Lead poisoning in childhood—comprehensive management and prevention

A symposium was held at Happy Hills Hospital in Baltimore on April 24, 1967, to call attention to the need for a cooperative community approach to the social, environmental, and psychological aspects of the problems of children with lead intoxication. Comprehensive care is just as urgent for the asymptomatic child with an increased body burden of lead as it is for the child with manifest acute plumbism. While chelation therapy for the acute toxic episodes of chronic lead poisoning is deservedly emphasized, hospitalization in a chronic disease facility which has a positive program of child and family rehabilitation serves an important role in the total care of the affected child. Experience in Baltimore and other large cities has shown that coordinated and sustained efforts by health departments, pediatricians, medical social workers, and child guidance workers are essential for an effective program for the prevention and treatment of childhood lead intoxication.

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This work was supported in part by United States Public Health Service Grant No. UI00399 from the National Center for Urban and Industrial Health.

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LEAD POISONING in the young child is a chronic disease. It results from the impact upon the urban slum child, in particular, of a variety of causative factors—pica and environmental exposure to lead, cultural and behavioral patterns of parents, and certain aspects of lead metabolism. Current knowledge with respect to each of these factors, although imperfect and limited, is sufficient to provide the basis for an effective approach to the problem.

CAUSATIVE FACTORS

Pica. Centuries ago, pica was the Latin word for magpie, a bird of voracious and indiscriminate appetite. Today the term pica denotes the habitual, purposeful, and compulsive search for and ingestion of such unnatural food substances as clay, plaster, ashes, laundry starch, string, putty, paint chips, paper (especially newspaper), dirt, crayons,

Vol. 73, No. 6, pp. 942-950

cigarette butts, yarn, and matches. Although the list is long and varied, many children and adults with pica tend to be highly selective: Each exhibits a craving for only one (or very few) of the above items. Pica had been known to medicine since the time of Galen. Its distribution is worldwide. Incidence in the general population increases during times of stress. Historically, it seems to be related mainly to the relative availability of a diet adequate both in quantity and quality to the social group as a whole. Women (especially pregnant women) and young children are most vulnerable to pica. Rural groups are affected more often than urban groups.[6, 17*]

Lourie and his associates[10, 17] could find no evidence that any nutritional deficiency was etiologically related to pica among urban children in Washington, D. C. Preliminary studies suggest that pica in large cities of the United States is most prevalent among families—particularly Negroes—recently arrived from the rural South. Lourie postulated that such families respond to the stress of their social and economic deprivation in their traditional rural behavior patterns, one of which is clay-eating among women. After 3 or 4 generations as urban dwellers and with improved social and economic security, rural behavior patterns including pica are largely dropped.

The child. Infants are apparently born with differing innate levels of oral activity. During the first 12 months of life, this takes the form of mouthing—but not ingesting—almost anything the infant can place in his mouth. For reasons unknown, children between 12 and 18 months of age may begin to ingest foreign materials as an extension of normal mouthing activity. As many as 50 per cent of children carefully studied in both middle class and poverty groups habitually and selectively ingest objects other than food. Between 3 and 5 years of age, this behavior tends to disappear. During these years, the child may indoctrinate his younger siblings into the same activity.

*These numbers guide the reader to the use of the Selected Bibliography at the end of the article. They are not the usual "reference numbers" of this JOURNAL.

Interaction of child and parent(s)—the emotional climate. The interaction of child and mother is often a critical determinant of pica activity. As many as 50 per cent of mothers of children with pica may also have pica themselves. A child's high level of oral activity may be reinforced by a mother with similar oral interest. Thus, while a bottle or pacifier is offered the fussy child at first, later the mother may substitute the clay or laundry starch that she habitually ingests. The pattern of relieving the child's anxiety by oral activity may become fixed in this manner and may later continue as an aggressive form of behavior: "If you don't let me go out to play, I'll eat paint."

