis and many had severe high blood pressure, mental impairment, and various kinds of psychiatric disorders.³

Thus far studies on the effect of increased lead exposure have focused largely on the sequelae of overt poisoning. Little is known and little has been done to determine whether or not damage to the body occurs at a low level of exposure. Although clinical symptoms of lead poisoning often do not appear until

the blood lead level is .06 mg/100 gm. of blood or higher, it is generally agreed that the normal blood lead level should not exceed .04 mg/100 gm. Recently it has been suggested that chronic debilitation and damage, which may not be recognized for many years, may result from an intake of lead far below what has been assumed to be dangerous, and that lead may be harmful to the body even in the absence of clinical symptoms.

Lack of awareness

The foregoing facts about lead poisoning pose a compelling question: How can a disease so prevalent and with such serious results escape attention of both the public at large and the Nation's health workers? In looking for an answer, one must first realize that lead poisoning in children is an illness rooted in social, economic, educational, psychological, cultural, medical, and even political factors. A direct result of a child's environment, it is prevalent only among children whose families are least able to improve their living conditions and who are not generally informed. The well-informed segments of the population are seldom affected.

Moreover, the answer is that many health workers who work among the poor are not aware that lead poisoning in children is still a problem. They apparently think that the mandatory use of lead-free paint for toys, furniture, and interiors of dwellings during the past quarter century has eliminated the problem. Furthermore, childhood lead poisoning is a disease that health workers may not recognize, even though it exists in epidemic proportions, because it has no distinctive clinical features. The symptoms of childhood lead poisoning are nonspecific. Anemia, listlessness, excessive irritability, loss of appetite, abdominal pain, constipation—signs and symptoms that appear before obvious evidence of encephalopathy, such as vomiting and convulsions—can all be misinterpreted as indications of some other illness. Because children who suffer from lead poisoning come from the slums, their anemia may be considered to be the result of inadequate nutrition; their listlessness and excessive irritability, to be the results of a pathological home environment; their abdominal pain and vomiting, to be symptoms of indigestion or gastroenteritis. Even convulsions may be regarded as signs of epilepsy rather than as evidence of lead encephalopathy.

Routine physical examination, blood count, and urinalysis will not provide an unsuspecting health

worker with the correct diagnosis. Unless the worker inquires specifically whether the child has eaten chips of paint or plaster and draws a blood specimen for lead determination, he is likely to miss the diagnosis altogether and treat the child for something else, only to be confronted later with the same patient, who may then exhibit signs of brain injury, which may already be irreversible. In cities with a high incidence of lead poisoning, certain hospitals serving children from known "lead belts" report few or no cases of the disease.

Inadequate housing codes

In addition to poor housing conditions and a general lack of awareness of the problem, other elements contribute to the persistence of lead poisoning. Many cities in which lead poisoning is a public health problem do not have health or housing codes adequate to protect tenants from exposure to lead paint. Even in cities with codes specifically prohibiting lead paint in the interior of dwellings, enforcement of such codes is generally far from satisfactory. The currently available methods of paint removal are expensive and many landlords are not willing to undertake this expense. In cities with large slum areas and insufficient housing for people of low income, city officials sometimes hesitate to enforce the housing codes, reasoning that too rigorous enforcement would compel the landlords to abandon their slum buildings, thereby creating more problems for the city. Even when city officials are interested in enforcing codes, they frequently do not have enough inspectors and sanitarians to carry out the necessary procedures for enforcement—inspection of houses, collection of paint specimens, testing for lead content, and reinspection.

Another reason housing codes are not effective is that enforcement relies primarily on the criminal process, usually in the form of misdemeanor prosecution in the lower criminal courts. Criminal prosecution in such cases is fraught with many procedural and conceptual difficulties. First, summonses are often improperly served, being sent by mail rather than delivered by hand. Even when a landlord has been properly served with a criminal summons, he may fail to appear in court and thus force a postponement. Because a criminal court cannot proceed with a case until the defendant appears in court, housing cases often remain pending for months or even years. When the landlords do appear in court, adjournments and delays are frequent. Furthermore, proving the guilt of the offender beyond a reasonable doubt may be a

long and complicated procedure in a lead poisoning case.⁴

When a landlord is found guilty, there is still the so-called "conceptual hurdle"—the reluctance of criminal courts to recognize a housing violation as a true "crime." A distinction is often made between the so-called "true crime" such as murder, assault, and robbery, and the "social welfare offenses" or crimes of omission, which consist of failure to meet health and safety standards. Penalties imposed for the latter offenses are generally minimal and inconsequential. In New York City, for example, while the city's statutes allow the imposition of fines ranging up to \$1,000 per violation and provide for jail sentences of up to 1 year for repeated offenders against the housing code, in practice, jail sentences have practically never been imposed. In 1965 the average fine per case was less than \$14; the average fine per violation was about 50 cents. Of the cases that did draw fines, many involved violations that had been uncorrected for years. Such inconsequential penalties convince many landlords that it is cheaper to pay the fine than to do the repair.⁴