Anxiety in the child for which oral gratification may serve a relieving function is often a response to an absent or a poorly functioning mother. She may be absent in order to earn a living outside the home, and there may be no father to share the domestic responsibilities. She may be hospitalized by illness or repetitive childbirths. She may be overwhelmed by too many children to care for and thus be deficient in her caring role for the toddler. Indeed, the onset of pica in the toddler often coincides with the arrival of the next infant. Emotional difficulties in the mother often accompany a child with pica. Maternal dependency is the most common pattern observed: Such mothers have a life history of despair, passivity, and inactivity except in crisis. In such a family, children with plumbism may escape early detection and appear for health care only when seizures or coma demand urgent intervention. Another pattern encountered is the mother who is not aware of the child's pica. This may reflect a basic ignorance that ingestion of these materials could be harmful, or she may be absent from the home for much of the day and not realize that the persons who care for her child allow or support his pica activity. In a small number of families, the inadequacy of the maternal role results from the mother's own gross intellectual or psychiatric handicaps. To sum up, the symptom of pica is most likely to occur in children with a high level of mouth activity whose oral

relief of anxiety is reinforced by cultural patterns and to whom the mothering necessary to stop it is unavailable for a variety of reasons. When such a child is exposed to hazardous environmental sources of lead, the likelihood of plumbism is indeed great.[10, 12, 17, 18]

Environment. The vast majority of cases of plumbism in young children in the United States today are recognized in those who live in old, deteriorating urban housing. The interior woodwork, painted wallpaper, and painted plaster of houses built prior to 1940 and still in use may contain layers of lead-pigment paints which have never been removed.[2] A few small chips of such paint may contain 100 mg. or more of lead. (The safe daily intake of lead is < 0.5 mg.) Recent studies in Baltimore reveal that 50 to 70 per cent of old houses in selected slum areas contain dangerous quantities of flaking lead pigments on the painted interior surfaces.[10] The clear relationship between childhood plumbism and old urban housing is shown in Table I.[8] Studies indicate that a comparable situation exists in many large cities of the continental United States. It seems likely that small towns and rural areas may also contain children wth pica who dwell in dilapidated old houses; nevertheless, plumbism is infrequently recognized outside of large cities. The usual interior locations of leaded paints chewed by poisoned children are windowsills and painted plaster and wallpaper. Common exterior sources are door frames, fences, porches, and housewalls.

Table I. Environmental exposure of young children to lead in new and old urban housing*

Location of home	*No. of children studied*	*No. with abnormal urine†*	*No. with plumbism*
Old housing	801	216 (27%)	38 (4.7%)
New housing project	105	3	0

*Adapted from the data of Griggs, R. C., Sunshine, I., Newill, V. A., et al.: Environmental factors in childhood lead poisoning, J. A. M. A. 187:703, 1964, and based upon a prospective home survey of preschool children in Cleveland, Ohio.

†Concentration of both lead and coproporphyrin increased.

There is increasing concern over the problem of environmental pollution of all sorts. Much of the controversy with respect to lead was raised by Patterson, who inferred that the body-lead burden in urban dwellers may be 100 times greater than the burden would be under ideal but primitive living conditions. This difference, he surmised, is due to the continuing accumulation of lead wastes in urban areas from lead alkyls, lead arsenate, food can solder, paints, alloys, piping, glazing, and spent ammunition. Atmospheric lead pollution is greatest in the urban areas, with the chief contribution coming from motor vehicle exhausts.[9] On the basis of balance studies, atmospheric lead pollution, even in urban areas, has not yet reached toxic levels for the general population.[14]

Metabolism of lead. Lead intoxication results from *chronic* increased ingestion of lead. This is so because inorganic lead compounds are poorly absorbed into the body, retained lead is stored largely in bone, and an excessive body-lead burden is only very slowly excreted. It follows that repetitive ingestion (or inhalation) of small amounts of lead is usually far more dangerous than a single massive exposure. The meticulous long-term balance studies of Kehoe[14] in human adult volunteers indicate that the average adult in the United States today ingests about 0.3 mg. Pb daily in food and beverage, and in urban areas currently has a respiratory intake of 0.03 to 0.04 mg. Pb daily. Of this total exposure, approximately 40 to 50 μg Pb is absorbed into the body and promptly excreted by urinary and biliary tracts so that no net retention of lead results. Such "normal" lead exposure is associated with a concentration in blood of 15 to 40 μg Pb per 100 Gm. whole blood in both children and adults. As yet, no untoward effect of this "normal" exposure has been demonstrated. As mean daily lead ingestion increases beyond 0.5 mg. Pb, the entire load cannot be excreted, so that the accumulation of an excessive body-lead burden begins and will increase progressively as long as abnormal

ingestion continues. Comparable balance data for young children are not available; nevertheless, it is blatantly obvious that repetitive ingestion of paint chips containing 50 to 100 mg. Pb per few small chips constitutes truly massive exposure. Studies in human adult volunteers and in dogs indicate that once excessive intake of lead is stopped, it takes at least twice as long to excrete the excessive body burden of lead as it did to accumulate it. For example, if a child with pica ingests lead over a 2 year period (from 1 to 3 years of age), it will require at least 4 years (or until 7 years of age) to excrete the load by normal physiologic mechanisms.[3, 9, 14, 16]

The biosynthesis of heme is exquisitely sensitive to the toxic effects of lead.[11] Increased excretion of the heme precursors, coproporphyrin and δ-aminolevulinic acid, in the urine, is virtually always found prior to the onset of clinical symptoms. The qualitative urinary coproporphyrin test (UCP)[4] is well suited for clinic and emergency room use for the rapid presumptive diagnosis of manifest or incipient acute plumbism. This test can be performed in 5 minutes and should be available in all metropolitan hospitals serving high-risk urban areas. When blood-lead concentration exceeds 80 μg Pb per 100 Gm. whole blood in a toddler actively ingesting lead, the UCP test is strongly positive; but at lesser concentrations of lead in blood, this qualitative test is not sufficiently sensitive or discriminating, so that its usefulness as a screening test for the early detection of the child with asymptomatic increased lead absorption is quite limited. Detection of fluorocytes (erythrocytes fluorescent owing to increased content of protoporphyrin) does not distinguish between plumbism and iron deficiency anemia and requires specially skilled technicians; nevertheless, the rapidity and simplicity of the basic technique make it attractive as a screening technique where a skilled technician can be found.[20] In industry, serial measurement of δ-aminolevulinic acid (ALA) in urine provides one of the best means of monitoring occupational lead exposure.[11] The recent development by Davis[7] of a simplified technique employing commercially available prefilled disposable ion-exchange resin columns for the estimation of ALA in urine holds great promise: At the moment, this is the best mass-screening technique available. With it, children with blood-lead concentrations > 60 μg Pb per 100 Gm. whole blood can apparently be detected with a high degree of accuracy.

Fundamentally, the diagnosis of plumbism depends upon the demonstration of an excessive body burden of lead.[3, 14] The most useful and direct index of this is measurement of blood-lead content. Accurate determinations require specialized techniques and proper collection of samples. At the moment, blood-lead analyses are too difficult and time-consuming to permit their widespread use in mass-screening programs. Byers and Kopito[15] are developing a technique for measuring lead in hair and are currently evaluating its suitability as a screening technique. Urine-lead analyses require quantitative 24 hour collections of urine to yield useful data, so that this measure is most useful in clinical research and in the management of hospitalized cases. The EDTA mobilization test for lead also requires quantitative collection of urine. It appears to have its main use in the study of older children suspected of chronic plumbism.[4]

THE DISEASE

Natural course. Uncurbed, pica tends to persist until 3 to 5 years of age: In the brain-damaged child, the habit may persist much longer.[18] Such protracted abnormal lead ingestion is accompanied clinically by recurrent acute toxic episodes of symptomatic plumbism. For reasons not fully apparent, 80 per cent or more of acute toxic episodes occur during the summer months. Clinical manifestations tend to vary with both the age of the child and magnitude of the abnormal lead ingestion. Encephalopathy is most common in children 15 to 30 months of age, while intoxication without encephalopathy in this age range usually presents as some form of hyperirritable or aggressive be-

havior disturbance. Associated iron deficiency anemia is vitually always present. Careful questioning usually reveals delay or reversal in verbal maturation and loss of recently acquired motor skills. Vomiting and decreased interest in play are especially ominous signs, as they may portend incipient encephalopathy. As the preschool child grows older, acute toxic episodes tend to be less severe. Thus the 2 to 5-year-old child with unrecognized plumbism may present with a convulsive disorder (without features of encephalopathy and not distinguishable from idiopathic epilepsy), chronic impulsive, aggressive hyperkinetic behavior disorder, or mental retardation. Uncommon syndromes include progressive loss of mental function simulating degenerative cerebral diseases, peripheral neuropathy or bouts of acute crampy abdominal pain usually attended by vomiting, constipation and pain and tenderness in trunk and proximal girdle muscles.[3, 4]