Failure to get rid of lead paint in a house where a child is known to have developed lead poisoning usually means that a treated child returns to the same hazardous environment to be exposed to another episode of poisoning. The recurrence rate is high in lead poisoning. Among survivors of acute lead encephalopathy who are reexposed to an environment that contains lead paint, the incidence of severe permanent brain damage is almost 100 percent. *Thus early casefinding and treatment programs are virtually meaningless when treated children are returned to their old environment.* Even when a lead-free home is found for a treated child, this merely solves the problem for that particular child; it presents no solution to the problem of lead poisoning in general. If the lead paint in the house is not removed, the lethal heritage will soon pass on to other families with children, and lead poisoning among children multiplies.

Steps to eradication

While the obstacles to the eradication of lead poisoning are tremendous, they are not insurmountable. Slum clearance combined with provision of adequate housing for the poor is the most effective means of eliminating lead poisoning. But even before this measure is undertaken on the scale required, lead poisoning may be reduced through education, early detection, treatment, and follow-up programs that

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include removal of lead from houses wherever it is found. A few cities have demonstrated the value of such methods.

In Chicago, for example, there is a massive screening program for lead poisoning operated through the coordinated effort of local officials, health workers, and the community.

In the summer of 1965 a Citizens Committee to End Lead Poisoning was founded after the discovery of several cases of lead poisoning in the East Garfield Park District. With the help of the American Friends Service Committee, the Chicago Board of Health, and the Medical Committee for Human Rights, an educational and casefinding program was launched. A group of dedicated teenagers carried on a door-to-door campaign in the area, collecting urine specimens from children to be tested for lead poisoning.

In December of the same year the Chicago Board of Health announced plans for a large-scale screening program, and by the next September, 30,000 urine specimens had been tested. In October 1966 the Board of Health began an extensive casefinding program based on blood lead determination by atomic absorption spectroscopy—a screening method far superior to the urine test used earlier. The program has been conducted through the OEO-sponsored Chicago Committee for Urban Opportunity and supported in part by the Children's Bureau and State funds. Community representatives, working out of Urban Progress Centers, go from door to door distributing leaflets and alerting parents to the hazards and symptoms of lead poisoning. They also arrange for all children in the family between 9 months and 5 years of age to have a blood lead test made in the Urban Progress Center by a Board of Health physician. The Lead Poisoning Clinic, headed by Dr. Henrietta K. Sachs, was established for the diagnosis and treatment of children found in the screening program to have elevated blood lead levels.

By October 31, 1969, over 120,000 children had been tested, and over 1,500 children had been treated.

As a result of this intensive program, the incidence of elevated blood lead levels among children from the same areas declined from 8.5 percent in 1967 to 3.8 percent in 1968. Along with a rise in the number of cases of the disease that were detected came a decline in the fatality rate. In 1963, the first year the disease was made reportable, 203 cases of lead poisoning were reported in Chicago and the fatality rate was 2.9 percent. In 1968 the number of reported cases rose to 702 while the fatality rate dropped to 1.3 percent. But because many children were returned to the homes in which they had developed lead poisoning, some recurrence among treated children was reported.

Similar figures have been reported from New York City where physicians from all medical agencies in the city are encouraged to send blood specimens on all suspected cases of lead poisoning to the city health department laboratory for prompt and accurate blood lead analysis. In 1954, New York City reported 80 cases of lead poisoning and a fatality rate of 15 percent; in 1968, the fatality rate was less than 1 percent in the 725 cases reported. The city health department is currently working with the lead poisoning screening programs of the Montefiore Hospital and of the federally supported comprehensive health care projects for children and youth at the Albert Einstein College of Medicine, the Jewish Hospital and Medical Center of Brooklyn, the Brookdale Hospital Center in Brooklyn, and Roosevelt Hospital. Because approximately 75 percent of the children with lead poisoning reported in New York City are from families receiving public assistance, plans are underway to mail with public assistance checks a leaflet on lead poisoning written in both English and Spanish. It has also been proposed that the leaflet be mailed with birth certificates so parents will become aware of the problem before their children reach the age of risk.