Sequelae. Permanent central nervous system injury and late-onset renal insufficiency have been reported to follow the plumbism of early childhood.[1, 3] At least 25 per cent of the survivors of acute encephalopathy sustain severe permanent brain damage.[2] Whether children without overt encephalopathy sustain significant CNS injury as a result of plumbism in early childhood is not clear. Although the usual psychometric tests and performance in school indicate deficiencies in comparison with norms derived from more privileged groups of children, it has not been shown that they differ greatly from other underprivileged children not known to have had plumbism who also reside in deprived urban areas. In a long-term, follow-up study of childhood plumbism in Australia, Henderson found that 94 of 352 patients had died of chronic nephritis, 15 to 40 years after the initial intoxication.[3] Onset of renal insufficiency began during or after adolescence. Similar work in the United States has not revealed any link between childhood plumbism and chronic renal insufficiency. This and other evidence suggests that lead nephropathy may be a sequel limited to very protracted childhood plumbism.

Byers and others have delineated the the nature of the CNS injury which follows early childhood lead poisoning.[1, 12] Injury resulting from lead does not differ from that which follows any diffuse cerebral injury sustained during early childhood (i.e., encephalitis, meningitis, trauma). In its most severe form, acute encephalopathy may result in cortical atrophy, hydrocephalus ex vacuo, severe convulsive disorder, idiocy, and blindness: Such a result is becoming increasingly rare. Subtle neurologic deficits are the more common outcome, such as lack of sensory perception and perseveration despite I.Q. scores of 80 to 100 or better on the Stanford-Binet test. Form and proportion are distorted. The affected child tends to break a drawing down into its components rather than to recognize the design as a whole, integrated unit. Such children also perseverate: For example, if you teach the child that 5×5 is 25 and then ask him what 4×3 is, the child says 25. If, on the other hand, you ask him quite apart from the first question (5×5) what 4×3 is, he may be able to say 12. Once he learns a correct answer, he repeats it even when the question is changed. The unwitting teacher (or mother) may conclude that such a child is insolent, whereupon she will punish him and so reinforce and aggravate the behavioral problems often present in such children. They also have short attention spans and are easily distracted. Although it is difficult to determine how much is due to organic brain damage and how much represents response to environment, many lead-poisoned children develop hostile, aggressive, and destructive behavior patterns which in turn may precipitate exclusion from school and the demand for institutionalization. Such behavior as well as convulsions may abate as puberty approaches, but intellectual deficits persist.

Early recognition–mass screening. A systematic program is essential in each community for early detection of the high-risk toddler, the high-risk mother, and the high-risk dwelling. The program should be implemented throughout the year, but should be most intensive during the spring and sum-

mer months. For early recognition, efforts should be concentrated on children 12 to 18 months of age. In selected slum areas, the estimated incidence of excessive lead ingestion among preschool children is 10 to 25 per cent and of children requiring therapy for plumbism it is 2 to 5 per cent. In such areas, mass screening techniques are clearly needed. Currently, the simplified urinary-ALA test of Davis[7] is the best available: It is economically feasible, but requires the facilities of an analytical laboratory, which is the responsibility of the local health department. The analysis of lead in hair by atomic absorption spectrophotometry[15] and fluorescent erythrocyte[20] techniques warrant further evaluation to determine their suitability for mass screening. Each abnormal test result calls for thorough clinical evaluation of the patient and, at the very least, a confirmatory blood-lead determination.

In the absence of mass-screening programs, early recognition is dependent upon the development of interview and observational techniques designed to identify the child with incipient pica and his dependent, depressed, overwhelmed, or unaware mother. Clinical indications for blood-lead and other laboratory determinations include: (1) pica in the child by history or observation in the clinic waiting area, or evidence of chewing on windowsills, etc., reported by visiting health nurse; (2) symptoms of plumbism as outlined previously; (3) nutritional anemia, especially after 12 months of age; (4) aberrant behavior, especially hyperirritable or aggressive behavior in a toddler; (5) developmental delay, especially in speech development. Maternal indications for blood-lead determination in the child include: (1) working mother unable to account satisfactorily for her toddler's activities during her absence; (2) neglect of toddler due to arrival of newborn infant; (3) depressed, psychotic, or alcoholic mother; (4) history of pica in mother or in previous children of the mother; (5) in short, any evidence of lack of mothering of a toddler residing in a dilapidated pre-World War II house. Since a definitive diagnosis of plumbism can only be confirmed by laboratory tests, these clinical indications call for blood-lead determination and UCP or urine-ALA in asymptomatic children. Suggestive symptoms or the presence of a positive UCP call for complete diagnostic evaluation and hospitalization.

COMPREHENSIVE MANAGEMENT

In years past, mothers of children with plumbism were sternly admonished not to let their children eat lead paint. Only those children with acute lead encephalopathy were admitted to the hospital, and, following a brief course of chelation therapy, they were discharged to the same leaded environment with the same parental admonition. In the light of current knowledge, the inadequacy of this casual approach should be obvious, and its ineffectiveness in preventing severe neurologic sequellae not surprising.

Today every child with asymptomatic increased lead absorption should be hospitalized. A team approach to his problem should include the comprehensive efforts of the local health department, physician, medical social worker, and psychologist. For optimal results, a specific course of action can be outlined during an early conference by these various health personnel.

Local health department. It should inspect the housing, see that all hazardous paint is removed, and back this up with penalties on the owner who does not comply with local health ordinances. In Baltimore, paint in housing interiors of more than 1 per cent lead is illegal. The Baltimore City Health Department provides free laboratory service for the analysis of lead in blood, urine, and environmental samples. A public health nurse evaluates the family, refers all preschool children for medical examination, and obtains samples of paint from multiple sites in the home for chemical analysis.[13] Similarly, should the family decide to change dwellings, the new home is also inspected before occupancy.

Medical social worker. He works directly with the parent, helping her deal with the guilt, hostility, and dependency so often

present in this circumstance. The goal is to substitute constructive parental behavior for hopelessness or anger, and this is often possible through a parent's direct participation in the area of improving the dwelling or seeking a suitable new dwelling for her family. In this, and in other activities of social worker and mother, the parent is assisted in functioning to her best ability while she is secure in knowing that the child's safety will definitely be provided for. Hopefully, family behavior can be so modified by external supports and internal strengthening that the deleterious factors operating for continuous pica activity can be alleviated. In some instances, such goals are not realistic and foster home placement or other protective services must be utilized with court approval in order to insure a child's future safety.

Role of child psychologist and psychiatrist. These serve the pediatrician and medical social worker in a consultant capacity. In view of the behavioral aspects of pica, psychiatric advice may be especially needed in instances of severe maternal inadequacy. Psychometric testing early and just prior to entry into school is needed to assess the damage and to facilitate appropriate school placement, especially in survivors of encephalopathy. In this regard, far better communication between the school system and medical facilities is needed than is often practiced.

Role of the pediatrician. It is the pediatrician's function to establish the diagnosis and to assess the severity of intoxication in each case. This, in turn, determines the supportive medical care and selection of chelating agents required.[4, 5] He must determine the nature and extent of residual central nervous system injury sustained so that appropriate therapy and school placement can be selected early. In concert with the others he works to modify the emotional climate which initially promoted pica and he must make certain that hazardous lead exposure is terminated. Most important, however, the pediatrician must accept the final responsibility for seeing that the program of management selected in each case is effectively and continuously implemented.