In Philadelphia, lead poisoning was made a reportable disease in 1950. But from 1950 to 1960 only 278 cases were reported; these cases involved 53 deaths. In 1961 the Electric Storage Battery Co. began to provide free blood lead determinations for suspected cases. As a result, 109 cases and seven deaths were reported during 1961.

In 1966 the Philadelphia City Council adopted an ordinance amending the Health Code and the Board of Health issued regulations regarding the labeling, application, and removal of lead paint. In reported cases in which lead paint in the interior of dwellings proves to be the source of poisoning, the Board of Health requires all loose paint to be removed wher-

ever found and all intact lead paint accessible to children to be removed down to the bare surface of the wall. Premises may not be repainted until they are approved on reinspection.

Since the ordinance and regulations became effective, approximately 400 properties have been made safe in this way. During the same period—1967 to 1968—there were 176 cases of lead poisoning and two deaths reported.

In 1968, a urine screening program for lead poisoning among children 1 to 3 years of age was initiated in the child health conferences of three district health centers in Philadelphia's high-risk areas.

Baltimore was one of the first cities to recognize lead poisoning in children as a public health problem. For more than 30 years, it has demonstrated an interest in this problem through continuous detection and prevention programs that include enforcement of health and housing codes, epidemiological surveys, and intensive educational campaigns. Aided by the Property Owners' Association, the health department has obtained good cooperation from landlords.

Unlike other cities that have detected increasing numbers of cases, in recent years, as awareness of the disease has increased, Baltimore has reported a steady decline since 1958 when 133 cases and 10 deaths were reported. In 1968 only 13 cases and no deaths were reported. Only one death was reported for each of the years 1964, 1966, and 1967, and none in 1965. These figures probably do not represent the real incidence and fatality rate of lead poisoning in Baltimore, and should be interpreted with caution. It may not be entirely unreasonable, however, to speculate that the continuous existence of the various programs for many years, along with paint removal from many dwellings, has yielded encouraging results.

Voluntary action

Two years ago, the Urban League of Rochester, N.Y., persuaded 22 youths from its Project Uplift Youth Incentives Program to assist the Rochester Committee for Scientific Information (RCSI) in its study of lead poisoning. The young people collected paint samples from slum homes and turned them over to RCSI for analysis. Since then Project Uplift has assigned smaller groups of youths to work with the Rochester Neighborhood Health Center and with doctors from the University of Rochester's Strong Memorial Hospital Department of Pediatrics. Last summer the young people and the Rochester Neigh-

borhood Health Center distributed material explaining the campaign to all families registered with the health center. Teams, of two teenagers each, visited the homes to find out whether the children had eaten paint and to collect paint samples. If a paint sample showed a dangerous content of lead, the teenagers collected urine samples from the young children living in those dwellings for testing for lead poisoning. Subsequently, those children whose urine showed positive results were examined thoroughly. Nearly 7 percent of the children tested thus far have been found to have dangerous levels of lead in their systems.

In New York City the New York Scientists' Committee for Public Information has called the public's attention to lead poisoning by sponsoring meetings both for the population at risk and for health and community workers.

In Minneapolis the University of Minnesota Bio-

A member of Project Uplift, a teenage volunteer organization sponsored by the Urban League of Rochester, N.Y., collects a sample of peeling plaster from an innercity dwelling for analysis to determine whether the paint contains lead.



medical Student Committee for Social Responsibility and the Minnesota Committee for Environmental Information have sponsored a program to determine the prevalence of lead poisoning in one area of the city. Two University of Minnesota research assistants conducted a survey in early 1969 and found that 40 percent of the houses they examined had chipping interior paint with dangerous lead content. On the basis of this finding, a screening program was begun for children in the high-risk areas. In the summer of 1969 the Minneapolis City Council passed an ordinance prohibiting the use of lead paint in dwellings.

Many comprehensive health care projects for children sponsored by the Children's Bureau have set up programs to control lead poisoning. Among these are projects at the Hill Health Center in New Haven, the Children's Hospital of the District of Columbia, and the ones in New York City already mentioned.

The Johns Hopkins University School of Medicine has received a grant from the Children's Bureau for an urgently needed study of tests used in screening children for lead poisoning. The goal is to develop a simple, quick method of determining the amount of lead in human blood for use in large-scale screening programs. At present blood lead determination, in which a physician or skilled technician must puncture the vein to draw enough blood for analysis, is the only reliable test available. The successful use of this test with more than 120,000 children in Chicago is evidence of its feasibility as a large-scale method of screening for lead poisoning. However, scientists are seeking a quicker, but equally reliable, method that will require only the small amount of blood obtained from a finger prick.