Role of convalescent hospital. If the maxim of never returning a child with increased body-lead burden to a "leaded" home is to be implemented in good faith, a convalescent facility is vital to provide temporary safe residence for the affected child. This gives the health department, medical social worker, and child guidance personnel the time they need to initiate remedial action. For the child with asymptomatic increased lead absorption, such a facility together with appropriate amelioration of his home environment may prevent the occurrence of overt intoxication. The child recovering from acute encephalopathy often exhibits behavioral aberrations which his limited parents may be unable to cope with at home: A convalescent home provides personnel professionally trained to deal with such children. A convalescent hospital also provides a structured environment in which toddlers may learn orderly patterns of play and diet—a pattern which many children with plumbism have never known. In addition, an active Child Life Program provides the mechanism for terminating the habit of pica. Discharge from the convalescent hospital occurs when a safe new residence is available. Readmission from time to time as indicated can prevent the recurrence of pica during the times of crisis in the family. Baltimore is fortunate in having a community-supported facility such as Happy Hills Hospital which fulfills these protective and reconstructive needs of the child. Periodic follow-up in the outpatient clinic is continued at least until entry into school or longer in damaged children. Throughout these preschool years, the medical social worker and physician encourage the family to enroll the child in nursery school programs and assist the parents of "minimally brain-damaged children" to find the special facilities they need.

PRIMARY PREVENTION OF CHILDHOOD LEAD POISONING

Childhood lead poisoning is a preventable disease. An effective program of prevention has not yet been evolved for the disadvantaged urban child and family. Such a pro-

gram requires two essential ingredients: a clear recognition of the pattern of etiologic factors, and a clear commitment of responsibility by physician, public health worker, city official, and aroused public.

The etiologic pattern which results in irreversible lead encephalopathy consists of a triad: the child, the parent, and the place. The child is a toddler with exaggerated oral activities. The parent is a mother with inadequate resources to cope with her family's needs. The place is a neglected slum housing unit with lead flakes within reach of a small child's grasp.

Each of these three factors is readily visible to the trained professional and should signal appropriate action for early recognition and effective prevention of further risk to that individual child. However, the responsible commitment of these very professionals on behalf of the disadvantaged child and parent is not always forthcoming. The child's health needs are often provided in municipal well baby clinics where nutrition and immunization in the first year of life are stressed above questions of childhood behavior in the second year and above maternal difficulties in housing and child-rearing. For acute illness, the hospital emergency room or pediatric clinic may provide satisfactory symptom-related diagnosis and treatment, but the physician or nurse may remain inattentive to the possibility of pica in the child or the mother's inability to cope with this and other environmental hazards. Moreover, the usual fragmentation of medical and social services in our clinics and hospitals favor ineffective follow-up of already identified high-risk children.

The recent establishment of comprehensive health clinics for disadvantaged children in many cities provides a suitable module for more effective child and parent health care in this area. In high-risk areas, the incorporation of a screening test for excessive lead ingestion into the regular laboratory procedures in such clinics together with the development of interview techniques designed to identify the child with incipient pica offer the best current hope for the prevention of childhood plumbism. These procedures can be carried out by public health nurses both in the clinic and on home visits and should be concentrated on children 12 to 18 months of age and repeated as the family situation warrants.

Since the harmful effects of lead exposure in homes have been public knowledge for many years, many cities have passed local ordinances designed to eradicate such hazards to children. Unfortunately, enforcement often occurs after the poisoning of the child and is not always effective. Too often the disadvantaged family will move from one slum house to another which has not been properly screened for lead exposure. The neighborhoods, where the greatest number of susceptible children coincide with the greatest number of substandard housing units, may receive spasmodic exhortations or visits from local health units. Such spasmodic ex post facto action cannot be called "prevention" and will not suffice.

What families in such neighborhoods require are the imaginative and continuous efforts of health and social action teams directed at parent participation and education. Were such action coupled, as in Australia, with a strenuous public demand for the *systematic* elimination of the environmental lead exposure associated with old dwellings, childhood lead poisoning could be largely eradicated in the United States. Immediately following the birth of an infant of a family residing in a pre-World War II dwelling, the prospective dwelling could be inspected and sampled for lead so that hazardous conditions could be corrected before the infant reaches the age of pica.

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