The two currently used urine tests are far from satisfactory; the coproporphyrin test is often negative in lead poisoning and positive in other diseases, while the ALA (delta aminolevulinic acid) test is found to correlate poorly with blood lead levels. Moreover, it is difficult to collect urine from children from 1 to 3 years of age, the age group with the highest incidence of lead poisoning. Determination of the level of lead in hair has also been suggested as another screening test. Because the usefulness of this test has been questioned, further evaluation of it is needed.

In 1969 the Lead Industry Association published a booklet entitled "Facts About Lead and Pediatrics," presenting seven steps to the prevention of lead poisoning. The booklet is being distributed to physicians, public health authorities, social workers,

city officials, and others who can help achieve prevention and control of the disease in children.

The recent upsurge of interest in childhood lead poisoning among Federal and local agencies, citizens' groups and government officials, health and community workers, and private and public institutions encourages the hope for an eventual end of this preventable manmade disease. But much more needs to be done.

Steps ahead

The ideal solution to childhood lead poisoning is slum clearance and urban renewal with the provision of adequate housing for families of low incomes. But this goal cannot be achieved quickly. Meanwhile, control and prevention must depend on other means, such as:

1. Public education through all channels and all media of communication to point out the dangers of paint eating, to acquaint the public with the symptoms of lead poisoning, and to urge parents to seek help whenever lead poisoning is suspected, even in the absence of symptoms. Many parents who are aware that their children eat paint do not know that this is dangerous. Among women who themselves eat clay or starch—a common practice in certain cultural groups—a child's paint eating may not receive attention.
2. Education of physicians, nurses, social workers and all other health workers on the prevalence of lead poisoning among children so that they will always have an index of suspicion. Health workers should routinely inquire about pica and paint ingestion in all children 1 to 6 years old, particularly those from high-risk neighborhoods, and should look for lead poisoning even before overt symptoms appear.
3. Mass screening programs in "lead belts" for all children between 1 and 6 years of age, using blood lead determination, the only reliable screening test.
4. Immediate referral of children found to have elevated blood lead levels to a medical center for diagnosis and treatment if necessary; prevention of a treated child's reexposure to lead in the home; and followup and retesting of all treated children who continue to be exposed. The prevention of reexposures means that health workers must work closely with housing authorities to see that lead paint is re-

moved from every dwelling where poisoning has occurred.

5. The establishment of effective health and housing codes pertaining to lead and lead poisoning and the diligent enforcement of these codes. Where codes are not enforced, court action may be necessary. In New York City, for example, where the health department is said rarely to invoke a law allowing it to require landlords to correct lead hazards, a neighborhood health center is seeking court orders for landlords to repair as common law nuisances dwellings where children have suffered lead poisoning. Other measures that have been advocated are withholding rents to make the necessary repairs or fining landlords for each day the violations remain uncorrected.⁵

6. A concerted effort by research institutions to develop a simple, practical, and relatively inexpensive method for paint removal. The expense of currently available methods to make paint inaccessible to children—either by paint removal through burning, scraping, and sanding, or by covering the old paint with plasterboard or fiberglass—is an important deterrent to correction by landlords.

Research is also urgently needed to accomplish the following objectives:

- Development of a simple portable device for lead detection to make it possible systematically to identify houses containing lead paint. At present detection of lead paint in houses involves collection of paint samples and chemical analysis of such samples.

- Evaluation of available screening tests for lead poisoning and development of a reliable, simple, inexpensive method to determine blood lead level.

- Prospective studies of children with elevated blood lead levels who are "asymptomatic," to determine the subtle effects of lead that do not become immediately apparent.

- A uniform reporting system for all screening programs to facilitate the collection of pertinent

data, exchange of information, and comparison of results.

- Improved methods of treatment to reduce not only the number of deaths from lead poisoning, but also the residual effects in survivors.

- Increased knowledge about the causes and cures of pica to reduce the incidence of lead ingestion.

THE ESTIMATED COST of treatment and institutionalization to the age of 60 of a person who incurs severe permanent brain damage from lead poisoning in childhood is about \$222,000. Complete removal of old lead paint from an average rowhouse with 10 windows, two doors, and baseboards would cost \$250 to \$300; replacement of window and door units and baseboards in such a house would cost \$600 to \$1,200.⁶ These figures show only the difference in dollar costs between preventing lead poisoning through paint removal and permitting severe brain damage to occur in children. They do not take into consideration the suffering and heartache of affected families or the loss of useful manpower to the Nation.

Until society recognizes that permitting children to be killed and crippled by lead through paint ingestion is a crime not very different from permitting massacre and maiming of children by the use of lead bullets, this needless manmade disease will continue to victimize children.

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